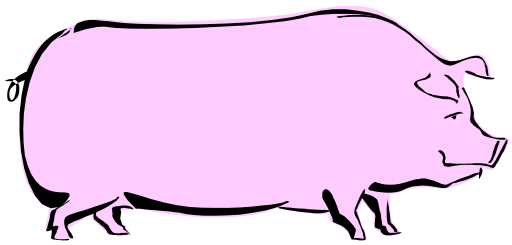
Australian Government

Department of Agriculture, Fisheries and Forestry

Generic Import Risk Analysis (IRA) for Pig Meat

Final Import Risk Analysis Report



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CONTENTS

LIST OF TABLES

LIST OF FIGURES

GLOSSARY OF TERMS AND ABBREVIATIONS

AAHL	Australian Animal Health Laboratory
ABPM	Animal Biosecurity Policy Memorandum
ADV	Aujeszky's disease virus
ALOP	Appropriate level of protection
AQIS	Australian Quarantine and Inspection Service
AQPM	Animal Quarantine Policy Memorandum
AQRC	Australian Quarantine Review Committee
ASF	African swine fever
AUSVETPLAN	Australian Veterinary Emergency Plan
AVA	Australian Veterinary Association
Biosecurity Australia	an operating group within the Commonwealth Department of Agriculture, Fisheries and Forestry
CSF	Classical swine fever
CSIRO	Commonwealth Scientific and Industrial Research Organisation
EEE	Eastern equine encephalomyelitis
EU	European Union
FMD	Foot-and-mouth disease
FSANZ	Food Standards Australia New Zealand
ICON	AQIS Import Conditions database
IPPC	International Plant Protection Convention
IRA	Import risk analysis
NAHIS	National Animal Health Information System
NAQS	Northern Australia Quarantine Strategy
OID	Oral infectious dose
OIE	Office International des Epizooties
OIE Code	OIE International Animal Health Code
PCR	Polymerase chain reaction
PED	Porcine epidemic diarrhoea
PMWS	Post-weaning multisystemic wasting syndrome
PRCV	Porcine respiratory coronavirus
PRRS	Porcine reproductive and respiratory syndrome
SPS	Sanitary and Phytosanitary
SPS Agreement	WTO Agreement on the Application of Sanitary and Phytosanitary Measures
SVD	Swine vesicular disease
TGE	Transmissible gastroenteritis
The Panel	Risk analysis panel
VEE	Venezuelan equine encephalomyelitis
VS	Vesicular stomatitis
WEE	Western equine encephalomyelitis
WTO	World Trade Organization

This *Final Import Risk Analysis (IRA) Report* describes the procedures followed to identify and assess the quarantine risks associated with imports to Australia of pig meat. It presents recommendations in relation to quarantine measures sufficient to ensure that Australia's appropriate level of protection (ALOP) is maintained.

This report contains the following:

- information on the background to this IRA, Australia's framework for quarantine policy and IRAs, the international framework for trade in animals and animal products, and Australia's current policy for importation of pig meat;
- an outline of the methodology and results of hazard identification, risk assessment and risk management;
- quarantine import conditions for pig meat;
- further steps in the IRA process; and
- a summary of stakeholder comments received on the *Technical Issues Paper, Draft Methods Paper* and *Draft IRA Report* and Biosecurity Australia's and the Panel's response.

In accordance with the process established by Biosecurity Australia for conducting IRAs as outlined in the *Import Risk Analysis Handbook* the *Final IRA Report* will be open to appeal for a period of 30 days after its release.

If there are no appeals, appeals are dismissed or once the identified deficiencies arising from any successful appeals are addressed, the recommended policy is submitted to the Director of Animal and Plant Quarantine for determination. Once the Director makes the final determination, the Australian Quarantine and Inspection Service (AQIS) is advised of the new policy and is responsible for its implementation.

Background

This IRA commenced in May 1998. The IRA is 'generic' in that it is not restricted to specific exporting countries; the import conditions recommended as a result of the IRA are applicable to any country provided that they can be met to the satisfaction of Australian authorities. The *Final IRA Report* examines the risks attributed to all disease agents of quarantine concern that may be introduced into Australia through the importation of pig meat.

For this IRA, the definition of 'pig meat' is limited to porcine muscle tissue, blood confined to muscle vasculature, bone and bone marrow, and any other tissues (for example, lymph nodes, skin, nerves) that may be considered inseparable from muscle. *Inter alia*, this approach means that the issues associated with the introduction of disease agents as a result of the importation of 'pig meat products' derived from offal, blood, bone or neurological tissue (such as brain, spinal cord) are not considered.

The IRA provides the basis for response to access requests for pig meat from Brazil, Canada, Chile, European Union (EU) Member States, Hungary, Korea, Mexico, New Zealand, South Africa, Taiwan and the United States of America (USA).

A risk analysis panel (the Panel) was established in 1999. The members are:

Dr David Banks (*Chair*)	General Manager, Animal Biosecurity, Biosecurity Australia
Dr Robyn Martin (*Secretariat*)	Manager, Animal Biosecurity, Biosecurity Australia
Dr Kevin Doyle	Veterinary Director, National Office, Australian Veterinary Association
Dr Ross Cutler	Consultant Specialist Veterinarian
Prof. Colin Wilks	Consultant Microbiologist

The Panel established two technical working groups for porcine reproductive and respiratory syndrome (PRRS) and post-weaning multi-systemic wasting syndrome (PMWS) to assist in its consideration of these diseases.

Current import policy for pig meat

Under current policy, uncanned, uncooked pig meat may be imported from the South Island of New Zealand, Canada and Denmark. Pig meat from Canada and Demark must, however, be imported deboned and be cooked on arrival in Australia in order to address the quarantine risk associated with the potential presence of the disease agent PRRS virus which does not occur in Australia. Pig meat cooked in Canada prior to export is also permitted. Imports of pig meat increased for the 12 months to November 2003 to $192 million. Canada supplies approximately 60 per cent by volume and Denmark 35 per cent, and together these nations account for 95 per cent of pig meat imports, the balance is from New Zealand and canned pig meat imports from various countries.

Pig meat may be imported from any country if the meat is canned (sealed container) and all portions of the contents have been heated to at least 100°C.

Further details of the current import requirements for pig meat are available at the ICON website http://www.aqis.gov.au/icon.

Hazard identification

A *Technical Issues Paper* was released on 8 January 2001 and a public meeting to discuss the paper was held in Canberra on 1 March 2001. The issues paper identified 28 disease agents for further consideration. These were:

- Foot-and-mouth disease virus
- Vesicular stomatitis virus
- African swine fever virus
- Classical swine fever virus
- Rinderpest virus
- Swine vesicular disease virus
- Aujeszky's disease virus
- Porcine reproductive and respiratory syndrome virus
- Transmissible gastroenteritis virus

- Trichinellosis (*Trichinella spiralis*)
- Cysticercosis (*Cysticercus cellulosae*)
- Nipah virus
- Post-weaning multisystemic wasting syndrome
- Salmonellosis (*Salmonella typhimurium* DT104)
- Swine influenza virus
- Porcine brucellosis (*Brucella suis*)
- Porcine epidemic diarrhoea virus
- Porcine respiratory coronavirus
- Rubulavirus (Mexican blue eye disease)
- Eperythrozoonosis (*Eperythrozoon suis*)
- Teschen disease (Enterovirus encephalomyelitis virus)
- Rabies virus
- Bovine tuberculosis (*Mycobacterium bovis*)
- Haemorrhagic septicaemia (*Pasteurella multocida*)
- Japanese encephalitis virus
- Surra (*Trypanosoma evansi*)
- Venezuelan, Eastern and Western equine encephalomyelitis
- Vesicular exanthema virus

Several responses were received on the *Technical Issues Paper*. Stakeholder comments were taken into consideration in preparing the *Draft* and *Final IRA Reports*.

Subsequently, it was decided not to consider two diseases. These were Eperythrozoonosis (*Eperythrozoon suis*) and vesicular exanthema virus. The first has been diagnosed in Australia and the second is no longer present in any country. The *Final IRA Report* recommends that exporting countries certify country freedom for vesicular exanthema. Accordingly 26 disease agents were identified of quarantine concern and were the focus of individual risk assessments.

Method for Import Risk Analysis

On 1 October 2002, Biosecurity Australia released a *Draft Methods Paper* that set out the approach to the method for undertaking the risk analysis. It outlined the release and exposure pathways, and the outbreak scenarios considered to be of importance in assessing the risk associated with importation of pig meat. The paper identified the major exposure pathways for disease introduction through waste from households and waste from food service establishments. Four groups of animals that may be directly exposed to uncooked pig meat scraps were identified and included feral pigs, backyard pigs, pigs in small commercial enterprises and susceptible species that will eat meat, i.e. dogs, cats and rodents. The IRA also examines the consequences of spread to large commercial piggeries and other animals such as horses and cattle although this is not considered a pathway for direct exposure. This IRA does not directly examine the public health risks to humans associated with the direct consumption of imported pig meat. Products intended for human consumption may undergo a separate risk assessment by Food Standards Australia New Zealand (FSANZ). The Australian Government Department of Health and Ageing has been consulted on the assessments for zoonotic pests or

diseases that may establish in Australia's animal population through the importation of pig meat.

Several stakeholders commented on the *Draft Methods Paper*. Those submissions were also considered in preparing the *Draft* and *Final IRA Reports*.

Draft Import Risk Analysis Report

The *Draft IRA Report* was released on 12 August 2003 and three public meetings were held (Bendigo, Young, Toowoomba) to discuss the paper during the 60 day comment period. At those meetings the requirements for PMWS related to processing were clarified to the effect that processing could take place on-shore under quarantine control or off-shore. Several responses were received on the *Draft IRA Report* and these comments were taken into account in preparing the *Final IRA Report*.

Assessment and management of risk

Risk management describes the process of identifying and implementing measures to mitigate risks so as to achieve Australia's ALOP, while ensuring that any negative effects on trade are minimised.

The unrestricted risk[1] of entry, establishment and/or spread was assessed for each disease agent of quarantine concern. In relation to the following disease agents the unrestricted risk of entry, establishment and/or spread was assessed as being too high to meet Australia's ALOP:

- Foot-and-mouth disease virus
- African swine fever virus
- Classical swine fever virus
- Rinderpest virus
- Swine vesicular disease virus
- Aujeszky's disease virus
- Porcine reproductive and respiratory syndrome virus
- Trichinellosis (*Trichinella spiralis*)
- Nipah virus
- Post-weaning multisystemic wasting syndrome

For all other disease agents, the unrestricted risk was assessed as being sufficiently low to meet Australia's ALOP.

In the case of *Trichinella spiralis*, Nipah virus, *Salmonella typhimurium* DT104 and *Brucella suis* the Australian Government Department of Health and Ageing has advised Biosecurity Australia that risk management measures would be required to address human health concerns which would arise should these diseases enter and establish or spread in the Australian animal population.

[1] Unrestricted risk estimates are those derived in the absence of specific risk management measures, or using only internationally accepted baseline risk management strategies. In contrast, restricted or mitigated risk estimates are those derived when 'risk management' is applied. In the case of this *Final IRA Report,* unrestricted risk is the risk associated with pig meat produced according to the relevant Australian Standards, in particular Australia's domestic requirements for ante-mortem, slaughter and post-mortem procedures for the production of meat for human consumption.

Summary of risk management measures

Foot-and-mouth disease virus

Country or zone freedom without vaccination, or canning of pig meat such that all portions have been heated to at least 100°C (shelf stable), would meet Australia's ALOP.

African swine fever (ASF) virus

Processing of pig meat by dry curing under specified conditions for Parma type hams (minimum curing time 399 days), Iberian type hams, loins or shoulders and Serrano type hams (minimum curing time 140 days), together with certification that the pigs had been sourced from premises which had been free from evidence of ASF infection for the 3 months prior to slaughter would reduce the risk of entry, establishment and/or spread of ASF virus to very low, which would meet Australia's ALOP.

Country or zone freedom, or canning of pig meat such that all portions have been heated to at least 100°C (shelf stable), would also meet Australia's ALOP.

Classical swine fever (CSF) virus

Processing of pig meat by dry curing under specified conditions for Parma type hams (minimum curing time 313 days), Iberian type hams, loins or shoulders and Serrano type hams (minimum curing time 252 days), together with certification that the pigs had been sourced from premises which had been free from evidence of CSF infection for the 3 months prior to slaughter would reduce the risk of entry, establishment and/or spread of CSF virus to very low, which would meet Australia's ALOP.

Country or zone freedom, or canning of pig meat such that all portions have been heated to at least 100°C (shelf stable), would also meet Australia's ALOP.

Rinderpest virus

Country or zone freedom, or canning of pig meat such that all portions have been heated to at least 100°C (shelf stable), would meet Australia's ALOP.

Swine vesicular disease (SVD) virus

Processing of pig meat by dry curing under specified conditions for Parma type hams (minimum curing time 360 days), together with certification that the pigs from which the meat was derived were sourced from herds serologically tested negative using either virus neutralisation or ELISA within the 6 months prior to slaughter and within the 6 months following slaughter would reduce the risk of entry, establishment and/or spread of SVD virus to very low, which would meet Australia's ALOP

Country or zone freedom, or canning of pig meat such that all portions have been heated to at least 100°C (shelf stable), would meet Australia's ALOP.

Aujeszky's disease virus

Removing the head and neck from the carcass would reduce the risk of entry, establishment and/or spread of Aujeszky's disease virus to very low, which would meet Australia's ALOP.

Deboning and processing (cooking or curing) of pig meat would reduce the risk of entry, establishment and/or spread of Aujeszky's disease to negligible, which would meet Australia's ALOP.

Country or zone freedom or canning of pig meat such that all portions have been heated to at least 100°C (shelf stable), would also meet Australia's ALOP.

Porcine reproductive and respiratory syndrome (PRRS) virus

Cooking of pig meat with or without bone to a minimum core temperature of 70°C for 11 minutes or dry curing pig meat under specified conditions for Parma type hams (minimum curing time 313 days), Iberian type hams, loins or shoulders and Serrano type hams (minimum curing time 140 days) would reduce the risk of entry, establishment and/or spread of PRRS virus to very low, which would meet Australia's ALOP. Imported pig meat may be cooked off-shore or in Australia on-shore provided that the latter occurs within the urban area of the port into which it is imported or if in a rural area is transported under appropriate secure arrangements (e.g. refrigerated container) by the most direct route from the nearest port of entry.

Country or zone freedom or canning of pig meat such that all portions have been heated to at least 100°C (shelf stable) would also meet Australia's ALOP.

Trichinella spiralis

Testing each carcass for *Trichinella* larvae, or processing of pig meat by cooking or freezing at temperatures to destroy larvae, or dry curing of pig meat under specified conditions for Parma type hams (minimum curing time 313 days), Iberian type hams, loins or shoulders and Serrano type hams (minimum curing time 140 days) would reduce the risk of entry, establishment and/or spread of *Trichinella spiralis* to very low (testing) or negligible (processing), which would meet Australia's ALOP.

Country or zone freedom in domestic pigs, or canning of pig meat such that all portions have been heated to at least 100°C (shelf stable), would also meet Australia's ALOP.

The Department of Health and Ageing has advised Biosecurity Australia that biosecurity measures would be required to manage the risk to human health associated with the importation of pig meat should the disease enter and establish or spread in the Australian animal population. Appropriate measures would include testing of carcasses or processing (cooking, curing, freezing), or herd or zone freedom.

Nipah virus

Country or zone freedom in domestic pigs, or canning of pig meat such that all portions have been heated to at least 100°C (shelf stable), would meet Australia's ALOP.

The Department of Health and Ageing has advised Biosecurity Australia that biosecurity measures would be required to manage the risk to human health associated with the importation of pig meat should the disease enter and establish or spread in the Australian animal population. Appropriate measures for a country or zone which has reported Nipah virus would include certification that the pigs from which the pig meat was derived originate from a herd which has been tested negative for the disease agent or canning of pig meat such that all portions have been heated to at least 100°C (shelf stable).

Post-weaning multisystemic wasting syndrome (PMWS)

Removing the head and neck and major peripheral lymph nodes and deboning, together with processing of pig meat (cooking or curing), would reduce the risk of entry, establishment and/or spread of PMWS to very low, which would meet Australia's ALOP. Imported pig meat may be cooked off-shore or in Australia on-shore provided that the latter occurs within the urban area of the port into which it is imported or if in a rural area is transported under appropriate secure arrangements (e.g. refrigerated container) by the most direct route from the nearest port of entry. Removal of the head and neck, major peripheral lymph nodes and bone must occur prior to export of pig meat to Australia for processing.

Country or zone freedom or canning of pig meat such that all portions have been heated to at least 100°C (shelf stable) would also meet Australia's ALOP.

Salmonella typhimurium DT104

The Department of Health and Ageing has advised Biosecurity Australia that biosecurity measures would be required to manage the risk to human health associated with the importation of pig meat should the disease enter and establish or spread in the Australian animal population. Appropriate measures would include compliance with the Food Standards Code including testing for *Salmonella*.

Brucella suis

The Department of Health and Ageing has advised Biosecurity Australia that biosecurity measures would be required to manage the risk to human health associated with the importation of pig meat should the disease enter and establish or spread in the Australian domestic animal population. Appropriate measures for countries where *B. suis* is endemic, in the case of uncooked pig meat (not subject to further processing in Australia, prior to retail sale), would be to require that the pigs from which the meat is derived be sourced from herds which have been tested negative, or are accredited free from *B. suis*.

Conclusion

This *Final IRA Report* recommends that import of pig meat be permitted subject to certain conditions depending on the health status of the exporting country or zone. Risk management measures include such things as country or zone freedom, testing of the carcass, cooking, freezing, curing, canning and removal of certain tissues or parts of the carcass (removal of the head and neck, major peripheral lymph nodes, deboning). Biosecurity Australia will consider other measures suggested by stakeholders that provide an equivalent level of quarantine protection.

INTRODUCTION

This section outlines:

- The legislative basis for Australia's biosecurity regime
- Australia's international rights and obligations
- Australia's Appropriate Level of Protection and risk management
- Import risk analysis
- Policy determination

AUSTRALIAN LEGISLATION

The *Quarantine Act 1908* and its subordinate legislation, including the *Quarantine Proclamation 1998*, are the legislative basis of human, animal and plant biosecurity in Australia.

Some key provisions are set out below.

Quarantine Act: Scope

Sub section 4 (1) of the *Quarantine Act 1908* defines the scope of quarantine as follows.

> *In this Act, quarantine includes, but is not limited to, measures:*
>
> *(a) for, or in relation to:*
>
> *(i) the examination, exclusion, detention, observation, segregation, isolation, protection, treatment and regulation of vessels, installations, human beings, animals, plants or other goods or things; or*
>
> *(ii) the seizure and destruction of animals, plants, or other goods or things; or*
>
> *(iii) the destruction of premises comprising buildings or other structures when treatment of these premises is not practicable; and*
>
> *(b) having as their object the prevention or control of the introduction, establishment or spread of diseases or pests that will or could cause significant damage to human beings, animals, plants, other aspects of the environment or economic activities.*

Section 5D of the *Quarantine Act 1908* covers the level of quarantine risk.

> *A reference in this Act to a level of quarantine risk is a reference to:*
>
> *(a) the probability of:*
>
> *(i) a disease or pest being introduced, established or spread in Australia or the Cocos Islands; and*
>
> *(ii) the disease or pest causing harm to human beings, animals, plants, other aspects of the environment, or economic activities; and*
>
> *(b) the probable extent of the harm.*

Section 5D of the *Quarantine Act 1908* includes harm to the environment as a component of the level of quarantine risk.

Environment is defined in Section 5 of the *Quarantine Act 1908,* in that it:

> *includes all aspects of the surroundings of human beings, whether natural surroundings or surroundings created by human beings themselves, and whether affecting them as individuals or in social groupings.*

Quarantine Proclamation

The *Quarantine Proclamation 1998* is made under the under the *Quarantine Act 1908*. It is the principal legal instrument used to control the importation into Australia of goods of quarantine (or biosecurity) interest. The Proclamation empowers a Director of Quarantine to grant a permit to import.

Section 70 of the *Quarantine Proclamation 1998* sets out the matters to be considered when deciding whether to grant a permit to import:

> *Things a Director of Quarantine must take into account when deciding whether to grant a permit for importation into Australia*
>
> *(1) In deciding whether to grant a permit to import a thing into Australia or the Cocos Islands, or for the removal of a thing from the Protected Zone or the Torres Strait Special Quarantine Zone to the rest of Australia, a Director of Quarantine:*
>
> *(a) must consider the level of quarantine risk if the permit were granted; and*
>
> *(b) must consider whether, if the permit were granted, the imposition of conditions on it would be necessary to limit the level of quarantine risk to one that is acceptably low; and*
>
> *(ba) for a permit to import a seed of a kind of plant that was produced by genetic manipulation -- must take into account any risk assessment prepared, and any decision made, in relation to the seed under the Gene Technology Act; and*
>
> *(c) may take into account anything else that he or she knows that is relevant.*

Development of Biosecurity Policy

As can be seen from the above extracts, the legislation establishes the concept of the level of biosecurity (quarantine) risk as the basis of decision-making under Australian quarantine legislation.

Import risk analyses are a significant contribution to the information available to the Director of Animal and Plant Quarantine - a decision maker for the purposes of the Quarantine Proclamation. Import risk analysis is conducted within an administrative process - known as the IRA process (described in the *IRA Handbook*[2]).

The purpose of the IRA process is to deliver a policy recommendation to the Director of Animal and Plant Quarantine that is characterised by sound science and by transparency, fairness and consistency. The key elements of the IRA process are covered in "Import Risk Analysis" below.

[2] Agriculture, Fisheries and Forestry - Australia (2003) *Import Risk Analysis Handbook*, Canberra.

AUSTRALIA'S INTERNATIONAL RIGHTS AND OBLIGATIONS

It is important that import risk analysis conforms with Australia's rights and obligations as a World Trade Organization (WTO) Member country. These rights and obligations derive principally from the World Trade Organization's *Agreement on the Application of Sanitary and Phytosanitary Measures* (SPS Agreement), although other WTO agreements may also be relevant. Specific international guidelines on risk analysis developed under the International Plant Protection Convention (IPPC) and by the Office International des Epizooties (OIE) are also relevant.

The SPS Agreement recognises the right of WTO Member countries to determine the level of sanitary and phytosanitary protection they deem appropriate, and to take the necessary measures to achieve that protection. Sanitary (human and animal health) and phytosanitary (plant health) measures typically apply to trade in or movement of animal and plant based goods within or between countries. The SPS Agreement applies to measures that may directly or indirectly affect international trade and that protect human, animal or plant life or health from pests and diseases or a Member's territory from a pest.

The SPS Agreement provides for the following:

- The right of WTO Member countries to determine the level of sanitary and phytosanitary protection (its appropriate level of protection, or ALOP) they deem appropriate;
- An importing Member has the sovereign right to take measures to achieve the level of protection it deems appropriate to protect human, animal or plant life or health within its territory;
- An SPS measure must be based on scientific principles and not be maintained without sufficient scientific evidence;
- An importing Member shall avoid arbitrary or unjustifiable distinctions in levels of protection, if such distinctions result in discrimination or a disguised restriction on international trade;
- An SPS measure must not be more trade restrictive than required to achieve an importing Member's ALOP, taking into account technical and economic feasibility;
- An SPS measure should be based on an international standard, guideline or recommendation where these exist, unless there is a scientific justification for a measure which results in a higher level of SPS protection to meet the importing Member's ALOP;
- An SPS measure conforming to an international standard, guideline or recommendation is deemed to be necessary to protect human, animal or plant life or health, and to be consistent with the SPS Agreement;
- Where an international standard, guideline or recommendation does not exist or where, in order to meet an importing Member's ALOP, a measure needs to provide a higher level of protection than accorded by the relevant international standard, such a measure must be based on a risk assessment; the risk assessment must take into account available scientific evidence and relevant economic factors;
- Where the relevant scientific evidence is insufficient, an importing Member may provisionally adopt SPS measures on the basis of available pertinent information. In such circumstances, Members shall seek to obtain the additional information necessary for a more objective assessment of risk and review the SPS measure accordingly within a reasonable period of time;

- An importing Member shall accept the measures of other countries as equivalent, if it is objectively demonstrated that the measures meet the importing Member's ALOP.

AUSTRALIA'S APPROPRIATE LEVEL OF PROTECTION (ALOP)

The SPS Agreement defines the concept of an 'appropriate level of sanitary or phytosanitary protection (ALOP)' as the level of protection deemed appropriate by the WTO Member establishing a sanitary or phytosanitary measure to protect human, animal or plant life or health within its territory.

Like many other countries, Australia expresses its ALOP in qualitative terms. Australia's ALOP, which reflects community expectations through government policy, is currently expressed as providing a high level of sanitary or phytosanitary protection aimed at reducing risk to a very low level, but not to zero.

ALOP can be illustrated using a 'risk estimation matrix' Table 1. The cells of this matrix describe the product of likelihood[3] and consequences — termed 'risk'. When interpreting the risk estimation matrix, it should be remembered that, although the descriptors for each axis are similar ('low', 'moderate', 'high' etc), the vertical axis refers to *likelihood* and the horizontal axis refers to *consequences*.

Table 1 Risk estimation matrix

Likelihood of entry and exposure	Negligible impact	Very low	Low	Moderate	High	Extreme impact
High likelihood	Negligible risk	Very low risk	Low risk	Moderate risk	High risk	Extreme risk
Moderate	Negligible risk	Very low risk	Low risk	Moderate risk	High risk	Extreme risk
Low	Negligible risk	Negligible risk	Very low risk	Low risk	Moderate risk	High risk
Very low	Negligible risk	Negligible risk	Negligible risk	Very low risk	Low risk	Moderate risk
Extremely low	Negligible risk	Negligible risk	Negligible risk	Negligible risk	Very low risk	Low risk
Negligible likelihood	Negligible risk	Negligible risk	Negligible risk	Negligible risk	Negligible risk	Very low risk

Consequences of entry, establishment or spread

The band of cells in Table 1 marked 'very low risk' represents Australia's ALOP, or tolerance of loss.

Risk Management and SPS Measures

Australia's plant and animal health status is maintained through the implementation of measures to facilitate the importation of products while protecting the health of people, animals and plants.

[3] The terms "likelihood" and "probability" are synonymous. "Probability" is used in the *Quarantine Act 1908* while "likelihood" is used in the WTO SPS Agreement. These terms are used interchangeably in this IRA Report.

Australia bases its national measures on international standards where they exist and where they deliver the appropriate level of protection from pests and diseases. However, where such standards do not achieve Australia's level of biosecurity protection, or relevant standards do not exist, Australia exercises its right under the SPS Agreement to take appropriate measures, justified on scientific grounds and supported by risk analysis.

Australia's approach to addressing requests for imports of animals, plants and their products, where there are biosecurity risks, is, where appropriate, to draw on existing sanitary and phytosanitary measures for similar products with comparable risks. However, where measures for comparable biosecurity risks have not previously been established, further action would be required to assess the risks to Australia and determine the sanitary and phytosanitary measures needed to achieve Australia's ALOP.

IMPORT RISK ANALYSIS

Description

In animal and plant biosecurity, import risk analysis identifies the pests and diseases relevant to an import proposal, assesses the risks posed by them and, if those risks are unacceptable, specifies the measures that could be taken to reduce those risks to an acceptable level. These analyses are conducted via an administrative process (described in the *IRA Handbook*) that involves, among other things, notification to the WTO, consultation and appeal.

Undertaking IRAs

Biosecurity Australia may undertake an IRA if:

- there is no relevant existing biosecurity measure for the good and pest/disease combination; or
- a variation in established policy is desirable because pests or diseases, or the likelihood and/or consequences of entry, establishment or spread of the pests or diseases could differ significantly from those previously assessed.

Environment and human health

When undertaking an import risk analysis, Biosecurity Australia takes into account harm to the environment as part of its assessment of biosecurity risks associated with the potential import.

Under the *Environment Protection and Biodiversity Conservation Act 1999*, the Australian Government Department of Environment and Heritage may assess proposals for the importation of live specimens and their reproductive material. Such an assessment may be used or referred to by Biosecurity Australia in its analyses.

Biosecurity Australia also consults with other Commonwealth agencies where they have responsibilities relevant to the subject matter of the IRA, e.g. Food Standards Australia New Zealand (FSANZ) and the Australian Government Department of Health and Ageing.

The IRA Process in summary

The process consists of the following major steps:

***Initiation*:** This is the stage where the identified need for an IRA originates.

***Scheduling and Scoping*:** At this stage, Biosecurity Australia considers all the factors that affect scheduling. Consultation with States, Territories and other Commonwealth agencies is involved. There is opportunity for appeal by stakeholders at this stage.

***Risk Assessment and Risk Management*:** Here, the major scientific and technical work relating to risk assessment is performed. There is detailed consultation with stakeholders.

***Reporting*:** Here, the results of the IRA are communicated formally. There is consultation with States and Territories. The Executive Manager of Biosecurity Australia then delivers the biosecurity policy recommendation arising from the IRA to the Director of Animal and Plant Quarantine. There is opportunity for appeal by stakeholders at this stage.

POLICY DETERMINATION

The Director of Animal and Plant Quarantine makes the policy determination, which is notified publicly.

BACKGROUND

The IRA commenced in May 1998, with Animal Quarantine Policy Memorandum (AQPM) 1998/45 entitled "*Import Risk Analysis: Pig Meat - Consultation on Approach*". The AQPM proposed a 'non-routine'[4] approach for the IRA. At that time, Australia had received requests to develop importation protocols for pig meat sourced from Canada, European Union (EU) Member States, Hungary, Korea, Mexico, New Zealand, South Africa, Taiwan, and the United States of America. Access requests have since been received from Brazil, Chile, Sweden and Finland.

Risk analysis panel

A risk analysis panel (the Panel) was established in 1999.

The risk analysis panel membership is:

Dr David Banks (*Chair*)	General Manager, Animal Biosecurity, Biosecurity Australia
Dr Robyn Martin (*Secretariat*)	Manager, Animal Biosecurity, Biosecurity Australia
Dr Kevin Doyle	Veterinary Director, National Office, Australian Veterinary Association
Dr Ross Cutler	Consultant Specialist Veterinarian
Prof. Colin Wilks	Consultant Microbiologist

Technical working group(s)

The Panel established the following technical working groups on porcine reproductive and respiratory syndrome (PRRS) and post-weaning multisystemic wasting syndrome (PMWS).

The technical working group for PRRS is:

Dr Geoff Gard *(Chair)*	Consultant Virologist
Dr Robyn Martin	Manager, Animal Biosecurity, Biosecurity Australia
Dr Chris Baldock	Consultant Epidemiologist
Dr Tony Forman	Consultant Virologist

All members of the Panel are members of the technical working group for PRRS.

[4] A 'non-routine' analysis is conducted by a risk analysis panel comprising scientific experts from Biosecurity Australia and other organisations who have expertise in quarantine risk assessment and disease agents relevant to the IRA.

The technical working group for PMWS is:

Dr Robyn Martin *(Chair)*	Manager, Animal Biosecurity, Biosecurity Australia
Dr Geoff Gard	Consultant Virologist
Dr Chris Baldock	Consultant Epidemiologist
Dr Tony Forman	Consultant Virologist
Dr Russell Rogers	Principal Veterinary Officer, Queensland Department of Primary Industries

The Panel first met on 25 February 1999. AQPM 1999/21 set out details of the proposed work program, and foreshadowed the release of the *Technical Issues Paper*. The *Technical Issues Paper* was released on 8 January 2001 under cover of Animal Biosecurity Policy Memorandum (ABPM) 2001/02. A public meeting to discuss the paper was held in Canberra on 1 March 2001. Several responses were received on the *Technical Issues Paper*. The risk analysis panel has considered the submissions - the submissions and the Panel's responses are at Annex A. Stakeholder comments were taken into consideration in preparing the *Draft* and *Final IRA Reports*.

At the request of the technical working group and the Panel, Biosecurity Australia commissioned Lelystad Id-dlo in the Netherlands to conduct research on the oral transmission of PRRS by feeding infected meat to pigs. The report of the results was sent to stakeholders in September 2001 (ABPM2001/25). The Panel considered further research on PRRS was warranted and Lelystad is now conducting that work.

On 1 October 2002, Biosecurity Australia released the *Draft Methods Paper* (ABPM 2002/45) that set out the approach to the method for undertaking the risk analysis. It outlined the release and exposure pathways, and the outbreak scenarios considered to be of importance in assessing the risk associated with importation of pig meat. Several stakeholders commented on the paper. Those submissions were considered in preparing the *Draft and Final IRA Reports* (Annex B).

The *Draft IRA Report* was released on 12 August 2003 (ABPM 2003/19) and combined the information provided in the *Technical Issues Paper* and included the method, the risk assessments, the proposed risk management measures and the draft import conditions. Several stakeholders commented on the paper. Those submissions were considered in preparing the *Final IRA Report* (Annex C).

This *Final IRA Report* combines the information from the above reports after taking into consideration stakeholder comments and includes quarantine requirements for the importation of pig meat.

ADMINISTRATION

Timetable

The "Further Steps in the Import Risk Analysis Process" section later in this document lists the steps to complete this IRA.

Scope

The IRA of pig meat is 'generic' in that it is not restricted to specific exporting countries, and that import conditions are applicable to any exporting country.

The *Final IRA Report* examines the risks attributed to all disease agents of quarantine concern that may be introduced into Australia through the importation of pig meat.

For this IRA, the definition of 'pig meat' is limited to porcine muscle tissue, blood confined to muscle vasculature, bone and bone marrow, and any other tissues (for example, lymph nodes, skin, nerves) that may be considered inseparable from muscle. *Inter alia*, this approach means that the issues associated with the introduction of disease agents as a result of the importation of 'pig meat products' derived from offal, blood, bone or neurological tissue (such as brain, spinal cord), will not be considered.

AUSTRALIA'S CURRENT QUARANTINE POLICY FOR IMPORTS OF PIG MEAT

Australia's quarantine policy

Under current policy uncanned, uncooked pig meat may be imported from the South Island of New Zealand, Canada and Denmark. Pig meat from Canada and Demark must, however, be imported deboned and be cooked on arrival in Australia in order to address the quarantine risk associated with the potential presence of the disease agent porcine reproductive and respiratory syndrome (PRRS) virus which does not occur in Australia or New Zealand. Pig meat cooked in Canada prior to export is also permitted.

Pig meat may be imported from any country providing the meat is canned and heated to a minimum internal temperature of 100°C (sealed container).

Further details of the import requirements for pig meat are available at the ICON website http://www.aqis.gov.au/icon.

Domestic arrangements

While the Commonwealth Government is responsible for regulating the movement of animals and their products into and out of Australia, State/Territory Governments have primary responsibility for animal health controls within their State or Territory. Legislation relating to resource management or animal health may be used by State/Territory Government agencies to control interstate movement of animals and their products.

THE PIG MEAT INDUSTRY IN AUSTRALIA

Production

The Australian pig industry produces around five and a half million pigs per year, largely supplying the Australian domestic market for pig meat. The industry comprises approximately 2,600 pig farmers and 332,000 sows. Whilst pig production occurs in all States and Territories except the ACT, of the total sows, NSW has 30%, Queensland 22%, Victoria 21%, South Australia 15% and Western Australia 11%.

By world standards the Australian pork industry is quite small, producing 0.4% of world pork production and accounting for 1.4% of world exports. Domestically, however, it is a significant industry and employer in regional Australia, that has over the past five years established significant export markets accounting for around 20 per cent of production.

Previously, Australian piggeries were often operated in association with grain production and most were family operated farms. Over the last decade, the number of farms has steadily declined and the number of extensive pig keeping systems has slowly expanded. The number of producers has fallen from almost 7,000 in 1990 to a little over 2,600. Many of the larger establishments are vertically integrated companies and the largest 2% of farms account for 40% of the sow population.

ABARE estimates the gross value of pig production in 2002-03 at $892 million, down slightly on the 2001-2002 of $968 million. According to the Western Research Institute, taking into account related industries, the pork industry provides 33,863 jobs.

Per capita pork consumption in Australia has increased in recent years to 21.46 kg/head[5]. Future growth will be as a result of an increase in market share at the expense of other meat.

The supply of pig meat is influenced by climatic conditions (drought and grain availability) and the value of the Australian dollar.

Exports

Currently exports account for around 16% of total pig meat production with total exports valued at around $230 million. Japan and Singapore are the main markets, with exports to Japan steadily increasing in recent years. In March 2003, farmed exports were a record, up 16% from the previous 12 months. The recent appreciation of the $A has impacted on the export market, with growth in returns from the Japanese market levelling off and volumes and values in the Singapore market falling consistently since December 2002. Industry focuses on niche markets based on Australia's favourable health status, proximity to markets and the ability to supply fresh chilled product. Priority is given to the higher valued export markets at the expense of domestic market with imports meeting the shortfall. The related industries have also changed in response to the export focus with the emergence of new export abattoirs and processors.

Imports

During 2002, imports of pig meat increased to new record levels ($211 million for the 12 months to August 2002). This was due to Australian producers/processors filling increasing demand from new export markets. Imports then decreased in late 2002 as Australian production capacity increased. Imports increased further during 2003 (52,937 tonnes valued at $192 million for the 12 months to November 2003) due to the drought limiting Australian production increases and the need to satisfy higher-valued export demand. The appreciation of the Australian dollar and an excess supply of Canadian pork is also encouraging imports.

Canada supplies 60 per cent by volume and Denmark 35 per cent, and together they account for 95 per cent of pig meat imports, the remainder coming from New Zealand and canned product which may come from any country subject to certain conditions.

[5] Ms Kathleen Plowman, Australian Pork Limited, submission on draft IRA report of pig meat.

OTHER PIGS IN AUSTRALIA

Australia has a significant wild population of pigs. Wild pigs inhabit approximately 38% of the continent with a total population fluctuating between approximately 3.5 and 23.5 million depending on seasonal conditions.

Wild pigs may act as hosts or vectors for many endemic diseases and potentially for exotic diseases. Diseases reported in localised sub-populations of wild pigs include brucellosis (*Brucella suis*), leptospirosis (*Leptospira* spp.), melioidosis (*Burkholderia pseudomallei*), tuberculosis (*Mycobacterium avium*), sparganosis (*Spirometra erinacei*), porcine parvovirus, toxoplasmosis, and Murray Valley encephalitis and other arboviruses.

Few pigs are kept as pets in Australia, partly due to local government laws. All pet pigs in Australia are derived from domestic swine.

Laboratory pigs have been imported but remain under quarantine control in the laboratory.

PIG HEALTH IN AUSTRALIA

As a result of geographical isolation and the application of sound quarantine procedures for imported livestock, genetic material and animal products, Australia remains free of the major epidemic diseases of livestock and many of the serious diseases of swine. African swine fever, Aujeszky's disease, classical swine fever and foot-and-mouth disease, porcine reproductive and respiratory syndrome, post-weaning multisystemic wasting syndrome, swine influenza and transmissible gastroenteritis do not occur in Australia. Australia is also free of many of the less significant or less widely distributed diseases of pigs, such as rubula virus and porcine epidemic diarrhoea.

Animal health surveillance

The Australian National Animal Health Information System (NAHIS), based on routine monitoring of selected diseases and supplemented by specific studies and surveys, has operated since 1993. NAHIS provides summary information on animal diseases and their importance in Australia, livestock numbers, slaughter statistics, and other related information. Sources of data for NAHIS include Commonwealth, State and Territory animal health authorities, diagnostic laboratories, eradication or control programs, herd monitoring systems, universities, research programs and veterinary practices.

In 1996, following negotiations with Canada regarding Australia's status with respect to porcine reproductive and respiratory syndrome (PRRS), a national serological survey was undertaken in order to confirm Australia's freedom from this disease. The results of this study supported the view that Australian domestic pigs are free of PRRS virus.

A preliminary survey of a limited number of domestic pigs has demonstrated the presence of porcine circovirus type 1 and porcine circovirus type 2 strains (97% homology to French and Canadian strains). The disease post-weaning multisystemic wasting syndrome (PMWS), in which porcine circovirus type 2 is considered an essential factor, is not present in Australia. Surveillance for this disease is currently being undertaken.

Under the Office International des Epizooties (OIE) Code, import risk analyses (IRAs) for animals and animal products are based on the following procedures:

- hazard identification
- risk assessment, incorporating:
  - release assessment
  - exposure assessment
  - consequence assessment
  - risk estimation
- risk management
- risk communication

METHOD FOR HAZARD IDENTIFICATION

Hazard identification, as documented in the *Technical Issues Paper*,[6] was carried out in two stages:

- Identification of a preliminary index of agents/diseases relevant to the importation of pigs or pig-derived products
- Refinement of the preliminary index in accordance with specified hazard identification criteria (hazard refinement)

A preliminary index of diseases/agents was derived by combining the relevant OIE List A and B diseases with unlisted diseases of swine considered by the risk analysis panel (the Panel) to be of potential quarantine concern.[7]

Hazard refinement was carried out in accordance with the criteria set out below. Where definitive data relevant to categorisation were lacking, the Panel made judgements that drew on scientific knowledge and observations from similar situations, and any other appropriate information.

- *The pathogenic agent is infectious*: the putative pathogenic agent must cause, or be causally associated with, a recognised disease and the disease must have been shown to have an infectious aetiology;
- *The pathogenic agent must have been found in association with pigs*: the pathogenic agent must be transmissible to susceptible hosts and may have been isolated. Ideally Koch's or Evans' (Thrusfield, 1995) postulates have been satisfied. This excludes diseases caused by environmental (for example, toxicosis), genetic or nutritional factors;
- *The pathogenic agent is exotic to Australia*: the pathogenic agent is considered to be exotic if there is no report of the disease or detection of the causal agent in animals in Australia. The level of confidence that can be attributed to such a determination depends on factors such as the virulence of the organism, severity of expression of clinical disease and nature of targeted surveillance applied to the disease/agent in question. Where a pathogenic agent

[6] Available at: http://www.daff.gov.au/

[7] Pathogenic agents not listed by OIE but relevant to this IRA were identified by the Panel or stakeholders, or by those within the Department of Agriculture Fisheries and Forestry.

is present in Australia, but the strain(s) present in other countries is/are significantly more virulent, these strains will be considered to be exotic to Australia and thus meet this criterion;

- *The pathogenic agent is present in Australia but subject to official control*: if a pathogenic agent or disease occurs in Australia, then either; (a) one or more State/Territory Government(s) must have enacted legislation and be taking action to control or eradicate the disease/agent, or, (b) control of the disease/agent must be the object of a mandatory industry-based control program
- *The pathogenic agent is listed by OIE*: the pathogenic agent causes a notifiable or other significant disease as listed by OIE;
- *The pathogenic agent would be expected to cause significant disease in Australia*: the pathogenic agent must satisfy one or more of the following criteria:
  - it would be expected to cause significant disease;
  - it would be expected to cause significant damage to the environment and/or native species; and/or
  - it would be expected to cause significant economic harm, for example, increased mortality, reduced growth rates, decreased product quality, loss of market access, increased management costs.

In summary, a pathogenic agent was given detailed consideration in the IRA if it was:

Infectious, *and* either,

exotic to Australia, *or*,

present in Australia but subject to official control, *and* either,

OIE listed, *and/or*,

likely to cause significant disease in Australia

METHOD FOR RISK ASSESSMENT

Risk assessment is defined in the OIE Code as:

> *… an evaluation of the likelihood and the biological and economic consequences of entry, establishment or spread of a pathogenic agent within the territory of an importing country.*

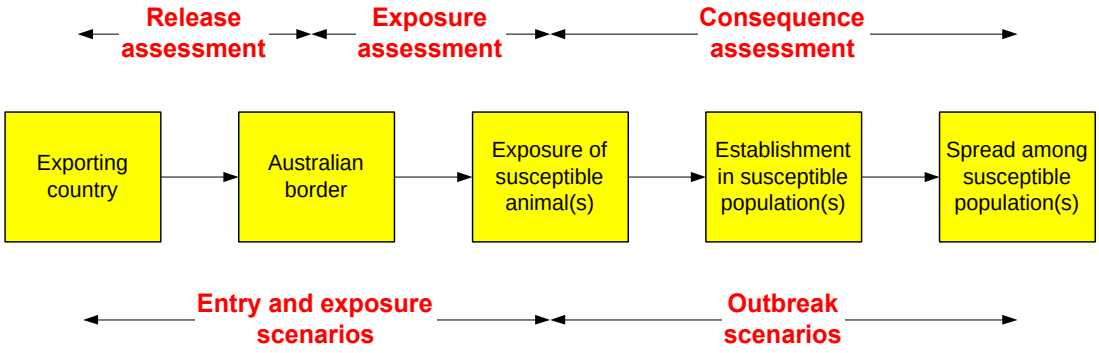
The likelihood that a pathogenic agent will enter an importing country and the likelihood that susceptible animals will be exposed to that agent were determined through a ‘release assessment’ and an ‘exposure assessment’, respectively.

The likelihood of establishment and/or spread and the biological and economic consequences of introducing a pathogenic agent were determined through a ‘consequence assessment’.

The risk assessment for each identified agent concluded with ‘risk estimation’, the combination of the likelihoods and consequences, and yielded the unrestricted risk estimate.

These steps are illustrated diagrammatically in Figure 1.

Figure 1 Components of a risk assessment



The principle of a 'generic' risk assessment

This IRA has been termed 'generic', in that the risks associated with the importation of uncooked pig meat from *any* exporting country were considered. In order to carry out release assessments that are relevant to all exporting countries, two assumptions were made:

1. That if a disease were present in a country, it would be present at a sustainable herd-level and within-herd level prevalence. This assumption was based on the premise that prevalence; (a) would be dictated by epidemiological characteristics of the disease, and, (b) is, by nature, dynamic and thus may not remain at the level cited by a particular country at the time that a particular assessment is carried out.

2. That because Australian standards relevant to the slaughter and processing of pigs provide the minimum biosecurity that Australia accepts for commodities for human consumption, the procedures outlined in these standards should be adopted as a benchmark during estimation of a generic unrestricted risk of entry. In this context, the 'relevant Australian standard' includes:
   - the Australian Standard for the Construction of Premises for Processing Meat for Human Consumption;
   - the Australian Standard for the Construction of Premises for Processing Animals for Human Consumption; and
   - the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption.

   Of these documents, the *Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption* which describes Australia's domestic requirements for the ante-mortem, slaughter and post-mortem procedures relevant to the production of meat for human consumption, is of key importance. This document is discussed further in the description of the release assessment.

Evaluating and reporting likelihood

The quantitative likelihood model

A quantitative likelihood model was used in this import risk analysis to represent pathways relevant to the importation and utilisation of pig meat, the disposal of pig meat waste, and the possible exposure of susceptible animals in Australia.

The quantitative model provided for the following four important technical facilities:

- a framework upon which to base the logical structure of each assessment;
- evaluation of the effect of the 'volume of trade' during a specified period;
- accommodation of 'uncertainty' and/or 'natural variation' in the likelihood estimate assigned to individual steps in pathways;
- the use of 'sensitivity analysis' to identify critical steps in each scenario, and thus focus information needs and (where relevant) risk management.

A framework upon which to base the logical structure of each assessment

Assessments in this import risk analysis were carried out according to carefully described importation and distribution scenarios, and a rigorous evaluation of consequences. Consequently, the assessments were complex and multifaceted, and required a framework that ensured all elements were combined in a transparent and consistent manner. One of the principal benefits of the quantitative spreadsheet-based model was that it provided such a framework.

Evaluation of the effect of the 'volume of trade' during a specified period

It is to be expected that as the volume of trade in a commodity during a prescribed period increases, so too will the likelihood of at least one introduction of a disease. Because the volume of trade in a prescribed period affects likelihood, it will also affect risk.

Without a quantitative framework it would be difficult to investigate and to demonstrate transparently or consistently the effect that projected volume of trade may have on the risks associated with the importation of uncooked pig meat.

Accommodation of uncertainty and/or natural variation in the likelihood estimate assigned to individual steps in pathways

One of the requirements of an assessment, is that any uncertainty and/or natural variation in individual estimates be incorporated. This is important because quantitative assessments may otherwise appear to convey a degree of 'precision' that is not present in either the underlying science, or in the model parameter being estimated.

The two simulation-based methods used to represent likelihood are explained in the following section (See, Representing Expert Judgements and Quantitative Data).

The use of 'sensitivity analysis' to identify critical steps in each scenario, and thus focus information needs and (where relevant) risk management

Sensitivity analysis is a procedure that can be performed using the output from a quantitative assessment. In this context, sensitivity analysis ranks the model variables (in this case, either

step likelihoods, or other variables such as test sensitivity that are used to calculate step likelihoods) according to their correlation with the output.

Estimates for variables that are highly correlated with the model output should be as robust as possible. In some situations, it was important to identify such variables and, where they could not be estimated with assurance, to re-model these using extreme values or probability distributions above and below those that are believed to be most realistic. Such manual re-analyses are termed 'sensitivity simulations', and provided a means by which to determine whether a lack of precise knowledge might have led to misrepresentation of the final risk.

Representing expert judgements and quantitative data

Each step in the quantitative model was estimated, and subsequently represented, using one of two interchangeable approaches:

- A simple Uniform probability distribution representing a *qualitative* expert judgement of probability, or likelihood;
- A more precise probability distribution representing *quantitative* data or other scientific evidence on a probability, or on estimates of other numeric quantities such as counts and volumes.

Modelling qualitative expert judgment

Quantitative data were not available to support many of the probabilities assigned to the pathway steps considered in this analysis. Likelihoods assigned to these steps were therefore based on expert judgements, and modelled using the qualitative descriptors described in Biosecurity Australia's *Guidelines for Import Risk Analysis*.[8]

These terms are outlined in Table 2.

Table 2 Nomenclature for qualitative likelihoods

Likelihood	Descriptive definition
High	The event would be very likely to occur
Moderate	The event would occur with an even probability
Low	The event would be unlikely to occur
Very low	The event would be very unlikely to occur
Extremely low	The event would be extremely unlikely to occur
Negligible	The event would almost certainly not occur

In order to ensure consistency in the usage and interpretation of these six terms and definitions, and to provide a framework under which they could be logically and transparently combined, the 0-1 interval for likelihood was divided into six categories. Events considered almost certain to occur were assigned a likelihood of 1.

High	> 0.7	→	1
Moderate	> 0.3	→	0.7
Low	> 0.05	→	0.3

[8] Available at: http://www.daff.gov.au/

Very low	> 0.001 →	0.05
Extremely low	> 10^{-6} →	0.001
Negligible	> 0 →	10^{-6}

The boundaries adopted for qualitative likelihoods were those described in the Biosecurity Australia *Guidelines for Import Risk Analysis*. In choosing these boundaries, it was important to provide a system that could be adopted by those whose task it is to review scientific evidence and estimate likelihoods. It was also important to ensure that the categories are neither overly precise nor constrictive, nor so broad as to lose the precision that may have been present in the original body of scientific evidence. Accordingly, it was *not* critical that the categories are of equal width, or that they are assigned according to a predefined arithmetic or logarithmic scale. Overall, the emphasis was on useability and, once defined, a system that would enable experts to use the corresponding terms and definitions (Table 2) consistently.

For example, an expert presented with the descriptors and probability ranges shown above might consider 'the likelihood that an infected animal will be sent to slaughter' to be 'low'.

In making this choice, the expert would have considered the likelihood to be less than the broad band representing an approximately even (moderate) probability, but not so low as to be in a range dominated by small fractions of a percent.

Likelihoods described under this nomenclature were subsequently combined using a spreadsheet-based simulation model. This model was constructed in Microsoft Excel, and run using the spreadsheet add-on software, @Risk (© 2001, Palisade Corporation, USA).

This was achieved by representing each of the six semi-quantitative likelihood categories as a 'Uniform probability distribution' (abbreviated 'Uniform distribution'). A Uniform probability distribution (also called a Rectangular probability distribution) is one that has a maximum and minimum value, but for which the continuous spectrum of values in between these limits each occurs with the same probability.

The parameters of each of these six Uniform distributions (their maximum and minimum values) were obtained from the boundaries of the corresponding probability category.

High	L ~ Uniform (0.7, 1)[9]
Moderate	L ~ Uniform (0.3, 0.7)
Low	L ~ Uniform (0.05, 0.3)
Very low	L ~ Uniform (0.001, 0.05)
Extremely low	L ~ Uniform (10^{-6}, 0.001)
Negligible	L ~ Uniform (0, 10^{-6})

An example of a Uniform distribution for a 'very low' likelihood (L) with minimum value of 0.001 and a maximum value of 0.05 is shown in Figure 2 below. Using the notation explained above, this distribution can be written in shorthand as L ~ Uniform (0.001, 0.05).

[9] This abbreviated syntax for likelihood (L) should be read as "*L is distributed uniformly between 0.7 and 1*".

Thus, a likelihood described by an expert presented with the descriptors and probability ranges shown above as 'Low', will be represented using a Uniform probability distribution with parameters, minimum = 0.05 and maximum = 0.30.

This would imply that the true likelihood might fall anywhere in the range 0.05 to 0.30, but that no particular value in this range is considered by the analyst to be more likely than any other.

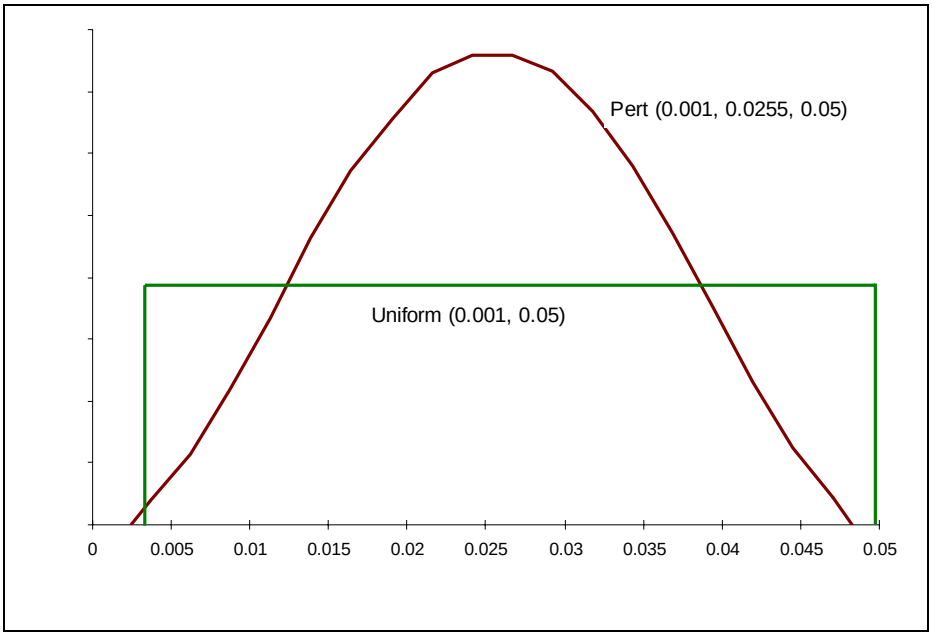
Modelling quantitative data

Quantitative data on a probability, or on estimates of other numeric quantities such as counts and volumes, were modelled either as a point estimate or, more commonly, as a probability distribution. The shape and parameters of this distribution depended on the nature of the variable being modelled and the completeness of available data. In many cases, however, the Pert distribution (a special case of the Beta distribution) was used.

The Pert distribution has three parameters, namely, its minimum, most likely and maximum values. The advantage of the Pert distribution over the very simple Uniform distributions described above is that it allows values that are considered more likely to occur, to be modelled as such. The distribution may resemble the familiar 'bell curve' although, unlike the Normal distribution upon which the bell curve is based, it need not be symmetrical and can be limited or constrained to designated maximum and minimum values.

An example of a Pert distribution for a likelihood (L) with a minimum value of 0.001, a most likely value of 0.0255 and a maximum value of 0.05 is shown in Figure 2 below. Using the notation explained above, this distribution can be written in shorthand as L ~ Pert (0.001, 0.0255, 0.05).

Figure 2 Uniform and Pert probability distributions



Summary: evaluating and reporting likelihood

The likelihood component of this analysis was based on a quantitative model. Simple Uniform probability distributions were used to represent expert judgements, whilst more precise probability distributions such as the Pert in Figure 2 above, were used where quantitative data was available.

The likelihood model is considered to be 'stochastic', because probability distributions rather than point estimates were used to represent likelihoods, proportions and other model inputs (such as volume of pig meat and numbers of waste units). The outcome of a stochastic model was also a distribution, rather than a point estimate. Interpretation of this probability distribution(s) was based on its correlation with Biosecurity Australia's six likelihood categories (see above). The median value (50^{th} percentile) was taken and the particular likelihood range within which this value falls was reported.

Release assessment

Steps in the release scenario

The 'biological pathway', or ordered sequence of steps undertaken in sourcing, processing and exporting a commodity, is termed its 'release scenario'. The initiating step for the release scenario for pig meat was the sourcing of slaughter age pigs in the exporting country, while the end-point was 'the arrival in Australia' of infected pig meat. In this context, 'arrival in Australia' was taken to mean the release of imported pig meat from the port of entry - whether this was an airport or a shipping port.

In the *Technical Issues Paper* it was stated that:

"… the definition of 'pig meat' is limited to porcine muscle tissue, blood confined to muscle vasculature, bone and bone marrow, and any other tissues (for example, lymph nodes) that may be considered inseparable from muscle.

And that;

Inter alia, this approach means that the issues associated with the introduction of disease agents as a result of the importation of 'pig meat products' derived from offal, blood, bone or neurological tissue, will not be considered."

This definition was continued in this method document, and in the disease risk assessments. It should be noted that a carcass could include the head, but with neurological tissue, tongue and tonsils removed.

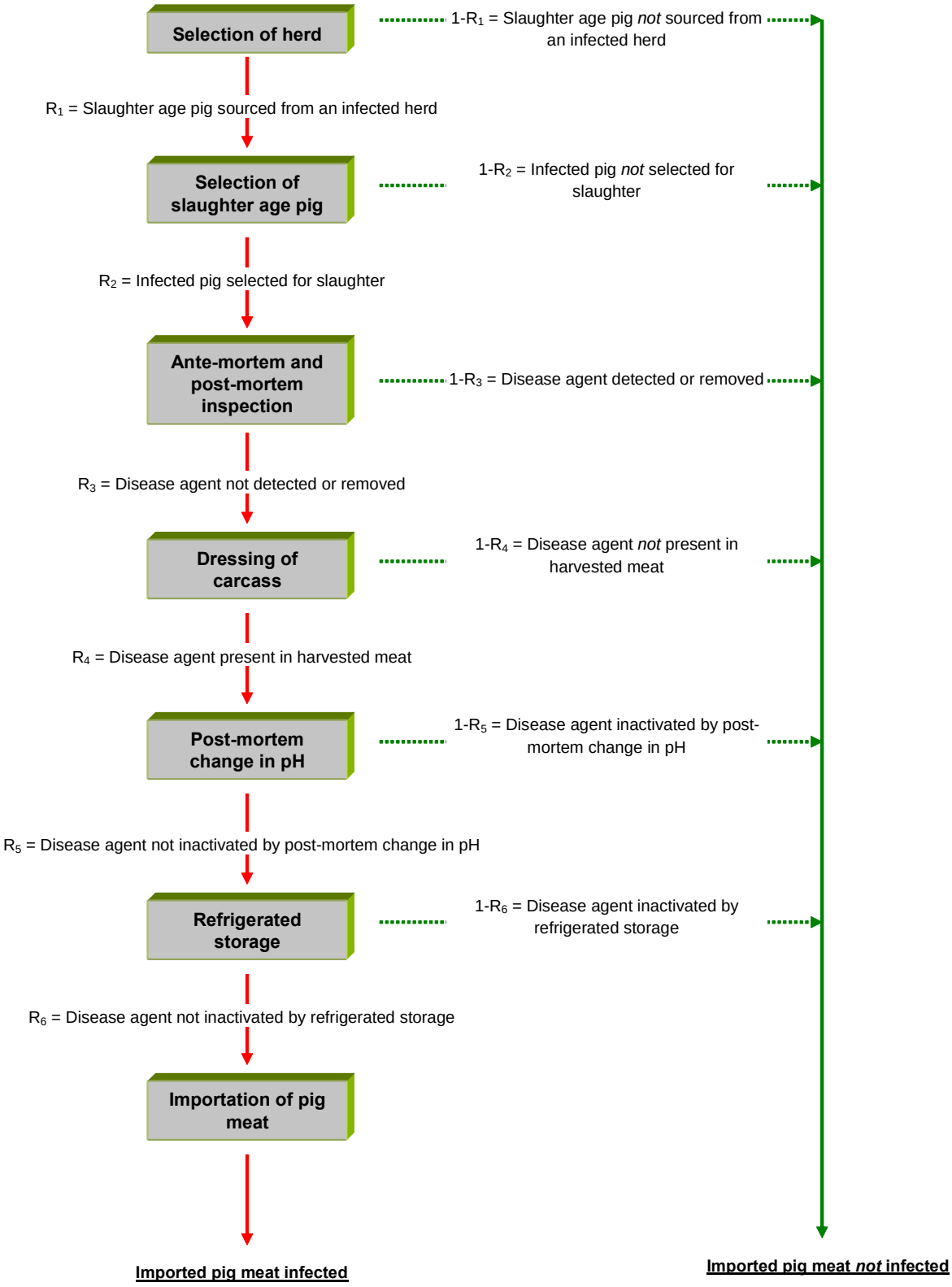
A conceptual representation of the release scenario for uncooked pig meat is presented in Figure 3. Likelihoods assigned to steps in the release scenario (R1 - R6) were evaluated and reported using the terms and definitions in Table 1. In each case the step-level likelihood represents 'the probability that infection will not be detected at that step, or that the infectious agent will not be inactivated'. The likelihood is 'conditional', because it is based on the assumption that the commodity has remained infected up until the start of the step in question.

- *Step 1* (R1): slaughter-age pigs[10] selected from an infected herd
- *Step 2* (R2): infected individual pig selected from an infected herd
- *Step 3* (R3): infected pig not detected, nor the pathogenic agent removed, as a result of ante-mortem and post-mortem requirements described in the Australian Standard
- *Step 4* (R4): pathogenic agent present in the meat harvested from an infected pig
- *Step 5* (R5): pathogenic agent in infected meat not destroyed by the post-mortem drop in muscle pH
- *Step 6* (R6): pathogenic agent in infected meat not destroyed by refrigerated storage and transport.

For some enteric organisms, on occasions it was additionally important to consider contamination of muscle tissue within the abattoir or meat processing plant. The likelihood that a pathogenic agent will contaminate muscle tissue at the time of slaughter, evisceration, de-boning or during the dressing of the carcass (or within any of the steps taken in the further processing of a meat product) will depend on the physical characteristics of the pathogenic agent and this was discussed within individual pathogenic agent assessments.

[10] The Panel considered that a slaughter-age pig would be at least 5 months of age.

Figure 3 Release scenario



R1: The likelihood assigned to *Step 1* represents the prevalence of infected herds within the country from which pig meat would be sourced. Regardless of the causative agent, herd prevalence is likely to fluctuate with changes in disease dynamics within an infected country

(the number of infectives, the number of susceptibles, the potential for adequate contact or transmission, etc). This will in turn be influenced by a range of environmental, human and epidemiological factors.

Given its dynamic nature, the herd prevalence of each identified disease was modelled by adopting a value considered sustainable in an endemically infected country, zone or region. It was recognised that serological evidence of infection often forms the basis of determining herd prevalence, and although this indicates exposure to the pathogenic agent it may not reflect active infection at the time of testing. Herd prevalence was discussed further within the risk assessment for each identified pathogenic agent.

R2: The likelihood assigned to *Step 2* represents the prevalence of infected animals within an infected herd. Given the many human, environmental and epidemiological factors that will influence group-level disease dynamics, this likelihood is unlikely to be stable within any given herd, or consistent among infected herds. For this reason, the within-herd prevalence of each identified disease was modelled by adopting a value considered to represent the prevalence sustainable within an endemically infected herd. Within-herd prevalence was discussed further in the risk assessment for each identified pathogenic agent.

R3: The likelihood assigned to *Step 3* represents the probability that each pathogenic agent would not be detected as a result of controls and procedures carried out accordance with requirements dictated in the relevant Australian standards.

Of particular importance are; (a) Part 3, article 8, and Schedule 3 (b) Part 3, article 10 and Schedule 2 and Schedule 3 of the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption, which describe the ante-mortem and post-mortem procedures (respectively) that Australia considers to provide the minimum level of sanitary protection acceptable for meat products.

More specifically, ante- and post-mortem procedures identified in the Australian Standard provide ‘criteria’ with which to assess the likelihood that each identified pathogenic agent or its associated disease syndrome would not be detected. These criteria, which are based on the visibility (ante-mortem and/or post-mortem) of pathological changes associated with each disease process, were discussed further within the assessment for each identified pathogenic agent.

R4: The likelihood assigned to *Step 4* represents the probability that each pathogenic agent would be present in meat harvested for export.

In the bacteraemic or viraemic phase of an infection, it is possible for a pathogen to ‘infect’ or to passively ‘contaminate’ muscle tissue.

- *Infection* of muscle tissue may occur as a result of a break in the barrier offered by skin and subcutaneous tissue, by translocation of the organism through the bloodstream or as a result of the migration of an organism from another site in the animal’s body.
- *Contamination* of muscle tissue may occur as a result of a break in the animal’s skin, or through the presence of contaminated blood or lymph in muscle vasculature at the time of slaughter. Depending on characteristics of the pathogenic agent and the stage of infection, organisms may be present in serum or extra-cellular fluid, or may invade the animal’s red or white blood cells. It follows that the successful bleeding of a carcass immediately following slaughter will tend to decrease the likelihood of muscle contamination by this route or, where contamination has occurred, to decrease the number of organisms per unit of contaminated muscle tissue.

In this analysis, meat described as 'infected' includes both the terms infection and contamination of muscle tissue as described above.

This likelihood was discussed further within the assessment for each identified pathogenic agent.

R5: The likelihood assigned to *Step 5* represents the probability that a pathogenic agent would not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation. The pH of muscle falls during the onset of rigor mortis as a result of the accumulation of lactic acid.

The final pH of muscle is affected by factors including breed, ante-mortem stress, and the processing system (Gregory, 2000; Tornberg, 2000). The pH of meat may also differ among different muscle groups. Finally, pH does not fall to the same level during rigor mortis in blood clots, bone marrow, lymph nodes and viscera and, for this reason, the antimicrobial properties of meat that has not been properly bled, or meat products that contain these carcass elements may differ (Farez & Morley, 1997).

Low pH values (< 5.7) are associated with pork of lesser quality (pale, soft, exudative; and red, soft, exudative pork) whereas pH values above 6.2 are associated with darker, less desirable pork called DFD (dark, firm and dry meat) (Tornberg, 2000; van Laack, 2001).

In view of these factors, it cannot be assumed that the pH of meat harvested for export would attain a pH lower than 6.2. This value was subsequently adopted as a benchmark for the purposes of this analysis, and the likelihood that each identified pathogenic agent would be inactivated at or above pH 6.2 was discussed further within the individual assessments.

R6: The likelihood assigned to *Step 6* represents the probability that a pathogenic agent would not be destroyed during cold storage and transport. It was difficult to be prescriptive about the period of storage prior to the arrival of the commodity in Australia, because this may vary substantially among pig meat products, consignments and exporting countries. It is reasonable, however, to expect that the period of storage would be at least 2-3 days.

It was also difficult to be prescriptive regarding the temperature during storage and, indeed, it is likely that a substantial proportion of imported pig meat will be frozen. It is, however, stated in the Australian Standard for Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard) that the surface temperature of carcasses should not be more than 7°C, and that the internal temperature of meat other than carcasses should not be more than 5°C. Because all exporting countries must at least equal these conditions, they were adopted in the analysis as a benchmark. This likelihood was discussed further within the assessment for each identified pathogenic agent.

Calculation of the likelihood of entry

Step likelihoods for the release assessment were combined using the spreadsheet-based simulation approach to give the overall likelihood that 'imported pig meat that has been derived from a single carcass will be infected'. This was termed the 'likelihood of entry', and was calculated as shown in Table 3 below.

It can be seen from this table that the 'unit' chosen for the likelihood of entry was 'meat derived from the carcass of a single infected pig'. Meat from the carcass of a single infected pig was chosen to be the unit for these assessments because;

- The infection status of an individual animal forms the basis for disease dynamics in a population;
- Infection, if present, is likely to affect all carcass cuts;
- The concept of a carcass, or a 'carcass equivalent', provides a simple and intuitive unit upon which estimates can be based.

Table 3 Calculation of the likelihood of entry

Variable	Description and calculation / estimation
LE	**The likelihood that imported pig meat that has been derived from a single carcass will be infected** $= R_{3.3} \times R_4 \times R_5 \times R_6$
R_1	The likelihood that a source herd is infected = disease specific
R_2	The likelihood that a slaughter age pig is infected = disease specific
R_3	The likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out accordance with requirements dictated in the relevant Australian standards $= 1 - R_{3.1}$
$R_{3.1}$	The sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard = disease specific
$R_{3.2}$	The specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard = 1- extremely low
$R_{3.3}$	The likelihood that a carcass will be infected, given that it has completed inspection = 1 - the 'Negative Predictive Value' for ante-mortem, slaughter and processing requirements described in the Australian Standard $= 1 - \frac{R_{3.2} \times (1 - R_1 \times R_2)}{R_{3.2} \times (1 - R_1 \times R_2) + (1 - R_{3.1}) \times (R_1 \times R_2)}$
R_4	The likelihood that the pathogenic agent will be present in meat harvested for export = disease specific
R_5	The likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation = disease specific
R_6	The likelihood that the pathogenic agent will not be destroyed during cold storage and transport = disease specific

LE = Likelihood of entry

R_n = The likelihood assigned to the n^{th} step in the release assessment

Exposure assessment

Projected volume of trade in uncooked pig meat

An estimate of the volume of uncooked pig meat that might be imported if trade were permitted without restrictions was based on current import trends and possible future market penetration.

Pig meat imports have been increasing since 1998 (Figure 4). Pig meat imports fluctuate, with most recently the highest monthly figure recorded for October 2003. The moving annual total to November 2003 is over 52,000 tonnes (shipping weight). Most imported pig meat is used for the manufacture of smallgoods due to the quarantine requirement that pig meat from Canada and Denmark be cooked on arrival in Australia. In 2001, total pig meat production in Australia was 377,889 tonnes. [11] This has since increased to approximately 405,000 tonnes. In the absence of quarantine restrictions (unrestricted risk) it is likely that the annual volume of imports would increase still further.

In considering likely future market penetration in the absence of quarantine restrictions such as post arrival processing, information was obtained from New Zealand, which until recently permitted bone-in frozen product (not subject to post arrival processing controls). In the 12 months prior New Zealand imposing processing controls imports constituted approximately 28% of New Zealand's total pig meat production.[12]

Based on the current trend of increasing imports of pig meat in Australia and market penetration in New Zealand, the Panel considered that unrestricted pig meat imports may increase to approximately 90,000 tonnes (shipped weight) per year. To accommodate these figures, and to take account of the uncertainty around them, the annual volume of trade in pig meat was modelled as a Pert distribution, with a minimum value of 50,000 tonnes, most likely value 90,000 tonnes and maximum value 151,160 tonnes. This distribution is illustrated in Figure 4.

[11] http://www.pork.gov.au

[12] Personal communication from Dr Allen Bryce, National Manager (Surveillance and Response) New Zealand Ministry of Agriculture and Forestry.

Figure 4 Australian pig meat imports — 12 month moving total and trend line

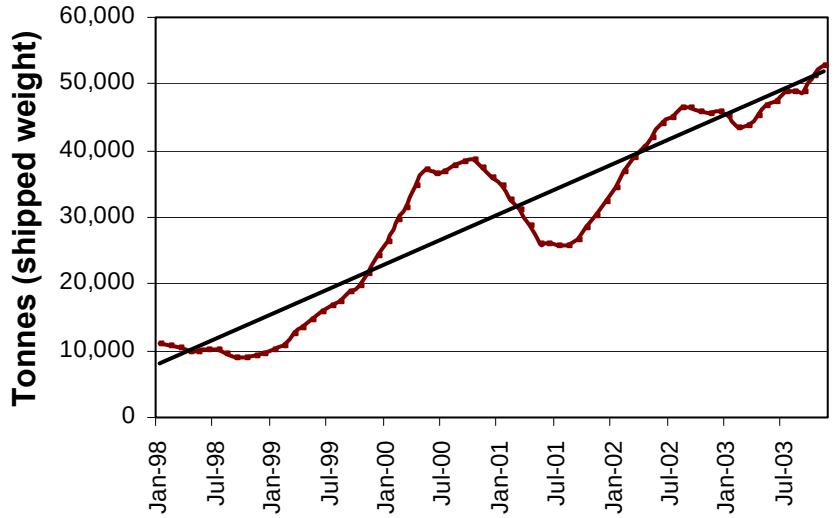
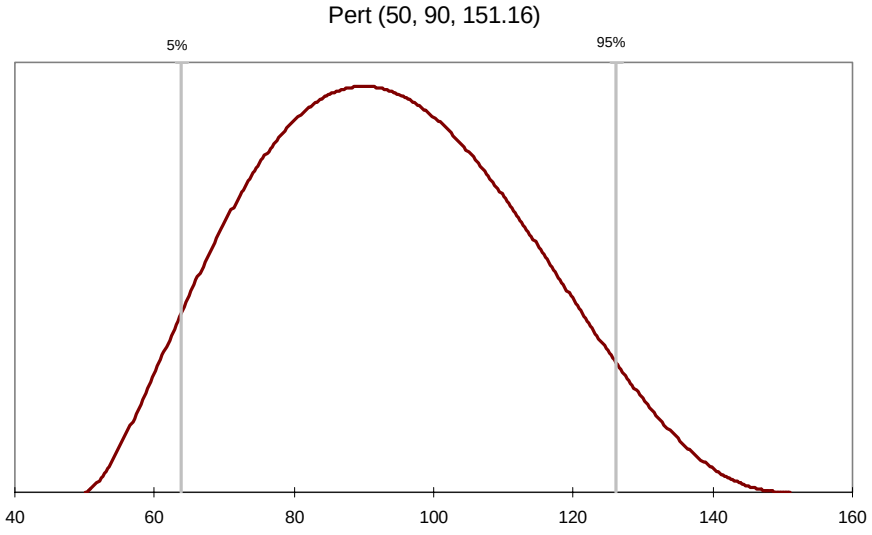


Figure 5 A Pert distribution for the annual volume of trade in pig meat (shipped weight x 1,000 tonnes)



Distribution and utilisation of pig meat in Australia

Distribution pathways

Under current quarantine requirements, uncooked pig meat imported into Australia must be cooked immediately on arrival. The exception is pig meat imported into Australia from the South Island of New Zealand. Consequently, imported pig meat is used almost exclusively for the manufacture of smallgoods. However, because the risk assessments in this analysis were undertaken firstly on an 'unrestricted'[13] basis, it was necessary to assume that imported pig meat would be distributed as if it were domestically produced, and that households and food service establishments (restaurants, cafes, take-away fast food outlets, institutions etc) would also have access to imported product.

Fresh or frozen pig meat might be imported directly to smallgoods manufacturers, or channelled to smallgoods via wholesalers. Smallgoods would then be sold directly or through retailers to food service establishments, and through retailers to households. In the research project *Pigs and Pigmeat* (Industry Commission (IC), 1995) estimates provided by the Australian Pork Corporation (APC) (now part of Australia Pork Limited) suggest that between 35 and 40% of domestically produced pig meat is sold to households and food service establishments as fresh meat. The remaining 60 to 65% of domestically produced pig meat is used in manufacture of smallgoods. Smallgoods are then sold on to households and food service establishments. These industry statistics are dated, but are likely to be reasonably relevant to current trends.[14]

Distribution pathways for pig meat are illustrated in Figure 6.

Proportion of pig meat purchased by food service establishments

Statistics on the proportion of pig meat (fresh meat and smallgoods) purchased by food service establishments are not available. However, it was reported that 19% of the total gross value of domestically produced fresh and frozen pig meat is purchased by food service establishments (BIS Shrapnel, 2002). More recently, it has been suggested that food service establishments could purchase as much as 40% of fresh pig meat[15] (note - fresh pig meat constitutes 40% of total pig meat production).

With regard to the proportion of smallgoods purchased by food service establishments, information from a smallgoods manufacturer indicates that approximately 13 to 20% of smallgoods are purchased by these establishments.

Some information is also available on the number of meals eaten away from home. In an Australian study (Cashel, 2001), it was reported that, on average, one out of seven evening meals (14%) were eaten away from home. Alternatively, a study of the Australian food service sector (Foreign Agricultural Service/USDA, 2000) estimated that in 1998, approximately 222 meals per head of population were served by the food service industry, with growth anticipated to be 4% annually over the next 2 years. If it is assumed that Australians would consume three meals a day over the 365 days of the year, then 222 meals per head per year represents

[13] In this context, the term *unrestricted* denotes an assessment carried out without consideration of the effect of risk management. The decision to implement risk management was based on the 'acceptability' or otherwise of the unrestricted risk (see, Risk Estimation). The efficacy of risk management was determined by obtaining an estimate of the restricted risk, and comparing this to both the original estimate and to Australia's appropriate level of protection (see, Risk Management).

[14] Personal communication from Mr Raymond North, General Manager, Australian Pork Corporation (APC), Sydney.

[15] Personal communication from Mr Terry Brown, General Manager (Marketing), Australian Pork Limited, Canberra.

approximately 20% of all meals. A more recent Australian survey estimated that approximately 30% of meals are eaten outside the home.[16] Because some of the meals provided by the food service industry are considered 'snacks', and thus in addition to the three main meals, the figure of 30% may be somewhat higher than the true proportion.

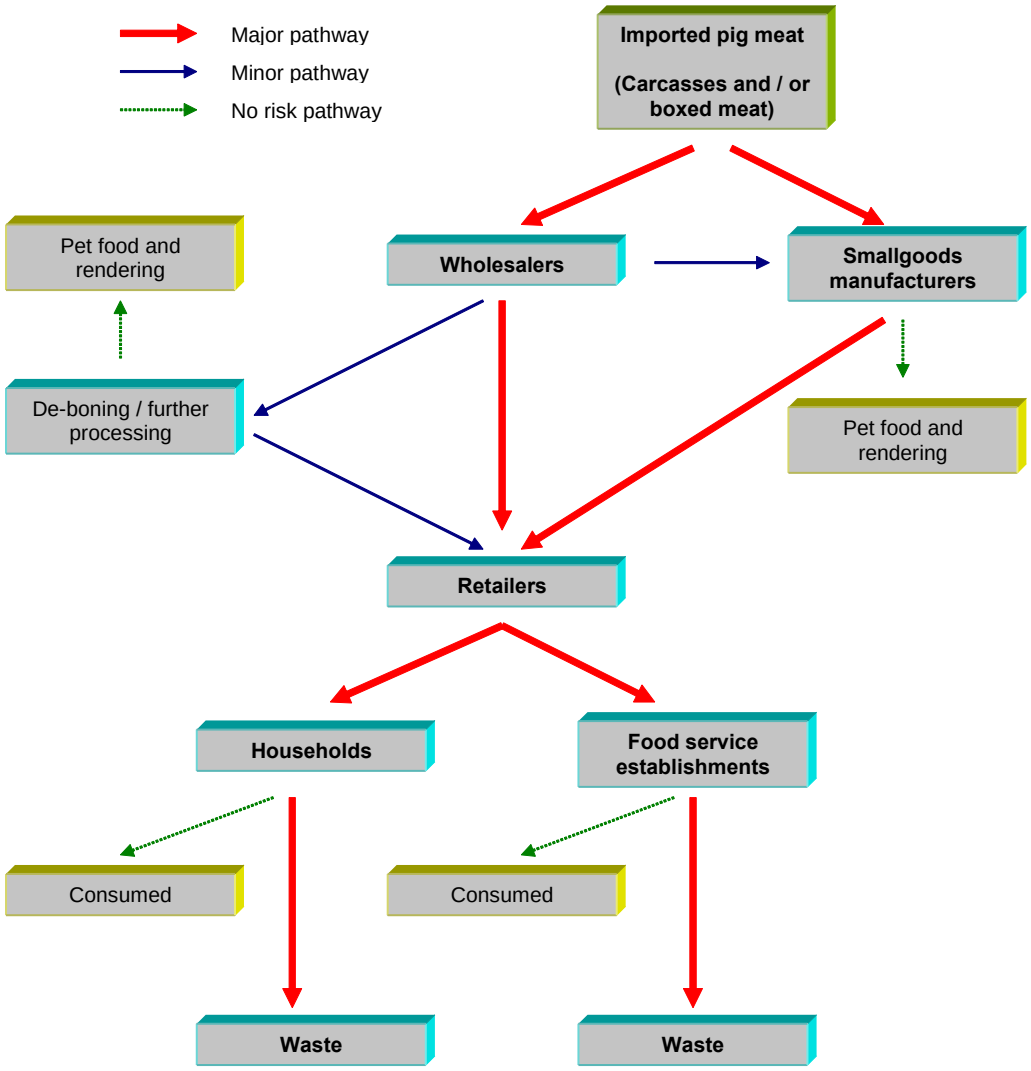
To accommodate these figures, and to take account of the uncertainty around them, the proportion of pig meat purchased by food service establishments as fresh meat and smallgoods were modelled as a Pert distribution with a minimum value 15%, most likely value 25%, and maximum value of 30%.

Proportion of pig meat purchased by households

As with food service establishments, statistics are not available on the proportion of pig meat (fresh meat and smallgoods) purchased by households. However, it follows that if food service establishments purchase approximately 25% then householders purchase the remainder. On this basis, the proportion of imported pig meat likely to be purchased by households was modelled as the complement of (i.e. one minus) the proportion likely to be purchased by food service establishments. Additional information provided by Australian Pork Limited[14] showed that, of total pork product serves purchased by a sample of grocery buyers, fresh pork, fresh ham, deli bacon, deli ham and pre-packaged rashers constituted 39%, 5%, 18%, 26% and 9%, respectively. These figures are consistent with the distribution generated by the model for the proportion of imported pig meat likely to be purchased by households in the form of fresh meat and smallgoods.

[16] Cited in *The Australian*, Friday September 20 2002, p.9.

Figure 6 Distribution pathways for imported pig meat



Proportion of pig meat discarded as waste

The Panel considers that susceptible animals in Australia would most likely gain access to uncooked, inadequately cooked or processed pig meat through scraps discarded by households or food service establishments. Cooked and processed pig meat scraps were included in the analysis because cooking and processing may not have been carried out to a level sufficient to inactivate the pathogenic agents under consideration. In addition, it is known that some pathogenic agents can persist in bone marrow and lymph nodes, and yet be inactivated in muscle tissue that is heat treated. It is also known that certain processes such as fermentation, and cooking at low temperatures or for short periods, may not result in inactivation (Blackwell, et al., 1985).

Wholesalers or smallgoods manufacturers generate very little waste, and that which is produced is generally diverted through a range of composite products, or heat rendered. Rendering in Australia utilises both wet and dry procedures, although in either case the minimum temperature is engineered to be approximately 120°C (Quinn & Fabiansson, 2001). The

pathogenic agents identified in this IRA would be inactivated by rendering under Australian conditions, and this pathway was not considered further.[17]

Although carcasses may be imported it is likely that the majority of pig meat imported would be as boxed meat either bone-in or de-boned, hence there would be minimal trimming. This assumption is supported by information available on types of pig meat imported into New Zealand. For example, in 2001, 84% of fresh, chilled or frozen pig meat imported into New Zealand was as boneless cuts, 15.3% as bone-in cuts and 0.7% as carcasses.[18]

Some carcass by-products may also be utilised by pet food manufacturers (pet food does not include stockfeeds for livestock). The Panel also recognised that there were several other minor pathways that may lead to exposure of susceptible animals in Australia including, but not limited to, contaminated packaging material, waste water contamination, contamination of clothing for those working at processing plants and transport accidents involving imported product.

The Panel considers that if imported pig meat poses a quarantine risk to Australia, this would become apparent through the major distribution pathways (i.e. households and food service establishments). Any potential risk of exposure to susceptible animals to infected imported pig meat via pet food products or through other minor pathways was examined in the context of risk management.

The proportion of pig meat purchased by food service establishments and discarded as waste

Statistics were not available on the proportion of pig meat purchased by food service establishments and subsequently discarded as waste. Indeed, this proportion is both complex and highly variable, because it incorporates factors associated with the amount of waste generated by different cuts of meat or smallgoods products, as well as the amount of waste generated from uneaten or partially eaten meals. The proportion is, however, likely to be higher than the equivalent proportion of pig meat waste from households, where meat is purchased for a smaller number of very specific meals, and where the cost associated with waste cannot be passed on to a consumer.

The proportion of pig meat purchased by food service establishments and subsequently discarded as waste was modelled in this analysis as a multiple of the Pert distribution used to model the equivalent proportion of pig meat waste from households (see below). The multiple used was allowed to vary between 110 and 150%, with a most likely value of 120%. The multiple was modelled as a Pert distribution with these parameters.

The proportion of pig meat purchased by households that is discarded as waste

As was the case for food service establishments, statistics were not available on the proportion of pig meat purchased by households and subsequently discarded as waste.

However, an informal survey of personnel in a Government department with households ranging from 1 to 6 persons revealed that most consumers may discard between 1 to 10% by volume of purchased pig meat. This will vary with the cuts of meat purchased. For example,

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17 If the risk assessment for a particular pathogenic agent demonstrated that imported pig meat poses an unacceptable level of risk, then risk management measures, including those involving disposal of wastes, were considered.

18 Statistics New Zealand, as reported by New Zealand Ministry of Agriculture and Forestry.

pork purchased for stir-fry dishes will generate very little waste, while pork chops will generate substantially more waste. Smallgoods, with exception of bone-in ham and bacon, are generally purchased in waste-free or consumer-ready form. As discussed above Australian Pork Limited advised that, of total pork product serves purchased by a sample of grocery buyers, fresh pork, fresh ham, deli bacon, deli ham and pre-packaged rashers constituted 39%, 5%, 18%, 26% and 9%, respectively. A small survey conducted in the United Kingdom assessed the proportion of purchased meat that is discarded uncooked from domestic kitchens (Gale, 2002)[19]. Eighteen of 39 respondents estimated that around 5% of meat purchased was discarded as uncooked, and a further 19 households discarded 1% or less. The remaining two estimates were 10% and 20%. Given this information, the proportion of pig meat purchased by households that is discarded as waste was modelled as a Pert distribution with minimum, most likely, and maximum values of 1%, 5%, and 10%, respectively.

The Panel sought to categorise pig meat cuts and products on the basis of the amount of waste generated, and to enumerate the proportion of each category that is purchased by households throughout a year. Assistance with this study was sought from the Australian industry and from other relevant parties; however, detailed information of this nature was not available.

Projected volume of pig meat discarded as waste

The projected volume of imported pig meat discarded as waste per year was calculated as the sum of the wastes generated by each group (households, food service establishments). Projected amounts of wastes generated by wholesalers and smallgoods manufacturers from imported pig meat were considered to be negligible in quantity and were not included in this calculation.

The amounts of waste generated by households *and* food service establishments were estimated independently, and subsequently summed, as shown in the formula below:

$$\text{Waste}_{\text{Total}} = \text{Waste}_{\text{HH}} + \text{Waste}_{\text{FSE}}$$

The amount of waste (kg) that may be generated by households in a 12-month period was calculated as shown in the formula below:

$$\text{Waste}_{\text{HH}} = \text{Total imports} \times \text{Prop}_{\text{HH}} \times \text{Propwaste}_{\text{HH}}$$

Where;

Total imports	=	the projected total volume of pig meat imported in a 12 month period (kg)
Prop_{HH}	=	the proportion of imported pig meat likely to be purchased by households as fresh meat and smallgoods
$\text{Propwaste}_{\text{HH}}$	=	the proportion of pig meat purchased by households that is discarded as waste (including cooked, uncooked and processed)

The amount of waste (kg) that may be generated by food service establishments in a 12-month period was calculated as shown in the formula below:

$$\text{Waste}_{\text{FSE}} = \text{Total imports} \times \text{Prop}_{\text{FSE}} \times \text{Propwaste}_{\text{FSE}}$$

[19] Note that this survey included all meat (that is, was not limited to pig meat), but was restricted to uncooked wastes.

Where;

Total imports	=	the projected total volume of pig meat imported in a 12 month period (kg)
$Prop_{FSE}$	=	the proportion of imported pig meat likely to be purchased by food service establishments as fresh meat and smallgoods
$Propwaste_{FSE}$	=	the proportion of pig meat purchased by food service establishments that is discarded as waste (including cooked, uncooked and processed)

Waste units

The annual likelihood of exposure of susceptible animals to infected imported pig meat waste will be related to the number of exposure opportunities that may occur. An exposure opportunity was considered to be the exposure of one susceptible animal to a quantity of pig meat waste ('waste unit') that was no larger than could be consumed by the animal in one day.

The number of waste units potentially generated in a 12-month period was calculated as shown in the formula below:

$$\text{Waste units}_{\text{Total}} = \frac{\text{Waste}_{\text{Total}}}{\text{Waste}_{\text{Size}}}$$

Where;

$Waste_{TOTAL}$	=	the total amount of waste from imported pig meat generated by households and food service establishments (kg)
$Waste_{SIZE}$	=	the size of a waste unit (kg)

The size of a unit of discarded waste was difficult to estimate, as it may vary amongst the cuts of meat and types of smallgoods, and with the behaviour of consumers.

Given this, the size of a waste unit is likely to lie between as little as 10g (or 0.010kg) and as large as a pile of carcasses, which may be discarded in the event of a freezer malfunction. In the context of this analysis, the maximum size of a waste unit is considered to be the maximum amount of meat that a pig would be likely to obtain and immediately ingest. Although not directly analogous, it is known that lactating feral sows may consume as much as 5kg of feed per day (Choquenot, et al. 1996). This was adopted as the maximum size of a waste unit.

The distribution for the size of a waste unit was bound by the minimum (10g) and maximum (5kg) discussed above, and allowed to vary between these with a most likely value of 250g. The size of a waste unit ($Waste_{Size}$) was subsequently modelled as a Custom probability distribution with those parameters.

Table 4 Calculation of the number of waste units

Variable	Description and calculation / estimation
Waste units $_{Total}$	The number of waste units potentially generated in a 12 month period $= \frac{Waste_{Total}}{Waste_{Size}}$
Waste $_{Total}$	The total amount of waste from imported pig meat generated by households and food service establishments (kg) $= Waste_{HH} + Waste_{FSE}$
Waste $_{Size}$	The size of a waste unit (kg) = Custom (0.01, 0.25, 5.0)
Waste $_{HH}$	The amount of waste (kg) that may be generated by households in a 12 month period $=$ Total imports x Prop $_{HH}$ x Propwaste $_{HH}$
Waste $_{FSE}$	The amount of waste (kg) that may be generated by food service establishments in a 12 month period $=$ Total imports x Prop $_{FSE}$ x Propwaste $_{FSE}$
Total imports	The projected total volume of pig meat imported in a 12 month period (kg) = Pert (50.00×10^6, 90.00×10^6, 151.16×10^6)
Prop $_{HH}$	The proportion of imported pig meat likely to be purchased by households as fresh meat and smallgoods $= 1 -$ Prop $_{FSE}$
Propwaste $_{HH}$	The proportion of pig meat purchased by households that is discarded as waste (including uncooked, cooked and processed) = Pert (0.01, 0.05, 0.10)
Prop $_{FSE}$	The proportion of imported pig meat likely to be purchased by food service establishments as fresh meat and smallgoods = Pert (0.15, 0.25, 0.30)
Propwaste $_{FSE}$	The proportion of pig meat purchased by food service establishments that is discarded as waste (including uncooked, cooked and processed) = Pert (1.10, 1.20, 1.50) x Propwaste $_{HH}$

Exposure groups

The term ‘exposure group’ denotes a category of animal (whether based on its species or the manner in which it lives or is managed) that may be susceptible to one or more of the pathogenic agents considered in the risk assessments.

Four groups of animals that may be directly exposed to uncooked pig meat scraps were identified.[20]

1. Feral pigs: wild porcines of the Sus scrofa species
2. Backyard pig-producers: non-commercial enterprises with less than 10 sows
3. Small pig-producing enterprises: commercial enterprises with between 10 and 99 sows
4. Other susceptible species: membership of this group varied among the identified pathogenic agents, but could include rats and other rodents, carnivorous or omnivorous bird life and other species that are either fed scraps, or have a propensity for scavenging

Commercial enterprises with more than 99 sows were not considered to be at risk of 'direct' exposure to meat scraps. Several issues support this contention. Firstly, evidence in Australia suggests that larger commercial piggeries are extremely unlikely to engage in illegal swill feeding. In recent years there have been few prosecutions for illegal swill feeding, of which nearly all involved backyard pig producers. For example, in 2002 there were a total of four prosecutions for illegal swill feeding and four warning letters. There has been one report of a small commercial piggery feeding illegal swill to up to 20 sows[21], and another involving up to 40 pigs[22]. Secondly, biosecurity is recognised by most commercial pig producers to be a critical management issue[23], such that pigs in large commercial herds are effectively 'quarantined' as regards new introductions or the feeding of substrates other than their prescribed diet. Finally, because growth rate is one of the important determinants of efficient pig production, most medium-to-large piggeries institute carefully designed feeding regimens.

The four exposure groups are illustrated in Figure 7.

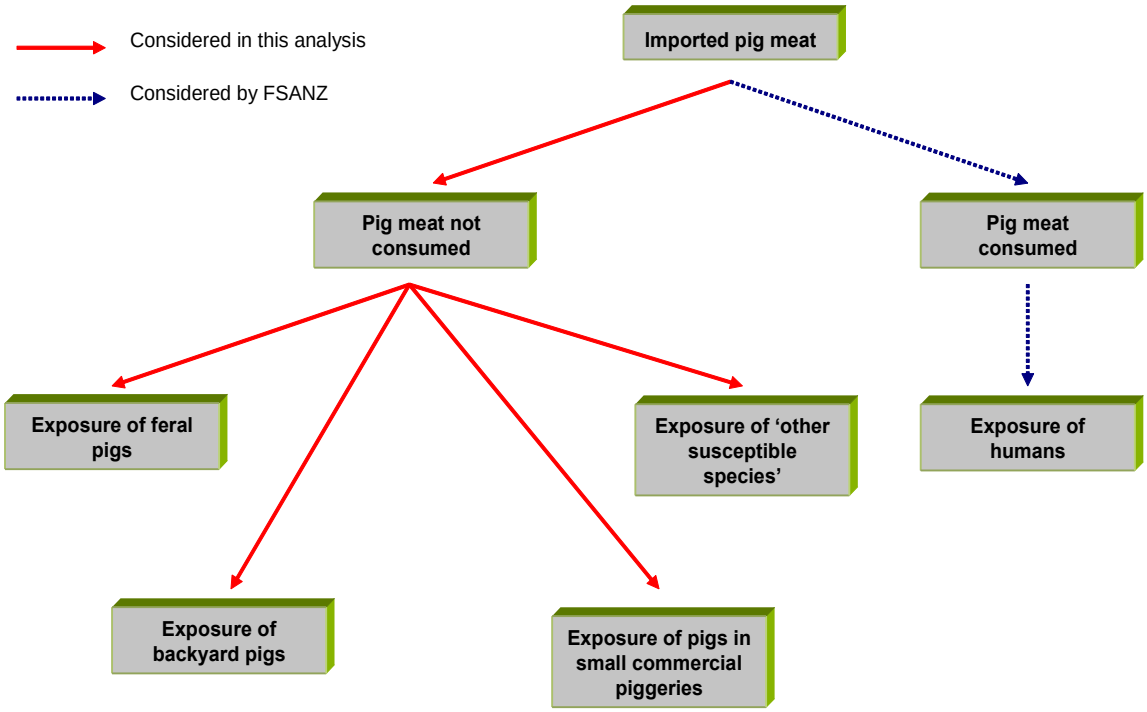
[20] In this context, the term *direct exposure* is taken to mean exposure resulting from the direct consumption of infected pig meat.

[21] Personal communication from Dr Hugh Millar, Chief Veterinary Officer, Department Natural Resources and Environment, Victoria (2000).

[22] Personal communication from Dr Kevin Dunn, Executive Director, Animal and Plant Health Service, Queensland Department of Primary Industries, Queensland (2003).

[23] In this context, *biosecurity* describes protection from diseases exotic to a given piggery, as well as diseases exotic to Australia.

Figure 7 Exposure groups for imported pig meat



Each of the four exposure groups (and the remaining group of medium-to-large piggeries) might also be exposed to imported pathogenic agents through a range of 'indirect' routes. For example, pigs kept in small piggeries might eat rats (one of the 'other susceptible species') that have consumed meat scraps infected with one of the identified hazards. Indirect exposures were considered in the assessment of 'establishment and/or spread' scenarios, or 'outbreak', scenarios, and are discussed elsewhere in the document (see, Consequence Assessment).

Finally, Figure 7 shows; (a) the consumption of pig meat 'scraps' by pigs, or by other animals excluding humans, and, (b) the consumption of imported pig meat by humans. This IRA did not directly examine the public health risks to humans associated with the direct consumption of imported pig meat. The IRA did, however, consider the role of humans in the epidemiology of exotic diseases and, where relevant, any consequences that may be associated with the indirect exposure of humans to exotic pathogenic agents (zoonoses) amplified or transmitted by susceptible animals. These issues were discussed in the context of 'outbreak scenarios' (see, Consequence Assessment).

Biosecurity Australia liaises with the Department of Health and Ageing and Food Standards Australia New Zealand (FSANZ) on public health issues. Products, intended for human consumption, may undergo a separate risk assessment by FSANZ to determine the public health risks. Imported food must comply with the *Imported Food Control Act 1992* and the *Food Standards Code developed under Food Standards Australia New Zealand Act 1991*. Consequently, AQIS may inspect, sample, hold and test imported pig meat based on issues of public health, including microbial agents or residues of public health concern, and compliance with the *Food Standards Code*.

Exposure assessment for feral pigs

Feral pigs may act as hosts and/or vectors for many of the pathogenic agents considered in this IRA.

The Panel considered that feral pigs would most likely gain access to infected imported pig meat through scavenging meat waste from refuse. Environmental Management Services Pty Ltd (EMS) carried out a consultancy project for the Australian Government Department of Agriculture Fisheries and Forestry entitled *Report on Factors Affecting the Exposure of Australian Animals to Imported Pig Meat* (1999). This report provided information concerning the potential for interaction between human and feral pig populations, and the management of Australian refuse dumps. As these factors vary across Australia, the assessment was stratified into three sectors of the population:

- Remote regions and properties
- Rural regions, towns and settlements
- Large towns

This stratification is based on the Accessibility/Remoteness Index of Australia (ARIA) (Australian Bureau of Statistics, 2001). In the context of this IRA, 'Remote regions and properties' encompasses 'Remote Australia' and 'Very Remote Australia'; 2.9% of the population inhabits these regions. 'Rural regions, towns and settlements' is equivalent to ARIAs 'Outer Regional Australia' classification, and is inhabited by 10.5% of the population. Finally, 'Large towns' comprises 'Major Cities of Australia' and 'Inner Regional Australia' and is inhabited by 86.5% of the population (Australian Bureau of Statistics, 2003).

The exposure assessment was also based on these three sectors. Importantly, a separate annual likelihood of exposing feral pigs was derived for each sector, and these subsequently combined to give an overall exposure assessment for feral pigs.

The exposure assessment for each sector was based on the binomial equation shown below. This equation calculates the likelihood that the generation and disposal of waste for a particular sector (A, B or C) will result in the exposure of at least one feral pig during the period of a year.

$$\text{Annual Likelihood of Entry and Exposure}_{\text{Sector (remote, rural, large towns)}} = 1 - (1 - P)^{N}$$

Where;

P = the probability that each unit of waste discarded in that sector will result in exposure

N = the number of waste units generated and discarded each year in that sector

Probability (P) that each unit of waste will result in exposure

P is a complex variable, and was calculated in the assessment for each of the three sectors as the *product* of the following:

- The likelihood that a waste unit is infected
- The likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection
- The likelihood that the pathogenic agent would remain viable during the period prior to scavenging
- The likelihood that the waste unit would be accessible to a feral pig

- The likelihood that a waste unit would be located by a feral pig

These variables are explained in turn in the text below.

The likelihood that a waste unit is infected

In probability terms, this is equivalent to the result of the release assessment. It follows that this likelihood will not differ amongst sectors (remote, rural and large towns), but was specific to individual disease assessments.

The likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

The quantity of infected meat sufficient to initiate infection will depend upon the concentration of an agent in meat and the oral infectious dose (OID) for that agent.

Both these quantities vary substantially among pathogenic agents although, in most cases, will be determined by the stage and severity of the viraemia / bacteraemia / parasitaemia in the animal from which the meat was derived and, for some disease agents, by the particular carcass cut. Infectious load may also be determined by the proportion of organisms that remain viable at the time the meat is consumed, such that the minimum infectious dose for fresh meat may be substantially different to the minimum infectious dose for discarded meat scraps. Virulence and infectivity are inherent properties of each pathogenic agent, and may also be important determinants of minimum infectious dose.

Where possible, estimates of the sufficient quantity of infected pig meat required to initiate infection were based on robust scientific data. However, there were instances where this value was either unknown or contentious. In these situations, conservative estimates were derived by comparing existing information with that obtained for similar or related pathogenic agents. As is the case for all variables in this analysis, uncertainty in this quantity was represented in the limits of each probability distribution

This likelihood did not differ in a predictable manner amongst the three sectors, but was specific to individual disease assessments.

The likelihood that the pathogenic agent would remain viable during the period prior to scavenging

The likelihood that a pathogenic agent would remain viable after exposure to the environment will depend on the inherent ‘stability’ of each agent. In particular, this likelihood will reflect the agent’s sensitivity to UV light, to ambient temperatures between approximately 10°C and 35°C[24] and to the putrefying effects of saprophytic organisms. It is recognised that pathogenic agents may be protected somewhat from exposure if they are sequestered within bone marrow or within substantial portions of muscle tissue.

This likelihood did not differ in a predictable manner amongst the three sectors, but was specific to individual disease assessments.

[24] While ambient temperature on rural Australian refuse dumps may be as low as -10°C or as high as 50°C (depending on the location and the time of the year), it is reasonable to assume that most discarded meat wastes would experience mean daily temperatures between approximately 10°C to 35°C.

The likelihood that the waste unit would be accessible to a feral pig

This likelihood encompassed factors associated with the security and management of refuse disposal sites. The Panel recognises that feral pigs may occasionally gain access to pig meat scraps other than those disposed of at refuse sites such as those discarded at barbeques or picnics. Nonetheless the vast majority of pig meat wastes will be disposed of at refuse sites and this pathway was considered in the analysis.

The management of refuse disposal in Australia is undergoing a systematic process of improvement as State Governments dictate, and local authorities implement, modern procedures. The EMS consultants found that the NSW Landfill Guidelines produced by the NSW Environment Protection Authority (EPA) to be the most comprehensive and advanced. This document describes four issues that may influence the ability of feral pigs to gain access to human refuse:

- The security of the site
- Compaction of waste
- The regular covering of waste
- Site capping - the final coverage of waste as a dumping area is sealed

The security of a refuse disposal site is the most significant barrier to scavenging by feral pigs. The NSW EPA recommends that (urban) sites receiving more than 250,000 tonnes per annum require a perimeter barrier of no less than 1.8m. Smaller rural sites require a stock-proof perimeter fence and a barrier of no less than 1.8m around active tipping areas. The EMS consultants concluded that few rural refuse disposal facilities achieved this level of protection.

Compaction of waste is carried out to minimise its dispersion and maximise the efficiency of land use. Compaction would also decrease the ability of animals to scavenge material that was not on the surface. The EPA recommends that sites receiving less than 50,000 tonnes per annum (the majority of sites) be compacted to 650kg/m3, and that compaction be carried out prior to covering and/or site capping (see below). The EMS consultants concluded that while compaction to this degree might discourage feral pigs, they are well adapted to digging and would not be deterred if sufficiently motivated by hunger.

The NSW EPA requires that a daily cover of at least 15cm be applied at all manned sites, and that a cover of at least 30cm be applied to sites that will be exposed for more than 90 days without capping (see below). The EMS consultants concluded that because many of the higher risk rural sites will not be manned, this measure is unlikely to reduce the likelihood that feral pigs will scavenge meat scraps. In addition, because feral pigs have an exceptional facility for scent location of food sources and are, as mentioned above, well adapted to digging, the covering of waste to a depth of 15cm is unlikely to be an effective safeguard.

Site capping is a procedure carried out to stabilise areas within a disposal facility where dumping has ceased. The EPA recommends that site capping include a seal-bearing surface, a gas drainage layer, a sealing layer, an infiltration drainage layer and a revegetation layer of at least 2.1m. The EMS consultants concluded that very few rural sites would achieve this degree of stabilisation. Where waste is not stabilised, potential exists for it to move and resurface.

The EMS consultants concluded that when these four factors associated with the management of refuse dumps were considered together, the likelihood of access by feral pigs is greatest for uncontrolled small dumps in remote and rural areas, and for private disposal sites on individual properties.

On the basis of this information the Panel considered that there was a high likelihood that refuse would be accessible to feral pigs in the remote sector. In the rural sector it was considered that there was a moderate likelihood but that there was a very low likelihood that refuse from large towns would be accessible to feral pigs.

The likelihood that a waste unit would be located by a feral pig

This likelihood was derived from factors associated with the abundance of feral pigs in each sector and their proclivity for scavenging from refuse sites, and the volume of other waste commonly found in refuse sites in each sector.

The correlation between the density of feral pigs and the density of humans in each Australian statistical subdivision (SSD) was examined by the EMS consultants. The EMS consultants concluded that whilst interaction was most likely to occur in Northern Queensland, South Western Queensland, the Murray Darling Basin in New South Wales and in various SSDs (statistical sub-division) in the northern part of the Northern Territory, there was a more general correlation between low human population density and high feral pig population density.

This correlation reflects clustering of feral pigs within those rural regions where food, water, topography, vegetation and other factors are most favourable and an inverse relationship between these factors and the density of human settlement. The correlation does not reflect a tendency for feral pigs to avoid human populations. Indeed, it is clear that while regions of maximal interaction can be identified, feral pigs occur throughout the non-arid rural regions of Australia where human habitation in most cases increases the availability and reliability of food and water and thus encourages the establishment of semi-permanent feral pig populations.

In addition to these factors, the Panel noted that pig meat waste is one of many components of refuse that would be attractive to feral pigs. It has been estimated that Australian households dispose of an average of 456 kg of organic compostable waste per household per year (Australian Bureau of Statistics, 2000), resulting in an annual amount of around 3,283 million kg (based on 7.2 million households). Pig meat waste generated per year by households alone is a very small portion of total organic compostable waste.

Overall, the Panel considered that that there was a very low likelihood that any individual pig meat waste unit would be located by a feral pig scavenging in remote regions, extremely low in a rural region, and negligible in a region with large towns.

Number (N) of waste units generated and discarded by each sector during a year

The number (N) of units of pig meat waste generated and discarded in each sector during a year is less complex than P (see above), and will be obtained as the product of the total number of waste units generated and discarded during a year and the proportion of the Australian population that resides in each sector.

The total number of units of pig meat waste was discussed previously (see, Projected Volume of Pig Meat Discarded as Waste). The proportion of the Australian population that resides in each sector (described above) was approximated as shown below:

- Remote regions and properties ≈ 3%
- Rural regions, towns and settlements ≈ 11%
- Large towns ≈ 86%

Table 5 Calculation of the annual likelihood of entry and exposure for feral pigs

Variable	Description and calculation / estimation
$LEE_{Feral\ pigs}$	The (annual) likelihood of entry and exposure for feral pigs $= 1 - (1 - LEE_{Remote\ regions}) \times (1 - LEE_{Rural\ regions}) \times (1 - LEE_{Large\ towns})$
$LEE_{Remote\ regions}$	The annual likelihood of entry and exposure for feral pigs in remote regions $= 1 - (1 - P_{Remote\ regions})^{N\ Remote\ regions}$
$LEE_{Rural\ regions}$	The annual likelihood of entry and exposure for feral pigs in rural regions $= 1 - (1 - P_{Rural\ regions})^{N\ Rural\ regions}$
$LEE_{Large\ towns}$	The annual likelihood of entry and exposure for feral pigs in regions with large towns $= 1 - (1 - P_{Large\ towns})^{N\ Large\ towns}$
$P_{Remote\ regions}$	The probability that each unit of waste discarded in a remote region will result in exposure $= L1 \times L2 \times L3 \times L4_{Remote\ regions} \times L5_{Remote\ regions}$
$N_{Remote\ regions}$	The number of waste units generated and discarded each year in remote regions $= Waste\ units_{Total} \times Population_{Remote\ regions}$
$P_{Rural\ regions}$	The probability that each unit of waste discarded in a rural region will result in exposure $= L1 \times L2 \times L3 \times L4_{Rural\ regions} \times L5_{Rural\ regions}$
$N_{Rural\ regions}$	The number of waste units generated and discarded each year in rural regions $= Waste\ units_{Total} \times Population_{Rural\ regions}$
$P_{Large\ towns}$	The probability that each unit of waste discarded in a large town will result in exposure $= L1 \times L2 \times L3 \times L4_{Large\ towns} \times L5_{Large\ towns}$
$N_{Large\ towns}$	The number of waste units generated and discarded each year in large towns $= Waste\ units_{Total} \times Population_{Large\ towns}$
L1	The likelihood that a waste unit is infected = Release assessment (estimate specific to each disease agent)
L2	The likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection (estimate specific to each disease agent)

Variable	Description and calculation / estimation
L3	The likelihood that the pathogenic agent would remain viable during the period prior to scavenging (estimate specific to each disease agent)
$L4_{\text{Remote regions}}$	The likelihood that the waste unit would be accessible to a feral pig in a remote region = High
$L4_{\text{Rural regions}}$	The likelihood that the waste unit would be accessible to a feral pig in a rural region = Moderate
$L4_{\text{Large towns}}$	The likelihood that the waste unit would be accessible to a feral pig in a region with large towns = Very low
$L5_{\text{Remote regions}}$	The likelihood that a waste unit would be located by a feral pig scavenging in a remote region = Very low
$L5_{\text{Rural regions}}$	The likelihood that a waste unit would be located by a feral pig scavenging in a rural region = Extremely low
$L5_{\text{Large towns}}$	The likelihood that a waste unit would be located by a feral pig scavenging in a region with large towns = Negligible
$\text{Waste units}_{\text{Total}}$	The total number of units of pig meat waste generated and discarded in a year This estimate was derived above (see, Table 4)
$\text{Population}_{\text{Remote regions}}$	The proportion of the Australian population that resides in remote regions = 3%
$\text{Population}_{\text{Rural regions}}$	The proportion of the Australian population that resides in rural regions = 11%
$\text{Population}_{\text{Large towns}}$	The proportion of the Australian population that resides in regions with large towns = 86%

Exposure assessment for backyard pigs

In this analysis, the colloquial term ‘backyard pig producers’ was used to describe enterprises with less than ten sows. This group of producers is very diverse as regards management and feeding practices and has, at least traditionally, been associated with a higher likelihood of illegal swill feeding than other categories of pig producers. Pigs kept in backyard enterprises

would generally be slaughtered and consumed, although it is recognised that some breeding and distribution of young pigs or slaughter age pigs may occur. For the purposes of this analysis, it was assumed that meat wastes fed to backyard pigs were derived from the household associated with those pigs.

The exposure assessment for backyard pigs was based on the binomial equation shown below. This equation calculates the likelihood that exposure of backyard pigs will result from the generation and disposal of waste by backyard pig producers during the period of a year.

$$\text{Annual Likelihood of Entry and Exposure}_{\text{Backyard pigs}} = 1 - (1 - P)^{N}$$

Where;

P = the probability that each unit of waste discarded by a backyard pig producer will result in exposure

N = the number of waste units that may be fed each year to backyard pigs

Probability (P) that each unit of waste will result in exposure

P is a complex variable, and was calculated in the assessment as the *product* of the following:

- The likelihood that a waste unit is infected;
- The likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection; and
- The likelihood that the pathogenic agent would remain viable during the period prior to ingestion.

These variables are explained in turn in the text below.

The likelihood that a waste unit is infected

In probability terms, this is equivalent to the result of the release assessment. It follows that this likelihood was specific to individual disease assessments.

The likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

The quantity of infected meat sufficient to initiate infection will depend upon the concentration of an agent in meat and the oral infectious dose (OID) for that agent.

Both these quantities vary substantially among pathogenic agents although, in most cases, will be determined by the stage and severity of the viraemia / bacteraemia / parasitaemia in the animal from which the meat was derived and, for some disease agents, by the particular carcass cut. Infectious load may also be determined by the proportion of organisms that remain viable at the time the meat is consumed, such that the minimum infectious dose for fresh meat may be substantially different to the minimum infectious dose for discarded meat scraps. Virulence and infectivity are inherent properties of each pathogenic agent, and may also be important determinants of minimum infectious dose.

Where possible, estimates of the sufficient quantity of infected pig meat required to initiate infection were based on robust scientific data. However, there were instances where this value was either unknown or contentious. In these situations, conservative estimates were derived by comparing existing information with that obtained for similar or related pathogenic agents. As

was the case for all variables in this analysis, uncertainty in this quantity was represented in the limits of each probability distribution.

This likelihood was specific to individual disease assessments.

The likelihood that the pathogenic agent would remain viable during the period prior to ingestion

The likelihood that a pathogenic agent will remain viable after exposure to the environment will depend on the inherent 'stability' of each agent. In particular, this likelihood will reflect the agent's sensitivity to UV light, to ambient temperatures between approximately 10°C and 35°C and to the putrefying effects of saprophytic organisms.

It is recognised that pathogenic agents may be protected somewhat from exposure if they are sequestered within bone marrow or within substantial portions of muscle tissue. However, it is also recognised that meat scraps may undergo some putrefaction in garbage during the period between trimming of meat for cooking, or the accumulation of table scraps, and the subsequent feeding of backyard pigs.

This likelihood was specific to individual disease assessments.

Number (N) of waste units fed to backyard pigs during a year

The number (N) of units of pig meat waste generated and fed to backyard pigs during a year is less complex than P (see above), and was obtained as the product of:

- The total number of waste units generated and discarded by households during a year;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

The total number of waste units generated and discarded by households during a year (Waste units $_{HH}$) was calculated as the product of the total number of waste units (Waste units $_{Total}$) and the proportion of these that was derived from households (Prop $_{HH}$). These component estimates were discussed elsewhere (see, Projected Volume of Pig Meat Discarded as Waste).

The proportion of the total waste that is generated by backyard pig producers was obtained by dividing the number of backyard pig producers by the total number of Australian households.

- Australian pig industry statistics[25] identify 778 premises with less than 10 sows. In order to incorporate the uncertainty about this estimate, the number of backyard pig producers were modelled as a Pert distribution with a minimum of 739 (95% of 778), a most likely value of 778 and a maximum of 817 (105% of 778).
- The Australian population of 7.2 million households were modelled similarly as a Pert distribution with a minimum of 6.8 million households (95% of 7.2 million), a most likely value of 7.2 million and a maximum of 7.6 million households (105% of 7.2 million).

Because feeding meat and table scraps is illegal in Australia, and prosecuted severely under State or Territory legislation, the proportion of backyard pig producers who participate in this practice was extremely difficult to estimate with precision. The proportion derived by the Panel from the history of prosecutions, and consideration of the difficulty in identifying and convicting perpetrators, was considered to be very low.

[25] Pig Stats 2000 and 2001. Australian Pork Limited, Canberra, 2002.

Table 6 Calculation of the annual likelihood of entry and exposure for backyard pigs

Variable	Description and calculation / estimation
$LEE_{Backyard\ pigs}$	The (annual) likelihood of entry and exposure for backyard pigs $= 1 - (1 - P_{Backyard\ pigs})^{N\ Backyard\ pigs}$
$P_{Backyard\ pigs}$	The probability that each unit of waste fed to backyard pigs will result in exposure $= L1 \times L2 \times L3_{Backyard\ pigs}$
$N_{Backyard\ pigs}$	The number of waste units that may be fed to backyard pigs during a year $= Waste\ units_{HH} \times Prop_{BP\ producers} \times Prop_{Feed\ swill\ BP}$
L1	The likelihood that a waste unit is infected = Release assessment (estimate specific to each disease agent)
L2	The likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection (estimate specific to each disease agent)
$L3_{Backyard\ pigs}$	The likelihood that the pathogenic agent would remain viable during the period prior to ingestion (estimate specific to each disease agent)
$Waste\ units_{HH}$	The total number of waste units generated and discarded by households during a year $= Waste\ units_{Total} \times Prop_{HH}$
$Waste\ units_{Total}$	The total number of units of pig meat waste generated and discarded in a year This estimate was derived above (see, Table 4)
$Prop_{HH}$	The proportion of imported pig meat likely to be purchased by households as fresh meat and smallgoods $= 1 - Prop_{FSE}$
$Prop_{FSE}$	The proportion of imported pig meat likely to be purchased by food service establishments as fresh meat and smallgoods = Pert (0.15, 0.25, 0.30)
$Prop_{BP\ producers}$	The proportion of the total waste that is generated by households that keep backyard pigs $= \frac{Pert(739, 778, 817)}{Pert(6.8\,million, 7.2\,million, 7.6\,million)}$
$Prop_{Feed\ swill\ BP}$	The proportion of backyard pig producers that may illegally feed waste to their pigs = Very low

Exposure assessment for small commercial piggeries

In this analysis, small commercial piggeries are those that keep less than 99 sows. Enterprises in this group are considered very diverse as regards their management (intensive or extensive) and feeding practices. In particular, pigs on small holdings may be housed intensively or allowed free range. This group of producers is generally considered less likely to feed scraps illegally on a casual basis, because the husbandry of at least 10 sows will generally require a planned approach to maintenance feeding. Swill feeding has, however, been reported in piggeries of this size, and may be more commonly associated with regular access to waste from restaurants or other food service establishments.

The exposure assessment for small commercial piggeries was based on the binomial equation shown below. This equation calculates the annual likelihood that exposure of pigs in small commercial piggeries will result from the feeding of pig meat waste during the period of a year.

$$\text{Annual Likelihood of Entry and Exposure}_{\text{Pigs in small commercial piggeries}} = 1 - (1 - P)^{N}$$

Where;

P = The probability that each unit of waste fed to pigs in small commercial piggeries will result in exposure

N = the number of waste units that may be fed each year to pigs in small commercial piggeries

Probability (P) that each unit of waste will result in exposure

P is a complex variable, and was calculated in the assessment as the *product* of the following:

- The likelihood that a waste unit is infected;
- The likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection; and
- The likelihood that the pathogenic agent would remain viable during the period prior to ingestion.

These variables are explained in turn in the text below.

The likelihood that a waste unit is infected

In probability terms, this is equivalent to the result of the release assessment. It follows that this likelihood was specific to individual disease assessments.

The likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

The quantity of infected meat sufficient to initiate infection will depend upon the concentration of an agent in meat and the oral infectious dose (OID) for that agent.

Both these quantities vary substantially among pathogenic agents although, in most cases, will be determined by the stage and severity of the viraemia / bacteraemia / parasitaemia in the animal from which the meat was derived and, for some disease agents, by the particular carcass cut. Infectious load may also be determined by the proportion of organisms that remain viable

at the time the meat is consumed, such that the minimum infectious dose for fresh meat may be substantially different to the minimum infectious dose for discarded meat scraps. Virulence and infectivity are inherent properties of each pathogenic agent, and may also be important determinants of minimum infectious dose.

Where possible, estimates of the sufficient quantity of infected pig meat required to initiate infection were based on robust scientific data. However, there were instances where this value was either unknown or contentious. In these situations, conservative estimates were derived by comparing existing information with that obtained for similar or related pathogenic agents. As was the case for all variables in this analysis, uncertainty in this quantity was represented in the limits of each probability distribution.

This likelihood was specific to individual disease assessments.

The likelihood that the pathogenic agent would remain viable during the period prior to ingestion

The likelihood that a pathogenic agent will remain viable after exposure to the environment will depend on the inherent 'stability' of each agent. In particular, this likelihood will reflect the agent's sensitivity to UV light, to ambient temperatures between approximately 10°C and 35°C and to the putrefying effects of saprophytic organisms.

It is recognised that pathogenic agents may be protected somewhat from exposure if they are sequestered within bone marrow or within substantial portions of muscle tissue. However, it is also recognised that meat scraps may undergo some putrefaction in garbage during the period between trimming of meat for cooking, or the accumulation of table scraps, and the subsequent feeding of pigs in small commercial piggeries.

This likelihood was specific to individual disease assessments.

Number (N) of waste units fed to pigs in small commercial piggeries during a year

The number (N) of units of pig meat waste generated and fed illegally to pigs in small commercial piggeries was difficult to estimate with precision, as it includes both household waste and waste from food service establishments.

The number of household waste units and the number of units from food service establishments were estimated independently, and subsequently summed.

The number of household waste units that may be fed to pigs in small commercial piggeries was calculated as shown in the formula below:

$$\text{Waste units}_{\text{HH source}} = \left[\frac{\text{No.}_{\text{SCP}} \times \text{Prop}_{\text{Feed swill SCP}} \times \text{No.}_{\text{Affiliated HH}}}{\text{HH}_{\text{Total}}} \right] \times \text{Waste units}_{\text{HH}}$$

Where;

No._{SCP} = the number of small commercial piggeries in Australia

$\text{Prop}_{\text{Feed swill SCP}}$ = the proportion of small commercial piggeries that may feed waste illegally

$No._{Affiliated\ HH}$ = the number of households whose waste may be used by a small commercial piggery that feeds waste illegally

HH_{Total} = the total number of Australian households

$Waste\ units_{HH}$ = the total number of waste units generated and discarded by households during a year

The number of waste units from food service establishments that may be fed to pigs in small commercial piggeries was calculated as shown in the formula below:

$$Waste\ units_{FSE\ source} = \left[\frac{No._{SCP} \times Prop_{Feed\ swill\ SCP} \times No._{Affiliated\ FSE}}{FSE_{Total}} \right] \times Waste\ units_{FSE}$$

Where;

$No._{SCP}$ = the number of small commercial piggeries in Australia

$Prop_{Feed\ swill\ SCP}$ = the proportion of small commercial piggeries that may feed waste illegally

$No._{Affiliated\ FSE}$ = the number of food service establishments whose waste may be used by a small commercial piggery that feeds waste illegally

FSE_{Total} = the total number of food service establishments

$Waste\ units_{FSE}$ = the total number of waste units generated and discarded by food service establishments during a year

Estimates for the terms in these formulae are as follows:

- **$No._{SCP}$**: Australian pig industry statistics[26] identify 1,212 premises with between 10 and 99 sows. In order to incorporate the uncertainty about this estimate, the number of small commercial pig producers was modelled as a Pert distribution with a minimum of 1,151 (95% of 1,212), a most likely value of 1,212 and a maximum of 1,272 (105% of 1,212).
- **$Prop_{Feed\ swill\ SCP}$**: Because feeding meat and table scraps is illegal in Australia, and prosecuted severely under State or Territory legislation, the proportion of small commercial pig producers who participate in this practice was extremely difficult to estimate with precision. The proportion derived by the Panel from the history of prosecutions, and in consideration of the difficulty in identifying and convicting perpetrators, was considered to be very low.
- **$No._{Affiliated\ HH}$**: The number of households whose waste may be used by a small commercial piggery that feeds waste illegally was difficult to estimate with precision. Recognising that small commercial piggeries may be associated with extended family groups, this number was modelled as a Pert distribution with a minimum value of 1, a most likely value of 3 and a maximum value of 5.
- **$No._{Affiliated\ FSE}$**: After consideration of the illegality of swill feeding, and the need for food service establishments to avoid prosecution, it was estimated that each small piggery would be extremely unlikely to obtain waste from more than a single food service establishment.

[26] Pig Stats 2000 and 2001. Australian Pork Limited, Canberra, 2002.

- **HH $_{Total}$**: The Australian population of 7.2 million households was modelled as a Pert distribution with a minimum of 6.8 million households (95% of 7.2 million), a most likely value of 7.2 million and a maximum of 7.6 million households (105% of 7.2 million).
- **FSE $_{Total}$**: The total number of food service establishments in Australia was estimated as 37,304 premises on the basis of Australian Bureau of Statistics surveys. This included cafes, restaurants, catering, and take-away premises, hotels, taverns, bars and clubs (hospitality). The number of food service establishments was modelled as a Pert distribution with a minimum of 35,439 premises (95% of 37,304), a most likely value of 37,304 and a maximum of 39,169 premises (105% of 37,304).
- **Waste units $_{HH}$**: The total number of waste units generated and discarded by households during a year was described in the assessment for backyard pigs (see above) as the product of the total number of waste units (Waste units $_{Total}$) and the proportion of these that derived from households (Prop $_{HH}$). These component estimates are, in turn, discussed elsewhere (see, Projected Volume of Pig Meat Discarded as Waste).
- **Waste units $_{FSE}$**: The total number of waste units generated and discarded by food service establishments during a year was calculated as the product of the total number of waste units (Waste units $_{Total}$) and the proportion of these that will be derived from food service establishments (Prop $_{FSE}$). These component estimates were discussed elsewhere (see, Projected Volume of Pig Meat Discarded as Waste).

Table 7 Calculation of the annual likelihood of entry and exposure for small commercial piggeries

Variable	Description and calculation / estimation
LEE_{SCP}	The (annual) likelihood of entry and exposure for small commercial piggeries $= 1 - (1 - P_{SCP})^{N\ SCP}$
P_{SCP}	The probability that each unit of waste fed to pigs in small commercial piggeries will result in exposure $= L1 \times L2 \times L3_{SCP}$
N_{SCP}	The number of waste units that may be fed to pigs in small commercial piggeries during a year $= Waste_{HH\ source} + Waste_{FSE\ source}$
L1	The likelihood that a waste unit is infected = Release assessment (estimate specific to each disease agent)
L2	The likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection (estimate specific to each disease agent)
$L3_{SCP}$	The likelihood that the pathogenic agent would remain viable during the period prior to ingestion (estimate specific to each disease agent)
$Waste_{HH\ source}$	The number of household waste units that may be fed to pigs in small commercial piggeries $= \left[\frac{No._{SCP} \times Prop_{Feed\ swill\ SCP} \times No._{Affiliated\ HH}}{HH_{Total}}\right] \times Waste\ units_{HH}$
$Waste_{FSE\ source}$	The number of waste units from food service establishments that may be fed to pigs in small commercial piggeries $= \left[\frac{No._{SCP} \times Prop_{Feed\ swill\ SCP} \times No._{Affiliated\ FSE}}{FSE_{Total}}\right] \times Waste\ units_{FSE}$
$No._{SCP}$	The number of small commercial piggeries in Australia = Pert (1151, 1212, 1272)
$Prop_{Feed\ swill\ SCP}$	the proportion of small commercial piggeries that may feed waste illegally = Very low
$No._{Affiliated\ HH}$	The number of households whose waste may be used by a small commercial piggery that feeds waste illegally = Pert (1, 3, 5)

Variable	Description and calculation / estimation
$No._{Affiliated\ FSE}$	The number of food service establishments whose waste may be used by a small commercial piggery that feeds waste illegally = 1
HH_{Total}	The total number of Australian households = Pert (6.8 million, 7.2 million, 7.6 million)
FSE_{Total}	The total number of food service establishments = Pert (35 439, 37 304, 39 169)
$Waste\ units_{HH}$	The total number of waste units generated and discarded by households during a year = $Waste\ units_{Total}$ x $Prop_{HH}$
$Waste\ units_{FSE}$	The total number of waste units generated and discarded by food service establishments during a year = $Waste\ units_{Total}$ x $Prop_{FSE}$
$Waste\ units_{Total}$	The total number of units of pig meat waste generated and discarded in a year This estimate was derived above (see, Table 4)
$Prop_{HH}$	The proportion of imported pig meat likely to be purchased by households as fresh meat and smallgoods = 1 - $Prop_{FSE}$
$Prop_{FSE}$	The proportion of imported pig meat likely to be purchased by food service establishments as fresh meat and smallgoods = Pert (0.15, 0.25, 0.30)

Exposure assessment for 'other susceptible species'

The final 'exposure group' is less clearly defined than are those above. This group excludes humans, which are not considered at risk from the ingestion of 'meat scraps' (Figure 7), but includes species such as rats, domestic carnivores, carnivorous bird life, etc. It was expected that the exposure assessment for this group would vary to some extent among the identified pathogenic agents, thus this was discussed in the assessments for the relevant pathogenic agents.

Summary: exposure assessments

The assessments detailed above gave rise to an annual likelihood of entry and exposure for each of the exposure groups.

It is explained elsewhere in this document (see, Risk Estimation) that these likelihoods provided the likelihood component in the calculation of 'partial annual risk' for each exposure group. The partial risks were subsequently combined to give an overall estimate of 'unrestricted annual risk'.

Consequence assessment

According to the *OIE Code*, a consequence assessment should '*describe the potential consequences of a given exposure, and estimate the probability of them occurring*'.

The 'potential consequences of an exposure' may be accrued in direct and indirect ways. The direct and indirect consequences considered in this analysis are shown below.

Direct consequences

These describe direct harm to:

- the life or health (including production effects) of production, domestic or feral animals; and
- the environment, including the life or health of native animals, and any direct impacts on the non-living environment (Annex D).

Indirect consequences

Indirect consequences are the costs resulting from natural or human processes associated with the incursion of a disease. These include:

- new or modified eradication, control, surveillance or monitoring and compensation strategies or programs;
- domestic trade or industry effects, including changes in consumer demand and impacts on other industries supplying inputs to, or utilising outputs from, directly affected industries;
- international trade effects, including loss of markets, meeting new technical requirements to enter or maintain markets and changes in international consumer demand;
- indirect impacts on the environment (see below), including biodiversity, endangered species, the integrity of ecosystems; and
- indirect impacts on communities, including reduced tourism, reduced rural and regional economic viability and loss of social amenity, and any 'side impacts' of control measures.

A range of factors is relevant to the consideration of harm to the environment. This includes harm arising from the impact of the disease agent itself, as well as from any treatments or procedures used to control it. The extent of harm was evaluated taking into account the circumstances of the particular hazard, and using the factors outlined below:

- all on-site and off-site impacts;
- the geographical scope and magnitude of the impact;
- the frequency and duration of the action causing the harm;
- the total impact which can be attributed to that action over the entire geographic area affected, and over time (i.e. cumulative impact);
- any synergistic effect of hazards on impact;
- reversibility of the impact;
- the sensitivity of the receiving environment (recognised environmental features of high sensitivity); and
- the degree of confidence with which the impacts of the action are known and understood.

The direct and indirect consequences described above collectively cover the *economic*, *environmental* and *social* effects of a disease. Given this, the consequences are also mutually exclusive — that is, an effect was not assessed more than once. In particular, the direct impacts of a disease on a native species were assessed under the criterion describing the '*environment, including the life or health of native animals and plants'*, whereas the indirect or 'flow-on' effects on the environment were assessed under the second last indirect criterion.

Describing direct and indirect disease effects

Each direct and indirect consequence was estimated at four levels, local, district or regional, State or Territory and national, and the values derived subsequently translated into a single qualitative score (A-G). In this context, the terms 'national', 'State or Territory', 'district or regional' and 'local', were defined as follows.

National:	Australia-wide;
State/Territory:	an Australian 'state' (New South Wales, Victoria, Queensland, Tasmania, South Australia or Western Australia) or 'territory' (the Australian Capital Territory, the Northern Territory, the Australian Antarctic Territory and other Australian Territories covered under the Act)[27];
District or region:	a geographically or geopolitically associated collection of aggregates — generally a recognised section of a state, such as the 'North West Slopes and Plains, NSW' or 'Far North Queensland'; and
Local:	an aggregate of households or enterprises — e.g. a rural community, a town or a local government area.

At each level, the magnitude of impact was described as 'unlikely to be discernible', of 'minor significance', 'significant' or 'highly significant':

- an '*unlikely to be discernible*' impact is not usually distinguishable from normal day-to-day variation in the criterion[28];
- an impact of '*minor significance*' is recognisable, but minor and reversible;
- a '*significant*' impact is serious and substantive, but reversible and unlikely to disturb either economic viability or the intrinsic value of the criterion; and
- a '*highly significant*' impact is extremely serious and irreversible and likely to disturb either economic viability or the intrinsic value of the criterion.

To estimate the consequences on a national scale, it was necessary to describe carefully the outbreak scenarios upon which the consequence assessments were to be based. The first step was to assess the magnitude of a direct or indirect impact on the national economy or the Australian community. This will often differ markedly from the effect of the disease on the local, district or regional, State/Territory or national population of directly affected parties. If, for the particular criterion, there was no discernible impact at a national level, then, in descending order, the magnitude of impact at the State/Territory, district or local level was investigated.

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[27] This excludes the Cocos Islands.

[28] Criterion refers to the individual direct and indirect consequences (i.e. animal health, environment, control programs etc).

The impact of a disease at a given level in more than one State/Territory, district or local area was considered to represent the same magnitude of impact at the level above. At each of the lower levels, an impact more serious than 'minor' was deemed to be discernible at the level above.

Estimates of the consequences of the introduction, establishment and/or spread at the national, State/Territory, district/region and local level were subsequently translated to an overall score (A-G) using the schema outlined in Table 8.

Table 8 The assessment of direct or indirect consequences on a national scale

Impact score	National	State or Territory	District or region	Local
G	**Highly significant**[1]	-	-	-
F	**Significant**	-	-	-
E	**Minor**	-	-	-
D	Unlikely to be discernible	**Minor**	-	-
C	-	Unlikely to be discernible	**Minor**	-
B	-	-	Unlikely to be discernible	**Minor**
A	-	-	-	**Unlikely to be discernible**
	National	*State or Territory*	*District or region*	*Local*

Level

[1] Shaded cells with bold font are those that dictate national impact scores

Consequence assessment for uncooked pig meat

Consequence assessments for each of the identified hazards were carried out in the following stages:

- identification of plausible 'outbreak scenarios' for each exposure group (feral pigs, backyard pigs, pigs in small commercial piggeries and 'other susceptible species');
- estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and/or spread);
- *for each outbreak scenario*, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- *for each outbreak scenario*, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- combination of the 'likelihood' and 'consequences' of each outbreak scenario, to give an estimate of 'likely consequences';

- combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group; and
- assessment of consequences to human life or health as described below.

These steps are discussed in turn.

Outbreak scenarios for each exposure group

In this analysis, an 'outbreak scenario' represents a particular level of 'establishment and/or spread'. While it was understood that the extent and direction of disease establishment and/or spread will be, in reality, both complex and continuous, it was none-the-less considered useful to categorise this aspect of the analysis so as to approach the assessment of consequences in a practical manner.

Outbreak scenarios for each of the exposure groups are outlined below. For each group, the first scenario denotes 'no further establishment or spread'. The purpose of this category was to ensure that the sum of likelihoods assigned to outbreak scenarios for that group would always be one. It was also acknowledged that for some diseases the consequences would be measurable even without establishment or spread. For some pathogenic agents, outbreak scenarios were used that differed from the generic scenarios described below. These were described within the individual assessments.

The descriptions of outbreak scenarios use the term 'secondary spread' to describe a range of means by which disease may be transmitted from pigs that have consumed infected meat scraps to other pigs or to other susceptible species (including humans[29]). In the terminology that is used throughout this analysis, animals infected as a result of secondary spread were considered to have been 'indirectly exposed' to the contaminating pathogenic agent. Mechanisms for secondary spread will vary among pathogenic agents, but include direct contact, fomites, aerosol plumes, insect vectors and iatrogenic means. Likewise, intermediate hosts and/or other more complex transmission or life cycle components may be relevant.

Outbreak scenarios for the exposure of feral pigs

The Panel acknowledged that a wide range of outbreak scenarios may arise from the exposure of feral pigs to infected pig meat scraps. Given this, the four possibilities outlined below were generally thought to encapsulate outcomes likely to be the most significant:

1. containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;

2. secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;

3. secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and

4. secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

[29] Humans were considered in this IRA if relevant as a species to the epidemiology of a disease or to the consequences of exposure of other susceptible species. The likelihood and consequences of the direct exposure of humans to contaminated pig meat were not considered.

Outbreak scenarios for the exposure of backyard pigs

In contrast to feral pigs, exposure of backyard pigs is likely to have outcomes that are more predictable. These were categorised as:

1. containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Outbreak scenarios for the exposure of small commercial piggeries

Outbreak scenarios for small commercial piggeries are likely to be similar to those described for backyard enterprises. The following scenarios were described:

1. containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;
2. secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Outbreak scenarios for the exposure of 'other susceptible species'

Given the range of 'other susceptible species' that may be directly exposed to infected pig meat, outbreak scenarios for this group were difficult to generalise. The following scenarios were generally used in the consequence assessments:

1. containment of the disease within the sub-population of directly exposed animals - no secondary spread - this option represents the 'no outbreak' scenario;
2. secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. secondary spread (via feral pigs or other means) to a local population of domestic pigs - containment within a local population of backyard enterprises or small commercial

piggeries - spread to other susceptible species - spread to humans if the agent is zoonotic; and

4. secondary spread (via feral pigs or other means) to a more general population of domestic pigs - exposure of medium-large commercial piggeries and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Estimating the likelihood of each outbreak scenario

An approximation, using the qualitative descriptors as outlined in Table 2, was provided for the likelihood that each identified outbreak scenario would occur. For any given pathogenic agent, the sum of these likelihoods equalled '1'.

Evaluating the consequences according to each direct and indirect criterion

The consequences according to each direct and indirect criterion were evaluated and reported using the qualitative method described at the start of this section (see, Describing direct and indirect disease effects).

Estimating the consequences associated with each outbreak scenario

The measure of impact obtained for each direct and indirect criterion was combined to give the overall consequences of a disease agent. The following rules were used for the combination of direct and indirect impacts. These rules are mutually exclusive, and were addressed in the order that they appear in the list — i.e. *if the first set of conditions did not apply, the second set was considered; if the second set did not apply, the third set was considered; and so forth, until one of the rules applied*.

1. Where the impact of a disease agent with respect to any direct or indirect criterion was G, the overall impact was extreme;
2. Where the impact of a disease agent with respect to more than one criterion was F, the overall impact was extreme;
3. Where the impact of a disease agent with respect to a single criterion was F and the impact with respect to each remaining criterion was E, the overall impacts was extreme;
4. Where the impact of a disease agent with respect to a single criterion was F and the impact with respect to remaining criteria was not unanimously E, the overall impact was high;
5. Where the impact of a disease agent with respect to all criteria was E, the overall impact was high;
6. Where the impact of a disease agent with respect to one or more criteria was E, the overall impact was moderate;
7. Where the impact of a disease agent with respect to all criteria was D, the overall impact was moderate;
8. Where the impact of a disease agent with respect to one or more criteria was D, the overall impact was low;
9. Where the impact of a disease agent with respect to all criteria was C, the overall impact was low;

10. Where the impact of a disease agent with respect to one or more criteria was C, the overall impact was very low;
11. Where the impact of a disease agent with respect to all criteria was B, the overall impact was very low;
12. Where the impact of a disease agent with respect to one or more criteria was B, the overall impact was negligible;
13. Where the impact of a disease agent with respect to all criteria was A, the overall impact was negligible.

Evaluating the 'likely consequences' associated with each outbreak scenario

The 'likely consequences' of an event describes the product of the likelihood that it will occur and the magnitude of its impact. In the context of this analysis, the likely consequences of an outbreak scenario represented the combination of the 'likelihood that the scenario would occur' and an estimate of the 'consequences associated with that scenario'. These measures were derived using the approach described in the discussions above and combined using the matrix in Table 9 to give an estimate of the 'likely consequences' associated with each outbreak scenario'.

Table 9 A matrix for estimating the 'likely consequences' for each outbreak scenario

Likelihood that the scenario would occur	Consequences associated with that scenario: Negligible	Very low	Low	Moderate	High	Extreme
High	Negligible	Very low	Low	Moderate	High	Extreme
Moderate	Negligible	Very low	Low	Moderate	High	Extreme
Low	Negligible	Negligible	Very low	Low	Moderate	High
V. Low	Negligible	Negligible	Negligible	Very low	Low	Moderate
E. Low	Negligible	Negligible	Negligible	Negligible	Very low	Low
Negligible	Negligible	Negligible	Negligible	Negligible	Negligible	Very low

Evaluating the 'likely consequences' of exposing each group of susceptible animals

Having obtained estimates for the 'likely consequences' associated with each outbreak scenario it thus then remained to combine these to give an estimate of the outcome expected when each of the four groups of susceptible animals was exposed.

For each of the four exposure groups, the likely consequences associated with outbreak scenarios were combined using the set of 11 rules outlined below. These rules are mutually exclusive, and were addressed in the order that they appear in the list. For example, *if the first set of conditions did not apply, the second set was considered. If the second set did not apply, the third set was considered* ..., and so forth until one of the rules applied.

1. Where the likely consequences for any outbreak scenario were ‘extreme’, the overall likely consequences were also considered to be ‘extreme’;
2. Where the likely consequences for more than one outbreak scenario were ‘high’, the overall likely consequences were considered to be ‘extreme’;
3. Where the likely consequences for a single outbreak scenario was ‘high’ and the likely consequences for each remaining scenario were ‘moderate’, the overall likely consequences were considered to be ‘extreme’;
4. Where the likely consequences for a single outbreak scenario was ‘high’ and the likely consequences for remaining scenarios were not unanimously ‘moderate’, the overall likely consequences were considered to be ‘high’;
5. Where the likely consequences for all outbreak scenarios were ‘moderate’, the overall likely consequences were considered to be ‘high’;
6. Where the likely consequences for one or more outbreak scenarios were ‘moderate’, the overall likely consequences were considered to be ‘moderate’;
7. Where the likely consequences for all outbreak scenarios were ‘low’, the overall likely consequences were considered to be ‘moderate’;
8. Where the likely consequences for one or more outbreak scenarios were ‘low’, the overall likely consequences were considered to be ‘low’;
9. Where the likely consequences for all outbreak scenarios were ‘very low’, the overall likely consequences were considered to be ‘low’;
10. Where the likely consequences for one or more outbreak scenarios were ‘very low’, the overall likely consequences were considered to be ‘very low’;
11. Where the likely consequences for all outbreak scenarios were ‘negligible’, the overall likely consequences were considered to be ‘negligible’.

The outcome of this final step in the consequence assessment will thus be an estimate for ‘the likely consequences of exposing each of the identified groups of susceptible animals’ to a given pathogenic agent.

Assessment of consequences to human life or health

The consequences of a pest or disease to human life or health were considered separately to its economic, environmental and social effects. This was because jurisdiction for regulation of trade on matters of human life or health does not rest with Biosecurity Australia.

Biosecurity Australia consults with the Australian Department of Health and Ageing and Food Standards Australia and New Zealand (FSANZ), on the assessments for ‘zoonotic’ pests or diseases that may establish in Australia’s animal population through the importation of pig meat. At the discretion of the Director of Human Quarantine, this may result in a requirement for biosecurity measures to manage the risk to human life or health associated with the importation of pig meat.

Risk estimation

In the context of this analysis, 'risk estimation' describes the integration of likelihood evaluation and consequence assessment, with the objective of deriving a unit to represent the risk associated with each pathogenic agent.

Risk estimation for each identified pathogenic agent was obtained in two stages:

- estimation of the 'partial annual risk' associated with each of the exposure groups
- combination of partial annual risks to give an estimate of 'overall annual risk'

Estimation of partial annual risks

The annual risk associated with *each exposure group* was obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' obtained from the consequence assessment for that exposure group.

Combination of likelihood and consequences was undertaken using the 'rules' shown in the risk estimation matrix in Table 10 below. The principle underlying this matrix is that the cells are expressed in the units, and represent the 'expected loss' associated with a particular combination of likelihood and consequences. It stands to reason that expected loss cannot exceed the consequence that would be accrued were the event not associated with a probability. Given this, the extent to which consequence was reduced by multiplying it by the probability of occurrence was determined by the magnitude of that probability.

In view of the imprecision inherent in an essentially qualitative assessment, it was assumed that probabilities greater than or equal to Biosecurity Australia's definition of 'Moderate' were not sufficiently small to reduce consequences *within the limits of measurement*. This means that the first two rows of the matrix mirror the consequence scale on the horizontal axis. The remaining levels of probability - that is, 'Low', 'Very Low', 'Extremely Low' and 'Negligible' - reduced the consequences by one, two, three and four categories, respectively, or to 'Negligible.

Table 10 Risk estimation matrix: estimation of the partial annual risk of exposure

Likelihood of entry and exposure	Negligible impact	Very low	Low	Moderate	High	Extreme impact
High likelihood	Negligible risk	Very low risk	Low risk	Moderate risk	High risk	Extreme risk
Moderate	Negligible risk	Very low risk	Low risk	Moderate risk	High risk	Extreme risk
Low	Negligible risk	Negligible risk	Very low risk	Low risk	Moderate risk	High risk
Very low	Negligible risk	Negligible risk	Negligible risk	Very low risk	Low risk	Moderate risk
Extremely low	Negligible risk	Negligible risk	Negligible risk	Negligible risk	Very low risk	Low risk
Negligible likelihood	Negligible risk	Negligible risk	Negligible risk	Negligible risk	Negligible risk	Very low risk

Consequences of entry, establishment or spread

Estimation of overall annual risk

The partial annual risk of exposure obtained for each of the four exposure groups were combined to give an overall estimate of annual risk. This was undertaken using the 11 rules outlined below. The rules are mutually exclusive, and were therefore addressed in the order that they appear in the list. For example, *if the first set of conditions did not apply, the second set was considered. If the second set did not apply, the third set was considered* ..., and so forth until one of the rules applied.

1. Where any one partial annual risk was extreme, the overall annual risk was also considered extreme;
2. Where more than one partial annual risk was high, the overall annual risk was considered extreme;
3. Where any one partial annual risk high and each remaining partial annual risk was moderate, the overall annual risk was considered extreme;
4. Where a single partial annual risk was high and the remaining partial annual risks were not unanimously moderate, the overall annual risk was considered high;
5. Where all partial annual risks were moderate, the overall annual risk was considered high;
6. Where one or more partial annual risks were moderate, the overall annual risk was considered moderate;
7. Where all partial annual risks were low, the overall annual risk was considered moderate;
8. Where one or more partial annual risks were considered low, the overall annual risk was considered low;

9. Where all partial annual risks were very low, the overall annual risk was considered low;
10. Where one or more partial annual risks were very low, the overall annual risk was considered very low;
11. Where all partial annual risks were negligible, the overall annual risk was considered negligible.

The result of this process was an estimate of the 'unrestricted annual risk of introducing a given disease into Australia as a result of the decision to import pig meat'. This was considered the final output of the risk assessment.

METHOD FOR RISK MANAGEMENT

Risk management is the process of identifying and implementing measures to mitigate risks so as to achieve Australia's appropriate level of protection, while ensuring that any negative affects on trade are minimised.

To implement risk management appropriately, it is necessary to formalise the difference between 'unrestricted' and 'restricted' risk estimates. Unrestricted risk estimates are those derived in the absence of any risk management or using only internationally accepted baseline risk management strategies. In contrast, restricted or mitigated risk estimates are those derived when 'risk management' is applied.

The result of the generic 'risk assessment' for uncooked pig meat was an unrestricted risk estimate for each of the disease agents identified as hazards. This was then be compared with Australia's appropriate level of protection, which is shown in the risk estimation matrix (Table 10) as the band of cells associated with a 'very low' risk. This step is termed 'risk evaluation'. An unrestricted risk that was either 'negligible' or 'very low' meets Australia's appropriate level of protection and was considered 'acceptable'. In this situation, risk management was not justified. Where an unrestricted risk was 'low', 'moderate', 'high' or 'extreme' however, risk management measures needed to be identified and applied and, for each of these, the 'restricted' risk was calculated. This process is termed 'option evaluation'.

In the case where the option involved processing of the product such as by cooking, curing or freezing an additional step was included in the release pathway (R7). The likelihood assigned to this step represents the probability that the pathogenic agent would not be destroyed by the specified processing.

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Hazard identification was carried out in two discrete stages:

- identification of a preliminary index of agents/diseases relevant to the importation of pigs or pig-derived products;
- refinement of the preliminary index in accordance with specified hazard identification criteria (hazard refinement).

PRELIMINARY INDEX OF DISEASES/AGENTS

A preliminary index of diseases/agents was derived by combining the relevant OIE Lists, and other pathogens/diseases of swine that are potentially of quarantine concern. Sixty-one diseases/disease agents of pigs were categorised according to their presence or absence in Australia, and their association with pig meat (Table 11). Where there was any doubt or contention about the occurrence of a disease agent, or its association with pig meat, that disease agent was retained on the list of potential quarantine hazards.

From this process 27 disease agents were identified and are listed below.

- Foot-and-mouth disease virus
- Vesicular stomatitis virus
- African swine fever virus
- Classical swine fever virus
- Rinderpest virus
- Swine vesicular disease virus
- Aujeszky's disease virus
- Porcine reproductive and respiratory syndrome virus
- Transmissible gastroenteritis virus
- Trichinellosis (*Trichinella spiralis*)
- Cysticercosis (*Cysticercus cellulosae*)
- Nipah virus
- Post-weaning multisystemic wasting syndrome
- Salmonellosis (*Salmonella typhimurium* DT104)
- Swine influenza virus
- Porcine brucellosis (*Brucella suis*)
- Porcine epidemic diarrhoea virus
- Porcine respiratory coronavirus
- Rubulavirus (Mexican blue eye disease)
- Teschen disease (Enterovirus encephalomyelitis virus)
- Rabies virus
- Bovine tuberculosis (*Mycobacterium bovis*)
- Haemorrhagic septicaemia (*Pasteurella multocida*)
- Japanese encephalitis virus

- Surra (*Trypanosoma evansi*)
- Venezuelan, Eastern and Western equine encephalomyelitis
- Vesicular exanthema virus

As vesicular exanthema virus is not present in any country a risk assessment was not carried out for this disease agent. The *Final IRA Report* recommends that exporting countries certify country freedom for this disease. Thus, 26 disease agents were identified as disease agents of quarantine concern and were the focus of individual risk assessments.

Table 11 Preliminary index - diseases/agents of possible concern

Disease/disease agent	Occurrence		
	Occurrence in Australia	Control measures in Australia	Include as an identified hazard?*
OIE List A Diseases/Agents			
Foot-and-mouth disease virus	Not present		Yes
Vesicular stomatitis virus	Not present		Yes
African swine fever virus	Not present		Yes
Classical swine fever virus	Not present		Yes
Rinderpest virus	Not present		Yes
Swine vesicular disease virus	Not present		Yes
OIE List B Diseases/Agents			
Anthrax (*Bacillus anthracis*)	Present	Movement controls during outbreaks	No
Aujeszky's disease virus	Not present		Yes
Leptospirosis (*Leptospira* spp.)	Present	No control measures	No
Rabies virus	Not present		Yes
Bovine tuberculosis (*Mycobacterium bovis*)	Declared free 31/12/97		Yes
Haemorrhagic septicaemia (*Pasteurella multocida*)	Not present		Yes
Japanese encephalitis virus	Serological evidence on Cape York (1998) but sentinel pigs negative to date		Yes
Surra (*Trypanosoma evansi*)	Not present. Diagnosed in imported camels in 1907 in north-west Australia - camels destroyed		Yes
Venezuelan, Eastern and Western equine encephalomyelitis	Not present		Yes

	Occurrence		
Disease/disease agent	**Occurrence in Australia**	**Control measures in Australia**	**Include as an identified hazard?***
Atrophic rhinitis of swine (*Pasteurella multocida* and *Bordatella bronchiseptica*)	Present	No control measures	No
Enterovirus encephalomyelitis / Teschen disease	Not present		Yes
Porcine brucellosis (*Brucella suis*)	Present in Qld	Notifiable. Movement restrictions for infected properties and States	Yes
Porcine reproductive and respiratory syndrome virus	Not present		Yes
Transmissible gastroenteritis virus	Not present		Yes
Trichinellosis (*Trichinella spiralis*)	Not present		Yes
Other diseases/agents			
Actinobacillus suis, *Actinomyces suis* and *A. equuli*	Present	No control measures	No
Bovine spongiform encephalopathy	Not present		No**
Bovine viral diarrhoea (Pestivirus)	Present	No control measures	No
Congenital tremors (unknown aetiology)	Present	No control measures	No
Cysticercosis (*Cysticercus cellulosae*)	Not present		Yes
Encephalo-myocarditis virus	Present	No control measures	No
Eperythrozoonosis (*Eperythrozoon suis*)	Present		No
Escherichia coli	Present	No control measures	No
Haemagglutinating encephalomyelitis virus	Present	No control measures	No

	Occurrence		
Disease/disease agent	**Occurrence in Australia**	**Control measures in Australia**	**Include as an identified hazard?***
Haemophilus parasuis	Present	No control measures	No
Inclusion body rhinitis (Porcine cytomegalovirus)	Present	No control measures	No
Intestinal adenomatosis complex, porcine proliferative enteropathies (*Lawsonia intracellulare*)	Present	No control measures	No
Listeriosis (*Listeria monocytogenes*)	Present	No control measures	No
Melioidosis (*Burkholderia pseudomallei*)	Present	No control measures	No
Mycoplasma hyopneumoniae	Present	No control measures	No
Mycoplasma hyorhinis	Present	No control measures	No
Mycoplasma hyosynoviae	Present	No control measures	No
Nipah virus	Not present		Yes
Porcine adenovirus	Present	No control measures	No
Porcine epidemic diarrhoea virus	Not present		Yes
Porcine paramyxovirus (Australian)	Not present in domestic pigs. Eradicated from infected pig herd		No
Porcine parvovirus	Present	No control measures	No
Porcine pleuropneumonia (*Actinobacillus pleuropneumoniae*)	Present	No control measures	No
Porcine respiratory coronavirus	Not present		Yes
Post-weaning multisystemic wasting syndrome (porcine circovirus type 2)	Unknown. A limited serological survey has demonstrated a PCV2 strain		Yes

	Occurrence		
Disease/disease agent	**Occurrence in Australia**	**Control measures in Australia**	**Include as an identified hazard?***
Reovirus infection	Probably present	No control measures	No
Rotavirus infection	Present	No control measures	No
Rubula virus	Not present		Yes
Salmonellosis (*Salmonella typhimurium* DT 104)	Not present		Yes
Sarcocystis spp.	Present	No control measures	No
Brachyspira pilosicoli	Present	No control measures	No
Streptococcus suis	Present	No control measures	No
Swine dysentery (*Serpulina hyodysenteriae*)	Present	No control measures	No
Swine erysipelas (*Erysipelothrix rhusiopathiae*)	Present	No control measures	No
Swine hepatitis E virus	Present	No control measures	No
Swine influenza virus	Not present		Yes
Swine pox virus	Present	No control measures	No
Toxoplasma gondii	Present	No control measures	No
Vesicular exanthema virus	Not present		Yes
Yersinia enterocolitica	Present	No control Measures	No

*Include as an identified hazard?

Yes: indicates that the characteristics of the disease and, specifically the role of pig meat in its transmission, will be examined more closely in the IRA

No: This indicates that at least one of the necessary criteria are void, and that there is no cause to further examine the disease

** Although BSE has been transmitted experimentally to pigs via intra-cranial, intravenous and intraperitoneal inoculation, it was not transmitted orally with high doses (Wells, et al., 2003) nor is there epidemiological evidence of transmission to pigs.

Wells, G.A.H., Hawkins, S.A.C. Austin, A.R., Ryder, S.J., Done, S.H., Green, R.B., Dexter, I., Dawson, M, & Kimberlin, R.H. (2003). Studies on the transmissibility of the agent bovine spongiform encephalopathy to pigs. *Journal of General Virology*, **84,** 1021-1031.

The detailed risk assessments conducted for the identified diseases and disease agents are presented in the following chapters.

Foot-and-mouth disease virus

Technical information

Background

Foot-and-mouth disease (FMD) is a highly contagious viral disease that primarily affects cloven-footed animals. The disease is characterised by the formation of vesicles (blisters) on the skin. The nostrils, lips, oral mucosa, coronary bands and interdigital space of the feet typically are affected. Affected animals often drool and may be lame. Significant mortality may occur in young animals and, in older animals, production losses may be severe. The most significant effect of an outbreak of foot-and-mouth disease in developed countries is the widespread restriction on trade in susceptible animals and animal products imposed by FMD-free trading partners. For this reason, FMD is regarded as the most important non-zoonotic animal disease.

Agent taxonomy

Foot-and-mouth disease virus belongs to the aphthovirus genus of the Picornaviridae family (Pensaert, 1989).

Agent properties

The virus is a single-stranded, positive-sense, nonenveloped RNA virus and is variable, both genetically and antigenically. Seven serotypes (A, O, C, Asia1, SAT 1, SAT 2 and SAT 3) have been identified (Saiz, et al., 2002).

The survival of viruses outside a living host is affected by factors that include the substrate, pH, temperature, relative humidity, and exposure to ultraviolet light. Foot-and-mouth disease virus is pH-labile and is rapidly inactivated below pH 6.0 and above pH 9.0 (Bachrach, et al., 1957). The virus is susceptible to increasing temperatures, with 90% viral inactivation reported at 30 seconds when held at pH 7.5 and heated to 61°C (Bachrach, et al., 1957). However, FMD virus persists for extended periods when chilled or frozen. Very little viral titre was lost after storage of bovine tongue epithelium for 11 years at -50°C (Cottral, 1969). Samples held at 4°C and pH 7.5 required 18 weeks for 95% viral inactivation to occur (Bachrach, et al., 1957). The virus is not particularly sensitive to the effects of ultraviolet light but is susceptible to desiccation, with poor survival reported below relative humidity levels of 55 to 60% (Donaldson & Ferris, 1975). The survival time of FMD virus in pig slurry has been reported to range from greater than 14 weeks at 5°C to 2 weeks at 20°C and 24 hours at 35°C (Haas, et al., 1995).

Host range

Foot-and-mouth disease occurs naturally in cloven-footed animals such as cattle, domestic buffaloes, yaks, sheep, goats, pigs, all wild ruminants and pigs. Camelids are less susceptible to infection with FMD virus[30]. A list of other species in which infection (although not clinical disease) with FMD virus has been reported to occur naturally or experimentally (including Australian native fauna (Snowdon, 1968)) has been compiled (USDA:APHIS:VS, 1994) and include such animals as kangaroos, wombats, hedgehogs, capybaras, rats, cats and dogs. None

[30] Foot and Mouth Disease Disease Card. Office International des Epizooties. www.oie.int/eng/maladies/fiches/a_A010.htm.

of these additional animals appears to be significant in the epidemiology of FMD and thus, although some are carnivorous and may potentially consume meat scraps, none will be considered as an additional direct exposure group. Very occasionally, infection of humans with FMD virus has been documented (Prempeh, et al., 2001). Disease signs in humans are mild and may include tingling blisters on the hands, feet and mouth, fever, and sore throat (Prempeh, et al., 2001). There has never been a case of a human transmitting FMD virus to an animal, although mechanical transmission can easily occur[31].

Epidemiology

Foot-and-mouth disease is endemic throughout much of the world, affecting countries in Africa, the Middle East, South and Centra America, Asia, and Eastern Europe. The Office International des Epizooties (OIE) maintains a list of the FMD status of member countries and this should be consulted for the most up-to-date information on world distribution of the disease[32].

Strain differences in the epidemiological behaviour of FMD virus exist; nevertheless, some generalizations can be made. Infection usually occurs by inhalation or ingestion. The incubation period varies with the strain of the virus, the number of viral particles ingested or inhaled, the species infected, and the age and health of the animal. Typically the incubation period ranges from 2 to 11 days, although it may be as short as 18 hours (Kitching & Alexandersen, 2002) or as long as 14 days[33]. Incubating and clinically affected animals excrete virus in breath, saliva, faeces, urine, milk and semen. Pigs excrete the greatest quantity of virus on their breath whilst cattle are the most susceptible to airborne infection as they have the largest tidal volume and thus take in more viral particles per breath than smaller animals. In addition, much less virus is required to infect cattle than pigs by the respiratory route (Donaldson & Alexandersen, 2001; Donaldson & Alexandersen, 2002). A carrier state is reported (Salt, 1993) to be a common sequel to clinical or subclinical infection with FMD virus in cattle (2.5 years) and buffalo (at least 5 years). Virus can be recovered from oropharyngeal secretions of carrier animals despite presence of circulating antibody. Vaccination does not prevent the development of a carrier state. The role that carrier animals play in the epidemiology of FMD is uncertain, although it has been shown that carrier buffalo may, on occasion, infect susceptible cattle (Thomson, 1995). The carrier state also occurs, albeit to a lesser extent, in sheep and goats (Salt, 1993), but is not described in pigs.

Outbreaks of FMD in immunologically-naïve populations are characterised by rapid spread of the virus within and between herds, extremely high morbidity amongst susceptible animals, and variable mortality (Gibbens, et al., 2001). Virus-related factors contributing to this include the wide and diverse host range, the low infectious dose, the large amounts of virus excreted by incubating and clinically affected animals, and the ability of the virus to persist in cold and temperate environments in fomites and on the wind (Salt, et al., 1998). A typical scenario for an outbreak in a previously free country is for pigs to become infected through consumption of inadequately cooked swill, followed by infection of ruminants via aerogenous spread (Blood, et al. 1989). However, in a country or region in which FMD is endemic, the epidemiological picture is different. In general, one or more strains of the virus are present within the region or country, and other strains of FMD are considered exotic (Arshadi & Maldjaii, 1976). Clinical

[31] www.daff.gov.au
[32] http://www.oie.int/eng/info/en_fmd.htm; http://www.oie.int/Cartes/world/a_Monde.htm
[33] Foot and Mouth Disease Disease Card. Office International des Epizooties. www.oie.int/eng/maladies/fiches/a_A010.htm

disease due to the enzootic strains is observed sporadically, when the population immunity has waned or the viral challenge is excessive.

Most reports of between and within herd prevalence of FMD, in particular seroprevalence, are confounded by control programs such as vaccination. Countries where the disease is enzootic but where there are no control programs (for instance, many countries in Africa) tend to either lack funds for the control of FMD, or have no inducement to do so (Vosloo, et al., 2002). In either case, there is little incentive to report the incidence or prevalence of infected herds. Nonetheless there are several reports on the incidence of disease in FMD enzootic countries. A review of the FMD situation in Nepal in the early 1990s (Ferris, et al., 1992) estimated that approximately 30% of the large ruminant population was affected annually and mentioned that FMD is widespread throughout Nepal at all times of the year. A survey undertaken in India in 1991 estimated the annual incidence rate of FMD throughout the country to be 23% (Saxena, 1995). The incidence was higher in local cattle (29%) than in cross-bred cattle (17%) or pigs (16%). In a region in Bolivia, where cattle but not sheep are vaccinated, antigen was detected in sheep on 56% of 81 farms (Fernandez, et al., 1976). This is similar to the results of a survey undertaken in 1978 in Brazil where 41% of 150 cattle properties had seropositive animals (Pavez, et al., 1981). There are few reports of within herd prevalence of FMD infection. In India, in 1994, the prevalence of FMD affected animals in five outbreaks varied from 45 to 100% (Sarma & Hazarika, 1996).

Clinical signs

Manifestations of clinical signs of disease vary according to strain of virus and species of host. In general terms, the disease is least apparent in sheep (Kitching & Hughes, 2002). However some strains exhibit species adaptation. For instance, the FMD virus type O isolated from an outbreak in Taiwan in 1997 (Taiwanese isolate TAW 9/97) caused typical lesions in pigs and spread readily but did not cause clinical or serological evidence of disease in cattle during the epidemic, despite their apparent exposure to the virus. The lack of transmission of this strain to cattle by natural routes was later confirmed experimentally (Dunn & Donaldson, 1997). Apparent species restriction of FMD infection may stem from managerial factors rather than reflecting a true species adaptation, as is illustrated in Thailand, where pigs, although susceptible to infection by the circulating strains, played a minor role in the epidemiology of the disease in the 1990s. This was thought to be due to the pig feeding and housing practices used that protected pigs from exposure to virus from infected cattle, buffalo, or their products (Chamnanpood, et al., 1995).

The typical clinical signs of FMD in pigs have been reviewed (Kitching & Alexandersen, 2002). Pigs develop vesicular lesions on the snout, in the mouth and on the tongue, and around the coronary bands of the feet and between the toes. Lesions may develop on the teats of lactating sows. An increase in body temperature may occur, but may not be remarkable. Affected pigs may be inappetant and lame. Young piglets may die acutely of myocarditis prior to development of vesicular lesions (Donaldson, et al., 1984). Abortions may be a feature of FMD infection in the farrowing herd (Mann & Sellers, 1989).

Pathogenesis

The initial site of viral infection and replication in pigs naturally infected by either the oral or respiratory route appears to be the pharynx, particularly the soft palate and tonsil (Alexandersen, et al., 2001). Virus is collected in the local lymph nodes and then enters the blood stream. This results in infection of stratified squamous cells and subsequent amplification

of the virus with increased viraemia and infection of epithelial cells on a wider scale. This process continues until typical vesicular lesions are apparent and/or the virus is controlled by the host's immune response. It is hypothesized that the airborne virus carried on the breath of pigs is derived mainly from stratified squamous epithelial cells of the skin, oral mucosa and pharynx (Alexandersen, et al., 2001). In this study, pigs were followed for 4 days post-exposure and the authors noted that peak viraemia coincided with onset of clinical signs (day three post-exposure in this study). Other workers monitored the presence of FMD virus in blood and muscle of a pig infected via direct contact with an experimentally-infected pig (Dhennin, 1979). They noted the appearance of virus in the blood 32 hours and the muscle 20 hours prior to the appearance of vesicles or the beginning of a rise in temperature.

Pathology

Most grossly visible lesions are confined to the oral mucosa, the coronary bands, interdigital skin and the skin of the snout. Lesions may also be present on the teats of nursing sows, and signs of mastitis may be observed. Lesions range from intact vesicles filled with straw-coloured fluid, to ruptured vesicles at various stages of healing. After rupturing, the epithelium of the vesicle detaches, disclosing a red, ulcerated surface that heals by granulation (Mann & Sellers, 1989).

Piglets dying from acute myocarditis may have small, greyish foci of irregular size in the wall and septum of the left ventricle. These may give the myocardium a striped appearance known as 'tiger heart' (Jones, et al. 1983).

Immunology

Immunity to one strain of FMD resulting from either vaccination or natural infection is not protective against other strains. Depending on the extent of viral challenge, the degree of homology between vaccinial and challenge strain, the formulation of the vaccine, and the time between vaccination and challenge, vaccination may not completely prevent infection with FMD virus in pigs (Salt, et al., 1998). However, vaccination is said to greatly reduce the amount of virus excreted by subsequently-infected pigs.

Transmission via meat

The transmission of FMD virus via meat or meat products is well documented. A review (USDA:APHIS:VS, 1994) of 627 known sources of FMD outbreaks throughout the world from 1870 to 1993 reported that 411 of the outbreaks (66%) were attributable to infected meat, meat products or garbage. Of the 411 outbreaks, all but 16 occurred more than 25 years ago.

The titres of FMD virus in muscle and associated tissues have been reported in several studies. The amount of virus present in tissues derived from an infected pig varies depending on several factors including the strain of virus; amount of virus initiating infection; stage of infection; presence and stage of host's immune response; conditions of processing and storage of the tissues after slaughter; and length of time since slaughter. For example, 62 pigs were each inoculated intravenously with 1 ml of a 1:10 dilution of stock FMD C serotype virus, titre $10^{8.9}$ $TCID_{50}$/ml (Mebus, et al., 1993). The pigs were slaughtered at 2 days post-inoculation, and the mean viral titres of blood, lymph node, bone marrow, fat and muscle were determined to be 3.6, 3.4, 1.9, 0.5 and 0.03, respectively, expressed in inverse $\log_{10}$ plaque forming units (PFU) per ml or per gram. Whereas another study reported viral titres in fat and muscle tissues of greater than 10^5 PFU/gram. In this study 10 pigs were inoculated in the coronary band with 1 ml of 1:5

dilution of FMD C1 serotype virus, titre $10^{7.5}$ PFU/ml and slaughtered 48 hours later (Panina, et al., 1989).

The stability of FMD virus in muscle and associated tissues in relation to changes in pH and temperature has been studied. Foot-and-mouth disease virus is pH labile, and is rapidly inactivated by pH levels below 6.0. Pig meat does not consistently reach as low an ultimate pH as does beef, thus the inactivation of FMD virus in pig meat may not be as complete as that occurring in beef (Farez & Morley, 1997). Importantly, fat, bone marrow, lymph nodes and blood clots are protected from the pH changes that occur in muscle tissue post slaughter.

Information concerning the survival of FMD virus in porcine tissues has been collated (Cottral, et al., 1960). Foot-and-mouth disease virus survived in the bone marrow of chilled pork for 42 days, and in frozen pork for 76 days. The virus was found in blood clots from pork that had been stored at 4°C for 70 days, and in fresh and frozen lymph nodes. Foot-and-mouth disease virus has been reported to survive for up to 190 days in salted bacon, up to 89 days in ham bone marrow, and up to 183 days in ham fat (McKercher, et al., 1987). Heating of samples of infected porcine lymph node, bone marrow and blood clot samples to 69°C inactivated the virus (McKercher, et al., 1980).

The survival of FMD virus in "Parma Hams" has also been examined (McKercher, et al., 1987). Virus was recovered from the bone marrow of hams at 30 days but not 108 days of ageing in one trial, and from the fat at 96 but not 170 days of ageing in another trial. The survival of FMD virus in dry cured pig meat products has also been reported (Mebus, et al., 1993). In this study, FMD virus was not detected in muscle after 14 days of processing, but was isolated from bone marrow of Iberian shoulder and Serrano ham for up to 84 days, and in the fat and lymph node of Serrano ham for up to 140 and 168 days, respectively[34]. In contrast, FMD virus was not isolated from muscle and fat of seven pigs infected 48 hours prior to slaughter either at the commencement of processing salami (72 hours after slaughter) or in salami tested 7 days after processing (Panina, et al., 1989).

The oral infectious dose for FMD virus for pigs has not been examined in detail (Farez & Morley, 1997). However, when 30 pigs were fed minced offal consisting of liver, kidney and lymph nodes with a viral titre of $10^{5.0}$ $TCID_{50}$, infection with FMD virus was confirmed in two of the pigs (Henderson & Brooksby, 1948). An estimate of $10^{6.0}$ $TCID_{50}$ (equivalent to $10^{5.0}$ PFU) for the pig oral ID_{50} has been extrapolated from this information (Gale, 2002).

Release assessment

R1 — the likelihood that a source herd is infected

The between herd prevalence of FMD infection in a country where the disease is endemic in the absence of a control program is difficult to estimate. There are little data on FMD prevalence in pig herds. Nonetheless the prevalence of FMD infection has been reported as ranging from 41 to 56% of sheep or cattle farms. Based on this information it was considered that there was a 'moderate' likelihood that the herd from which slaughter-age pigs were selected would be infected.

[34] To place this information in perspective, it should be noted that the normal curing time for these products in each case greatly exceeded the maximum number of days at which virus could be isolated from the product.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

A characteristic of FMD is its extremely rapid spread between animals. Pigs are less susceptible than cattle to infection by aerosol (Donaldson & Alexandersen, 2001) but infected pigs excrete vast quantities of virus. Transmission via aerosol, direct contact, and fomites would result in exposure of most pigs in a herd within days of entry of the virus into the herd. Persistent infections are not a feature of the disease in pigs. In a country where the disease was endemic, pigs are likely to be exposed to the virus after maternal antibody has waned. Considering this information, the likelihood that a slaughter-age pig is infected was considered to be 'moderate'.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

The clinical signs of FMD in pigs are characteristic, and clinically affected pigs are unlikely to pass ante-mortem inspection. However, pigs in the incubation stage of the disease would pass. The length of the incubation period varies, but is generally from 2 to 11days. The duration of clinical disease is also variable, but healing epithelial lesions are visible for more than a week (Geering, et al. 1995). The feet will show the after-effects of FMD infection for longer (even if the claws are not shed, affected horn must grow out) but this may not be detected at ante-mortem inspection. Subclinical or persistent infections are not a feature of FMD in pigs.

The clinical signs of FMD lend themselves to detection at ante-mortem inspection. Post-mortem examination of the carcass is more likely to confirm suspicions rather than reveal unsuspected infection.

In light of this information, the sensitivity of ante-mortem, slaughter and processing requirements was considered to be 'moderate'.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with FMD virus and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a 'one minus the background rejection rate'. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered 'extremely low'. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the FMD virus will be present in the meat harvested for export

Infection with FMD virus is characterised by replication in stratified squamous cells and subsequent amplification of the virus, with viraemia resulting in a more widespread infection of epithelial cells. The virus does not have a predilection for muscle tissue and its presence in muscle, lymph nodes and fat is due to the vascular perfusion of these areas. Foot-and-mouth

disease virus is easily isolated from muscle tissue from infected animals immediately after slaughter and bleeding out (that is, prior to the pH changes in the muscle tissue that occur post-mortem) (McKercher, et al., 1987; Panina, et al., 1989; Mebus, et al., 1993).

The likelihood that FMD virus would be present in the meat harvested for export from an infected pig was considered to be 'high'.

R5 — the likelihood that the FMD virus will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

Foot-and-mouth disease virus is susceptible to inactivation at pH levels below 6.0. However, the post-mortem decrease in pH in pig meat is not as pronounced as that occurring in beef. In addition, the microenvironments of lymph nodes, bone marrow, fat and blood clots are not subject to the same pH changes as those that take place in muscle tissue. In this IRA it has been assumed that pig meat does not obtain a pH below 6.2.

On the basis of this information, the likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation was considered to be 'high'.

R6 — the likelihood that the FMD virus will not be destroyed during cold storage and transport

Foot-and-mouth disease virus is very stable under cold conditions. For example, samples held at 4°C and pH 7.5 required 18 weeks for 95% viral inactivation to occur (Bachrach, et al., 1957), and very little viral titre was lost after storage of bovine tongue epithelium for 11 years at -50°C (Cottral, 1969).

Thus, the likelihood that meat infected at the completion of carcass maturation would remain infected during transport and storage was considered to be 'high'.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a 'low' likelihood that imported pig meat derived from an individual carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the FMD virus to initiate infection

Pigs are easily infected by the oral route. High concentrations of virus are present in the tissues of incubating and clinically affected pigs. Historically, outbreaks of FMD are often associated with infection of pigs following ingestion of contaminated meat or meat products in swill.

Clearly, sufficient virus can persist in carcass tissues to result in infection. This is supported by experimental evidence of high quantities of virus being detected in small amounts of infected porcine tissue (>10^5 PFU g^{-1}), and a relatively low estimate for the oral ID_{50} ($10^{5.0}$ PFU) for pigs.

Given this, it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of FMD virus to initiate infection was 'high'.

L3 — the likelihood that the FMD virus would remain viable during the period prior to scavenging

Foot-and-mouth disease virus is not very sensitive to the effects of sunlight, but is affected by desiccation and heat. The inactivation time for FMD virus in pig slurry ranges from 14 weeks at 5°C to 24 hours at 35°C (Haas, et al., 1995). Survival of FMD virus in the environment was reviewed in Australia's exotic disease contingency plan, AUSVETPLAN (Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 2002). The virus reportedly can survive 50 days in water, 74 days on pasture at 8 to 18°C and high relative humidity, 26 to 200 days in soil or hay depending on storage or climatic conditions and 35 days on cardboard or wood contaminated with blood or tissue.

Considering this, the likelihood that FMD virus would remain viable in meat scraps discarded in refuse for the period of time required for pigs to locate and subsequently scavenge the material was estimated to be 'high'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = High
- Rural regions = High
- Large towns = Very low

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be 'high'.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs, it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall the Panel considered that there was a 'high' likelihood that the FMD virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'high'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall the Panel considered that there was a 'high' likelihood that the FMD virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'high'.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the three exposure groups - that is, feral pigs, pigs in backyard enterprises and pigs in small commercial piggeries. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Foot-and-mouth disease is an extremely contagious disease. It is characterised by rapid spread in temperate and cold climates. Incubating and clinically affected pigs produce vast quantities of virus that is excreted in their bodily fluids and carried on their breath. Transmission from pigs to pigs or to other susceptible species may occur via direct contact, via aerosol spread over distances that can be large (depending on conditions), via fomites, or via consumption of infected meat or meat products. The environmental conditions throughout much of Australia may not be conducive for prolonged survival of the virus outside of a host.

A recent review of the risks posed to Australia with respect to FMD by feral pigs and other feral animals noted that, as pigs are relatively difficult to infect via the aerosol route, the contact rate between groups of feral pigs is important in determining the likelihood of spread within a feral pig population (Black, 2002). Feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous.

An outbreak of FMD in a local population of feral pigs may not, initially, be suspected. However, it is likely that a more widespread outbreak of FMD in feral pigs would be identified, as inspection of feral pigs harvested for export purposes should result in detection of vesicular lesions. For example, an exotic disease investigation was instigated after a hunter reported

seeing sores on the mouth and feet of two boars in the Northern Territory in 2002 (Small, 2002).

Spread of FMD from feral pigs to backyard pigs is feasible. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs, however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves. However, the authors of the review mentioned above noted that, although caution is required when extrapolating from the situation in other countries, experience overseas demonstrates that wild pigs are rarely involved as sources of infection for domestic livestock (Black, 2002).

Transmission of FMD virus from pigs to other susceptible species could also occur by close contact, or under suitable environmental circumstances, by wind-borne spread. Pigs are 'amplifier' hosts, in that they generate vast quantities of virus in aerosol, and cattle are very susceptible to infection via aerosol. The risk of spread by this means is proportional to the density of livestock downwind from the excreting pigs (Cannon & Garner, 1999). Nonetheless the stocking density of susceptible species in many areas is quite low, which may slow the spread of the virus. Should FMD virus spread from feral pigs to backyard pigs, then infection of associated livestock is quite likely to occur. However, it is very likely that the disease would be noted at this point and that strict measures would be put in place to contain and eradicate the disease, thus minimising the spread to a more general population of domestic pigs and other susceptible species.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: low

Scenario 3: low

Scenario 4: low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The classical signs of FMD in pigs are such that it is possible that they will be recognised in the directly exposed backyard herd, and reported. Nonetheless it is feasible that signs such as lameness may not be fully investigated. In addition, the signs of disease apparent in the directly exposed herd may not be those of 'classical' FMD. Other signs, such as sudden death in piglets, or abortions in sows, may predominate (Donaldson, et al., 1984). Further, it is possible that the owner of the backyard herd may elect not to report the disease, again increasing the likelihood of spread of the disease beyond the index herd.

Pigs excrete large quantities of virus in the incubation period. This, in combination with the minimal levels of biosecurity in most backyard herds, would reduce the likelihood of the outbreak being restricted in the directly exposed backyard herd. Spread via fomites to other pig herd or susceptible species may also occur, with this method of transmission frequently implicated.

The spread of FMD from backyard pigs to feral pigs is possible, given that contact between the two populations does occur, as noted above. However, it is the experience of other countries that wild pigs, in general, do not play an important role in the epidemiology of FMD (Black, 2002).

It was considered that the presence of FMD in a wider population of backyard pigs and other susceptible species would be noted and eradication measures implemented, thus reducing the likelihood that the disease would spread to a more general population of domestic pig operations and livestock.

On balance, the following liklihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: moderate

Scenario 4: low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a

pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

It was considered likely that detection of FMD would occur in a small commercial piggery. Managers, owners and veterinarians are well aware of the clinical signs of FMD with active awareness campaigns for exotic diseases occurring in Australia. Nonetheless as pigs excrete large quantities of virus, and with up to a 100 sows in a small commercial piggery, infection would be amplified with possible spread to other piggeries or to other susceptible species. Other important considerations include the larger number of live pigs transported from small commercial piggeries and the potential spread via fomites. In FMD outbreaks involving pigs the disease can spread quickly. For example during the initial outbreak of FMD in Taiwan 60% of pigs were infected on 20% of farms despite implementation of control measures such as slaughter and movement restrictions (Yamane, et al., 1997).

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: moderate

Scenario 4: moderate

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus, while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this scenario, FMD would have established in the directly exposed animal, or group of animals, but would not have spread to other pig herds or other animals. In the case of a feral pig herd or backyard pig enterprise being infected, this 'no outbreak' scenario would have resulted from a low probability of contact between infected and susceptible animals, rather than from human intervention. Indeed, it was assumed that it would not have been identified in these

exposure groups. In the case of a small commercial piggery, the disease would have been identified and contained due to implementation of a control and eradication program.

The direct impact of foot-and-mouth disease

Animal life or health

Foot-and-mouth disease infection may result in high mortalities (especially in piglets) and severe lameness. In addition, abortion may occur in sows. The epithelial lesions in the mouth and on the teats may be quite painful, and pyrexia is common. However, most pigs recover from the disease.

On this basis the direct impact of FMD on animal health was considered unlikely to be discernible except at the local level. Thus, this criterion was rated as 'B'.

Environment

Because FMD is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of foot-and-mouth disease

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

This criterion was not rated identically for each exposure group.

It is likely that if the disease was contained within a feral pig herd or a single backyard enterprise, FMD may not be diagnosed within either of these herds. However if the primary outbreak involved a small commercial piggery it was considered that pigs showing clinical signs of FMD would be investigated.

If FMD was identified in Australia in a small commercial piggery the measures to be implemented, as outlined in AUSVETPLAN, are to eradicate FMD in the shortest possible period while limiting economic impact using a combination of strategies including stamping out, pre-emptive depopulation of susceptible animals, quarantine and movement controls, decontamination of facilities, tracing and surveillance, zoning, a public awareness campaign, and (possibly) vaccination (Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 2002). The disease is classed as Category 2 under the Australian Emergency Animal Disease Cost-Sharing Agreement[35], and thus the cost of the response is to be covered by government and relevant industries by contributions of 80% and 20%, respectively. Category 2 diseases have the potential to cause major national socio-economic consequences through very serious international trade losses, national market disruptions and very severe production losses in the livestock industries that are involved.

In this scenario where FMD has not spread it was considered that the disease would be eradicated promptly. Nonetheless there would need to be extensive surveillance of the domestic pig population, feral pig population and the local ruminant populations.

[35] http://www.aahc.com.au/eadp/response.htm

Overall, it was considered that the indirect impact of new eradication and control programs was unlikely to be discernible at any level when the primary exposure group was a feral pig herd or a backyard pig enterprise. This resulted in a rating of 'A' for this criterion. However, when the primary exposure group was a small commercial piggery, it was considered that the indirect impact of new eradication and control programs was of minor importance at the national level. This gave the disease a rating of 'E' for this criterion.

Domestic trade or industry effects

This criterion was not rated identically for each exposure group.

As discussed above it was considered unlikely that the disease would be detected in the initially exposed herd of feral pigs or single backyard enterprise, thus no domestic trade or industry effects would be expected, and the rating assigned to this criterion was 'A'.

In the case of a small commercial piggery it was considered that the index herd might be detected, in which case an eradication program would be implemented. Depopulation of the infected herd and any dangerous contact herds would be carried out. Movement restrictions would be imposed to ensure that any product from infected or in-contact animals was disposed of and suspect product was detained. These controls would affect all susceptible species within the restricted area and other controls likely would be imposed in the control area. Movements of animals to sale and slaughter would also be affected. Initially a standstill order may apply to all susceptible animals in Australia. It is likely that following detection of FMD in one State of Australia, other States and Territories may close their borders to all susceptible animals and products until the extent of the outbreak was ascertained.

Due to the disruption to exports, large quantities of meat would enter the domestic market, with domestic prices likely to fall. As a result revenue for affected and associated industries would fall. With the detection of an exotic disease in Australia it is likely that consumers may initially decrease their consumption of pork. A publicity campaign may need to be undertaken to reassure the public that there were no health concerns.

When these issues were taken into account, the indirect impact of FMD on domestic trade and industry when the primary exposure group was a small commercial piggery was considered to be of minor importance at the national level. This resulted in a rating of 'E' for this criterion.

International trade effects

This criterion was not rated identically for each exposure group.

As the disease is unlikely to be detected in the initially exposed herd of feral pigs or backyard enterprise, the indirect effects of FMD on international trade for these exposure groups was unlikely to be discernible at any level, and thus, the rating assigned to this criterion was 'A'.

The diagnosis of FMD in a single small commercial piggery, would likely result in initial cessation of trade in agricultural products including live animals susceptible to FMD (Productivity Commission, 2002). Although in this scenario the disease could be promptly eradicated, the OIE Code states when an FMD outbreak occurs in an FMD free country or zone where vaccination is not practised, a 3 months waiting period after the last case, where a stamping-out policy and serological surveillance are applied, is required to regain the status of FMD free country or zone. Export of some agriculture commodities may not resume for some time.

In light of this information, it was considered that the indirect effects on international trade when the primary exposure group was a small commercial piggery would be significant at the national level, and thus this criterion was rated as 'F'.

Indirect impact on the environment

In this scenario, FMD is unlikely to lead to any discernible indirect impacts on the environment such as affecting biodiversity, or from the disposal of carcasses from a single premises and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

This criterion was not rated identically for each exposure group.

As FMD is unlikely to be diagnosed in a feral pig herd or single backyard enterprise it was considered that for these exposure groups there was unlikely to be any discernible indirect impact on communities, and a rating of 'A' was thus assigned to this criterion.

The diagnosis of FMD in a single premises (small commercial piggery) would cause disruption to domestic and international trade. In turn this would affect rural communities reliant on livestock industry revenue whilst bans on exports were in place. Employment could be affected over a range of farming and associated industries and businesses in the local area. Given this, the indirect impact on rural communities was considered unlikely to be discernible at the national level, but of minor importance at the State level. This gave the disease a rating of 'D' for this criterion.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, FMD would have established in a broader population of feral pigs. The disease would be contained through the diagnosis of disease in feral pigs and the mounting of an eradication program.

The direct impact of foot-and-mouth disease

Animal life or health

With this scenario the disease spreads to a general population of feral pigs but not to domestic pigs. Foot-and-mouth disease may result in high mortalities (especially in piglets) and severe lameness. In addition, abortion may occur in sows. The epithelial lesions in the mouth and on the teats may be quite painful, and pyrexia is common. However, most pigs recover from the disease. Overall, the direct impact on animal health is unlikely to differ from that of the direct primary exposure group and thus, this criterion was rated as 'B'.

Environment

Because FMD is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of foot-and-mouth disease

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this scenario, where the disease has spread to a more general population of feral pigs it was considered that the outbreak would be detected regardless of the exposure group from which the disease originated.

Following detection of FMD AUSVETPLAN recommendations would be implemented. These include eradication, control, surveillance/monitoring and compensation programs, directed at both feral and domestic pig populations, and including other susceptible species. Animals destroyed would need to be disposed of in a way that prevents scavenging by feral pigs. Eradication of the disease in feral pigs could be difficult due to inaccessibility of some areas and ensuring safe disposal of carcases. AUSVETPLAN recommends that if FMD has spread into the feral pig population, the eradication program could involve establishing the limits of the identified zone, creating an infected depopulation zone and reducing the population density within the infected zone. Modelling suggests that total elimination of a feral population may not be necessary to achieve FMD eradication. Biosecurity of farms would need to be increased to ensure feral pigs could not gain access to livestock.

After consideration of these issues, the indirect impact of control and eradication programs was deemed to be of minor importance at the national level. This gave the disease a rating of ‘E’ for this criterion.

Domestic trade or industry effects

In the case of spread to a more general population of feral pigs there is likely, at least initially, to be an order preventing movement of all susceptible species. Once the standstill is lifted, movement controls will remain in place in affected areas, which will prevent all susceptible animals moving to slaughter or sale. States that are not affected with FMD will likely close their borders to susceptible animals and products. The harvesting of feral pigs by hunters would also cease.

As export markets for meat will close, the extra volume of meat will be redirected onto the local market. This could result in a reduction in domestic red meat and pork prices. In addition consumers may initially decrease consumption of these meats following a disease outbreak. Publicity campaigns may need to be undertaken to reassure the public that there was no risk from meat.

Given this, the impact on domestic trade or industry was considered to be of minor importance at the national level, thus resulting in a rating of ‘E’ for this criterion.

International trade effects

An outbreak of FMD in Australia, even in this scenario where there is no secondary spread to domestic livestock, would have an immediate impact on international trade with the cessation of export of live susceptible animals and their products. It was considered that the indirect effects on international trade would be similar to those described for scenario 1, in the case of FMD in a small commercial piggery. As such it was considered to be significant at the national level, and thus this criterion was rated as ‘F’.

Indirect impact on the environment

In this scenario, FMD is unlikely to lead to any discernible indirect impacts on the environment except at the local level, such as may result from the disposal of carcasses, and a rating of 'B' was thus assigned to this criterion.

Indirect impact on communities

It was considered that the economic effects on rural and regional viability of an outbreak of FMD, even on a small scale, would be similar to that discussed above for scenario 1 for a small commercial piggery. Given this, the indirect impact on rural communities was considered unlikely to be discernible at the national level, but of minor importance at the State level. This gave the disease a rating of 'D' for this criterion.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, FMD would have established in a local population of backyard piggeries or small commercial piggeries and to other susceptible species such as cattle or sheep. The disease would be contained through the diagnosis of disease in pigs and/or other susceptible species, and the mounting of an eradication program.

The direct impact of foot-and-mouth disease

Animal life or health

This scenario is characterised by the spread of FMD to a local population of domestic pigs and other susceptible species. In pigs the predominant sign is lameness. Epithelial lesions in the mouth and on the teats may be quite painful, and pyrexia is common. However, most pigs recover from the disease. In the case of other susceptible species such as cattle the signs include such things as fever, poor appetite, salivation, lameness, reduced lactation, mastitis and abortion. Mortality in adults is rare but there may be high mortality in calves. In sheep and goats the clinical signs may be mild but include lameness, reluctance to stand and significant mortality in lambs can occur.

Taking these factors into consideration, the direct effect on animal health was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. This resulted in a rating of 'C' for this criterion.

Environment

Because FMD is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of foot-and-mouth disease

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

It was considered that the consequences on this criterion would be similar to that described for scenario 2 although the major focus of the eradication, control, surveillance/monitoring

programs and compensation programs would be directed at domestic pig and ruminant populations. Nonetheless, some surveillance of the feral pig population would also be necessary. Significant numbers of animals may need to be destroyed. One study conducted in the 1990s estimated that with a local outbreak of FMD in Northern Victoria involving 36 infected premises, some 26,000 stock would be slaughtered whereas in Northern New South Wales when 29 premises were infected some 69,000 stock would be slaughtered to achieve eradication (Garner & Lack, 1995). The same study estimated that the cost of compensation and control would be approximately $4.25 million and $1.44 million respectively for an outbreak in Northern New South Wales. A more recent examination of the costs of an FMD outbreak in Australia in a wheat sheep zone of Western Australia, when 15 premises were infected, found that control and compensation costs could be about $30 million (Productivity Commission, 2002).

Considering this, the indirect impact of control and eradication programs was deemed to be of minor importance at the national level. This gave the disease a rating of ‘E’ for this criterion.

Domestic trade or industry effects

Due to secondary spread of FMD to a local population of domestic livestock there would be more movement controls on animals and products over a greater area, but still a local area, and for a greater period of time that that described for scenario 1 or 2. This would not only affect livestock producers but could also affect associated industries such as transport, meat processing, milk manufacturing and wool processing. Movement of livestock and livestock products between States in Australia may be disrupted for a period of time.

Product destined for export will be redirected to the domestic market, likely resulting in a decrease in domestic red meat and pork prices.

Overall it was considered that the indirect effect on domestic trade was of minor importance at the national level, and a rating of ‘E’ was assigned to this criterion.

International trade effects

As described for scenarios 1 and 2 the detection of FMD in Australia would result in other countries placing an immediate ban on Australian livestock and products. It may be possible to renegotiate conditions for some livestock and products to countries where FMD is endemic, although this may depend on the strain of FMD. Eradication and demonstration of freedom from FMD together with acceptance by our trading partners may take longer to achieve with secondary spread of FMD into a local population of domestic pigs and other susceptible species than that described for the scenarios above.

The loss of national income from closure of Australia’s export markets has been estimated at $3333 million for a 3 month outbreak involving one area in Western Australia (Productivity Commission, 2002).

Taking these factors into consideration the likely indirect effect of FMD on international trade was considered highly significant at the national level, and a rating of ‘G’ was assigned to this criterion.

Indirect impact on the environment

An outbreak of FMD as described by this scenario (local spread) is likely to have indirect environmental impacts resulting mainly from the disposal of animal carcasses. Additional

impacts could arise from the disposal of other livestock products such as milk, the widespread use of disinfectants to decontaminate infected properties, and a reduction in on-farm environmental improvement measures (for example, soil conservation, tree planting, salinity reduction) due to decreases in farm cash flow (Productivity Commission, 2002). Given this, the indirect impact on the environment was considered unlikely to be discernible at the national and State levels, and of minor importance at the district or regional level. This resulted in a rating of 'C' for this criterion.

Indirect impact on communities

An outbreak of FMD would also affect the rural and regional economic viability including such things as businesses reliant on livestock revenue, employment, local governments together with social costs to individuals and communities. One study estimated that for an outbreak of FMD in Northern Victoria approximately 954 jobs would be lost, with losses in income of approximately $22 million (Garner & Lack, 1995). The more recent Productivity Commission report stated that for a 3 month outbreak encompassing one area within a State the Australian Gross Domestic Product (GDP) would decline by $900 million in the first year and over 10 years by $2 billion to $3 billion.

When these issues were collated, the indirect impact of FMD on rural communities was considered to be significant at the national level. Overall this resulted in a rating of 'F' for this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, FMD would have established in a broader population of commercial piggeries (including medium-large piggeries) and spread to other susceptible species. An eradication program would have been mounted in response to the diagnosis of the disease in pigs and/or other susceptible species.

The direct impact of foot-and-mouth disease

Animal life or health

In this scenario, FMD has spread to a more general population of domestic pigs, and to other susceptible species (in particular, domestic ruminants) on a wider scale. Foot-and-mouth disease may result in high mortalities (especially in piglets and calves) and severe lameness. The epithelial lesions in the mouth and on the teats may be quite painful, and pyrexia is common. Quite apart from illness and death, the widespread and likely prolonged movement restrictions could cause serious overcrowding and associated animal health problems as pigs, for example, outgrow their accommodation and cannot be moved on. Given this, it was considered that the direct impact on animal health would be of minor importance at the national level, resulting in a ranking of 'E' for this criterion.

Environment

Because FMD is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of foot-and-mouth disease

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

The Productivity Commission recently estimated eradication and control costs for outbreaks of FMD in Australia. These costs for a 12-month outbreak involving three States (Victoria, South Australia, and New South Wales) were estimated at between $360 and 420 million. The compensation costs for this outbreak were estimated to be an additional $41 million without vaccination and $68 million with vaccination (Productivity Commission, 2002).

Australia's policy for FMD is to eradicate by stamping out even if the disease were present in a number of areas. Zoning would be employed in those endemic areas, together with stamping out and associated control measures. Vaccination may be used as a control measure but eradication will be the primary strategy.

In light of this information, the indirect impact of control and eradication programs was considered to be significant at the national level, resulting in a ranking of 'F' for this criterion.

Domestic trade or industry effects

The restrictions imposed on the movement of animals and animal products with a more generalised outbreak are likely to cause disruption to the local marketing of meat. Interstate trading restrictions may apply on meat until tracing and surveillance were completed. Initially, if there was a shortage of product there could be a price increase for meat. However, as export of meat would cease, meat would be redirected to the local market. Overall, this is likely to cause a reduction in meat prices. Consumers may also decrease consumption of meat following an outbreak and a publicity campaign would likely need to be conducted to reassure the public that there were no health concerns. There would loss of genetics if breeding herds were involved in the outbreak.

Associated industries such as abattoirs, processors, transport, and stock feed manufacturers would also be affected if the outbreak were prolonged. Unemployment may result.

There are likely to be increased feed costs and welfare concerns for those producers whose premises are not infected but which are subject to movement restrictions.

The Productivity Commission report on the impact of an outbreak of FMD in a single zone in Western Australia stated that domestic market revenue would be reduced by approximately $2373 million (Productivity Commission, 2002).

In view of these factors, the indirect effect of a more generalised outbreak of FMD on the domestic trade or industry was considered to be significant at the national level, thus resulting in a ranking of 'F' for this criterion.

International trade effects

As described in the 2002 Productivity Commission report, it is likely that many agricultural markets would close after the diagnosis of FMD in Australia. Australia is a large agricultural exporter. Annual livestock exports constitute 6 per cent of total exports by value - almost $10 billion in 2000 - 2001 (Productivity Commission, 2002). After initial closure of export markets, the consequences for international trade will depend on factors including the extent of the outbreak and the rapidity with which it is contained and/or disease free zones are established.

The report estimated that Australia's beef and live cattle exports are valued at around $4,500 million per year; mutton, lamb and live sheep exports at $1,200 million; exports of pig meat are worth over $180 million per year; dairy exports are valued at over $3 billion annually; and wool exports are around $3.8 billion per year (Productivity Commission, 2002). The initial loss and likely prolonged disruption of these export markets is estimated to have a highly significant impact at the national level, resulting in a ranking of 'G' for this criterion.

Indirect impact on the environment

An outbreak of FMD of the magnitude described by this scenario is likely to have indirect environmental impacts resulting mainly from the disposal of large number of animal carcasses. Additional indirect environmental impacts could arise as described for scenario 3. Overall it was considered that the indirect impact on the environment was of minor importance at the national level. This resulted in a rating of 'E' for this criterion.

Indirect impact on communities

Factors to be considered under this criterion include the rural and regional economic viability. The Productivity Commission used the MONASH Multi-Regional Forecasting (MMRF) model to estimate the indicative impacts on the Australian economy of changes in production and demand that would result from an FMD outbreak. It was concluded that for the outbreak scenarios studied there would be a significant effect on the Australian economy. In particular, for a 12 month outbreak encompassing three states it was estimated that the Australian Gross Domestic Product (GDP) would decline by $2 billion in the first year and over 10 years by $8 billion to $13 billion.

When these issues were collated, the indirect impact of FMD on the environment and rural communities was considered to be highly significant at the national level, resulting in a ranking of 'G' for this criterion.

The overall impact of foot-and-mouth disease virus

When the direct and indirect impacts of FMD were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible (feral pigs, backyard pigs), high (small commercial piggeries)

Scenario 2: Consequences high

Scenario 3: Consequences extreme

Scenario 4: Consequences extreme

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 12, Table 13 and Table 14. It can be seen that the likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps in each case were considered to be 'extreme'.

Table 12 FMD: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Low	High	Moderate
Scenario 3	Low	Extreme	High
Scenario 4	Low	Extreme	High
		Overall likely consequences	**Extreme**

Table 13 FMD: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Very low	High	Low
Scenario 3	Moderate	Extreme	Extreme
Scenario 4	Low	Extreme	High
		Overall likely consequences	**Extreme**

Table 14 FMD: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	High	Moderate
Scenario 2	Very low	High	Low
Scenario 3	Moderate	Extreme	Extreme
Scenario 4	Moderate	Extreme	Extreme
		Overall likely consequences	**Extreme**

Human life or health

Separate to the above is consideration of the consequences to human life or health. Foot-and-mouth disease virus may very occasionally infect humans (Prempeh, et al., 2001). Disease signs in humans are mild and may include tingling blisters on the hands, feet and mouth, fever, and

sore throat. There has never been a case of a human transmitting FMD virus to an animal, although mechanical transmission can easily occur[36].

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with foot-and-mouth disease virus.

Table 15 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly higher than Australia's ALOP (very low), risk management would be required for FMD virus.

Table 15 FMD: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Low	High	Extreme	Extreme
Backyard pigs	Low	High	Extreme	Extreme
Small commercial piggeries	Low	High	Extreme	Extreme
			Overall annual risk	**Extreme**

[36] www.daff.gov.au

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Vesicular stomatitis virus

Technical information

Background

Vesicular stomatitis occurs only in the Americas. The disease has not established elsewhere despite horses being exported to Europe from North America in 1915 where clinical signs consistent with vesicular stomatitis were observed (Webb & Holbrook, 1989). Serological evidence of infection with vesicular stomatitis virus has been reported in many species of wildlife and domestic animals; however, clinical disease is usually observed only in horses, cattle and pigs. The vesicular lesions in cattle and pigs are clinically indistinguishable from those caused by foot-and-mouth disease (FMD). For this reason vesicular stomatitis is classified as an OIE List A disease.

Agent taxonomy

The vesicular stomatitis virus is a member of the *Vesiculovirus* genus of the Rhabdoviridae family (Pensaert, 1989).

Agent properties

The virus is a single-stranded, enveloped RNA virus with a negative-sense genome (Pensaert, 1989). Two main serotypes occur; (1) vesicular stomatitis virus New Jersey (VSV-NJ) and (2) vesicular stomatitis virus Indiana (VSV-IN, 3 subtypes). The virus is reported to be stable within a pH range of 4 to in excess of 10 (Fong & Madin, 1954; Patterson, et al., 1958). The virus is sensitive to heat, being inactivated in porcine, equine or bovine serum when heated for 30 minutes at 58°C and 60°C (Shahan, 1946). However, the virus is stable for prolonged periods at low temperatures (Galasso, 1967). Infectivity of vesicular stomatitis virus in 'defibrinated hog cholera blood' was maintained during refrigerated storage for at least 40, but less than 52, days (Shahan, 1946). The half-life of the virus stored as a cell lysate at -30, 4, 23 and 37°C was 123 days, 51 days, 2.7 days, and 7.2 hours, respectively (Galasso, 1967). Tissues from pigs experimentally infected with VSV-NJ retained infectivity for 2 but not 4 weeks when stored at 7°C (Patterson, et al., 1955). The survival of vesicular stomatitis virus in fermented edible waste material has also been examined after incubation at 5, 10, 20 or 30°C for 96 hours. The virus was rapidly inactivated at all four temperatures (Wooley, et al., 1981). The virus is quite sensitive to the effects of ultraviolet light. Infectivity of vesicular stomatitis virus suspensions were reduced by up to five log units after exposure to light under a variety of conditions (Skinner & Bradish, 1954).

Host range

Clinical disease resulting from infection with vesicular stomatitis virus has been reported to occur most often in horses, followed by cattle and pigs. Vesicular stomatitis is a mild zoonosis (Reif, et al., 1987; Hugh-Jones, et al. 1995). Antibodies to vesicular stomatitis virus have been detected in a wide range of vertebrate species such as humans, other primates, bovines, murines, hamsters, marsupials, reptiles, fish and birds (Johnson, et al., 1969; Jimenez, et al., 1996). In addition, the virus has been isolated from many haematophagous and non-haematophagous insect species including sand flies, black flies, mosquitoes, culicoides, house flies and eye gnats (Rodriguez, 2002).

Epidemiology

Sporadic outbreaks of clinical disease occur periodically in horses and cattle in the western United States of America, typically sweeping from the south near the Mexican border up through the Rocky Mountain region during summers and disappearing with the first severe frost (Webb & Holbrook, 1989). The virus is endemic from northern South America to southern Mexico (Rodriguez, 2002). An endemic focus of VSV-NJ has been identified in the eastern United States of America (Ossabaw Island, Georgia) where the virus repeatedly has been isolated from feral and domestic pigs and phlebotamine sandflies (Stallknecht, 2000).

The epidemiology of vesicular stomatitis is not well understood. Viral reservoirs, amplification hosts, and natural modes of transmission are unclear (Cornish, et al., 2001). In clinically affected animals, vesicular fluids contain extremely high concentrations (in excess of 10^8 $TCID_{50}$/ml) of virus (Clarke, et al., 1996) and susceptible animals may be exposed to these fluids by direct contact, contact with contaminated fomites, or (possibly) aerosol (Johnson, et al., 1969; Stallknecht, et al., 2001). The virus gains access to a vertebrate host via minor abrasions or trauma to skin or mucosal surfaces. However, particularly in endemic areas, where subclinical infection is common transmission is unlikely to be due to contamination with vesicular fluids. Insects have been implicated as both mechanical and biological vectors of the disease. Epidemiological support for the involvement of insects in the disease cycle has been summarised (Schmidtmann, et al., 1999) and includes the following: (1) seasonal incidence, with outbreaks coinciding with warm temperatures that promote insect activity; (2) livestock on pasture are generally at highest risk for exposure; and (3) during epizootics in western United States of America, VSV-NJ has been isolated from several species of Diptera including species recognised as mechanical and biological vectors of other arboviruses. Transmission of virus from infected sand flies and black flies to susceptible vertebrates has also been demonstrated experimentally (Comer, et al., 1990; Mead, et al., 1999). However, arguments against the insect transmission of vesicular stomatitis virus include (Webb & Holbrook, 1989; Schmidtmann, et al., 1999) (1) the repeated 'affliction' of animals in certain pastures but not those in adjacent pastures, although insects move freely between pastures; (2) the occasional sudden involvement of a large proportion of a herd and brief duration within a herd (indicating common exposure); and (3) the general absence of secondary waves of infection during the vector season. In addition, viraemia has never been detected in domestic animals, thus these animals do not appear to act as amplifying hosts for vesicular stomatitis virus. Viraemia (after experimental infection) has been reported only in rodents, including laboratory mice, spiny rats, Syrian hamsters and deer mice (Cornish, et al., 2001), and it is suggested that deer mice and/or other native American rodents may be involved in the epidemiology of vesicular stomatitis.

Pigs are susceptible to infection by both VSV-NJ (Stallknecht, 2000) and VSV-IN (Yedloutschnig & Dardiri, 1977); however, VSV-NJ has been the predominant serotype isolated from pigs (Stallknecht, et al., 1986; House & House, 1999). The history of vesicular stomatitis in pigs in North America has been summarised (Carbrey, 1989). Clinical vesicular stomatitis was first observed in a hog cholera antiserum production plant in Missouri in 1943. An extensive outbreak occurred in pigs in Colorado in 1944, and in 1952 and 1954 vesicular stomatitis was diagnosed in pigs in Georgia. In 1967, the only clinical outbreak of the disease in the United States of America was in a herd of pigs in Louisiana. Infection of pigs has not been a feature of the sporadic outbreaks of clinical vesicular stomatitis (mentioned above) that occur periodically in the western United States of America (McCluskey, et al., 1999).

In contrast, infection of feral pigs with VSV-NJ is a feature of the endemic focus of the disease on Ossabaw Island off the coast of Georgia, although lesions are detected only rarely

(Stallknecht, 2000). Ongoing, long-term studies of the island and its ecosystem have been conducted. Serum neutralising antibodies to VSV-NJ have been detected in six of 17 mammalian species studied, but in none of the avian, reptile or amphibian species examined. The virus appears to be maintained and transmitted only on a small portion of the island, apparently associated with the presence of old growth maritime forest and thence likely also with the distribution of the sandfly species *Lutzomyia shannoni* (Fletcher, et al., 1991; Stallknecht, 2000). Serial bleeding of sentinel pigs on the island in 1984 and 1985 showed that viral activity began during mid-May, varied between years, and differed between island locations (Stallknecht, et al., 1987). A capture/recapture technique was used to serially monitor the proportion of captured feral pigs that were seropositive. The proportion of seropositive pigs trapped in the southern part of the island (viral activity appeared absent in the northern part) increased in 1984 from 5% (one of 19 pigs) in May, to 83% (five of six pigs) in September. Similarly, in 1985, the proportion increased from 10% (nine of 92 pigs) in May to 51% (22 of 42 pigs) in September (Stallknecht, et al., 1987).

Clinical signs

Clinical signs of vesicular stomatitis in pigs, when present and severe, are indistinguishable from those of FMD. These include fever (40°C to 41°C), drooling, and vesicle formation. Vesicles may be present on the tongue, snout and coronary band, and may reach 3 cm in diameter. Severe lameness may result from the foot lesions, and secondary bacterial infection frequently occurs (Carbrey, 1989). The OIE describes the incubation period as lasting up to 21 days; however, after experimental infection, virus may be recovered within 24 hours of inoculation of pigs from some or all of tonsils, nasal swabs or vesicular lesions (Patterson, et al., 1955; Stallknecht, et al., 1999; Stallknecht, et al., 2001). Absence of clinical signs is characteristic of natural infection of domestic and feral pigs with the Ossabaw Island strain of VSV-NJ, although vesicles may be detected at sites of experimental inoculations, depending on the amount of virus administered (Clarke, et al., 1996; Howerth, et al., 1997).

The clinical signs of vesicular stomatitis in cattle and horses are similar to those observed in pigs; in cattle (as in pigs) these are indistinguishable from those of FMD. Subclinical infection occurs in these species (Letchworth, et al., 1999).

Serological evidence of infection with vesicular stomatitis virus has been reported in many vertebrate species, (as mentioned above), including carnivorous mammals such as dogs and foxes (Letchworth, et al., 1999; Miller, et al., 2000). However, signs of clinical disease in these animals are not mentioned.

In humans, infection with vesicular stomatitis virus is characterised by an influenza-like illness that is usually mild and of short duration (Reif, et al., 1987). Occasionally, vesicles may be observed in the mouth, pharynx, or on the hands (Hugh-Jones, et al. 1995). There are no reports of human to human transmission.

Pathogenesis

The pathogenesis of vesicular stomatitis in pigs after experimental or natural infection is not well understood. Evidence of infection with vesicular stomatitis virus as evidenced by seroconversion has been demonstrated after inoculation of pigs by a variety of routes including intradermal (snout, ear, coronary band), intravenous, oral, and application to scarified oral mucosa, and skin of the snout and coronary band (Howerth, et al., 1997; Stallknecht, et al., 1999; Stallknecht, 2000). However, application to the conjunctiva or as a nasal aerosol did not

result in evidence of infection (Stallknecht, et al., 1999). Absence of detectable viraemia was a consistent feature of these studies. Virus was isolated from tonsillar swabs from at least one pig after all routes of inoculation with the exception of the conjunctival route. In some cases, this may be secondary to direct contact following the swallowing of virus-contaminated fluids. However, the possibility of systemic spread exists, particularly as virus was isolated from tonsillar swabs from pigs in which no vesicular lesions were observed (Stallknecht, et al., 1999). In another study, in one of three pigs, virus was isolated from a tonsillar swab 5 days after inoculation of the coronary band of the right rear foot of one pig, however, a vesicular lesion was also noted at this time and it is possible that ingestion of virus occurred (Howerth, et al., 1997).

The amount of VSV-NJ detected in swabs from the nasal planum, nasal cavity, saliva, tonsils and faeces from infected pigs in one study consistently exceeded 10^2 $TCID_{50}$, which was the dose used to infect the pigs via a break in the skin or mucous membranes. Shedding of virus reached a maximum by day four post-inoculation, and was difficult to detect after the sixth day (Stallknecht, et al., 1999). In another study, virus was isolated 8 days after inoculation from the tonsil of only one of 24 pigs at necropsy. All other tissues examined (multiple lymph nodes, skin samples, brain) from these pigs were negative (Howerth, et al., 1997). Another worker (Redelman, et al., 1989) reported isolating infective virus from the salivary gland, tonsil, and skin near the site of inoculation at 4 days post-inoculation. However, at 6 days post-inoculation, virus was recovered only from skin sampled near the inoculation sites. Virus has been isolated 10 days after infection from the tonsils of pigs that have seroconverted (Clarke, et al., 1996).

Pathology

Vesicles on the skin of the lips, snout, coronary band and interdigital space are characteristic, but are clinically indistinguishable from other vesicular diseases such as foot-and-mouth disease, swine vesicular disease, and vesicular exanthema of swine. Subclinical infections are common, and gross pathological changes may be absent. On occasion, congestion of the liver may be detected. Microscopically, histopathological changes may be observed at the sites of the lesions, extending to the dermal layers (Chow, 1953).

Immunology

Following natural or experimental infection with vesicular stomatitis virus, pigs produce specific neutralising antibodies, the levels of which may be detectable as early as 4 days (Stallknecht, et al., 1999) and which peak around 3 to 5 weeks following infection (Redelman, et al., 1989). It is likely that in pigs, as in horses and cattle, a protective response to one serotype will not provide cross-protection against the other (McCluskey, et al., 1999). To date, however, all natural infections with vesicular stomatitis virus in feral swine in the United States have involved the New Jersey serotype (Stallknecht, et al., 1986). However, in another study, the frequency of viral isolation decreased dramatically after seroconversion (Stallknecht, et al., 1999). Virus was isolated from 19% of 435 swabs collected prior to seroconversion in contrast to isolation of virus from less than 1% of 195 swabs collected after seroconversion. The presence of high levels of circulating antibody in cattle is not sufficient to prevent clinical disease in cattle, as one review cites evidence that most animals with clinical vesicular stomatitis in an endemic area have neutralising antibody titres prior to onset of disease (Letchworth, et al., 1999).

Transmission via meat

There are few data available on the oral infectious dose of vesicular stomatitis virus. It has been postulated that pigs may become infected via swallowing virus contaminated fluid. Moreover it is known that application of virus to the oral mucosa can result in infection. In one experiment, pigs were orally infected by application to the surface of the oral mucosa of 10^6 (but not 10^4) $TCID_{50}$ VSV-NJ (Stallknecht, et al., 1999). The three infected pigs did not develop detectable vesicular lesions, but virus was isolated from the saliva of one, and the tonsils of all three pigs. All pigs seroconverted between 5 and 7 days post-inoculation.

The potential for transmission of vesicular stomatitis virus via meat has been studied (Patterson, et al., 1955). Briefly, eight pigs were inoculated intravenously with VSV-NJ and slaughtered 54 hours later. Vesicular lesions were observed in seven of eight inoculated pigs. Three categories of carcass material were collected from these pigs: 1) snout, feet and skin; 2) viscera, including lymph nodes, heart, spleen, kidney, liver, lungs, section of intestines and crushed bone, and 3) chopped muscle tissue. Twenty-four recipient pigs were housed in pairs and fed 4.5 kg carcass materials per category per pig after fasting for 48 hours. One recipient pig of each pair was scarified on the snout and two front feet prior to feeding. Materials were fed immediately, or after 1, 2 or 4 weeks of storage at 7°C. Recipient pigs were monitored for clinical signs of infection, and 3 weeks later any that did not develop clinical signs of infection were challenged directly with vesicular stomatitis virus to determine whether immunity had developed.

Clinical signs of vesicular stomatitis infection were observed only in pigs fed snout-feet-skin material (one of the two pigs fed fresh material, and one of the two pigs fed 2 week old material). Although not demonstrating clinical signs, one of the pigs fed snout-feet-skin material stored for 1 week appeared immune when subsequently challenged with vesicular stomatitis virus. None of the pigs fed the viscera-bone material either displayed clinical signs or appeared to develop immunity to subsequent challenge. However, one pig fed fresh muscle tissue appeared to be immune when challenged three weeks later. Pigs fed muscle tissue stored for 1 week or more did not demonstrate immunity to challenge. It should be noted that when this experiment was conducted no serological tests were available and hence it is unknown if the recipient pigs were truly naïve, and thus susceptible to infection.

In an additional experiment reported in this study, tissue scraps (pooled tissues containing foot and snout tissues, meat, viscera, blood and crushed bone) were obtained from pigs inoculated intravenously and then slaughtered from 6 hours up to 15 days post-inoculation. The pooled tissue scraps collected from pigs slaughtered from 30 hours up to 8 days post-inoculation were found to be infective to recipient pigs, either by the development of vesicular lesions or immunity to subsequent challenge. However, the authors noted that in all cases where lesions were observed after feeding infective tissues, the pig that developed lesions had been scarified prior to feeding. They concluded that although vesicular stomatitis virus may be spread by the feeding of infective tissues, transmission appeared to have resulted from these tissues coming in contact with abraded skin of the recipient pigs, rather than by ingestion of the material.

There are no reports of outbreaks of vesicular stomatitis being associated with trade in meat or meat products.

Release assessment

R1 — the likelihood that a source herd is infected

The likelihood that a source herd of pigs is infected is difficult to assess, as detailed information concerning vesicular stomatitis virus infection in pigs in an endemic area is limited to reports of infection in feral and sentinel domestic pigs from Ossabaw Island, in Georgia (USA). Some information is available on the prevalence of infection in cattle. One study of 22 dairy farms in Costa Rica (a vesicular stomatitis virus endemic area) showed that cattle from nine farms became infected during the study period (Vanleeuwen, et al., 1995). Another study in Costa Rica determined the overall seroprevalence of vesicular stomatitis virus in cattle, finding 46% and 21% were seropositive for VSV-NJ and VSV-IN respectively (Atwill, et al., 1993). In 1995 in the United States of America during an epidemic of vesicular stomatitis, it was determined that at least one animal on 41% of 890 premises investigated in 6 states was infected. Of the infected animals, horses were identified on 78% of the 362 'infected' premises, and cattle were identified on the remaining 22%. One vesicular stomatitis positive llama was identified during the outbreak (Bridges, et al., 1997).

Given this, the likelihood that slaughter-age pigs have been selected from an infected herd in a country where vesicular stomatitis virus is endemic was considered to be 'moderate'.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

The incidence of seroconversion amongst feral pigs on Ossabaw Island has been studied using a capture/recapture technique. The true incidence of infection is impossible to determine from this approach; nevertheless, it appeared that a moderate-to-high proportion of susceptible pigs were infected each year. In dairy cattle, one study reported that on three farms in Costa Rica the seroprevalence of VSV-NJ was 94.2%, which did not differ significantly between herds. However, the seroprevalence of VSV-IN averaged 15.2%, and was significantly higher in one herd. Nonetheless the annual incidence rate of clinical infection was only 9% (Rodriguez, et al., 1990). This is in agreement with another study where the annual incidence rate of clinical infection was found to be 11.1% (Vanleeuwen, et al., 1995). In Mexico the seroprevalence of VSV-NJ and VSV-IN for two herds averaged 36% and 13% respectively (Hernadez de Anda, et al., 1992).

Although within herd seroprevalence can be high, the length of time for which a pig may be infectious to others appears to be quite short, approximately one week (Patterson, et al., 1955; Howerth, et al., 1997; Stallknecht, et al., 1999). Combining this information, it was considered that the likelihood of selecting an infected pig in an infected herd was 'low'.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

It was considered that pigs exhibiting signs of classical vesicular stomatitis virus infection would be detected and removed from processing. However, natural infection of pigs with VSV-

NJ is often clinically undetectable. Thus, the sensitivity of the ante-mortem, slaughter and processing requirements was considered to be ‘extremely low’.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with vesicular stomatitis virus and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a ‘one minus the background rejection rate’. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered ‘extremely low’. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

Vesicular stomatitis virus has a tropism for epithelial cells. Viraemia, if it occurs in domestic mammals, is extremely short and/or occurs at very low levels. Nonetheless one study conducted many years ago demonstrated that fresh muscle tissue obtained from recently infected pigs appeared to result in subclinical infection when fed to a naïve pig (Patterson, et al., 1955). It should be noted that most muscle tissue was obtained from pigs with vesicular lesions. These pigs would be very unlikely to pass ante-mortem inspection.

It is also known that virus may be isolated from the tonsillar tissue of pigs that do not exhibit vesicular lesions. In one study in which pigs were infected with vesicular stomatitis virus by a variety of routes and monitored for seroconversion and the presence of lesions, 20 pigs seroconverted and lesions were noted in 10 of these (Howerth, et al., 1997). Virus was isolated from the tonsillar swabs of six of the pigs without lesions (and from seven of those that had lesions). Nonetheless most tonsillar tissue will be removed at slaughter.

In view of the above factors, it was considered that the likelihood that vesicular stomatitis virus would be present in meat harvested from an infected pig was ‘very low’.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

Vesicular stomatitis virus is stable within a pH range of 4 to 10, and has been shown to survive in porcine tissues after slaughter. Thus, it was considered that there was a ‘high’ likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

The virus survives storage at low temperatures for prolonged periods. Tissues from pigs experimentally infected with VSV retained infectivity for at least two weeks when stored at 7°C. In view of this, it was considered that there was a ‘high’ likelihood that meat infected at the completion of carcass maturation would remain infected during transport and cold storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a 'very low' likelihood that imported pig meat derived from an individual carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

When vesicular stomatitis virus was applied directly to the oral mucosa of each of three pigs, a viral quantity of 10^6 $TCID_{50}$ resulted in infection of all three pigs, whereas none was infected after application of 10^4 $TCID_{50}$ (Stallknecht, et al., 1999). Although infection of pigs after exposure to infected meat scraps has been demonstrated, the authors of the study suggested that infection occurred via contact of the virus-containing meat with scarified skin of the recipient pig, rather than by ingestion (Patterson, et al., 1955). The donor pigs in this study were infected by intravenous inoculation; viraemia is not a feature of natural infection. The viral titre of muscle was not determined. It has been shown that viral titres exceeding 10^2 $TCID_{50}$ have been sufficient to infect pigs when applied to scarified skin or scarified mucous membranes (Stallknecht, et al., 1999).

Taking these factors into consideration, the likelihood that a waste unit from an infected pig would contain a sufficient dose of vesicular stomatitis virus to initiate infection was considered to be 'extremely low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

Vesicular stomatitis virus is susceptible to increasing temperatures and to ultraviolet light. The half-life of vesicular stomatitis virus stored as a cell lysate at 23°C and 37°C was 2.7 days and 7.2 hours, respectively (Galasso, 1967). In light of this information, it was considered that the likelihood that vesicular stomatitis virus would survive in meat scraps discarded in refuse for the period of time required for feral pigs to locate and subsequently scavenge the material was 'low'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High

- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = Extremely low
- Rural regions = Extremely low
- Large towns = Negligible

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be ‘extremely low’.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a ‘very low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was ‘extremely low’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall the Panel considered that there was a 'moderate' likelihood that vesicular stomatitis virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'extremely low'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected would contain a sufficient dose of the pathogenic agent to initiate infection was 'extremely low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall the Panel considered that there was a 'moderate' likelihood that vesicular stomatitis virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'extremely low'.

Exposure assessment for other susceptible species

Disease resulting from infection with vesicular stomatitis virus is most often reported in horses and, to a lesser extent, cattle. Pigs are clinically affected only rarely, as are humans. Serological evidence of infection is reported in a wide range of other vertebrate species; however, convincing evidence of their involvement in the epidemiology of the disease is lacking, with the possible exception of the deer mouse (a rodent) (Cornish, et al., 2001). The deer mouse is not present in Australia. It is unclear how these species are infected with the virus; however, insects are suspected as likely mechanical or biological vectors. Natural infection of any other susceptible species (such as rodents, foxes, dogs, cats) via ingestion of meat scraps has never been reported nor implicated in the establishment or spread of vesicular stomatitis in the Americas or any other country. On the basis of this information, the annual likelihood of entry and exposure for other susceptible species was considered to be 'negligible'.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the four exposure groups - that is, feral pigs, pigs in backyard enterprises pigs in small commercial piggeries and other susceptible species. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs, however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

For transmission of vesicular stomatitis virus to occur via contact, an infected pig would need to shed extremely large quantities of virus (that is, from vesicular fluids) (Howerth, et al., 1997). Vesicular lesions are rarely manifest in naturally-infected pigs on Ossabaw Island in Georgia, and thus contact transmission is not thought to be an important factor in the epidemiology of the disease in this location. Moreover, pigs are thought to be able to transmit the virus for only about 1 week. In view of this information, it was considered quite unlikely that a feral pig consuming infected meat scraps would exhibit vesicular lesions as a consequence of infection and transmit the virus to herd mates or to other groups of feral pigs.

Insects have been implicated as both mechanical and biological vectors of the disease. It is unknown if Australia has suitable invertebrate and vertebrate hosts for establishment of the virus. Importantly the disease has not become established outside the Americas, which may indicate that a special ecological niche is required. Nonetheless if suitable hosts were present in Australia, wider spread of the virus could occur such as to other feral pigs and other susceptible hosts. This in turn may lead to sweeping outbreaks of the disease in the domestic livestock population as seen periodically in the southwestern United States of America.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: high

Scenario 2: very low

Scenario 3: very low

Scenario 4: very low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

As described above for feral pigs, it was considered unlikely that a pig, ingesting infected meat scraps, would develop vesicular lesions. However, should this occur, there may be transmission amongst the backyard herd. Despite the significant publicity campaigns to report any signs of vesicular disease to State animal health authorities, it is likely that, either disease would not be recognised in a single premises or would go unreported. If pigs with vesicles from a backyard herd were moved to another premises, for example, in the case of speciality breeds or unusual breeds live pigs transferred for breeding purposes or alternatively, pigs raised for personal consumption transferred between backyard holdings for growing out or fattening, further transmission of the disease could occur.

The spread of vesicular stomatitis from backyard herds to feral pigs, or to a wider population of domestic pigs and other susceptible species such as horses and cattle, would depend to a large degree on the establishment of the virus in an Australian environmental niche, with adequate reservoirs of infection.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: high

Scenario 2: very low

Scenario 3: very low

Scenario 4: very low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

As described above for feral and backyard pigs, it was not considered likely that a pig, ingesting infected meat scraps, would develop vesicular lesions. However, should this occur, transmission amongst the small commercial herd may result. Should vesicular lesions develop, managers of small commercial piggeries are likely to contact a veterinarian or State animal health authority. Managers, owners and veterinarians are well aware of the clinical signs of foot-and-mouth disease (these are indistinguishable from those of vesicular stomatitis) with active publicity campaigns for exotic diseases occurring in Australia. The emergency response to a vesicular disease would be effective in limiting the spread of the disease, provided that the virus had not become established in a suitable environmental niche, with alternative vertebrate and possibly invertebrate hosts.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: high

Scenario 2: very low

Scenario 3: very low

Scenario 4: very low

Other susceptible species

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of other susceptible species:

1. Containment of the disease within the sub-population of directly exposed animals - no secondary spread - this option represents the 'no outbreak' scenario;

2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of domestic pigs - containment within a local population of backyard enterprises or small commercial piggeries - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs - exposure of medium-large commercial piggeries and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Other susceptible species, which are carnivorous, include animals such as some rodents and foxes. Vesicular lesions are not generally reported as a consequence of natural infection in species other than domestic animals, and in the absence of an established ecological niche with appropriate reservoir hosts, it is difficult to envisage the spread of the virus beyond the initially-infected animal. If suitable vertebrate and invertebrate hosts exist in Australia vesicular stomatitis may spread to other susceptible species including feral pigs and domestic livestock.

In areas where the virus is present such as the United States of America a survey of kit fox for serological evidence of vesicular stomatitis virus infection detected antibodies to VSV-NJ and VSV-IN in 20% and 14% of animals respectively (Miller, et al., 2000). A survey of small mammals from the order Rodentia, located in a vesicular stomatitis virus enzootic focus, found that 43% (9 of 21) of Hispid Cotton rats (*Sigmodon hispidus*) had antibodies to the virus (Jimenez, et al., 1996).

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: high

Scenario 2: very low

Scenario 3: very low

Scenario 4: very low

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes ‘no further establishment or spread’. The remaining three outbreak scenarios describe ‘secondary spread to other pigs, humans or to other susceptible species’. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this scenario, vesicular stomatitis would have established in the directly exposed animal, or group of animals, but would not have spread to other animals. In the case of a feral pig herd or backyard pig enterprise or other susceptible species being infected, this ‘no outbreak’ scenario would have resulted from low probability of contact between infected and susceptible animals, rather than from human intervention. Indeed, because the disease may be of low

pathogenicity for pigs and carnivorous or omnivorous other susceptible species, it was assumed that it would not have been identified in these exposure groups. In the case of a small commercial piggery, due to the closer observation, it was assumed that the disease would have been identified and contained due to implementation of a control and eradication program.

The direct impact of vesicular stomatitis

Animal life or health

Subclinical infections with vesicular stomatitis virus are common in pigs. However, fever and vesicular lesions may occur, and these lesions may be accompanied with difficulty eating or lameness, depending on location of the lesions. Most pigs recover fully within two weeks. Generally there is no clinical evidence of infection in other susceptible species. Due to the restricted extent of this scenario, and the variable manifestations of the disease, the likely impact of vesicular stomatitis on animal health was considered unlikely to be discernible at any level. This resulted in a rating of 'A' for this criterion.

Environment

Under this outbreak, vesicular stomatitis is confined to the primary exposure group and in the case of other susceptible species may include carnivorous Australian native animals. Although the susceptibility of these animals to vesicular stomatitis virus is unknown, it would appear that in the Americas most animals do not show clinical signs of infection. In view of this, the direct impact on the environment was considered unlikely to be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of vesicular stomatitis

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

This criterion was not rated identically for each exposure group.

It is likely that if the disease were contained within a feral pig herd or a single backyard enterprise, or another susceptible species, vesicular stomatitis would not be diagnosed within these herds. However, if the primary outbreak involved a small commercial piggery it was considered that pigs showing clinical signs of a vesicular disease would be investigated.

If vesicular stomatitis was identified in Australia in a small commercial piggery, the policy as outlined in AUSVETPLAN (Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 1996) is to eradicate vesicular stomatitis, recognising that the virus may be transmitted by a variety of insect vectors and that the disease does not always follow predictable transmission and distribution patterns. However, if eradication cannot be achieved, the policy will be modified to contain the disease and to minimise the effects on trade. A combination of strategies will be employed, including judicious slaughter of clinically affected animals, quarantine and movement controls, tracing and surveillance, vector control, decontamination, epidemiological investigations, and a public awareness campaign. The disease is classed as Category 2 under the Australian Emergency Animal Disease Cost-Sharing Agreement[37], and thus the cost of the response is to be covered by government and relevant industries by contributions of 80% and 20%, respectively. Category 2 diseases have the

[37] http://www.aahc.com.au/eadp/response.htm

potential to cause major national socio-economic consequences through very serious international trade losses, national market disruptions and very severe production losses in the livestock industries that are involved.

In this scenario where vesicular stomatitis has not spread beyond the initial small commercial piggery, it is likely that the disease would be eradicated promptly. Nonetheless there would need to be extensive surveillance of the domestic and feral pig populations and the local ruminant and horse populations, and possibly wildlife.

Overall, it was considered that the indirect impact of new eradication and control programs was unlikely to be discernible at any level when the exposure group was a feral pig herd, a backyard pig enterprise, or another susceptible species. This resulted in a rating of 'A' for this criterion. However, when the exposure group was a small commercial piggery, it was considered that the indirect impact of new eradication and control programs was unlikely to be discernible at the national level, but of minor significance at the State level. This resulted in a rating of 'D' for this criterion.

Domestic trade or industry effects

This criterion was not rated identically for each exposure group.

As discussed above, it was considered unlikely that the disease would be detected in the initially exposed herd of feral pigs, single backyard enterprise, or other susceptible species, thus no domestic trade or industry effects would be expected, and the rating assigned to this criterion was 'A'.

In the case of a small commercial piggery it was considered that the pigs with vesicular lesions would likely be investigated. Once diagnosed, an eradication program would be implemented as discussed above. Movement restrictions would be imposed. These controls would affect all susceptible species within the restricted area, not just pigs, and other controls likely would be imposed in the control area. Movements of animals to sale and slaughter would also be affected. It is possible that following detection of vesicular stomatitis in one State of Australia, other States may close their borders to all susceptible animals and products until the extent of the outbreak was ascertained.

Taking these issues were taken into account, when the exposure group was a small commercial piggery, it was considered that the indirect impact of vesicular stomatitis on domestic trade and industry was unlikely to be discernible at the national or State level, but of minor significance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

International trade effects

This criterion was not rated identically for each exposure group.

As the disease is unlikely to be detected in the initially exposed herd of feral pigs, backyard enterprise, or other susceptible species, the indirect effects of vesicular stomatitis on international trade for these exposure groups was unlikely to be discernible at any level, and thus, the rating assigned to this criterion was 'A'.

The identification of vesicular stomatitis in a single small commercial piggery would likely result in some disruption to exports, particularly in the case of live animals, until the extent of the outbreak was known. After the resumption of trade, animals may need to be tested for

vesicular stomatitis. There may be some disruption to trade in meat, however, this was considered likely to be minor and of a short duration.

On balance, it was considered that the indirect effects on international trade when the exposure group was a small commercial piggery would be unlikely to be discernible at the national or State level and of minor significance at the district or regional level. Thus, this criterion was rated as 'C'.

Indirect impact on the environment

In this scenario, vesicular stomatitis is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, vesicular stomatitis would have established in a broader population of feral pigs. In the case of spread from a feral pig herd, backyard pig enterprise or other susceptible species, the disease would have been contained due to low probability of contact between infected and susceptible animals, rather than from human intervention. Indeed, because the disease may be of low pathogenicity for pigs, it was assumed that it would not have been identified in these exposure groups and feral pigs. In the case of spread from a small commercial piggery to feral pigs, it was assumed that the disease would have been identified in the small commercial piggery and contained due to implementation of a control and eradication program.

The direct impact of vesicular stomatitis

Animal life or health

With this scenario, the disease spreads to a general population of feral pigs but not to domestic pigs. Clinical signs in pigs may vary from undetectable to severe. Nevertheless, mortality is rare and generally due to secondary complications, and most animals showing clinical signs will recover within two weeks. Overall, the direct impact on animal health is unlikely to differ from that of outbreak scenario 1 and thus, this criterion was rated as 'A'.

Environment

As with outbreak scenario 1, it was considered that the direct impact on the environment was unlikely to be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of vesicular stomatitis

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

This criterion was not rated identically for each exposure group.

In this scenario, although the disease has spread to a more general population of feral pigs it was considered unlikely the outbreak would be detected when the primary exposure group was feral pigs, backyard pigs, or other susceptible species. Signs of infection with VSV-NJ in feral pigs on Ossabaw Island are seen extremely rarely, although many of these animals are scrutinised closely after natural or experimental infection.

When the direct exposure group is a small commercial piggery it was considered that the disease would be diagnosed and eradication and control programs implemented as discussed for scenario 1. However, the extent and costs of any eradication or control programs will depend on the results of surveillance and assessment of the role of feral pigs in the epidemiology of the disease in domestic animals.

After consideration of these issues, the indirect impact of new eradication and control programs was unlikely to be discernible at any level when the direct exposure group was a feral pig herd, a backyard pig enterprise, or another susceptible species. This resulted in a rating of 'A' for this criterion. However, when the direct exposure group was a small commercial piggery, it was considered that the indirect impact of new eradication and control programs was unlikely to be discernible at the national level, but of minor significance at the State level. This resulted in a rating of 'D' for this criterion.

Domestic trade or industry effects

This criterion was not rated identically for each exposure group.

As discussed above, it was considered unlikely that the disease would be detected when the direct exposure group was a feral pig herd, backyard enterprise, or other susceptible species, thus no domestic trade or industry effects would be expected, and the rating assigned to this criterion was 'A'.

However, if the source of the outbreak was a small commercial piggery, the indirect effect on domestic trade or industry would be similar to that described in outbreak scenario 1 and a rating of 'C' was assigned for this criterion.

International trade effects

This criterion was not rated identically for each exposure group.

As the disease is unlikely to be detected in the initially exposed herd of feral pigs, backyard enterprise, or other susceptible species, nor with secondary spread to feral pigs, the indirect effects of vesicular stomatitis on international trade for these exposure groups was unlikely to be discernible at any level, and thus, the rating assigned to this criterion was 'A'.

As with scenario 1, the identification of vesicular stomatitis in any pigs could result in disruption to exports of live animals and possibly initially some markets for meat. As under this scenario, the disease has not spread from the small commercial piggery to other domestic livestock but only to feral pigs, it was considered that the indirect impacts on international trade would be the same as for scenario 1. Hence, when the direct exposure group was a small commercial piggery a rating of 'C' was assigned for this criterion.

Indirect impact on the environment

In this scenario, vesicular stomatitis is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, vesicular stomatitis would have established in a local population of backyard piggeries or small commercial piggeries, cattle and horses. The disease would be contained through the diagnosis of disease in any of the affected species, and the mounting of an eradication program.

The direct impact of vesicular stomatitis

Animal life or health

In this scenario, vesicular stomatitis spreads to a local population of domestic pigs and other susceptible species including cattle and horses. Clinical signs indistinguishable from foot-and-mouth disease may be seen in cattle, and horses may exhibit similar signs. Morbidity may be high, and most animals will recover. If dairy cattle were affected, milk production would decrease.

On this basis the direct effect on animal health was considered unlikely to be discernible at the national or State level, but would be of minor significant at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

Environment

In areas where vesicular stomatitis is endemic, many species of vertebrate wild animals have serological evidence of infection with the virus; however, signs of disease are not reported. Although it is not known if Australian native fauna and insects are susceptible to infection with the virus, clinical disease was considered unlikely. In view of this, the direct impact on the environment was considered unlikely to be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of vesicular stomatitis

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

Australia's policy as outlined in AUSVETPLAN (Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 1996) is to eradicate vesicular stomatitis, recognising that the virus may be transmitted by a variety of insect vectors and that the disease does not always follow predictable transmission and distribution patterns. However, if eradication cannot be achieved, the policy will be modified to contain the disease and to minimise the effects on trade. As discussed previously, the disease is classed as Category 2 under the Australian Emergency Animal Disease Cost-Sharing Agreement. Consultation between government and industry will be required to determine whether eradication is feasible; this decision will influence the costs of associated programs. In this scenario where vesicular

stomatitis has only limited spread, eradication of the disease in the livestock would be possible, however, if the disease established in a vertebrate and/or invertebrate reservoir hosts, periodic outbreaks could occur. Regardless, it was considered that the indirect impact of new or modified control programs was unlikely to be discernible at the national level, and of minor significance at the State level. This resulted in a rating of 'D' for this criterion.

Domestic trade or industry effects

As a result of the detection of vesicular stomatitis in a local population of domestic pigs or cattle or horses, movement restrictions would be imposed. These controls would affect all susceptible species within the restricted area and other controls likely would be imposed in the control area. Movements of animals to sale and slaughter would also be affected. It is possible that following detection of vesicular stomatitis in one State of Australia, other States may close their borders to all susceptible animals and products until the extent of the outbreak was ascertained. The likely involvement of cattle in this outbreak does potentially increase the severity of the impacts as clinically-affected cattle would not be accepted for slaughter for human consumption. Depending on the location of the outbreak, horse racing and horse events may be prohibited.

Taking these issues into account, it was considered that the indirect impact of vesicular stomatitis on domestic trade and industry was of unlikely to be discernible at the national level, and of minor significance at the State level. This gave the disease a rating of 'D' for this criterion.

International trade effects

The effects on international trade of a confirmed outbreak of vesicular stomatitis in Australia would be similar to that described for scenarios 1 and 2. However, with the involvement of cattle and horses, export markets for these animals may be disrupted for a greater period of time. In light of this information, it was considered that the indirect effect of vesicular stomatitis on international trade would be unlikely to be discernible at the national level, and of minor significance at the State level. This resulted in a rating of 'D' for this criterion.

Indirect impact on the environment

In this scenario, vesicular stomatitis is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

One of the considerations within this criterion was the indirect impact of a disease on rural and regional economic viability. In this scenario, where a local population of domestic pigs and other susceptible species such as cattle and horses are infected with vesicular stomatitis virus, it was considered that where these industries were important to the local economy, aspects of the rural community may be threatened. Given this, the indirect impact of vesicular stomatitis on rural communities was considered of minor significance at the district or regional level. This resulted in a rating of 'C' for this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, vesicular stomatitis would have established in a broader population of commercial piggeries (including medium-large piggeries) and cattle, sheep and horses. An eradication and control program would have been mounted in response to the diagnosis of the disease in the affected animals.

The direct impact of vesicular stomatitis

Animal life or health

In this outbreak scenario, vesicular stomatitis spreads to a more general population of domestic pigs and other susceptible species such as cattle and horses. Clinical signs indistinguishable from foot-and-mouth disease may be seen in cattle, and horses may exhibit similar signs. Morbidity may be high, but most animals will recover. Milk production of dairy cows infected would be reduced. One study investigated the economic impacts of vesicular stomatitis on dairy herds in the United States of America. The greatest losses were due to increased culling, followed by reduced milk production and increased mortality (Goodger, et al., 1985). Vesicular stomatitis can cause significant production losses in affected herds and performance losses in affected stables. Primary financial losses for beef herds have been attributed to among other things increased culling rates and death of pregnant cows (Hayek, et al., 1998). In dairy herds infected with vesicular stomatitis virus the greatest loss was due to cows culled, with decreased milk production second (Alderink, 1985).

Given the extent of the outbreak and the numbers of animals likely involved, the direct impact on animal health was considered unlikely to be discernible at the national level, but of minor significance at the State level. This resulted in a rating of ‘D’ for this criterion.

Environment

In areas where vesicular stomatitis is endemic, many species of vertebrate wild animals have serological evidence of infection with the virus; however, signs of disease are not reported. Although it is not known if Australian native fauna and insects are susceptible to infection with the virus, clinical disease was considered unlikely. In view of this, the direct impact on the environment was considered unlikely to be discernible at any level. This resulted in a rating of ‘A’ for this criterion.

The indirect impact of vesicular stomatitis

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

As discussed for outbreak scenario 3, AUSVETPLAN recommends stamping out, however, in this scenario the disease is widespread, and as such stamping out is unlikely to be effective. If insect vectors are believed to be involved and the virus is present in the wildlife population, AUSVETPLAN recommends that slaughtering be used sparingly. In some instances, for example if vesicular stomatitis occurs in a valuable horse racing or breeding establishment eradication by stamping out may not be a suitable control strategy. Quarantine and movement controls would be implemented in the control and restricted areas. An area of at least 100 km around the infected premises is recommended. Restrictions on movements of vehicles and

equipment would also apply. Milk from infected animals must be pasteurised and slaughtered clinically infected animals cannot be used for human consumption, but may be rendered into meatmeal. Equipment and infected premises would need to be decontaminated.

Extensive tracing and surveillance would be required, together with ongoing surveillance particularly if the disease cannot be eradicated to assist with zoning. Vaccination may be an option if the disease cannot be eradicated.

There would need to be publicity campaigns to inform people handling infected animals that the virus can cause disease in humans.

Taking these factors into account, it was considered that the indirect impact of new or modified control programs was of minor significance at the national level. This resulted in a rating of 'E' for this criterion.

Domestic trade or industry effects

As with outbreak scenario 3, there would be restrictions on movements of animals, holding of horse races and other equestrian events. Industries supporting the pig, cattle and horse industries such as stockfeed manufacturers', veterinarians and farriers could also be affected. Additional labour would be involved on infected premises caring for sick animals. One study conducted in the United States of America in 1995 estimated that an average of 833 hours of additional labour was required to care for vesicular stomatitis infected beef cattle (Hayek, et al., 1998).

Taking these issues into account, it was considered that the indirect impact of vesicular stomatitis on domestic trade and industry was considered of minor significance at the national level. This gave the disease a rating of 'E' for this criterion.

International trade effects

The indirect impact on international trade was considered to be similar to that described for outbreak scenario 3, although there may be greater disruption to Australia's live cattle exports and possibly sheep exports. If the disease could not be eradicated, the OIE Code recommends that for the importation of animals from countries considered infected with vesicular stomatitis, the animals should be held in quarantine and protected from insects for 21 days and be serologically negative and healthy at the time of shipment. No requirement for meat or other animal products are specified. Quarantine and protection from insects of feeder cattle prior to export may not be feasible in Australia. We would need to adopt zoning to assist in the international marketing of these animals. Given this, it was considered that the indirect impact of vesicular stomatitis on international trade was of minor significance nationally. This gave the disease a rating of 'E' for this criterion.

Indirect impact on the environment

Vesicular stomatitis is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

A widespread outbreak of vesicular stomatitis involving horses with disruption of horse events would have social consequences for the many thousands of people involved in horse riding. Moreover, horse racing contributes significantly to government revenue.

Where domestic pigs, cattle or sheep were important to the local economy, aspects of the rural community may be threatened. Given this, the indirect impact of vesicular stomatitis on rural communities was considered unlikely to be discernible nationally and of minor significance at the State level. This resulted in a rating of 'D' for this criterion.

The overall impact of vesicular stomatitis

When the direct and indirect impacts of vesicular stomatitis were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible (feral pigs, backyard pigs, other susceptible species), low (small commercial pigs)

Scenario 2: Consequences negligible (feral pigs, backyard pigs, other susceptible species), low (small commercial pigs)

Scenario 3: Consequences low

Scenario 4: Consequences moderate

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 16, Table 17, Table 18, and Table 19. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were considered 'very low', 'very low' and 'low' respectively. The likely consequences associated with the exposure of other susceptible species to infected pig meat scraps were considered 'very low'.

Table 16 Vesicular stomatitis: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	High	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Very low	Low	Negligible
Scenario 4	Very low	Moderate	Very low
		Overall likely consequences	**Very low**

Table 17 Vesicular stomatitis: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	High	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Very low	Low	Negligible
Scenario 4	Very low	Moderate	Very low
		Overall likely consequences	**Very low**

Table 18 Vesicular stomatitis: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	High	Low	Low
Scenario 2	Very low	Low	Negligible
Scenario 3	Very low	Low	Negligible
Scenario 4	Very low	Moderate	Very low
		Overall likely consequences	**Low**

Table 19 Vesicular stomatitis: summary of the consequences of exposure of other susceptible species

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	High	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Very low	Low	Negligible
Scenario 4	Very low	Moderate	Very low
		Overall likely consequences	**Very low**

Human life or health

Separate to the above is consideration of the consequences to human life or health. Vesicular stomatitis is a mild zoonosis (Hugh-Jones, et al. 1995). Clinical cases have been reported for laboratory workers and persons who have had contact with infected animals. It appears that

vesicular stomatitis virus produces a febrile “flu-like’ illness in adults. A biphasic fever accompanied by malaise, myalgia, headache and chills develops in the majority of cases. In a few cases vesicles on the tongue, buccal and pharyngeal mucosa, lips and nose have been reported. Transmission can occur by direct inoculation, virus contact with skin wounds, and possibly by inhalation of infectious aerosols(Reif, et al., 1987). Antibodies to vesicular stomatitis virus have been detected in humans without disease (Johnson, et al., 1969).

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the ‘partial annual risk’ associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of ‘overall annual risk’.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of ‘likely consequences’ for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with vesicular stomatitis virus.

Table 20 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly less than Australia’s ALOP (very low), risk management would not be required for vesicular stomatitis virus.

Table 20 Vesicular stomatitis: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Very low	Extremely low	Very low	Negligible
Backyard pigs	Very low	Extremely low	Very low	Negligible
Small commercial piggeries	Very low	Extremely low	Low	Negligible
Other susceptible species	Very low	Negligible	Very low	Negligible
			Overall annual risk	**Negligible**

The Department of Health and Ageing has advised Biosecurity Australia that biosecurity measures for vesicular stomatitis virus would not be required to manage the risk to human life or health associated with the importation of pig meat.

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African swine fever virus

Technical information

Background

African swine fever virus is the cause of African swine fever (ASF), a highly contagious, systemic haemorrhagic disease of pigs. African swine fever is an OIE List A disease. The disease is present in most of sub-Sahara Africa with high prevalence in a zone between the equator in the north and South Africa in the south. In 1957, ASF spread to Portugal, probably via meat products from Angola (Hess, 1981). Further outbreaks of ASF occurred in Europe and several years ago in Portugal in 1999. An endemic focus of ASF remains in Sardinia, Italy. Outbreaks have also occurred previously in Cuba, Brazil, the Dominican Republic and Haiti. Madagascar experienced ASF outbreaks for the first time in 1997. Except for Sardinia, ASF has been eradicated from all countries outside Africa.

Agent taxonomy

African swine fever virus is a DNA virus, being the sole member of the family Asfarviridae. Although there is only a single serotype recognised, numerous strains of varying virulence have been identified using nucleic acid detection techniques (Dixon, et al., 2000).

Agent properties

The virus is unique among the DNA viruses in that it behaves as a true arbovirus, having the ability to multiply in both vertebrate and invertebrate hosts. In pigs, ASF virus replicates in monocytes and macrophages (Wardley & Wilkinson, 1978).

African swine fever virus is stable over a wide pH range. In serum-free medium, ASF virus is inactivated at pH 3.9 or lower and at pH 11.5 or higher. Nonetheless, in a suitable serum medium, the virus has been shown to remain active at lower and higher pH values for a few hours to several days. In the presence of 25% serum, ASF virus has persisted for 7 days at pH 13.4. Thus ASF virus is relatively resistant to the pH changes that accompany *rigor mortis* (Plowright & Parker, 1967).

The virus is stable at low temperatures but is inactivated at high temperatures. African swine fever virus can survive freezing at -70°C indefinitely, in refrigerated blood for 6 years (De Kock, et al., 1940), in serum at room temperature for 18 months (Montgomery, 1921), and in blood at 37°C for a month (Neitz, 1963). Heating at 60°C for 30 minutes will inactivate the virus, whereas at 56°C ASF virus is not inactivated. In the absence of a protein medium, viability is reduced with storage at -20°C producing gradual inactivation.

Host range

Pigs are the only natural vertebrate hosts. Clinical disease occurs in domestic pigs (*Sus scrofa*) and in the European wild boar (*Sus scrofa ferus*). Subclinical infection occurs in warthogs (*Phacochoerus aethiopicus*), bushpigs (*Potamocherus porcus*) and giant forest hogs (*Hylocherus meinerzhageni*).

Epidemiology

African swine fever is a highly contagious disease in domestic pigs. It is transmitted by direct contact with infected pigs, by ingestion of products from infected pigs, or by the soft ticks, *Ornithodoros* spp. Within the soft ticks, ASF virus can be transmitted transstadially and transovarially. *O. moubata* contributes to the endemicity of ASF in eastern and southern Africa as had *O. erraticus* in the Iberian Peninsula before ASF was eradicated. *Ornithodoros* ticks collected from Haiti, the Dominican Republic and southern California have been shown to be capable vectors of ASF virus. The *Ornithodoros* ticks found in Australia, *O. gurneyi*, the inornate kangaroo tick, and *O. capensis*, the penguin tick, are not known to feed on pigs.

Ticks are the principal ectoparasites involved in transmission of ASF virus, however, experimentally, stable flies, *Stomoxys* species, were able to maintain or transmit the virus for 24 to 48 hours (Mellor, et al., 1987).

In Africa ASF virus is maintained either in a sylvatic cycle between ticks (*O. moubata*) living in warthog burrows and newborn warthogs or in a domestic cycle involving local pigs, with or without tick involvement. Outbreaks have occurred when domestic pigs come into contact with the sylvatic cycle, most likely with the *Ornithodoros* tick. Historically, ASF in Africa has been readily controlled by keeping pigs in pig-proof enclosures away from warthogs and their ticks and not feeding pigs infected materials.

In domestic pigs large amounts of virus are shed in all secretions and excretions during the acute stage of disease with the principal route of infection being the oral-nasal route. Aerial transmission does not appear to play a role in the spread of the disease. There is some circumstantial field evidence that pigs that survive ASF may become carriers, although their role in the spread of the disease is unclear. Recovered pigs do not appear to shed the virus 1 month after infection nor is the virus transmitted by their secretions and excretions (McVicar, 1984). Nonetheless infective levels of virus can be found in spleen, kidneys and bone marrow of recovered pigs about 85 days after an acute ASF infection and in lymph nodes, spleen and lungs about a year after infection (McVicar, 1984). Other tissues are unlikely to sustain detectable infective levels of ASF virus for more than two months after infection (Mebus, 1988).

The prevalence of ASF infected herds within a country have been determined by serological surveys. Between 1994 and 1996 in the province of Nuoro on the island of Sardinia 3.9% of pig farms tested positive to ASF virus. Prevalence ranged from 29.4% of free-range farms where disease control was difficult to 1.2% of farms where pigs were permanently confined and considered likely to have been fed pork scraps. None of the intensive piggeries in this province had seropositive pigs (Mannelli, et al., 1997). In Spain, between 1979 and 1981, 4.7% of farms surveyed were positive for ASF (Ordas, et al., 1983).

Several studies report the prevalence of infection or antibodies within a herd. In the survey reported above conducted in the province of Nuoro seroprevalence on ASF infected farms averaged 31% (range 12.5% to 66.7%) (Mannelli, et al., 1997). In an ASF outbreak in a pig herd in Spain, 60% of pigs became infected within 3 weeks of the first case of ASF (Bech-Nielsen, et al., 1995).

Clinical signs

The clinical signs of ASF can vary depending on the strain of the disease with peracute, acute, subacute and chronic forms occurring.

The incubation period varies between 5 and 15 days. In the peracute form sudden death with very few clinical signs is the main feature. Moribund pigs with high fever may also be seen shortly before death. Mortality can reach 100% with virulent strains. With less virulent strains, there is persistent or fluctuating fever to 42°C. One to two days after fever appears, affected animals have reduced appetite, are reluctant to move, huddle together and become recumbent. Affected pigs often have cyanotic blotching, especially on the extremities, mucopurulent ocular and nasal discharges, abdominal pain causing back arching, vomiting, either constipation or bloody diarrhoea, ataxia, and dyspnoea. Pregnant sows may abort. Nervous signs, including convulsions, may develop after several days. Clinical signs usually last 2 to 7 days with mortality reaching up to 100%. Survivors have poor body condition with respiratory distress and moist coughing due to the interstitial pneumonia, and painful and swollen joints. Death may follow after a period of weeks or even months, usually due to secondary bacterial infections, otherwise they recover or progress to the chronic form of disease characterised by stunting, emaciation, dull coat and sometimes lameness and ulcers on extremities.

A low virulent form of ASF has evolved characterised by fever and malaise and low mortality of around 5% (Mebus, et al., 1978).

Pathogenesis

The virus invades through the tonsils and respiratory tract, or by direct inoculation from feeding ticks, and replicates in draining lymph nodes. Replication and the onset of viraemia generally occur within 48 to 72 hours of infection (Hamdy & Dardiri, 1984). Infected pigs become thrombocytopenic over the following 48-hour period. Immune mediated thrombocytopenia and associated coagulation defects lead to the development of haemorrhages, serous exudates, infarcts and tissue oedema (Edwards, et al., 1985). In addition, the virus causes serious lymphopenia as a result of the widespread destruction of lymphocytes, and has a significant effect on members of the mononuclear macrophage system (Gomez-Villamandos, et al., 1997).

Pathology

Where sudden death due to ASF has occurred, the carcass itself is usually in good condition. The most outstanding feature is haemorrhages throughout the internal organs. Common gross lesions are swollen and haemorrhagic lymph nodes, especially those associated with the gastrointestinal tract and the head, which usually persist until death in subacute and chronic cases, capsular petechiation of the kidneys, ecchymoses of the cardiac surfaces and the gastric and intestinal serosa, and pulmonary oedema with hydrothorax (Rodriguez, et al., 1996). Renal haemorrhages, splenomegaly and oedematous gall bladder may sometimes be evident. Lesions seen with chronic cases include pericarditis, interstitial pneumonia, lymphadenitis and severe submucosal congestion of the colon with occasional button ulcers of the large intestines. Pigs that have recovered from infection with the low virulent strain have no lesions suggestive of ASF (Mebus & Dardiri, 1979).

Immunology

Pigs develop antibodies detectable by serological tests between 7 and 12 days post-infection which can persist in recovered pigs for long periods after infection, sometimes for life. However, there is an absence of virus-neutralising antibodies and there is no effective vaccine available against ASF. Chronically infected pigs often develop hypergammaglobinaemia (Pan, et al., 1970). Recovered pigs are usually resistant to reinfection with the homologous strain, but

not heterologous strains, and repeated reinoculation with the homologous strain increases protection against related strains.

Transmission via meat

International spread of ASF has been primarily through pork products in waste fed to pigs. Experimentally high virus titres have been detected in fat, muscle and bone marrow from acutely infected pigs. Five days after infection virus titres were $10^{5.4}$, $10^{6.6}$ and $10^{9.5}$ HAd_{50} (50% haemadsorbing units) per gram of tissue in fat, muscle and bone marrow respectively (Mebus, et al., 1993).

In studies of the survivability of ASF virus in chilled or frozen pork the virus persisted in muscle tissue for 104 days at 4°C and at -4° C, and in bone marrow for 188 days at -4°C (Kovalenko, et al., 1967; Botija, 1982). ASF virus was inactivated in hams by retort cooking to an internal temperature of 69°C (McKercher, et al., 1980).

ASF virus was not detected in cured meat products (smoked salami and pepperoni sausages) after 30 days of curing (McKercher, et al., 1978). While traditional curing periods exceed 30 days, processing pepperoni can be completed by around 22 days after slaughter. Parma hams were negative on culture for ASF virus at 300 days (Italian study) and 399 days (US study). In the US study, the loss of infectivity occurred between 291 and 399 days. As the parma cured ham goes through at least 365 days of maturing, the authors concluded that ASF virus is inactivated by this commercial curing process (McKercher, et al., 1978). Serrano hams, Iberian hams and shoulder hams were demonstrated to be free of viable ASF virus by day 140 of curing, all within the standardised serrano and Iberian curing periods. Iberian loin hams, which have a commercial curing period of 90 to 130 days, were found to be free of ASF virus by day 112 of curing (Mebus, et al., 1993). Finally it has been shown that ASF virus can be inactivated in infected muscle tissue using 20 kilograys of ionising gamma radiation (McVicar, et al., 1982).

Although pigs can be infected through the oral route, infectivity appears to depend on virus contact with the tonsils and upper respiratory tract epithelium. In one study it was demonstrated that pigs could be infected by placing ASF virus infected material, such as faeces and urine in their mouth but were unable to be infected when the infected material was placed in hollowed out sweet potatoes or bananas prior to feeding (Montgomery, 1921).

The oral infectious dose is unknown, however, experimentally at least $10^{4.3}$ HAd_{50} by the oronasal route is necessary for infection whereas a dose as small as 0.13 HAd_{50} can be infectious to pigs if given intravenously or intramuscularly.

Release assessment

R1 — the likelihood that a source herd is infected

Serological surveys in areas where low virulence ASF is or was endemic show an average of 3% to 5% of farms to be infected with ASF. Infection ranged from 0% on farms with intensive pig production to 30% on farms with free-range pigs. Severe epizootics, usually occurring every 10 to 12 years in parts of Africa, can result in over 30% of farms infected. Given the above, it was considered that, in ASF endemic areas, the likelihood of selecting slaughter-age pigs from an infected herd was ‘low’.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

ASF is highly contagious where pigs are kept in close contact and 60% of pigs can become infected within three weeks of the first case of ASF. Irrespective of the virulence of ASF virus, morbidity can reach 100% in previously unexposed herds. In ASF endemic areas, morbidity averages 30%. Given this, it was considered that the likelihood of selecting an infected animal in an infected herd was 'moderate'.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

Ante-mortem and post-mortem inspections involve observing pigs for clinical signs of ill-health before slaughter and gross pathological changes during the slaughter and dressing processes.

Clinical signs vary with the virulence of ASF virus and may be present less than a day in virulent cases when sudden death occurs or, in subacute cases, as long as 3 to 4 weeks. Affected animals have a fever, are reluctant to move, may huddle together, and cyanotic skin blotching can occur. Recovered carrier pigs may not show clinical signs.

Few pathological changes may be present in peracute cases, lesions may be sparse with few haemorrhagic spots seen; however, such pigs are usually found dead. The main feature of acute ASF is haemorrhages throughout the internal organs. Where systemic gastro-intestinal tract inflammation and/or lymphadenitis are observed in a carcass during post-mortem inspection, the Australian standard requires that the carcass be condemned as unfit for human consumption. Pigs that have recovered from infection due to low virulent strains and become carriers usually have no gross lesions.

On the basis of this information, the sensitivity of ante-mortem, slaughter and processing requirements in detecting and removing pigs infected with ASF is considered to be 'moderate'.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with ASF virus and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a 'one minus the background rejection rate'. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered 'extremely low'. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

High titres of ASF virus have been detected in fat, muscle and bone marrow of carcasses of recently infected pigs as well as in organs and blood removed during the carcass dressing

procedure. Hence, the likelihood that ASF virus will be present in meat harvested for export was ‘high’.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

ASF virus is stable over a wide range of pH values, from pH 3.9 to 11.5 and thus it was considered that there was a ‘high’ likelihood that meat infected at the time of slaughter would remain so after carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

ASF virus is stable at low temperatures, and is still infectious after 15 weeks in chilled or frozen meat. Given this, it was considered that there was a ‘high’ likelihood that chilling or freezing of infected carcasses would not destroy ASF virus.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a ‘very low’ likelihood that imported pig meat derived from a carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a ‘very low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

There are limited data on the oral infectious dose of ASF virus. Nonetheless swill feeding of infected meat is frequently linked to outbreaks of disease. High virus titres have been detected in muscle, bone marrow and fat of acutely infected pigs. Experimentally, infection by the oronasal route required at least $10^{4.3}$ $HAd_{50;}$ whereas virus titre in muscle has been reported to be as high as $10^{6.6}$ HAd_{50} per gram.

Given the above, it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent was ‘high’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

The likelihood that a pathogenic agent would remain viable after exposure to the environment will depend on the inherent ‘stability’ of each agent. In particular, this likelihood will reflect the agent’s sensitivity to ultraviolet light, to ambient temperatures between approximately 10°C and 35°C and to the putrefying effects of saprophytic organisms. It has been demonstrated that

ASF virus is stable at low temperatures, in serum for 18 months at room temperature and in blood at 37°C for 1 month.

Taking the above factors into consideration, the likelihood that ASF virus would survive within meat scraps discarded in refuse for the period of time required for pigs to locate and subsequently scavenge the material was considered 'high'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = High
- Rural regions = High
- Large towns = Very low

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be 'high'.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage. Nonetheless as ASF virus is a relatively stable virus, the period of time between discarding scraps and ingestion by either feral pigs or domestic pigs is unlikely to have a significant effect on virus viability.

Overall the Panel considered that there was a 'high' likelihood that ASF virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'low'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage. Nonetheless as ASF virus is a relatively stable virus, the period of time between discarding scraps and ingestion by either feral pigs or domestic pigs is unlikely to have significant effect on virus viability.

Overall the Panel considered that there was a 'high' likelihood that ASF virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'high'.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the three exposure groups - that is, feral pigs, pigs in backyard enterprises and pigs in small commercial piggeries. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps and in accordance with the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;

- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As ASF is highly contagious, there is potential for spread and establishment with the dissemination rate dependent on pig population dynamics. Feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and at mating, and some populations are contiguous. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs, however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

Feral pigs are extensively hunted in Australia, particularly at night, and normally gutted in the field where it may be difficult to observe mild lesions if pigs were infected with a low virulent strain. Nonetheless lesions are marked when pigs are infected with highly virulent strains. High mortality and morbidity would be expected with virulent strains of ASF virus.

Ornithodorus ticks in Africa are involved in the transmission of ASF and act as reservoirs. It is unknown if *Ornithodorus* ticks present in Australia are capable vectors of ASF virus but other species of this family of ticks in North and South America have been shown to be capable of transmitting ASF virus. Nonetheless it would appear that *Ornithodorus* ticks present in Australia do not feed on pigs.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: high

Scenario 3: low

Scenario 4: very low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

Veterinary attention for sick or dying pigs in backyard enterprises may be rarely sought, due to the costs involved. Backyard pigs are subject to a diverse range of feeding and management practices and are usually kept where biosecurity is poor. They are likely to be kept in simply constructed pens or allowed to roam in purpose-fenced paddocks or both. Iatrogenic spread of ASF virus by farmers has been reported (Bironț, et al., 1987). As most backyard pigs are kept on small hobby farm sized holdings, where neighbours are likely to share equipment, iatrogenic spread may occur. Spread of ASF virus by fomites is also likely as infected pigs shed virus in all secretions. Faeces is the most likely environmental contaminant.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: low

Scenario 3: moderate

Scenario 4: low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;

3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic;
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

Due to the high mortality associated with virulent strains of ASF virus, and greater awareness of managers of small commercial piggeries of animal health issues it was considered that there was a higher likelihood of the disease being diagnosed at an early stage. Even low virulent strains would result in increased mortality and pigs with fever and malaise. Moreover veterinarians are more likely to be called to investigate cases of mortality and sick pigs in a small commercial piggery than for backyard enterprises. In Spain an outbreak of ASF was confirmed 16 days after the first pig became sick, although the initial tentative diagnosis was swine erysipelas, highlighting the possibility of misdiagnosis with diseases endemic in Australia. Thirty-nine of sixty-one pigs became sick before the herd in Spain was slaughtered, but none died from the disease itself and the disease did not spread to other local pig herds. A feature of this outbreak was the absence of vector ticks in the herd (Bech-Nielsen, et al., 1995).

Movement of pigs from small commercial piggeries are considered to be more common than movement of pigs from backyard enterprises. Movement of contaminated materials by trucks and people could rapidly spread the disease

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: very low

Scenario 3: moderate

Scenario 4: low

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this scenario, ASF would have established in the directly exposed animal, or group of animals, but would not have spread to other animals. In the case of a feral pig herd or backyard pig enterprise being infected, this 'no outbreak' scenario would have resulted from low probability of contact between infected and susceptible animals, rather than from human intervention. Indeed, because the disease may be of low pathogenicity for pigs, it was assumed that it would not have been identified in these exposure groups. In the case of a small commercial piggery, due to the closer observation, it was assumed that the disease would have been identified and contained due to implementation of a control and eradication program.

The direct impact of African swine fever

Animal life or health

African swine fever is a highly contagious disease where virulent strains of the virus can cause up to 100% mortality, especially when the disease is spread by vector ticks. Even infection with low virulent strains can result in increased mortality, ill thrift, fever, pneumonia and abortion.

Given that under this scenario the disease only affects a single feral pig herd, a backyard enterprise or a small commercial piggery, the likely impact of ASF on animal health and welfare was unlikely to be discernible at any other level except the local level. This resulted in a rating of 'B' for this criterion.

Environment

African swine fever is not known to affect native Australian species. Although it is unknown if *Ornithodoros* ticks within Australia are capable as acting as reservoir there is no evidence that the virus affects the tick vector. Hence, it was considered that the direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of African swine fever

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

This criterion was not rated identically for each exposure group.

It is likely that, if the disease was contained within one herd of feral pigs or a single backyard enterprise, ASF would not be diagnosed within either of these herds. However, if the primary outbreak involved a small commercial piggery it is considered likely that pigs showing clinical signs of ASF would be investigated.

Diagnosis of ASF in Australia would trigger an emergency animal disease response under the Australian Emergency Response Plan[38]. Australian policy, as defined in AUSVETPLAN (Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 1996), is to eradicate ASF in the shortest possible period, while limiting economic impact, using a combination of strategies including stamping out (slaughter and disposal of destroyed

[38] http://www.aahc.com.au/eadp/response.htm

animals), quarantine and movement controls, decontamination, tracing and surveillance, zoning and a public awareness campaign.

African swine fever is listed as Category 3 under the Emergency Animal Disease Response Agreement. In this agreement the government and livestock industries have agreed to share costs based on the conduct of an agreed response plan for an outbreak of a disease that falls in one of four categories. Category 3 diseases are funded 50% by governments and 50% by the relevant industry.

Taking the above factors into account it was considered that the indirect impact of eradication programs was unlikely to be discernible at any level when the direct exposure group was a feral pig herd or a backyard pig enterprise. This resulted in a rating of 'A' for this criterion. Whereas when the direct exposure group was a small commercial piggery it was considered that the indirect impact of new eradication programs was unlikely to be discernible at the national or State level, and of minor significance at the district or regional level. Thus, a rating of 'C' was assigned to this criterion.

Domestic trade or industry effects

This criterion was not rated identically for each exposure group.

As discussed above, it was considered unlikely that ASF would be diagnosed in either a single feral pig herd or one backyard enterprise. On this basis, the indirect impact of ASF on domestic trade and industry was considered unlikely to be discernible at any level for these two exposure groups, and thus the rating assigned to this criterion was 'A'.

In the case of ASF being diagnosed in a small commercial piggery, under AUSVETPLAN all animals would be slaughtered and disposed of together with contaminated animal products. There would be quarantine and movement controls on animals and animal products in restricted and control areas surrounding the infected premises. In this scenario, the disease would be eradicated promptly. After consideration of these issues, the indirect impact of ASF on domestic trade and industry when the direct exposure group was a small commercial piggery was considered unlikely to be discernible at any level, except at the local level, and a rating of 'B' was assigned to this criterion.

International trade effects

This criterion was not rated identically for each exposure group.

As the disease is unlikely to be diagnosed in a single feral pig herd or backyard enterprise the indirect effects of ASF on international trade for these exposure groups was unlikely to be discernible at any level, and a rating of 'A' was therefore assigned to this criterion.

The diagnosis of ASF in Australia would lead to bans on import of Australian pig meat (farmed and feral), pig meat products, live pigs and pig semen into most countries until Australia had regained freedom. In this scenario, the disease would be promptly eradicated, however, the OIE Code states that a country shall be considered free if the disease has not been present for 12 months after a stamping-out policy is practised. In this period of time Australia would likely have lost market share to Singapore and Japan, our major export markets for pig meat. Export markets for pig meat are worth approximately $230 million per year. On this basis the likely impact of ASF on international trade, when the direct exposure group was a small commercial piggery, was considered to be of minor importance at the national level. This gave the disease a rating of 'E' for this criterion.

Indirect impact on the environment

In this scenario, ASF is unlikely to lead to any discernible indirect impacts on the environment such as affecting biodiversity, or from the disposal of carcasses from a single premises and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, ASF would have established in a broader population of feral pigs. The disease would be contained through the diagnosis of disease in feral pigs and the mounting of an eradication program.

The direct impact of African swine fever

Animal life or health

Although in this scenario, the disease spreads to a general population of feral pigs, it was considered that the direct impact on animal health was unlikely to differ to that described for scenario 1, and hence a rating of 'B' was assigned to this criterion.

Environment

African swine fever is not known to affect native Australian species. Although it is unknown if *Ornithodoros* ticks within Australia are capable as acting as reservoir there is no evidence that the virus affects the tick vector. Hence, it was considered that the direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of African swine fever

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

Due to the clinical signs of ASF it is considered likely that the disease would be diagnosed if it spread to a general population of feral pigs. Following the diagnosis, an emergency animal disease response aimed at eradicating the disease would be activated under AUSVETPLAN (see above) there would need to be extensive surveillance of both the feral pig population and the domestic pig population to determine the extent of the disease.

Eradication of ASF in the feral pig population would be difficult unless the disease was localised. Disposal of feral pigs may not be possible in some cases due to difficult terrain and the method of eradication such as aerial shooting or baiting. This would influence the type of control and eradication measures required as it would be important to ensure that feral pigs did not have access to infected carcasses. It has been estimated that it would cost between $4 and $65 per feral pig if eradication was undertaken (Hassall and Associates, 1993). Eradication of the disease in the feral pig population could be a lengthy process.

Some piggeries would need to improve their biosecurity to ensure that feral pigs could not gain access.

Overall the indirect impact of eradication and control programs was considered unlikely to be discernible at the national level, and of minor importance at the State level. This gave the disease a rating of 'D' for this criterion.

Domestic trade or industry effects

With secondary spread of ASF virus to feral pigs there may be some restrictions applied to local producers regarding movement of pigs in the those localities where infected feral pigs were detected. Controls would need to be implemented for hunters of feral pigs such that wastes from pig carcasses were disposed of appropriately. It was considered that the indirect impact on domestic trade and industry would be unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

International trade effects

As described above diagnosis of ASF in domestic or a more generalised population of feral pigs would lead to import bans on Australian pig meat (farmed and feral), pig meat products, live pigs and genetic material. The OIE Code requires that domestic and feral pigs are free from ASF for a period of 12 months after stamping-out prior to a country being considered free. The indirect effect of ASF on international trade is as described above for scenario 1, and a rating of 'E' was assigned for this criterion.

Indirect impact on the environment

Environmental issues would need to be taken into consideration if disposal of large numbers of slaughtered feral pigs was required. Nevertheless eradication of large numbers of feral pigs could benefit the environment in terms of rehabilitation of vegetation and native animal species. Overall it was considered that the indirect impact of ASF on the environment was unlikely to be discernible except at the local level, and a rating of 'B' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, ASF would have established in a local population of backyard piggeries or small commercial piggeries. The disease would be contained through the diagnosis of disease in pigs, and the mounting of an eradication program.

The direct impact of African swine fever

Animal life or health

The third scenario is characterised by spread of ASF to a local population of domestic pigs, but containment within this population. High morbidity and mortality may occur in herds infected with ASF. Overall, it was considered that the direct impact of ASF on animal health was

unlikely to be discernible at the national or State level, and of minor significance at the district or regional level. Overall this resulted in a rating of 'C' for this criterion.

Environment

African swine fever is not known to affect native Australian species. Although it is unknown if *Ornithodoros* ticks within Australia are capable as acting as reservoir there is no evidence that the virus affects the tick vector. Hence, it was considered that the direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of African swine fever

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

The response to a diagnosis of ASF would be the same for the third scenario as that described above for scenario 2. The indirect impact of eradication and control programs was considered unlikely to be discernible at the national level and minor at the State level, which would be responsible for its delivery. This gave the disease a rating of 'D' for this criterion.

Domestic trade or industry effects

With the disease spreading to a local population of small commercial piggeries or backyard enterprises, pigs in restricted and control areas surrounding the infected premises could only move direct to slaughter, subject to permit. This could result in welfare issues associated with over-stocking and increased feed costs for those affected piggeries. There would be loss of income for producers whose herds are destroyed and those subjected to quarantine controls and, possibly, detrimental effects on the health and welfare of those producers and their families.

Taking these factors into consideration, the indirect effect on domestic trade and industry was considered to be unlikely to be discernible at the national level and of minor impact at the State level. This resulted in a rating of 'D' for this criterion.

International trade effects

Were the third scenario to occur, the indirect effect on international trade would be as described above for scenario 2, of minor significance at the national level. This gave the disease a rating of 'E' for this criterion.

Indirect effects on the environment

The disposal of pigs by burial or cremation can present environmental problems. However in this scenario the disease has spread only to a local population of backyard enterprises or small commercial piggeries. Hence the numbers slaughtered would not be great and it was considered unlikely to lead to any discernible indirect impact on the environment other than at the local level. Thus, a rating of 'B' was assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, ASF would have established in a broader population of commercial piggeries (including medium-large piggeries). An eradication program would have been mounted in response to the diagnosis of the disease in pigs.

The direct impact of African swine fever

Animal life or health

An outbreak of ASF to a more general population of domestic pigs including to medium and large commercial piggeries would cause significant production losses to those piggeries affected. Even if the strain was not highly virulent there would be increased mortality, and such things as ill thrift, fever, pneumonia and abortion. Overall the direct effect on animal health was considered of minor importance at the national level. This resulted in a rating of 'E' for this criterion.

Environment

African swine fever is not known to affect native Australian species. Although it is unknown if *Ornithodoros* ticks within Australia are capable as acting as reservoir there is no evidence that the virus affects the tick vector. Hence, it was considered that the direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of African swine fever

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

The response to a diagnosis of ASF in the fourth scenario would be similar to that described for scenarios 2 and 3 but more extensive and prolonged.

In view of this, the indirect impact of eradication and control programs was considered of minor significance at the national level. This gave the disease a rating of 'E' for this criterion.

Domestic trade or industry effects

With a widespread outbreak involving a general population of domestic pigs there would be loss of income for producers in the affected areas, increased feed costs and welfare issues where stock were subject to movement restrictions. Some interstate trading restrictions may apply on meat until tracing and surveillance was completed. These restrictions could disrupt the marketing system. Unemployment could result at the farm and processor levels. The transport industry could also be disrupted. There would be the loss of genetics if breeding animals were involved.

There would be loss of income for producers whose herds are destroyed and those subjected to quarantine controls and, possibly, detrimental effects on the health and welfare of those producers and their families.

The public would need to be reassured that they were not at risk from ASF by consuming pork and pig meat products. The destruction of large numbers of pigs may have a detrimental impact on the public reaction to pig meat and pig meat products.

Taking these issues into account, the indirect impact of ASF on domestic trade and industry was considered of minor importance at the national level. Overall this resulted in a rating of 'E' for this criterion.

International trade effects

Were the fourth scenario to occur the indirect effect on international trade would be as described above for scenarios 2 and 3, of minor significance nationally. This gave the disease a rating of 'E' for this criterion.

Indirect impact on the environment

The disposal of pigs by burial or cremation can present environmental problems, particularly in this scenario where large numbers of pigs would need to be disposed. In view of this, the indirect impacts on the environment were considered unlikely to be discernible at national and State level, but of importance for the affected districts and regions. Thus, a rating of 'C' was assigned to this criterion.

Indirect impact on communities

One of the considerations with this criterion was the indirect impact of ASF on rural and regional economic viability. The pig industry is important to the economies of several localities and districts in New South Wales, Victoria , Queensland, Western Australia and South Australia. It has been estimated that in general terms, for every one employee working in the pig industry, there will be another two people employed in providing goods and services to the pig industry directly or to the employees in the pig industry (Alliance Consulting and Management, 2000).

As discussed previously, associated industries such as processors and the transport industry may also be affected. Where the pig industry was highly significant to the local economy, aspects of these communities may be threatened.

When these issues were collated, the indirect impact of ASF on rural communities was considered unlikely to be discernible at the national or State level, but of importance to affected districts or regions. Overall this resulted in a rating of 'C' for this criterion.

The overall impact of African swine fever

When the direct and indirect impacts of ASF were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible (feral pigs, backyard pigs); moderate (small commercial piggeries)

Scenario 2: Consequences moderate

Scenario 3: Consequences moderate

Scenario 4: Consequences moderate

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 21, Table 22, and Table 23. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were considered 'moderate', for each exposure group.

Table 21 ASF: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	High	Moderate	Moderate
Scenario 3	Low	Moderate	Low
Scenario 4	Very low	Moderate	Very low
		Overall likely consequences	**Moderate**

Table 22 ASF: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Low	Moderate	Low
Scenario 3	Moderate	Moderate	Moderate
Scenario 4	Low	Moderate	Low
		Overall likely consequences	**Moderate**

Table 23 ASF: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Moderate	Moderate
Scenario 2	Very low	Moderate	Very low
Scenario 3	Moderate	Moderate	Moderate
Scenario 4	Low	Moderate	Low
		Overall likely consequences	**Moderate**

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with ASF virus.

Table 24 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly higher than Australia's ALOP (very low), risk management would be required for ASF virus.

Table 24 ASF: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Very low	High	Moderate	Moderate
Backyard pigs	Very low	Low	Moderate	Low
Small commercial piggeries	Very low	High	Moderate	Moderate
			Overall annual risk	**Moderate**

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Classical swine fever virus

Technical information

Background

Classical swine fever (CSF) has been eradicated from much of western Europe (where vaccination is no longer permitted), although sporadic outbreaks in wild boar and domestic pigs still occur. Persistent foci of infection remain in Germany, France and some eastern European countries (Laddomada, 2000). Classical swine fever is also present on the island of Sardinia, Italy, in East and Central Africa, the Indian subcontinent, China, East and South-East Asia, regions of Mexico and most other countries in Central America, and throughout most of South America. Recent outbreaks of CSF in domestic pigs in the European Union occurred in France in 2002, Luxembourg in 2002, Germany in 2001 and 2002, Spain in 2001 and 2002. There were outbreaks in the Netherlands in 1997 and in England in 2000, both of which were eradicated. Classical swine fever is not present in Australia.

Agent taxonomy

Classical swine fever, also called “hog cholera” or “swine fever”, is a highly contagious and generalised viral disease caused by a porcine pestivirus of the family *Flaviviridae*. There are three groups of CSF virus identified, with three or four subgroups in each group (Paton, et al., 2000). Group identification of the virus in new outbreaks can help to determine the source of infection (Biagetti, et al., 2001). Classical swine fever is an OIE List A disease.

Agent properties

There is a single serotype of CSF virus although strains vary considerably in virulence and antigenicity.

The thermal stability of CSF virus is partly dependent on the medium containing the virus. Classical swine fever virus in cell culture fluid is inactivated when raised to 60°C for 10 minutes whereas in defibrinated blood the virus is not inactivated after 30 minutes at 68°C. Nonetheless as a general rule the higher the temperature, the quicker CSF virus is inactivated. CSF virus can survive 3 days at 50°C and 7 days at 37°C (Farez & Morley, 1997). There is no change in virus titre when kept at 4°C, -30°C or -80°C for 180 days (Harkness, 1985). The virus is relatively stable within a pH range of 4 to 10 (Depner, et al., 1992). Above and below these pH values, infectivity is relatively rapidly destroyed. These authors showed that, at neutral pH and at a temperature of 21°C, CSF virus had an average half-life of 50 hours. The virus is also rapidly inactivated by ultraviolet light (Kubin, 1967 as cited by (Edwards, 2000)) although may survive for some time in manure.

Host range

Domestic pigs and wild boar are the only natural hosts for CSF virus.

Epidemiology

Direct contact between infected and susceptible pigs is the most important means of transmission of CSF virus. The disease commonly spreads within a herd or population by the movement of viraemic animals. This is especially common for strains of low virulence in pigs

with congenital infection (Dahle & Liess, 1992). These piglets can shed virus for months without showing signs of disease. Viral shedding can also occur before the onset of clinical signs of disease and during the acute stage of disease. Pigs that survive acute and sub-acute infection develop antibodies and no longer shed virus. However pigs that develop a chronic infection may excrete the virus continuously or intermittently until death, a period that can be several months.

The most important means of natural transmission appears to be via oral and nasal secretions, although the virus is present in lacrimal secretions, urine and faeces (Ressang, 1973). More recently it has been demonstrated that CSF virus can be spread via semen (de Smit, et al., 1999; Hennecken, et al., 2000). It has been suggested that insect vectors may transmit CSF virus by contact with eyes or open wounds (Reuss, 1959 as cited by (Dahle, et al., 1992)). In areas of high pig density, the indirect transmission of CSF virus between herds by veterinarians and their equipment, farmers, vehicles, and infected clothing has been an important means of spread (Dahle & Liess, 1992; Radostits, et al. 1994). Airborne transmission does not seem to play a role in the spread of disease from farm to farm.

Pork and pork products are also important in the transmission of CSF and several reviews cite the feeding of infected meat scraps as a cause of outbreaks (particularly the first outbreak) in several countries (Helwig & Keast, 1966; Timoney, et al. 1988; Laude, et al., 1993; Radostits, et al. 1994; Geering, et al. 1995; Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 1996).

Collection of accurate data on the prevalence of CSF within a country is often complicated by control and eradication programs being undertaken. Vaccination, often used as a control measure, makes the interpretation of seroprevalence difficult. It is known that from 1993 to 1997, 455 outbreaks of CSF were reported in Cuba, causing the death or slaughter of more than 70,000 pigs from a population of 414,000 (17%). In Mexico, in the State of Jalisco, during May to November 1998, 35 of 691 herds (5.1%) experienced an outbreak of CSF (Martens, 2003). In 1996 or 1997 there were 5,879 cases of CSF reported in the Philippines where there is a pig population of 7.5 million and 15,313 cases in Indonesia with a pig population of 7.8 million. However, the total number of animals affected or number of herds is not provided (Edwards, 2000). A serological survey for CSF was conducted in four districts in Lao People's Democratic Republic where vaccination was rarely practiced. In these districts the prevalence of antibodies was 12% (Khounsy, et al., 2000).

Within the European Union (EU), where eradication programs are in place, from 1996 to 2001, each year between 5 and 600 herds of pigs (average 122) experienced outbreaks of CSF each year, with most of these herds having more than nine pigs. In 1997 there were 337,000 pig farms recorded in the EU with more than nine pigs (European Commission, 2001). As such, less than 0.2% of herds within the EU have had an outbreak of CSF annually.

Within the wild boar population of the EU there can be significant year-to-year variation in the incidence and prevalence of CSF in endemic areas. A serological survey of French free and farmed wild boars from 1991 to 1998 showed 80 of 12,025 sera were positive for CSF, 66 of which came from the endemic area in eastern France (Albina, et al., 2000). The pattern of CSF in Sardinia is interesting in that the disease does not show a tendency to spread far from the core endemic areas, despite the high density of the wild boar population all over the island and the lack of major geographical barriers. Results of a serological survey for CSF antibodies in wild boar shot in Sardinia over a 3 year period from 1988 to 1992 demonstrated an overall prevalence of 11% (Laddomada, et al., 1994).

Pigs on farms in the Philippines with enzootic CSF generally have a significant percentage of animals with positive titres (Geerts, et al., 1995). Even in vaccinated populations up to 30% of young pigs in the Philippines showed clinical signs of CSF within the next 10 months. The prevalence of CSF within a pig herd in Vietnam was reported to be 35% (Bui Quang Anh , et al., 2000). In the 1998 outbreak in Mexico, the within herd prevalence of clinical disease in four unvaccinated herds varied between 14% and 49.6%, with an average of 37.9% (Martens, 2003).

Clinical signs

Clinical signs of CSF can vary markedly from high mortality and morbidity to very mild disease (Van Oirschot & Terpstra, 1989). In the acute or subacute form of the disease clinical signs appear after a short incubation period of 2 to 6 days. Initially there may be fever, dullness, reluctance to move and reduced appetite. If disturbed and made to stand, some pigs will have arched backs and others may appear chilled. Conjunctivitis often develops early in the course of the disease. Other signs include constipation followed by diarrhoea or vomiting, purplish abdominal skin, necrotic tips of ears, tail and vulva, incoordination, tremors, convulsions and/or circling. The mortality rate from acute CSF is 95 to 100% and most pigs die between 10 to 20 days post-infection. In subacute CSF, pigs show less severe signs of disease and succumb within 30 days.

However, some infected pigs survive longer than 30 days and these infections are termed persistent. Persistence of CSF can give rise to different clinical forms of the disease. Pigs that overcome the initial infection can develop chronic CSF where after initial clinical improvement they relapse and die. These pigs are often retarded in growth, have skin lesions and arched backs. These pigs can survive for several months but eventually die.

There is also a late-onset chronic form of CSF as a sequel of congenital CSF virus infection. These pigs remain relatively healthy for a longer period (several months) after infection. Clinical signs consist of mild anorexia, conjunctivitis, dermatitis, diarrhoea, runting and locomotion disturbances. These pigs are persistently infected and often survive for more than 6 months, but eventually die.

Persistent, subclinical infection can also develop if pigs are infected postnatally with low virulent strains of CSF virus.

Classical swine fever virus can cause stillbirth or mummification of foetuses when sows are infected during early pregnancy, or healthy-looking but infected piglets when sows are infected during mid or late pregnancy.

Pathogenesis

The tonsil is the primary site of virus invasion following oral exposure. Primary multiplication occurs in the tonsil within hours of invasion. The virus is subsequently transferred through lymphatics and capillaries, resulting in viraemia at approximately 24 hours post-infection (Cheville & Mengeling, 1969). At this time, CSF virus can be found in the spleen, peripheral lymph nodes, bone marrow and Peyer’s patches. The virus exerts its cytopathic effect on endothelial cells, lymphoreticular cells and macrophages, and epithelial cells. The generalised insult to the vascular system results in widespread congestion, arteriolar thrombosis, haemorrhages and infarction, with the most severe lesions found in the lymph nodes, spleen, kidneys and gastrointestinal tract. A leukopaenia is common in the early stages of the disease, followed by anaemia and thrombocytosis. In many cases secondary bacterial infection occurs and plays a role in the development of lesions and clinical signs (Cheville & Mengeling, 1969).

Pathology

In peracute cases of CSF, pathologic lesions are often absent. However the pathologic picture can be quite marked in acute and subacute CSF, being that of a septicaemic disease characterised by multiple haemorrhages of various sizes. Haemorrhagic lesions are most commonly found in the lymph nodes and kidneys. Petechial to ecchymotic haemorrhages also occur in the bladder, skin, heart, larynx, intestinal mucosa and serosa. Infarction of the spleen, which occur as dark blebs raised slightly above the surrounding surface, is almost pathognomic for acute CSF.

In persistent CSF haemorrhages and infarctions are generally less pronounced or absent. The most prominent lesions in persistent CSF are atrophy of the thymus and severe depletion of lymphocytes in tonsil, lymph nodes and spleen. Intestinal button ulcers, 1 to 2 cm diameter with necrotic centres, and rib lesions are also frequently associated with persistent CSF (Van Oirschot & Terpstra, 1989).

Immunology

Pigs that have recovered from CSF have antibodies to CSF virus and are immune against subsequent infection. Antibodies can also be produced during an acute or subacute fatal infection. Pigs with chronic CSF, which eventually die, are also capable of mounting an immune response, resulting in the simultaneous occurrence of virus and antibody in the blood. However, pigs with congenital persistent infection seldom produce a specific antibody response. Piglets born to seropositive sows obtain antibodies via colostrum. This passive immunity generally protects piglets against mortality for the first 5 weeks of life, but not against virus replication and shedding (Van Oirschot & Terpstra, 1989).

Transmission via meat

As discussed above infected pork and pork products are an important means of transmission of CSF virus. The titres of CSF virus in muscle were determined in the tissues of experimentally inoculated pigs slaughtered between 7 and 25 days after infection (Wood, et al., 1988). Pigs were orally infected with $10^{6.5}$ $TCID_{50}$/pig, with the titre of virus recovered from muscle and lymph nodes being $10^{3.4}$ and $10^{4.9}$ $TCID_{50}$/gram respectively.

Virus was also recovered from muscle and lymph nodes of pigs infected with CSF virus . In addition high titres of virus were isolated from bone marrow. In this study 64 pigs were inoculated intravenously with 1 ml of a 1:100 dilution of a stock virus having a titre of $10^{5.3}TCID_{50}$/ml and slaughtered 4 to 5 days later. The mean viral titres detected in muscle, lymph node and bone marrow were $10^{1.0}$, $10^{3.9}$, and $10^{1.0}$ plaque forming units (PFU)/g.

It has been demonstrated that the oral infectious dose of CSF virus is very low. An oral infectious dose less than 10 $TCID_{50}$ was able to cause fatal disease in weaner pigs (Dahle & Liess, 1992). It has been stated that only a few grams of infected tissue would be required to orally infect pigs .

Classical swine fever virus has a stability in carcass components similar to that of rinderpest virus, surviving in skin for 33 days and in muscle for 73 days, when stored at room temperature (Blackwell, 1984). Classical swine fever virus was inactivated by retort heating muscle, lymph node tissue and bone marrow to 65°C for 15 minutes. These results concur with those of others who found that CSF was inactivated by heating to 71°C for 1 minute (Stewart, et al., 1979). In studies of the inactivation of CSF in blood, the virus was inactivated in whole blood after

preheating at 60°C for 120 minutes, then heating to 68°C for 30 minutes, or preheating for 3 minutes then heating to 66°C for 60 minutes. When blood was defibrinated, heating for 30 minutes at 69°C inactivated CSF virus (Torrey & Prather, 1963).

With regard to cured hams, uncooked ham remained infective for between 34 and 85 days, while cooked ham did not contain active virus. The virus was destroyed when the centre of the cooked ham product was maintained at 65°C for at least 30 minutes (Helwig & Keast, 1966). Likewise, another study found that hams from CSF-infected pigs were no longer infective after being heated to 69°C (McKercher, et al., 1978). In a joint US-Italian project, hams produced using the 'Prosciutto de Parma' process were negative on culture for CSF virus at 189 days (Italian study) and 313 days for the US study (samples were not tested between 189 and 312 days) (McKercher, et al., 1987). In other salted/dried products, CSF virus survived for 70 days in ham bone marrow and 90 days in ham muscle and fat (original paper in French - results as cited by (Mebus, et al., 1993)). Alternatively, Iberian hams were shown to be free of CSF virus after 252 days of curing (curing time 365-730 days), Iberian shoulder hams after 140 days (curing time 240 to 420 days), Iberian loins by 126 days (curing time 90 to 130 days) and white serrano hams by 140 days (curing time 180 to 365 days) (Mebus, et al., 1993). These authors recognised the differences in inactivation times for CSF virus between meat products cured by different processes and stated that the protective efficacy of each process should be considered.

Concerning sausage products, sausage casings held at 39°C and salted according to one commercial procedure remained infective for up to 86 days . Casings salted using another (commercial) procedure remained infective for 17 days. In another early study, CSF virus could be inactivated by heating 29-31mm 'Bratwurst' to 80°C to 82°C for 10 minutes, by smoking 22-33mm 'Vienna' at 80°C for 45 minutes and scalding at 80°C for 8 minutes, and by smoking 59-62mm 'Lyonerwurst' at 82°C to 85°C for 50 minutes and scalding at 81°C to 82°C for 45 minutes (Leresche, 1956, as cited by (Torrey & Prather, 1963)). Finally, pepperoni and Italian salamis prepared according to traditional protocols and from CSF-infected tissues contained viable virus 22 and 21 days after slaughter respectively (McKercher, et al., 1978).

Release assessment

R1 — the likelihood that a source herd is infected

The prevalence of CSF within endemic countries is difficult to ascertain. However it is known that in wild boar populations the prevalence can be as high as 11%. Within Cuba a prevalence of 17% was reported and 12% in Lao People's Democratic Republic. In Mexico, the between herd prevalence was reported as 5.1%. Based on this information, it was considered that in CSF endemic areas the likelihood of selecting slaughter-age pigs from an infected herd was 'low'.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

Classical swine fever is a highly contagious disease. The rate of spread of virus within a herd appears to depend on the virulence of the strain. Pigs infected with virulent CSF virus shed high quantities of virus during the entire disease period, whereas infections with low virulent strains are characterised by short periods of virus multiplication and excretion. Hence virulent CSF virus will generally spread faster in an infected herd and induce a higher morbidity than low virulent strains (Van Oirschot & Terpstra, 1989). In CSF endemic areas, within herd prevalence of disease has been reported as 30% where vaccination was practised. In Mexico, in herds that were unvaccinated the prevalence of clinical disease averaged 37.9%.

In an endemic situation it is likely that pigs will be infected shortly after weaning, however, with congenital infections pigs can be persistently infected such that slaughter-age pigs could be viraemic and not be serologically positive.

Given the above, it was considered that the likelihood of selecting an infected animal in an infected herd was 'moderate'.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

The clinical signs of CSF were outlined above. While these vary with the stage of infection and the virulence of the strain, those pigs that are showing clinical signs of acute, subacute or chronic infection would not pass ante-mortem inspection. However slaughter-age pigs may also be subclinically and persistently infected either with low virulent strains or as a sequel of congenital infection. These pigs would pass ante-mortem inspection.

The pathological picture is quite marked in acute and subacute CSF. Nonetheless the macroscopic lesions associated with subclinically infected animals are generally less pronounced or absent. There may be atrophy of the thymus, intestinal button ulcers and rib lesions. This may result in the condemnation of the thymus and/or intestines but not the whole carcass.

On the basis of this information, the sensitivity of the ante-mortem, slaughter and processing requirements in detecting and removing pigs infected with CSF was considered to be 'moderate'.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with CSF virus and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a 'one minus the background rejection rate'. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered 'extremely low'. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

High titres of CSF virus have been detected in muscle and lymph nodes of recently infected pigs. Since pigs slaughtered and processed in accordance with Australia's requirements for ante-mortem inspection are extremely unlikely to be showing clinical signs at the time of slaughter, the presence of CSF virus in meat will be dependent on subclinical persistent viraemia. Persistent CSF infections are known to occur as a result of either congenital infections or low virulent strains.

This evidence suggested that the likelihood that CSF virus would be present in meat harvested for export was ‘high’.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

Classical swine fever virus is stable between pH 4 and 10 and, thus, it was considered that there was a ‘high’ likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

Classical swine fever virus is stable at low temperatures, and is known to be stable for extended periods when stored at 4°C, or when frozen. In view of this, it was considered that there was a ‘high’ likelihood that meat infected at the completion of carcass maturation would remain infected during transport and cold storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a ‘very low’ likelihood that imported pig meat derived from an individual carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a ‘very low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

It has been demonstrated that the oral infectious dose of CSF virus is very low. An oral dose as little as 10 $TCID_{50}$ can cause fatal disease in pigs. Virus has been detected in muscle, lymph node and bone marrow at titres generally exceeding the oral infectious dose. It has been concluded that only a few grams of infected tissue would be required to orally infect pigs. It is also known that meat has been frequently implicated in the spread of the disease to free countries or regions.

Taking the above factors into consideration, the likelihood that a waste unit from an infected pig would contain a sufficient dose of CSF virus to initiate infection was considered ‘high’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

This step describes the sensitivity of CSF virus to ultraviolet light, to ambient temperatures ranging from 10ºC to 35ºC and to the putrefying effects of saprophytic organisms. It is known that CSF virus can survive for 3 days at 50ºC and 7 days at 37ºC. It can be assumed that at lower temperatures, the virus will persist for a longer period. The virus is susceptible to ultraviolet light but may be partially protected if present in bone marrow or meat wastes covered by other refuse.

This information led the Panel to consider that the likelihood that CSF virus would survive within meat scraps discarded in refuse for the period of time required for feral pigs to locate and subsequently scavenge the material was 'high'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of the annual likelihood for each region was explained in Table 5.

- Remote regions = High
- Rural regions = High
- Large towns = Very low

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be 'high'.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of CSF virus to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be discarded due to spoilage. Nevertheless as CSF virus is a relatively stable virus, the period of time between discarding scraps and ingestion by either feral pigs or backyard pigs is unlikely to have a significant effect on agent viability.

Overall the Panel considered that there was a 'high' likelihood that CSF virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'low'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of CSF virus to initiate infection was ‘high’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be discarded due to spoilage. However, due to the relative stability of CSF virus there is unlikely to be a significant difference in the viability of the virus with different exposure groups.

Overall the Panel considered that there was a ‘high’ likelihood that CSF virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be ‘high’.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete ‘outbreak scenarios’ for each of the three exposure groups - that is, feral pigs, pigs in backyard enterprises and pigs in small commercial piggeries. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;

- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Classical swine fever is a contagious disease in which infected pigs are viraemic at least as long as clinical signs persist. In the case of chronic infection this can be several months. Moreover congenital infections can result in persistently infected pigs. These factors would assist in the spread of the disease. Nonetheless feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs; however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves. It is also known that CSF spreads from wild or feral pigs to domestic pigs, as has been the case in Europe.

Classical swine fever virus could also be spread indirectly to domestic pigs from contaminated clothing and equipment belonging to farmers who are hunters as well.

Depending on the strain on CSF virus clinical signs can be very mild, although virulent strains would cause high mortality and morbidity. It is considered likely that the disease could be present for a period of time prior to be recognised increasing the likelihood of its spread.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: very low

Scenario 2: moderate

Scenario 3: low

Scenario 4: moderate

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

As discussed above it is feasible that backyard pigs may under some circumstances come in contact with nocturnal foraging feral pigs. It is also feasible that there may be some mixing of backyard pigs between herds. Pigs may be transferred between holdings, for example, as part of a barter system, or for growing out or fattening. In addition there may be movement of pigs for breeding purposes in the case of small breeders for example with rare breeds of pigs. Pig meat products may also be distributed between backyard operators, which could spread the disease if illegal swill feeding is practised.

There may also be indirect spread of CSF by fomites to other piggeries. Classical swine fever virus in urine and faeces could contaminate clothing, boots, vehicles and equipment.

Owners of backyard pigs are less likely than commercial operators to seek veterinary attention for sick pigs due to the costs involved. In addition the clinical signs of CSF may be mild such that there could be a delay in diagnosing the disease. Even within commercial piggeries recognition of CSF has been delayed. It is estimated that CSF was present in the Netherlands in 1997 at least 5 weeks prior to being diagnosed (Elbers, et al., 1999).

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: moderate

Scenario 4: low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

High mortality and morbidity resulting from infection with virulent strains of CSF are more likely to be investigated in a small commercial piggery than backyard enterprises. Nonetheless, depending on the clinical picture, it may be quite some time before the disease is diagnosed. As stated above, it was at least 5 weeks before the disease was diagnosed in the Netherlands. The last outbreak of CSF in Australia in 1961, caused by a strain of low virulence, only came to official attention as result of higher than normal condemnation rate for 'septicaemia' of pig carcasses in abattoirs, and increased mortality rates in poorly run piggeries with a high prevalence of secondary bacterial infections (Geering, et al. 1995).

Although most pigs from a small commercial piggery will go directly to slaughter, it is known that CSF virus can be spread indirectly via contaminated trucks, personnel and equipment. A small number of pigs may also be purchased as stores for other piggeries.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: moderate

Scenario 4: moderate

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes ‘no further establishment or spread’. The remaining three outbreak scenarios describe ‘secondary spread to other pigs, humans or to other susceptible species’. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this scenario, CSF would have established in the directly exposed animal, or group of animals, but would not have spread to other pig herds. This ‘no outbreak’ scenario would have resulted from low probability of contact between infected and susceptible animals, rather than from human intervention. Indeed, because clinical signs of the disease can be mild, it was assumed that it would not have been identified.

The direct impact of classical swine fever

Animal life or health

Classical swine fever is a highly contagious disease where virulent strains can cause high mortality and morbidity. Pigs that develop persistent infection may show such signs as runting, diarrhoea and depression. Pregnant sows that survive infection may later abort, or produce mummified, stillborn and/or weak piglets. Nonetheless there are low virulent strains of CSF where the clinical signs of disease can be very mild or not apparent.

On this basis, and for this scenario the likely impact of CSF on animal health was considered unlikely to be discernible at any level except the local level. This resulted in a rating of ‘B’ for this criterion.

Environment

Because CSF is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of ‘A’ for this criterion.

The indirect impact of classical swine fever

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

Under this scenario, it is likely that the disease would not be diagnosed, particularly if the strain was not highly virulent as the disease is only present within a single herd. As discussed above it was at least 5 weeks before the CSF outbreak in the Netherlands was diagnosed and it was considered that it could be a period of time before the disease was diagnosed in Australia. Given this, the overall indirect impact of eradication and control programs was considered unlikely to be discernible at any level, and thus the rating assigned to this criterion was ‘A’.

Domestic trade or industry effects

As discussed above, it was considered unlikely that the disease would be diagnosed in a single feral pig herd, a backyard enterprise or a small commercial piggery. As such the indirect impact of CSF on domestic trade and industry was considered unlikely to be discernible at any level, and a rating of 'A' was therefore assigned to this criterion.

International trade effects

As the disease is not likely to be diagnosed in any single directly exposed herd the indirect effect of CSF on international trade was considered unlikely to be discernible at any level, and a rating of 'A' was assigned to this criterion.

Indirect impact on the environment

In this scenario, CSF is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, CSF would have established in a broader population of feral pigs. It is unlikely that the CSF would be diagnosed in feral pigs as often the clinical signs of disease are mild. Containment of the disease in feral pigs would result from the low probability of contact between infected and susceptible animals, rather than by human intervention.

The direct impact of classical swine fever

Animal life or health

Under this scenario, the disease spreads to a general population of feral pigs but not to other domestic pigs. Depending on the strain of CSF virus clinical signs may be very mild although a virulent strain of CSF virus may result in high mortality within a feral pig population. Overall the direct impact on animal health is unlikely to differ to that of the direct exposure group and hence a rating of 'B' was assigned to this criterion.

Environment

Because CSF is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of classical swine fever

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

It is unlikely that CSF would be diagnosed in a general population of feral pigs. Pigs hunted are generally older animals that, on post-mortem inspection for export purposes, would likely not have marked pathological changes. Piglet mortality associated with CSF infection in feral pigs may go unnoticed or attributed to other causes including drought. Experience overseas and in Australia in the 1960s demonstrated that even in commercial pigs the disease may not be detected for some time. It is feasible that the disease could exist in the feral pig population and may only be diagnosed if there was spill-over into the domestic pig population. Surveillance for CSF within the feral pig population is undertaken in the northern parts of Australia, from Broome to Cairns; however, there are large populations of feral pigs elsewhere in Australia. Hence the indirect impact of CSF on eradication and control programs was considered unlikely to be discernible at any level, and thus the rating assigned to this criterion was 'A'.

Domestic trade or industry effects

As discussed above it was considered that CSF was unlikely to be diagnosed in a more general population of feral pigs. On this basis the indirect impact of CSF on domestic trade and industry was considered unlikely to be discernible at any level, and thus the rating assigned to this criterion was 'A'.

International trade effects

As it was considered likely that CSF would not be diagnosed under this scenario the indirect impact on international trade is unlikely to be discernible at any level, and a rating of 'A' was assigned to this criterion.

Indirect impact on the environment

In this scenario, CSF is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, CSF would have established in a local population of backyard piggeries or small commercial piggeries. The disease would be contained through the diagnosis of disease in pigs, and the mounting of an eradication program.

The direct impact of classical swine fever

Animal life or health

This scenario is characterised by the spread of CSF to a local population of domestic pigs. Depending on the strain of CSF virus clinical signs of disease can vary from high mortality and morbidity to mild disease. Pregnant sows can abort. In the chronic form of the disease ill thrift is one of the symptoms. However, subclinical infection can also be a feature of CSF infection. Given this, the direct impact of CSF on animal health was considered unlikely to be discernible at the national or State level, and of minor significance at the district or regional level. Overall this resulted in a rating of 'C' for this criterion.

Environment

Because CSF is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of classical swine fever

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

Managers and owners of small commercial piggeries are more aware of exotic diseases and seek veterinary advice. It may be a period of time before the disease was diagnosed as the clinical signs presented may be confused with endemic diseases but disease investigations would likely occur and it was considered that were the third scenario to occur the disease would be diagnosed.

Diagnosis of CSF in Australia would trigger an emergency animal disease response under the Australian Emergency Response Plan (Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 1996). Australian policy, as defined in AUSVETPLAN, is to eradicate CSF in the shortest possible period, while limiting economic impact, using a combination of strategies including stamping out (slaughter and disposal of destroyed animals), quarantine and movement controls, decontamination, tracing and surveillance, zoning and a public awareness campaign. Vaccination is unlikely to be used, but may be approved in exceptional circumstances if stamping out is failing to control the spread of infection. If the outbreak was due to a low virulence strain causing negligible production loss, a modified policy might be applied.

Classical swine fever is listed as Category 3 under the Emergency Animal Disease Response Agreement[39]. In this agreement the government and livestock industries have agreed to share costs based on the conduct of an agreed response plan for an outbreak of disease that falls in one of four categories. Category 3 diseases are funded 50% by governments and 50% by the relevant industry.

To establish the extent of the disease surveillance would need to be undertaken. Depending on the location of the outbreak, feral pigs may also need to be tested. There may need to be additional surveillance of feral pigs to demonstrate freedom, in particular to the European Union regarding export of feral pig meat. Efforts would need to be made to minimise contact

[39] http://www.aahc.com.au/eadp/response.htm

between feral pigs and domestic pigs such as by fencing and by reducing the number of feral pigs in the locality.

After consideration of these issues, the indirect impact of control and eradication programs was considered unlikely to be discernible at the national level and minor at the State level, which would be responsible for its delivery. This gave the disease a rating of 'D' for this criterion.

Domestic trade or industry effects

Were the third scenario to occur, there is unlikely to be major disruption to domestic trade in meat or associated industries such as transport or stock feed manufacturers. There would be restrictions on movement of pigs and pig products in the area where the outbreaks occurred. Producers whose herd were destroyed and others whose herds were subject to movement restrictions would suffer loss of income. There would be the cost of replacing the breeding herd. There could be increased feed costs for those piggeries unable to freely market pigs. As the export market of pig meat would at least be temporarily disrupted extra volume may be redirected onto the local market. This could result in a reduction in domestic pork prices.

Overall it was considered that the indirect effect on domestic trade was unlikely to be discernible at the national level and of minor impact at State level. Hence the rating assigned to this criterion was 'D'.

International trade effects

An outbreak of CSF would lead to temporary bans on the import of Australian pigs, pig semen and pig meat (farmed and feral) into most countries. According to OIE guidelines should a CSF outbreak occur in an establishment of a free country or zone, the status of the country or zone may be restored at least 30 days after completion of stamping out. There is provision within the OIE guidelines for a country to be free from CSF within domestic pigs but with infection in wild pigs. Under this scenario, in which only a local outbreak occurs, the disease should be eradicated promptly. It has been estimated that an epidemic event involving spread from a backyard enterprise to commercial piggery and then to a number of farms within the surrounding area within either regions of the Darling Downs or Northern Victoria would last approximately 3 weeks (Garner, et al., 2001).

Australia does not export significant quantities of semen or live pigs. Australia's biggest market for live pigs is the Philippines where CSF is endemic, however, due to possible strain differences it is likely that all trade in live animals and genetic material would cease until the disease was eradicated.

Export markets for pig meat are valued at approximately $230 million per year. With prompt eradication of CSF, our major markets in Singapore and Japan may only be temporarily disrupted and market share may not be lost.

Taking these factors into consideration the likely indirect effect of CSF on international trade was considered of minor significance at the national level. This resulted in a rating of 'E' for this criterion.

Indirect impact on the environment

The disposal of pigs by burial or cremation can present environmental problems. However in this scenario the disease has spread only to a local population of backyard enterprises or small commercial piggeries. Hence the numbers slaughtered would not be great and it was considered

unlikely to lead to any discernible indirect impact on the environment other than at the local level. Thus, a rating of 'B' was assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, CSF would have established in a broader population of commercial piggeries (including medium-large piggeries). An eradication program would have been mounted in response to the diagnosis of the disease in pigs.

The direct impact of classical swine fever

Animal life or health

An outbreak of CSF to a more general population of domestic pigs may cause significant production losses although it is noted that the severity of clinical signs are dependent on the virulence of the strain. High mortalities can occur in young pigs. Abortion, stillbirths and mummified foetuses can also be a feature of the disease. Infected pigs that develop the chronic form of the disease often have stunted growth. Given this, the direct effect on animal health was considered of minor importance at the national level, and a rating of 'E' was assigned to this criterion.

Environment

Because CSF is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of classical swine fever

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

As discussed above in scenario three, diagnosis of CSF in Australia would trigger an emergency animal disease response under AUSVETPLAN aimed at eradication in the shortest possible time.. The cost of such a program to Governments and industry would be significant where the disease has spread to a more general population of domestic pigs. The cost of carcass disposal and piggery decontamination for a small outbreak involving commercial piggeries was estimated as $13.8 million for those affected in the Darling Downs and $22.5 million for those affected in Northern Victoria (Garner, et al., 2001). Separate costs were not provided for surveillance; these could be extensive, involving both domestic and feral pig populations.

The cost of compensation, destruction of animals and decontamination following the severe outbreak (500 infected premises) in the Netherlands in 1997 was estimated at 400 million Euros (Saatkamp, et al., 2000). Previously in the United Kingdom approximately £12.3 million was spent between 1963 and 1966 to eradicate CSF and maintain a surveillance program

(Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 1996). During the outbreak of CSF in the United Kingdom in 2000, where 16 premises were infected, approximately £4.4 million compensation was paid to pig farmers for the slaughter of potentially infected pigs or dangerous contacts (approximately 75,000 pigs). In addition another £13 million was paid to assist producers meet welfare obligations with nearly 190,000 pigs slaughtered (Department for Environment Food and Rural Affairs (UK), 2001).

If an outbreak was widespread or the disease became endemic, vaccination may be considered as part of the control program. The advantages of vaccination would need to be weighed up in relation to ongoing export trade restrictions.

Taking these issues into consideration, the indirect impact of an eradication and control program was of minor importance at the national level. Overall this resulted in a rating of 'E' for this criterion.

Domestic trade or industry effects

The restrictions imposed on the movement of pigs and pig products with a more generalised outbreak may cause disruption to the local marketing of pig meat. Some interstate trading restrictions may apply on meat until tracing and surveillance was completed. Initially, if there was a shortage of product there could be a price increase in pork. However, as export of pig meat would cease, meat would be redirected to the local market. Overall this is likely to cause a reduction in pork prices. Consumers may also decrease consumption of pork following an outbreak and a publicity campaign would likely need to be conducted to reassure the public that there were no health concerns. There would loss of genetics if breeding herds were involved in the outbreak.

Associated industries such as abattoirs, processors, transport, and stock feed manufacturers would also be affected if the outbreak was prolonged. Unemployment may result.

There are likely to be increased feed costs and welfare concerns for those producers whose premises are not infected but which are subject to movement restrictions.

On-farm costs associated with an epidemic of CSF in either the Darling Downs or Northern Victoria was estimated at $2 million and $3 million respectively excluding compensation and piggery decontamination (Garner, et al., 2001). Further costs of $2 million and $3.8 million would be incurred associated with movement restrictions for herds in restricted and control areas for the Darling Downs and Northern Victoria respectively. Overall it was calculated that the gross income of the national pig industry would fall by 9% if there was an epidemic of CSF within these areas. This figure included lost production, cost of disposal and price effects. Should CSF become established (endemic) the above authors calculated that losses to the national pig industry would be approximately 11% per year, based on productivity considerations alone.

Taking these factors into consideration, the indirect impact of CSF on domestic trade and industry was considered of minor importance at the national level. Overall this resulted in a rating of 'E' for this criterion.

International trade effects

If the disease were widespread then the process of eradication could be prolonged and it is likely that Australia would lose market share for pig meat exports to Singapore and other markets. Nonetheless although this would be of importance at a national level it was considered

that this would not be expected to threaten economic viability. Overall it was considered that the indirect effect on international trade would be as described for scenario 3, of minor significance nationally. This gave the disease a rating of 'E' for this criterion.

Indirect impact on the environment

An important consideration would be the environmental issues associated with the slaughter and disposal of large numbers of pigs associated with an outbreak involving several pig producing regions. The environmental issues would need to be addressed prior to disposal. In view of this, the indirect impacts on the environment were considered unlikely to be discernible at the national and State levels, but of importance for the affected districts or regions. This resulted in a rating of 'C' for this criterion.

Indirect impact on communities

One of the considerations with this criterion was the indirect impact of CSF on rural and regional economic viability. The pig industry is important to the economies of several localities and districts in New South Wales, Victoria, Queensland, Western Australia and South Australia. It has been estimated that in general terms, for every one employee working in the pig industry, there will be another two people employed in providing goods and services to the pig industry directly or to the employees in the pig industry (Alliance Consulting and Management, 2000).

As discussed above associated industries such as processors, the transport industry and stockfeed manufacturers may also be affected. Where the pig industry was highly significant to the local economy, aspects of these communities may be threatened.

When these issues were collated, the indirect impact of CSF on rural communities was considered unlikely to be discernible at the national or State level but of importance to affected districts or regions. Overall this resulted in a rating of 'C' for this criterion.

The overall impact of classical swine fever

When the direct and indirect impacts of CSF were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences negligible

Scenario 3: Consequences moderate

Scenario 4: Consequences moderate

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 25, Table 26, and Table 27. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were each considered 'moderate'.

Table 25 CSF: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Very low	Negligible	Negligible
Scenario 2	Moderate	Negligible	Negligible
Scenario 3	Low	Moderate	Low
Scenario 4	Moderate	Moderate	Moderate
		Overall likely consequences	**Moderate**

Table 26 CSF: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Moderate	Moderate	Moderate
Scenario 4	Low	Moderate	Low
		Overall likely consequences	**Moderate**

Table 27 CSF: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Moderate	Moderate	Moderate
Scenario 4	Moderate	Moderate	Moderate
		Overall likely consequences	**Moderate**

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with classical swine fever virus.

Table 28 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly higher than Australia's ALOP (very low), risk management would be required for CSF virus.

Table 28 CSF: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Very low	High	Moderate	Moderate
Backyard pigs	Very low	Low	Moderate	Low
Small commercial piggeries	Very low	High	Moderate	Moderate
			Overall annual risk	**Moderate**

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Rinderpest virus

Technical information

Background

Rinderpest is an acute viral disease, principally of cattle and buffaloes, but which can also infect a number of non-domestic species (Anderson, et al. 1996). Clinically, the disease is characterised by inflammation and necrosis of mucous membranes, and a very high mortality rate in susceptible animals. Rinderpest is the legendary 'cattle plague' which historically caused devastating epidemics for centuries in Asia and Europe and, since the 1890s, in Africa. Following resurgence of the disease in the 1980s, regional control programs have reduced the distribution of the disease to a few isolated areas in Africa, the Middle East and South Asia. The Food and Agriculture Organization of the United Nations (FAO) has launched a Global Rinderpest Eradication Programme (GREP) calling for global eradication of the virus by the year 2010.

Agent taxonomy

Rinderpest virus is a member of the morbillivirus genus of the family Paramyxoviridae. Other viruses in this genus are those of measles, canine distemper, phocine (seal) distemper and peste des petits ruminants. These viruses have similar physicochemical properties and are serologically related. There is only one serotype of rinderpest, but strains vary in virulence.

Agent properties

Rinderpest virus is not very stable and probably does not survive in dried secretions, excretions or carcases for more than a few days (Geering, et al. 1995). It is relatively heat sensitive, being rapidly inactivated at 56°C. The OIE reports that small amounts of virus resist 56°C for 60 minutes or 60°C for 30 minutes (Office international des épizooties, 2002).

The virulent WZ-78 strain of rinderpest virus has been studied (Ata, 1982). At 56°C, infectivity of the virus disappeared in 50 minutes with a half-life of 2 minutes 50 seconds. It was not affected by eight cycles of freezing and thawing. The virus was inactivated by lipid solvents, trypsin and a pH of 3 (Ata, 1982).

Rinderpest virus is reported as being stable between pH 7.2 to 7.9, but is inactivated at pH values of less than 5.6 or greater than 9.6 (Geering, et al. 1995). In contrast, the OIE disease card for rinderpest indicates that it is stable between pH 4.0 and 10.0.

Host range

Rinderpest affects cattle, water buffalo and many species of wild animals including African buffalo, eland, kudu, wildebeest, various antelope, bushpig, warthog and giraffe (Office international des épizooties, 2002).

When the disease occurred in Asia, native breeds of pigs were quite susceptible, but European breeds resistant (Geering, et al. 1995). The Asian domestic sway-backed pig suffers from and succumbs to rinderpest, whilst European pigs experience inapparent infections when exposed experimentally (Anderson, et al. 1996).

Cattle and buffalo are highly susceptible and rinderpest is mostly seen in these species. *Bos indicus* breeds of cattle are generally less severely affected in Africa. The virus has also been reported as causing disease in sheep and goats in India, but the relative contribution made by rinderpest and peste des petits ruminants to the disease syndrome seen in those species was not always clear. Clinical rinderpest in sheep and goats is rare in Africa, although subclinical infection may occur in association with disease outbreaks in cattle.

Epidemiology

Most strains of rinderpest virus are reported to be highly contagious (Geering, et al. 1995), although some spread slowly. Infection is commonly transmitted by direct contact between animals by virus in aerosol droplets, but airborne spread up to 100 metres is possible. The virus is present in high concentrations in the expired air, tears, nasal discharges, saliva, faeces and urine of infected animals. Excretion of the virus may commence 1 to 2 days before onset of clinical signs and can continue for a maximum of 14 days. There is no chronic carrier state.

In experimentally infected pigs scrapings from tongue, gum and skin, saliva, nasal discharges and corneal smears were examined. The highest virus concentrations were found in nasal discharges and saliva 10 to 15 days after inoculation. All samples were negative by day 45 (Roy, et al., 1997).

Major epidemics, with rapid spread and high morbidity and mortality rates, occur when the virus is introduced into susceptible cattle populations. This almost invariably follows from the introduction of infected animals.

In endemic areas the disease tends to spread more slowly, affecting mainly younger animals, with flare-ups occurring at intervals of about 3 to 4 years. The nomadic movement of animals through communal watering places and markets is important in the maintenance of the disease in such situations.

Mild forms of rinderpest occur in the Horn of Africa (Rossiter, 1996). In parts of Kenya there has been confirmed rinderpest in wildlife that has not been seen in nearby cattle.

Animal products are not considered a source of natural infection. Indirect transmission has on rare occasions allegedly occurred through contaminated bedding, fodder or water, but is not considered important (Geering, et al. 1995). Anderson et al (1996) report that an analysis of valid records of virgin-soil epizootics from 1851 to 1950 clearly revealed that all instances were traceable to the importation of live animals.

Due to control programs in place for rinderpest virus in endemic areas the true prevalence of infection is difficult to ascertain. Prevalence is generally reported in a country or area rather than within or between herd. There is little information on prevalence of infection in pigs. In Egypt, pigs were surveyed for antibodies at slaughter following severe outbreaks in cattle and buffalo (Youssef, et al., 1991). Twenty-eight percent of these pigs (36 of 128) had neutralising antibodies. Similar results were obtained in a survey for antibodies in pig sera in Tamil Nadu and Andhra Pradesh in India where rinderpest was endemic and in which 33 out of 134 pigs (25%) at slaughter had antibodies specific to rinderpest (Krishnaswamy, et al., 1981). Also in India rinderpest antibodies were demonstrated in 37% of 2400 sheep and 29.5% of 1000 goats (Babu & Rajasekhar, 1988). In another study conducted in India, 46.2% of 723 sheep and goats sampled mostly at abattoirs were seropositive to a strain of rinderpest virus (Sudharshana, et al., 1995).

Clinical signs

Rinderpest disease in pigs, particularly in European breeds, may be clinically inapparent whereas Asian pigs may develop the typical clinical disease and suffer high mortality as described below for cattle (Anderson, et al. 1996). Nonetheless there are occasional reports of fatal rinderpest in European breeds of pigs (Govindarajan, et al., 1996).

Rinderpest in cattle and buffalo may be peracute, acute or subacute (Anderson, et al. 1996). The incubation period in susceptible animals is 2 to 6 days but may be as long as 15 days in native breeds of livestock that have some innate resistance.

In the acute disease there is sudden onset of high fever, which lasts 2 to 7 days, and which is often biphasic. Clinical signs appear a day or so after the onset of fever and include cessation of milk production, depression, restlessness, partial loss of appetite, nasal and ocular discharges (at first serous but later mucopurulent), rapid and shallow respiration, congested mucous membranes, dull coat, retarded rumination and constipation. Within another 1 to 2 days, lesions appear on the mucous membranes of the mouth, nostrils and urogenital tract. At first these lesions are raised necrotic pinpoints but they rapidly enlarge and coalesce. The necrotic tissue sloughs easily leaving irregular, well demarcated shallow erosions. At this stage, salivation is profuse, the animal is very obviously ill, its breath is foetid and its breathing laboured with characteristic grunting expiration. Lacrimal secretions become mucopurulent. Superficial lymph nodes may become markedly enlarged.

Diarrhoea starts 1 or 2 days after the appearance of mucosal lesions. The fluid faeces are profuse, dark, foetid and may contain mucus, blood and fragments of necrotic mucosa. There is frequent straining, exposing congested and eroded rectal mucosa. Dehydration follows rapidly, with collapse and death.

Most animals die 6 to 12 days after the onset of clinical signs. Some die as early as 2 days after a peracute illness. In such cases visible mucous membranes may be very congested but death usually supervenes before mucosal erosions develop. Other sick animals may linger for up to 3 weeks. Some animals recover, but convalescence is prolonged.

Subacute cases are seen in endemic areas, but could also occur in susceptible populations with the introduction of less virulent strains of virus. Varying combinations of the above symptoms may occur in a milder form. There may be a mild febrile reaction only with temporary anorexia, malaise and catarrhal inflammation of mucous membranes.

Pathogenesis

Rinderpest virus has a core affinity for lymphoid tissues and secondary affinity for the epithelium of the alimentary, upper respiratory and urogenital tracts (Anderson, et al. 1996). The latter tropism is well developed in highly contagious strains of the virus but is muted or absent in strains serially passaged by parenteral injection of suspensions of infected tissues. Most natural cases of rinderpest exhibit grossly more pronounced changes in epithelial linings than in lymphoid organs. Microscopic examination reveals the opposite. During disease the virus is also found in non-lymphoid organs such as the lungs, liver and kidneys (Rossiter, 1995).

The selective destruction of lymphocytes by rinderpest virus induces significant haematological changes and the severity of the changes appears to be linked to the virulence of the virus. A transient leucocytosis often precedes the onset of fever but subsequently there is a dramatic and

profound leucopenia. The lowest level, reached during the erosive-mucosa phase of the clinical reaction, is followed by a gradual return over several weeks to normal levels in survivors.

In surviving animals the erythrocyte count fluctuates within the normal range, but in fatal cases there is an apparent increase attributable to the effects of terminal dehydration. This terminal change is manifested also by a packed cell volume (PCV) reaching 40 to 65%. As a result, the loss of body water approaches 40% and the blood at death is dark, thick and slow to coagulate.

Pathology

The pathology in pigs, which has been described, is similar to that in cattle, with stomatitis, gastritis, lesions in the Peyer's patches of the small intestine and more prominent lesions in the caecum and colon (Anderson, et al. 1996). Lymphoid organs exhibit a variety of necrotic lesions that are particularly conspicuous in the gut-associated lymphoid tissues. The spleen is usually grossly normal although it may be swollen.

Immunology

Infected animals produce a high-titre antibody response against the mass of virus antigens in the lymphoid tissues (Anderson, et al. 1996). The response is essentially the same in all species of susceptible animals and in infections with virulent and avirulent (vaccine) strains of the virus. These antibodies are a major component of active immunity against infection and play an important role in recovery. Their appearance during disease corresponds closely with the disappearance of viraemia and virus antigen in the tissues.

Antibodies start to develop between 2 and 5 days after the onset of clinical disease in virulent infections and 6 to 10 days after infection with avirulent strains. The titres rise until death or 3 to 4 weeks after infection, at which stage the animal is usually well advanced into convalescence. The majority of animals will maintain high levels of humoral antibody, detectable by ELISA, throughout their lives.

Transmission via meat

The most common means of spread has been through movement of infected animals. There is little information available on the ability of rinderpest virus to survive in animal products. It has been mentioned that pigs may become infected through eating contaminated offal but animal products are not a common source of infection (Geering, et al. 1995). Experimentally pigs have been infected with rinderpest virus when fed infected bovine, rabbit and goat spleens (Scott, et al., 1962). No data were found on the minimum infective oral dose of rinderpest virus in pigs or other species.

The length of persistence of rinderpest virus in the meat of infected animals has been the subject of controversy. In chilled meat it is more than 9 days but certain observations indicate that it could reach 33 days or even 18 weeks (Drieux, 1975). Blackwell (1987) cites a report that rinderpest virus has survived for up to 6 weeks in carcases of experimentally infected cattle but the reference source contained no information to that effect. In contrast it has been stated that rinderpest infected carcases are rendered safe relatively quickly (Anderson, et al. 1996). It should be noted that beef may reach a lower pH than that of pork, so extrapolation may not be directly applicable.

Freezing of meat would appear to have negligible effect on the virus as trials have shown that it loses no infectivity during storage of 6 to 9 months between -25°C and - 70°C and it has

survived for up to 3 years in frozen spleen. The virus may survive in chilled spleen for up to 7 months and chilled blood for 3 months (Drieux, 1975).

In skins, rinderpest virus does not survive prolonged exposure to sunlight. It disappears after 24 hours salting and after 48 hours of drying in darkness. The virus does not survive the drying in horns and hooves (Drieux, 1975).

Release assessment

R1 — the likelihood that a source herd is infected

The prevalence of infection of pigs with rinderpest virus varies widely because of breed predilection. Rinderpest in indigenous pigs has been identified on several occasions in the South-Asian zone but the first report of infection of European breeds was only relatively recent (Govindarajan, et al., 1996). In countries where pigs are susceptible and the disease is endemic, prevalence of antibodies has been reported as ranging between 25% and 28% and in other species between 13.5% and 37%. Based on this information, it was considered that where rinderpest is endemic in the pig population, the likelihood of selecting slaughter-age pigs from an infected herd is ‘low’.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

Although good data were unavailable on within herd prevalence, it is known that morbidity is high within an infected herd. Adult animals that have previously been infected are likely to be immune, as are their sucking young, however, all other pigs would be susceptible to infection. In a country where the disease is present, animals are likely to become infected some time after weaning. Prolonged viraemia and excretion of virus is not a feature of the disease, with animals generally excreting virus for about 2 weeks.

Given this, it was considered that the likelihood of selecting an infected animal in an infected herd was ‘moderate’.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

Ante-mortem and post-mortem inspections involve observing pigs for clinical signs of ill-health before slaughter and gross pathological changes during the slaughter and dressing processes.

An acutely affected pig would show clinical signs which would be detected at ante-mortem inspection. However, a pig in the incubation or recovery phase or one that is more refractive to clinical disease (but not infection with the virus) would not be showing clinical signs. It should be noted, however, that no persistent carrier state exists for rinderpest. The same situation would also apply to post-mortem inspection at slaughter.

On the basis of this information, the sensitivity of ante-mortem, slaughter and processing requirements in detecting and removing pigs infected with rinderpest was considered to be 'extremely low'.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with rinderpest virus and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a 'one minus the background rejection rate'. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered 'extremely low'. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

Rinderpest virus has an affinity for lymphoid tissue and less so for the epithelium of the alimentary, upper respiratory and urogenital tracts. This suggests that virus present after dressing of the carcass is likely to be derived from lymphoid tissue, or from the blood perfusing the tissues rather than muscle tissue *per se*. It is not known if meat from infected animals can initiate infection if consumed by a naïve pig. In view of the above factors, and since a dressed carcass includes lymph nodes, it was considered that the likelihood that rinderpest virus would be present in meat harvested from an infected pig was 'moderate'.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

The pH range for survival of rinderpest virus is quoted as between 4 and 10 by the OIE and in another review as between 5.6 and 9.6 (Geering, et al. 1995). The sensitivity to pH varies with the strain and the half-life is reduced at the extremes of this range (Scott, 1967) eg below pH 5.6. For the purposes of this IRA, meat is not assumed to reach a pH lower than 6.2. Thus, it was considered that there was a 'high' likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

It has been stated that rinderpest virus can survive at least 9 days in chilled meat, but may be up to 18 weeks. The virus is stable for months when frozen. In view of this, it was considered that there was a 'high' likelihood that meat infected at the completion of carcass maturation would remain infected during transport and storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a 'very low' likelihood that imported pig meat derived from an individual carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

It has been stated (although details were not provided) that an infectious dose as low as one $TCID_{50}$ of rinderpest virus induces clinical rinderpest with 100% mortality in unvaccinated cattle (Yilma, et al., 1996). Experimentally, in one study, the protective effect of a vaccine was tested in cattle through inoculation with $10^{3.5}$ $TCID_{50}$ of a virulent strain of rinderpest virus (Samanta & Pandey, 1995). In other experiments involving intra-nasal inoculation of live virus vaccine strains, the dose required to stimulate a response was in the order of $10^{2.5}$ to $10^{3.0}$ $TCID_{50}$ (Anderson, et al., 2000; Murugan & Ramkrishna, 1996). However, the oral infectious dose of rinderpest virus is unknown, although it is known that transmission to pigs can occur via the feeding of infected spleen.

The amount of virus in the waste unit would also depend on the tissues of which the waste unit were comprised. If lymph node were included, the amount of virus present would be expected to be higher than muscle. Waste from the stifle, neck, or axillary areas would be more likely to contain lymphoid tissue than would waste from other parts of the carcass.

When these factors were combined, the likelihood that a waste unit from an infected pig would contain a sufficient dose of rinderpest virus to initiate infection was considered to be 'low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

Survival of rinderpest virus in non-refrigerated meat would appear to be of a short duration. This is particularly so in lymphoid tissue where the half life of rinderpest virus has been estimated as 6 hours at 25°C and 2 hours at 37°C. Sunlight is highly effective in inactivating the virus, the half-life being measured in seconds (Scott, 1967).

In light of this information, it was considered that the likelihood that rinderpest virus would survive within meat scraps discarded in refuse for the period of time required for feral pigs to locate and subsequently scavenge the material was 'very low'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = High
- Rural regions = Very low
- Large towns = Extremely low

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be ‘high’.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a ‘very low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of rinderpest virus to initiate infection was ‘low’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall the Panel considered that there was a 'low' likelihood that rinderpest virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'very low'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of rinderpest virus to initiate infection was 'low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall the Panel considered that there was a 'low' likelihood that rinderpest virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be ‘high’.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete ‘outbreak scenarios’ for each of the three exposure groups - that is, feral pigs, pigs in backyard enterprises and pigs in small commercial piggeries. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of ‘consequences’ for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the ‘no outbreak’ scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Rinderpest is a highly contagious disease of cattle and other Artiodactyla with most clinical cases occurring in cattle. Infection in European breeds of pigs is often subclinical. Feral pigs in Australia are of both Asian and European breeds and as such the disease could go unnoticed in some areas for a period of time until spread to cattle and other highly susceptible species occurred. Cattle rather than pigs are the principal animal involved in spread of the disease.

Nonetheless spread from infected pigs to in-contact pigs has been reported as has spread to cattle. In one experimental study the disease appeared to spread from infected pigs to pigs more readily than to cattle. Forty pigs and 40 cattle were exposed to rinderpest infected pigs of European origin, with virus recovered from 32.5% and 10% of the in-contact pigs and cattle respectively (Scott, et al., 1962).

Feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs, however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

As the virus is not very stable in the environment and a carrier status does not exist, it is feasible that the disease could die out if only a small population of feral pigs were infected.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: high

Scenario 2: low

Scenario 3: low

Scenario 4: extremely low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

As discussed above, it is feasible that backyard pigs kept in rural areas may come in close contact with nocturnally foraging feral pigs and that transmission of rinderpest virus from one group to the other may result. It is also feasible that some mixing between pigs from an infected backyard herd and other domestic pigs may occur. For example, in the case of speciality breeds or unusual breeds pigs or semen may be transferred from one herd to another for breeding purposes. Alternatively, pigs raised for personal consumption may be transferred between

backyard holdings for growing out or fattening. However, often backyard pigs will be raised for consumption by that household.

As rinderpest virus often results in subclinical infection in European breeds of pigs, it is feasible that the disease may not be recognised until there is spread to other susceptible species. However, it is likely that the disease would be rapidly diagnosed should this occur, thus preventing further spread.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: high

Scenario 4: extremely low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

As discussed above, European breeds of pigs infected with rinderpest virus often do not show clinical signs of disease. There have been occasional reports of high mortality in European breeds of pigs following infection. If this was the case managers of small commercial piggeries will detect the disease in an early stage, and the consulting veterinarian will be alerted to the potential of an exotic disease epidemic. However, if infection results in subclinical disease, as is generally the case, further spread is likely to other piggeries and other susceptible species

before the disease is diagnosed and eradicated. AUSVETPLAN states that it is highly likely that rinderpest virus would be quickly eradicated from Australia (Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 1996). Rinderpest has previously been introduced into Australia, in 1923, however, it was quickly eradicated.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: high

Scenario 4: extremely low

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this scenario, rinderpest would have established in the directly exposed animal, or group of animals, but would not have spread to other animals. This 'no outbreak' scenario would have resulted from low probability of contact between infected and susceptible animals, rather than from human intervention. Indeed, because the disease may be of low pathogenicity for European breeds of pigs, it was assumed that it would not have been identified and would not, under a 'no outbreak' scenario, have any discernible direct or indirect impacts.

On this basis, a rating of 'A' was assigned to each of the direct and indirect criteria.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, the disease is most likely to have established within a population of feral pigs, and to have run its course without identification. As explained above, this is because the disease may be subclinical in pigs. As such, under this scenario there would not be any discernible direct or indirect impacts.

On this basis, a rating of 'A' was assigned to each of the direct and indirect criteria.

Outbreak scenario 3 — secondary spread to a local population of backyard enterprises or small commercial piggeries - spread to other susceptible species

Under this scenario, rinderpest would have established in a local population of backyard piggeries or small commercial piggeries, and other susceptible species such as cattle. The disease would be contained through the diagnosis of disease in any of the affected species, but particularly cattle, and the mounting of an eradication program.

The direct impact of rinderpest

Animal life or health

The third scenario is characterised by spread of rinderpest virus to a local population of domestic pigs in backyard enterprises or small commercial piggeries and to other susceptible species such as cattle but containment within this population. Although in pigs clinical signs of disease may be inapparent, in cattle clinical signs of infection are marked, with high mortality. Hence the direct impact on animal health was, under this scenario, considered unlikely to be discernible at the national level, but of minor importance at the State level. Overall, this resulted in a rating of 'D' for this criterion.

Environment

Because Rinderpest virus is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of rinderpest

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

Under this scenario, it was considered that rinderpest would be diagnosed and a control, eradication and compensation program would be implemented immediately. Rinderpest is listed as Category 2 under the Emergency Animal Disease Response Agreement[40]. In this agreement the government and livestock industries have agreed to share costs based on the conduct of an agreed response plan for an outbreak of a disease that falls in one of four categories. Category 2 diseases are funded 80% by governments and 20% by the relevant industries. AUSVETPLAN recommends eradication by destruction of all infected and exposed susceptible animals on infected premises, movement controls and quarantine (Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 1996). Facilities, products and things like equipment would be decontaminated to eliminate the virus on infected premises and to prevent spread in declared areas. The policy is to eradicate rinderpest in the shortest possible period.

There would need to be tracing and surveillance including possibly the feral pig population to determine the source and extent of infection and provide proof of freedom from the disease.

Overall the indirect impacts of control and eradication programs were considered to be of minor significance at the national level. This resulted in a rating of 'E' for this criterion.

Domestic trade or industry effects

In the case of rinderpest being diagnosed all infected and exposed susceptible animals on infected premises would be slaughtered and disposed of together with contaminated animal products. There would be quarantine and movement controls on animals and animal products in restricted and control areas surrounding the infected premises. AUSVETPLAN states that milk from restricted areas may be permitted to be marketed subject to heat treatment for milk powder. Clinically free animals from non-infected premises in restricted and control areas may

[40] http:/www.aahc.com.au/eadp/response.htm

move direct to slaughter for local consumption subject to certain conditions. Crops and grains may be removed providing they are not fed immediately to livestock (Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 1996).

States not affected with rinderpest would likely close their borders to susceptible animals and products.

As export markets for meat would likely close, the extra volume of meat would be redirected onto the local market. This could result in a reduction in domestic red meat and pork prices. In addition, consumers may initially decrease consumption of these meats following a disease outbreak. Publicity campaigns may need to be undertaken to reassure the public that there was no risk from meat.

Given this, the impact on domestic trade or industry was considered to be of minor significance at the national level. This resulted in a rating of 'E' for this criterion.

International trade effects

The diagnosis of rinderpest would prohibit the export of meat, meat products, live cattle, sheep, goats, camels, pigs and their genetic material until Australia had regained freedom. In this scenario, the disease would be promptly eradicated, however, the OIE Code states that a country shall be considered free if the disease has not been present for 6 months after a stamping-out policy and serological surveillance is practiced. In this period of time Australia would likely have lost market share to Japan, USA, Korea, Canada and the EU, our major export markets for beef. The value of Australia's total annual beef exports is of the order of $4 billion and of live cattle exports $600 million. Pig meat exports are currently valued at approximately $230 million annually (2003). On this basis, the likely indirect impact of rinderpest on international trade was considered to be highly significant at the national level. This gave the disease a rating of 'G' for this criterion.

Indirect impact on the environment

An outbreak of rinderpest as described by this scenario is likely to have indirect environmental impacts resulting mainly from the disposal of animal carcasses. Additional impacts could arise from the widespread use of disinfectants to decontaminate infected properties.

Given this, the indirect impact on the environment was considered unlikely to be discernible at the national and State levels, and of minor importance at the district or regional level. This resulted in a rating of 'C' for this criterion.

Indirect impact on communities

An outbreak of rinderpest would affect the rural and regional economic viability including such things as businesses reliant on livestock revenue, employment, local governments together with social costs to individuals and communities.

Considering these factors, the indirect impact of rinderpest on rural communities was considered to be significant at the national level. Overall this resulted in a rating of 'F' for this criterion.

Outbreak scenario 4 — secondary spread to a more general population of domestic pigs - spread to other susceptible species

Under this scenario, rinderpest would have established in a broader population of commercial piggeries (including medium-large piggeries) and cattle. An eradication and control program would have been mounted in response to the diagnosis of the disease in the affected animals, particularly cattle.

The direct impact of rinderpest

Animal life or health

An outbreak of rinderpest on a wider scale involving a more general population of domestic pigs, and other susceptible species (in particular, domestic ruminants) would likely result in high mortalities particularly in cattle. Pyrexia, necrosis of the mouth lining and diarrhoea may occur. The widespread and likely prolonged movement restrictions could cause serious overcrowding and associated animal health problems as pigs, for example, outgrow their accommodation and cannot be moved on. Given this, it was considered that the direct impact on animal health would be significant at the national level. This resulted in a ranking of 'F' for this criterion.

Environment

Because Rinderpest virus is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of rinderpest

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

As described for scenario 3, Australia's policy for rinderpest is to eradicate by stamping out even if the disease were present in a number of areas. Zoning would be employed in those areas, together with stamping out and associated control measures. All the measures described above would be applicable to this scenario.

In light of this information, the indirect impact of control and eradication programs was considered to be significant at the national level. Hence the rating assigned to this criterion was 'F'.

Domestic trade or industry effects

The restrictions imposed on the movement of animals and animal products with a more generalised outbreak is likely to cause disruption to local marketing of animals and products. Interstate trading restrictions may apply on meat until tracing and surveillance was completed. As export of meat would likely cease, meat would be redirected to the local market. Overall, this is likely to cause a reduction in meat prices. An outbreak in an area involving dairies may result in short-term shortages of milk. Consumers may also initially decrease consumption of meat following an outbreak which resulted in high cattle mortalities. There would loss of genetics if breeding herds were involved in the outbreak.

Associated industries such as abattoirs, processors, transport, and stock feed manufacturers would also be affected if the outbreak were prolonged. Job losses both on farms and in associated industries may result with a widespread outbreak.

There are likely to be increased feed costs and welfare concerns for those producers whose premises are not infected but which are subject to movement restrictions.

In view of these factors, the indirect effect of a more generalised outbreak of rinderpest on the domestic trade or industry was considered to be significant at the national level, thus resulting in a ranking of 'F' for this criterion.

International trade effects

The response by Australia's trading partners to the diagnosis of rinderpest in either a local population of animals (scenario 3) or a more widespread outbreak is likely to be the same. Exports of susceptible animals and their products would cease until Australia could provide proof of freedom. On this basis, the likely impact of rinderpest on international trade was considered to be highly significant at the national level. This gave the disease a rating of 'G' for this criterion.

Indirect impact on the environment

An outbreak of rinderpest in a general population of susceptible animals, as described by this scenario, is likely to have indirect environmental impacts resulting mainly from the disposal of animal carcasses. Additional impacts could arise from the widespread use of disinfectants to decontaminate infected properties.

Overall it was considered that the indirect impact on the environment was of minor importance at the national level. This resulted in a rating of 'E' for this criterion.

Indirect impact on communities

An outbreak of rinderpest would affect the rural and regional economic viability including such things as businesses reliant on livestock revenue, employment, local governments together with social costs to individuals and communities. Those communities that are highly dependent on livestock industries would be significantly affected with associated job losses and social consequences.

Considering these factors, the indirect impact of rinderpest on rural communities was considered to be highly significant at the national level. Overall this resulted in a rating of 'G' for this criterion.

The overall impact of rinderpest

When the direct and indirect impacts of rinderpest were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences negligible

Scenario 3: Consequences extreme

Scenario 4: Consequences extreme

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 29, Table 30, and Table 31. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were considered 'high', 'extreme' and 'extreme' respectively.

Table 29 Rinderpest: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	High	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Low	Extreme	High
Scenario 4	Extremely low	Extreme	Low
		Overall likely consequences	**High**

Table 30 Rinderpest: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	High	Extreme	Extreme
Scenario 4	Extremely low	Extreme	Low
		Overall likely consequences	**Extreme**

Table 31 Rinderpest: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	High	Extreme	Extreme
Scenario 4	Extremely low	Extreme	Low
		Overall likely consequences	**Extreme**

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with rinderpest virus.

Table 32 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly higher than Australia's ALOP (very low), risk management would be required for rinderpest virus.

Table 32 Rinderpest: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Very low	High	High	High
Backyard pigs	Very low	Very low	Extreme	Moderate
Small commercial piggeries	Very low	High	Extreme	Extreme
			Overall annual risk	**Extreme**

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Swine vesicular disease virus

Technical information

Background

Swine vesicular disease (SVD) was first recognised in Italy in 1966, where initially it was assumed to be foot-and-mouth disease (FMD). Clinical signs of SVD in pigs, when present, are indistinguishable from those of FMD, vesicular stomatitis and vesicular exanthema of swine. For this reason, the Office Internationale des Épizooties (OIE) placed the disease in 'List A'.

Agent taxonomy

The virus is a member of the enterovirus genus of the Picornaviridae family (Pensaert, 1989).

Agent properties

The virus is a single-stranded, positive-sense, nonenveloped RNA virus (Pensaert, 1989). Differences in pathogenicity exist amongst isolates of SVD virus; however, antigenic differences are slight and the virus is considered to occur as a single serotype (Dekker, 2000). Studies of the molecular epidemiology of the virus suggest that it arose from a single transfer of the human coxsackie B5 virus to pigs sometime between 1945 and 1965 (Zhang, et al., 1999).

Persistence of SVD virus outside the host is due to its ability to withstand changes and extremes in temperature and pH. The virus is stable between pH 2.5 to 12, depending on temperature and time (Herniman, et al., 1973). Infectivity was maintained for 164 days when virus was added to inorganic buffers with pH ranging between 5.10 and 7.54 and maintained at 5°C. Virus has been shown to persist in infected carcass tissues for at least 11 months when these were stored at -20°C (Dawe, 1974). In the same study, faeces from infected pigs were stored in 50 kg plastic bags (ambient temperature varied between 12°C and 17°C) and sampled periodically for presence of viable virus, the last isolation of which was at 138 days of storage. The virus resists desiccation in the presence of organic material (Loxam & Hedger, 1983).

The virus is heat-labile; however, the temperature at which it is inactivated depends on the substrate or solution in which the virus is contained, and the duration of heating. For instance, at 65°C and 70°C, detectable infectivity was noted for several minutes for virus held in serum-free F15 medium but for only 0.5 minutes for virus held in Tris buffered saline (Cunliffe, 1974). Virus in alkaline pig slurry (pH 7.8 to 8) was inactivated by heating the slurry to 50°C to 55°C whereas in acidic slurry (pH 6.4), inactivation occurred between 55°C and 60°C (Turner, et al., 1999).

Host range

Swine vesicular disease occurs naturally only in the pig. Intracerebral or intraperitoneal inoculation of infant mice with the SVD virus results in neurological signs (tremor, paralysis) and high mortality (Burrows, et al., 1974a). Virus can be recovered from pharyngeal and rectal swabs of cattle and sheep, when these animals are closely confined with infected pigs that are excreting large quantities of virus (Burrows, et al., 1974b). In this study, no evidence of active infection was detected in the cattle. In contrast, the sheep seroconverted and the amount of virus recovered from the sheep pharynxes indicated active growth of the virus. Nonetheless, no clinical signs of SVD have ever been reported in either sheep or cattle, and animals other than

pigs have never been implicated in the epidemiology of the disease. Laboratory workers exposed to the virus may seroconvert, but SVD is not usually described as a zoonosis although a mild influenza-like illness may be associated with human exposure to the virus (Lin & Kitching, 2000). Thus, there are considered to be no 'other susceptible species' for swine vesicular disease.

Epidemiology

Swine vesicular disease virus is thought to have originated in Asia prior to its initial identification in Italy in 1966 (Lin & Kitching, 2000); however, little information is available concerning the current status of most Asian countries with respect to this virus. The virus was detected in Hong Kong in 1971, in the United Kingdom in 1972 and in many European countries thereafter (Burrows, et al., 1974a; Lin & Kitching, 2000). Swine vesicular disease has been eradicated from most of the European Union, with the exception of regions in southern Italy, where the virus continues to circulate despite an eradication program since 1995 (Brocchio, et al., 2002). The virus was last reported in Spain in 1993, the Netherlands in 1994 and Portugal in 1995[41]. The last outbreak reported in Taipei China was 1999.

The incubation period for SVD varies from 2 to 7 days depending on dose and route of exposure (Lai, et al., 1979; Loxam & Hedger, 1983). Extremely large quantities of virus (up to $10^{9.8}$ PFU/g)[42] are shed from vesicular lesions (Dekker, et al., 1995). Virus is also shed in faeces and is present in expired air (Sellers & Herniman, 1974). However, aerosol spread is not a feature of this disease as the particle size results in precipitation of the particles after only a short distance. Transmission occurs after direct contact with an infected pig, indirectly via exposure to contaminated environment or equipment or after consumption pig meat derived from an infected pig. An examination of SVD in Great Britain from 1972 to 1981 determined the source for 76% of 518 outbreaks - Table 33 (Hedger & Mann, 1989).

Table 33 Origin of outbreaks of swine vesicular disease in Great Britain 1972 - 1981

Origin	Number of outbreaks	Percentage of total outbreaks (%)
Movement of pigs	82	16
Contaminated hauliers' vehicles	107	21
Contact at markets	57	11
Movement of equipment of personnel	35	7
Local spread	16	3
Recrudescence	15	3
Feeding of contaminated waste food	80	15
Obscure origin	126	24

Infection may occur by a variety of routes but abraded skin is most vulnerable to infection (Mann & Hutchings, 1980). Abraded skin can be infected by as little as $10^{3.6}$ PFU of virus

[41] Source of information: Handistatus II (http://www.oie.int/hs2/report.asp)
[42] PFU: plaque-forming units

(three of four pigs infected), whereas application of $10^{6.8}$ PFU was required for infection by the oral route (three of six pigs), direct application to the tonsil and instillation into the nose or eye (four of six pigs in each case).

The epidemiology of SVD in a country in which the disease is endemic is confused by control programs. Swine vesicular disease is a relatively 'new' disease, and the inability to clinically distinguish between vesicular lesions caused by SVD and FMD, in particular, has ensured that concerted efforts are made to eradicate the disease when detected. The disease is often described as a 'pen' disease, rather than a 'herd' disease, as morbidity within a pen is typically very high, yet the proportion of pens infected per herd varies widely according to movement of animals and equipment and, in particular, the existence of a common drainage system between the pens (Lin & Kitching, 2000).

Clinical signs

When present, the clinical signs of SVD are indistinguishable from those of FMD. Initially a pyrexia of up to 41°C lasts 2 to 3 days. Vesicles may develop on the coronary bands of the feet and, less frequently, on the snout, lips, tongue and teats. On occasions, the vesicles on the feet may extend up the legs. Early symptoms include anorexia and lameness, the latter resolves when vesicles rupture (Loxam & Hedger, 1983). The severity of systemic effects varies; the disease tends to be more severe in younger pigs but even in these animals, recovery is rapid and mortality negligible (Loxam & Hedger, 1983). The direct losses caused by the disease (weight loss, piglet mortality) are generally insignificant[43]. Some strains of SVD virus cause no clinical disease and are detected only through laboratory surveillance. The 2002 outbreak of SVD in Italy involved subclinical infection in all but one of 10,312 pigs (Brocchio, et al., 2002).

Pathogenesis

After gaining entry to the body via abraded skin, mucous membranes or the intestinal tract, the virus replicates at the site of entry. The virus has a tropism for epithelial cells (Lai, et al., 1979) and vesicles develop as a result of coagulative necrosis that begins in the stratum spinosum then spreads to all layers of the epithelium (Lenghaus & Mann, 1976). High concentrations of the virus are found in draining lymphatics, lymph nodes and tonsils. Viraemia ensues, with subsequent dissemination of virus throughout the body. Viraemia may be present as early as 24 hours following exposure to the virus (preceding the development of clinical signs) and declines with the development of neutralising antibodies approximately 4 to 7 days after infection (Lai, et al., 1979; Dekker, et al., 1995). Concentrations of virus in epithelial tissues, myocardium, and brain are higher than those detected in plasma and is thought to indicate viral replication in these tissues (Dekker, 2000). There is no evidence that SVD virus has a tropism for skeletal muscle cells. Virus is present in the faeces of pigs for 20 to 30 days following infection (Brocchio, et al., 2002). The identification of virus in faeces, after it can no longer be detected in tissues, may indicate persistence in the intestinal tract (Lin & Kitching, 2000). Occasionally, pigs are reported to harbour virus for up to 4 months (Escribano-Romero, et al., 2000), but this has been difficult to reproduce and it is concluded that persistence of infection with SVD virus is rare (Lin, et al., 2001). Nonetheless, one study demonstrated that virus or viral RNA could be isolated in the faeces, nasal swabs or tonsillar tissues up to 63 days post-infection. Although virus could not detected between 63 and 119 days post-infection, the virus was detected in the faeces of the pigs for 7 days after stressing the pigs at 119 days post-

[43] Press release, Office International des Épizooties, 22 September 2000. http://www.oie.int/eng/publicat/press/a_000922.htm

infection (Lin, et al., 1998). Viral RNA has also been detected in somatic muscle in one study for 35 days and in another for 25 days (Lin, et al., 1998; Niedbalski, 1999); however, it is not known if the tissues are still infectious.

Pathology

The pathology associated with infection of pigs by SVD virus has been described (Lenghaus & Mann, 1976; Lai, et al., 1979). Vesicles, when present, may be the only grossly apparent lesions. After experimental infection, significant microscopic changes were apparent in the skin, snout, tongue, tonsil, gastrointestinal tract, kidney, salivary gland and pancreas, with minor changes observed in many other tissues. In addition, a nonsuppurative meningoencephalomyelitis has been reported (Lai, et al., 1979).

Immunology

Neutralising antibodies may be detected as early as 4 days post-infection (Lai, et al., 1979; Dekker, et al., 1995), peak around 21 to 28 days post-infection and are thought to remain high for years (Hedger & Mann, 1989).

Transmission via meat

The transmission of SVD virus via meat or meat products is well documented. As mentioned above, 80 of 518 outbreaks of SVD (15%) occurring in Great Britain between 1972 and 1981 were attributed to the feeding of contaminated waste food to pigs (Hedger & Mann, 1989).

The amount of virus present in pork or pork products depends on factors including the amount of virus with which the infected pigs were challenged, number of days post-infection, method of slaughter, efficiency of exsanguination and treatment of the product. Viral titres in muscle and associated tissues of infected pigs have been reported. In one study, the virus content of meat from infected pigs slaughtered when clinical signs were most severe (2 to 3 days post-inoculation) ranged from 10^3 to $10^{4.5}$ $TCID_{50}$ per gram (McKercher, et al., 1974). The virus content of hams from infected pigs after storage at 0 to 4°C for 72 hours varied between $10^{4.4}$ to $10^{4.6}$ PFU/g (McKercher, et al., 1985). However, virus was not detectable from hams from similarly infected pigs that were exsanguinated following stunning rather than anaesthesia. In another study, virus was isolated from the blood of 26 of 32 Iberian black pigs and 31 of 32 Spanish white pigs slaughtered 3 days post-inoculation with SVD virus. However, virus was isolated from the muscles of only two of the Iberian pigs and four of the Spanish pigs (Mebus, et al., 1993).

The persistence of SVD virus in pork and pork products has been examined. Carcass material frozen at -20°C for 11 months was reported to have 10^6, 10^4, 10^3, and 10^3 $TCID_{50}$ per gram in skin, intercostal muscle, rib bone and kidney cortex, respectively (Dawe, 1974). No virus was detectable in cooked, canned hams prepared using meat from infected pigs; the canning process involved heating the products up to an internal temperature of 69°C over a 5 hour period (McKercher, et al., 1974). However, during the same trial, virus was recovered for at least 200 days after processing from dried salami products, dried pepperoni sausage and intestinal casings derived from infected pigs. Similarly, other workers have shown the prolonged persistence of SVD virus in artificially-contaminated salami sausages for at least 42 days but not in similarly-contaminated ‘mortadelle’ hams in which an internal temperature of 60°C was reached after about 8 hours of processing (Frescura, et al., 1976). The survival of the virus in salted, dried ham products has been assessed. ‘Parma hams’ derived from SVD virus-infected

pigs slaughtered at peak viraemia were free of infective virus by the end of the official curing period of 12 months (McKercher, et al., 1985). The duration of viral persistence varied between Italian and United States replicates of the experiment and is likely related to the different viral content of the samples at processing. Other workers have studied the persistence of SVD virus in Iberian hams, shoulders and loins, and Serrano hams (Mebus, et al., 1993). A process of controlled salting and drying is used to produce these items. In this study, the Iberian loins, shoulder hams and hams were free of viable SVD virus by days 28, 112, and 560, respectively whereas Serrano hams were free of viable SVD virus by day 539, exceeding the maximum commercial curing time for this product.

An oral infective dose sufficient to infect 50% of pigs (three of six) of $10^{6.8}$ PFU has been reported . In the same study, lesser amounts of virus ($10^{3.6}$ PFU) infected three of four pigs when applied to abraded skin(Mann & Hutchings, 1980).

Release assessment

R1 — the likelihood that a source herd is infected

It is difficult to estimate the between herd prevalence of SVD infection in a country where the disease is endemic in the absence of a control program. In nationwide surveys conducted in Japan in 1973 and 1975 after outbreaks of SVD positive sera were found in 11.9% and 42.6% of the pigs respectively (Saito, et al., 1977). Given the extraordinary persistence of the virus, and the variety of means of transmission (direct contact, fomites, infected pork products), it was considered that, in the absence of any control programs, the likelihood that slaughter-age pigs have been selected from an infected herd was ‘moderate’.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

Swine vesicular disease is described as a ‘pen’ rather than a ‘herd’ disease. The within-pen morbidity is usually high; however, the proportion of infected pens is highly variable and depends on factors such as amount of movement of pigs and equipment throughout the farm, and the presence of a common drainage system between pens. It is considered that pigs generally do not become persistently infected (Lin, et al., 2001), although there are occasional reports of persistently infected pigs. Viraemia is only present for about 7 days post-infection (Dekker, et al., 1995). Although viral RNA has been detected at 28 days post-inoculation, these pigs were not infectious to sentinels (Lin, et al., 2001). In somatic muscle viral RNA has been detected at 35 days post-infection. Depending on the age at which pigs are infected they may no longer be viraemic or contain viable virus in tissues at the time of slaughter.

On the basis of this information, it was considered that the likelihood of selecting an infected pig in an infected herd was ‘moderate’.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

The clinical signs of SVD are characteristic of vesicular diseases and clinically-infected pigs are unlikely to pass ante-mortem inspection. However, subclinical infection is a feature of SVD, with lesions rarely detected in pigs in Italy where the condition is endemic. Post-mortem inspection of the carcass is more likely to confirm suspicions rather than reveal unsuspected infection.

In light of this information, the sensitivity of the ante-mortem, slaughter and processing requirements in detecting and removing pigs infected with SVD virus was considered to be ‘extremely low’.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with SVD and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a ‘one minus the background rejection rate’. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered ‘extremely low’. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

Infection with SVD virus is characterised by its affinity for epithelial cells coupled with viraemia. The viraemia results in widespread distribution of the virus throughout the tissues of the body. The virus does not have a predilection for muscle tissue and its presence in muscle, lymph nodes and fat is due to the vascular perfusion of these areas. Nonetheless SVD virus is easily isolated from muscle tissue from infected animals after slaughter and bleeding out; however, the viral titres vary depending on factors such as amount of virus to which the infected pigs were exposed, number of days post-infection, method of slaughter, and efficiency of exsanguination.

Taking these factors into consideration, the likelihood that SVD virus would be present in the meat harvested for export from an infected pig was considered to be ‘high’.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

Swine vesicular disease virus is particularly hardy and resistant to pH changes between 2.5 and 12. Thus, the likelihood that SVD virus will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation was considered to be ‘high’.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

Swine vesicular disease virus is very stable under cold conditions. For example, carcass tissues retained infectivity for at least 11 months when stored at -20ºC (Dawe, 1974). Even at ambient temperatures the virus can persist for a significant period of time.

Thus, it was considered that there was a 'high' likelihood that meat infected at the completion of carcass maturation would remain infected during transport and storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a 'low' likelihood that imported pig meat derived from an individual carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

High concentrations of SVD virus are present in the tissues of viraemic pigs. The presence and persistence of virus has been documented in a variety of pig meat products. Historically, outbreaks of SVD are associated with feeding of contaminated meat or meat products in swill. Pigs may be infected by contact with, or ingestion of, meat or meat products derived from pigs infected with SVD virus. Pigs scavenging for food may have abraded snouts and other areas of skin, and these may come in contact with food scraps, thus providing an alternative route of transmission for SVD virus. In one experimental study, some pigs became infected when fed as little as 2 ounces (56.7 g) of infected meat in which the viral titres were between 10^3 and $10^{4.5}$ PFU/g (McKercher, et al., 1974).

Given this, it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of SVD virus to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

Swine vesicular disease virus is described as being extraordinarily robust. It is highly resistant to inactivation, and is able to persist in the farm environment and on equipment for extended periods of time (Loxam & Hedger, 1983). It resists desiccation in the presence of organic material. Taking these factors into consideration, the likelihood that SVD would remain viable during the period prior to scavenging was estimated to be 'high'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = High
- Rural regions = High
- Large towns = Very low

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be ‘high’.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a ‘low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a 'high' likelihood that SVD virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'high'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in

small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall the Panel considered that there was a 'high' likelihood that SVD virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'high'.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the three exposure groups - that is, feral pigs, pigs in backyard enterprises and pigs in small commercial piggeries. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;

2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Swine vesicular disease virus is extremely robust. Vast quantities of virus are shed from vesicular lesions, when present. Lesser quantities of virus are shed in other bodily secretions and excretions, and virus may be detected in the faeces for 20 to 30 days. Rarely, some individuals are infected for a longer period of time. Transmission of SVD is by direct contact with an infected pig, or indirectly by exposure of susceptible pigs to contaminated environment, equipment, or meat products.

The lameness associated with SVD is generally not severe, and resolves upon rupture of the vesicles. Subclinical infection is common, and the clinical disease, when present, is limited in its severity. In general, the clinical symptoms of this disease would not be expected to limit the movement of feral pigs and hence, opportunity to create direct and indirect exposure opportunities for other pigs and pig herds. As such, disease may not be recognised in this population for a considerable period of time. Nonetheless, unlike classical swine fever, SVD is rarely detected in wild boar in Europe and does not appear to regularly spill-over into the domestic pig population.

Feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs, however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: moderate

Scenario 3: low

Scenario 4: low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the ‘no outbreak’ scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;

3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

Swine vesicular disease is transmitted easily between pigs within a pen and thus it is very likely that transmission amongst the backyard herd would occur, if at least one pig from the herd has been infected by exposure to pig meat scraps. As clinical signs of the disease may be inapparent or mild the owners of the pigs may not recognise infection. It is feasible that some mixing between pigs from an infected backyard herd and other domestic pigs may occur resulting in further spread of the disease, particularly in light of the excretion of virus in faeces for up to 30 days. For example, in the case of speciality breeds or unusual breeds live pigs may be transferred from one herd to another for breeding purposes. Alternatively, pigs raised for personal consumption may be transferred between backyard holdings for growing out or fattening.

As discussed above, the spread of SVD virus from domestic pigs to wild boar or vice versa is not a feature of the epidemiology of SVD in Italy, or when outbreaks occur elsewhere.

Indirect spread by fomites or by mechanical vectors is a feature of SVD transmission due to the robustness of the virus and its persistence on equipment and vehicles. Swine vesicular disease may be transferred from an infected backyard herd to other domestic pigs through inadequately cleaned boots and trucks.

If the strain of SVD virus introduced was virulent, resulting in vesicular lesions, it is likely that the disease would be diagnosed when further spread to small commercial piggeries occurred. However, if a low virulent strain was introduced, spread to large commercial piggeries is feasible. The disease could spread within the domestic pig population with pig movements before coming to the attention of regulatory authorities.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: moderate

Scenario 4: low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the ‘no outbreak’ scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between ‘small commercial piggeries’ and ‘backyard piggeries’ is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

As discussed above, it was considered more likely that the disease would be diagnosed (if clinical signs were present) within the initially-exposed small commercial piggery, but otherwise, the pattern of disease spread was considered similar. Nonetheless it should be noted that in the United Kingdom, due to the mildness of clinical signs, only 50% of outbreaks were reported by owners (Watson, 1981). An important consideration was the larger number of pigs moved from small commercial piggeries and the contamination of trucks increasing the likelihood of further spread of the virus. Again, in the absence of knowledge concerning the virulence of the outbreak strain, it is difficult to predict when the virus might be detected and contained, but the virus could well spread within the domestic pig population before diagnosis.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: low

Scenario 4: moderate

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes ‘no further establishment or spread’. The remaining three outbreak scenarios describe ‘secondary spread to other pigs, humans or to other susceptible species’. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this scenario, SVD would have established in the directly exposed animal, or group of animals, but would not have spread to other pigs. In the case of a feral pig herd or backyard pig enterprise being infected, this 'no outbreak' scenario would have resulted from low probability of contact between infected and susceptible animals, rather than from human intervention. Indeed, because signs of the disease may be mild, it was assumed that it would not have been identified in these exposure groups. In the case of a small commercial piggery, due to closer observation, it was assumed that the disease would have been identified and contained due to implementation of a control and eradication program.

The direct impact of swine vesicular disease

Animal life or health

Infection of pigs with SVD virus can result in fever and vesicular lesions and these lesions may be accompanied with lameness or difficulty eating. The systemic effects of SVD varies, however, recovery is generally rapid. Moreover some strains of SVD virus cause no clinical disease.

On this basis the direct effects of infection with SVD virus on animal health, where the disease is contained within the directly exposed group, was considered unlikely to be discernible at any level. Thus, the criterion was rated as 'A'.

Environment

Because swine vesicular disease is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of swine vesicular disease

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

This criterion was not rated identically for each exposure group.

It is likely that if the disease was contained within a feral pig herd or a single backyard enterprise, SVD would not be diagnosed within these herds. However, if the primary outbreak involved a small commercial piggery it was considered that pigs showing clinical signs of a vesicular disease would be investigated.

If SVD was identified in Australia in a small commercial piggery, the policy as outlined in AUSVETPLAN (Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 1996) is to eradicate SVD in the shortest possible period, while limiting economic impact, using a combination of strategies including stamping out, quarantine and movement controls, decontamination, tracing and surveillance, zoning, and a public awareness campaign. The disease is classed as Category 3 under the Australian Emergency Animal Disease Cost-Sharing Agreement[44], and thus the cost of the response is to be covered by

[44] http://www.aahc.com.au/eadp/response.htm

government and relevant industries by contributions of 50% each. Category 3 diseases are of moderate public impact and have the potential to cause significant (but generally moderate) national socio-economic consequences through international trade losses, market disruptions involving two or more States, and severe production losses to affected industries, but have minimal or no effect on human health or the environment.

In this scenario, where SVD has not spread beyond the small commercial piggery, it is possible that the disease would be eradicated promptly. Nonetheless there would need to be extensive surveillance of the domestic and feral pig populations.

Overall, it was considered that the indirect impact of new eradication and control programs was unlikely to be discernible at any level when the primary exposure group was a feral pig herd or a backyard pig enterprise. This resulted in a rating of 'A' for this criterion. However, when the primary exposure group was a small commercial piggery, it was considered that the indirect impact of new eradication and control programs was unlikely to be discernible at the national level, but would have a minor impact at the State level, which would be responsible for its delivery. This gave the disease a rating of 'D' for this criterion.

Domestic trade or industry effects

This criterion was not rated identically for each exposure group.

As discussed above, it was considered unlikely that the disease would be detected in the initially exposed herd of feral pigs or single backyard enterprise, thus no domestic trade or industry effects would be expected, and the rating assigned to this criterion was 'A'.

In the case of a small commercial piggery it was considered that the index herd may be detected (if clinical signs are present), in which case an eradication program would be implemented as discussed above. Restrictions on the movement of pigs would be imposed. It is possible that following detection of SVD in one State of Australia, other States may close their borders to all pigs and pig meat products until the extent of the outbreak was ascertained. As pig meat exports would cease at least in the short term, this product would enter the domestic market, resulting in an oversupply.

Taking these issues into account, when the primary exposure group was a small commercial piggery, it was considered that the indirect impact of SVD on domestic trade and industry was unlikely to be discernible at the national level, but would have a minor impact at the State level. This resulted in a rating of 'D' for this criterion.

International trade effects

This criterion was not rated identically for each exposure group.

As the disease is unlikely to be detected in the initially exposed herd of feral pigs or backyard enterprise the indirect effects of SVD on international trade for these exposure groups was unlikely to be discernible at any level, and thus, the rating assigned to this criterion was 'A'.

On confirmation of SVD in a small commercial piggery trade in live pigs, genetic material and pig meat (farmed and feral) would cease. International trade in animals and animal products other than those derived from pigs should not be affected. Australia exports few live pigs. Total exports of farmed pig meat in 2003 were valued at approximately $230 million. In the short term, it is expected that exports of pork would cease, however it might be possible to negotiate with Singapore concerning acceptance of Australian pork, as Singapore has no domestic pig

industry. Singapore's imports of farmed pig meat were valued at over $120 million in 2002 (Australian Pork Limited, 2003). The OIE Code Chapter on SVD recommends that for fresh meat imports, the entire consignment of meat comes from animals, which have not been kept or slaughtered in an abattoir situated in a SVD infected zone. With this restricted outbreak, the infected zone would be limited, and export may be able to continue from elsewhere in Australia. The OIE considers that a zone shall be considered as infected until at least 60 days have elapsed after confirmation of the last case of SVD and completion of a stamping-out policy and disinfection procedures.

Any confusion with FMD, if reported internationally, is likely to affect ruminant exports at least initially.

In light of this information, it was considered that the indirect effects of SVD on international trade when the primary exposure group was a small commercial piggery, were of minor significance at the national level. This resulted in a rating of 'E' for this criterion.

Indirect impact on the environment

In this scenario, SVD is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, SVD would have established in a broader population of feral pigs. In the case of spread from a feral pig herd or backyard pig enterprise, the disease would have been contained due to low probability of contact between infected and susceptible animals, rather than from human intervention. Indeed, because the disease may be of low pathogenicity for pigs, it was assumed that it would not have been identified in these direct exposure groups and feral pigs. In the case of spread from a small commercial piggery to feral pigs, it was assumed that the disease would have been identified in the small commercial piggery and contained due to implementation of a control and eradication program.

The direct impact of swine vesicular disease

Animal life or health

With this scenario, the disease spreads to a general population of feral pigs but not to domestic pigs. However, clinical signs (when present) are rarely severe, and mortality is uncommon. Overall, the direct impact on animal health is unlikely to differ from that of the direct primary exposure group and thus, this criterion was rated as 'A'.

Environment

Because SVD is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

This criterion was not rated identically for each exposure group.

In this scenario, although the disease has spread to a more general population of feral pigs it was considered that the outbreak is unlikely to be detected, as infection may be subclinical and feral pigs are not closely observed. Thus, as spread from a localised to a more general population of feral pigs or spread from backyard pigs to feral pigs may go undetected, no new or modified eradication, control, surveillance/monitoring and compensation strategies/programs would be implicated. Considering this, this criterion was rated as 'A' when the primary exposure group was feral pigs or a backyard pig enterprise.

As discussed above for scenario 1, it was considered that the disease may be diagnosed within a small commercial piggery. Nonetheless spread to feral pigs may occur before the diagnosis is confirmed. As such, eradication and control programs, as previously discussed, would be implemented. However, the extent and costs of any eradication or control programs would depend on the results of surveillance and assessment of the role of the feral pigs in the epidemiology of the disease in domestic animals. Feral pig populations may need to be contained or reduced to a level where the disease is unlikely to be transmitted and may die out.

After consideration of these issues, when the primary exposure group was a small commercial piggery, it was considered that the indirect impact of eradication and control programs was unlikely to be discernible at the national level, but would have a minor impact at the State level. This gave the disease a rating of 'D' for this criterion.

Domestic trade or industry effects

This criterion was not rated identically for each exposure group.

As for scenario 1, the indirect effect on domestic trade or industry, when the primary exposure group was feral pigs or a backyard pig enterprise was unlikely to be discernible at any level, hence the raying assigned to this criterion was 'A'.

However, if the primary exposure group was a small commercial piggery, the disease may be detected and the indirect effect on domestic trade or industry would be similar to that described above for scenario 1. This resulted in a ranking of 'D' for this criterion.

International trade effects

As SVD was considered unlikely to be diagnosed with further spread to feral pigs when the primary exposure group was feral pigs or a backyard pig enterprise, the indirect impact on international trade was considered unlikely to be discernible at any level. This resulted in a rating of 'A' for this criterion.

In the case where the primary exposure group was a small commercial piggery, it was considered that the disease would be detected and hence trade in pigs, genetic material and pig meat would, at least initially, cease until either Australia could claim freedom from the disease or renegotiate access based on such things as zoning, testing or quarantine. Thus, as for outbreak scenario 1, it was considered that the indirect effect of SVD on international trade,

when the primary exposure group was a small commercial piggery, was of minor significance at the national level. This resulted in a rating of 'E' for this criterion.

Indirect impact on the environment

In this scenario, SVD is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, SVD would have established in a local population of backyard piggeries or small commercial piggeries. The disease would be contained through the diagnosis of disease in pigs, and the mounting of an eradication program.

The direct impact of swine vesicular disease

Animal life or health

With this scenario, the disease spreads to a local population of domestic pigs. However, clinical signs (when present) are rarely severe, and mortality is uncommon. Overall, it was considered unlikely that the direct impact on animal health would be discernible at other than the local level. Hence the rating assigned to this criterion was 'B'.

Environment

Because SVD is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of swine vesicular disease

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

As discussed previously, the Australian policy is to eradicate SVD in the shortest possible period whilst limiting economic impact. This would be achieved using a combination of strategies including stamping out, quarantine and movement controls, decontamination, tracing and surveillance, zoning and a public awareness campaign.

In this scenario, SVD has spread to a local population of domestic pigs in backyard enterprises or small commercial piggeries but not to large commercial piggeries. Hence the disease could be eradicated promptly from the domestic pig population. Nonetheless, there would need to be extensive surveillance of the domestic and feral pig populations to demonstrate freedom.

Overall, it was considered that the indirect impact of new eradication and control programs was unlikely to be discernible at the national level but would have a minor impact at the State level. This gave the disease a rating of 'D' for this criterion.

Domestic trade or industry effects

The indirect effects on domestic trade or industry would be similar for this outbreak as for that described above for the outbreak involving one small commercial piggery due to the limited spread of the disease. There is likely to be some disruption to domestic trade in meat. States may initially close their borders to pigs and pig products, and meat destined for the export market may be redirected to the local market. There would be restrictions on movement of pigs and pig products in the area where the outbreak occurred. Producers whose herd were destroyed and others whose herds were subject to movement restrictions would suffer loss of income. There would be the cost of replacing the breeding herd. There could be increased feed costs for those piggeries unable to freely market pigs.

Overall, it was considered that the indirect effect on domestic trade, whilst not discernible at the national level, would have a minor impact at the State level. Hence the rating assigned to this criterion was 'D'.

International trade effects

The effects on international trade of a confirmed outbreak of SVD in Australia would be similar to those described for outbreaks scenarios 1 and 2 when the primary exposure group was a small commercial piggery. Thus, it was considered that the indirect effect of SVD on international trade was of minor significance at the national level, resulting in a rating of 'E' for this criterion.

Indirect impact on the environment

The disposal of pigs by burial or cremation can present environmental problems. However, in this scenario the disease has spread only to a local population of backyard enterprises or small commercial piggeries. Hence the numbers slaughtered would not be great and it was considered unlikely to lead to any discernible indirect impact on the environment other than at the local level. Thus, a rating of 'B' was assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, SVD would have established in a broader population of commercial piggeries (including medium-large piggeries). An eradication program would have been mounted in response to the diagnosis of the disease in pigs.

The direct impact of swine vesicular disease

Animal life or health

Clinical signs (when present) are rarely severe, and mortality is uncommon. With this scenario, the disease spreads to a more general population of domestic pigs, thus larger numbers of pigs will be affected and productivity losses might become apparent in some cases due to resulting lameness and reluctance to eat. Taking this into consideration, the direct impact on animal health was considered unlikely to be discernible at any level, except locally. This resulted in a rating of 'B' for this criterion.

Environment

Because SVD is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of swine vesicular disease

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

An eradication program for SVD involving destruction of animals and contaminated animal products, decontamination, and compensation would be a significant cost to governments and industry. With an extensive outbreak involving several States, considerable surveillance and monitoring would need to be undertaken of the domestic pig population. Some surveillance of the feral pig population may also be required. If the disease was widespread in the feral pig population, SVD may only be able to be eradicated from the domestic pig population. If this was the case pig producers may need to improve biosecurity to prevent contact with feral pigs. Zoning may also be an option following a widespread outbreak.

In view of this, the indirect impact of new eradication and control programs was considered to have a minor impact at the national level. This gave the disease a rating of 'E' for this criterion.

Domestic trade or industry effects

The restrictions imposed on the movement of pigs and pig products with a more generalised outbreak may cause disruption to the local marketing of pig meat. Some interstate trading restrictions may apply on meat until tracing and surveillance was completed. Meat destined for the export market would enter the domestic market. Consumers may also decrease consumption of pork following an outbreak and a publicity campaign would likely need to be conducted to reassure the public that there were no health concerns. There would loss of genetics if breeding herds were involved in the outbreak. There are likely to be increased feed costs and welfare concerns for those producers whose premises are not infected but which are subject to movement restrictions. For those producers whose premises were infected, the long delay in repopulation will result in financial loss.

Associated industries such as abattoirs, processors, transport, and stock feed manufacturers would also be affected if the outbreak was prolonged. Unemployment may result.

Taking these factors into consideration, the indirect impact of SVD on domestic trade and industry was considered of minor significance at the national level. Overall this resulted in a rating of 'E' for this criterion.

International trade effects

The effects on international trade of an outbreak of a confirmed outbreak of SVD in Australia would be similar to that described above, although likely more prolonged due to the extent of the outbreak.

In the unlikely event that SVD was not eradicated and became endemic, due to the possibility of confusion with FMD, there is the potential for sporadic disruptions to international trade in cattle, sheep and their products.

Overall it was considered that the indirect effect on international trade was of minor significance at the national level. This gave the disease a rating of 'E' for this criterion.

Indirect impact on the environment

An important consideration would be the environmental issues associated with the slaughter and disposal of large numbers of pigs associated with an outbreak involving several pig producing regions. The environmental issues would need to be addressed prior to disposal. In view of this, the indirect impacts on the environment were considered unlikely to be discernible at the national and State levels, but of importance for the affected districts or regions. This resulted in a rating of 'C' for this criterion.

Indirect impact on communities

One of the considerations with this criterion was the indirect impact of SVD on rural and regional economic viability. The pig industry is important to the economies of several localities and districts in New South Wales, Victoria, Queensland, Western Australia and South Australia. It has been estimated that in general terms, for every one employee working in the pig industry, there will be another two people employed in providing goods and services to the pig industry directly or to the employees in the pig industry (Alliance Consulting and Management, 2000).

As discussed above, associated industries such as processors, the transport industry and stockfeed manufacturers may also be affected. Where the pig industry was highly significant to the local economy, aspects of these communities may be threatened.

Taking these factors into account, the indirect impact of SVD on rural communities was considered unlikely to be discernible at the national or State level, but of importance at the district or regional level. This resulted in a rating of 'C' for this criterion.

The overall impact of swine vesicular disease

When the direct and indirect impacts of swine vesicular disease were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible (feral pigs, backyard pigs), moderate (small commercial pigs)

Scenario 2: Consequences negligible (feral pigs, backyard pigs), moderate (small commercial pigs)

Scenario 3: Consequences moderate

Scenario 4: Consequences moderate

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 34, Table 35, and Table 36. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were considered 'low', 'moderate' and 'moderate' respectively.

Table 34 SVD: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Moderate	Negligible	Negligible
Scenario 3	Low	Moderate	Low
Scenario 4	Low	Moderate	Low
		Overall likely consequences	**Low**

Table 35 SVD: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Moderate	Moderate	Moderate
Scenario 4	Low	Moderate	Low
		Overall likely consequences	**Moderate**

Table 36 SVD: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Moderate	Low
Scenario 2	Very low	Moderate	Very low
Scenario 3	Low	Moderate	Low
Scenario 4	Moderate	Moderate	Moderate
		Overall likely consequences	**Moderate**

Human life or health

Separate to the above is consideration of the consequences to human life or health. Laboratory workers exposed to the virus may seroconvert, but SVD is not usually described as a zoonosis although a mild influenza-like illness may be associated with human exposure to the virus (Lin & Kitching, 2000).

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with SVD virus.

Table 37 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly higher than Australia's ALOP (very low), risk management would be required for SVD virus.

Table 37 SVD: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Low	High	Low	Low
Backyard pigs	Low	High	Moderate	Moderate
Small commercial piggeries	Low	High	Moderate	Moderate
			Overall annual risk	**Moderate**

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Aujeszky's disease virus

Technical information

Background

Aujeszky's disease (pseudorabies or 'mad itch') is predominately a disease of pigs but was first identified in cattle in the United States of America in 1813 (Kluge, et al., 1999). This viral disease affects the nervous, respiratory and reproductive systems, depending on the age of the pig when infected. Prior to the 1960s the disease was regarded as being of limited clinical or economic significance, however, with the emergence of virulent strains, more accurate diagnostic techniques and changes in management systems, the severity of the disease, prevalence and distribution have increased (Kluge, et al., 1999).

Agent taxonomy

Aujeszky's disease virus (ADV) is a member of the alphavirus subfamily of the family Herpesviridae (Pensaert & Kluge, 1989; Kluge, et al., 1999).

Agent properties

Aujeszky's disease virus is an enveloped, double-stranded DNA virus. Strains of varying pathogenicity have been reported; however, there is only one serotype of ADV. The survival of ADV outside a living host is affected by factors that include the substrate, pH, temperature, relative humidity and exposure to ultraviolet light.

In vitro experiments (Davies & Beran, 1981) have shown that ADV suspended in a physiological saline-dextrose-lactalbumin hydrolysate medium is quite stable at pH levels between 5 and 9 when maintained at stable temperatures between 4°C and 25°C. The sensitivity of the virus to increasing temperatures was demonstrated by an exponential increase in the inactivation rate as temperature increased from 4°C to 37°C. Rapid and repeated freezing and thawing of viral suspensions (various pH levels between 5 and 9.3) resulted in loss of viral titre. Loss of viral titre was also noted in frozen viral suspensions (various pH levels between 5.1 and 9.1) where the temperature fluctuated between -90°C and -13°C. Viral suspensions (pH levels between 5.1 and 9.1) held at a constant temperature of -13°C lost greater than 3 $\log_{10}$ of titre in three days. Drying inactivated virus suspensions, as did ultraviolet light. The survivability of ADV in saliva at infectious levels on a variety of different fomites has been reviewed and summarised (Kluge, et al., 1999); survival times were 4 days or less, with the exception of 7 days for both well water and green grass.

Host range

The pig is the natural host. Infection with ADV has been reported in many domestic and wild animal species including dogs, cats, ruminants, rodents, mink, ferrets and bears, but has not been substantiated in humans. In species other than the pig, infection with Aujeszky's disease virus is generally fatal within 1 to 3 days. However, it has been reported that some cats and rodents may survive infection with ADV and may possibly act as temporary vectors for transmission of the virus (Weigel, et al., 2000). Aujeszky's disease in species other than pigs is only reported to occur when the disease is endemic in the pig population (Vandevelde, 1998).

Epidemiology

Aujeszky's disease has had an almost worldwide distribution. Many countries, however, have either eradicated the disease, or are in the process of doing so. The disease has never been reported in Australia.

Pigs are reported to shed virus oronasally for 2 to 3 (or more) weeks following infection (Maes, et al., 1983), depending on strain of ADV. Pigs are usually infected via the oronasal route after direct contact with nasal secretions or aerosolised virus, but venereal and vertical transmission also occurs (Pensaert & Kluge, 1989; Hahn, et al., 1997). Virus may also enter a susceptible host via abraded skin. Experimentally, pigs may be infected after inoculation via intramuscular, -venous, -cerebral, -gastric, -nasal, -tracheal, -conjunctival, -uterine, -testicular, and oral routes. However, pigs are reported to be least susceptible to inoculation via the intragastric route (Kluge, et al., 1999).

The prevalence of infected herds within a country is affected by factors including density of pig population, size of pig herds, distance between herds, contact between herds, and the existence and characteristics of official Aujeszky's disease control programs. For the purposes of this IRA, the prevalence of infected herds will be considered in countries without official eradication control programs or in which vaccination against the disease is not practised. In the United States of America (prior to the initiation of control programs) the overall prevalence of infected herds in 1983 to 1984 was estimated to be 8.78% (Bech-Nielsen, et al., 1995). Aujeszky's disease was first diagnosed in the North Island of New Zealand in 1976. An abattoir survey conducted in 1988 to 1989 showed a 5.2% herds in the North Island were infected (Pannett, et al., 1999). Aujeszky's disease was first diagnosed in Argentina in 1979. A survey conducted during 1987 to 1988 found 25.7% herds infected (Echeverria, et al., 1992).

The prevalence of antibodies to Aujeszky's disease (seroprevalence) in pigs within an infected herd depends on factors such as infection history of the herd, herd vaccination status, herd structure, age group of pigs assessed, herd size and miscellaneous management practices. A study conducted in Minnesota USA (Anderson, et al., 1989) (Morrison & Thawley, 1989) of 15 farrow-to-finish herds in which vaccination was not practised, and in which at least 75% of sows were seropositive, showed that 4 of the 15 herds had no seropositive finishing pigs whilst the remaining 11 herds had 75% or more seropositive finishing pigs at some or all of the sampling points in the study. Pigs are more likely to be infected around weaning, rather than later during growth (Morley, 1993).

Clinical signs

The clinical signs of Aujeszky's disease have been reviewed (Pensaert & Kluge, 1989; Kluge, et al., 1999) and depend on the strain, the dose of virus and the age of the pigs at infection. The incubation period in slaughter-age pigs is 3 to 6 days, after which fever, depression, anorexia, sneezing and nasal discharge may occur. Pneumonia may develop. Neurological signs occur occasionally and range from mild muscle tremors to convulsions. Mortality is usually low in slaughter-age pigs (1 to 2%), but morbidity may approach 100%. Clinical recovery occurs in 6 to 10 days. The clinical signs described, although typical, do not occur with all strains of ADV. Respiratory signs were never a feature of Aujeszky's disease in the North Island of New Zealand, where the disease was manifest as a fatal neurological disorder of piglets, or as a reproductive disease in pregnant sows (Pannett, et al., 1999).

Pathogenesis

Aujeszky's disease virus is typical of herpesviruses in having an affinity for neurological tissue. The virus tends to spread via the lymphatics from the site of entry to the regional lymph nodes, where replication occurs (Kluge, et al., 1999). Spread may also occur via the nerves from the primary site of infection to the central nervous system (CNS). It is thought that all strains have an affinity for the upper respiratory tract and CNS, but that more virulent strains have a wider dissemination throughout the body and likely spread via infected peripheral blood mononuclear cells (Chinsakchai & Molitor, 1994). The duration of viraemia is short and cell-free viraemia is reported to be rare. High virulence strains have been isolated from tissues including alveolar macrophages, epithelium of terminal bronchioles, hepatocytes, lymphoid cells of the spleen and lymph nodes, adrenal cortical cells, trophoblasts and embryos from the gravid uterus, and luteal cells of the ovary (Iglesias, et al., 1992; Kluge, et al., 1999).

The virus may persist in pigs as a latent infection that can reactivate under conditions of natural or experimentally-induced stress, such as parturition, transportation or administration of exogenous corticosteroids (Davies & Beran, 1980). The lag time for recrudescence and viral shedding noted in one sow after farrowing was 3 days (Davies & Beran, 1980). The trigeminal nerve ganglia are a major site of ADV latency; in addition, ADV has been shown to persist in tonsils and olfactory bulbs of 10 and 9, respectively, of 11 pigs examined 64 days post-inoculation (Wheeler & Osorio, 1991), although tonsillar tissue was not confirmed as a site of persistence by other workers (Balasch, et al., 1998). An interesting finding in this later work was the identification of ADV genome in the bone marrow of 5 of 15 persistently infected pigs.

Pathology

Gross lesions may be inapparent. When present, they may include a serous to fibronecrotic rhinitis that may extend to the larynx and down the trachea. Necrotic tonsillitis is a feature of the disease and lymph nodes of the oral cavity and upper respiratory tract may be swollen and haemorrhagic. On occasions, pulmonary oedema and scattered, small foci of consolidation of the lungs may be observed. Mild to severe keratoconjunctivitis is frequently present (Pensaert & Kluge, 1989; Kluge, et al., 1999).

Immunology

The porcine immune response to ADV involves both humoral and cell-mediated components. The immune response is not able to clear the body of latent virus (Chinsakchai & Molitor, 1994). Vaccines against ADV protect pigs against clinical disease and the duration, and amount of viral shedding, is reduced. This serves to reduce the amount of virus circulating in a herd, and thus, vaccination can be an important component of an eradication program. Vaccines have been developed with selected deletion markers which enable vaccinated animals to be differentiated from those naturally infected. However, inactivated, modified live vaccines and the gene-deleted vaccines, developed to date, are unable to prevent infection with wild strains of ADV. In addition, they may not necessarily prevent of establishment of latent infection with the wild strain (Kluge, et al., 1999).

Transmission via meat

Aujeszky's disease virus has been detected at very low titres in the muscle of experimentally infected pigs. In one experiment the presence of ADV in porcine muscle, lymph node and bone marrow was assessed 60 h after intranasal inoculation of three weaner pigs with 10^6 $TCID_{50}$

and 90 minutes after intravenous inoculation of an additional three pigs with 10^8 $TCID_{50}$ (Durham, et al., 1980). Virus was detected at very low titres in the fresh muscle tissue of the pigs infected intravenously, and only in the tonsillar tissue of two of the three pigs infected intranasally. In another study, tissues were examined from two pigs from each of three groups on days 3 and 7 after experimental intranasal infection with three different strains of ADV (Donaldson, et al., 1983). Virus was not isolated from muscle tissue in any of the 12 pigs, although virus with titres ranging from 10^1 to 10^6 $TCID_{50}$ was isolated from other tissues (mainly neurological and lymphoid tissues from the head and neck).

The study described above (Durham, et al., 1980) also investigated inactivation of ADV in tissues. The virus titres in muscle were considered inadequate for these studies, so 10^8 $TCID_{50}$ ADV was infused into a hindquarter of a freshly killed weaner pig. Muscle tissue, bone marrow, and lymph node samples from the hindquarter were then frozen at -18°C. Most of the virus in the muscle and bone marrow samples was rapidly inactivated (approximate half life 5 h), but the remaining virus appeared to be more heat stable, with a half-life of 4 days. Samples from the lymph node did not show this biphasic response, rather followed a simple inactivation curve with a half-life of 4 days. Virus was not detectable in any tissue after 35 days. In contrast to these results other workers (Pirtle & Beran, 1991) describe unpublished work showing that ADV could be recovered from 80% lean ground pork sausage (pH 5.85) stored at 4°C for 14 days and at -20°C for 40 days. However, neither the amount of virus mixed with the sausage nor the amount recovered was reported.

The transmission of ADV to other susceptible species after the consumption of porcine head or offal tissues is well documented. For example, (Horvath & Papp, 1967) linked the feeding of uncooked pork offal and scraps of pork meat to 58 cats diagnosed with Aujeszky's disease. In addition, many of the cats were successful hunters of rats and mice, which have also been implicated in the transmission of Aujeszky's disease to carnivores. Five bears travelling in Spain with a circus died acutely after being fed raw pigs' heads. In this case, the ADV strain was isolated from the bears and confirmed to be that circulating in pigs in northern Spain some years earlier (Banks, et al., 1999).

The transmission of ADV to pigs via consumption of tissues from heads of pigs that died acutely from Aujeszky's disease has been described, however, consumption of tissues from heads of latently-infected pigs did not result in transmission of the disease (Hahn, et al., 1997). Transmission of ADV to other susceptible species after the consumption of porcine head or offal tissues has also been described (Horvath & Papp, 1967; Banks, et al., 1999).

The introduction of Aujeszky's disease into a previously-free country or area is generally associated with movements of live animals or infected genetic material rather than the importation of carcass meat (that is, excluding offal and heads). For example, it has been estimated that Canada imported carcass meat derived from 56,048 to 79,511 ADV-infected pigs from the United States of America between 1975 to 1992, and no outbreaks of the disease have ever been reported in Canada (Morley, 1993).

The oral infectious dose of ADV infection for pigs has been estimated to be 10^1 to 10^3 $TCID_{50}$ for piglets, 10^4 $TCID_{50}$ for young pigs and 10^4 to 10^5 $TCID_{50}$ for adult pigs (Wittmann, 1991). These values are much larger than those required for infection via the intranasal route, and may vary according to strain of virus considered.

Release assessment

R1 — the likelihood that a source herd is infected

The prevalence of Aujeszky's disease within countries in which the disease is endemic has been reported as ranging from 5 to 26%. Based on these figures it was considered that there was a 'low' likelihood that the herd from which slaughter-age pigs were selected would be infected.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

A study of 15 infected farrow-to-finish herds showed that in 11 of the herds at least 75% of slaughter-age pigs had been exposed to ADV, based on serological evidence, but that no slaughter-age pigs were infected with ADV in the remaining four herds (Anderson, et al., 1989). It is recognised that the majority of pigs will be infected as weaners, however, in the case of Aujeszky's disease persistent, latent infections are a feature. Using these figures as a guide, the likelihood that an infected pig was selected from an infected herd was considered to be 'moderate'.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

The clinical signs of Aujeszky's disease in slaughter-age pigs are usually those of fever, depression, and anorexia. Sneezing and nasal discharge may occur and pneumonia may develop. Neurological signs occur occasionally, and range from mild muscle tremors to convulsions.

Pigs infected with ADV will be condemned and removed from further processing if they are febrile, if they have acute encephalitis or meningitis, or if they have peracute pneumonia. Less severe pneumonia results in condemnation of the lungs, but not the carcass. In the early stages of acute infection animals may be viraemic, yet show limited clinical signs and it is likely that these animals will pass inspection procedures.

Nonetheless, subclinical infection can be a feature of Aujeszky's disease as are latent and persistent infections. These pigs are very unlikely to be condemned during ante-mortem and post-mortem inspection.

On the basis of this information, the sensitivity of ante-mortem, slaughter and processing requirements in detecting and removing ADV-infected pigs was considered to be 'very low'**.**

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with ADV virus and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a 'one minus the background rejection rate'. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered

'extremely low'. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

Aujeszky's disease virus has been detected, with difficulty, in muscle tissues in very low titres from pigs slaughtered at peak pyrexia. The virus does not appear to have an affinity for muscle tissue, but might be found in muscle during the brief period of viraemia, perhaps associated with infected peripheral blood mononuclear cells. However, Aujeszky's disease virus is consistently recoverable from latently infected pigs from some or all of the trigeminal ganglia, olfactory bulb, and tonsils, i.e. those tissues associated with the head. Viral genome has also been detected on occasions in bone marrow.

This evidence suggests that the likelihood that a carcass including the head, from a latently infected pig would be infected with ADV was 'moderate'.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

Aujeszky's disease virus is quite stable in the pH range of 5 to 9 and thus the likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation was 'high'.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

There is evidence that virus survival is adversely affected by frozen storage, however, studies have shown that the virus is quite stable at 4°C *in vitro* at a range of pH values. Moreover ADV inoculated into pork sausage could be isolated after the product was stored at 4°C for 14 days. On the basis of this information, it was considered that there was a 'high' likelihood that meat infected or contaminated with ADV at the completion of carcass maturation would remain infected during storage and transport.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a 'very low' likelihood that imported pig meat derived from an individual carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

There are limited data on the oral infectious dose of ADV, however, it is known that pigs develop infection after oral inoculation. It has been estimated that infection by the oral route requires about $10^4 TCID_{50}$ in young pigs and $10^4 TCID_{50}$ to $10^5 TCID_{50}$ in adult pigs. Experimentally pigs are reported to be least susceptible to infection via the intra-gastric route of inoculation.

Aujeszky's disease virus has rarely been detected in muscle tissue even in acutely infected animals and then only by rabbit inoculation. However, the virus has been isolated from neurological and lymphoid tissues from the head and neck of acutely infected animals. Acutely infected animals showing clinical signs of disease would not pass inspection procedures, however, those in very early stages of infection would pass. It has been reported that the feeding of heads of acutely infected pigs to naïve pigs has resulted transmission of ADV, although consumption of tissues from heads of latently infected pigs did not result in transmission. It is unknown whether the brain was fed in either study. The virus can spread along the trigeminal and olfactory nerves to the medulla and pons. It should be noted that in this IRA the brain is not included in the definition of a carcass, although neurological tissue that cannot be separated from muscle is considered.

The likelihood that a waste unit would contain a sufficient dose of ADV to initiate infection, given that it was derived from an infected pig, was based on the source of the waste unit (head and neck region or the rest of the carcass).

Carcass region from which waste unit derived	Weighting factor	Likelihood that waste unit would contain a sufficient dose of ADV to initiate infection
Head and neck	10%	'Moderate'
Rest of the carcass	90%	'Very low'

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

This step describes the sensitivity of ADV to ultraviolet light, ambient temperatures ranging from 10°C to 35°C and to the putrefying effects of saprophytic organisms. The virus is known to be highly susceptible to drying and to the effects of ultraviolet light. It is inactivated in 4 days or less on most environmental fomites, and *in vitro* studies have shown an exponential pattern of inactivation as temperatures rise above 4°C.

This information led the Panel to consider the likelihood that ADV would survive within meat scraps discarded in refuse for the period of time required for pigs to locate and subsequently scavenge the material was 'low'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered 'very likely' that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a 'moderate' likelihood but that it was 'very unlikely' that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs was derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of annual likelihood for each region was explained in Table 5.

- Remote regions = High
- Rural regions = Very low
- Large towns = Extremely low

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be 'high'.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit would contain a sufficient dose of ADV to initiate infection, given that it was derived from an infected pig, would be based on the source of the waste unit (head and neck region or the rest of the carcass), as follows:

Carcass region from which waste unit derived	Weighting factor	Likelihood that waste unit would contain a sufficient dose of ADV to initiate infection
Head and neck	10%	'Moderate'
Rest of the carcass	90%	'Very low'

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a 'moderate' likelihood that ADV would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'very low'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit would contain a sufficient dose of ADV to initiate infection, given that it was derived from an infected pig, would be based on the source of the waste unit (head and neck region or the rest of the carcass), as follows:

Carcass region from which waste unit derived	Weighting factor	Likelihood that waste unit would contain a sufficient dose of ADV to initiate infection
Head and neck	10%	'Moderate'
Rest of the carcass	90%	'Very low'

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a 'moderate' likelihood that ADV would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'high'.

Exposure assessment for other susceptible species

Aujeszky's disease has only ever been reported in species other than pigs in areas where the disease is endemic in the pig population. Sporadic cases occur in other carnivorous or omnivorous species such as dogs, cats, raccoons, foxes and rats. As dogs can be infected, it is likely that dingoes could also be infected. Other species are generally regarded as 'dead-end hosts'. Other susceptible species generally become infected as a spill-over from infected pigs.

The transmission of ADV to other susceptible species after the consumption of porcine head or offal tissues is well documented. For example, (Horvath & Papp, 1967) linked the feeding of uncooked pork offal and scraps of pork meat to 58 cats diagnosed with Aujeszky's disease. In addition, many of the cats were successful hunters of rats and mice, which have also been implicated in the transmission of Aujeszky's disease to carnivores. Five bears travelling in Spain with a circus died acutely after being fed raw pigs' heads. In this case, the ADV strain was isolated from the bears and confirmed to be that circulating in pigs in northern Spain some years earlier (Banks, et al., 1999).

Experimentally rats have been infected orally with ADV at a dose of approximately 10^6 $TCID_{50}$ (McFerran, & Dow, 1970).

Given this, it was considered that the annual likelihood of entry and exposure for other susceptible species was 'moderate'.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the four exposure groups - that is, feral pigs, pigs in backyard enterprises pigs in small commercial piggeries and other susceptible species. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criterion, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and

4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Aujeszky's disease is usually spread via direct contact of susceptible pigs with acutely infected pigs that are shedding virus particles in respiratory and nasal secretions. Aerosol spread has been reported, but only in regions with dense pig populations. It is highly unlikely that conditions favourable for windborne spread of the virus will occur in Australia.

The likely signs of ADV in a pig herd will depend on the strain of ADV involved. Clinical signs of disease can be very mild and subclinical and persistent latent infections occur. Nonetheless, there can be high mortality in young pigs (prior to weaning), and respiratory disease with high morbidity but low mortality in older pigs. Reproductive failure (resorption or abortion) may occur in pregnant sows.

Feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs, however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

The spread of Aujeszky's disease from feral pigs to domestic pigs has occasionally been reported. One worker (Beran, 1993), citing a United States Department of Agriculture report, mentions that contact with infected feral pigs was the probably source of infection in 1.5% of newly infected herds reported in the United States of America in 1990. More recently, contact with feral pigs was reported as being the source of infection in a newly infected herd in Virginia (Taft, 2002). Close contact (nose-to-nose or, depending on strain, venereal) is required for transmission to occur.

Were transmission to a piggery to occur, it is likely that the disease would be amplified and spread regionally by fomites, live pigs, semen or other means to other piggeries before a diagnosis was established and controls to limit spread were initiated.

Although other susceptible species such as rats may become infected with ADV via consumption of carcass material from infected pigs they do not seem to play a major role in the spread of the disease. The infection of species such as cattle and sheep is unlikely under Australian conditions, as these animals are rarely housed in close, confined contact with pigs. Infection in all these species is usually short and self-limiting.

On balance, the following likelihoods were assigned to the four outbreak scenarios.

Scenario 1: moderate

Scenario 2: low

Scenario 3: very low

Scenario 4: low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

As discussed above, it is feasible that backyard pigs kept in rural areas may come in close contact with nocturnally foraging feral pigs, and that transmission of Aujeszky's disease from one group to the other may result. It is also feasible that some mixing between pigs from an infected backyard herd and other domestic pigs may occur. For example, in the case of speciality breeds or unusual breeds live pigs or semen may be transferred from one herd to another for breeding purposes. Alternatively, pigs raised for personal consumption may be transferred between backyard holdings for growing out or fattening. However, often backyard pigs will be raised for consumption by that household.

Indirect spread by fomites or by mechanical vectors is also feasible. For example, ADV in saliva and/or urine may be transferred from an infected backyard herd to other domestic pigs through inadequately cleaned trucks or footwear. Alternatively, ADV may be transferred on inadequately cleaned and disinfected equipment, such as buckets or ropes, since the clinical signs of this disease are not pathognomonic or sufficiently distinctive to ensure its immediate diagnosis.

It was stated above that were transmission to a piggery to occur, it is likely that the disease would be amplified and spread regionally by fomites, live pigs, semen or other means to other piggeries before diagnosis was made and controls to limit spread were initiated. If large commercial piggeries were also situated within the region it is conceivable that spread to these might occur, and that this would subsequently lead to a more general outbreak.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: very low

Scenario 3: moderate

Scenario 4: low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

In particular, the likelihood of a more rapid diagnosis of Aujeszky's disease was considered higher for small commercial piggeries, since the effects of the disease will likely be more obvious within a bigger herd and, particularly, in a herd that includes breeding sows. In addition, it is more likely that the managers of small commercial piggeries will detect the disease in an early stage, and that the consulting veterinarian will be alerted to the potential of an exotic disease epidemic. Other important considerations include the larger number of live pigs moved between small commercial piggeries than backyard enterprises, and the increased potential for movement of pig semen. Likewise, it is more conceivable that infection would be amplified within a small commercial herd to the extent necessary for transmission via fomites or mechanical vectors.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: low

Scenario 4: moderate

Other susceptible species

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of other susceptible species:

1. Containment of the disease within the sub-population of directly exposed animals - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of domestic pigs - containment within a local population of backyard enterprises or small commercial piggeries - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs - exposure of medium-large commercial piggeries and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Most species other than pigs infected with Aujeszky's disease are generally regarded as incidental hosts, dying within a short period of being infected. There is inconclusive evidence as to the involvement of rats in the spread of ADV, possibly acting as a reservoir for the disease if it is endemic in pigs. Feral pigs would need to consume an acutely infected rat or other susceptible species to be exposed to ADV. Commercial enclosed piggeries generally practice rodent control and have biosecurity measures in place to minimise access by other animals.

On balance, the following likelihhods were assigned to the four scenarios.

Scenario 1: high

Scenario 2: very low

Scenario 3: very low

Scenario 4: very low

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this 'no outbreak' scenario, the disease is most likely to have established amongst the directly exposed animals, and to have run its course without identification. This is because the clinical symptoms of this disease, may be mild and could be confused with endemic diseases.

The direct impact of Aujeszky's disease

Animal life or health

Depending on the strain of ADV a range of clinical signs may be seen, some of which may be very mild. For example, in New Zealand it was estimated that the disease was present for several years before being diagnosed (Pannett, et al., 1999). There can be mortality in young pigs and in other susceptible species.

On this basis, the likely impact of Aujeszky's disease in terms of *animal health* was considered unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

Environment

In this scenario, Aujeszky's disease is contained within the directly exposed group and as such it was considered that its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of Aujeszky's disease

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this outbreak scenario where there is containment of the disease within the directly exposed group and in the case of pigs the clinical signs of disease can be mild or non-specific it was considered unlikely that the primary case would be diagnosed. In the case of other susceptible species, the number of animals showing clinical signs of disease would likely be small and the cause of death or disease likely to be undiagnosed. Given this, it was considered likely that the indirect impact of new or modified control programs would be undiscernible at any level, and the rating assigned to this criterion was therefore 'A'.

Domestic trade or industry effects

As discussed above, it was considered unlikely that disease would be diagnosed in a single herd or an individual or small group of other susceptible species. On this basis, the indirect impact of Aujeszky's disease on domestic trade and industry was considered unlikely to be discernible at any level, and a rating of 'A' was thus assigned to this criterion.

International trade effects

As the disease is unlikely to be diagnosed in a single herd, it was considered that the indirect effect of Aujeszky's disease on international trade was unlikely to be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on the environment

Aujeszky's disease is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, the disease is most likely to have established within a population of feral pigs, and to have run its course without identification. As explained above, this is because the clinical symptoms of this disease can be mild.

The direct impact of Aujeszky's disease

Animal life or health

As with the first scenario, the impact on animal health was considered unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

Environment

In this scenario, Aujeszky's disease spreads to feral pigs but not to other susceptible species such as native carnivorous or omnivorous animals. In view of this, it was considered that the direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of Aujeszky's disease

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

It is likely that the disease would remain undiagnosed if contained within a more general population of feral pigs due to the nature of the disease and limited opportunities for close observation of feral pigs. Feral pigs harvested for meat are mature animals and unlikely to be showing marked clinical signs of Aujeszky's disease. Accordingly, the consequences for this criterion would be similar to that described above for the first scenario, resulting in a rating of 'A'.

Domestic trade or industry effects

In this outbreak scenario Aujeszky's disease spreads to a more general population of feral pigs but not to domestic pigs. As the disease may likely remain undiagnosed within feral pigs for a significant period of time the indirect effects on domestic trade and industry would be unlikely to be discernible at any level. Overall, this resulted in a rating of 'A' for this criterion.

International trade effects

As described above, the indirect effect of Aujeszky's disease on international trade was considered unlikely to be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on the environment

Aujeszky's disease is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, the disease has established within a local population of small commercial piggeries or backyard enterprises and spread to other susceptible species. It is likely that the disease would be diagnosed and contained through an eradication program.

The direct impact of Aujeszky's disease

Animal life or health

The third scenario is characterised by spread of Aujeszky's disease to a local population of domestic pigs, but containment within this population, and spread to other susceptible species. Other susceptible domestic species would be affected generally on an individual basis. Due to the potential for spread within a herd and mortality associated with young pigs, it was considered that the direct impact on animal health would be unlikely to be discernible at the national or State level, but of minor importance at district or regional level. This gave the disease a rating of 'C' for this criterion.

Environment

In the scenario, Aujeszky's disease spreads to a local population of domestic pigs and other susceptible animals, which may include native Australian animals such as dingoes. Generally individual animals are infected, however, infection in dogs is generally fatal. In light of this information, the direct impact on the environment was considered unlikely to be discernible except at the local level. Hence, the rating assigned to this criterion was 'B'.

The indirect impact of Aujeszky's disease

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

If identified in Australia, ADV would require a control, eradication and compensation program. Aujeszky's disease is listed as Category 4 under the Cost Sharing Agreement. In this agreement the government and livestock industries have agreed to share costs based on the conduct of an agreed response plan for an outbreak of a disease that falls within one of four categories. Category 4 diseases are funded 20% by governments and the remaining 80% by the relevant industry. AUSVETPLAN recommends that disease be eradicated as quickly as possible. In this scenario, where Aujeszky's disease has only limited spread within the domestic pig population (a local population of backyard or small commercial piggeries), it is considered that the disease would be eradicated promptly.

There would need to be some surveillance of the domestic pig population and feral pig population. If the disease was present in the feral pig population, depopulation may be feasible in the case of a localised outbreak. If the disease was unable to be eradicated in the feral pig population, individual farmers would need to improve biosecurity by appropriate fencing. Close contact between feral pigs and domestic pigs is required for spread of the disease between these populations.

Overall the indirect impact of new or modified control programs was considered unlikely to be discernible at the national or State level, and of minor importance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

Domestic trade or industry effects

Under AUSVETPLAN, the diagnosis of Aujeszky's disease in a local population of domestic pigs would result in restrictions on the movement of breeding pigs and semen from infected premises and those within a 10 km radius. This is unlikely to affect small commercial piggeries or backyard piggeries significantly. Pigs would be permitted to go direct to an approved abattoir for immediate slaughter.

With the detection of an exotic disease in Australia it is likely that consumers may initially decrease pork consumption. A publicity campaign may need to be undertaken to reassure the public that there are no health concerns.

When these issues were taken into account, the indirect impact of ADV on domestic trade and industry was considered unlikely to be discernible at the national level, but of minor importance at the State level. This resulted in a rating of 'D' for this criterion.

International trade effects

The diagnosis of Aujeszky's disease in domestic pigs, even within a local population, would likely result in initial cessation of trade in live pigs and semen to some markets. Australia exports only small numbers of breeding pigs and quantities of semen. Trade should only be interrupted temporarily under this scenario with testing of pigs and semen donors for Aujeszky's disease prior to export an option.

With a limited small outbreak it is unlikely that export of meat would be significantly disrupted. There may be an initial reaction from some trading partners to halt meat imports in the short term, however, as there is no human health risk and the disease is endemic in some markets, trade should resume quickly. The OIE does not consider risk management measures for meat are warranted in regard to Aujeszky's disease but recommends measures with regard to offal. The export trade in offal would be affected, however, Australia exports only small quantities of this commodity.

After consideration of these issues, the indirect effect of ADV on international trade was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

Indirect impact on the environment

Aujeszky's disease is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, Aujeszky's disease would have established in a broader population of commercial piggeries (including medium-large piggeries) and spread to other susceptible species. A control and eradication program would have been mounted in response to the diagnosis of the disease in pigs or any other affected animal.

The direct impact of Aujeszky's disease

Animal life or health

In this scenario, Aujeszky's disease has spread within the domestic pig population. The clinical signs would include neonatal mortality, respiratory disease and reproductive disorders within piggeries. Production would be reduced.

In the United States of America, the estimates of the annual economic impact of Aujeszky's disease range from US $21 to nearly $33 million, although there is considerable variation in the severity of losses in different years (Bech-Nielsen, et al., 1995). In contrast, in New Zealand it has been stated that there was no significant direct economic impact of Aujeszky's disease in the North Island, although New Zealand undertook an eradication program[45].

Based on this information, it was considered that the direct impact on animal health would be unlikely to be discernible at the national level, but of minor importance at the State level. This gave the disease a rating of 'D' for this criterion.

Environment

In the scenario, Aujeszky's disease spreads to other susceptible animals, which may include native Australian animals such as dingoes. Generally individual animals are infected, however, infection in dogs is generally fatal. In light of this information, the direct impact on the environment was considered unlikely to be discernible except at the local level. Hence the rating assigned to this criterion was 'B'.

The indirect impact of Aujeszky's disease

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

As stated in the above scenario, diagnosis of Aujeszky's disease in Australia would require a comprehensive control, eradication and compensation program. Infected premises and those in the restricted area would be subject to quarantine and movement controls. The recommended strategy is immediate depopulation with salvage through abattoirs. Nonetheless, it is likely that stock unsuitable for slaughter such as older sows would need to be disposed of on-farm.

[45] MacDiarmid SC (1999) Pers. Comm. (AQIS T87/1670)

Vaccination may be considered for breeding animals or alternatively if the disease establishes. Biosecurity would include measures to prevent contact with wild pigs, eliminate or exclude rodents and prevent the migration of rodents to other premises. If the disease was widespread eradication may be prolonged and costly to producers and governments. In the United States of America, the State-Federal-Industry Pseudorabies eradication program has been in effect since 1989.

There would be surveillance of the domestic and feral pig population. If the disease was detected in the feral pig population, eradication may be feasible if localised but otherwise would be difficult.

On this basis, it was considered that the likely indirect impact of new or modified control programs would be unlikely to be discernible at the national level, and of minor importance at the State level. Overall this resulted in a rating of 'D' for this criterion.

Domestic trade or industry effects

The presence of ADV within commercial piggeries would result in restrictions on the movement of pigs and semen within the restricted area. Pigs would still be able to move for slaughter and trade in meat would not be restricted in Australia. Replacement breeding stock would need to be purchased following decontamination of the infected piggery.

When these issues were collated, the indirect impact of Aujeszky's disease on domestic trade and industry was considered unlikely to be discernible at the national level, and of minor importance at the State level. This gave the disease a rating of 'D' for this criterion.

International trade effects

As discussed above under scenario 3, trade in pigs and semen may be halted following detection of Aujeszky's disease although this is likely to be temporary. It is likely that a few individual consignments would be affected while conditions were renegotiated.

Australia's major markets for pig meat are Singapore and Japan. Both these markets would be sensitive to any significant disease outbreak involving the commercial pig population. It is likely that trade may be temporarily halted while reassurances were provided that there were no public health implications. Singapore does not have a pig industry, and Japan has Aujeszky's disease, so there should be no animal health disease concerns. The export trade in offal would be affected, however, Australia exports only small quantities of this commodity.

In light of this information, it was considered that the indirect impact of Aujeszky's disease on international trade was unlikely to be discernible at the national or State level, and of minor significance at the district or regional level. Overall, this gave the disease a rating of 'C' for this criterion.

Indirect impact on the environment

Aujeszky's disease is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

If Aujeszky's disease became established there would be significant ongoing production costs for producers, affecting the viability of some and damaging to their communities. The pig industry is important to the economies of several localities and districts in New South Wales,

Victoria, Queensland, Western Australia and South Australia. It has been estimated that in general terms, for every one employee working in the pig industry, there will be another two people employed in providing goods and services to the pig industry directly or to employees in the pig industry.

It is clear that the viability of some producers would be affected if there was a widespread outbreak of Aujeszky's disease or if the disease became established within the pig industry. Where the pig industry was highly significant to the local economy, aspects of these communities may be threatened.

Taking these issues into consideration, the indirect impact of Aujeszky's disease on rural communities was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. This resulted in a rating of 'C' for this criterion.

The overall impact of Aujeszky's disease

When the direct and indirect impacts of Aujeszky's disease were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences negligible

Scenario 3: Consequences low

Scenario 4: Consequences low

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 38, Table 39, Table 40, and Table 41. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were considered 'very low', 'low' and 'low' respectively. The likely consequences associated with the exposure of other susceptible species to infected pig meat scraps were considered 'negligible'.

Table 38 Aujeszky's disease: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Very low	Low	Negligible
Scenario 4	Low	Low	Very Low
		Overall likely consequences	**Very Low**

Table 39 Aujeszky's disease: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Moderate	Low	Low
Scenario 4	Low	Low	Very Low
		Overall likely consequences	**Low**

Table 40 Aujeszky's disease: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Low	Low	Very low
Scenario 4	Moderate	Low	Low
		Overall likely consequences	**Low**

Table 41 Aujeszky's disease: summary of the consequences of exposure of other susceptible species

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	High	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Very low	Low	Negligible
Scenario 4	Very low	Low	Negligible
		Overall likely consequences	**Negligible**

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with ADV.

Table 42 shows the results obtained for each of the exposure groups and for the overall annual unrestricted annual risk. Since the overall annual risk is clearly higher than Australia's ALOP (very low), risk management would be required for ADV.

Table 42 Aujeszky's disease: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Very low	High	Very Low	Very low
Backyard pigs	Very low	Very low	Low	Negligible
Small commercial piggeries	Very low	High	Low	Low
Other susceptible species	Very low	Moderate	Negligible	Negligible
			Overall annual risk	**Low**

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Porcine reproductive and respiratory syndrome virus

Technical information

Background

The syndrome that later became known as porcine reproductive and respiratory syndrome (PRRS) was first observed in the United States of America during the late 1980s (Goyal, 1993), and in Europe in 1990 (Wensvoort, 1993). It is characterised by outbreaks of varying degrees of reproductive loss and respiratory disease (Rossow, 1998). The disease has spread throughout much of the pig-producing areas of the world, causing significant production losses.

To date, PRRS has been identified in the United States of America, most Member States of the European Union, Canada, Malta, Russia, the Philippines, the Republic of Korea, Taiwan and Japan. Countries that are reportedly free from PRRS include Australia (Garner, et al., 1997), New Zealand (Animal Biosecurity, 1996), Norway (Norwegian Animal Health Authority, 2000), Finland (Veijalainen & Tapiovaara, 2000), Sweden (Orava, 2001) and Switzerland (Canon, et al., 1998).

Agent taxonomy

PRRS virus belongs to the newly defined *Arteriviridae* family. This family, which also contains equine arteritis virus, simian haemorrhagic fever virus and lactate dehydrogenase-elevating virus of mice, was combined with the *Coronaviridae* family to form the *Nidovirales* order (Meulenberg, 2000).

Agent properties

PRRS virus is a small, enveloped, positive strand RNA virus. Strains of differing antigenicity and varying pathogenicity exist. The survival of viruses outside a living host is affected by factors that include the substrate, pH, temperature, relative humidity, and exposure to ultraviolet light.

The European strain of PRRS virus appears to be stable for at least 72 hours when chilled at 4°C or frozen at -20°C, although 93% of infectivity was lost after storage of tissue samples at 25°C for a similar period (Van Alstine, et al., 1993). American studies using aliquots of virus samples in Eagles's minimum essential media have indicated that the United States strain of PRRS virus is stable at -70°C for at least 18 months and at 4°C for at least 1 month, while viability is reduced by 50% after storage at 37°C for 12 hours. Complete inactivation of the virus occurred within 48 hours at 37°C and by 45 minutes at 56°C (Benfield, et al., 1992).

Another study reported that in culture medium at pH 7.5, the half-life of the European strain of PRRS virus was 140 hours at 4°C, 20 hours at 21°C, 3 hours at 37°C and 6 minutes at 56°C (Bloemraad, et al., 1994). Rapid alterations in pH decreased the half-life. Bloemraad and co-workers concluded that PRRS virus is most stable between pH values 5.5 and 6.5. These results concur with those of others (Benfield, et al., 1992) who found that virus infectivity was reduced by over 90% at a pH less than 5 or greater than 7.

Host range

Porcine reproductive and respiratory syndrome has been reported only to occur naturally in pigs.

Epidemiology

In 1991, a causative agent for PRRS was isolated in the Netherlands and was initially named Lelystad virus (Wensvoort, et al., 1991). In the same year, a similar virus was isolated in the United States of America (Collins, et al., 1992). It was subsequently shown that PRRS virus existed as a number of distinct strains and that although the groups of strains from North America and Europe shared similar morphological and physico-chemical characteristics, they were antigenically distinct (Wensvoort, et al., 1992; Nelsen, et al., 1999). Some strains isolated in North America are closely related to the Lelystad virus and the use of modified live vaccines, based on American strains of PRRS virus, in Europe, has resulted in American strains occurring in Europe (Bøtner, et al., 1997). Antigenic differences between isolates appear to be associated with variable disease syndromes (Halbur, et al., 1996; Norwegian Animal Health Authority, 2000).

Typically, PRRS virus spreads rapidly within herds. The rate of transmission of PRRS virus infection between herds is dependent on factors including herd density, size and biosecurity, with area spread being slowest in areas of low pig density and where there are few pig movements between herds. Infected pigs shed virus in saliva, urine, semen, and mammary secretions, and direct contact with an infected pig is the primary means of transmission of the PRRS virus (Benfield, et al., 1999). However, aerosol transmission of the PRRS virus, particularly in conditions of high humidity, low wind-speed and low ambient temperature, has been reported (Mortensen, et al., 2002).

Serological evidence of PRRS virus infection has been reported in wild boar in France (Albina, et al., 2000) and Oklahoma, United States of America (Saliki, et al., 1998). However, none of the 20 feral pigs tested from November 1993 to February 1994 in Kansas, United States of America, were seropositive to the PRRS virus (Saliki, et al., 1998).

The role of fomites is uncertain although it is known that the virus is excreted in the urine and faeces of affected animals (Yoon, et al., 1993). Experimentally, transmission of PRRS virus to each other and thence to pigs by mallard ducks has been reported (Zimmerman, et al., 1997) although this has not been substantiated in either field observations or epidemiological studies. Further efforts to investigate the susceptibility of non-swine species to PRRS virus have been described (Wills, et al., 2000). The ability of PRRS virus to infect and replicate in dogs, cats, skunks, raccoons, rats, mice, opossums, sparrows and starlings was investigated. The authors note that the results do not support the hypothesis that the animals tested are likely hosts or reservoirs of PRRS virus. It has been shown (experimentally) that mechanical transmission of PRRS virus from viraemic to susceptible pigs via mosquitoes (Otake, et al., 2002b), needles (Otake, et al., 2002a) and houseflies (Otake, et al., 2003) may occur. A survey of rats and mice collected from pig sheds during epidemic and endemic phases of PRRS has indicated that rodents are not a reservoir for the disease (Hooper, et al., 1994).

Transmission of PRRS virus via artificial insemination of semen from infected boars has been demonstrated (Yaeger, et al., 1993; Gradil, et al., 1996; Mortensen, et al., 2002). PRRS virus has been found in semen up to 92 days post-infection (Christopher-Hennings, et al., 1995). PRRS virus may also be transmitted in-utero to live-born and stillborn piglets (Christianson, et al., 1993).

There are many reports of chronic or persistent infection of pigs with PRRS virus, up to 157 days post-infection, in spite of the presence of measurable anti-PRRS virus circulating antibody (Wills, et al., 1997; Zimmerman, et al., 2000; Allende, et al., 2000; Horter, et al., 2002).

However, viraemia is generally much shorter. Virus has been detected in serum for up to 35 days post-infection in 38 of 60 pigs by virus isolation (Horter, et al., 2002).

In one study involving four 4-week-old pigs, PRRS virus was intermittently isolated from the sera of two pigs for up to 23 days post-infection but was isolated from the oropharynx of three of the four pigs at 87 days post-infection, and from one of the four pigs at 157 days post-infection, in an experimental period of 213 days (Wills, et al., 1997). A much larger study has been reported where sixty 3-week-old pigs were inoculated with PRRS virus. A number of these pigs were periodically euthanised between 63 and 105 days post-inoculation and examined for the presence of infectious PRRS virus in serum and tissues (oropharyngeal scrapings or tonsillar tissue), using virus isolation and swine bioassays. Infectious virus was detected from 100% (12/12) of those pigs necropsied at 63 days post-inoculation and from 90% (10/11) of those necropsied at 105 days (Horter, et al., 2002). In another study, 10 pigs were inoculated at 1 to 2 months of age with PRRS virus, monitored for 150 days, then euthanised (Allende, et al., 2000). One pig was shown to be infected at 84 days post-infection via virus isolation. However, using the bioassay technique five pigs were shown to harbour PRRS virus at 84 days and two pigs at 150 days.

Another study followed 28 pigs from inoculation at 35 days of age to euthanasia 251 days later (Wills, et al., 2003). Serum and tonsil biopsy samples were collected periodically, and tonsil, lymph node and lung samples at euthanasia. Samples were examined for the presence of virus or viral RNA by several techniques. Viraemia was evident at 14 days in 20/28 pigs, but in only 2/28 pigs at 28 days post-inoculation. Virus could be isolated from tonsillar tissue from 23/28 pigs at 14 days, 9/28 at 28 days, and 4/28 pigs at 56 days post-inoculation. In general, detection of viral RNA by RT-PCR was more sensitive than viral isolation in detecting infected pigs, although the authors recognised that positive RT-PCR results do not necessarily indicate the presence of viable virus. Viral RNA was detected in 20/28 (71%) of tonsillar samples on day 84 but from only 1/28 (4%) on day 119 post-inoculation. In addition, serum or tonsillar tissue was RT-PCR positive on at least one occasion between days 119 and 251 in nine of the 28 inoculated pigs. Bioassays performed on tissues collected at necropsy at 251 days from all 28 pigs and one control pig were all negative.

In contrast to the above studies where very young pigs were experimentally infected, one study examined the persistence of PRRS virus in breeding age gilts. One hundred and twenty 4-month-old gilts were inoculated with PRRS virus, and 30 of them were subsequently tested for presence of virus in serum by virus isolation and PCR. Virus was detected by virus isolation and PCR up to 14 days post-infection but not at 30 days. Forty of the pigs were slaughtered at 120, 150 and 180 days post-infection and tissues collected for virus isolation and PCR. All pigs were negative for PRRS virus at slaughter as were sentinel pigs housed in contact with them (Batista, et al., 2002).

The possibility that persistently infected pigs might, when stressed, become viraemic has been addressed. Transmission of PRRS virus to contact pigs by two 22-week-old pigs that had been infected in utero, and subjected to both transport stress and exogenous corticosteroids, has been demonstrated (Albina, et al., 1994). Shipping stress alone did not induce shedding or viraemia in two adult boars that had been experimentally-infected 106 days previously (Christopher-Hennings, et al., 1995).

Prevalence studies

The National Animal Health Monitoring System (NAHMS) study showed that in the United States of America in 1995, the overall between herd prevalence, in herds in which a PRRS vaccine was not used, was 59.4% (129/217) (USDA:VS, 1997). It has been estimated that approximately 80% of Canadian pig herds are infected (Dewey, 2000). A high prevalence of PRRS virus infection between herds has also been reported for Venezuela (86%) (Rolo, et al., 1998) and the Republic of Korea (87%) (Han, et al., 1998).

In Europe, PRRS virus infection is also very prevalent. PRRS virus infection is reported to be endemic amongst breeding herds in the Netherlands, as illustrated by a report in which 31 of 32 (96.9%) breeding farms examined in 1995 contained serologically positive pigs (Nodelijk, et al., 1997). PRRS virus is believed to be widespread throughout Italy where serological evidence of infection was found in 37 of 39 (94.9%) typical pig farms located in the Po valley (Sala, et al., 1998). Seven of ten (70%) large breeding herds of pigs in Croatia were seropositive for PRRS virus (Lipej, et al., 1998). In contrast, in the Pays de la Loire region of France, in which there is a medium density of pig farms, limited movements of pigs between farms, and a commitment towards control and eradication of PRRS from the area, the herd prevalence has been held below the 2.7% level recorded in 1993 (Le Potier, et al., 1997).

The prevalence of PRRS virus within an infected herd is affected by such things as production systems, farm biosecurity, and localisation (or not) of circulating virus to particular production units on the farm. Farms that have continuous-flow production systems may find that older pigs infect young pigs as they enter shared facilities.

In PRRS-infected Danish pig herds, the seroprevalence to PRRS virus amongst finisher pigs typically is very high (Mortensen, et al., 2001). All of 158 serum samples (collected at slaughter between December 1997 and February 1998) from finisher pigs from five PRRS-infected herds were seropositive for PRRS virus. These authors also refer to an (unpublished) larger study of 1,603 infected herds in which an overall within-herd seroprevalence of 83% was calculated. Similarly, another worker (Wang, 1999) reported that 205 of 240 serum samples (85.4%), collected at slaughter from finisher pigs in Taiwan between October 1995 and March 1996, were seropositive for PRRS virus. If it is assumed that some pigs infected soon after weaning revert to seronegative status by slaughter age, then these results are consistent with an estimate of 100% exposure of pigs to PRRS virus, given that the virus is circulating within the herd (Blanquefort & Benoit, 2000).

Clinical signs

The nature and severity of clinical signs associated with PRRS infection vary considerably and are affected by the age of the pig at infection, the strain of virus, the presence of other infectious agents, genetic susceptibility of the pig, and environmental and management factors (Done & Paton, 1995; Done, et al., 1996; Rossow, 1998).

In immunologically naïve herds, all ages of pigs are susceptible to PRRS virus infection. Clinical signs of acute infection include inappetence, fever and dyspnea. Sows may farrow prematurely and affected litters born early, full term or late may be composed of the following: stillborn pigs, mummified foetuses, late term dead foetuses, variably sized weak-born pigs, and variably sized, apparently normal pigs. Preweaning mortality is high.

In endemically infected herds, clinical signs are most severe in younger pigs, and include ill thrift, dyspnea, exacerbation of other endemic diseases, and increased mortality. Clinical signs

may be mild or inapparent in pigs infected in the finishing stages. Clinical signs, when present in slaughter-age pigs, are reported to include transient fever and inappetence (Rossow, 1998). It has been suggested that infection with PRRS virus is inapparent in most finishing herds (Done, et al., 1996). Nevertheless, particularly in herds that have recently become infected, influenza-like signs may be observed in finishing pigs (Albina, et al., 1994).

Pathogenesis

PRRS virus gains access to its host via mucosal surfaces, after which replication occurs in local macrophages with subsequent viraemia and distribution to regional lymphoid tissues. PRRS virus has a tropism for macrophages. It has been shown that the virus replicates mainly in macrophages of the lymphoid tissues and lungs in the acute phase of infection and persists in lung macrophages (Duan, et al., 1997). Other workers (Thanawongnuwech, et al., 2000) agree that the lymphoid and respiratory system are probably the major sites for PRRS virus replication in the pig, but also note that PRRS antigen has been found, on occasion (by others), in the resident macrophages of a variety of tissues as well as other cells including muscle tissues.

Pathology

Gross lesions are usually observed in only a few organ systems such as the respiratory and lymphoid and are most marked in neonatal and nursery pigs. In these cases, PRRS virus infected lungs are mottled tan and red and fail to collapse. Lymph nodes are moderately to severely enlarged and tan in colour.

The gross pathology observed after uncomplicated infection of PRRS in finishing pigs may be unremarkable (Rossow, 1998). Lesions may be limited to some consolidation of the cranial lobe of the lungs, accompanied by slightly or moderately enlarged lymph nodes, particularly the tracheobronchial nodes (Done & Paton, 1995). Under field conditions, most PRRS virus infected pigs are co-infected with one or more pathogens, which complicates the diagnosis of PRRS based on pathology.

Immunology

The immune response that develops following infection with the PRRS virus protects the clinically-recovered pig from subsequent challenge, but does not prevent the establishment of persistent infection (Benfield, et al., 1999).

Vaccines have been developed and are widely used; however, the use of modified live vaccines, based on American strains of PRRS virus, in Europe, has resulted in American strains occurring in Europe (Bøtner, et al., 1997).

Transmission via meat

The tissue tropism of the PRRS virus for macrophages suggests that virus present in meat is likely to be derived from associated lymphoid tissues, or from blood perfusing the tissues. PRRS virus has been isolated from muscle of experimentally-infected, viremic pigs (Bloemraad, et al., 1994; Mengeling, et al., 1995; van der Linden, et al., 2003). PRRS virus has also been detected in commercially-packaged pork (Magar, et al., 1995; Frey, et al., 1995b), but only rarely and at very low titres (Frey, et al., 1995a; Larochelle & Magar, 1997).

In one study, low levels of PRRS virus were recovered from muscle of experimentally infected viraemic pigs slaughtered 5 and 10 days post-infection. PRRS virus was isolated from some samples of muscle 0 and 24 hours after slaughter ($10^{2.8}$ to $10^{3.7}$ $TCID_{50}$/g) (Bloemraad, et al., 1994). In another study in which 21 pigs were exposed to PRRS virus, the virus was isolated from the ham muscles of only one pig, slaughtered 7 days post-infection (Mengeling, et al., 1995). PRRS virus (both European and American strains) has also been demonstrated in pooled samples of ham muscle and bone marrow in pigs slaughtered 6 days post-infection (Frey, et al., 1995b). A further experiment detected PRRS virus in muscle samples collected 7 days post-infection from 2 pigs, but not at 14 days (Magar, et al., 1995).

Several groups have investigated the presence of PRRS virus in commercially slaughtered pork. One group examined 1049 sample pools taken from 178 lots of fresh pork (40,000 lbs per lot) for PRRS virus, finding 6 of the sample pools positive for virus (Frey et al 1995a). The levels of virus in the positive samples were low, because most isolates were only obtained after multiple cell culture passage and re-isolation was not always successful. In another study, muscle samples were collected from 44 abattoir pigs derived from seropositive herds. No virus was isolated and no viral antigens detected by immunogold silver staining (Magar, et al., 1995). This same research group subsequently expanded the study examining, by virus isolation, 73 lots of frozen packaged pig meat, each composed of 6 pools of meat samples. Meat samples were also tested by reverse transcription - polymerase chain reaction (RT-PCR) (Larochelle & Magar, 1997). All samples were negative by both virus isolation and RT-PCR. For that reason, the investigators concluded that pig meat does not retain detectable amounts of PRRS virus.

The effects of cold storage on the persistence of the PRRS virus in meat have also been examined. PRRS virus was detected from all three pooled samples of ham muscle and femoral bone marrow (obtained from pigs slaughtered 6 to 7 days post-inoculation) and stored at 4°C for up to18 days post-inoculation. One pooled sample (from six pigs) contained detectable virus after storage at 4°C for at least 28 days. However, virus could not be isolated from one of two pooled samples (each from three pigs) after 25 days of storage, nor from either of these two pooled samples after 32 days of cold storage (Frey, et al., 1995a). Other workers (Bloemraad, et al., 1994) attempted viral isolation on muscles from two pigs slaughtered at 5 and 10 days post-inoculation, respectively. Virus was isolated from the muscles of the pig slaughtered at 5 days post-inoculation after 0 and 24 hours storage at 4°C, but not after 48 hours.

The virus appears to be stable in meat frozen for prolonged periods. The virus was detected from three of three pooled samples of ham muscle and femoral bone marrow (obtained from pigs slaughtered 6 to 7 days post-inoculation) and stored at -20°C for 28 to 32 days post-inoculation (Frey, et al., 1995a). Similarly, other workers have determined that meat frozen for 13 to 14 days retains infectivity when fed to susceptible pigs, even when the initial levels of virus were below the level of detection by virus isolation (van der Linden, et al., 2003).

The transmission of PRRS virus to pigs via the ingestion of infected meat has been confirmed by research commissioned by Biosecurity Australia and performed by Lelystad ID-DLO, the Netherlands. Twenty-four 8-week-old pigs were infected by intranasal inoculation with either a European or American strain of PRRS virus (12 pigs per group). The pigs were all viraemic 5 days post-inoculation (serum virus titres $10^{2.3}$ to $10^{4.8}$ $TCID_{50}$/ml). The pigs were slaughtered 11 days post-inoculation and the semimembranosus muscle was assayed to determine PRRS viral titres. Virus was detected in the semimembranosus muscle from seven of twelve pigs infected with the European strain and from five of 12 pigs infected with the American strain ($10^{3.3}$ to $10^{4.3}$ $TCID_{50}$/g). The muscle was frozen until use in the feeding experiment. Muscle virus titres were determined prior to feeding. In most samples, muscle virus titres decreased

following freezing (below $10^{1.8}$ to $10^{3.8}$ $TCID_{50}$/g). Muscle samples were positive for viral RNA with the exception of one sample.

Five hundred grams of raw semimembranosus muscle from each of the experimentally infected pigs was fed over a two day period (250 g/d) to each of two receiver pigs (48 receiver pigs). Sera were collected for virus isolation and antibody detection for three weeks post-feeding. Oral transmission of the European and American strains of PRRS virus to receiver pigs via the feeding of meat was demonstrated. In addition, there was evidence of horizontal transmission, with sentinel pigs in contact with the receiver pig becoming viraemic (van der Linden, et al., 2003).

The oral infectious dose for PRRS virus has not been determined, however, the Lelystad study showed that meat containing virus at titres less than the limit of detection of the assay ($10^{1.8}$ $TCID_{50}$ per gram tissue) was infectious when fed to pigs. The pigs received 250 grams of infected meat at each of two feedings, thus the oral infectious dose is probably less than $10^{4.5}TCID_{50}$.

Release assessment

R1 — the likelihood that a source herd is infected

In those countries in which PRRS virus is endemic, infection is generally widespread as demonstrated by serological surveys. The between herd prevalence of PRRS virus infection has been reported as ranging from 60% to 97%. Given this, the likelihood that slaughter-age pigs have been selected from an infected herd in a country where PRRS is endemic was considered to be ‘high’.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

The seroprevalence of PRRS virus infection in finishing herds has been reported as ranging from 85 to 100%. Typically, pigs are infected soon after weaning, however, it has been reported that some pigs may harbour virus for at least 157 days, although the proportion that are persistently infected generally decreases over time. For example only one of four pigs was still infected at 157 days and two of ten pigs at 150 days. Nonetheless, in one experimental study 11 of 12 pigs were still infected 105 days post-inoculation. Recently, it has been suggested that although pigs can remain persistently infected for several months, most pigs clear the virus between three and four months after infection (Wills, et al., 2003). In this study none of 28 pigs was still infected at 251 days post-inoculation as determined by swine bioassay. It is also recognised that infection may occur later in the finishing herd, particularly if a previously-negative farm has just experienced an outbreak of PRRS (Albina, et al., 1994; Dee, et al., 1998). It has also been shown that stress, such as regrouping and transport, may lead to re-excretion of the virus.

A study conducted in Canada with slaughter-age pigs determined that of 1039 blood samples and 1027 meat samples 4.3% and 1.9% were positive for PRRS virus respectively as determined by PCR[46].

On the basis of this information, it was considered that the likelihood of selecting an infected pig in an infected herd was ‘low’.

[46] Personal communication from Dr Brian Evans Chief Veterinary Officer, Canadian Food Inspection Agency

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

The clinical signs of PRRS vary with the stage of infection in the individual pig. Most recent infections in slaughter-age pigs are subclinical. Persistently infected pigs generally show no clinical signs. Pigs that are recently infected may show signs of fever, and respiratory disease. Pathological changes in infected slaughter-age pigs tend to be mild, limited to the lungs and associated lymph nodes, and at most may result in the condemnation of the lungs but not the associated carcass.

Considering this, the sensitivity of ante-mortem, slaughter and processing procedures in detecting and removing pigs infected with PRRS virus was considered ‘extremely low’.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with PRRS virus and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a ‘one minus the background rejection rate’. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered ‘extremely low’. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

The tissue tropism of the PRRS virus for macrophages suggests that virus present after division of the carcass is likely to be derived from lymphoid tissues associated with the product, or from blood perfusing the tissues rather than muscle tissue *per se*. It is known that PRRS virus may be present in meat harvested from a viraemic pig, and that virus within meat may initiate infection if consumed by a naïve pig. It has also been shown that PRRS virus can on occasions be present in commercially packaged pork. Virus or viral antigen has also been isolated from oropharyngeal scrapings or tonsillar tissues from a proportion of pigs up to 157 days post-infection. It is not known if virus can be isolated from other lymphoid tissues for extended periods.

In view of the above factors, and that fact that a carcass will contain lymphoid tissue and may include the head and hence contain residual tonsillar and other associated lymphoid tissues, it was considered that the likelihood that PRRS virus would be present in meat harvested from an infected pig was ‘moderate’.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

PRRS virus is most stable between pH values 5.5 and 6.5. For the purposes of this IRA, meat is not assumed to reach a pH lower than 6.2. Thus, it was considered that there was a 'high' likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

Although the proportion of viable virus may be reduced by cold storage, both the European and American strains of PRRS virus are known to be stable for extended periods when stored at 4°C or frozen. In view of this, it was considered that there was a 'high' likelihood that meat infected at the completion of carcass maturation would remain infected during transport and cold storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a 'very low' likelihood that imported pig meat derived from an individual carcass will be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit will be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

The likelihood assigned to this step will be determined by the load of viable virus in discarded meat scraps, and its virulence and infectivity. The oral infectious dose for PRRS virus has not been determined, however, it is known that 500 grams of meat derived from a pig recently infected with PRRS virus provided a sufficient oral dose to infect a naïve pig. In this experiment, meat derived from some of the pigs contained virus at titres less than the limit of the detection of the assay ($10^{1.8}TCID_{50}$/gram tissue). It is not known if ingestion of smaller quantities of meat would have resulted in infection. In commercially packaged pork PRRS virus has been found, but only at very low titres. Nonetheless, it is clear that a feral pig could consume 500 grams of meat relatively easily.

The oral infectious dose of PRRS virus is unknown. One study reported that as few as 10 virions by intranasal inoculation were sufficient to achieve infection (Yoon, et al., 1998), indicating that the disease is highly infectious, at least by that route.

When these factors were combined, the likelihood that a waste unit from an infected pig would contain a sufficient dose of PRRS virus to initiate infection was considered to be ‘high’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

This step describes the sensitivity of PRRS virus to ultraviolet light, to ambient temperatures ranging from 10°C to 35°C and to the putrefying effects of saprophytic organisms. PRRS virus has been characterised as a fairly labile virus. It is known that within 48 hours at 37°C, PRRS virus is inactivated. It is also known that the proportion of viral particles surviving a given period of exposure is greater at a lower temperature. It is anticipated that the effect of exposure to the environment (heat, ultraviolet light, and desiccation) would reduce the survival time for the virus still further. In the light of this information, it was considered that the likelihood that PRRS virus would survive within meat scraps discarded in refuse for the period of time required for feral pigs to locate and subsequently scavenge the material was ‘low’.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = High

- Rural regions = Moderate
- Large towns = Extremely low

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be ‘high’.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a ‘very low’ likelihood that a waste unit will be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was ‘high’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a ‘moderate’ likelihood that PRRS virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be ‘low’.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a ‘very low’ likelihood that a waste unit will be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a 'moderate' likelihood that PRRS virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'high'.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the three exposure groups - that is, feral pigs, pigs in backyard enterprises and pigs in small commercial piggeries. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and

- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

PRRS is a highly infectious disease for which the primary means of spread is the infected pig. Transmission occurs principally as a result of close contact (including mating and artificial insemination) between infected and susceptible animals, although aerosol transmission may occur over short distances in conditions of high humidity, low wind speed and low ambient temperature. The role of fomites in the transmission of the disease is uncertain, although the virus has been detected in saliva, urine and faeces. The rate of spread of PRRS virus is greatest in high density populations of susceptible animals. Persistent infection is a feature of PRRS which would assist in the spread of the disease.

The likely signs of PRRS in a pig herd include abortions, stillbirths and the birth of weak or sickly piglets. In young pigs, respiratory disease may be severe, with less pronounced clinical signs in older pigs including subclinical infection.

Feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. Feral pigs are widespread in Australia and there is very limited close contact with domestic pigs. However, there have been several reported cases of feral pigs gaining access to piggeries, particularly outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves. In either case, it is conceivable that nocturnally foraging feral pigs may be attracted to an enclosure housing domestic pigs, and that while mixing *per se* is unlikely, contact sufficient for the transmission of PRRS virus may occur.

Serological surveys of wild boars have demonstrated that PRRS virus infection is present in some populations (Albina, et al., 2000), although it would appear at a low prevalence (3.6%).

If transmission to a backyard or small commercial piggery occurred, it is likely that the disease would be amplified and spread regionally by live pigs, semen, or other means to other such piggeries before diagnosis was made and controls to limit spread were initiated. If large commercial piggeries were also situated within the region, spread to these might occur, and that this may subsequently lead to a more general outbreak.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: very low

Scenario 2: moderate

Scenario 3: low

Scenario 4: moderate

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

As discussed above, it is feasible that backyard pigs kept in rural areas may come in close contact with nocturnally foraging feral pigs, and that transmission of PRRS virus from one group to the other may result. Backyard pigs are often raised for consumption by the household but it is feasible that some mixing of pigs between backyard herds may occur. For example, in the case of speciality breeds or unusual breeds, live pigs or semen may be transferred from one herd to another for breeding purposes. Alternatively, pigs raised for personal consumption may be transferred between backyard holdings for growing out or fattening.

The clinical signs of this disease are not pathognomonic or sufficiently distinctive to ensure its early diagnosis. Indirect spread by fomites or by mechanical vectors is also feasible and has been demonstrated experimentally (Dee, et al., 2002; Otake, et al., 2002a; Otake, et al., 2002b Otake, et al., 2002c; Dee, et al., 2003). For example, it is theoretically possible that PRRS virus in saliva and/or urine may be transferred from an infected backyard herd to other domestic pigs through inadequately cleaned vehicles, equipment or footwear.

It was stated above that if transmission to a piggery occurred, it is likely that the disease would be amplified and spread regionally by pigs, semen or fomites to other piggeries before diagnosis was made and controls to limit spread were initiated. If large commercial piggeries were also situated within the region, spread to these might occur, and this may lead to a more general outbreak.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: low

Scenario 3: low

Scenario 4: moderate

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

In particular, the likelihood of a more rapid diagnosis of PRRS was considered higher for small commercial piggeries, since the effects of the disease will likely be more obvious within a bigger herd and, particularly, in a herd that includes breeding sows. In addition, it is more likely that the managers of small commercial piggeries will detect the disease in an early stage, and that the consulting veterinarian will be alerted to the potential of an exotic disease epidemic. Nonetheless, several farrowing cycles may have been completed before the diagnosis of an exotic disease increasing the likelihood of spread to other piggeries. Other important considerations include the larger number of pigs moved between small commercial piggeries than backyard enterprises, and the increased potential for movement of pig semen. Likewise, it is more conceivable that infection would be amplified within a small commercial herd to the extent necessary for transmission via fomites or mechanical vectors.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: low

Scenario 3: moderate

Scenario 4: moderate

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus, while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this 'no outbreak' scenario, the disease is most likely to have established amongst the directly exposed animals, and to have run its course without identification. This is because the clinical symptoms of this disease are not distinct, may be mild and could be confused with endemic diseases.

The direct impact of PRRS

Animal life or health

A range of clinical signs are seen with PRRS infection, from reproductive losses in sows, mortality in very young piglets, and respiratory disease in older pigs. Infections can also be subclinical.

On the basis of this, the likely impact of PRRS in terms of *animal health*, where the disease is contained within the directly exposed group, was considered unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

Environment

Because PRRS is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of PRRS

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this outbreak scenario where there is containment of the disease within the directly exposed group and in the case of pigs the clinical signs of disease can be mild or non-specific, it was considered unlikely that the primary case would be diagnosed. Given this, it was considered unlikely that the indirect impact of new or modified control programs would be discernible at any level, and the rating assigned to this criterion was therefore 'A'.

Domestic trade or industry effects

As discussed above, it is unlikely that disease would be diagnosed in a single herd. On this basis, the indirect impact of PRRS on domestic trade and industry was considered unlikely to be discernible at any level, and a rating of 'A' was thus assigned to this criterion.

International trade effects

As the disease is unlikely to be diagnosed in a single herd, it was considered that the indirect effect of PRRS on international trade was unlikely to be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on the environment

In this scenario, PRRS is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, PRRS is most likely to have established within a population of feral pigs, and to have run its course without identification. As explained above, this is because the clinical symptoms of this disease are not distinct and can be mild.

The direct impact of PRRS

Animal life or health

As with the first scenario, the impact on animal health is unlikely to be discernible at any level except the local level. Indeed, it is likely that the disease would remain undiagnosed if contained within a more general population of feral pigs due to the nature of the disease and limited opportunities for close observation of feral pigs. Feral pigs harvested for meat are mature animals and are unlikely to be showing marked clinical signs of PRRS. This resulted in a rating of 'B' for this criterion.

Environment

Because PRRS is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of PRRS

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

The consequences on this criterion would be similar to that described above for the first scenario, resulting in a rating of 'A'.

Domestic trade or industry effects

In this outbreak scenario PRRS spreads to a more general population of feral pigs but not to domestic pigs. As the disease may remain undiagnosed within feral pigs for a significant period of time the indirect effects on domestic trade and industry were considered unlikely to be discernible at any level, and thus the rating assigned to this criterion was 'A'.

International trade effects

As described above the indirect effects of PRRS on international trade were considered unlikely to be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on the environment

PRRS in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, PRRS establishes within a local population of small commercial piggeries or backyard enterprises. It is likely that PRRS would be diagnosed due to reproductive disease in sows, increased piglet mortality and respiratory disease in weaned and finishing pigs. The disease would be contained following the implementation of control measures such as quarantine and movement restrictions.

The direct impact of PRRS

Animal life or health

The third scenario is characterised by spread of PRRS to a local population of domestic pigs in backyard enterprises or small commercial piggeries, but containment within this population. In those herds with breeding sows infection with PRRS virus can result in premature farrowings, stillborn piglets, mummified foetuses and high piglet mortality. In weaned and finishing pigs, respiratory disease is the predominant symptom and mortality can also occur. Production is significantly decreased for months.

On this basis, the direct effect on animal health was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

Environment

Because PRRS is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of PRRS

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

If PRRS was diagnosed in Australia, a control and eradication program would be implemented. PRRS is listed as Category 4 under the Emergency Animal Disease Sharing Agreement. In this agreement, the government and livestock industries have agreed to share costs based on the conduct of an agreed response plan for an outbreak of a disease that falls within one of four categories. Category 4 diseases are funded 20% by governments and the remaining 80% by the relevant industry. Such a plan (AUSVETPLAN) is being finalised. The draft plan affirms that Australian policy will be to eradicate PRRS by the most cost-effective method using modified stamping out, i.e. slaughter and salvage. In this scenario, where PRRS has only limited spread within the domestic pig population (a local population of backyard or small commercial piggeries), it was considered likely the disease would be eradicated promptly.

There would need to be some surveillance of the domestic pig population and possibly the feral pig population. If the disease was present in the feral pig population, depopulation may be feasible in the case of a localised outbreak. If eradication in the feral pig population was not possible, farmers would need to improve biosecurity.

Overall, the indirect impact of new or modified control programs was considered unlikely to be discernible at the national and State levels, and of minor significance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

Domestic trade or industry effects

Under the draft AUSVETPLAN requirements, the diagnosis of PRRS in a local population of domestic pigs would result in restrictions on the movement of breeding pigs and semen from infected premises and those within a 10 km radius. This is unlikely to affect small commercial piggeries or backyard piggeries significantly. Pigs would be permitted to go directly to an approved abattoir for immediate slaughter, but cooking of the meat would be required.

With the detection of an exotic disease in Australia consumers may initially decrease pork consumption. A publicity campaign may need to be undertaken to reassure the public that there are no health concerns.

In this scenario, with a local outbreak, other industries associated with the pig industry such as feed, transport, meat processing and pharmaceutical industries are unlikely to be significantly affected.

When these issues were taken into account, the indirect impact of PRRS on domestic trade and industry was considered unlikely to be discernible at the national level, but would have a minor impact at the State level. This resulted in a rating of 'D' for this criterion.

International trade effects

The diagnosis of PRRS in domestic pigs, even within a local population, would likely result in initial cessation of trade in live pigs and semen to some markets. Australia exports only small numbers of breeding pigs and quantities of semen. Trade should only be interrupted temporarily under this scenario with testing of pigs and semen donors for PRRS prior to export an option.

With a limited small outbreak it is unlikely that export of meat would be significantly disrupted. There may be an initial reaction from some trading partners to halt meat imports in the short term, however, as there is no human health risk and the disease is endemic in most markets, trade should resume quickly. In the case of pig meat exports to New Zealand, meat may have to be processed by cooking either prior to export to, or on arrival in, New Zealand.

After consideration of these issues, the indirect effect of PRRS on international trade was considered unlikely to be discernible at the national or State level, but would have a minor impact at the level of district or region. Overall, this resulted in a rating of 'C' for this criterion.

Indirect impact on the environment

PRRS diagnosed in a local population of domestic pigs is unlikely to lead to any significant indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, PRRS would have established in a broader population of commercial piggeries (including medium-large piggeries). A control and eradication program would have been mounted in response to the diagnosis of the disease in pigs.

The direct impact of PRRS

Animal life or health

An outbreak of PRRS to a more general population of domestic pigs may cause significant production losses, although in some cases infections can be inapparent. On a herd basis, most acute outbreaks are estimated to decrease annual production 5 to 20% (Christianson & Joo, 1994). In the United States of America cost estimates for direct losses in an acute outbreak range up to US$250 per sow and over US$500 per sow in chronic infections[47]. In the United Kingdom losses of £102 per sow have also been reported (Christianson & Joo, 1994). PRRS infection can result in reproductive disorders such as increased number of abortions, stillbirths and mummified foetuses. Respiratory disease can occur in pigs in all stages of production. In one reported acute outbreak the mortality rate for term piglets (including stillborn) increased from 6% prior to the outbreak, to a maximum of approximately 76% up to weaning (Pejsak, et al., 1997). However, after about 5 months, production returned to normal levels.

Given this, the direct impact on animal health was considered to be unlikely to be discernible at the national level, but would be of minor significance at the State level. This resulted in a rating of 'D' for this criterion.

[47] http://www.porkscience.org/documents/other/positionprrs.pdf

Environment

Because PRRS is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of PRRS

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

As stated in the above scenario, diagnosis of PRRS in Australia would lead to a comprehensive control and eradication program. Infected premises and those in the restricted area would be subject to quarantine and movement controls. The recommended strategy is immediate depopulation with salvage through abattoirs. Nonetheless, it is likely that stock unsuitable for slaughter such as older sows would need to be disposed of on farm. Vaccination may be considered for breeding animals or, alternatively, if the disease became established. Biosecurity would include measures to prevent contact with wild pigs. If the disease was widespread eradication may be prolonged and costly to producers and governments. The expected economic impact of PRRS outbreaks in two regions of Australia have been estimated (Garner, et al., 2001). In this study, a value of $600 per pig was estimated to approximate the costs of destocking a piggery and managing an eradication response at a local level. The expected overall nation-wide costs of epidemics in the Darling Downs and Northern Victoria was approximately $33.5 million and $45 million respectively.

There would be surveillance of the domestic and feral pig population. If the disease was detected in the feral pig population, eradication may be feasible if localised but otherwise would be difficult.

On this basis, the likely indirect impact of new or modified control programs would be unlikely to be discernible at the national level, but would have a minor impact at the State level. Overall this resulted in a rating of 'D' for this criterion.

Domestic trade or industry effects

The diagnosis of PRRS within commercial piggeries would result in restrictions on the movement of live pigs and semen within the restricted area. Pigs would still be able to move for slaughter, but, until the disease was eradicated or eradication was abandoned, there would be restrictions on the trade in uncooked pig meat in Australia. Replacement breeding stock would need to be purchased following decontamination of the infected piggery.

If PRRS became established there would be significant ongoing production costs for producers, affecting the viability of some and damaging their communities. Reductions in piggery outputs of around 15% have been estimated, based on simulations of PRRS outbreaks in Australia where the disease becomes endemic in five States (Garner, et al., 2001). This study estimated that the gross national income of the pig industry would fall by 5% (an annual estimated cost of $34 million in lost production).

If there was an epidemic of PRRS within the Darling Downs or Northern Victoria, it was calculated that the gross national income of the pig industry would fall by 6%, based on lost production, cost of disposal and price effects. In the United States of America, PRRS is reported to be the most important disease for pork producers from an economic impact

standpoint[48]. The National Pork Board, United States of America has stated that PRRS costs the industry approximately $600 million a year[49].

Veterinary costs would increase during the outbreak and there would be ongoing costs if the disease became endemic together with additional costs associated with preventing and treating secondary infections.

Industries supplying inputs to the pig industry, such as feed manufacturers and pharmaceutical industries would also be affected together with those utilising the outputs from the pig industry such as abattoirs, processing plants and transport.

The presence of PRRS in a breeding herd may affect the marketability of breeding stock. Abattoirs should be willing to slaughter and process pigs from infected piggeries, although some trade practices may be disrupted. Pigs would be permitted to go directly to an approved abattoir for immediate slaughter, but cooking of the meat would be required. Fresh pig meat for domestic consumption may be in short supply and the price may increase.

When these issues were collated, the indirect impact of PRRS on domestic trade and industry was considered unlikely to be discernible at the national level, but would have a minor impact at the State level. This resulted in a rating of 'D' for this criterion.

International trade effects

As discussed above under scenario 3, trade in live pigs and semen may be halted following detection of PRRS although this is likely to be temporary. It is likely that a few individual consignments would be affected while conditions were renegotiated.

Australia's major markets for pig meat are Singapore and Japan. New Zealand is also a significant export market for Australian pig meat. These markets would be sensitive to any significant disease outbreak involving the commercial pig population. It is likely that trade may be temporarily halted while reassurances were provided that there were no public health implications. Singapore does not have a pig industry, and Japan has PRRS. Both countries import pig meat from countries where PRRS is endemic so trade should be able to be renegotiated. New Zealand requires pig meat to be processed by cooking when imported from countries where PRRS is present and would likely require this of Australia.

In light of this information, it was considered that the indirect impact of PRRS on international trade was unlikely to be discernible at the national or State level and of minor significance at the district or regional level. Overall, this gave the disease a rating of 'C' for this criterion.

Indirect impact on the environment

PRRS diagnosed in a local population of domestic pigs is unlikely to lead to any significant indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

One of the considerations within this criterion was the possible indirect impact of a disease on rural and regional economic viability. The pig industry is important to the economies of several localities and districts in New South Wales, Victoria, Queensland, Western Australia and South Australia. It has been estimated that in general terms, for every one employee working in the

[48] http://www.porkscience.org/documents/other/positionprrs.pdf
[49] http://www.agriculture.com

pig industry, there will be another two people employed in providing goods and services to the pig industry directly or to employees in the pig industry.

It is clear that the viability of some producers would be affected if there was a widespread outbreak of PRRS or if the disease became established within the pig industry. Where the pig industry was highly significant to the local economy, aspects of these communities may be threatened.

Taking these issues into consideration, the indirect impact of PRRS on rural communities was considered unlikely to be discernible at the national or State level, but of importance to affected districts or regions. This resulted in a rating of 'C' for this criterion.

The overall impact of PRRS

When the direct and indirect impacts of PRRS were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences negligible

Scenario 3: Consequences low

Scenario 4: Consequences low

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 43, Table 44, and Table 45. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were considered 'low', 'low' and 'low' respectively.

Table 43 PRRS: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Very low	Negligible	Negligible
Scenario 2	Moderate	Negligible	Negligible
Scenario 3	Low	Low	Very low
Scenario 4	Moderate	Low	Low
		Overall likely consequences	**Low**

Table 44 PRRS: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Low	Low	Very low
Scenario 4	Moderate	Low	Low
		Overall likely consequences	**Low**

Table 45 PRRS: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Moderate	Low	Low
Scenario 4	Moderate	Low	Low
		Overall likely consequences	**Low**

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with PRRS.

Table 46 shows the results obtained for each of the exposure groups and for the overall annual unrestricted annual risk. Since the overall annual risk is clearly higher than Australia's ALOP (very low), risk management would be required for PRRS.

Table 46 PRRS: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Very low	High	Low	Low
Backyard pigs	Very low	Low	Low	Very low
Small commercial piggeries	Very low	High	Low	Low
			Overall annual risk	**Low**

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Transmissible gastroenteritis virus

Technical information

Background

Transmissible gastroenteritis (TGE) was first described in 1946 in the United States of America. Subsequently, it has been reported in most pig-producing regions of the world. It is characterised by profuse diarrhoea in young piglets, with a high mortality rate. However, in recent years, particularly in Europe, the impact of TGE has lessened due to the presence of a deletion mutant of TGE virus, porcine respiratory coronavirus (PRCV). PRCV was detected in the 1980s and provides partial protection against infection with TGE virus whilst being itself of limited pathogenicity (Saif & Wesley, 1999).

Agent taxonomy

Transmissible gastroenteritis virus is a member of the family Coronaviridae, genus Coronavirus (Benfield, et al., 1991).

Agent properties

Transmissible gastroenteritis virus is a single-stranded (positive), nonsegmented, enveloped RNA virus (Pensaert, 1989). Strains of differing pathogenicity have been described, but only one serotype has been reported (Kemeny, 1976). Antigenically, TGE virus is closely related to canine coronavirus and feline infectious peritonitis virus (Pensaert, 1989).

The survival of viruses outside a living host is affected by factors that include the substrate, pH, temperature, relative humidity, and exposure to ultraviolet light.

No significant loss in infectivity was detected after storage for 365 days of TGE virus in culture medium at -20°C, -40°C, and -80°C. At 4°C, a significant decrease in infectivity was observed after 180 days of storage. At room temperature and at 37°C, loss of infectivity was observed after 45 and 4 days, respectively (Harada, et al., 1968). The inactivation times for TGE virus in liquid manure are reported to be in excess of 8 weeks for material held at 5°C, but only 24 hours and 30 minutes when held at 35°C and 55°C respectively (Haas, et al., 1995). The virus is highly photosensitive. Transmissible gastroenteritis virus in small quantities of liquid manure was inactivated within 6 hours after exposure to sunlight (temperature range 21°C to 31°C) (Haelterman, 1963). Transmissible gastroenteritis virus is stable within a pH range of 3 to 9 (Harada, et al., 1968).

Host range

Infection with TGE virus causes clinical disease only in pigs.

It has been shown that dogs, cats and foxes may shed infective virus in their faeces for a variable length of time after ingestion of the virus (Haelterman, 1962; Larson, et al., 1979; Reynolds & Garwes, 1979). However, clinical disease does not occur in these species and although it was originally suggested they may act as carriers or serve as a reservoir for TGE virus, this has not been confirmed (Haelterman, 1962; McClurkin, et al., 1970; Schulman, et al., 1980).

Epidemiology

Transmissible gastroenteritis has been identified in most pig-producing areas, with exceptions including Australia, New Zealand and Norway. Ireland was free of TGE virus until an outbreak in 1984; this outbreak was contained and the disease eradicated (Gunn, 1996).

Pigs of all ages are susceptible to infection with TGE virus. Outbreaks of TGE have a seasonal incidence, with more than 90% of outbreaks in the United States of America reported to occur during winter and spring (Haelterman, 1963). Initial introduction of the virus into a herd usually results in infection of most pigs but its presence may only be suspected when young piglets are exposed to the virus. Infection with TGE virus in piglets less than 3 weeks of age is characterised by an acute onset of profuse, watery, foul-smelling diarrhoea, vomiting, dehydration, depression, anorexia and death (Haelterman, 1963). In excess of 1,200 piglets died over a 7 week period in a 650 sow piggery during the outbreak of TGE in Ireland in 1984 (Gunn, 1996). The mortality rate amongst piglets declines dramatically when they are born to previously-infected sows as the colostrum and milk of immune sows contain antibodies that neutralise ingested TGE virus in the intestinal lumen of the piglet (Saif & Wesley, 1999). Cessation of piglet mortality does not necessarily imply eradication of TGE infection from the herd. Endemic TGE is common and depends on a continual supply of susceptible pigs as may occur with the introduction of naïve sows or feeder pigs. In this situation, piglets are usually protected in whole or part whilst nursing (thus mortality and clinical signs are greatly reduced), but are susceptible to infection after weaning, with less severe disease and mortality in this age group (Pritchard, 1987). Infection of weaned pigs with TGE virus may result in a reduced growth rate (Maes & Haelterman, 1979).

The incubation period for TGE can be as short at 18 to 24 hours. The virus is shed in large quantities in the faeces, with 10^5 infectious doses per ml of pooled faeces obtained from six 9 to 11 day-old piglets 3 days after experimental inoculation (Haelterman, 1963). Transmission is usually via the faeco-oral route, although virus may also be shed in milk and nasal secretions (Kemeny & Woods, 1977). The copious amounts of diarrhoea produced by affected piglets and its tenacious nature promote the dissemination of the virus and the spread of the disease, particularly amongst intensively housed animals. Infected pigs are reported to shed virus in faeces for up to 2 weeks (Saif & Wesley, 1999), although as TGE virus could be recovered from the jejunal contents (but not the rectal faeces) of two of seven feeder pigs 35 days after experimental inoculation, virus might occasionally be shed in the faeces for longer periods (Morin, et al., 1974). It has been proposed that the virus may be transmitted between herds, particularly in winter due to the greater stability of the virus, on inanimate objects (Saif & Wesley, 1999). The potential for mechanical transmission of TGE virus by starlings (Pilchard, 1965) and house flies (Gough & Jorgenson, 1983) has been suggested, but their importance in the epidemiology of TGE has not been confirmed.

The epidemiology of TGE has been complicated by the emergence in the 1980s of PRCV (Pensaert, et al., 1986). PRCV, a respiratory virus of relatively low pathogenicity, has become extremely widespread throughout most of the pig-producing regions of the world with exceptions including Australia and New Zealand (Saif & Wesley, 1999). The two viruses cross-neutralize and thus cannot be distinguished by the classical serum neutralisation test for TGE (Pensaert, et al., 1993). Immunity resulting from infection with PRCV is reported to provide partial protection against the clinical effects of TGE viral infection in piglets (Laude, et al., 1993). Clinical outbreaks of TGE have been reported less frequently in Europe since the emergence of PRCV. For example, TGE virus was diagnosed in Belgium 68 times during the period of 1982 to 1983, 61 times in 1985 to 1986, and only seven times in 1988 and 1989

(Laude, et al., 1993). Transmissible gastroenteritis virus was still circulating in Belgian pig herds at this time, as 15% of 81 breeding herds, and 15% of 33 fattening farms contained pigs that were seropositive for TGE virus by competitive ELISA in 1990 (Pensaert, et al., 1993). Similarly, subsequent to the establishment of PRCV in Britain, clinical outbreaks of TGE infection are extremely rare, although a survey conducted in the East Anglia region in 1997 showed that 21% of herds in this region were serologically positive for TGE virus (Pritchard, et al., 1999).

Four-hundred and eighty herds were sampled in 1987 in the Murcia region of south eastern Spain, and the overall prevalence of TGE-infected herds was determined to be about 5% (Cubero, et al., 1993).

Prior to the emergence of PRCV, the prevalence of TGE infected herds in Quebec was estimated to be 19% (Gagnon, et al., 1974). In major pig-producing areas of the United States of America more than 50% of farms were reported to be infected (Egan, et al., 1982; Hill, et al., 1983) but it is not known whether PRCV was then present. In 1989 to 1990, 46% of 392 herds sampled in the United States of America's National Swine Survey were positive by the serum neutralisation test (Yanga, et al., 1995); however, no distinction was made between TGE and PRCV which was first noted in the United States of America around this time (Wesley, et al., 1990). In 1995, 22 Iowa pig herds in another study were sampled for the presence of porcine coronavirus antibodies (Wesley, et al., 1997). Twelve herds were infected with PRCV, six with TGE virus, and four with both. A national study conducted in the United States of America has shown clinical TGE to be a problem in approximately 2% of breeding sows, 3% of suckling pigs, and 1% of nursery-age pigs in TGE infected herds (USDA, 2002).

In Spain the within herd seroprevalence on infected farms ranged from 5 to 60% (Cubero, et al., 1993). These authors discuss other studies in which wide within herd seroprevalences were reported (ranging from 0.5 to 96%). Although within herd prevalence was not determined, the prevalence of antibody to TGE virus in a sample of slaughter-age pigs in the United States of America has been reported as 34.8% (Cook, et al., 1991), which was consistent with an earlier study that showed a prevalence of 30.9% (Egan, et al., 1982).

The prevalence of infected slaughter-age pigs within a TGE-infected herd depends on factors including the chronology of infection in the herd, management practices (continuous versus batch systems), and herd structure (farrow to finish operations versus fattening operations). In many cases, particularly in herds that are endemically infected with TGE virus, pigs are infected soon after weaning. However, in the case of herds that have not previously been exposed to TGE virus, or in endemically-infected herds (particularly large herds) where pigs may not have been exposed to the virus at an early age, infection of slaughter-age pigs does occur, particularly when groups of pigs are commingled (Morin, et al., 1978; Maes & Haelterman, 1979; Gunn, 1996).

Clinical signs

The clinical signs associated with infection by TGE virus have been reviewed (Bohl, 1989; Saif & Wesley, 1999). Severity of disease is inversely related to age at infection. As mentioned above, infection with TGE virus in piglets less than 3 weeks of age born to non-immune sows is characterised by the acute onset of profuse, watery, foul-smelling diarrhoea, vomiting, dehydration, depression, anorexia and death (Haelterman, 1963). Older piglets are less severely affected, as are those born to immune sows, as the colostrum and milk of such sows contain antibodies that neutralise ingested TGE virus in the intestinal lumen of the piglet (Saif &

Wesley, 1999). Grower and adult pigs, whilst susceptible to infection with TGE virus, may exhibit no clinical signs. However, transient inappetence, very occasional vomiting, and some loosening of the faeces may be observed.

Pathogenesis

The pathogenesis of TGE has been summarised (Bohl, 1989; Saif & Wesley, 1999). After ingestion, the virus passes through the stomach to the small intestine, the primary site of replication. Viral particles attach to the columnar epithelial cells of the villi. These cells are shed, causing shortening or atrophy of the villi. The loss of these functional enterocytes results in the characteristic maldigestion and malabsorption associated with TGE. Although the virus is predominately an enteric virus, some limited extra-enteric activity may occur. Shedding of the virus from nasal secretions and the mammary gland of lactating sows after intravenous inoculation is thought to result from haematogenous spread (Kemeny & Woods, 1977). Virus has been recovered from the lung of an experimentally infected piglet, and viral antigen was detected by immunocytochemistry in bronchiolar epithelial cells, alveolar macrophages and hepatocytes of three similarly infected piglets (O'Toole, et al., 1989). Virus has been isolated from the tonsils of naturally-infected, asymptomatic slaughter pigs (Kemeny, 1978; Cook, et al., 1991).

The level and duration of viraemia in naturally infected pigs is not well defined, but is likely of very low levels and of short duration. Virus was not detectable by viral isolation in blood samples or from pharyngeal swab, muscle, lymph node or bone marrow samples from acutely infected six-month-old pigs, yet homogenates of muscle, lymph node and bone marrow from these pigs were infectious when fed to recipient piglets (Forman, 1991).

Pathology

Gross pathologic findings tend to be unremarkable in all but very young piglets. The carcass of young piglets is dehydrated, and the stomach may be distended with curdled milk. The small intestine may be thin walled, and distended with foamy fluid. The mucosa of both stomach and small intestine may be congested (Haelterman, 1963).

Immunology

An effective immunity is induced following infection with TGE virus; however, the duration of immunity is unknown, and effective vaccines have not, to date, been developed (Saif, et al., 1994).

Transmission via meat

Although TGE virus has not been detected in muscle and bone marrow by virus isolation, homogenates of these tissues including lymph node were infectious when fed to naïve pigs (Cook, et al., 1991; Forman, 1991). Virus has been isolated from the tonsils of slaughter age pigs and sows, with the prevalence of infection reported as 0.8 and 3% respectively (Kemeny, 1978; Cook, et al., 1991).

In the first study (Forman, 1991), 16 pigs (4 to 6 months of age) were exposed to 12 piglets (1 week of age) that had been orally infected with TGE virus. Fourteen of the 16 pigs were sequentially slaughtered over a period of 6 days following contact challenge. Muscle from the hindquarter (approximately 1 kg), lymph nodes (internal iliac, sub-maxillary and cervical) and femoral bone marrow were harvested from each pig and frozen at -25°C for at least 30 days.

Virus isolation was attempted on these samples prior to freezing, and on intestinal samples and pharyngeal swabs. In addition, during the slaughter period, pigs remaining were bled and nasal passages were swabbed for purposes of viral isolation. Neither clinical signs of TGE infection nor viraemia was detected in any of the 16 pigs. Virus was isolated from a single nasal swab (collected on the first post-contact day) but from none of the pharyngeal swabs, muscle, lymph node or bone marrow samples. However, virus was detected in intestinal samples, and/or virus antigen in intestinal mucosa of some of the pigs. The two remaining pigs not slaughtered had neutralising antibody titres to TGE virus 15 days after the commencement of the challenge.

A total of 17 kg of tissue (muscle, bone marrow and lymph nodes) harvested from the 14 pigs was thawed, minced and fed to 12 three-week-old piglets over 5 days so that they received approximately 1.5 kg each (50% of their daily intake). In addition, 12 one-week-old piglets were each inoculated orally with 5 ml of a 10% w/v homogenate of the carcass tissues. All piglets were held in separate pens in the same room. No clinical signs were observed in the three-week-old piglets, but four of the one-week-old piglets died between 10 and 17 days post-inoculation after exhibiting non-specific signs of anorexia and emaciation 1 to 2 days prior to death. Transmissible gastroenteritis virus was isolated from the intestine of one of the dead piglets, and villous atrophy was apparent histologically in two of them. All surviving piglets had neutralising antibody to TGE virus by 28 days post-exposure. It was concluded that TGE virus was present, albeit likely at very low levels, in the carcass tissues from one or more of the acutely infected 4 to 6 month old pigs.

In the second study (Cook, et al., 1991), 500 heads were collected from pigs commercially and routinely slaughtered in Iowa, United States of America. Tonsil, brachiocephalic muscle and parotid lymph node samples were collected from each head. Muscle and lymph node samples from each of 25 pigs were pooled, resulting in 20 homogenates. Viral isolation was attempted on individual homogenates of the tonsil samples and pooled muscle and lymph node homogenates. Virus was isolated from 4 of the 500 tonsil samples (0.8%), but from none of the pooled muscle and lymph node homogenates.

Two groups of 10 piglets were dosed orally with 5 ml per day of the muscle and lymph node homogenate over a period of 4 days. All 20 piglets developed typical signs of TGE infection by day 7 post-inoculation and five piglets died. The presence of TGE virus was confirmed in the dead piglets, and all 15 surviving piglets had neutralising antibody to TGE virus by day 21 post-inoculation. Due to the housing arrangements for each group of piglets, horizontal transmission of TGE may have occurred. However, at least one homogenate fed to each group contained virus, and therefore, it was concluded that the carcass tissues of at least two of the 500 slaughtered pigs contained viable TGE virus.

Release assessment

R1 — the likelihood that a source herd is infected

Taking into account the presence of PRCV, estimates of the prevalence of infected herds in countries in which TGE virus infection is endemic include 5% in Spain in 1987, 15% of sampled farms in Belgium in 1990, 19% in Quebec in the 1970s, and 21% in the East Anglia region of England in 1997. Given this, the likelihood that a source herd is infected was considered to be ‘low’.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

The within herd seroprevalence of TGE on infected farms has been reported to range from 5 to 96%. Within slaughter age pigs, the prevalence of antibody to TGE virus has been reported from two studies as 30.9% and 34.8%.

Pigs from infected herds are usually exposed to, and infected with, TGE virus soon after weaning. Most pigs shed virus in their faeces for less than 2 weeks, thus are less likely to be infected at slaughter-age. However, particularly in large herds, some pigs may not be exposed to the virus until later in the growing period, and thus may be infected at slaughter. This is supported by the isolation of virus from the tonsils of 0.8% of slaughter-age pigs in Iowa in 1990 and from 3% of slaughtered sows in the United States of America, with a day-to-day infection rate varying from 0 to 22.9% as reported earlier.

Taking the above factors into account it was considered that the likelihood of selecting an infected animal in an infected herd was ‘low’.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

Slaughter-age pigs infected with TGE virus usually show no clinical signs. However, transient inappetence, very occasional vomiting, and loosening of the faeces may occur. If diarrhoea is sufficient to cause excessive soiling of the animal, it may be withheld from slaughter subject to cleaning. The pathology of this condition is unremarkable in slaughter-age pigs, thus infected pigs are unlikely to be identified at post-mortem inspection.

Considering these factors, the sensitivity of ante-mortem, slaughter and processing requirements in detecting and removing TGE virus-infected pigs was estimated to be ‘extremely low’.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with TGE virus and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a ‘one minus the background rejection rate’. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered ‘extremely low’. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

Transmissible gastroenteritis virus is an enteric virus, with the small intestine being the primary site of replication. The intestinal tract and associated lymph nodes are removed during processing. Viraemia is difficult to demonstrate and, if present, it is likely to be at very low

levels and of short duration. The virus has been isolated from tonsils of infected pigs. Tonsils are removed at slaughter; however, residual tissue may remain and it was considered that associated draining lymph nodes might also harbour infectious virus. Ingestion of combined muscle and lymph node samples, or muscle, lymph node and bone marrow samples derived from infected pigs has resulted in transmission of TGE virus to susceptible pigs, although the amount of virus present in these tissues was below the level detectable by isolation in tissue culture in (at least) one study. The virus does not appear to have an affinity for muscle tissue, so its presence in muscle tissue is likely due to infected blood or lymph perfusing the tissue.

The possibility of contamination of the carcass with TGE virus as a result of faecal or intestinal spillage during processing was considered. It is a requirement of processing to the Australian Standard that any evidence of visible contamination of the carcass with faecal material be removed (Agriculture and Resource Management Council of Australia and New Zealand. Standing Committee on Agriculture and Resource Management, 2002). Any of the few viral particles remaining could neither penetrate the carcass nor multiply on the skin.

Based on this information, the likelihood that TGE virus would be present in a carcass including the head was estimated to be ‘low’.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

TGE virus is stable throughout the range of pH 3 to 9, thus the likelihood that TGE virus will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation was considered to be ‘high’.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

Transmissible gastroenteritis virus is stable at low temperatures for prolonged periods particularly when frozen. At 4°C infectivity of TGE virus decreased significantly but this was after 180 days storage. Given this, it was considered that there was a ‘high’ likelihood that meat infected or contaminated with TGE virus at the completion of carcass maturation would remain infected during storage and transport.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a ‘very low’ likelihood that imported pig meat derived from an individual carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a ‘very low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

Although transmission of TGE virus to susceptible piglets has been demonstrated after oral administration of homogenates prepared from carcass tissues, evidence for the transmission of the virus in commercial pork or pork products has not been documented. In the study discussed above (Forman, 1991), piglets were fed large quantities of (approximately 300 g/day for 5 days) of minced carcass tissues derived from pigs acutely infected with TGE virus. Although the recipient piglets were fully susceptible and young enough (3 weeks of age) that clinical signs of infection would have been expected, neither clinical evidence of infection nor isolation of virus from the faeces of these piglets was reported. However, neutralising antibodies to TGE virus were present at 28 days post-exposure. The amount of virus present in carcass tissues from acutely infected pigs is thus likely to be extremely low, particularly when contrasted with that contained in faeces, where in excess of 10^5 infectious doses per ml has been reported.

In light of this information, it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of TGE virus to initiate infection was estimated to be ‘very low’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

Transmissible gastroenteritis virus is reported to be susceptible to sunlight and, although preserved at low temperatures, is rapidly inactivated as temperatures increase. Nonetheless, at room temperature the virus required 45 days before loss of infectivity was observed. This susceptibility to increased temperatures has been suggested as the reason that, historically, TGE infection is a winter problem in pigs in the United States of America.

Given this, the likelihood that the TGE virus would remain viable during the period prior to scavenging was considered to be ‘moderate’.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = Low
- Rural regions = Very low
- Large towns = Extremely low

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be 'low'.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'very low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a 'high' likelihood that the TGE virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;

- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'extremely low'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'very low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a 'high' likelihood that the TGE virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'low'.

Exposure assessment for other susceptible species

Dogs, cats and foxes may shed infective virus in their faeces for a variable length of time after ingestion of the virus (Haelterman, 1962; Larson, et al., 1979; Reynolds & Garwes, 1979). However, clinical disease does not occur in these species and they have not been shown to act as carriers or serve as a reservoir for TGE virus. On the basis of this information, the Panel concluded that these species did not warrant further consideration as an exposure group.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the three exposure groups - that is, feral pigs, pigs in backyard enterprises and pigs in small commercial piggeries. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Transmissible gastroenteritis is usually spread to susceptible pigs by ingestion of faeces containing viable virus. The disease spreads particularly quickly within farrowing units, where pigs are intensively housed with a high population density, and where the profuse diarrhoea

produced by piglets enhances the spread of contagious faecal material throughout the unit. The disease spreads more slowly amongst extensively managed pigs and similarly would be expected to spread slowly within a feral pig herd. One mathematical model predicted that for feral pigs, if the transmission coefficient for TGE virus was low, it was highly likely that the disease will disappear before it was detected in these animals. However, if the transmission coefficient was higher the disease was predicted to persist over 2 years (Hone, 1994). It was estimated that an infected feral pig could be in contact with over 161 other feral pigs in areas as low as 15 km^2 in the wetlands of New South Wales or 26 km^2 in the Northern Territory, both areas within potential home range sizes.

Although pigs of all ages are susceptible to TGE infection, a scavenging feral pig that ingests infected porcine waste is unlikely to develop diarrhoea, and the concentration of viral particles in the faeces may be quite low. In addition, the ambient temperature of much of the home range of Australian feral pigs may limit the survival of the virus in excretions. High environmental temperatures are hypothesized as a reason that feral pigs in southern areas of the United States of America do not serve as a reservoir for TGE virus (Woods, et al., 1990).

Feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs; however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: low

Scenario 3: low

Scenario 4: low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the ‘no outbreak’ scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

Transmissible gastroenteritis infection in backyard pigs may go unnoticed if no newborn piglets are present at the time of introduction. In adult pigs the clinical signs of disease can be very mild or inapparent. Generally the virus is excreted in faeces for a few weeks. Spread of the disease to other backyard herds or small commercial piggeries could occur via movement of recently infected pigs or indirectly via faecal contamination of items such as trucks, crates, feed or boots. Owners of backyard pigs are less likely to seek veterinary attention which could result in a delay in diagnosis and spread of the virus. One study concluded that piggeries at most risk of not correctly diagnosing TGE infection appear to be those with older pigs such as for fattening, where there is little or no veterinary involvement; and where endemic or sporadic diarrhoea associated with other pathogens occurs (Hone, 1994).

If transmission from backyard pigs to a small commercial piggery were to occur, it is likely that the disease would be spread via movement of live pigs and fomites to other piggeries before a diagnosis was made.

Due to the close contact required for spread of the virus and the relatively short excretion time spread of the disease to feral pigs from a backyard herd is regarded as very unlikely.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: low

Scenario 3: moderate

Scenario 4: low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the ‘no outbreak’ scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other

domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between ‘small commercial piggeries’ and ‘backyard piggeries’ is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

An outbreak of TGE virus infection is more likely to be diagnosed in a small commercial piggery than in a backyard piggery, since the effects of the disease will be more obvious, particularly in a herd with piglets on the premises at the time of the introduction. Moreover, managers of small commercial piggeries are more likely to seek veterinary advice. However, the increased movement of pigs from small commercial piggeries and the greater amount of infectious material produced, may result in further spread of the disease via contaminated boots, trucks, feed, or other fomites.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: low

Scenario 3: moderate

Scenario 4: moderate

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes ‘no further establishment or spread’. The remaining three outbreak scenarios describe ‘secondary spread to other pigs, humans or to other susceptible species’. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this ‘no outbreak’ scenario, the disease is most likely to have established amongst the directly exposed animals, and to have run its course without identification. This is because the clinical symptoms of this disease are not distinct, may be mild and could be confused with endemic diseases.

The direct impact of transmissible gastroenteritis

Animal life or health

Transmissible gastroenteritis virus infection results in high mortalities in young piglets. However, in older animals infected with the virus clinical signs may be absent or mild. Due to the limited extent of this scenario, the likely impact of TGE on animal health was considered unlikely to be discernible except at the local level. This resulted in a rating of ‘B’ for this criterion.

Environment

Because TGE is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of transmissible gastroenteritis

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this outbreak scenario the disease is contained within the directly exposed group. Clinical signs of infection can be very mild for older pigs infected with the virus although there can be high mortality in young piglets. On this basis, it was considered unlikely that the primary herd infected would be diagnosed with TGE infection.

Given this, it was considered likely that the indirect impact of new or modified control programs would be undiscernible at any level, and the rating assigned to this criterion was therefore 'A'.

Domestic trade or industry effects

As discussed above, it was considered unlikely that TGE would be diagnosed in a single herd. On this basis, the indirect impact of TGE on domestic trade and industry was considered unlikely to be discernible at any level, and a rating of 'A' was thus assigned to this criterion.

International trade effects

As it was considered unlikely that TGE would be diagnosed under this scenario, it was considered that the indirect effect of TGE on international trade was unlikely to be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on the environment

Transmissible gastroenteritis in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, the disease is most likely to have established within a population of feral pigs, and to have run its course without identification. As explained above, this is because the clinical symptoms of this disease are not distinct and can be mild.

The direct impact of transmissible gastroenteritis

Animal life or health

As with the first scenario, the impact on animal health is likely to be minor at the local level. Indeed, it is likely that the disease would remain undiagnosed if contained within a more general population of feral pigs due to the limited opportunities for close observation of feral pigs. Feral pigs harvested for meat are mature animals and unlikely to be showing clinical signs of TGE. This resulted in a rating of 'B' for this criterion.

Environment

Because TGE is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of transmissible gastroenteritis

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this outbreak scenario, TGE spreads to feral pigs, but does not spread to domestic pigs. The indirect impact of new and modified control programs was considered similar to that described above for the first scenario, resulting in a rating of 'A' for this criterion.

Domestic trade or industry effects

It was considered likely that the disease may remain undiagnosed within feral pigs for a significant period of time. As such, it was considered that the indirect effects on domestic trade and industry were unlikely to be discernible at any level, hence this criterion was rating 'A'.

International trade effects

As described above the indirect effect of TGE on international trade was unlikely to be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on the environment

Transmissible gastroenteritis in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, the disease has established within a local population of small commercial piggeries or backyard enterprises. It is likely that the disease would be diagnosed due to the

increased piglet mortality and diarrhoea. The disease would be contained following the implementation of control measures such as quarantine and movement restrictions.

The direct impact of transmissible gastroenteritis

Animal life or health

The third scenario is characterised by spread of TGE to a local population of domestic pigs in backyard enterprises or small commercial piggeries, but containment within this population. Due to the potential for spread within a herd and high mortality in piglets less than three weeks of age, it was considered that the direct effects on animal health would be unlikely to be discernible at the national or State level but would be of minor importance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

Environment

Because TGE is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of transmissible gastroenteritis

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

Under this scenario, the disease spreads to a local population of domestic pigs at backyard enterprises or small commercial piggeries. It was considered that the TGE would be diagnosed under this scenario.

The Australian policy for TGE as outlined in AUSVETPLAN (Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 1996) is 'to eradicate TGE by the most cost-effective method using one of more of three strategies in infected piggeries'. These strategies include stamping out, salvage and slaughter out or eradication by controlled exposure. They are supported by quarantine and movement controls, decontamination, tracing and surveillance, and a public awareness campaign.

Stamping out involves quarantine, slaughter of all infected and exposed susceptible animals on infected premises and safe disposal of destroyed animals and contaminated animal products. Salvage and slaughter-out involves quarantine, slaughter of all saleable infected pigs at an abattoir and destruction of animals that are not saleable. The third strategy involves quarantine followed by rapid dissemination of the virus throughout the infected herd with the aim of eliminating the virus from the piggery. Pigs would be permitted immediate slaughter at an abattoir.

Transmissible gastroenteritis virus infection is classed as a Category 4 'production loss' disease under the Emergency Animal Disease Response Agreement[50], thus the costs of the response program are to be shared between governments (20%) and industry (80%).

Pig producers may also need to improve biosecurity to prevent access by feral pigs, birds, foxes, dogs and cats.

[50] http://www.aahc.com.au/eadp/response.htm

When these issues were taken into account, the indirect impact of new or modified control programs was considered unlikely to be discernible at the national or State level, and of minor importance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

Domestic trade or industry effects

AUSVETPLAN recommends that movement of pigs from the restricted area only be permitted to move directly to slaughter. Thus the movement of live pigs from property to property or to saleyards would be prohibited. Under this scenario, where only a local population of pigs from backyard enterprises or small commercial piggeries is infected, movement restrictions are unlikely to significantly affect domestic trade. There would be significant costs for those producers affected. In addition, with the detection of an exotic disease of pigs in Australia, consumers may initially decrease pork consumption.

When these issues were taken into account, the indirect impact of TGE on domestic trade was considered unlikely to be discernible at the national and State levels and minor at the district or regional level. This resulted in a rating of 'C' for this criterion.

International trade effects

The diagnosis of TGE virus in domestic pigs would likely result in initial cessation of trade in live pigs and semen to some markets. Australia exports only small numbers of breeding pigs and quantities of semen. Export trade may only be interrupted temporarily under this scenario, with testing of pigs and semen donors for evidence of infection with TGE virus prior to export an option.

With an outbreak limited to a local population of herds, it is unlikely that export of meat would be significantly disrupted. There may be an initial reaction from some trading partners to halt meat imports in the short term, however, as there is no human health risk and the disease is endemic in many markets, trade should resume quickly. The OIE does not consider risk management measures for meat are warranted for TGE.

After consideration of these issues, the indirect effect of TGE on international trade was considered unlikely to be discernible at any level, except at the local level. Overall, this resulted in a rating of 'B' for this criterion.

Indirect impact on the environment

Transmissible gastroenteritis diagnosed in a local population of domestic pigs is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, TGE would have established in a broader population of commercial piggeries (including medium-large piggeries). A control and eradication program would have been mounted in response to the diagnosis of the disease in pigs.

The direct impact of transmissible gastroenteritis

Animal life or health

In this scenario, where TGE has spread to a more general population of domestic pigs including medium to large commercial piggeries, the direct impact on animal health of initial high piglet mortality, reduced growth rate amongst weaner pigs and disturbance on the breeding program was considered unlikely to be discernible at the national level, but of minor importance at the State level. This gave the disease a rating of 'D' for this criterion.

Environment

Because TGE is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of transmissible gastroenteritis

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

The eradication, control, surveillance/monitoring and compensation strategies/programs initiated in this scenario were considered to be similar, albeit more intense, than those described for scenario 3 above. The strategy for control and eradication is to use stamping out sparingly and to salvage as many animals as possible. Nonetheless, clinically affected animals and stock unsuitable for slaughter would need to be destroyed on farm.

The process of eradication could be prolonged and costly to producers and governments and may not be feasible if the disease became widespread. It should be noted that TGE has rarely been eradicated from a country. Nonetheless, the authors of AUSVETPLAN state that if tracing and surveillance can identify infected herds the disease could be eradicated provided there is strict adherence to movement controls.

On this basis, it was considered that the likely indirect impact of new or modified control programs would be unlikely to be discernible at the national level, and of minor importance at the State level, thus this criterion was rated as 'D'.

Domestic trade or industry effects

The quarantine and movement controls, including restrictions on the sale of live pigs within the restricted areas, will affect domestic trade in pigs, particularly breeding pigs. Domestic trade in meat should not be significantly disrupted as most pigs will still be able to be sent to slaughter.

Productivity on infected piggeries will be significantly reduced. It has been estimated (Mullan, et al., 1994) that with a 'moderate' outbreak of TGE where piglet mortality was 50%, net revenue would be reduced by 70% in the 6 months after the outbreak. Were the outbreak

'severe' (100% piglet mortality) a net reduction in revenue of 100% was predicted. AUSVETPLAN cites a 1990 report by Baldock and Webster who predicted that in the first year following infection, the annual cash surplus of a 100 sow piggery would be reduced by at least 50%.

In addition, with the detection of an exotic disease of pigs in Australia, it is likely that consumers may initially decrease pork consumption.

When these issues were taken into account, the indirect impact of TGE on domestic trade was considered unlikely to be discernible at the national or State level and of minor importance at the district or regional level. This resulted in a rating of 'C' for this criterion.

International trade effects

The diagnosis of TGE virus in domestic pigs would likely result in initial cessation of trade in live pigs and semen to some export markets. Australia exports only small numbers of breeding pigs and quantities of semen. Trade should only be interrupted temporarily with testing of pigs and semen donors for evidence of infection with TGE virus prior to export an option.

With an outbreak extending to a more general population of domestic pigs including medium and large commercial piggeries, export of meat may initially be disrupted. The initial reaction from some trading partners may be to halt meat imports in the short term, however, as there is no human health risk and the disease is endemic in many markets, trade should be able to resume. The OIE does not consider risk management measures for meat are warranted in regard to TGE.

After consideration of these issues, it was considered that the indirect effect of TGE on international trade would be unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

Indirect impact on the environment

The environmental issues associated with disposal of pigs slaughtered on infected premises would need to be addressed. However, as the strategy for eradication is to minimise the use of stamping out and salvage as many animals as possible, the number of animals to be disposed of should not be great. In view of this, it was considered that the indirect impact on the environment was unlikely to be discernible, and a rating of 'A' was thus assigned.

Indirect impact on communities

The economies of major pig producing areas could be affected if there was a widespread outbreak of TGE or if the disease became established. The productivity of infected farms will be reduced and in some cases producers may go out of business. This will impact on the economy of the local area. It has been estimated (Garner, et al., 2001) that, in general terms, for every one employed working in the pig industry, there will be another two people employed in providing goods and services to the pig industry directly or to employees in the pig industry.

Taking these issues into consideration, the indirect impact of TGE on rural communities and the environment was considered unlikely to be discernible at the national or State level, and of minor importance at the district or regional level. This resulted in a rating of 'C' for this criterion.

The overall impact of transmissible gastroenteritis

When the direct and indirect impacts of TGE were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences negligible

Scenario 3: Consequences very low

Scenario 4: Consequences low

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 47, Table 48 and Table 49. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were considered 'very low', 'very low' and 'low' respectively.

Table 47 TGE: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Low	Very low	Negligible
Scenario 4	Low	Low	Very low
		Overall likely consequences	**Very low**

Table 48 TGE: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Moderate	Very low	Very low
Scenario 4	Low	Low	Very low
		Overall likely consequences	**Very low**

Table 49 TGE: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Moderate	Very low	Very low
Scenario 4	Moderate	Low	Low
		Overall likely consequences	**Low**

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with TGE virus.

Table 50 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk meets Australia's ALOP (very low), risk management would not be required for TGE virus.

Table 50 TGE: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Very low	Low	Very low	Negligible
Backyard pigs	Very low	Extremely low	Very low	Negligible
Small commercial piggeries	Very low	Low	Low	Very low
			Overall annual risk	**Very low**

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Trichinella spiralis

Technical information

Background

Trichinellosis (or trichinosis or trichiniasis) is a parasitic zoonosis due to infection by the nematode, *Trichinella spiralis*. Trichinellosis in humans is acquired by the consumption of infected uncooked, or insufficiently cooked meat, especially pork.

Although the parasite is associated with temperate rather than tropical regions, it is present in most parts of the world. It does not occur in Australia. Old calcified trichinellosis cysts have occasionally been found at autopsy in humans in Australia, although these have invariably been in people who have immigrated from, or visited, enzootic countries.

Trichinella pseudospiralis, a non-encapsulating species of the genus *Trichinella*, has been identified in several wild animals (Tasmanian devils, spotted tail quolls and Eastern quolls) in Tasmania. Of 163 Tasmanian devils examined State-wide, 30% were infested (Obendorf, et al., 1990). This species is not known to be present in production animals in Australia (Geering, et al., 1995). Overseas a few cases of human infection due to *Trichinella pseudospiralis* have been reported (Ranque, et al., 2000). Recently another non-encapsulated *Trichinella* species (*Trichinella papuae*) was discovered in domestic and wild pigs in one remote area of Papua New Guinea (Owen, et al., 2000).

Agent taxonomy

Trichinella spiralis belongs to the family Trichinellidae, order Trichurida, class Enoplea, phylum Nematoda. Several subtypes of *Trichinella spiralis* have been identified.

Agent properties

The larvae are susceptible to heat (core temperature of meat of 60° C) and freezing (-15°C for 20 days or -25°C for 10 days) (Steele, 1982; Acha, et al. 1987). Arctic strains of the *Trichinella spiralis,* now identified as *Trichinella nativa*, are cold resistant. Low level gamma irradiation (0.15 kGy) can effectively kill the larvae. Infected meat can remain so for quite long periods. In some instances *Trichinella* larvae in muscle, can resist degradation for several weeks when stored at room temperature at 100% humidity (von Koller, et al., 2001). Larvae in pig muscle tissue buried in the ground for at least 90 days retained infectivity (Jovic, et al., 2001).

Some anthelmintics are effective against trichinellae. Thiabendazole is effective against the parasites in the intestinal mucosa while mebendazole is effective against the larvae in muscle.

Host range

Probably all mammals are susceptible to *Trichinella spiralis* infection, although infestation is most common in omnivores and carnivores. Of the domestic animal species, pigs are the main host followed by cats and dogs, although the incidence in horses is increasing. Horses are thought to become infested through eating chaff or milled rations that have been contaminated by rat or mouse carcasses. In wild animal species, infestations of bears, walruses, wild pigs, foxes, rats and mice are of the greatest epidemiological significance. Humans are susceptible and are regarded as end-hosts.

Epidemiology

Infestation occurs by ingesting undercooked or raw meat containing encysted larvae. Pigs have traditionally been the most important source of human infection. The disease perpetuates in pig populations generally through the feeding of uncooked or poorly cooked contaminated swill, or from eating rodent carcasses. Occasionally, transmission to pigs may occur via ingestion of larvae passed in faeces of an infested host (Hill, 1968). Congenital infection has been reported in humans and rats (Cosoroaba & Orjanu, 1998; Dubinsky, et al., 2001). Person to person transmission does not occur.

The incidence of trichinellosis in humans has been reduced to almost negligible levels in several countries where control programs are in place. Austria, Belgium, Denmark, Finland, Great Britain, Ireland, Portugal, Sweden, Switzerland and the Netherlands have not reported human trichinellosis due to eating local domestic or wild animals for over 20 years (Pozio, et al., 1996; Gottstein, et al., 1997).

In a recent epidemiological study undertaken in the United States of America (Gamble, et al., 1999), risk factors for infection of pig herds with trichinellae were identified. The exposure of pigs to wildlife, and to wildlife carcasses was significantly associated with infection. These authors concluded that both rodents and other small carnivores or omnivores (skunks, raccoons, opossums, etc), and the group of larger carnivores (foxes, bears, etc) provide a reservoir of infection. Spill-over from this sylvatic cycle into pig herds would then represent a chance event dictated by managerial practices and pig housing arrangements.

Following widespread adoption of modern intensive husbandry systems, the incidence of *Trichinella spiralis* in pigs and pig herds has declined markedly. The prevalence of *Trichinella spiralis* in domestic pigs varies significantly from country to country. Some of this variation may be due to the detection methods employed. Herd accreditation programs are present in some countries.

In North America, in the mid 1980s, serologic examination of pigs at slaughterhouses in the United States of America indicated that the prevalence of *Trichinella* infestation in commercial pork ranged from 0 to 0.7% (Hill, et al., 1985; Duffy, et al., 1985). More recently a serologic survey of 4078 pigs from 156 pig farms employing various management styles in northeastern United States of America showed a between herd prevalence of 0.15% and a within herd prevalence of 6.4% (Gamble, et al., 1999). Risk factors significantly associated with the seropositivity included access of pigs to wildlife and wildlife carcasses on the farm. The United States of America has recently conducted a *Trichinella* certification pilot study. As part of the development of this program diaphragm digestion and serological testing were performed on 221,123 pigs from midwestern United States of America. None of the pigs was found to be positive for *T. spiralis*. Of 14,121 pig sera collected for the National Animal Health Monitoring System (NAHMS) in 2000 only one serologically positive was found[51]. In Canada, a national serologic survey of sows during 1996 to 1997 showed that the domestic swine herd was free of trichinellosis (Appleyard, et al., 2002).

Trichinella is present in Mexico and parts of South America. A survey conducted in Toluca, Mexico, where pigs from commercial farms, as well as backyards pigs, are slaughtered did not detect *Trichinella* larvae by trichinoscopy or artificial digestion. However, specific antibodies were detected in 12.4% of sera (Monroy, et al., 2001). A review on trichinellosis in Mexico,

[51] Dr Ron DeHaven, Deputy Administrator Veterinary Services, United States Department of Agriculture. Submission on *Draft IRA Report* of pig meat.

Central and South America reported that 2.5% of pigs were seropositive in one area in Mexico and 13.4% of pigs were seropositive in Bolivia (Ortega-Pierres, et al., 2000). In Chile, *Trichinella* in pigs has progressively declined over the last 20 years from 0.683% in 1980 to 1984 to 0.0115% in 1990 to 1996 (Schenone, et al., 1999).

Within the European Union the prevalence of *Trichinella* infestation in pigs is also low, generally less than 0.001%. Nonetheless, in some regions a higher prevalence has been reported. For example in Finland prevalence in slaughter pigs ranged from 0% to 0.01% between 1995 and 2000, and in Spain prevalence ranged from 0.008% to 0.02% in the Extremadura region (Scientific Committee on Veterinary Measures relating to Public Health, 2001).

In contrast, in parts of central and eastern Europe, a higher prevalence of *Trichinella* in domestic pigs has been reported. In Croatia, the prevalence in slaughtered domestic pigs in four known endemic regions has risen from 0.24% in 1995 to 0.95% in 1999. In some of these pig herds, within herd seroprevalence ranged from 10% to 33% (Marinculic, et al., 2001). In Serbia between 1991 and 1993, *Trichinella* spread from three endemic regions to almost the whole country and, in 1999, prevalence in domestic pigs at slaughterhouses had risen from 0.03% in 1994 to 0.1% (Cuperlovic, et al., 2001). In Romania, between 1983 and 1993, human cases of trichinellosis increased from 217 to 3,649 before falling to 848 in 1999. Prevalence in slaughtered pigs fell slightly from 0.158% in 1993 to 0.150% in 1999 (Olteanu, 2001). In contrast to those countries above, only 0.00036% of over 18 million pigs were found to be infected in Poland in 1998 (Ramisz, et al., 2001).

In China, the seroprevalence of *Trichinella* in pigs slaughtered in abattoirs varied greatly between provinces, ranging from 0.0001% to a high of 34.2% while the seroprevalence of unauthorised domestic slaughtered pigs sold at village markets varied between provinces from 0.29% to 5.6% (Wang & Cui, 2001).

Trichinellae can also be found in wildlife, even where there is no evidence of the parasite in domestic pigs. In the Netherlands, where pig farming management practices and meat inspection have prevented trichinellosis in humans for over 20 years, wildlife remains a reservoir for *Trichinella* with prevalence in wild pigs estimated to be 6.8% in 1998 (van der Giessen, et al., 2001). Outbreaks of trichinellosis in humans sometimes occur in the European Union, particularly in France and Spain after the consumption of wild boar meat (Scientific Committee on Veterinary Measures relating to Public Health, 2001). Occasional cases of trichinellosis in humans have occurred in New Zealand following the consumption of home-killed pigs (Institute of Environmental Science and Research Ltd., 1997; Ministry of Agriculture and Forestry, New Zealand, 2001).

Clinical signs

In general, the incubation period ranges from 1 to 2 days for the intestinal phase to 2 to 8 weeks for the muscular phase. Pigs generally tolerate the parasite well and rarely show any clinical signs unless extremely heavily infected. Experimentally infected pigs have been observed to show signs similar to human infection (Corwin & Stewart, 1999). In other animals clinical signs of infestation are often absent or similar to those described for humans below, but milder (Geering, et al. 1987).

In humans, clinical signs occur when the larvae are being produced in the intestines, then migrating to, and encysting in, the muscle tissues. In many light infections, the host is never aware of the infection. The number of infective larvae ingested usually determines the severity

of the condition. In severe infections, initial signs, which may include upset stomach, vomiting, and diarrhoea, due to parasite activity in the intestinal mucosa, may be seen within 24 to 48 hours of infection. Symptoms associated with mass larval migration in the body a few days later include muscular pain, fever, headache and prostration. Symptoms associated with larvae encysting in the muscle cells include facial swelling, haemorrhages, fever and dyspnoea. Death can result from cardiovascular and neurological complications (Scientific Committee on Veterinary Measures relating to Public Health, 2001).

Pathogenesis

After the host ingests encysted larvae in raw or insufficiently cooked muscle tissue, the larvae are liberated in the small intestines by proteolytic digestion and become sexually mature in 2 to 6 days. The mature parasites are intracellular, within the intestinal mucosa. Mating occurs within the intestinal mucosa and the males die soon afterwards. Females penetrate deeply into the glands of Lieberkühn and may live for about six weeks. Larvae, about 1500 per female adult, pass through the lymphatics into the bloodstream and finally into skeletal muscles where they encyst. They are most numerous in the blood 8 to 25 days after ingestion. Cysts form around the larvae within 3 months and begin to calcify in 6 to 9 months. The larvae may remain alive for as long as 11 years in cysts, but do not develop any further until the muscle is eaten by another host (Geering, et al., 1995).

Pathology

The only pathology noted in natural infestation of pigs is the cyst formed by the muscle fibre around the larvae. Very heavy infections may be seen with the naked eye as minute sand-like specks in the muscle. Old lesions are calcified.

Immunology

The host can act against the parasite through non-specific mechanisms of resistance and through specific immune responses. Host species that become infected with *Trichinella spiralis* make antibodies against the antigens of the first stage larvae (Denkers, et al., 1991). While these responses are useful for diagnosing infections, these antibodies are not considered protective. The larvae can also invoke an immune response characterized by infiltration of polymorphonucleocytes, predominated by eosinophils. However, the parasite appears to successfully avoid host defences in a variety of ways and can survive for very long periods in the host.

Diagnosis

Two methods are commonly used to determine if muscle tissue is infected with *Trichinella*, trichinoscopy, a microscopic examination method where meat samples are examined under low powered microscopes, and the digestion methods, where meat is digested in artificial digestive juices and the free encysted larvae counted. Infections under three larvae per gram of tissue are not reliably detected by these methods (Scientific Committee on Veterinary Measures relating to Public Health, 2001).

For ante-mortem diagnosis, serology is preferable to biopsy, however serology will not detect antibodies until at least 3 weeks after initial infection. The ELISA is the test most commonly used and can detect infections as low as 1 larva per gram of tissue.

Transmission via meat

As discussed above, infestation occurs by ingesting undercooked or raw meat infected with encysted larvae. Pig meat has historically been the most important source of human infection, and large outbreaks still occur. In Yugoslavia, at the end of 2001 and early 2002 a large outbreak was reported that involved 247 people who had eaten smoked pork sausage infested with 23 larvae per gram. No deaths were recorded (Gamble, 1996; WHO, 2002). Heavy infestations can occasionally cause deaths in humans, with fewer than 2% of all reported cases being fatal. Heavily infested meat can contain over 3600 larvae per gram (Serrano, et al., 1999).

An European Commission report summarises information on the minimal infective dose of *Trichinella* larvae able to cause clinical trichinellosis in a person (Scientific Committee on Veterinary Measures relating to Public Health, 2001). One study reported that 70 live larvae were sufficient to cause clinical disease. Another paper suggested that meat containing at least 1 larva per gram is necessary which would correspond to an infective dose of approximately 150 larvae. The report estimated that the minimum infective dose could be around 100 to 300 larvae.

In pigs, an experimental oral dose of 20 larvae was enough to establish a low level of infestation in pigs without causing clinical signs (Gamble, 1996). Another study reported that experimentally an infection dose of 10 larvae caused infestation in one pig (Haralabidis, et al., 1989).

Trichinellae in meat can be destroyed by sufficient heating, refrigerating or some curing procedures. The larvae is destroyed by heating all parts of the pork muscle tissue to 60°C for at least one minute, or by freezing it at -17.8°C for at least 106 hours (US Department of Agriculture Code of Federal Regulations Title 9 Chapter III 318.10). Microwaving and irradiation can also destroy trichinellae (Zimmerman, 1983; Steele, 2000). During the dry curing of pork products, proscuitto, proscuittini, and Genoa salami, *Trichinella* larvae were progressively destroyed (Smith, et al., 1989). Rat bioassay was positive for viable trichinae in prosciutto prepared using a sodium chloride mixture at day 34 but not at day 48 of the curing process. No viable trichinae were detected for Genoa salami between 13 and 42 days post preparation, and for proscuittini between days 27 and 69.

Release assessment

R1 — the likelihood that a source herd is infected

The prevalence of *Trichinella* infestation in pigs varies considerably for different countries, dependent on pig management practices, from less than 0.001% in the European Union to 12.4% in a region of Mexico and up to 32.4% for one province in China. In one region of the United States of America a between herd prevalence of 0.15% was reported. Based on this information, it was considered that where *Trichinella* is endemic in the pig population, the likelihood of selecting slaughter-age pigs from an infested herd was ‘low’.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

Pigs kept in intensive production systems with good rodent control practices are less likely to become infested with *Trichinella* than those fed raw garbage or where wildlife have access. In the United States of America, the reported within herd prevalence of *Trichinella* was 6.4%. There was an association between seropositive herds and having access to live wildlife and

wildlife carcasses on the farm. In Croatia, the within herd seroprevalence for sows ranged from 10% to 33%.

Given the above information, it was considered that the likelihood of selecting an infested animal in an infested herd was ‘low’.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

Pigs infested with *Trichinella* do not show clinical signs. Pigs would need to be heavily infested to the point that minute sandy specks in the muscle can be seen with the naked eye, but this is likely to be a very rare occurrence. Larvae are detected at slaughter by trichinoscopy or digestion methods, but, as these constitute risk management measures, they are not considered in determining the unrestricted risk estimate. On the basis of this information, the sensitivity of ante-mortem, slaughter and processing requirements in detecting and removing pigs infested with *Trichinella* was considered to be ‘negligible’.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infested with *Trichinella* and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a ‘one minus the background rejection rate’. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered ‘extremely low’. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

Infective parasitic cysts are found only in muscle tissues. Consequently, it was considered that the likelihood that *Trichinella* would be present in meat harvested for export was ‘certain’.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

Trichinella larvae in meat are very resistant to physical and chemical factors even retaining infectivity in rotten meat for several months and, thus, it was considered that the likelihood that meat infested at the time of slaughter would remain so after the process of carcass maturation was ‘certain’.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

Trichinella larvae are susceptible to freezing and cooking at 60°C, but retain infectivity at temperatures in between for a considerable period of time. In this IRA it was assumed that chilled meat may be imported providing it met the Australian Standard for Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (Agriculture and Resource Management Council of Australia and New Zealand. Standing Committee on Agriculture and Resource Management, 2002). Thus, it was considered that there was a 'high' likelihood that meat infested with *Trichinella* at the completion of carcass maturation would remain infectious during transport and storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was considered that, in the absence of risk management and without considerations regarding the exporting country, there was a 'very low' likelihood that imported pig meat derived from an individual carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

Pig meat waste may be composed of bone, fat and/or muscle, however, *Trichinella* larvae are only present in muscle tissues. Although *Trichinella* larvae have a predilection for certain muscles, including the diaphragm, tongue and masseter, of which the tongue and most of the diaphragm is removed during the slaughter process, the larvae are distributed throughout other muscles. Meat may have as little as 0.003 to 0.021 larvae per gram (Gamble, et al., 1999) to as much as 3634 larvae per gram although these were experimental conditions (Serrano, et al., 1999). Infestation of pigs has resulted experimentally from a dose of 10 larvae. As such, in some instances pigs may need to consume a relatively large quantity of pig meat waste and in others a very small quantity to become infested. Given the range in the number of larvae per gram of pig meat, the composition of pig meat waste and the volume of waste consumed by a pig, it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of *Trichinella* to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

The likelihood that a pathogenic agent would remain viable after exposure to the environment will depend on the inherent 'stability' of each agent. In particular, this likelihood will reflect the agent's sensitivity to ultraviolet light, to ambient temperatures between approximately 10°C

and 35°C and to the putrefying effects of saprophytic organisms. It is recognised that pathogenic agents may be protected somewhat from exposure if they are sequestered within substantial portions of muscle tissue or not exposed directly to the environment.

As *Trichinella* larvae in meat can survive storage at room temperature and high relative humidity and burial, it is likely to survive in meat scraps at refuse sites for long periods under Australian environmental conditions.

This information led the Panel to consider that the likelihood that *Trichinella* would survive within meat scraps discarded in refuse for the period of time required for feral pigs to locate and subsequently scavenge the material was 'high'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = High
- Rural regions = High
- Large towns = Extremely low

When the annual likelihood for each region were combined, the overall annual likelihood of entry and exposure for feral pigs was found to be 'high'.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs, it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be discarded due to spoilage. Nevertheless as *Trichinella* larvae are quite resistant to physical factors, the period of time between discarding scraps and ingestion by either feral pigs or backyard pigs is unlikely to have a significant effect on agent viability.

Overall, the Panel considered that there was a 'high' likelihood that *Trichinella* larvae would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the median annual likelihood of entry and exposure for backyard pigs was found to be 'low'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was 'high' likelihood that *Trichinella* larvae would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'high'.

Exposure assessment for other susceptible species

Foxes, wild dogs, feral cats, Tasmanian devils, rats and mice are other susceptible species that could become directly infested with *Trichinella* larvae after eating contaminated pig meat scraps disposed at refuse sites or elsewhere. All these animals could gain access to discarded meat scraps with rats and mice often found at refuse sites. As discussed above, *Trichinella* larvae are quite resistant to physical factors and are likely to survive in meat scraps for a considerable period of time. Domestic dogs and cats may also be fed pig meat scraps infested with larvae as part of their diet. Other susceptible species, in particular, rats are often involved in the epidemiology of the disease and transmission to pigs. Rat populations appear to be able to maintain the infection, even in the absence of a known source of infested meat, probably through cannibalism. High numbers of larvae per gram of muscle have been reported from rats, an average of 293 larvae per gram with one third of the rats with greater than 1000 larvae per gram (Leiby, et al., 1990).

Based on this information, the annual likelihood of entry and exposure for other susceptible species was considered 'high'.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the four exposure groups - that is, feral pigs, pigs in backyard enterprises pigs in small commercial piggeries and other susceptible species. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs, but no subsequent spread to other domestic pig herds, other susceptible species or humans;
3. Secondary spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to domestic pigs (including backyard, small and/or medium-large commercial piggeries) - spread to other susceptible species - spread to humans if the agent is zoonotic.

Infestation occurs only by eating undercooked or raw muscle tissues containing the encysted larvae. It cannot spread directly from pig to pig. Secondary spread to other feral pigs could occur by cannibalism amongst these feral pigs. Carnivores or omnivores, such as rats, foxes, dingoes, wild dogs, foxes and feral cats may become infested by scavenging on feral pig carcasses. Crocodiles taking feral pigs may become infested. A natural cycle occurs in sylvatic carnivores. These animals represent an important reservoir for *Trichinella*. Overseas feral pigs represent an important reservoir, being a significant link between the sylvatic cycle and man, allowing transmission of *Trichinella* from wildlife to humans. Transmission from wildlife (such as rats) to domestic pigs may occur in situations such as outdoor commercial piggeries or backyard pigs. Secondary spread may also occur by feeding uncooked feral pig meat scraps to other pigs and dogs and cats.

The prevalence of *Trichinella* in wild boars varies in the different geographic regions and has been reported by the Netherlands as 6.8%, Finland 1.3%, France 0.02% to 0.03% and Spain 0.08% to 0.48% (Scientific Committee on Veterinary Measures relating to Public Health, 2001). In the United States of America, one survey of feral pigs in South Carolina found that 39% were seropositive for *Trichinella* (Gresham, et al., 2002). The prevalence of *Trichinella* in other wildlife also varies from region to region and species. In the Netherlands, the prevalence in foxes ranged from 3.9% in the eastern part of the country, 13.1% in the central part and 1.3% in the most western part (van der Giessen, et al., 2001). In Egypt, the overall prevalence of infection in rodents was 13.3%, with a higher prevalence in older rodents and those near to abattoirs (Loutfy, et al., 1999). In Tasmania, Australia the prevalence of *Trichinella pseudospiralis* has been reported as 30% in Tasmanian devils.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: low

Scenario 3: low

Scenario 4: low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds, other susceptible species or humans;
3. Secondary spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to domestic pigs (including backyard, small and/or medium-large commercial piggeries) - spread to other susceptible species - spread to humans if the agent is zoonotic.

As stated above, infestation occurs by eating undercooked or raw muscle tissues containing the encysted larvae. It cannot spread directly from pig to pig, except via cannibalism. Secondary spread could occur by carnivores and omnivores, such as rodents, feral pigs, dingoes, wild dogs, foxes and feral cats, scavenging on backyard pig carcasses that have been inadequately disposed of. Nonetheless, often pigs that die on the farm are buried or burned. Secondary spread may also occur by the feeding of uncooked pig meat scraps to dogs and cats on the property or by other susceptible species, such as rodents gaining access to inadequately disposed of meat scraps derived from the backyard pigs.

As backyard pigs are generally slaughtered for home consumption, any person that consumes *Trichinella* infested meat, which is inadequately cooked, is at risk of infection. Adequate freezing and cooking destroys the larvae.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: very low

Scenario 3: moderate

Scenario 4: low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds, to other susceptible species or humans;
3. Secondary spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to domestic pigs (including backyard, small and/or medium-large commercial piggeries) - spread to other susceptible species - spread to humans if the agent is zoonotic.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

As stated above, infestation occurs by eating undercooked or raw muscle tissues containing the encysted larvae. It cannot spread directly from pig to pig, except via cannibalism. Only those pigs within the piggery that consume the infested meat would initially become infested. There may be some further spread in the piggery by such means as tail biting. Nonetheless it is likely that the prevalence within the piggery would be very low. Secondary spread could occur by carnivores and omnivores, such as rodents, feral pigs, dingoes, wild dogs, foxes and feral cats, scavenging on pig carcasses that were inadequately disposed.

Pigs from small commercial piggeries are more likely to be sold and slaughtered at abattoirs. Infested meat purchased by the public may result in infection if inadequately cooked or if the meat has not been frozen. Secondary spread to other susceptible animals, such as rodents may occur if they gain access to these inadequately cooked pig meat scraps at, for example, refuse sites. Rodents may become a reservoir. Uncooked pig meat scraps may also be fed to household pets.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: very low

Scenario 3: moderate

Scenario 4: low

Other susceptible species

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of other susceptible species:

1. Containment of the disease within the sub-population of directly exposed animals - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds, to other susceptible species or humans;
3. Secondary spread to other susceptible species - but no subsequent spread to humans; and
4. Secondary spread to domestic pigs (including backyard, small and/or medium-large commercial piggeries) - spread to other susceptible species - spread to humans if the agent is zoonotic.

Other susceptible species such as rodents, dingoes, wild dogs, foxes and feral cats may scavenge discarded pig meat scraps. Some of these species, such as rodents and foxes, may become natural reservoirs with a sylvatic cycle occurring. Rats living in garbage dumps were found to be infested with *Trichinella*, however, rats in a rural area were not found to be infected (Acha, et al. 1987). Nonetheless, rodents close to piggeries have been found to be infested with high numbers of larvae and are apparently capable of maintaining infection without continuing access to meat scraps. From these reservoirs, spill-over to feral pigs or domestic pigs may occur and hence to humans. A survey of animals in Estonia showed that *Trichinella spiralis* infests brown rat and foxes as well as wild boars and domestic pigs (Jarvis, et al., 2001). Infestation is more likely to occur with outdoor piggeries and pigs in backyard enterprises where contact between wildlife and domestic pigs cannot be prevented. Domestic pigs housed indoors where there is good rodent and wildlife control are less likely to become infested.

Domestic dogs and cats may also become infested with *Trichinella* following the consumption of uncooked or inadequately cooked pig meat scraps, however, in these instances further spread was considered unlikely to occur.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: extremely low

Scenario 2: very low

Scenario 3: high

Scenario 4: low

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each exposure group generally follows a similar pattern with the exception of other susceptible species. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this 'no outbreak' scenario, *Trichinella* would have established in the directly exposed animal, or group of animals, but would not have spread to other animals or to humans. Because the disease is of low pathogenicity for pigs, it was assumed that it would not have been identified, and was contained for reasons other than human intervention. As such, under a 'no outbreak' scenario, the disease would not have any discernible direct or indirect impacts.

On this basis, a rating of 'A' was assigned to each of the direct and indirect criteria.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, *Trichinella* would have established in a broader population of feral pigs. The disease would be contained through identification of *Trichinella* as part of the routine testing of feral pig carcasses for export, and the mounting of an eradication program.

The direct impact of trichinellosis

Animal life or health

As described previously, clinical signs are very rarely seen in animals infested with *Trichinella* including wildlife. Hence, the likely impact of *Trichinella* in terms of animal health was considered to be unlikely to be discernible at any level and a rating of 'A' was assigned to this criterion.

Environment

As with outbreak scenario 1, it was considered that the direct impact on the environment was unlikely to be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of trichinellosis

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

It is considered likely that *Trichinella* would be diagnosed if spread occurred to a more general population of feral pigs as feral pig meat exports from Australia to the European Union are screened for trichinellae. Considering that around 3,000,000 kg of feral pig meat is exported to the European Union yearly and that if carcass weight averaged less than 60kg, at least 50,000 pigs are tested for *Trichinella* each year.

Under Australia's Exotic Animal Disease Response (EADR) Cost Sharing Agreement, trichinellosis is a Category 3 disease, that is, a disease of moderate public impact having the potential to cause significant (but generally moderate) national socio-economic consequences through international trade losses, market disruptions involving two or more States and severe production losses to affected industries, but have minimal or no effect on human health or the environment. If an outbreak occurred in animals in Australia, the control and eradication program will be funded 50% by governments and 50% by the applicable industry(s). No AUSVETPLAN manual is published for this disease.

Surveillance would need to be undertaken to determine the spread of the disease agent. If tracing indicated that a backyard enterprise or small commercial piggery was involved pigs may be tested and those positive destroyed. Depending on the extent of spread within the feral pig population or other susceptible species, it may not be possible to eradicate the disease. Control programs, such as rodent control and measures to preclude wildlife including feral pigs may need to be put in place by pig producers to prevent spread to domestic pigs.

Ongoing surveillance will likely be required to meet the recommendations of the OIE Code Chapter for trichinellosis for zone or country freedom of the domestic pig population. This Chapter recommends that meat is either tested for *Trichinella* or processed or comes from domestic pigs that were born and bred in a free country or zone. To establish that *Trichinella* does not exist in domestic pig population of the country or zone, a serological survey should be undertaken within a 5 year period and be carried out every third year, and there should be ongoing annual testing of the slaughter pig population.

If the disease cannot be eradicated from feral pigs there may need to be a public health campaign to educate hunters that meat from feral pigs must be well cooked.

After consideration of these issues, the indirect impact of control and eradication was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

Domestic trade or industry effects

The diagnosis of a zoonotic disease may initially affect consumption of pig meat. Although in this outbreak scenario where there is little involvement of domestic pigs, the public should be reassured quickly that there is little public health concern. Given this, the indirect impact of *Trichinella* on domestic trade and industry was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

International trade effects

Australia is free from *Trichinella spiralis*, and consequently trichinoscopy or artificial digestion methods are not practised at domestic abattoirs. Despite this, in order to comply with European Commission directives, feral pig meat and horse meat exports to the EU are screened for *Trichinella* prior to export and sent frozen. It is likely that other trading partners may impose additional measures on pork and or horse meat exports, such as testing of meat for larvae by artificial digestion methods. These measures may be able to be lifted if Australia could demonstrate that *Trichinella* had not established in the domestic pig population. This may require an extensive survey.

Given these factors, it was considered likely that the indirect impact of *Trichinella* on international trade would be unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

Indirect impacts on the environment

In this scenario, Trichinella is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reduced rural and regional economic viability, and a rating of 'A' was assigned this criterion.

Outbreak scenario 3 — secondary spread to other susceptible species and humans. Where the direct exposure group is other susceptible animals — secondary spread to other susceptible species only

In this scenario, *Trichinella* spreads to other susceptible animals and humans where the direct exposure group was a feral pig herd, backyard pig enterprise or small commercial piggery. Because *Trichinella* infestation in pigs is subclinical, it was assumed that the disease was diagnosed due to human illness and contained by a control program. In the case where the direct exposure group was another susceptible species such as rodents, in this scenario, *Trichinella* only spreads to other susceptible species not humans. Due to the subclinical nature of the disease in animals, it was considered that the disease would not be recognised.

The direct impact of trichinellosis

Animal life or health

As described previously, clinical signs are very rarely seen in animals infested with *Trichinella* including wildlife. Hence, the likely impact of *Trichinella* in terms of animal health was considered to be unlikely to be discernible at any level and a rating of 'A' was assigned to this criterion.

Environment

As with outbreak scenarios 1 and 2, it was considered that the direct impact on the environment was unlikely to be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of trichinellosis

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

This criterion was not rated identically for each exposure group.

Under this scenario, there is no secondary spread to domestic or feral pigs but there is spread to humans if the direct exposure group involves pigs. Because clinical signs are usually not evident in animals, the disease is likely to be detected following zoonotic spread. Once the disease has been diagnosed, the Commonwealth Emergency Animal Disease Response Cost Sharing Agreement may be implemented. It is likely that some surveillance would be undertaken to determine the spread of the disease agent. If tracing indicated that a backyard enterprise or small commercial piggery were involved, pigs are likely to be tested and those positive destroyed. Depending on the extent of secondary to other susceptible species, it may not be possible to eradicate the disease. Control programs such as rodent control and measures to preclude wildlife including feral pigs may need to be put in place by pig producers to prevent spread to domestic pigs.

After consideration of these issues, the indirect impact of control and eradication programs when the primary exposure group was feral pigs, a backyard enterprise or small commercial piggery, was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

When the primary exposure group was other susceptible animals, secondary spread has only occurred to other susceptible animals in this scenario and it was considered unlikely that the disease would be diagnosed, thus a rating of 'A' was assigned for this criterion.

Domestic trade or industry effects

This criterion was not rated identically for each exposure group.

As discussed above, it was considered likely that trichinellosis would be detected as a result of zoonotic spread when the primary exposure group involved pigs. As with scenario 2 there may initially be a decrease in pig meat consumption. A publicity campaign may be required to reassure the public (in this outbreak scenario there is no secondary spread to other pigs) and to remind consumers not to consume raw or inadequately cooked pork.

Given this, the indirect impact of *Trichinella* on domestic trade and industry, when the primary exposure group was feral pigs, a backyard enterprise or small commercial piggery, was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

When the primary exposure group was other susceptible animals, secondary spread has only occurred to other susceptible animals in this scenario and it was considered unlikely that the disease would be diagnosed, thus a rating of 'A' was assigned for this criterion.

International trade effects

This criterion was not rated identically for each exposure group.

Australia is free from *Trichinella spiralis* and testing of pig meat exports is not required for several markets including Singapore and Japan. Other markets such as the European Union and Russia currently require that pig meat and horse meat exported from Australia be screened for larvae. The diagnosis of trichinellosis in a human in Australia, which is linked to the consumption of local pig meat may affect our exports to countries such as Singapore and Japan. Surveillance of the domestic pig population may need to be undertaken to reassure our trading partners and testing of pig meat for export may be required.

Taking these factors into consideration, the likely indirect impact of *Trichinella* on international was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

When the primary exposure group was other susceptible animals, secondary spread has only occurred to other susceptible animals in this scenario and it was considered unlikely that the disease would be diagnosed, thus a rating of 'A' was assigned for this criterion.

Indirect impact on the environment

The indirect impacts on the environment of *Trichinella* such as an effect on biodiversity were considered unlikely to be discernible at any level, and a rating of 'A' was assigned this criterion.

Indirect impact on communities

The indirect impacts on the environment of *Trichinella* such as reduced rural and regional economic viability were considered unlikely to be discernible at any level, and a rating of 'A' was assigned this criterion.

Outbreak scenario 4 — secondary spread to domestic pigs

Under this scenario, *Trichinella* would have established in a broader population of commercial piggeries (including medium-large piggeries), and other susceptible species such as rodents and some consumers of pork would have become infested. A control program would have been mounted in response to the diagnosis of trichinellosis in humans.

The direct impact of trichinellosis

Animal life or health

As described previously, clinical signs are very rarely seen in animals infested with *Trichinella* including wildlife. Hence, the likely impact of *Trichinella* in terms of animal health was considered to be unlikely to be discernible at any level and a rating of 'A' was assigned to this criterion.

Environment

The consequences on this criterion would be similar to that described above for the previous scenarios, thus a rating of 'A' was assigned for this criterion.

The indirect impact of trichinellosis

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

As discussed above trichinellosis is covered by the Emergency Animal Disease Response Agreement. Industry and governments share the costs. It is likely that an eradication program would be implemented to ensure that Australia's commercial pig population was free from *Trichinella*. Depending on how widespread *Trichinella* is within the domestic pig population industry may take responsibility to control trichinellosis through official pig herd freedom accreditation programs similar to that being adopted in the United States of America. Abattoir surveillance through use of digestion methods would assist in identifying infected herds. It would not be feasible to control the disease in wildlife and so measures would need to be adopted to prevent contact between infected wildlife, particularly rodents, and commercial pigs.

If Australia wants to meet the OIE Code Chapter guidelines for a free country or zone in domestic pigs, ongoing surveillance of pigs at slaughter would be required.

Given this, the indirect impact of an eradication and control program was considered unlikely to be discernible at the national level, but likely to have a minor impact at the State level. This resulted in a rating of 'D' for this criterion.

Domestic trade or industry effects

As discussed above in the previous scenario, pork consumption may initially decrease and assurances would need to be provided to the public on the management of this disease.

The burden will be on the pig industry to provide the public with 'safe' pork. Consequently abattoirs may need to screen all carcasses for larvae and the costs of tests would have to be borne by either the government or the pig industry or both. To minimise costs of ongoing abattoir tests, accredited piggeries may become exempt from abattoir testing with full costs being borne by non-accredited piggeries. Organic pig farms and open range type commercial piggeries are unlikely to meet accreditation requirements and may find it difficult to remain economically viable. Overall, it was considered that the indirect impact of *Trichinella* on domestic trade and industry would be of minor significance at the national level. This resulted in a rating of 'E' for this criterion.

International trade effects

Trichinella in the domestic pig population is likely to have some impact on our export markets. Our market for chilled pork to Singapore may be affected at least initially. Nonetheless risk management measures, such as sourcing pigs for exports from accredited disease free herds and selecting carcasses which tested negative to abattoir surveillance programs such as digestion methods is likely to allow exports to those markets to resume after a period of time. However, it is possible that in the intervening time these markets could be lost.

Given this, the overall indirect impact of *Trichinella* on international trade was considered of minor significance at the national level. This resulted in a rating of 'E' for this criterion.

Indirect impact on the environment

The consequences on this criterion would be similar to that described above for the previous scenarios, thus a rating of 'A' was assigned for this criterion.

Indirect impact on communities

The indirect impacts on the environment of *Trichinella*, such as reduced rural and regional economic viability, were considered unlikely to be discernible at any level, and a rating of 'A' was assigned this criterion.

The overall impact of trichinellosis

When the direct and indirect impacts of *Trichinella* were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences very low

Scenario 3: Consequences very low (feral pigs, backyard pigs, small commercial piggeries), negligible (other susceptible species)

Scenario 4: Consequences moderate

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 51, Table 52, Table 53, and Table 54. It can be seen that the likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were considered 'low' in all cases. The likely consequences associated with the exposure of other susceptible species to infected pig meat scraps were considered 'low'.

Table 51 ***Trichinella spiralis*: summary of the consequences of exposure of feral pigs**

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Low	Very Low	Negligible
Scenario 3	Low	Very Low	Negligible
Scenario 4	Low	Moderate	Low
		Overall likely consequences	**Low**

Table 52 ***Trichinella spiralis*: summary of the consequences of exposure of backyard pigs**

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Very low	Very low	Negligible
Scenario 3	Moderate	Very low	Very low
Scenario 4	Low	Moderate	Low
		Overall likely consequences	**Low**

Table 53 ***Trichinella spiralis*: summary of the consequences of exposure of small commercial piggeries**

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Very low	Very low	Negligible
Scenario 3	Moderate	Very low	Very low
Scenario 4	Low	Moderate	Low
		Overall likely consequences	**Low**

Table 54 *Trichinella spiralis*: summary of the consequences of exposure of other susceptible species

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Extremely low	Negligible	Negligible
Scenario 2	Very low	Very low	Negligible
Scenario 3	High	Negligible	Negligible
Scenario 4	Low	Moderate	Low
		Overall likely consequences	**Low**

Human life or health

Separate to the above is consideration of the consequences to human life or health. *Trichinella spiralis* is considered a zoonosis. In humans, the clinical signs of infection may vary in intensity, depending on the extent of *Trichinella* invasion, the species involved and the immune response of the host. In many light infections, the host is never aware of the infection. In severe infections, initial signs, which may include upset stomach, vomiting, and diarrhoea, due to parasite activity in the intestinal mucosa, may be seen within 24 to 48 hours of infection. Symptoms associated with mass larval migration in the body a few days later include muscular pain, fever, headache and prostration. Symptoms associated with larvae encysting in the muscle cells include facial swelling, haemorrhages, fever and dyspnoea. Death can result from cardiovascular and neurological complications (Scientific Committee on Veterinary Measures relating to Public Health, 2001). Treatment for the disease includes the use of anthelmintics.

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with *Trichinella spiralis*.

Table 55 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly higher than Australia's ALOP (very low), risk management would be required for *Trichinella spiralis*.

Table 55 *Trichinella spiralis*: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Very low	High	Low	Low
Backyard pigs	Very low	Low	Low	Very low
Small commercial piggeries	Very low	High	Low	Low
Other susceptible species	Very low	High	Low	Low
			Overall annual risk	**Low**

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Cysticercus cellulosae

Cysticercus cellulosae is the larval stage of the tapeworm *Taenia solium* (Phylum *Nemathelminthes*, Class *Cestoda*, Order *Cyclophyllidea*, Family *Taeniidae*). The larval cysts or cysticerci occur in the muscles of the pig. Humans become infested by ingesting raw or undercooked pork containing viable cysticerci. Humans are the host for the adult tapeworm (taeniasis) and tapeworm segments and eggs are shed in human faeces. The life cycle of the parasite is completed when pigs ingest *T. solium* eggs and develop cysticercosis. Humans may also develop cysticercosis, by ingestion of *T. solium* eggs or by autoinfection, which can occur when a gravid segment of an intestinal tapeworm enters the stomach by reverse peristalsis with release of the oncospheres after digestion (Urquhart, et al. 1996). Cysticercosis in pigs is of little clinical significance; however, cysticercosis in humans can be associated with serious health consequences, depending upon sites of localisation.

Pigs do not develop taeniasis following ingestion of porcine cysticerci (Maravilla, et al., 1998). In this study, researchers seeking an experimental model for taeniasis other than humans or non-human primates evaluated animals including hamsters, gerbils, chinchillas, rabbits, cats, pigs and rhesus monkeys for suitability as definitive hosts for *Taenia solium*. Tapeworms failed to develop in rabbits, cats, pigs and rhesus monkeys, and developed (with the assistance of steroid treatment) only in hamsters, gerbils and chinchillas. As these latter species are not carnivorous, and are not permitted import into Australia, except in the case of hamsters for laboratory purposes, they cannot be regarded as an exposure group for imported pig meat.

As pigs can become infected only through exposure to human faeces and not via exposure to pig meat, the importation of pig meat or meat products infected with *Cysticercus cellulosae* will not be considered further in this IRA. Biosecurity Australia has advised Food Standards Australia New Zealand (FSANZ) of this matter. FSANZ may consider whether biosecurity measures for *Cysticercus cellulosae* are required to manage the risk to human life or health associated with the consumption of imported pig meat.

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Nipah virus

Technical information

Background

Nipah virus disease was diagnosed in Peninsula Malaysia in 1999 during an investigation of a major outbreak of a severe encephalitic disease in adult humans. Between October 1998 and May 1999, 265 human cases of encephalitis were reported from three states of Malaysia, with 105 fatalities (Parashar, et al., 2000). A previously undiscovered paramyxovirus, closely related to the Hendra virus, and named Nipah virus, after a village where the first case was reported, was found to be the cause of severe febrile encephalitis in people having close contact with infected pigs.

Over 1 million pigs out of a total population of 2.4 million pigs were slaughtered to contain the outbreak. It is believed the pigs had become infected as a result of the virus spilling over into the pig population from fruit bats of the genus *Pteropus*. It has been suggested that some pigs in Malaysia were previously infected with Nipah virus, probably in late 1996, but no evidence was provided to support this statement (Mackenzie, et al., 2001).

Nipah virus disease has been reported only in Peninsula Malaysia and Singapore. In the case of Singapore, Nipah virus infection was diagnosed in several workers in abattoirs that imported pigs from Malaysia (Paton, et al., 1999).

Nipah virus has been recovered from the urine of the fruit bat, *Pteropus hyomelanus*, and antibodies cross-reactive to Nipah virus have been detected in several members of the genus *Pteropus* in Malaysia and Cambodia (Yob, et al., 2001; Olson, et al., 2002). Bats of the genus *Pteropus* include fruit bats and flying foxes. These bats are found throughout South-east Asia, and from the west Indian Ocean islands of Mauritius, Madagascar and Comoro, along the sub-Himalayan region of Pakistan and India, through Indonesia, New Guinea, the South-west Pacific Islands (to Cook Islands) and Australia (Field, et al. 2002).

Agent taxonomy

Nipah virus is an enveloped single negative stranded sense RNA virus of genus Henipavirus, subfamily Paramyxovirinae, family Paramyxoviridae, order Mononegavirales.

Agent properties

No formal studies have been done on the physico-chemical properties of Nipah virus.[52] The virus is reported to be relatively unstable *in vitro* and can be readily disinfected with common detergents (Mohd Nor, et al., 2000).

Host range

Fruit bats and flying foxes of the genus *Pteropus* are considered to be the natural reservoirs for this virus (Mohd Nor, et al., 2000). Pigs, dogs, cats, horses and humans are known to be susceptible to infection with Nipah virus (Chua, et al., 2000). Once pigs are infected by direct or indirect contact with infected fruit bats, they become intermediate amplifying hosts for the

[52] Personal communication from Dr Deborah Middleton, Acting Diagnosis and Epidemiology Project Leader, CSIRO Livestock Industries, Australian Animal Health Laboratory (AAHL), Geelong, Australia.

virus and transmit it by direct contact to other pigs, animals and humans. Rodents and birds appear to be resistant to infection (Yob, et al., 2001).

Epidemiology

The main mode of transmission of Nipah virus between pig herds is by pig movements. At the time when early human cases were being reported in Malaysia, the 'fire sales' of pigs within the area where human and pig cases were occurring resulted in widespread dispersal of infected pigs in several regions within Malaysia. With active pig trading in many areas, the disease spread to a large number of pig herds. The sharing of boar semen between farms, use of unsterilised needles and equipment and transmission by dogs and cats were suspected to be other possible modes of transmission between pig herds (Mohd Nor, et al., 2000).

Within pig herds, transmission was by direct contact with excretory and secretory fluids such as urine, saliva, and the pharyngeal and bronchial secretions of infected pigs. Experimentally 4 days after infection, high levels of virus appear in the fluids and discharges of infected pigs even in those pigs without clinical signs. Excretion of virus continues in both diseased and asymptomatic animals until neutralising antibodies appear at 14 and 18 days post-infection (Middleton, et al., 2002).

Most human cases were pig workers who had direct contact with infected pigs. The case-fatality rate was 40%. Some cases (8%) had reported no contact with pigs, although the outbreak stopped once farms in infected areas were slaughtered out and pigs buried. However, other sources of infection in humans, such as infected dogs and cats, could not be excluded, especially in those people reporting no contact with pigs (Parashar, et al., 2000). There was no evidence of secondary cases in families, friends or contacts of pig farmers, abattoir workers or military personnel involved in the stamping out program, giving credence to the theory that humans are end-hosts for Nipah virus (Paton, et al., 1999; Parashar, et al., 2000; Ali, et al., 2001).

There are no published data illustrating the prevalence of infection between and within pig herds. Mortality is generally between 1% and 5%, but morbidity can approach 100% (Mohd Nor, et al., 2000). Over 900,000 pigs from 896 farms in infected areas (areas where human cases were reported) were destroyed to control the epidemic. This 'stamping out' policy ceased once an ELISA became available and a national swine testing and surveillance program commenced. A total of 889 farms were tested nationwide with 50 of these farms (5.6%) found to be positive to infection with Nipah virus. These positive farms were 'stamped out'. After the outbreak, new government regulations were introduced, permitting pig farming only in designated areas so as to minimise the risk of further outbreaks. In 2000, about 829 pig farms remained in Malaysia (Mohd Nor, et al., 2000).

Clinical signs

In pigs, the incubation period varies from 6 to 14 days (Mohd Nor, et al., 2000; Middleton, et al., 2002). Clinical signs may be very subtle or unrecognisable, and vary according with the age of the pig. Sows display a primarily neurologic syndrome, whilst respiratory signs predominate in growers (4 weeks to 6 months of age).

In growers the initial signs include fever and depression, followed by rapid and laboured breathing to a harsh non-productive and loud barking cough. Serious cases may expectorate blood and breathe with an open mouth. Neurological signs, such as trembling, twitches, spasms, myoclonus, paresis, incoordination or pain may accompany the respiratory syndrome. The loud

barking cough was regarded to be a characteristic feature of Nipah virus in pigs, with the disease commonly called 'barking pig syndrome' (Mohd Nor, et al., 2000).

Pathogenesis

Nipah virus has tropism for vascular and parenchymal tissues. While investigations into the sites of viral replication are not yet complete,[53] the virus elicits transitory syncytia, a characteristic feature of the morbilliviruses, also members of the paramyxovirus family, especially in systemic vascular tissues where the syncytial cells are usually found localised in the endothelium, in nervous tissues and in tracheobronchial epithelial tissues. The virus can also cause proteinaceous oedema, resulting in syndromes such as pulmonary oedema and meningitis (Hooper, et al., 2001).

Pathology

There are no pathognomic lesions ascribable to Nipah virus in pigs (Field, et al. 2002). However, systemic vasculitis, alveolitis and meningitis are common. In the lungs, lesions can be mild to severe with varying degrees of consolidation and emphysema, and petechial or ecchymotic haemorrhages. The bronchi and trachea may be filled with a frothy fluid, with or without blood, and there may be congestion and oedema in the brain. Occasionally the surface and cortex of the kidneys are congested (Mohd Nor, et al., 2000).

Immunology

Neutralising antibodies appear as early as 14 days post-infection in clinically and asymptomatically infected pigs (Middleton, et al., 2002).

Transmission via meat

There is no published information on the transmission of Nipah virus through ingestion of infected meat nor whether the virus is present in meat. It is known that the virus can be transmitted experimentally to pigs via the oral route using a dose of 50,000 $TCID_{50}$ (Middleton, et al., 2002).

The virus has been isolated from the tonsil, nose, blood, lung, and spleen of experimentally infected pigs inoculated orally or subcutaneously, between 2 to 10 days post-infection (Middleton, et al., 2002). Use of immunohistochemistry techniques by specific immunolabelling for Nipah virus antigen has resulted in identification of Nipah virus antigen in the muscular and endothelial layers of inflamed systemic blood vessels as well as in the tissues of the respiratory tract and the brain (Middleton, et al., 2002).

The Panel is unaware of any information linking human infection with the consumption of pig meat from infected pigs. Abattoir workers can become infected as a result of handling infected pigs during slaughter. Screen testing of 1469 Singaporeans, including abattoir workers, turf club workers, health care workers, meat inspectors, zoo workers, laboratory workers, public butchers, recreational staff workers and customs inspectors, confirmed that all 22 seropositive cases were only in abattoir workers known or suspected to have direct contact with live or freshly killed pigs at the two pig abattoirs in Singapore (Chan, et al., 2002). One abattoir worker in Singapore died following infection with Nipah virus.

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[53] Personal communication from Dr Deborah Middleton, Acting Diagnosis and Epidemiology Project Leader, CSIRO Livestock Industries, Australian Animal Health Laboratory (AAHL), Geelong, Australia.

Release assessment

R1 — the likelihood that a source herd is infected

Nipah virus disease has been reported in pigs only in Peninsular Malaysia in 1998 to 1999. To control the outbreak, an eradication program was put in place where 946 infected and at-risk farms were slaughtered out. Following the eradication program, there have been no known human cases of Nipah virus infection. Based on this information, it was considered that the likelihood of selecting slaughter-age pigs from an infected herd was ‘extremely low’.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

In pig herds that were infected with Nipah virus morbidity approached 100%. All ages of pigs can become infected. Persistent infection does not appear to be a feature of the disease, with virus isolated in blood or tissues for 2 to 3 weeks post-infection. Given this, it was considered that the likelihood of selecting an infected animal from an infected herd was ‘high’.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1—the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

Slaughter-age pigs infected with Nipah virus may be asymptomatic. If clinical disease is occurring, then it is usually expressed as a loud barking cough with other respiratory symptoms or, less commonly, neurological signs. Importantly, despite the systematic examination of all pigs of Malaysian origin that entered two of Singapore’s abattoirs, no clinical or pathological signs of Nipah virus infection were reported for these pigs. In view of these observations, the sensitivity of ante-mortem, slaughter and processing requirements was considered to be ‘extremely low’.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with Nipah virus and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a ‘one minus the background rejection rate’. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered ‘extremely low’. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

Viral antigen has been detected in the endothelium of the systemic blood and lymphatic vessels, and also in tonsillar tissues and lymph nodes. Furthermore, Nipah virus has been isolated from the blood of infected pigs, some of which is still retained in pig carcasses after exsanguination

at slaughter. Given this, the likelihood that Nipah virus would be present in the meat harvested for export was considered to be 'high'.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

There is no published information on the effect of pH on Nipah virus. However, other paramyxoviruses, such as rinderpest virus or Newcastle disease virus, are not inactivated at the pH (approximately pH 6.2) that accompanies carcass maturation (Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 2000). On this basis, a 'high' likelihood was assigned to this step.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

There is no information on the effects of cold storage on Nipah virus. However, paramyxoviruses are known to remain viable for long periods when kept at low temperatures. Rinderpest virus, for example, can survive in culture for at least 4 months at -20°C, 8 weeks at 4°C, and 1 week at 20°C to 25°C (Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 1996). In light of this, it was considered that there was a 'high' likelihood that meat infected at the completion of carcass maturation would remain infected during transport and cold storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was an 'extremely low' likelihood that imported pig meat derived from an individual carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was an 'extremely low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

There is no information on the oral infective dose of Nipah virus, or on the concentration of virus within the infected tissues of a pork carcass. However, oral administration of 50,000 $TCID_{50}$ of Nipah virus has, under experimental conditions, resulted in viraemia and virus excretion for 2 to 10 days. On this basis, it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of Nipah virus to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

This step describes the sensitivity of Nipah virus to ultraviolet light, to ambient temperatures ranging from 10°C to 35°C and to the putrefying effects of saprophytic organisms, bearing in mind that some time may be required before meat scraps are located by scavenging feral pigs.

There is no information on the impact of these factors on the viability of Nipah virus. Other members of the paramyxovirus family are known to be readily inactivated by heating, ultraviolet light and desiccation (Bellini, et al., 1998). Given this, it was considered that the likelihood that Nipah virus would survive within meat scraps discarded in refuse for the period of time required for feral pigs to locate and subsequently scavenge the material was ‘low’.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = Low
- Rural regions = Very low
- Large towns = Extremely low

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be 'low'.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was an 'extremely low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

On balance, the likelihood that Nipah virus would remain viable during the period prior to ingestion was considered to be 'moderate'.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'very low'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was an 'extremely low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

On balance, the Panel considered that there was a 'moderate' likelihood that Nipah virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'low'.

Exposure assessment for other susceptible species

The henipaviruses, of which Nipah virus and Hendra virus are the only discovered members, are capable of infecting several animal species including dogs and cats. However, rodents and birds trapped on infected farms in Malaysia were seronegative to Nipah virus, and may be resistant to infection. Wild dogs, dingoes and feral cats may gain access to discarded pig meat scraps and domestic dogs and cats may be fed pig meat scraps as part of their diet.

Experimentally it has been shown that cats can be infected when inoculated by both the oral and intranasal routes with 50,000 $TCID_{50}$ of Nipah virus.

Given this, it was considered that the annual likelihood of entry and exposure for other susceptible species was 'moderate'.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the four exposure groups - that is, feral pigs, pigs in backyard enterprises pigs in small commercial piggeries and other susceptible species. Outbreak

scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Nipah virus is highly contagious within a domestic pig population, with transmission occurring through close or direct contact between pigs. It was considered that the disease would not spread as effectively in sparser feral pig populations, where there is less opportunity for direct contact. Indeed, none of the feral pigs tested in Malaysia tested positive to Nipah virus (Yob, et al., 2001).

In Australia, feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs; however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

Infected pigs shed the virus to in-contact animals for 2 to 3 weeks. This would lead to spread within the immediate feral pig herd. If this herd is of limited size, the infection may die out. Additionally, respiratory or neurological symptoms may restrict the movement of infected

animals, and, thus, the opportunity for spread to other feral herds or to domestic pigs. If transmission to domestic pigs were to occur, it is likely that the disease would amplify and spread to other piggeries before a diagnosis was established. If domestic pigs became infected, pig producers, farm and abattoir workers may become infected. In fact the disease may be diagnosed in humans first, particularly if infected pigs are asymptomatic.

Other susceptible species can become infected with Nipah virus, in particular, feral dogs and cats. Additionally, pig hunters and their dogs may become infected. Dogs were suspected of spreading Nipah virus in Malaysia. With a widespread outbreak, horses may also become infected.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: high

Scenario 2: low

Scenario 3: very low

Scenario 4: very low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

Pigs infected with Nipah virus may be asymptomatic, or very subtly affected. Yet, as discussed above, Nipah virus is highly contagious in the domestic pig population, with transmission occurring through close or direct contact. Additionally, owners of backyard pigs are less likely to seek veterinary advice than commercial operators, and thus it is likely the virus will be amplified and spread to other piggeries may occur via pig movements. Nonetheless, whilst some pigs raised for personal consumption may be transferred between backyard holdings, most are raised for on-farm consumption.

As there is often close contact between backyard pig farmers and their pigs, zoonosis is likely. Similarly, the disease may spread to dogs, cats and horses located on the same premises as infected pigs.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: low

Scenario 3: moderate

Scenario 4: low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios. In particular, pig production at small commercial piggeries is generally more intensive, and includes a continual flow of pigs in and out of the facility. Most pigs from a small commercial piggery will go directly to slaughter, although some are purchased as stores or breeders for other commercial piggeries or for backyard piggeries. It is also relevant that managers of small commercial piggeries are more likely to observe and report unusual illness to a veterinarian, and that exotic disease events are likely to be identified earlier than might be the case for backyard piggeries. Workers on infected piggeries and abattoir workers would likely be exposed to the virus and may develop disease.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: low

Scenario 3: moderate

Scenario 4: low

Other susceptible species

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of other susceptible species:

1. Containment of the disease within the sub-population of directly exposed animals - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of domestic pigs - containment within a local population of backyard enterprises or small commercial piggeries - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs - exposure of medium-large commercial piggeries and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Dogs, cats, horses, goats and fruit bats have been shown to be susceptible to Nipah virus (Hooper, et al., 2001) (Mohd Nor & Ong, 2000; Hooper, et al., 2001; Middleton, et al., 2002). Fruit bats appear to serve as a reservoir of infection for pigs. In the field only one cat was confirmed as being infected with Nipah virus although there were reports of cats dying on infected premises (Bunning, et al., 2000; Hooper, et al., 2001). Dogs infected with Nipah virus may also show severe clinical signs of infection, resulting in death. However, a serological survey demonstrated that apparently healthy dogs from infected farms had antibodies to Nipah virus (Hooper, et al., 2001). Farmers raised the possibility that dogs and cats may play a role in spread of the virus from farm to farm (Bunning, et al., 2000). Owners of dogs and cats are likely to seek veterinary attention if the animals are showing clinical signs of disease. Nonetheless, the disease may not initially be diagnosed until either a human was infected or several animals became infected.

If Nipah virus infection was eradicated from the pig population but infection became widespread in fruit bat populations in Australia, rare sporadic outbreaks may occur in domestic pigs due to spill-over of virus from bats.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: high

Scenario 2: low

Scenario 3: very low

Scenario 4: extremely low

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each

exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed primary exposure group - no secondary spread

Under this scenario, Nipah virus would have established in the directly exposed animal, or group of animals, but would not have spread to other animals or to humans. This 'no outbreak' scenario would have resulted from low probability of contact between infected and susceptible animals or humans, rather than from human intervention. Indeed, because the disease may be of low pathogenicity for pigs, it was assumed that it would not have been identified.

The direct impact of Nipah virus infection

Animal life or health

Nipah virus infection in pigs may be asymptomatic. Some pigs may develop clinical signs with sows generally presenting a neurologic syndrome and growers a respiratory syndrome. While morbidity can be very high, mortality increases only marginally.

Dogs and cats are susceptible to infection with Nipah virus. Some dogs may develop symptoms resembling distemper while cats may become febrile and develop severe dyspnoea (Hooper, et al., 2001). Because the symptoms are not specific Nipah virus may not be identified in an individual animal.

On this basis, the direct impact of Nipah virus on animal health was considered to be unlikely to be discernible at any level except locally. This resulted in a rating of 'B' for this criterion.

Environment

Although Nipah virus infects fruit bats (it was considered unlikely that this would occur via contact with infected pigs), there is no evidence that the virus causes clinical disease in these animals. If dingoes were infected, a few may show clinical signs of disease. Overall, it was considered that the direct impact on the environment was unlikely to be discernible except locally, hence the rating assigned to this criterion was 'B'.

The indirect impact of Nipah virus infection

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this scenario, where the disease is contained within the initial exposure group with no secondary spread, it was considered unlikely that the primary case of Nipah virus infection would be diagnosed either in pigs or other susceptible animal species. Given this, it was considered unlikely that the indirect impact of new or modified control programs would be discernible at any level, and the rating assigned to this criterion was therefore 'A'.

Domestic trade or industry effects

As discussed above, it was considered unlikely that Nipah virus would be diagnosed in a single pig herd or a small group of other susceptible animal species. On this basis, the indirect impact

of Nipah virus on domestic trade and industry was considered unlikely to be discernible at any level, and thus the rating assigned to this criterion was 'A'.

International trade effects

As the disease is unlikely to be diagnosed if contained within the direct exposure group, it was considered that the indirect effects of Nipah virus on international trade was unlikely to be discernible at any level, and a rating of 'A' was therefore assigned to this criterion.

Indirect impact on the environment

In this scenario, Nipah virus is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this scenario, Nipah virus is unlikely to lead to any indirect impacts on the community, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, Nipah virus would have established in a broader population of feral pigs, and spread to feral pig hunters, pig handlers (owners) or abattoir workers in contact with the secretions and viscera of affected animals. The disease would be contained in pigs following the diagnosis of illness in humans and the mounting of an eradication program.

The direct impact of Nipah virus infection

Animal life or health

Nipah virus may be of low pathogenicity in pigs, although in some cases respiratory or neurological signs may be present. As such, the direct effect on animal life or health is, under this scenario, unlikely to be discernible at any level except locally. This resulted in a rating of 'B' for this criterion.

Environment

Although Nipah virus infects fruit bats (it was considered unlikely that this would occur via contact with infected pigs), there is no evidence that the virus causes clinical disease in these animals. If dingoes were infected, some may show clinical signs of disease. Overall, it was considered that the direct impact on the environment was unlikely to be discernible except locally, hence the rating assigned to this criterion was 'B'.

The indirect impact of Nipah virus infection

New or modified eradication, control, surveillance/monitoring and compensation strategies/programs

Nipah virus is listed as a Category 1 disease under the Emergency Animal Disease Response Agreement[54]. Category 1 diseases are funded 100% by governments and are those that predominantly seriously affect human health and/or the environment but may only have

[54] http://www.aahc.com.au/eadp/response.htm

minimal direct consequences to the livestock industry. A control and eradication program would be mounted. Because of the risk to public health, pig farms located within areas known or suspected to contain infected feral pigs are likely to be quarantined and tested, and biosecurity arrangements reviewed and strengthened. Serosurveys of feral pig populations would be carried out. Although it would not be feasible to eradicate feral pigs, thinning the population may lower the reproduction rate and thus encourage the disease in feral pigs to die out.

On balance, the indirect impact of Nipah virus on eradication and control programs was, under this scenario, considered unlikely to be discernible at the national or State level, and of minor significance at the district or regional level. This resulted in a rating of 'C' for this criterion.

Domestic trade or industry effects

If Nipah virus was identified in feral pigs, then movement restrictions on domestic pigs within and from affected areas would be likely. Pig hunters may be less inclined to hunt feral pigs because of public health risks, and some abattoirs that process feral pigs may close. This may be followed by a restructuring of sections of the pig industry to reduce risk of contact between feral pigs and domestic pigs. Because it is a serious zoonosis, the disease might also create negative perceptions about the consumption of pig meat, and thus reduce domestic sales.

On balance, the indirect impact of Nipah virus on domestic trade and industry was, under this scenario, considered unlikely to be discernible at the national level and of minor impact at the State level. This resulted in a rating of 'D' for this criterion.

International trade effects

An outbreak of Nipah virus in Australia is likely to have an immediate impact on international trade, including the temporary cessation of exports of pigs, pig semen and pig meat (domestic and feral). Export of horses, cats and dogs may also be affected, as was the case in Malaysia. In particular, it might be difficult for Australia to regain access for feral pig meat to Europe, especially if the disease was not eradicated from feral pigs. Even if Australia was able to prove disease freedom (compartmentalisation) in domestic pigs, then trading partners may continue to resist importing Australian pigs and their products. Although there are no OIE guidelines for Nipah virus, response of the world trading community to Malaysia's outbreak was a ban by many countries on Malaysian pigs and pig products and horses until after Malaysia was able to demonstrate that its entire pig population was free from disease. Export markets for Australian pig meat are valued at approximately $230 million with Singapore and Japan the major markets. Both of these markets would be highly sensitive to an outbreak of Nipah virus in pigs in Australia. Australia exports horses worldwide, with over 2200 exported in 2001/02 (Australian Racing Board, 2002).

Taking these factors into consideration, the likely indirect effect of Nipah virus on international trade was, under this scenario, considered to be of minor impact at the national level. This resulted in a rating of 'E' for this criterion.

Indirect impact on the environment

In this scenario, Nipah virus is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

Due to the zoonotic potential of Nipah virus there is likely to be some public health concerns within the local community, which might result in people leaving the affected area. The indirect impacts of Nipah virus on the sustainability of rural communities, were considered unlikely to be discernible at the national, State or district level, but of minor importance to the local area, and a rating of 'B' was assigned this criterion.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, Nipah virus would have established in a local population of backyard piggeries or small commercial piggeries, and spread to pig handlers or abattoir workers and other susceptible species. The disease would be contained through the diagnosis of illness in humans or animals, and the mounting of an eradication program.

The direct impact of Nipah virus infection

Animal life or health

Nipah virus may be of low pathogenicity in pigs but respiratory and neurological signs can be present. In naïve pigs severe coughing, fever and depression may be evident. In other susceptible species, such as dogs and cats clinical disease can be severe. Because, in this outbreak scenario spread has only occurred to a local population of animals, its direct effect on animal life or health was considered likely only to be discernible at the local level. This gave the disease a rating of 'B' for this criterion.

Environment

Although Nipah virus infects fruit bats (it was considered unlikely that this would occur via contact with infected pigs), there is no evidence that the virus causes clinical disease in these animals. If dingoes were infected, some may show clinical signs of disease. Overall, it was considered that the direct impact on the environment was unlikely to be discernible except locally, hence the rating assigned to this criterion was 'B'.

The indirect impact of Nipah virus infection

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

It was explained above that although there are no specific AUSVETPLAN strategies in place for Nipah virus, it is listed as a Category 1 public health risk disease under the Emergency Animal Disease Response Agreement and, thus, the costs of a response program would be funded by the governments. This program would involve targeted slaughter of domestic and feral pig populations in affected areas, and movement restrictions on pigs at risk. Disposal would normally be by burial or cremation. Garner and others (Garner, et al., 2001) estimated that in an epidemic situation the cost of disposal of carcasses and piggery clean-up was $600 per pig. If the outbreak had occurred in a coastal region, investigations and control programs are likely to extend to the flying fox and fruit bat populations.

In consideration of this, the indirect impact of eradication and control programs was, under this scenario, considered unlikely to be discernible at the national or State levels, and of minor significance at the district or regional level. This resulted in a rating of 'C' for this criterion.

Domestic trade or industry effects

If Nipah virus were identified in backyard piggeries or small commercial piggeries then it is likely that movement and sale restrictions would be imposed as a part of the control and eradication program. It is also important that Nipah virus has serious public health implications, and that an outbreak of any magnitude is likely to reduce demand for pig meat and lead to a fall in market price and consumption.

On balance, the indirect impact of Nipah virus on domestic trade and industry was, under this scenario, considered to be of minor significance to the affected State. Thus, a rating of 'D' was assigned to this criterion.

International trade effects

The impact of Nipah virus disease on international trade is likely to be similar under this scenario as was described for scenario 2 (see above). A rating of 'E' was assigned to this criterion.

Indirect impact on the environment

The disposal of pigs by burial or cremation can present environmental problems. However in this scenario the disease has spread only to a local population of backyard enterprises or small commercial piggeries. Hence the numbers slaughtered would not be great and it was considered unlikely to lead to any discernible indirect impact on the environment other than at the local level. Thus, a rating of 'B' was assigned to this criterion.

Indirect impact on communities

Due to the zoonotic potential of Nipah virus there is likely to be some public health concerns within the local community, which might result in people leaving the affected area. The indirect impacts of Nipah virus on the sustainability of rural communities, were considered unlikely to be discernible at the national, State or district level, but of minor importance to the local area, and a rating of 'B' was assigned this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, Nipah virus would have established in a broader population of commercial piggeries (including medium-large piggeries), and is likely to have spread to pig handlers or abattoir workers. An eradication program would have been mounted in response to the isolation of the agent from affected humans, or from the diagnosis of the disease in pigs or other animals.

The direct impact of Nipah virus infection

Animal life or health

Although Nipah virus may be of low pathogenicity in pigs, a large-scale outbreak in a naive population would likely result in clinical illness such as coughing pigs, a reduction in feed

conversion and a general decrease in the efficiency of affected piggeries. Morbidity is high and mortality is usually low.

It is also relevant that in a large-scale outbreak, a few domestic dogs and cats may be affected. A serological survey carried out in Malaysia during the 1998 to 1999 outbreak showed that approximately 50% of dogs from infected farms had antibodies to Nipah virus. The disease is not invariably fatal in dogs, although deaths of farm dogs were reported during the Malaysian outbreak (Hooper, et al., 2001; Field, et al. 2002). The disease appears to generally be fatal for cats. Horses may also be infected.

On balance, the direct effects on animal health were, under this scenario, considered unlikely to be discernible at the national or State level and of minor impact at the district or regional level. This resulted in a rating of 'C' for this criterion.

Environment

Although Nipah virus infects fruit bats (it was considered unlikely that this would occur via contact with infected pigs), there is no evidence that the virus causes clinical disease in these animals. If dingoes were infected, some may show clinical signs of disease. Overall, it was considered that the direct impact on the environment was unlikely to be discernible except locally, hence the rating assigned to this criterion was 'B'.

The indirect impact of Nipah virus infection

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

Although on a larger scale, the control, monitoring and surveillance programs initiated under this scenario are likely to be similar to those described for scenario 2 and scenario 3. These would involve targeted slaughter of domestic and feral pig populations in affected areas, and movement restrictions on pigs at risk. If the outbreak had occurred in a coastal region, investigations and control programs are likely to extend to the flying fox and fruit bat populations. If other susceptible animals such as horses, dogs and cats were infected these animals may be euthenased. The cost of these programs is unlikely to be discernible at the national level, but of minor impact at the State level. This resulted in a rating of 'D' for this criterion.

Domestic trade or industry effects

If Nipah virus were widespread in commercial piggeries then it is likely that movement and sale restrictions would be imposed as a part of the control and eradication program (see above). Garner and others (Garner, et al., 2001) have estimated that if it were necessary to control infection in two regions, then approximately 15 farms would be subject to stamping out, and, due to loss of export markets exacerbated by negative perceptions among local consumers, the gross income of the national pig industry would fall by around 3%. Should Nipah virus become established the study estimated that the gross national income of the pig industry would decrease by 0.1% annually. Outbreaks may also reduce demand for pig meat, leading to a fall in prices and a fall in overall consumption.

Depending on the location of the outbreak, and the involvement of horses, horse racing and horse events may be prohibited. Horse racing contributes significantly to government revenue.

On balance, the indirect impact of Nipah virus on domestic trade and industry was, under this scenario, considered unlikely to be discernible at the national level and of minor impact at the State level, which resulted in a rating of 'D' for this criterion.

International trade effects

The indirect impact of Nipah virus on international trade is likely to be similar under this scenario as was described for scenario 2 and scenario 3 (see above). A rating of 'E' was thus assigned to this criterion.

Indirect impact on the environment

The disposal of pigs by burial or cremation can present environmental problems particularly where large numbers of pigs may be involved, which may be the case under this scenario. Hence the indirect impact on the environment was considered unlikely to lead to any discernible impact at the national or State level, but would have a minor impact at the district or regional level. Thus, a rating of 'C' was assigned to this criterion.

Indirect impact on communities

Under this scenario, slaughter programs and movement restrictions are likely to threaten the economic viability of pig producers in affected areas, as well as the viability of some support industries. These would include the pig transport industry, the various feed industries and the slaughter and meat processing industries. Collectively, these industries provide support for many rural communities in New South Wales, Victoria, Queensland, Western Australia and South Australia. Moreover, the threat of disease in humans might result in reduced tourism and lead to people leaving affected districts. A widespread outbreak in which horses were involved could have social consequences for people involved in horse riding.

On balance, the indirect impact of Nipah virus on rural communities was considered unlikely to be discernible at the national level, but would have a minor impact at the State level. This resulted in a rating of 'D' for this criterion.

The overall impact of Nipah virus

When the direct and indirect impacts of Nipah virus were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences moderate

Scenario 3: Consequences moderate

Scenario 4: Consequences moderate

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 56, Table 57, Table 58, and Table 59. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were considered 'low', 'moderate' and 'moderate', respectively. The likely consequences associated with the exposure of other susceptible species to infected pig meat scraps were considered 'low'.

Table 56 Nipah virus: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	High	Negligible	Negligible
Scenario 2	Low	Moderate	Low
Scenario 3	Very low	Moderate	Very low
Scenario 4	Very low	Moderate	Very low
		Overall likely consequences	**Low**

Table 57 Nipah virus: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Low	Moderate	Low
Scenario 3	Moderate	Moderate	Moderate
Scenario 4	Low	Moderate	Low
		Overall likely consequences	**Moderate**

Table 58 Nipah virus: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Low	Moderate	Low
Scenario 3	Moderate	Moderate	Moderate
Scenario 4	Low	Moderate	Low
		Overall likely consequences	**Moderate**

Table 59 Nipah virus: summary of the consequences of exposure of other susceptible species

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	High	Negligible	Negligible
Scenario 2	Low	Moderate	Low
Scenario 3	Very low	Moderate	Very low
Scenario 4	Extremely low	Moderate	Negligible
		Overall likely consequences	**Low**

Human life or health

Nipah virus can cause fatal encephalitis in people who come into direct contact with infected pigs. The main clinical signs in humans included fever, headache, dizziness, vomiting and a reduced level of consciousness and seizures (Goh, et al., 2000). The virus does not generally seem to be spread from person to person. Family members of the affected individuals were not ill, and people living in the same area, but who were not directly involved in pig farming did not develop the disease (Farrar, 1999).

In Peninsular Malaysia in 1998 to 1999, 265 human cases of Nipah virus infection were diagnosed, of which 105 died (a case-fatality rate of almost 40%). Most cases of Nipah virus infection in Malaysia were pig farmers, however, a few cases were reported in people with other occupational exposure to pigs (Sahani, et al., 2001). Five cases were reported among abattoir workers who slaughtered pigs. Following this, an investigation was conducted to determine the prevalence of exposure to Nipah virus among abattoir workers in Malaysia (Sahini et al, 2001). Seven of 435 (1.6%) abattoir workers who slaughtered pigs showed antibody to Nipah virus. In Singapore, 22 of 521 abattoir workers involved with slaughtering pigs from Malaysia tested positive to Nipah virus and, of these, 12 (2.3%) had clinical signs and one died (Paton, et al., 1999; Chan, et al., 2002). Of the 12 people with clinical signs, nine presented with encephalitis, two with pneumonia and one with both encephalitis and pneumonia.

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;; and
- Combination of partial annual risks to give an estimate of 'overall annual risk'..

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10). The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with Nipah virus.

Table 60 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly greater than Australia's ALOP (very low), risk management would be required for Nipah virus.

Table 60 Nipah virus: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Extremely low	Low	Low	Very low
Backyard pigs	Extremely low	Very low	Moderate	Very low
Small commercial piggeries	Extremely low	Low	Moderate	Low
Other susceptible species	Extremely low	Moderate	Low	Low
			Overall annual risk	**Low**

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Post-weaning multisystemic wasting syndrome

Technical Information

Background

Post-weaning multisystemic wasting syndrome (PMWS) is an emerging disease that was first reported in Canada in 1996 (Harding, 1997). Retrospective analysis of tissue samples from Spain in 1986 demonstrated PMWS-associated lesions, indicating that the disease was present in the 1980s (Rosell, et al., 2000b). The syndrome has now been described in most countries (Allan & Ellis, 2000), but not in Australia. PMWS has recently been reported on the North Island of New Zealand.

PMWS is considered to be a multi-factorial disease of pigs, in which a necessary, but apparently not sufficient factor alone is the presence of porcine circovirus type 2 (PCV2) (Ellis, et al., 1998; Kennedy, et al., 2000; Bolin, et al., 2001). Porcine circovirus type 2 is present worldwide including Australia (Buddle, et al., 2003). Porcine circovirus type 2 has also been implicated in other conditions, including porcine dermatitis nephropathy syndrome (PDNS) (Rosell, et al., 2000a), reproductive disorders in sows (O'Connor, et al., 2001), respiratory disease in weaned and fattening pigs (Harms, et al., 2002) and congenital tremors (Stevenson, et al., 2001). The role of PCV2 in these other conditions has not been fully elucidated and, in some cases, the link is controversial. Congenital tremors occurs in Australia, as does PDNS, although the disease would appear to be rare, being reported sporadically (Cameron, 1994).

Agent taxonomy

Porcine circovirus (PCV) was first detected as a contaminant of a pig kidney cell line (PK-15) in Germany in 1974 and was noteworthy due to the novelty of its circular, single-stranded DNA genome (Tischer, et al., 1982). Porcine circovirus has been assigned to the family *Circoviridae*. Under experimental conditions this virus did not produce disease in pigs (Tischer, et al., 1986). The original PCV contaminant of the PK-15 cell line is now referred to as PCV1.

The porcine circoviruses (PCV2) associated with the wasting syndrome in pigs are closely related, exhibiting greater than 95% nucleotide sequence identity. However, they are significantly different from PCV1, as is shown by less than 76% homology (Hamel, et al., 2000).

Agent properties

There is limited information on the physico-chemical properties of PCV. One study demonstrated that PCV1 was stable at pH 3 and stable at 56°C and 70°C for 15 minutes (Allan, et al., 1994). Chicken anaemia virus, which also belongs to the family *Circoviridae* resisted heating at 80°C for 15 minutes (Yuasa, et al., 1979). Porcine circovirus 2 is readily isolated from tissue samples that have been stored at -70°C (Ellis, et al., 1998). Porcine circovirus 2 was shown to be resistant to some disinfectants but virus titres were significantly reduced by sodium hydroxide and Virkon S (Royer, et al., 2001).

Host range

Pigs are considered the principal host for porcine circoviruses. There is one report of a circovirus isolate from a bovine which is genetically closely related to PCV2 (Fenaux, et al.,

2000). These authors suggested that the bovine circovirus may be of porcine origin. Several studies were unable to detect antibodies to PCV1 or PCV2 in a wide range of animals including cattle, horses, sheep, cats, dogs, mice, rabbits, ducks and humans using indirect immunofluorescent testing, ELISA and immunoperoxidase monolayer assay (Allan, et al., 1994; Ellis, et al., 2001; Rodriguez-Arrioja, et al., 2002b). In contrast, one study reported antibodies to PCV in humans, mice and cattle, as determined by indirect immunofluorescence assay, suggesting that these species may have been exposed to a PCV-like virus (Tischer, et al., 1995).

Epidemiology

Increasing evidence continues to support the hypothesis that PCV2 is essential for the development of PMWS. Initial attempts to reproduce clinical PMWS with PCV2 inocula alone were generally unsuccessful; however, histological lesions typical of PMWS were experimentally reproduced on several occasions (Kennedy, et al., 2000). Studies also demonstrated that clinical PMWS could be consistently reproduced by co-inoculation with PCV2 and porcine parvovirus or porcine reproductive and respiratory syndrome (PRRS) virus (Ellis, et al., 1999; Allan, et al., 1999; Kennedy, et al., 2000). More recently, clinical PMWS has been induced after experimental inoculation with PCV2 alone in caesarean derived colostrum deprived or conventional pigs (Reynaud, et al., 2000; Harms, et al., 2001; Bolin, et al., 2001; Ladekjaer-Mikkelsen, et al., 2002a). In the field, other infections or diseases such as PRRS virus, porcine parvovirus, encephalomyocarditis virus and hepatitis E virus are often found in farms experiencing PMWS (Ellis, et al., 2001).

It is generally considered that although PCV2 is essential for the development of PMWS, other factors are required to induce the full spectrum of clinical signs and lesions associated with advanced PMWS in conventional pigs. These factors may include co-infecting pathogens, immune stimulation such as vaccines, environmental factors and stress such as transport and mixing of pigs (Allan & Ellis, 2000). It is not known if different PCV2 isolates differ in virulence. A recent study demonstrated that PMWS could be produced experimentally in pigs using a PCV2 isolate from a region (Sweden) apparently free, until recently, from PMWS (Allan, et al., 2003). The authors suggested that the status of the host and its environment is an important factor in the development of clinical disease. It has also been hypothesised that an unknown disease agent may be the trigger for activation of PCV2 and hence expression of the disease (Rathkjen, et al., 2003).

PMWS has been described in most types of farms, ranging in size from 30 sows to 10,000 sow herds. Individual expression of the disease seems to be a key point; in a given pen only some individual pigs exhibit clinical signs (Segales & Domingo, 2002). Although morbidity can be low, evidence of PCV2 infection is often widespread within a herd (Quintana, et al., 2001).

The mode of transmission of porcine circoviruses has not been properly investigated. Several studies have isolated PCV2 nucleic acid from nasal secretions, faeces, urine, tonsillar and bronchial swabs suggesting that the virus could be transmitted by oronasal, faecal and urinary routes (Harms, et al., 2001; Resendes, et al., 2002; Calsamiglia, et al., 2002). Direct contact transmission of virus has been demonstrated; pigs inoculated 42 days previously transmitted virus to control pigs (Bolin, et al., 2001). Although transmission studies have not been conducted with semen, PCV2 nucleic acid has been detected in boar semen, up to 47 days post-infection (Larochelle, et al., 2000).

There is evidence that some animals may be persistently infected with virus or viral nucleic acid detected in secretions and serum for prolonged periods. Under experimental conditions, viral nucleic acid was detected in nasal, faecal and urine samples, tonsillar swabs and serum of clinically healthy pigs up to 69 days post-inoculation, at which time the experiment concluded (Resendes, et al., 2002). These authors concluded that PCV2 could cause subclinical long lasting infection. Another study found 3 of 29 pigs were viraemic (based on PCR) for 16 weeks (Rodriguez-Arrioja, et al., 2002a). Viral nucleic acid was also detected in serum and tissues of clinically healthy pigs 2 months after an outbreak of PMWS. However, no microscopic lymphoid lesions of PMWS nor PCV2 nucleic acid in serum was found in pigs of the same batch at slaughter, suggesting that the virus had cleared from the pigs by the time they were slaughtered at approximately 26 weeks of age (Quintana, et al., 2001). In contrast, PCV2 nucleic acid was detected in sera collected from 203 of 368 (52.6%) healthy slaughter-age pigs (Liu, et al., 2002).

Viral nucleic acid has also been detected in tissues from the cerebrum, spleen, mesenteric lymph node, thymus and liver at 52 days post-inoculation (Bolin, et al., 2001). In the same experiment at 125 days post-inoculation, viral nucleic acid was detected in the spleen and distal ileum. Virus was also isolated from these tissues indicating that persistent infection was established.

Virus titres in tissues have rarely been reported. One study found virus titres of between $10^{4.5}$ to $10^{5.7}$ $TCID_{50}$/g in pooled inguinal and prescapular lymph nodes at 21 days post-inoculation (Ladekjaer-Mikkelsen, et al., 2002b) but the virus titre was less than $10^{1.5}$ $TCID_{50}$/g at 36 days post-inoculation. In clinically affected piglets high PCV2 titres ($10^{4.3}$ $TCID_{50}$/g to $10^{6.2}$ $TCID_{50}$/g) were detected in inguinal lymph nodes whereas in piglets with subclinical infection titres were less than $10^{2.0}$ $TCID_{50}$/g (Meerts, et al., 2003).

Very few countries have conducted surveys to determine the prevalence of PMWS. Serological surveys for PCV2 indicate that the virus is widespread globally, however, this is not an indication of disease prevalence (Segales & Domingo, 2002). In England and Wales recent surveys indicate that between 18% to 20% of all larger holdings (greater than 100 sows and/or greater than 200 growers) have been affected with either PMWS or PDNS (Gresham & Thomson, 2001). An earlier survey in England and Wales estimated that approximately 9.6% of larger holdings were affected with either PMWS or PDNS (Gresham, et al., 2000). In the United States of America a survey conducted in 2000 indicated that 5.7% of herds had experienced PMWS in nursery-age pigs during the previous 12 months (USDA, 2002). This varied with herd size, with 20.9% of large herds surveyed (10,000 or more pigs). In growers or finishers, PMWS or PCV was reported in 3.6% of sites surveyed, with 12.4% of large herds affected (USDA, 2002). In the Netherlands in 2000 about 15% of pig herds were affected with PMWS, whereas in 2002 this has increased to more than half the pig herds (de Jong, et al., 2003).

Within herds, morbidity of PMWS is generally reported to be low (Harding & Clark, 1997; Harding, 1997) although investigations into a herd affected with PMWS in central Spain reported that the prevalence of disease was 30% in 8 to 10 week old pigs (Rodriguez-Arrioja, et al., 2002a).

Clinical signs

The most frequent signs of PMWS are wasting or failure to thrive, dyspnoea, enlarged lymph nodes and, less frequently, diarrhoea, pallor and jaundice (Harding & Clark, 1997). The clinical

signs appear to be restricted to the post-weaning age group. Original reports of PMWS were in nursery stage pigs, 3 to 6 weeks post-weaning (Harding & Clark, 1997), but PMWS has also been reported in older pigs in the grow-finish period (10 to 20 week old pigs) (Sorden, 2000). In an acute outbreak, the monthly mortality associated with PMWS may be 10%, whereas morbidity and mortality may be considerably less in endemically affected herds (Harding & Clark, 1997). These authors suggested that the expression and severity of disease depend on stress and commingled ages. Mortality rates have been reported to be up to 40% higher in weanling pigs in herds with PMWS (Krakowka, et al., 2000). In the United Kingdom data from 62 herds were examined to determine post-weaning mortality before, during, at peak, and in some cases after the outbreak of PMWS and/or PDNS (Veterinary Laboratories Agency, 2002). Average mortality figures were 3.7% prior to PMWS, 12.6% during PMWS, peaking at 19.9%, then declining to 6% after the outbreak. The average duration of PMWS outbreaks was 274 days.

Porcine circovirus 2 infection has been linked to other syndromes including respiratory disease in pigs of 16 to 20 weeks of age, which is characterised by reduced growth rates, coughing and pneumonia (Segales & Domingo, 2002).

Pathogenesis

Lymphoid tissues appear to be the primary target tissues for PCV2. High levels of PCV2 antigen/nucleic acid are found in lymphoid tissues and lungs of diseased pigs (Clark, 1997). Virus or viral antigen has also been found in the brain, spleen, liver, kidney, tonsil, pancreas, bone marrow, distal ileum and adrenal gland (Bolin, et al., 2001). Target cells for PCV replication include monocyte/macrophage lineage cells and to a lesser extent epithelial cells such as renal tubular and bronchial cells. Nucleic acid and PCV antigen are detected primarily in the cytoplasm and rarely in the nuclei of macrophages (Rosell, et al., 1999). There would appear to be a strong correlation between the amount of PCV2 nucleic acid or antigen and the severity of the lesions in lymphoid tissues (Rosell, et al., 1999; Quintana, et al., 2001). However, PCV2 nucleic acid or antigen can also be found in clinically healthy pigs (Quintana, et al., 2001). Viral load in tissues and serum is consistently higher in PMWS affected pigs than those of pigs either not clinically affected or from PMWS free herds (Sibila, et al., 2003; Wellenberg, et al., 2003). It appears that clinical expression of disease depends on the generation and accumulation of “critical mass” of infectious virus in target tissues (Krakowka, et al., 2003).

It has been suggested that PCV2 enters through the tonsillar macrophages, with viraemia following within a few days. Replication of PCV2 and porcine parvovirus occurs to some extent in the circulating peripheral monocytes, contributing to cell-associated viraemia and to viral distribution throughout the lymphoid tissue (Kim, et al., 2003).

Immunostimulation may play an important role in the development of PMWS in some circumstances. Several studies have demonstrated that clinical PMWS can be consistently reproduced in germ-free or colostrum-deprived piglets by co-inoculation with PCV2 and porcine parvovirus or PRRS virus (Ellis, et al., 1999; Allan, et al., 1999; Kennedy, et al., 2000). These disease agents are believed to stimulate the immune system and promote PCV2 replication. More recently, the reproduction of severe clinical disease in germ-free pigs inoculated with PCV2 and then systemically immunised with keyhole limpet haemocyanin, emulsified in Freund’s incomplete adjuvant (KLH/ICFA) was reported (Krakowka, et al., 2001). These researchers concluded that immune activation is a key component of the pathogenesis of PCV2 associated PMWS in pigs. In contrast, one study found that

immunostimulation did not play a critical role in the development of PMWS in specific pathogen free (SPF) piglets (Ladekjaer-Mikkelsen, et al., 2002b).

Pathology

At necropsy, enlargement of lymph nodes is often the most notable feature in PMWS affected pigs. Other lesions include non-collapsing lungs sometimes with mottling and increased firmness, the liver is often atrophic, and the spleen is often enlarged. The kidneys may also be enlarged (Clark, 1997). However, these lesions are not always present.

Lymphocyte depletion is the most characteristic histopathologic finding in lymphoid tissues of PMWS affected pigs (Rosell, et al., 1999).

Immunology

The development of antibodies to PCV2 infection shows a pattern typical of viral infections that affect pigs. In one study colostral antibodies decreased during the nursery period, with the lowest levels at 7 weeks of age, active seroconversion of pigs occurred during the grower period and a relationship between the mortality associated with PMWS and the low serologic titres at 7 weeks of age was noted (Rodriguez-Arrioja, et al., 2002a). This pattern of seroconversion appeared to occur in herds with and without PMWS. Antibodies detected by immunoperoxidase monolayer assay in healthy pigs were unable to neutralise virus.

Transmission via meat

The Panel is unaware of any studies that have examined skeletal muscle for the presence of PCV2 viral antigen or virus. Viral antigen from chicken anaemia virus, has been detected in muscle at 11 and 12 days post-inoculation and in bone marrow up to 13 days (Smyth, et al., 1993). Porcine circovirus type 2 viral antigen has also been detected in the bone marrow of seven of 14 pigs, 20 to 28 days after experimental infection, and in one of five pigs, 35 days post-infection. Viral antigen was not detected in bone marrow at 52 days post-infection (Bolin, et al., 2001). In contrast, PCV1 was not detected in bone marrow 9 days after inoculation (Allan, et al., 1995). Porcine circovirus type 2 viral antigen has been found in the heart (Kennedy, et al., 2000; Bolin, et al., 2001).

It is unknown if pigs can be infected orally with PCV2. The detection of the virus in oronasal secretions and faeces is compatible with an oral route of transmission. Generally, under experimental conditions, pigs are infected intranasally. In one study, pigs were infected oronasally with 4.3 x 10^6/0.5ml PCV2 (Krakowka, et al., 2001).

Recently it has been postulated that the PMWS outbreak in New Zealand may be linked to the feeding of imported uncooked or inadequately cooked pig meat to pigs.[55,56] To date this has not been substantiated.

[55] New Zealand Ministry of Ariculture and Forestry media release, 4 February 2004.

[56] Professor Roger Morris, Massey University, New Zealand – submission dated 10 February 2004 to the Senate Rural and Regional Affairs and Transport Committee.

Release assessment

R1 — the likelihood that a source herd is infected

There are few data on the between herd prevalence of PMWS in affected countries. In England and Wales where the disease is significant and spreading, between 18% to 20% of all larger holdings are affected with either PMWS or PDNS. In the United States of America 5.6% of holdings reported PMWS in nursery-age pigs, with over 20% of large holdings affected. In the Netherlands over half of the pig herds reportedly are affected with PMWS. If it is assumed that a virulent strain of PCV2 is responsible for PMWS, it is likely that between herd prevalence may be higher than that reported, as expression of the disease may depend on co-factors. Given this, the likelihood that slaughter-age pigs have been selected from an infected herd in a country where PMWS is endemic was considered to be 'moderate'.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

In herds affected with PMWS morbidity has been reported as low, however, seroprevalence of PCV2 is high. If it is assumed that in those herds affected with PMWS the strain of PCV2 circulating within that herd is virulent, then within herd prevalence of PCV2 infection in PMWS affected herds is an important consideration. It has been demonstrated that most pigs become infected with PCV2 following weaning. There is increasing evidence that persistent infections may be a feature of PCV2, with viral nucleic acid detected in a few tissues for up to 125 days post-inoculation. In one study, which followed a batch of pigs from a PMWS affected herd through to slaughter, workers were unable to demonstrate microscopic lymphoid lesions or viral nucleic acid at slaughter, suggesting that these pigs had cleared the virus. However, another study detected PCV2 nucleic acid in sera from 52.6% of 386 healthy slaughter-age pigs.

On the basis of this information, it was considered that the likelihood of selecting an infected pig in an infected herd was 'moderate'.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

The clinical signs of PMWS and gross pathology may be quite marked and it was considered unlikely that clinically affected pigs would pass the inspection procedures in the unlikely event they were sent for slaughter. However, in affected herds, subclinical infection is a feature of PCV2. In particular, pigs infected during fattening may show no signs of disease or possibly respiratory disease. In the case of pigs showing signs of respiratory disease, pathological changes would tend to be limited to the lungs, and at most may result in the condemnation of the lungs but not the associated carcass.

Considering this, the sensitivity of ante-mortem, slaughter and processing procedures in detecting and removing infected pigs was considered 'extremely low'.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with PMWS and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a 'one minus the background rejection rate'. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered 'extremely low'. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

The Panel is unaware of any reports where muscle has been examined for the presence of PCV2. It is known that the virus, as is the case with many viruses, has an affinity for lymphoid tissues and has been detected in bone marrow for up to 35 days in one of five pigs and at low levels in lymph nodes at 36 days post-inoculation. Viral nucleic acid has also been detected in serum for up to 16 weeks. It has been postulated that PMWS may have entered New Zealand via imported uncooked pig meat which was then fed to pigs.

In view of the fact that a carcass will contain some lymphoid tissue, bone and blood, as well as muscle, it was considered that the likelihood that the pathogenic agent would be present in meat harvested from an infected pig was 'moderate'.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

Porcine circovirus is stable at pH 3. For the purposes of this IRA, meat is not assumed to reach a pH lower than 6.2. Thus, it was considered that there was a 'high' likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

There are limited data on the stability of PCV2. Porcine circovirus 2 is readily isolated from tissues that have been stored at -70°C (Ellis, et al., 1998). It is known that PCV1 is stable at higher temperatures. In view of this, it was considered that there was a 'high' likelihood that meat infected at the completion of carcass maturation would remain infected during transport and cold storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a 'low' likelihood that imported pig meat derived from an individual carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

Porcine circovirus type 2 is easily spread between pigs and transmission appears to occur via oronasal secretions, faeces and urine. Hence it is likely that pigs are infected with PCV2 orally, however, no information is available on the oral infectious dose. Experimentally a high dose of virus has generally been administered intranasally, but this does not always result in expression of the disease. PMWS has been transmitted to pigs by direct contact with PMWS affected pigs. As stated above it has been postulated that PMWS may have entered New Zealand via imported uncooked imported pig meat which was then fed to pigs. If this is shown to be the case, this would demonstrate that oral transmission can occur resulting in PMWS.

The levels of virus in bone marrow have not been determined. It is known that lymph nodes are strongly positive for nucleic acid or antigen (Rosell, et al., 1999; Allan, et al., 1999). Virus titres of approximately 10^5 to 10^6 $TCID_{50}$/g of lymph node have been reported from clinically healthy pigs experimentally infected with PCV2 (Meehan, et al., 2001). It is not known when the samples were collected post-infection. High levels of PCV2 DNA have been found in serum, reaching a peak at 21 days post-infection (10^9 DNA copies/ml serum), and decreasing to 10^6 DNA copies/ml serum at day 35 (Ladekjaer-Mikkelsen, et al., 2001). Levels in lymph node were high at 21 days post-inoculation but were less than $10^{1.5}$ $TCID_{50}$/g at 36 days. In experimentally infected pigs, necropsied 70 days post-inoculation PCV2 DNA load was less than 10^3 DNA copies/µl of organ suspension (Stockhofe-Zurwieden, et al., 2003). In persistently subclinically infected pigs levels of virus in tissues are likely to be low.

On balance, the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was considered to be 'moderate'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

This step describes the sensitivity of PCV2 to ultraviolet light, to ambient temperatures ranging from 10°C to 35°C and to the putrefying effects of saprophytic organisms. Circoviruses are considered fairly resistant viruses, although little data are available. It is known that PCV is stable at a low pH and for at least 15 minutes at 56°C and for 15 minutes at 70°C.

In the light of this information, it was considered that the likelihood that the pathogenic agent would survive within meat scraps discarded in refuse for the period of time required for feral pigs to locate and subsequently scavenge the material was 'high'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = High
- Rural regions = High
- Large towns = Very low

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be ‘high’.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a ‘low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was ‘moderate’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a ‘high’ likelihood that the pathogenic agent would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be ‘high’.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a ‘low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was ‘moderate’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in

small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a 'high' likelihood that the pathogenic agent would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'high'.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the three exposure groups - that is, feral pigs, pigs in backyard enterprises and pigs in small commercial piggeries. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;

2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs, however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

Antibodies to PCV2 have been detected in feral pig populations. In Belgium and Spain 37% and 34.6% of feral pigs tested positive (Sanchez, et al., 2001; Segales, et al., 2002). Infection with PCV2 appears to be less widespread in feral pigs than in domestic pigs. PMWS in wild boars has recently been described (Segales, et al., 2003).

In domestic pig herds affected with PMWS, the disease appears to occur on an individual basis within a pen, despite widespread infection with PCV2. In many animals PCV2 infection is subclinical. The virus has been detected in faeces, urine, bronchial, tonsillar and nasal swabs, suggesting an oronasal route of infection. Persistent infections may also occur increasing the likelihood of spread of the virus. The virus appears to be hardy and is likely to persist in the environment.. Transmission of PMWS has occurred via direct contact. Based on this information, it is feasible that infection would spread in a feral pig population, however, it may be that the additional factors required to trigger PMWS are not present. This situation could also apply if an unknown agent was involved in PMWS

It is conceivable that nocturnally foraging feral pigs may be attracted to an enclosure housing domestic pigs, and that while mixing *per se* is unlikely, contact sufficient for the transmission of infection may occur, although this in itself may not result in the development of PMWS.

If transmission to a backyard or small commercial piggery occurred, it is likely that the virus could be spread regionally by live pigs, or possibly by fomites or semen (this has not been demonstrated at present) to other such piggeries. If PMWS developed as a result of spread of the virus, diagnosis would likely occur when several piggeries were affected. If large commercial piggeries were also situated within the region, spread to these might occur, and that this may subsequently lead to a more general outbreak.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: very low

Scenario 2: moderate

Scenario 3: moderate

Scenario 4: low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

As discussed above, it is feasible that backyard pigs kept in rural areas may come in close contact with nocturnally foraging feral pigs, and that transmission of virus from one group to the other may result, although PMWS may not necessarily occur. Backyard pigs are often raised for consumption by the household but it is feasible that some mixing of pigs between backyard herds may occur. For example, in the case of speciality breeds or unusual breeds, live pigs or semen may be transferred from one herd to another for breeding purposes. Alternatively, pigs raised for personal consumption may be transferred between backyard holdings for growing out or fattening.

The clinical signs of this disease are not sufficiently distinctive to ensure its early diagnosis. Young weaned pigs may not be on the premises or only a few pigs out of a group may be affected. Hence, movement of pigs may result in spread of the disease to other backyard premises and then further spread to commercial piggeries.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: low

Scenario 4: high

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

An outbreak of PMWS in small commercial piggeries is more likely to result in veterinary attention, but it may be some time before the disease is accurately diagnosed, mortality rates may not be high and endemic diseases may be ruled out first. Veterinarians in Australia are well aware of PMWS and to date, there has been active submission of samples from pigs showing clinical symptoms similar to those of PMWS. Due to the larger herd size and introductions and movements of pigs within the small commercial piggery, the outbreak may persist for a period of time. This factor, together with the relatively high level of movements of pigs, personnel and fomites between piggeries, may result in further spread of disease.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: very low

Scenario 2: very low

Scenario 3: low

Scenario 4: high

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each exposure group follow a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this scenario, the disease is most likely to have established amongst the directly exposed animals, and to have run its course without identification. This is because the clinical symptoms of this disease are not distinct and may be confused with endemic diseases.

The direct impact of PMWS

Animal life or health

In PMWS affected herds, weaned pigs can show signs of unthriftiness, wasting, dyspnoea and sometimes diarrhoea. Mortality can greater than 10% in weaned pigs. In this scenario, the disease has only affected the directly exposed herd, hence the direct impact on animal health was considered unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

Environment

Because PMWS is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of PMWS

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this outbreak scenario where there is containment of the disease within the directly exposed group and in the case of pigs the clinical signs of disease can be mild or non-specific, it was considered unlikely that the primary case would be diagnosed. Given this, it was considered unlikely that the indirect impact of new or modified control programs would be discernible at any level, and the rating assigned to this criterion was therefore 'A'.

Domestic trade or industry effects

As discussed above, it is unlikely that disease would be diagnosed in a single herd. On this basis, the indirect impact of PMWS on domestic trade and industry was considered unlikely to be discernible at any level, and a rating of 'A' was thus assigned to this criterion.

International trade effects

As the disease is unlikely to be diagnosed in a single herd, it was considered that the indirect effect of PMWS on international trade was unlikely to be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on the environment

In this scenario, PMWS is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, the disease is most likely to have established within a population of feral pigs, and to have run its course without identification. As explained above, this is because the clinical symptoms of this disease are not distinct.

The direct impact of PMWS

Animal life or health

As with the first scenario, the impact on animal health is unlikely to be discernible at any level except the local level. Indeed, it is likely that the disease would remain undiagnosed if contained within a more general population of feral pigs due to the nature of the disease and limited opportunities for close observation of feral pigs. This resulted in a rating of 'B' for this criterion.

Environment

Because PMWS is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of PMWS

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

The consequences on this criterion would be similar to that described above for the first scenario, resulting in a rating of 'A'.

Domestic trade or industry effects

In this outbreak scenario, PMWS spreads to a more general population of feral pigs but not to domestic pigs. As the disease may remain undiagnosed within feral pigs for a significant period of time the indirect effects on domestic trade and industry were considered unlikely to be discernible at any level, and thus the rating assigned to this criterion was 'A'.

International trade effects

As described above, the indirect effects of PMWS on international trade were considered unlikely to be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on the environment

In this scenario, PMWS is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, the disease has established within a local population of small commercial piggeries or backyard enterprises. As Australian veterinarians are actively looking for the disease, it was considered likely that the disease would be diagnosed due to the increased mortality in weaned pigs. Control measures such as quarantine and movement restrictions may be applied to limit spread.

The direct impact of PMWS

Animal life or health

This scenario is characterised by spread of PMWS to a local population of domestic pigs in backyard enterprises or small commercial piggeries, but containment within this population. PMWS can result in increased mortalities in weaned pig. Porcine circovirus type 2 has also been linked to respiratory disease in grower and fattening pigs and reproductive disorders. Given this, it was considered that the direct effect on animal health would be unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

Environment

Because PMWS is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of PMWS

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this scenario, PMWS spreads to a local population of domestic pigs in backyard enterprises or small commercial piggeries. It was considered that PMWS would be diagnosed under this scenario.

PMWS is not included in Australia's Emergency Animal Disease Response Agreement and as such the cost of control measures and eradication would likely have to be met by industry. Should the disease occur in Australia, it may be considered as an emerging disease and the outbreak managed by adopting cost-effective strategies to control and, if feasible, eradicate the disease. In this limited outbreak this may be possible with quarantine and movement controls, such that animals only move to slaughter.

When these issues were taken into account, the indirect impact of new or modified control programs was considered unlikely to be discernible at the national or State level, and of minor

importance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

Domestic trade or industry effects

If movement controls are implemented, pigs from infected herds may be restricted to moving directly to slaughter. Thus, the movement of live pigs for breeding purposes or to saleyards may be prohibited. As this is likely to affect only those producers within the local area, the indirect impact of PMWS on domestic trade and industry was considered unlikely to be discernible at the national or State level and of minor significance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

International trade effects

The diagnosis of PMWS in domestic pigs may result in cessation of live pigs and semen to a few markets. PMWS is already present in the major export markets. Australia exports only small numbers of breeding pigs and small quantities of semen. PMWS is not an OIE Listed disease. The detection of PMWS in Australia is unlikely to affect the export of meat. Australia's major export markets are Japan and Singapore. The disease is present in Japan, Singapore does not have a pig industry, and PMWS has no human health implications. Thus the indirect effect of PMWS on international trade was considered unlikely to be discernible, except locally. Overall, this resulted in a rating of 'B' for this criterion.

Indirect impact on the environment

PMWS in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, PMWS would have established in a broader population of commercial piggeries (including medium-large piggeries) and be identified. If the disease was not widespread in the Australian pig population, a control program may be implemented, alternatively, if widespread, control would likely be left to individual producers.

The direct impact of PMWS

Animal life or health

In this scenario, PMWS has spread to a more general population of domestic pigs including medium to large commercial piggeries. Mortality rates can be high as in the case of the United Kingdom, where the disease is associated with significant economic costs (Veterinary Laboratories Agency, 2003). In other countries such as Canada and the United States of America the impact of the disease would appear to be less significant (Sorden & Halbur, 2002). In Germany, mortality rates increased by approximately 6%, daily weight gain decreased by 13

g/d and feed conversion rate increased 0.06 kg/kg in a herd affected with PMWS (Hardge, et al., 2003).

On balance, the direct impact on animal health was considered unlikely to be discernible at the national level, but would be of minor importance at the State level. This gave the disease a rating of 'D' for this criterion.

Environment

Because PMWS is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of PMWS

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this outbreak scenario, PMWS has spread to a more general population of domestic pigs, including medium and large commercial piggeries. Surveillance may be undertaken to determine the extent of the spread of the disease and the feasibility of any control program. As PCV2 is already present in Australia, testing of pig herds for this agent would not assist in assessing spread of the disease. Control and surveillance would need to be based on presence of clinical disease in a herd. It may not be feasible to implement an overall control program and management of the disease may be left to individual producers.

On this basis, it was considered that the indirect impact of new or modified control programs would be unlikely to be discernible at the national or State level, but of minor importance at the district or regional level, thus this criterion was rated as 'C'.

Domestic trade or industry effects

If quarantine and movement controls were implemented, this would mainly affect the sale of breeding pigs. The supply of pork to the domestic market should not be disrupted as most pigs will still be able to be sent to slaughter. Associated industries such as abattoirs, manufacturers of small goods and transport are unlikely to be affected. As production may be significantly reduced in affected piggeries, revenue of individual producers will also be reduced. There would be additional costs of treatment. In one herd affected with PMWS the gross margin decreased from €11.56 per pig (pre PMWS) to €7.62 per pig during PMWS (Hardge, et al., 2003). Veterinary costs rose from €3.18 per pig to €3.53 per pig. In the United Kingdom losses to the industry due to PMWS have been estimated to be in excess of €50 million per year. Overall losses in the European Member States are estimated to be currently running in excess of €562 million per year (Allan, 2003). It has been estimated that an epidemic of PMWS in Australia, similar to that overseas, could add 15% to the cost of pig meat production in affected herds.[57]

When these issues were taken into account, the indirect impact of PMWS on domestic trade and industry was considered unlikely to be discernible at the national level, but of minor importance at the State level. This resulted in a rating of 'D' for this criterion.

[57] Australian Pork Limited – submission dated 4 February 2004 to the Senate Rural and Regional Affairs and Transport Committee.

International trade effects

As discussed in scenario 3, the diagnosis of PMWS in domestic pigs may result in cessation of live pigs and semen to some markets but is unlikely to affect the export of meat. Thus, the indirect effect of PMWS on international trade was considered unlikely to be discernible, except at the local level. Overall, this resulted in a rating of 'B' for this criterion.

Indirect impact on the environment

PMWS in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

Where the pig industry is significant to the local area, some aspects of that community may be affected. Some piggeries will suffer a loss of revenue due to the decreased production. This would likely have a flow on affect for that community. On balance, the indirect impact of PMWS on rural communities was considered unlikely to be discernible at the national or State level, but of minor importance to affected districts and regions. This resulted in a rating of 'C' for this criterion.

The overall impact of PMWS

When the direct and indirect impacts of PMWS were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences negligible

Scenario 3: Consequences very low

Scenario 4: Consequences low

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 61, Table 62 and Table 63. It can be seen that the overall likely consequences associated with the exposure of feral pigs were considered 'very low', and those for backyard pigs and pigs in small commercial piggeries to infected pig meat scraps were considered to be 'low'.

Table 61 PMWS: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Very low	Negligible	Negligible
Scenario 2	Moderate	Negligible	Negligible
Scenario 3	Moderate	Very low	Very low
Scenario 4	Low	Low	Very low
		Overall likely consequences	**Very low**

Table 62 PMWS: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Low	Very low	Negligible
Scenario 4	High	Low	Low
		Overall likely consequences	**Low**

Table 63 PMWS: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Very low	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Low	Very low	Negligible
Scenario 4	High	Low	Low
		Overall likely consequences	**Low**

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with PMWS.

Table 64 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly higher than Australia's ALOP (very low), risk management would be required for PMWS.

Table 64 PMWS: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Low	High	Very low	Very low
Backyard pigs	Low	High	Low	Low
Small commercial piggeries	Low	High	Low	Low
			Overall annual risk	**Low**

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***Salmonella typhimurium* DT104**

Technical information

Background

Clinical and subclinical *Salmonella* infections in animals have long been a source of concern to the veterinary, food and public health industries worldwide. Of the many serotypes identified to date, some can cause gastro-enteritis in animals and food poisoning in people. Several serotypes have worldwide distribution while others have not been reported in Australian livestock. One such pathogenic serotype not reported in Australian livestock is *Salmonella enterica* subspecies enterica serovar Typhimurium variant Definitive Type 104 R-*ACSSuT*[58] (commonly referred to as DT104) which first emerged in the United Kingdom in 1984 and remained an infrequent isolate until 1990. *Salmonella typhimurium* DT104 is primarily a pathogen of cattle (Poppe, et al., 1998).

Salmonella typhimurium DT104 has since been identified in all continental European countries, where the incidence of human cases has increased in a similar manner to that in the United Kingdom (World Health Organization, 1997). The bacteria has also been identified in the United States of America, Canada and Middle-Eastern and Southeast Asian countries. In 1995, it was the most commonly isolated strain found in human cases of salmonellosis in England and Wales (World Health Organization, 1997). In the United States of America, the National Antimicrobial Resistance Monitoring System (NARMS) reported that in 1996 and 2001, 34% and 29% of human cases of *S. typhimurium* were ACSSuT resistant phenotypes respectively (NARMS, 2001).

In Australia, *Salmonella typhimurium* DT104 has been isolated only from travellers returning from overseas or from people who became ill after eating infected imported food (Davos, 2001; Fisher, et al., 2001).

Agent taxonomy

More than 2200 serovars of salmonellae, based on its somatic (O) antigen groups and flagellar (H) antigen groups, have been identified (Coetzer, et al., 1994; World Health Organization, 1997).

Agent properties

Members of the genus *Salmonella* are morphologically and biochemically homogenous groups of gram-negative, motile, facultatively anaerobic bacilli. Salmonellae are considered to be hardy and ubiquitous pathogens. They multiply at 7°C to 45°C, survive freezing and desiccation and can persist for years in suitable organic substrates (Schwartz, 1999). *Salmonella typhimurium* can remain viable for up to 7 months in soil, water, faeces or on pasture (Ramírez, et al., 2002). Salmonellae are rapidly inactivated by heat and sunlight, do not sporulate and are destroyed by common phenolic, chlorine and iodine based disinfectants (Ramírez, et al., 2002).

A *S. typhimurium* DT104 clone now widespread has multiple antimicrobial resistance patterns to ampicillin (A), chloramphenicol (C), streptomycin (S), sulphonamides (Su) and tetracyclines (T) due to strain resistance genes being chromosomally encoded. This type of encoding

[58] R-ACSSuT = resistant to ampicillin (A), chloramphenicol (C), streptomycin (S), sulphonamides (Su) and tetracyclines (T)

suggests that any removal of the selective pressure from antimicrobials is not likely to reverse resistance as can happen with extra-chromosomal or plasmid mediated resistance. This clone has spread considerably throughout the northern hemisphere in the past decade and, in some cases, has developed resistance to other antibiotics, including kanamycin, spectinomycin, trimethoprim and/or some fluoroquinalone antibiotics (Baggesen, et al., 2000). However, antibiotics, selected on the basis of culture and sensitivity tests and best management practices, can be used to treat animals infected with *S. typhimurium* DT104 (Beaudin, et al., 2002).

Host range

Salmonella typhimurium DT104 is not host specific. While most commonly associated with cattle, the pathogen has been isolated from sheep, pigs, goats, poultry, dogs, cats, emus, rodents, wildlife including wild birds, such as starlings, and various food products including processed food (Besser, et al., 1997; Poppe, et al., 1998). Poultry chicks can be infected with *S. typhimurium* DT104 without showing clinical signs but the bacteria can persist in the environment and become the dominant strain in the poultry house (Fedorka-Cray, et al., 2001). Salmonellosis due to *S. typhimurium* DT104 is a serious zoonosis (Wall, et al., 1994).

Epidemiology

Transmission of *S. typhimurium* DT104 via faeces from an infected pig is the principal means of spread between or within pig herds. Bacteria are shed in the faeces of clinically affected pigs or carrier pigs, particularly when they are stressed by movement, poor nutrition, overcrowding or concurrent disease (Schwartz, 1999). Infection is via the faeco-oral route. Pig producers frequently cite the faeces of rodents or birds as a source of infection, although it has been reported that these species are generally infected by exposure to a contaminated piggery (Newell & Williams, 1971). Infection can also occur as a result of indirect transmission from contaminated feed and water supplies, pasture contaminated by slurry or sewerage, and wildlife vectors (Bagger, et al., 2001).

Pigs exposed to *S. typhimurium* can shed low numbers of bacteria in the faeces for up to 28 weeks after infection (Wood, et al., 1989). Twenty-four pigs experimentally dosed with 1×10^{11} CFU (colony forming units) of *S. typhimurium* DT104 by intragastric inoculation developed diarrhoea with 4×10^2 to 6×10^6 CFU/g in faeces for the first 7 days after infection. Faecal shedding then decreased to less than 4×10^2 CFU/g over the next 7 days. When these pigs were sent to slaughter 21 days after infection, 92% of pigs subjected to 8 hours of transportation prior to slaughter shed the bacteria in their faeces while only 58% of pigs not subjected to transportation prior to slaughter shed bacteria (Marg, et al., 2001). This suggests the stress of transport and lairage may cause healthy carrier pigs that were not shedding at the farm of origin to resume shedding bacteria at slaughter. The lairage is a significant source, and transport a significant contributor, of contamination of pigs from uninfected herds (Swanenburg, et al., 2001). This finding is supported by a study which determined that the percentage of *Salmonella*-excreting Danish pigs before and after transport was high (Berends 1993, as quoted in (Berends, et al., 1998b)). The number of excreting pigs before (X) and after (Y) transport being described by the function: $Y = (1.72 \pm 0.18)X$.

Animals can also intermittently shed the bacteria in their saliva, contaminating the environment (Sharp & Rawson, 1992; Fone & Barker, 1994). *Salmonella typhimurium* DT104 can also be recovered from the ileum, caecum and the ileo-caecal lymph nodes in concentrations of at least 2.7×10^2 CFU/g of tissue (Springer, et al., 1999).

Human outbreaks of *S. typhimurium* DT104 have been linked to a broad range of infected foodstuffs, including pork and pork products (World Health Organization, 1997). Human outbreaks, particularly involving young children living on farms, have also been linked to contact with infected animals (Fone & Barker, 1994). Evidence suggests direct transmission from animal to people may occur more frequently with *S. typhimurium* DT104 than with other *Salmonella* strains (Wall, et al., 1994; Fone & Barker, 1994). In the United Kingdom during 1995, this strain is associated with hospitalisation rates which are twice that of other zoonotic food-borne *Salmonella* infections and with ten times higher case fatality rates (World Health Organization, 1997). People most at risk are those at the extremes of age, the young (less than 4 years) and the elderly, and immunosuppressed individuals.

Data representing the prevalence of *S. typhimurium* DT104 are very limited. The data that were available were examined on a national, between herd and within herd basis. In Great Britain, a national study to determine the prevalence of *Salmonella* infections in pigs was carried out between 1999 and 2000 (Davies, et al., 2000). Caecal contents and carcass surface swabs were collected from 2059 pigs. Salmonellae were identified in 23% of the caecal content samples, of which 11.1% were *S. typhimurium* and of these 21.9% were DT104. Overall, 0.56% of the pigs tested positive for *S. typhimurium* DT104.

In 1995, the United States Department of Agriculture conducted a national study of the pig industry. The study showed that 38.2% of swine operations had evidence of salmonellae in faecal samples (National Animal Health Monitoring System (NAHMS), 1995). Considering that (a) *S. typhimurium* was the second most commonly reported *Salmonella* serotype, accounting for 24% of over 40,000 *Salmonella* isolates reported that year; (b) the ACSSuT resistance pattern was present in 28% of a national sample of nearly 1000 *S. typhimurium* isolates tested and (c) that several of the ACSSuT isolates were sent to the United Kingdom for phage typing and 83% of these isolates were DT104 (Hosek, et al., 1997), the prevalence of herds with *S. typhimurium* DT104 was estimated to be around 2.13% (that is, 38.2% x 0.24 x 0.28 x 0.83).

Between 1998 and 2000, in Denmark where mandatory salmonella control programs are in place, between 2% and 4% of slaughtered pigs tested positive for salmonellae (Danish Zoonosis Centre, 2001). Denmark's stamping out policy of *S. typhimurium* DT104 infected pig herds, introduced in 1996, was terminated during 2000 when 49 infected pig herds were identified and a new reduction strategy was implemented (Danish Zoonosis Centre, 2001). In view of the fact that there were over 21,000 pig herds in Denmark in 2000 (Danish Zoonosis Centre, 2001), the between herd prevalence of *S. typhimurium* DT104 can be estimated to be 0.23%, noting that a stamping out program had been in place. Another survey in Denmark conducted in 1998 with 1962 slaughter pig herds estimated the herd apparent prevalence of *S. typhimurium* DT104 to be 0.05% (95% confidence interval 0.011% to 0.25%) (Christensen, et al., 2002).

The level of infection can vary considerably from farm to farm (Mogelmose, et al., 1999). A survey to estimate the within herd prevalence of salmonellae in caecal contents of infected Irish pig herds showed that prevalence varied from 10% to 19% (Quirke, et al., 2001). In another report, expert opinion on infection status in pigs at the farm of origin indicated that between 21% to 33% of pigs coming from a chronically infected farm would be infected with salmonellae, but only one-third of the infected pigs, that is, 7% to 11%, would be shedders (Stark, et al., 2002). The prevalence of *S. typhimurium* DT104 in four infected pig herds in Denmark varied from less than 10% up to 100%, although the 100% test positive animals were only 14 samples from weaned piglets (Mogelmose, et al., 1999).

Clinical signs

The morbidity rate of *S. typhimurium* DT104 infection can be high in cattle and poultry and mortality rate high in cattle. Clinical signs in cattle include watery diarrhoea, loss of appetite and loss of condition (Evans & Davies, 1996). A cat infected with *S. typhimurium* DT104 presented with diarrhoea, fever and vomiting (Wall, et al., 1995). Sub-clinical infection is common, particularly in pigs, and healthy carrier animals may harbour subclinical infections and shed the pathogen in the faeces for several months after infection.

Infection in pigs is usually detected as a result of intensive abattoir surveillance programs for salmonellae. However, the pathogen has caused nervous symptoms and death in 1-week-old piglets (van der Wolf, et al., 2001). Experimentally a dose of 1 x 10^{10} CFU *S. typhimurium* DT104 given via the intragastric route to pigs did not produce any clinical signs, while 5 x 10^{10} CFU orally resulted in two of four pigs developing mild clinical signs and a dose of 1 x 10^{11} CFU by intragastric route to 24 pigs resulted in all pigs having diarrhoea within 24 to 36 hours post-infection (Springer, et al., 1999; Marg, et al., 2001).

Salmonella choleraesuis is the most common cause of salmonellosis in swine. Disease is associated with septicaemia, enterocolitis, or bacteraemic localisation as pneumonia and hepatitis or occasionally as meningitis encephalitis and abortion (Schwartz, 1999).

In humans, the pathogen causes diarrhoea, sometimes bloody stools, vomiting, abdominal pain, and fever. Some people require hospitalisation, the length of stay ranging from 1 to 35 days (median 5 days). Human infections due to *S. typhimurium* DT104 have resulted in higher rates of admissions to hospitals and mortality than many other *Salmonella* spp (Wall, et al., 1994).

Pathogenesis

The clinical and pathologic features of salmonellosis reflect a host-parasite interaction that can be influenced by serotype, virulence, natural and acquired resistance and route and size of infectious dose. Salmonellae are normally a small part of an extremely complex and competitive bacterial environment in the intestinal tract.

Salmonellae can invade and replicate in the epithelial cells of the gastrointestinal tract. The exact mechanism by which *Salmonella* spp. causes diarrhoea is not well known. The pathogenesis of diarrhoea typical of enteric salmonellosis and of later stages of septicaemic salmonellosis has traditionally been attributed to malabsorption and net fluid leakage from a necrotic and inflamed bowel. Further studies demonstrate that at least early in the disease, the diarrhoea is the result of decreased sodium resorption and increased chloride secretion due to cholera-like and shiga-like enterotoxins. The systemic signs and lesions of septicaemic salmonellosis in pigs are most commonly attributed to endotoxaemia from bacterial dissemination (Schwartz, 1999).

Pathology

Where pigs have died of salmonellosis, focal or diffuse necrotic colitis and typhlitis is usually seen, with the necrosis appearing as button ulcers. Often, mesenteric lymph nodes, particularly the ileocaecal nodes, are grossly enlarged, being two to five times normal size, and there is splenomegaly. Sometimes, the only evidence of subacute disease is a slight reddening and roughening of the ileal mucosa. The liver usually appears normal, being enlarged only in some cases of terminal congestion (Schwartz, 1999).

Immunology

Newborn animals are immunologically immature and do not respond serologically to the somatic (O) antigen until they are 2 to 3 weeks of age. They do, however, produce a serological response to the flagellar (H) antigens of the *Salmonella* bacteria (Office international des épizooties, 2000). Animals can acquire maternally derived antibodies via the colostrum. Following *Salmonella* infections, immunoglobulin concentrations remain elevated for around 2 to 3 months.

Immunisation has been used for many years to control *Salmonella* infections in farm animals, and if diagnostic serology is to be used, it may be necessary to differentiate the vaccine response from that of actual infection.

Transmission via meat

Salmonellae have been rarely isolated from meat of infected pigs. Of 24 pigs orally dosed with 1×10^{11} CFU, *S. typhimurium* DT104 was isolated from muscles of one pig at slaughter 21 days after infection (Marg, et al., 2001). The most common means of 'infection' of meat is via contamination with infected faeces or blood during the slaughter process i.e. contamination of carcasses from infected pigs and cross-contamination of uninfected carcasses. One study found that there was a strong correlation between the proportion of pigs with salmonellae in their faeces and the proportion of contaminated carcasses at the end of the line (Berends, et al., 1997). Seventy-three percent of carcasses from pigs that had salmonellae in their faeces tested positive at the end of the line. Furthermore, it was demonstrated that about 70% of all carcass contamination resulted from the pigs themselves being carriers and about 30% because other pigs in the line were carriers (cross-contamination).

The slaughter line is an important source of cross-contamination of carcases through manual or mechanical handling (Swanenburg, et al., 2001). In a survey of abattoirs, an estimated 5% to 15% of all carcass contamination occurred during polishing after singeing; 55% to 90% occurred during evisceration; and 5% to 35% during dressing, splitting and inspection (Berends, et al., 1997). According to data collected by Brekelmans and others (as reported in (Berends, et al., 1997)), the overall effect of contamination was that the number of contaminated carcasses at the end of the slaughter process exceeded the number of pigs with detectable levels of faecal salmonellae entering the slaughter process by 13%.

In a survey of 12 abattoirs in the European Union, salmonellae were not isolated from five of the abattoirs, but were isolated from 5.3% of the products sampled at the other seven abattoirs (Hald, et al., 1999). This report also noted that the probability of recovering a positive sample was more than three times as high at the end of the slaughter day than at the beginning of the day.

The application of HACCP[59] methods within the abattoir or processing plant appears to be only marginally effective in the control of cross-contamination in well-managed abattoirs (Berends, et al., 1998b). Nonetheless it has been shown that washing carcasses with water heated to 79°C to 81°C for 15 seconds prior to chilling reduced contamination to below detectable levels in 90% of cases (Jensen & Christensen, 2001). The temperature of the water must be maintained at or above 62°C, as salmonellae can survive below this temperature (Hald, et al., 1999).

[59] HACCP – Hazard Analysis and Critical Control Point. HACCP consists of a set of principles which are applied to the food processing industry as a component of a food safety program.

Release assessment

R1 — the likelihood that a source herd is infected

There is little information available on the between herd prevalence of *S. typhimurium* DT104 in pigs. In Denmark, in 2000, the between herd prevalence of *S. typhimurium* DT104 was calculated to be between 0.011% and 0.25%. However, a control program was in place prior to this survey. Available data indicated that the prevalence of *S. typhimurium* DT104 between pig herds in the United States of America in 1995 was 2.13%. In Great Britain, in a national survey, 0.56% of pigs were positive for *S. typhimurium* DT104. Nonetheless, the increasing number of human cases suggests that the prevalence of *S. typhimurium* DT104 worldwide has increased in recent years. On balance, it was considered that the likelihood of selecting slaughter-age pigs from an infected herd was ‘very low’.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

The level of infection can vary considerably within herds. Expert opinion on infection status of *Salmonella* in pigs at the farm of origin estimated that 21% to 33% of pigs coming from a chronically infected farm would be infected, but only one-third of the infected pigs, that is, 7% to 11%, would be shedders. Another survey on the prevalence of salmonellae in caecal contents of infected Irish pig herds showed that within herd prevalence varied from 10% to 19%. In Denmark, within herd prevalence of *S. typhimurium* DT104 varied from less than 10% up to 100%, in the case of 14 weaned piglets.

Stress of transport and overcrowding during transport and holding at saleyards and/or abattoirs may cause infected pigs that were not shedding at the farm of origin to resume shedding bacteria and spread infection to uninfected pigs whilst being held at the lairage. One modelling study showed the number of excreting pigs increased by 54% to 90% after transport (Berends, et al., 1996; Berends, et al., 1998a).

Based on this information, it was considered that the likelihood of selecting an infected animal either from an infected herd or infected during transport and/or at the lairage was ‘moderate’.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

Slaughter-age pigs infected with *S. typhimurium* DT104 are generally healthy carriers of the organism. Clinical disease is rare. If diarrhoea is noted during ante-mortem inspection, these pigs are likely to be either withheld from processing pending treatment for, or recovery from, diarrhoea, or processed but under restrictions which prevent unacceptable contamination of the processing floor. Normally where diarrhoea is apparent on post-mortem inspection, only the gastro-intestinal tract is condemned, unless there is peritonitis or other systemic involvement, in which case the whole carcass is condemned. The sensitivity of ante-mortem, slaughter and processing requirements in detecting and removing *S. typhimurium* DT104 infected pigs was estimated to be ‘extremely low’.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with *S. typhimurium* DT104 and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a 'one minus the background rejection rate'. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered 'extremely low'. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

One study has shown that *S. typhimurium* can be isolated from muscle of around 4% of bacteraemic pigs. Generally the carcasses of infected pigs are not themselves infected unless contaminated by infected faeces or blood. The risks of contamination during the slaughter process are well recognised. Abattoirs meeting the requirements of the Australian Standard have procedures and mechanisms in place to minimise the risk of infected faeces contaminating carcasses of infected pigs. Even so, once infected pigs have been slaughtered, a high proportion of these carcasses may become contaminated.

Taking these factors into consideration, the likelihood that *S. typhimurium* DT104 would be present in meat harvested for export from an infected or contaminated carcass was considered to be 'high'.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

Salmonella are hardy and ubiquitous pathogens, having been isolated from semi-dried, fermented beef sausage products and dry-cured sausages. They can survive a wide range of pH associated with processed and fermented food. Given this, it was considered that there was a 'high' likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

Salmonellae not only survive in chilled carcasses but have been reported to multiply at 7°C. Given this, it was considered that there was a 'high' likelihood that meat infected or contaminated with *S. typhimurium* DT104 at the completion of carcass maturation would remain infected during transport and storage.

Conclusions — release assessment

Based on the likelihoods above, the likelihood of entry was found to be 'very low'. Nonetheless, the likelihood of cross-contamination occurring on the slaughter line also needed to be considered. One study showed that the overall effect of contamination resulted in up to 13% more carcasses contaminated at the end of the slaughter line compared with the number of shedding pigs entering the slaughter process. Another study demonstrated that 30% of carcasses that tested positive for salmonellae at the end of the line were due to cross-contamination.

Moreover, it is known that carcasses are more likely to be contaminated at the end of the day than at the beginning.

After examination of the probability distribution that reflected the 'very low' likelihood of entry, and accounting for contamination, the likelihood that imported pig meat derived from an individual carcass would be infected remained 'very low'.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit will be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

Transmission of *Salmonella* organisms occurs via the oral route. Experimentally pigs have been infected orally with a dose of 10^{10} CFU *S. typhimurium* DT104. The minimum oral infectious dose for pigs is unknown, however, transmission appears to occur readily between pigs or in a contaminated environment. Humans can be infected with 10^2 to 10^5 CFU of salmonellae (Hogue, et al., 1997). Contaminated pig feed has also been implicated in the transmission, survival and multiplication of salmonellae (van Winsen, et al., 1999). On balance, it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of *S. typhimurium* DT104 to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

The likelihood that a pathogenic agent would remain viable after exposure to the environment will depend on its inherent 'stability'. Salmonellae multiply at 7°C to 45°C and can persist for years in suitable organic substrates. Although the agent's sensitivity to ultraviolet light and to the putrefying effects of saprophytic organisms may adversely affect bacterial growth and survival, it is recognised that salmonellae may be protected from exposure if meat waste is buried by other waste.

It was considered that the likelihood that *S. typhimurium* DT104 would remain viable after exposure to sunlight, putrefaction and the environment in meat scraps discarded in refuse for the period of time for feral pigs to locate and subsequently scavenge the material was 'high'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate

- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of the annual likelihood for each region was explained in Table 5.

- Remote regions = High
- Rural regions = High
- Large towns = Extremely low

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be ‘high’.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a ‘very low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was ‘high’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Although backyard pigs are likely

to be fed scraps or spoiled meat collected on a daily basis, such scraps are not usually kept chilled or frozen before being fed and growth of *S. typhimurium* DT104 may have occurred before being consumed by pigs.

Overall, the Panel considered that there was a 'high' likelihood that *S. typhimurium* DT104 would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'low'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection was 'high'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Although pigs in small commercial piggeries are likely to be fed scraps or spoiled meat collected on a daily basis, such scraps are not usually kept chilled or frozen before being fed and growth of *S. typhimurium* DT104 may have occurred before being consumed by pigs.

Overall, the Panel considered that there was a 'high' likelihood that *S. typhimurium* DT104 would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service

establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'high'.

Exposure assessment for other susceptible species

A wide range of carnivorous or omnivorous animal species, including wild birds, poultry, dogs, cats and rodents, can be infected with *S. typhimurium* DT104. Although there is no report of foxes being infected with *S. typhimurium* DT104, it is likely they too are susceptible to infection. Rodents, wild dogs, dingoes, foxes and feral cats are also known to scavenge refuse tips. In addition, rodents are likely to eat morsels of scraps left behind by backyard pigs and pigs in small commercial piggeries, while backyard poultry may be fed food scraps which contain pig meat. Dogs and cats, in cities, towns and on farms, may be fed meat scraps that may contain *S. typhimurium* DT104.

Given this, it was considered that the annual likelihood of entry and exposure for other susceptible species was 'high'.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the four exposure groups - that is, feral pigs, pigs in backyard enterprises pigs in small commercial piggeries and other susceptible species. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Pigs infected with *S. typhimurium* DT104 can become carriers and shed the bacteria in their faeces for relatively long periods of time. This, together with persistence within the environment, assists in wide distribution of the organism. High animal density is considered to increase the shedding by carriers as well as the susceptibility of exposed pigs (Schwartz, 1999). In the case of feral pigs, they tend to maintain small discrete groups, with some mixing between groups at times of low feed and water availability and at mating. They occupy a wide range of habitats and their movements are largely driven by food availability. As the faeces of feral pigs will be exposed to ultra-violet light this may affect the viability of salmonellae. These factors may limit the spread of the bacteria within the feral pig population.

Several studies have examined feral pig carcasses for the presence of salmonella infection. In Australia, one study found that while 53 (34.4%) of 154 feral pig carcasses were contaminated, only 9 (5.84%) were contaminated with *S. typhimurium* (Bensink, et al., 1991). Another study reported that 7% (11 of 156) of feral pig carcasses in Poland were contaminated with *Salmonella* spp (Wisniewski, 2001). A serological survey of wild boars in Spain found that 4% were positive for *Salmonella* serogroup B, which includes *S. typhimurium* (Vicente, et al., 2002).

Wildlife sharing the same habitat as feral pigs could also become infected if the environment became contaminated. Moreover, feral pig hunters and people that consume feral pig meat may be exposed to *S. typhimurium* DT104.

Spread of *S. typhimurium* DT104 to domestic pigs was considered less likely to occur as, while feral pigs are widespread in Australia, there is very limited close contact with domestic pigs. Nonetheless there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: moderate

Scenario 3: moderate

Scenario 4: low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

Backyard pigs are subject to a diverse range of feeding and management practices and may be kept where biosecurity is poor. Many are kept in simply constructed pens or allowed to roam in purpose-fenced paddocks or both. This may enable contact with other susceptible animals and/or feral pigs.

As most pigs infected with *S. typhimurium* DT104 are healthy carriers, it is not likely the infection would be detected before further spread, if any, has occurred. Spread to other animal species, particularly rodents, sharing the same pastures may occur, particularly if there is a concentration of animals sharing a common infected water supply. Environmental contamination is a feature of salmonellosis and spread via contaminated fomites, feed and transport occurs. Spread may also occur via the movement of pigs such as with the sharing of boars, and buying and selling breeder pigs on the local market. Infection of humans could occur either by direct contact with infected animals, or indirectly through the consumption of meat.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: very low

Scenario 3: moderate

Scenario 4: low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios. Generally, small commercial producers provide better biosecurity for their herds than do backyard pig producers. However, the greater movement of pigs, potential for environmental contamination, and spread via contaminated feed and trucks increases the likelihood of widespread distribution of Salmonella to pigs and other susceptible species including to humans via contaminated pork.

In some instances infection of pigs with *S. typhimurium* DT104 can result in clinical disease, although more often infected pigs are clinically healthy carriers of the bacteria. If disease did occur, this is more likely to be investigated in a small commercial piggery, but a confirmed diagnosis may take quite some time as endemic diseases causing diarrhoea, including salmonellosis due to non-exotic strains of *S. typhimurium*, would be considered initially.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: moderate

Scenario 4: moderate

Other susceptible species

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of other susceptible species:

1. Containment of the disease within the sub-population of directly exposed animals - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;

3. Secondary spread (via feral pigs or other means) to a local population of domestic pigs - containment within a local population of backyard enterprises or small commercial piggeries - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs - exposure of medium-large commercial piggeries and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

A wide range of animal and bird species can be infected by *S. typhimurium* DT104, especially rats, mice, feral cats, wild dogs and birds. Detection of infection in these species is unlikely unless secondary spread to domestic livestock, particularly cattle, or humans has occurred. *Salmonella typhimurium* DT104 is primarily a pathogen of cattle (Poppe, et al., 1998). Investigation of salmonellosis outbreaks on a farm often reveals infection in more than one species. The characteristics of *Salmonella* infections including multiple reservoir hosts, long term carriers and faecal shedding, persistence within the environment and effective use of transmission vectors generally ensure wide distribution (Schwartz, 1999).

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: very low

Scenario 2: low

Scenario 3: moderate

Scenario 4: moderate

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes ‘no further establishment or spread’. The remaining three outbreak scenarios describe ‘secondary spread to other pigs, humans or to other susceptible species’. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this scenario, the disease would have established in the directly exposed animal, or group of animals, but would not have spread to other animals or to humans. This ‘no outbreak’ scenario would have resulted from low probability of contact between infected and susceptible animals or humans, rather than from human intervention. Indeed, because the disease may be of low pathogenicity for pigs, it was assumed that it would not have been identified.

In view of this, it would not, under this scenario, have any discernible direct or indirect impacts and a rating of ‘A’ was assigned to each of the direct and indirect criteria.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, the disease would have established in a broader population of feral pigs, and possibly spread to humans (such as feral pig hunters, owners and family members of

backyard enterprises) either via direct contact with contaminated carcasses or indirectly through the consumption of contaminated pig meat. The disease would likely be diagnosed through illness in humans, however, it was considered unlikely that eradication programs would be implemented with feral pigs. Some control programs may be implemented such as to minimise contamination of feral pig meat. Containment, under this scenario, would be due natural means rather than through human intervention.

The direct impact of Salmonella typhimurium DT104 infection

Animal life or health

Infection with *S. typhimurium* DT104 in pigs is often subclinical. In some cases, pigs can develop diarrhoea. There is limited information on the clinical signs of infection in other carnivorous or omnivorous susceptible species such as rodents, and dogs. Exposure to the organism is often demonstrated in surveys. A case report of *S. typhimurium* DT104 in a cat described diarrhoea, fever and vomiting.

Based on this information the direct impact of *S. typhimurium* DT104 on animal health, under this scenario, was considered unlikely to be discernible at any level, and a rating of 'A' was given to this criterion.

Environment

Although *S. typhimurium* DT104 may infect native animals, it was considered, based on the behaviour of other salmonellae, that disease would be mild or subclinical. Hence, it was considered that the direct impact on the environment was unlikely to be discernible at any level, and the rating assigned to this criterion was 'A'.

The indirect impact of Salmonella typhimurium DT104 infection

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

Detection of *S. typhimurium* DT104 in feral pig hunters or other people who consumed contaminated pig meat may result in additional testing of pig meat carcasses to determine the prevalence of infection. Education programs may be undertaken to alert hunters to appropriate handling procedures to minimise contamination to themselves and other carcasses. Salmonellosis is notifiable in humans, and the disease is subject to surveillance and intensive epidemiological investigation.

Overall, it was considered that the indirect impact of control and eradication programs was unlikely to be discernible at any level, except locally, and a rating of 'B' was assigned for this criterion.

Domestic trade or industry effects

With the disease contained within feral pigs it was considered that indirect impact on domestic trade or industry would be unlikely to be discernible at any level. Hence, a rating of 'A' was assigned for this criterion.

International trade effects

The spread of *S. typhimurium* DT104 to the general population of feral pigs may result in additional testing of feral pig meat carcasses for export. Australia exported over $24 million (3,600 tonnes) in feral pig meat in 2001, mainly to Europe. If an outbreak occurred overseas that was linked to exported Australian feral pig meat, this would likely result in a recall of the consignment, an audit of the export process, and implementation of effective risk management measures to minimise carcass contamination.

In view of this, the likely indirect effect on international trade was, under this scenario, considered unlikely to be discernible except locally. Thus a rating of 'B' was assigned to this criterion.

Indirect impact on the environment

In this scenario, *S. typhimurium* DT104 infection is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, *S. typhimurium* DT104 would have established in a local population of backyard piggeries or small commercial piggeries, and possibly spread to pig handlers, abattoir workers or consumers and other susceptible species such as cattle. The disease would be diagnosed through illness in humans, or animals. Control programs may be implemented to contain the disease in domesticated livestock.

The direct impact of Salmonella typhimurium DT104 infection

Animal life or health

Although subclinical infection is a feature of infection in pigs, morbidity and mortality can be high for infected cattle. In cattle, clinical infections with *S. typhimurium DT104,* usually present with loss of appetite, loss of condition, decreased milk production and watery diarrhoea, and may subsequently abort. Because, in this outbreak scenario spread has only occurred to a local population of animals, its direct effect on animal life or health was considered likely only to be discernible at the local level. This gave the disease a rating of 'B' for this criterion.

Environment

Although *S. typhimurium* DT104 may infect native animals, it was considered, based on the behaviour of other salmonellae, that disease would be mild or subclinical. Hence, it was considered that the direct impact on the environment was unlikely to be discernible at any level, and the rating assigned to this criterion was 'A'.

The indirect impact of Salmonella typhimurium DT104 infection

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

When *S. typhimurium* DT104 was first detected in Denmark, a stamping-out program was implemented, and premises disinfected before restocking. Fifteen herds were slaughtered out before it was realised that this option was rapidly becoming uneconomical (Nielsen, et al., 2001). Now control is by managing risks effectively and improving biosecurity of piggeries. Programs involving changing management practices at the farm, in transporting pigs, and at abattoirs were introduced. These programs also required regular on-farm testing of stock to monitor progress and to determine if freedom from disease has been achieved.

There is no official control, eradication or compensation program for salmonellosis in livestock in Australia. Nevertheless, in this limited outbreak, eradication may be feasible and a control program similar to that applied in Denmark could be introduced. Given this, the indirect effect of control and eradication programs was considered unlikely to be discernible at the national or State level, but would be of minor significance at the district or regional level. Thus a rating of 'C' was assigned to this criterion.

Domestic trade or industry effects

Outbreaks of *S. typhimurium* DT104 in various animal species in North America and Europe would appear to have had little effect on the domestic trade or associated industries. Nonetheless recall of contaminated products can result in damaged consumer confidence and future sales of these products may be affected.

Given this, the indirect impact of *S. typhimurium* DT104 outbreaks on domestic trade was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. This resulted in a rating of 'C' for this criterion.

International trade effects

Salmonellosis of livestock is not an OIE listed disease, though the OIE Code does provide guidelines for salmonellosis in poultry. Outbreaks of *S. typhimurium* DT104 in the United States of America and Europe do not appear to have had any significant effect on international trade in meat and livestock. Nonetheless additional risk management measures may need to be implemented to minimise carcass contamination in all export-approved abattoirs.

In view of this, the likely indirect effect on international trade was considered unlikely to be discernible at the national or State level, but of minor significance at the district or regional level. Thus a rating of 'C' was assigned to this criterion.

Indirect impact on the environment

In this scenario, *S. typhimurium* DT104 infection is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, the disease has become widespread, including spread to commercial piggeries and other susceptible species such as cattle, sheep and wildlife. Spread to humans could occur either through direct contact with infected animals or indirectly through the consumption of infected products. Control measures on farm and in abattoirs and processing plants are likely to be implemented.

The direct impact of Salmonella typhimurium DT104 infection

Animal life or health

In cattle the disease is likely to cause productivity losses from livestock deaths, increased cull rates, reduced feed efficiency and decreased weight gain. As clinical signs are rarely seen in pigs, productivity losses would be minimal in this species.

On balance, the direct effects on animal health were, under this scenario, considered unlikely to be discernible at the national or State level and of minor impact at the district or regional level. This resulted in a rating of 'C' for this criterion.

Environment

Although *S. typhimurium* DT104 may infect native animals, it was considered, based on the behaviour of other salmonellae, that disease would be mild or subclinical. Hence, it was considered that the direct impact on the environment was unlikely to be discernible at any level, and the rating assigned to this criterion was 'A'.

The indirect impact of Salmonella typhimurium DT104 infection

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

Because *S. typhimurium* DT104 is a zoonosis and can possess resistance to a range of antibiotics commonly used in treatment of salmonellosis, various risk management programs to reduce herd infections, spread of infection between herds, stress in livestock whilst being transported to saleyards and abattoirs and contamination of carcasses in abattoirs are likely to be implemented, to minimise the zoonotic impact of this disease. Some of these measures may already be in place for other endemic *Salmonella* infections.

Given this, the indirect effect of control and eradication programs was considered unlikely to be discernible at the national or State level, but would be of minor significance at the district or regional level. Thus a rating of 'C' was assigned to this criterion.

Domestic trade or industry effects

The indirect effect of *S. typhimurium* DT104 outbreaks on domestic trade or industry, under this outbreak scenario, was considered unlikely to differ to that described above for scenario 3. Thus a rating of 'C' was assigned to this criterion.

International trade effects

The indirect impact of *S. typhimurium* DT104 on international trade was considered likely to be similar under this scenario as was described for scenario 3 (see above). A rating of 'C' was thus assigned to this criterion.

Indirect impact on the environment

In this scenario, *S. typhimurium* DT104 is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

The overall impact of *Salmonella typhimurium DT104*

When the direct and indirect impacts of *S. typhimurium* DT104 were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences negligible

Scenario 3: Consequences very low

Scenario 4: Consequences very low

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 65, Table 66, Table 67, and Table 68. It can be seen that the likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were all 'very low'. The likely consequences associated with the exposure of other susceptible species to infected pig meat scraps were considered 'very low'.

Table 65 ***S. typhimurium*** **DT104: summary of the consequences of exposure of feral pigs**

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Moderate	Negligible	Negligible
Scenario 3	Moderate	Very low	Very low
Scenario 4	Low	Very low	Negligible
		Overall likely consequences	**Very low**

Table 66 ***S. typhimurium*** **DT104: summary of the consequences of exposure of backyard pigs**

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Moderate	Very low	Very low
Scenario 4	Low	Very low	Negligible
		Overall likely consequences	**Very low**

Table 67 ***S. typhimurium*** **DT104: summary of the consequences of exposure of small commercial piggeries**

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Moderate	Very low	Very low
Scenario 4	Moderate	Very low	Very low
		Overall likely consequences	**Very low**

Table 68 *S. typhimurium* DT104: summary of the consequences of exposure of other susceptible species

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Very low	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Moderate	Very low	Very low
Scenario 4	Moderate	Very low	Very low
		Overall likely consequences	**Very low**

Human life or health

Separate to the above is consideration of the consequences to human life or health. *Salmonella typhimurium* DT104 is a zoonosis. This variant created concern among the public health community due to its broad antimicrobial resistance patterns. The genes encoding this broad antibacterial resistance are integrated into the bacterial chromosome. This type of encoding suggests that any removal of the selective pressure from antimicrobials is not likely to reverse resistance as can happen with extra-chromosomal or plasmid mediated resistance. This clone has spread considerably throughout the northern hemisphere in the past decade and, in some cases, has developed resistance to other antibiotics including fluroquinolone (Baggesen, et al., 2000).

Outbreaks of *S. typhimurium* DT104 in humans as a result of eating pork products have been reported in Denmark (Baggesen, et al., 1999). Human infections as a result of direct exposure to infected animals or carcasses have also been reported (Wall, et al., 1994).

Clinical signs of infection with multiple-drug resistant *S. typhimurium* DT104 in humans include diarrhoea, fever, headache, nausea, vomiting and abdominal pain, and in some cases, bloody diarrhoea (Akkina, et al., 1999). Human infections due to *S. typhimurium* DT104 have resulted in higher rates of admissions to hospitals and mortality than many other *Salmonella* spp (Wall, et al., 1994). In a case-control study of 83 cases in the United Kingdom that were attributed to *S. typhimurium* DT104, 34 (41%) patients were hospitalised, and 10 of 295 (3%) patients identified during the study period died (Wall, et al., 1994). In contrast, the case fatality rate for *Salmonella* infections other than multiple-drug resistant *S. typhimurium* DT104 was approximately 0.1 %. In the United Kingdom during 1995, this strain was associated with hospitalisation rates which were twice that of other zoonotic food-borne *Salmonella* infections and with ten times higher case fatality rates (World Health Organization, 1997).

In 1998 in the United States of America, the estimated economic cost of human illness due to food borne *Salmonella* infections was $US 2.3 billion. As between 6% and 9% of infections were due to consumption of infected pork and pork products, the economic costs of salmonellosis due to consumption of pork was $US 0.1 to $US 0.2 billion. Breakdown of costs showed medical expenses was $US 3.8 million per fatal case, $US 5452 per hospitalised case, $US 316 per case visiting a physician only, and $US 24 recovering without medical care (Frenzen, et al., 1999). This is substantially lower than $US 1.1 billion annual costs of illness due to *Salmonella* contaminated eggs (Frenzen, et al., 1999). In Netherlands, pork and pork products account for around 5% to 25% (average 15%) of all human salmonellosis cases.

A cluster of 14 human cases of *S. typhimurium* DT104 infection occurred in Australia in 2001, following consumption of food imported from Turkey. The outcomes of the outbreak included successful medical treatment of all cases and the identification and international recall of the imported product. The same product had also caused human cases in Sweden and Germany.[60]

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with *S. typhimurium* DT104.

Table 69 shows the results obtained for each of the exposure groups and for the overall annual unrestricted annual risk. Since the overall annual risk meets Australia's ALOP (very low), risk management would not be required for *S. typhimurium* DT104.

Table 69 *S. typhimurium* DT104: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Very low	High	Very low	Very low
Backyard pigs	Very low	Low	Very low	Negligible
Small commercial piggeries	Very low	High	Very low	Very low
Other susceptible species	Very low	High	Very low	Very low
			Overall annual risk	**Very low**

The Department of Health and Ageing has advised Biosecurity Australia that biosecurity measures would be required for *S. typhimurium* DT104 to manage the risk to human health associated with the importation of pig meat should the disease enter and establish in the Australian animal population. An appropriate measure would include imported processed (cooked, cured) pig meat classified as 'Risk' must comply with the Food Standards Code

[60] Eurosurveillance Weekly 2001 http://www.eurosurv.org/2001/010816 .htm

including testing for *Salmonella*. No additional measures are required for imported uncooked pig meat which is processed in Australia prior to retail sale.

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Swine influenza virus

Technical information

Background

Swine influenza can cause outbreaks of acute respiratory disease of high morbidity and low mortality in pigs. As with many viral respiratory diseases, swine influenza is often complicated by concurrent or secondary bacterial infection.

Swine influenza is not reported in Australia and New Zealand, although it occurs in most other pig-producing countries of the world. A serosurvey of pigs across northern Australia between 1997 and 1999 showed all 421 pigs tested to be seronegative to swine influenza.[61]

Agent taxonomy

Swine influenza is caused by an enveloped RNA type A influenza virus belonging to the Orthomyxoviridae family. However, an influenza virus type C has been isolated from pigs in China (Kimura, et al., 1997).

Influenza viruses are classified according to the two groups of structural proteins present in the virus, fifteen haemagglutinin (H) and nine neuraminidase (N) surface antigens. According to these criteria, swine influenza is associated with H1N1 and, more recently, H3N2 strains. Other strains isolated from pigs in recent times include H1N2 in Indiana (Karasin, et al., 2000b), H4N6 (avian influenza strain) in Ontario (Karasin, et al., 2000a) and H9N2 in south eastern China (Peiris, et al., 2001).

Agent properties

Swine influenza virus is relatively labile, susceptible to heat (56°C for at least 30 minutes) and radiation, and can survive for several hours in dried mucus (Health Canada, 2001). It is rapidly inactivated at extremes of pH, being stable between pH 5.5 and 8.0 (Stallknecht, et al., 1990; Health Canada, 2001; Takahashi, et al., 2001). Survival of the virus outside the animal is prolonged by low relative humidity and low temperature in aerosols. Swine influenza virus, maintained at 21°C in aerosol form, was inactivated after 15 hours (Mitchell & Guerin, 1972). Although there are little data on inactivation of swine influenza in the environment, equine influenza virus has been shown to maintain viability for up to 24 hours in soil, in the dark at a temperature of 15 to 18°C, and for 8 hours in the presence of sunlight (Yadav, et al., 1993). Duck strains of avian influenza virus persisted for up to 100 days in water at 17°C but persistence decreased with increasing salinity and pH (Stallknecht, et al., 1990).

Host range

Pigs are the principal hosts of swine influenza virus, although there is increasing evidence for the transmission of some strains between avian and mammalian hosts. Swine influenza virus or swine influenza like virus has also been isolated from ducks, turkeys and humans (Bachman, 1989). Swine respiratory tract epithelial cells have receptors for both avian and mammalian influenza viruses and pigs are believed to serve as mixing vessels for development of new reassorted strains of influenza viruses (Ito, et al., 1998).

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[61] Personal communication from Tim Kerlin, Senior Scientific Officer, Northern Australia Quarantine Strategy,Cairns, Queensland.

It is currently believed that a mammalian strain, probably swine influenza virus, was responsible for the 1918 pandemic of influenza in humans (Taubenberger, et al., 1997). The public health aspects of swine influenza have been reviewed (Easterday & Van Reeth, 1999). In 1976 the zoonotic potential of swine influenza was confirmed when the caretaker of a pig farm became ill several days after the pigs had shown signs of influenza. Characterisation of the viruses from the man and pigs were identical. Prior to this case several hundred military recruits had been infected with an influenza virus closely related to swine influenza. There have been several other isolated reports of humans infected with swine influenza virus, although generally there has been minimal spread from human to human. Some of these infections have resulted in acute, fatal respiratory disease in humans.

It should be noted that human influenza viruses and avian influenza viruses can also infect pigs (Ito, 2000).

Epidemiology

Swine influenza is transmitted by the nasopharyngeal route as a result of close or direct contact between pigs. Nasal secretions of pigs have a high concentration of virus during the acute stages of the disease and the virus is transmitted by aerosols over a short distance. The disease usually spreads to new areas and farms by the movement of infected pigs (Leman, et al., 1974; Ramírez, et al., 2002). Infection with, or vaccination against, one strain does not necessarily provide cross-protection against other strains, as outbreaks of H3N2 have been reported in pigs previously infected with, or vaccinated, against H1N1 (Yoon, et al., 1999).

Most infected pigs shed the virus for only 5 to 7 days in their nasal secretions, but some can shed the virus for up to 30 days post-infection (Vannier, et al., 1985). Virus titres of up to $10^{7.5}$ EID_{50}/ml (egg infectious $dose_{50}$) in nasal mucus have been reported for 5 to 7 days after infection (Larsen, et al., 2000). In one experiment, one pig excreted swine influenza virus for over 4 months (Blaskovic, et al., 1970). However, there is no evidence of a true carrier state in pigs. Susceptible pigs have been experimentally infected with $10^{4.4}$ to $10^{5.3}$ EID_{50} units to initiate classical signs of swine influenza (Brown, et al., 1993; Lee, et al., 1995; Larsen, et al., 2000).

Climatic stress influences the expression of clinical disease. Outbreaks can be seasonal, tending to occur in late autumn and early winter. Epidemics are often explosive, with outbreaks occurring on most pig farms in a locality over a short period. Virological and serological surveys have shown that swine influenza virus can circulate at low levels in pig populations at other times of the year without causing overt disease (Ramírez, et al., 2002).

Serological surveys conducted for swine influenza virus demonstrate widespread exposure of pigs and herds. However, in some cases, animals may be seropositive due to vaccination. A National Animal Health Monitoring System (NAHMS) survey in the United States of America suggested that 31.0% of breeding females and 15.3% of weaned market pigs, from 6.2% of swine herds were vaccinated against swine influenza virus (USDA, 2002). In Korea, a survey of 22 to 24 week old finisher pigs in 130 herds showed 71.5% of herds were positive against H1N1 strain (Jung, et al., 2002). In Belgium in a region with high density of pigs, 50 to 100% of unvaccinated pigs from all 17 herds under study seroconverted to H1N1 soon after entering finishing facilities (Geering, et al. 1995). In Denmark, 20-week-old pigs in 4 and 7 of 9 herds surveyed were seropositive to H3N2 and H1N1 viruses respectively (Andreasen, et al., 2000). In the United States of America, of 188 herds surveyed in 18 states, 56% were seropositive to H3N2 viruses (Webby, et al., 2000). Following the emergence of H3N2 in Iowa in December

1998, a serosurvey of 1064 finishing pigs from 129 Iowa pig herds by June 1999 showed that 64% of pigs and 92.2% of herds had serological evidence of exposure to H3N2 (Yoon, et al., 2000). A vaccine against H3N2 became available in June 1999.

A survey of nearly 4400 pigs in 23 states in the United States of America showed that 28.3% had been exposed to H1N1 viruses and 20.5% exposed to the H3N2 viruses, with 9.8% of pigs having antibody to both strains. Overall, 39% of pigs were seropositive to swine influenza virus (Webby, et al., 2000). In Minnesota, 22.8% of 111,418 pig sera tested positive for both H1 and H3 swine influenza virus antibody between 1998 and 2000 (Choi, et al., 2002). In north-central United States of America, 27.7% of 2375 pigs tested seropositive to H1 influenza virus. (Olsen, et al., 2000). In a smaller survey of 323 young pigs on 16 farms, 31.3% and 7.4% were seropositive to H1N1 and H3N2 respectively (Lee, et al., 1993). A serosurvey of pigs of all ages in north-central United States of America in 1988 to 1989 showed 51% to be seropositive to swine influenza (Chambers, et al., 1991). In another study conducted in the United States of America, the within herd seroprevalence of 16 week old pigs averaged 63% and for 24 week old finisher pigs averaged 70% (Regula, et al., 2000).

In a study conducted more than 20 years ago on slaughter swine in the United States of America, swine influenza virus (H1N1) was recovered from nasal secretions of approximately 5% of 9400 pigs tested. Antibodies against swine influenza virus were found in 21% of those pigs (Hinshaw, et al., 1978a). More recently in the United States of America samp1es of nasal secretions were collected from 1200 pigs at the time of slaughter over a period of 1 year for virus isolation. Overall 2.2% of pigs were positive for swine influenza virus, although virus shedding rates were up to 16% between October and January (Olsen, et al., 2000).

Clinical signs

In naïve herds infection with swine influenza virus results in sudden onset of disease in pigs of all ages. The clinical signs include high fever, anorexia, inactivity, huddling, nasal and ocular discharge and a barking cough. Unless secondary bacterial infections develop, pigs usually recover within a week. Morbidity can often approach 100% but mortality is uncommon (around 1%) unless infection is complicated by secondary bacterial infections (Easterday & Van Reeth, 1999).

The enzootic form is now more common, particularly in the United States of America, as most herds are not naïve. Clinical signs include mild but chronic respiratory problems in grower and finisher pigs and decreased feed efficiency in all pigs. There have been reports that infection with swine influenza virus may result in occasional abortion, causing a 5 to 10% drop in farrowing rates, although data are not available to support these reports (Easterday & Van Reeth, 1999). The enzootic form of swine influenza is often seen in conjunction with other diseases (Janke, et al., 2001). Annual re-infection is common and morbidity is high in the younger naïve pigs.

In addition to clinically apparent disease, subclinical infections occur frequently, as indicated by the high seroprevalence reported in finishing pigs.

Pathogenesis

The incubation period ranges from 1 to 3 days but can be as short as 4 hours. Following intranasal uptake of swine influenza virus, the virus attaches to cilia and adsorbs to the membranes of the nasal, tracheal and bronchiol mucosa cells. After primary replication in single epithelial cell, swine influenza virus spreads throughout the respiratory tract within 1 to 3

days. Viraemia has only rarely been detected with the lungs being the major target organ. The amount of virus that reaches the lower respiratory tract and the resulting production of infectious virus in the lungs seem to determine the severity of illness (Bachman, 1989).

Pathology

In uncomplicated cases of swine influenza, lesions are normally restricted to the respiratory tract. Observations include lungs with a few firm lobular lesions with interlobular oedema and enlarged and haemorrhagic bronchial and mediastinal lymph nodes (Easterday & Van Reeth, 1999). Swine influenza virus can cause widespread, well-defined interstitial pneumonia, with lungs that appears purplish and firm. The virus can also damage the mucociliary lining of the trachea making it difficult for the lungs to clear the infection. Consequently, this results in congestion of the mucosae in the upper respiratory tract.

Immunology

Swine influenza virus can elicit both cell-mediated immune and humoral responses in pigs. Antibodies (IgM and IgG) start to rise around 3 days post-infection. IgG becomes routinely detectable around 7 to 8 days post-infection, peaks around 10 to 17 days later, although in some cases as long as 3 weeks later, and persists at fairly high levels for 4 to 6 months post-infection, before becoming undetectable after 16 to 18 months (Lee, et al., 1993; Larsen, et al., 2000). IgM usually peaks around 7 days post-infection at which time serum values start to fall. Maternal antibodies transferred from the dam to newborn piglets can persist for 2 to 4 months, depending on the initial level. Because of the antigenic heterogeneity of swine influenza virus, cross-protection from other strains can vary. Humoral response to vaccination is similar to that seen in infection, but induces a lower level of antibody response.

Transmission in meat

Virus can be isolated from nasal swabs and from tissues of the respiratory tract for up to 4 days after infection, but not from faeces, liver and spleen. Virus has been isolated from the turbinates and tonsils of experimentally infected pigs killed 1, 2 and 3 days after infection but not 7 days after infection (Brown, et al., 1993). In the same study virus was isolated from serum samples ($10^{0.4}$ to $10^{4.9}$ EID_{50}/ml) from infected animals for a period of only 1 day between 1 and 3 days post-infection. In other studies viraemia has not been detected or virus has only been isolated occasionally from serum samples in the first few days post-infection (Styk, et al., 1971; Wallace & Elm, 1979). Virus was not isolated from tissues other than those of, or associated with, the respiratory tract.

Although swine influenza virus has not been detected in tissues beside those of, or associated with, the respiratory tract, one study examined the effects of storage on virus titres of meat immersed in, and injected with, a suspension of swine influenza virus of $10^{3.7}$ to $10^{7.7}$ EID_{50} per ml. The study demonstrated, that there was a progressive loss of virus during the storage period, slightly faster when stored refrigerated at 4°C than deep frozen at -20°C. Virus could be recovered from some contaminated meat 8 days later but no virus could be recovered from contaminated meat after 15 days storage at 4°C (Romijn, et al., 1989).

Release assessment

R1 — the likelihood that a source herd is infected

Serological surveys for swine influenza virus demonstrate widespread exposure of herds with prevalence reported as ranging from 56 to 100%. Based on this information, it was considered that there was a 'high' likelihood that the herd from which slaughter-age pigs were selected would be infected.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

The within herd seroprevalence of finisher pigs has been reported in several studies. In Belgium 50 to 100% of pigs seroconverted soon after entering finishing facilities. Whereas in the United States of America one study reported that 63% of pigs within a herd had seroconverted by 16 weeks of age with an additional 7% of pigs seroconverting in the next 8 weeks (70% seroprevalence for 24 week old finisher pigs). These figures concur with those reported in Iowa, where 64% of finisher pigs had serological evidence of exposure to swine influenza virus. It is recognised that swine influenza causes acute infection with viral shedding only for about 5 to 7 days. As such, pigs exposed to the virus during the finishing period may no longer be infected at the time of slaughter. Nonetheless in a study conducted many years ago virus was isolated from nasal secretions of approximately 5% of 9400 pigs at slaughter. More recently, swine influenza virus was isolated from nasal secretions of 2.2% of slaughter-age pigs although on a seasonal basis this could be as high as 16%. Given this, it was considered that the likelihood that an infected pig was selected from an infected herd was 'low'.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

Ante-mortem inspections include observing pigs for clinical signs of ill health before slaughter. According to the Australian Standard, pigs with fever are to be condemned or withheld from slaughter until recovered, provided there is no risk of spread of disease and recovery is likely. Pig farmers are unlikely to send pigs with clinical signs of acute fever due to swine influenza to abattoirs due to the high costs of condemnation. Where the clinical signs are mild, as is often the case in finishing pigs, these pigs would pass ante-mortem inspection but the lungs and associated lymph nodes may be condemned. As such, the sensitivity of ante-mortem, slaughter and processing requirements in detecting and removing pigs infected with swine influenza virus was considered to be 'extremely low'.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with swine influenza virus and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a 'one minus the background rejection rate'.

Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered ‘extremely low’. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

Swine influenza virus exhibits marked tropism for the respiratory tract, which is removed after slaughter. The virus has not been recovered from lymphoid tissues, such as liver and spleen, or the faeces of infected pigs. Viraemia, if it occurs, is extremely short, virus being isolated from serum for only 1 day from some pigs in the first few days of infection. Moreover, bleeding the carcass should remove, to a large extent, the virus contaminating muscle due to viraemia. Given this, the likelihood that swine influenza virus would be present in meat harvested for export was considered to be ‘extremely low’.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

Influenza virus is stable over a pH range of 5.5 to 8.0. As stated in the Method for Import Risk Analysis, it has been assumed that pig meat harvested for export will not attain a pH lower than 6.2. Thus, it was considered that there was a ‘high’ likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

There are little data on the effects of temperature on the viability of swine influenza virus. One review reported that the virus was not stable at -20°C and recommended storing samples at -70°C (Easterday & Van Reeth, 1999). In meat that was artificially contaminated with swine influenza virus there was progressive loss of virus when meat was stored at 4°C. Virus could be recovered from some experimentally contaminated meat 8 days later but not after 15 days storage. In light of this information, it was considered that there was a ‘moderate’ likelihood that meat infected at the completion of carcass maturation would remain infected during transport and storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was an ‘extremely low’ likelihood that imported pig meat derived from an individual carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was an 'extremely low' likelihood that a waste unit will be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

No information was found on the oral infectious dose of swine influenza virus. It is known that transmission occurs via the nasopharyngeal route. Generally under experimental conditions pigs are infected by instillation of virus suspensions into the nostrils. However, pigs have been infected with swine influenza virus at a dose of $10^{4.4}$ EID_{50} units by inoculation in three equal doses given intranasally, aerogenically by nebuliser, and orally (Brown, et al., 1993).

As discussed previously, swine influenza virus has only been detected in respiratory tissues and hence it was considered that any virus, if present in meat, would be due to blood contamination. Nevertheless most blood is removed at slaughter during the process of exsanguination. In a small experimental study involving five pigs, virus was detected in sera on only 1 day from each of the pigs. Three of the pigs had a titre of $10^{1.4}$ EID_{50}/ml, one pig a titre of $10^{0.4}$ EID_{50}/ml and the other a titre of $10^{4.9}$ EID_{50}/ml.

Given this, it was considered the likelihood that a waste unit from an infected pig would contain a sufficient dose of swine influenza virus to initiate infection was 'extremely low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

There is very little information on the viability of swine influenza virus in porcine tissues at ambient temperatures. The virus is reported to be relatively labile and susceptible to heat and radiation. As swine influenza virus is an enveloped virus, adverse conditions such as ultraviolet irradiation may affect the envelope and consequently destroy the virus.

Given this, the Panel considered the likelihood that swine influenza virus would survive within meat scraps discarded in refuse for the period of time required for pigs to locate and subsequently scavenge the material was 'low'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = Extremely low
- Rural regions = Negligible
- Large towns = Negligible

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be ‘extremely low’.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was an ‘extremely low’ likelihood that a waste unit will be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was ‘extremely low’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed

scraps collected on a daily basis although in some instances pig meat may be discarded due to spoilage.

Overall, the Panel considered that there was a 'moderate' likelihood that swine influenza virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'negligible'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was an 'extremely low' likelihood that a waste unit will be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'extremely low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be discarded due to spoilage.

Overall, the Panel considered that there was a 'moderate' likelihood that swine influenza virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food

service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be ‘extremely low’.

Exposure assessment for other susceptible species

Other susceptible species that could become directly infected with swine influenza virus after eating contaminated pig meat scraps include some avian species. Ducks and other waterfowls are considered to be the principal natural hosts of influenza A viruses. Waterfowls can be either vegetarian or carnivorous, with many species eating both plants and animals, depending on their habitat, food availability and season. Waterfowls are rarely seen at refuse tips except at times of food shortage. Pigs have been known to be infected by waterfowls (Karasin, et al., 2000a) and swine influenza like viruses have been isolated from ducks (Butterfield, et al., 1978; Hinshaw, et al., 1978b) and turkeys (Wood, et al., 1997). It is possible that backyard turkeys may be fed meat scraps, however, there is no epidemiological evidence to indicate that transmission of swine influenza virus has occurred by this means. Generally outbreaks in turkeys with viruses similar to swine influenza occurred when turkeys were in close proximity to pig herds (Mohan, et al., 1981).

Based on this information, the annual likelihood of entry and exposure for other susceptible species was considered ‘extremely low’.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete ‘outbreak scenarios’ for each of the four exposure groups - that is, feral pigs, pigs in backyard enterprises and pigs in small commercial piggeries and other susceptible species. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of ‘consequences’ for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Swine influenza virus is highly contagious in the domestic pig population. However, in the more dispersed feral pig populations only 3 of 78 wild boars (4%) in Spain (Vicente, et al., 2002) and 13 of 120 wild swine (11%) in Oklahoma (Saliki, et al., 1998) were seropositive to swine influenza virus.

Transmission of the virus occurs through close or direct contact between pigs. In the case of pigs infected with swine influenza virus shedding occurs for only a relatively short period, for about 1 week. In naive pigs the clinical signs of swine influenza infection include high fever, inactivity, huddling and coughing.

In Australia, feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs, however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves. Spread of swine influenza virus from feral pigs to domestic pigs has not been reported in other countries.

It is likely that the virus would spread quickly within the directly exposed feral pig herd. However, due to the changes in behaviour that could occur if pigs were infected with swine influenza virus, inactivity, huddling, these pigs may not move the distances that healthy pigs move, and indeed might not move very much at all whilst infectious.

Were transmission to a piggery to occur, it is likely that the disease would be amplified and would spread to other piggeries before a diagnosis was established and controls to minimise spread implemented.

It is possible that other susceptible species may become infected with swine influenza virus. Infection in humans is rare, despite the high prevalence of infection in pigs worldwide, and generally has occurred in people who have had close contact with infected pigs. Where humans have been infected with swine influenza virus, there has been limited secondary spread to other humans. Sporadic cases of swine influenza virus infection in turkeys have been reported when birds have been in close proximity to pig herds.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: moderate

Scenario 3: very low

Scenario 4: low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

As discussed above, it is feasible that backyard pigs kept in rural areas may come in close contact with nocturnally foraging feral pigs, and that transmission of swine influenza virus from one group to the other may result. There may also be some movement of backyard pigs to other premises which could result in spread of the virus, for example, in the case of speciality breeds or unusual breeds being transferred from one herd to another for breeding purposes. It is unlikely that sick pigs would be moved, but pigs incubating the virus would not be showing clinical signs. Alternatively, pigs raised for personal consumption may be transferred between backyard holdings for growing out or fattening. However, often backyard pigs will be raised for consumption by that household.

Indirect spread of swine influenza virus could also occur through local sharing of equipment contaminated with nasal secretions, although the virus is unlikely to persist on equipment for long periods.

It was stated above that were transmission to a piggery to occur, it is likely that the disease would be amplified and could spread to other piggeries by live pigs or other means. If large commercial piggeries were also situated within the area, spread to these might occur, and that this would subsequently lead to a more general outbreak.

Owners of backyard pig are less likely to seek veterinary advice, than commercial operators. In addition with a small number of pigs and fewer introductions of naïve pigs in backyard herds, clinical signs of disease may not be ongoing i.e. there may be only an initial bout of respiratory disease, from which most pigs will recover.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: moderate

Scenario 4: low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

The high morbidity resulting from infection with swine influenza virus is more likely to be investigated in a small commercial piggery than backyard enterprises. In addition, in herds with breeding sows there will be a steady introduction of naïve piglets with ongoing evidence of clinical disease. As endemic causes of respiratory disease will be investigated first, it is likely that there will be a delay in diagnosis which could result in spread of the disease to other piggeries.

Most pigs from a small commercial piggery will go directly to slaughter, however, the virus could be spread via contaminated trucks and equipment. Moreover, some pigs may be purchased as stores for other piggeries or as breeders particularly in the case of rare breeds.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: low

Scenario 4: moderate

Other susceptible species

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of other susceptible species:

1. Containment of the disease within the sub-population of directly exposed animals - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to other susceptible species, but no spread to feral or domestic pig herds - no spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to domestic pigs - exposure of medium-large commercial piggeries and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Ducks and turkeys have been shown to be susceptible to swine influenza or swine influenza-like virus. Reported cases in turkeys appear to have been localised and showed no tendency to spread (Mohan, et al., 1981). Nonetheless it has been stated that ducks and possibly turkeys, as well as swine may serve as a reservoir for influenza virus for man. Infection in ducks would not be diagnosed on clinical signs as none has been reported. In turkeys it has been documented that infection with swine influenza virus can cause respiratory disease, a decline in egg production and an increase in the number of abnormal eggs (Mohan, et al., 1981). It is feasible that diagnosis may only occur if the virus spreads to the human or pig population.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: very low

Scenario 3: moderate

Scenario 4: very low

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each exposure group follows a similar pattern with the exception of 'other susceptible species'. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this scenario, swine influenza would have established in the directly exposed animal, or group of animals, but would not have spread to other pig herds, humans or other susceptible animals. This 'no outbreak' scenario would have resulted from low probability of contact between infected and susceptible animals, rather than from human intervention. Indeed, because clinical signs of the disease could be confused with endemic disease, it was assumed that it would not have been identified.

The direct impact of swine influenza

Animal life or health

Infection with swine influenza virus can cause acute respiratory disease in pigs of all ages. Severity of clinical disease is influenced by a range of factors including virus strain and co-occurrence of secondary infections. The clinical signs include high fever, inactivity, huddling and coughing. Morbidity is high often approaching 100%, but mortality is uncommon. Pigs usually recover within a week.

In other susceptible species known to have been infected with swine influenza or swine influenza-like virus, there may be no clinical signs of disease as is the case with ducks, or respiratory disease and a decline in egg production in the case of infection in turkeys.

On this basis, the likely impact of swine influenza virus in terms of *animal health* was considered unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

Environment

Swine influenza virus may infect native Australian waterfowl, however, clinical signs are likely to be absent. Given this, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of swine influenza

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this scenario, where the disease is contained within the initial exposure group with no secondary spread, it was considered unlikely that the primary case of swine influenza would be diagnosed either in pigs or other susceptible species. Swine influenza virus infection in pigs results in high morbidity but, mortality is very low and most pigs recover within a short period. Acute respiratory disease is likely to be investigated in small commercial piggeries, however, it may be some time before there was a diagnosis of swine influenza with endemic respiratory disease needing to be ruled out first. Given this, it was considered unlikely that the indirect impact of new or modified control programs would be discernible at any level, and the rating assigned to this criterion was therefore 'A'.

Domestic trade or industry effects

As discussed above, it was considered unlikely that swine influenza would be diagnosed in a single pig herd or a small group of other susceptible species. On this basis, the indirect impact of swine influenza on domestic trade and industry was considered unlikely to be discernible at any level, and thus the rating assigned to this criterion was 'A'.

International trade effects

As the disease is unlikely to be diagnosed if contained within the primary exposure group, it was considered that the indirect effects of swine influenza on international trade was unlikely to be discernible at any level, and a rating of 'A' was therefore assigned to this criterion.

Indirect impact on the environment

In this scenario, it was considered unlikely that swine influenza virus would have a indirect impact on the environment (such as an effect on biodiversity) that would be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as a reduction in rural and regional viability, and thus a rating of 'A' was assigned to this criterion.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, swine influenza would have established in a broader population of feral pigs. It is unlikely that the swine influenza would be diagnosed in feral pigs. Containment of the disease in feral pigs would result from the low probability of contact between infected and susceptible animals, rather than by human intervention.

The direct impact of swine influenza

Animal life or health

In this scenario, swine influenza spreads to a more general population of feral pigs. However, as with the first scenario, it was considered unlikely that the disease would be diagnosed in this population. Pigs infected with swine influenza virus only show clinical signs for about 1 week and may not be observed during this period by hunters. Pigs that are sick are more likely to remain in one location. Feral pigs harvested for meat, even if infected with swine influenza virus, are unlikely to show marked gross pathological changes.

Given this, it was considered likely that the direct impact of swine influenza on animal life, health or welfare was unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

Environment

Swine influenza virus may infect native Australian waterfowl, however, clinical signs are likely to be absent. Given this, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of swine influenza

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

The indirect impact of swine influenza on new or modified control programs for this scenario was considered similar to that described above for the first scenario, resulting in a rating of 'A' for this criterion.

Domestic trade or industry effects

As discussed above, it was considered unlikely that swine influenza would be diagnosed if scenario 2 occurred. On this basis, the indirect impact of swine influenza on domestic trade and industry was considered unlikely to be discernible at any level, and a rating of 'A' was assigned to this criterion.

International trade effects

As discussed above, the indirect effect of swine influenza on international trade was unlikely to be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on the environment

In this scenario, it was considered unlikely that swine influenza virus would have a indirect impact on the environment (such as an effect on biodiversity) that would be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as a reduction in rural and regional viability, and thus a rating of 'A' was assigned to this criterion.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries. In the case of other susceptible species as the direct exposure group — secondary spread to other susceptible species

Under this scenario, when the direct exposure group was a feral pig herd, backyard pig enterprise or a small commercial piggery, swine influenza would have established in a local population of backyard piggeries or small commercial piggeries and may have spread to other susceptible species and humans. The disease would be contained through the diagnosis of disease in pigs and/or illness in humans, and the mounting of a control program involving quarantine and movement controls.

When ducks and waterfowl were the direct exposure group, swine influenza would have spread to more general population of these species but due to the often subclinical nature of infection the disease would not be identified.

The direct impact of swine influenza

Animal life or health

This criterion was not rated identically for each exposure group.

The third scenario, where the primary exposure group is pigs, is characterised by spread of swine influenza virus to a local population of domestic pigs in backyard enterprises or small commercial piggeries, but containment within this population, and spread to other susceptible species.

Due to the high morbidity within affected pig herds, it was considered that the impact of swine influenza virus on animal life or health would be unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion when the primary exposure group was feral pigs, backyard pigs or pigs in small commercial piggeries.

Turkeys are the only known reported susceptible species, other than humans, likely to show clinical signs of infection with swine influenza virus. These signs may include those of respiratory disease and a decrease in egg production. Given this, it was considered that the direct effects on animal health would be unlikely to be discernible except at the local level. Thus, when the primary exposure group was other susceptible species, a rating of 'B' was assigned to this criterion.

Environment

Swine influenza virus may infect native Australian waterfowl, however, clinical signs are likely to be absent. Given this, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of swine influenza

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

This criterion was not rated identically for each exposure group.

The spread of infection to other local pig herds is likely to lead to the diagnosis of swine influenza. Swine influenza is listed as Category 4 'production loss' disease under the Emergency Animal Disease Response Agreement, thus the costs of any response program are to be shared between governments (20%) and industry (80%).

If the disease is diagnosed before spread occurs to large commercial piggeries, it is likely that infected premises and those in the restricted area would be subject to quarantine and movement controls. It is possible that pigs could still be moved directly to slaughter, however, there would be restrictions on the movement of breeding pigs. Surveillance of the domestic and feral pig population would need to be undertaken to determine the extent of the outbreak. If surveillance indicates that the disease is widespread in feral pigs, then a control program using vaccination may be considered as an option.

Due to the occupational health and safety issues associated with influenza virus an education program would likely be implemented for piggery workers. There would need to be close liaison with the Department of Health and Ageing regarding the public health issues.

On this basis, the indirect effect of new or modified control programs when the direct exposure group was feral pigs, backyard pigs or pigs from small commercial piggeries was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

In the case of other susceptible species being the direct exposure group with secondary spread to other susceptible species but not to the pig population, it is unlikely that the disease would be diagnosed. Hence, it was considered that the indirect effect of new or modified control programs was unlikely to be discernible at any level resulting in a rating of 'A'.

Domestic trade or industry effects

This criterion was not rated identically for each exposure group.

Although there are likely to be some movement restrictions at least initially, these are unlikely to significantly affect backyard or small commercial piggeries. Movement restrictions could affect larger commercial piggeries if located in the restricted area, although animals may still be able to be sent to slaughter.

With the detection of an exotic disease in Australia, one with public health implications, consumers may initially decrease pork consumption. A publicity campaign may need to be undertaken to reassure the public that there are no health concerns with the consumption of pig meat.

After considering these issues, the indirect impact of swine influenza on domestic trade and industry, when the direct exposure group involved pigs, was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

As discussed above, in the case of other susceptible species being the primary exposure group swine influenza is unlikely to be diagnosed. Hence the indirect impact on domestic trade and industry was considered unlikely to be discernible at any level, and a rating of 'A' was assigned to this criterion.

International trade effects

This criterion was not rated identically for each exposure group.

Swine influenza is widespread elsewhere in the world and does not appear to inhibit trade in meat. Nonetheless, an outbreak of any disease that may have public health implications could result in a temporary suspension in trade of meat to sensitive markets such as Japan and Singapore, particularly if any human cases of swine influenza also occurred in Australia. Trade should be able to be renegotiated as Japan has swine influenza and imports pig meat from other countries with the disease. Singapore does not have a pig industry and meat is not considered a public health concern for swine influenza.

There may be temporary disruption to the export of live pigs. Pigs could be tested to demonstrate that they had not been exposed to the swine influenza virus prior to being exported. Alternatively if vaccination has been used for control, pigs may need to undergo a period of quarantine prior to export. On this basis, the indirect effect of swine influenza on international trade when the primary exposure group was feral pigs, backyard pigs or pig in small commercial piggeries was considered unlikely to be discernible except at the local level. Overall, this resulted in a rating of 'B' for this criterion.

Whereas if the primary exposure group is other susceptible species, the indirect effect on international trade is unlikely to be discernible at any level. This resulted in a rating of 'A' for this criterion.

Indirect impact on the environment

In this scenario, it was considered unlikely that swine influenza virus would have a indirect impact on the environment (such as an effect on biodiversity) that would be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as a reduction in rural and regional viability, and thus a rating of 'A' was assigned to this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, swine influenza would have established in a broader population of commercial piggeries (including medium-large piggeries), other susceptible species. The disease would be identified. Humans may also have been infected. If the disease was not widespread in the Australian pig population, a control program would likely be implemented, alternatively, if widespread, control may be left to individual producers.

The direct impact of swine influenza

Animal life or health

In naïve herds, infection with swine influenza virus can result in acute respiratory disease, in pigs of all ages. If the disease became endemic, it is likely that further outbreaks would not appear as explosive, with maternal immunity protecting piglets, and waning maternal immunity minimising clinical signs in weaner pigs. Nevertheless, in herds where the disease is endemic, chronic respiratory problems in grower and finisher pigs and decreased feed efficiency are seen. Even subclinical infection can adversely affect average daily gain (Regula, et al., 2000).

In turkeys, infection with swine influenza virus can result in respiratory disease and decreased egg production.

Given this, the direct effect on animal health was considered unlikely to be discernible at the national level, but would have a minor impact at the State level. This resulted in a rating of 'D' for this criterion.

Environment

Swine influenza virus may infect native Australian waterfowl, however, clinical signs are likely to be absent. Given this, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of swine influenza

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

Initial reaction to an outbreak of swine influenza is likely to result in the Emergency Animal Disease Response Agreement being invoked. The control, monitoring and surveillance programs initiated in this scenario would be similar to those described in scenario 3, although more widespread. If surveillance demonstrated that the disease was widespread in Australia, eradication may not be possible. In such a case, the Agreement is likely to be suspended, with producers within the industry taking responsibility for the costs of disease management in their herds. Management could include vaccination and biosecurity measures.

Taking these issues into consideration, the indirect impact of new or modified control programs was considered unlikely to be discernible at the national level, but would have a minor impact at the State level. This resulted in a rating of 'D' for this criterion.

Domestic trade or industry effects

The spread of swine influenza to the commercial pig population may initially result in movement restrictions, although it is likely that pigs could still move to slaughter. Even if movement to slaughter is permitted there may be some disruption to domestic trade in pig meat. It is known that in herds infected with swine influenza virus, it takes longer for pigs to reach slaughter weight (Kay, et al., 1994). There would be costs to affected producers.

If the disease became endemic, costs would be ongoing and may include vaccination, veterinary treatments and additional feed costs. The national yearly financial losses incurred as a result of widespread swine influenza in the United Kingdom through on-farm mortality, rejected carcasses at abattoirs, delay in reaching slaughter weight, and reduced reproductive efficiency in sows has been estimated, using a model simulation, as ranging from £7,117,000 to £16,548,000[62]. In this simulation vaccination was not used for control.

As discussed above with the detection of swine influenza virus consumers may initially decrease pork consumption. In the unlikely event that any human cases of swine influenza occurred as a result of the outbreak in domestic pigs, pork consumption could remain reduced for quite some time. A publicity campaign may need to be undertaken to reassure the public that there are no health concerns with the consumption of pig meat.

If a decrease in pig meat consumption occurred, this would not only affect producers but also those associated industries such as processors and transport companies.

Given these factors, it was considered likely that the indirect impact on domestic trade and industry would be unlikely to be discernible at the national level, but would have a minor impact at the State level. This resulted in a rating of 'D' for this criterion.

International trade effects

The indirect effect on international trade was considered to be the same as described for scenario 3. Even with a more generalised outbreak of swine influenza within the domestic pig population, the export of pig meat should only be temporarily disrupted. Trade should be able to be renegotiated. There is likely to be some initial disruption to trade in breeding pigs. As

[62] http://www.rdg.ac.uk/livestockdisea/pigs/swinflu.htm

such, the indirect effect on international trade was considered unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

Indirect impact on the environment

Again, it was considered unlikely that swine influenza virus would have a indirect impact on the environment (such as an effect on biodiversity) that would be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on communities

The rural economies of major pig producing areas could be affected if swine influenza became widespread. The viability of some producers could be affected and some producers may go out of business. This would impact on the local economy, on businesses directly supplying the pig industry and those supplying the local community.

In light of this information, the indirect impact of swine influenza on rural economies was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

The overall impact of swine influenza

When the direct and indirect impacts of swine influenza were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences negligible

Scenario 3: Consequences very low (feral pigs, backyard pigs, small commercial piggeries), negligible (other susceptible species)

Scenario 4: Consequences low

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 70, Table 71, Table 72 and Table 73. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs, pigs in small commercial piggeries and other susceptible species to infected pig meat scraps were considered 'very low', 'very low', 'low' and 'negligible', respectively.

Table 70 Swine influenza: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Moderate	Negligible	Negligible
Scenario 3	Very low	Very low	Negligible
Scenario 4	Low	Low	Very low
		Overall likely consequences	**Very low**

Table 71 Swine influenza: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Very Low	Negligible	Negligible
Scenario 3	Moderate	Very low	Very low
Scenario 4	Low	Low	Very low
		Overall likely consequences	**Very low**

Table 72 Swine influenza: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Low	Very low	Negligible
Scenario 4	Moderate	Low	Low
		Overall likely consequences	**Low**

Table 73 Swine influenza: summary of the consequences of exposure of other susceptible species

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Moderate	Negligible	Negligible
Scenario 4	Very low	Low	Negligible
		Overall likely consequences	**Negligible**

Human life or health

Separate to the above is consideration of the consequences to human life or health. Swine influenza is regarded as a zoonosis. In 1976, the zoonotic potential of swine influenza was confirmed when the caretaker of a pig farm became ill several days after the pigs had shown signs of influenza. Characterisation of the viruses from the man and pigs were identical. Prior to this case several hundred military recruits had been infected with an influenza virus closely related to swine influenza. There have been several other isolated reports of humans infected with swine influenza virus, although generally there has been minimal spread from human to human. Some of these infections have resulted in acute, fatal respiratory disease in humans (Easterday & Van Reeth, 1999).

It would appear that although disease is rare in humans, exposure to swine influenza virus occurs more frequently. In Wisconsin, United States of America, 17 of 74 (23%) people having regular contact with pigs were seropositive to swine influenza virus while only one of 114 urban residents was seropositive (Olsen, et al., 2002).

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with swine influenza virus.

Table 74 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly less than Australia's ALOP (very low), risk management would not be required for swine influenza virus.

Table 74 Swine influenza: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Extremely low	Extremely low	Very low	Negligible
Backyard pigs	Extremely low	Negligible	Very low	Negligible
Small commercial piggeries	Extremely low	Extremely low	Low	Negligible
Other susceptible species	Extremely low	Extremely low	Negligible	Negligible
			Overall annual risk	**Negligible**

The Department of Health and Ageing has advised Biosecurity Australia that biosecurity measures for swine influenza virus would not be required to manage the risk to human life or health associated with the importation of pig meat. It should be noted that the annual likelihood of entry and exposure of swine influenza via pig meat is extremely low or negligible. The virus could enter Australia via wild waterfowl or humans.

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Brucella suis

Technical information

Background

Porcine brucellosis, an infectious bacterial disease caused by *Brucella suis*, is characterised by sterility and abortion in sows, mortality in piglets and orchitis in boars.

The disease occurs in most countries where wild or domestic swine exist including the United States of America and most countries in continental Europe.

Brucella suis biovar[63] 1 is present in Australia (Crichton & Medveczky, 1987). Isolates collected from native rodents in northern Queensland in the 1960s and classified as *B. suis* biovar 3 (Cook, et al., 1966) were later reclassified as *B. abortus* (Crichton & Medveczky, 1987). In Australia, porcine brucellosis is a notifiable disease. It is restricted to feral pigs in central and south west Queensland. The organism has also been found in domestic cattle in Queensland but has not been cultured from domestic pigs for many years (Rogers, et al., 1989). Testing of 16,000 breeders from piggeries throughout Queensland between 1996 and 2001 found no evidence of *B. suis* infection in commercial pigs (Animal Health Australia, 2002). Commercial piggeries in other parts of Australia are free of this disease. Movement restrictions are applied to pigs from infected areas in Australia. These require that breeding pigs be tested or originate from accredited-free herds.

Agent taxonomy

Brucella suis, a member of the family Brucellaceae, order Rhizobiales, class Rhodospirilli, kingdom Proteobacteria, are aerobic to microaerophilic, gram negative, non-motile, non-spore-forming coccobacillary rods. There are 5 biotypes identified; biovars 1 and 3 occur primarily in pigs, biovar 2 primarily in pigs and European hares, biovar 4 occurs primarily in reindeer and caribou, and biovar 5 in rodents of the Subfamily Murinae, causing murine brucellosis.

Agent properties

Brucellae can survive in carcasses and organs for up to 135 days, in soil for 125 days and in blood at 4°C for 180 days (Health Canada, 2001). *Brucella suis* survived in organic matter at freezing or near freezing temperatures for over two years (MacMillan, 1999). Although possibly not directly applicable to meat, *B. suis* survived for 16 days at approximately 30°C and a pH of 4 to 5 when inoculated into buffaloes' milk yoghurt (Ghoniem, 1972). In contrast, *Brucella suis* survived more than 50 days at 20°C to 25°C when added to buffaloes' milk inoculated with yoghurt starter (Nour, et al., 1975). *Brucella suis* is readily destroyed by pasteurisation, when exposed to direct sunlight for 2 to 4 hours, and by commonly used disinfectants.

The optimum pH for growth of *B. suis* is between 6.6 and 7.4. Unless the bacterium is first adapted to an acidic environment, it is inactivated at pH 5.5 (Kulakov, et al., 1999).

[63] Some texts refer to biotype or biogroup rather than biovar.

Host range

Swine are the most common hosts for *B. suis* biovars 1 and 3, both of which have a worldwide distribution. Biovar 2, commonly found in Europe, occurs in hares as well as pigs. Pigs infected with biovars 1, 2, and 3 can serve as a source of infection for other domestic animal species such as horses, cattle, and dogs. Biovar 4 occurs in reindeer and caribou, particularly in Siberia, Alaska and Canada, and is not pathogenic for swine. Biovar 5 causes murine brucellosis only.

Brucellosis, including porcine brucellosis, is a zoonosis and has public health significance.

Epidemiology

Within a piggery, ingestion and coitus commonly spread disease. Pigs can become infected by eating food or pastures, or drinking fluids, contaminated with discharges (urine, semen, foetal membranes, uterine discharges) from infected pigs. The introduction of infected pigs or the use of contaminated semen is the most important means of spread. Most infected sows cease shedding the bacteria within 30 days of infection, however, some can continue to shed for as long as 30 months. Boars on the other hand tend to become persistently infected and can disseminate large numbers of bacteria in their semen, transmitting the disease to the female by natural service or through artificial insemination. Feral pigs and hares (biovar 2) are also capable of transmitting the disease to pigs in piggeries.

Prevalence of infection is usually much higher in sexually mature adults than in young pigs, and spread of the disease through a herd can be quite rapid because of the intensive husbandry conditions under which pigs are kept (Badiola, 1985; van der Leek, et al., 1993; Rossi, et al., 2002).

Although not directly relevant to porcine brucellosis, human cases of brucellosis have occurred as a result of people eating raw bone marrow or raw meat from reindeer or caribou infected with biovar 4 (Acha, et al., 1987). Dogs have also been infected with biovar 4 by eating contaminated reindeer meat (Neiland, 1975). Most human cases of brucellosis due to *B. suis* are due to occupational contact or, if cattle are infected, the consumption of contaminated unpasteurised milk and dairy products (MacMillan, 1999).

Latin America and South East Asia are considered the regions where porcine brucellosis is most prevalent. There are little data on the between herd prevalence of *B. suis* infection for countries where the disease is endemic. In South East Asia, most figures are dated but overall country prevalence has been reported as 15% in Indonesia, 6.6% in the Philippines, and 7% in Thailand (Blajan & Melendez, 1984; Badiola, 1985; Priadi, et al., 1985). Pig farming in India is restricted to certain parts and seroprevalence in these parts has reportedly ranged from 3.2% to 16.7% (Saini, et al., 1994; Renukaradhya, et al., 2002).

In South America, surveys suggest overall country prevalence to be 14.2% to 25% in Argentina, and 4 to 35% (depending on disease control practices) in Venezuela (Lord, et al., 1997; Samartino, 2002). In Brazil, a national survey conducted in 1981 on 66,770 porcine serum samples showed a seroprevalence of 2.19%. Commercial breeding herds in Brazil have since been subject to official control programs and a more recent survey reported by the Ministry of Agriculture showed that national seroprevalence had dropped to 0.34% (as quoted in (Poester, et al., 2002).

In the United States of America, porcine brucellosis was once a major disease of domestic pigs but changes in pig management and regulatory activity to control the disease in domestic pigs

have reduced porcine brucellosis to very low levels. In 1980, the control program had reduced the prevalence to 0.05% (Blajan & Melendez, 1984). As of 31 May, 2003, there were no brucellosis affected swine herds in the United States of America (USDA:APHIS, 2003).

Serological surveys in Venezuela show the within herd prevalence of *B. suis* to be highly variable among farms, ranging from 5% up to 89% on one farm (Lord, et al., 1997). A serological survey conducted in India reported that the within herd prevalence of brucellosis ranged from 11.1% to 58.3% while the prevalence of infection in market pigs was reported to be 3.2% (Saini, et al., 1994). Despite control measures, seroprevalence within infected herds in the United States of America reached levels of up to 66% (Cornell, et al., 1989). *Brucella suis* was isolated at slaughter from 21% of 221 pigs from 39 known infected herds (Ferris, et al., 1995).

Porcine brucellosis has been reported in feral pigs in Australia and elsewhere. In the United States of America, *B. suis* is present in feral pigs in Hawaii and the southern States. A serological survey in feral pigs in Florida found an overall prevalence of 23.4% in which 33.3% (6/18) of sites sampled contained seropositive pigs (Becker, et al., 1978; van der Leek, et al., 1993). In South Carolina approximately 44% of feral pigs at three locations tested positive for *B. suis* antibodies. This prevalence was higher than that found in previous years of 28% in 1976 and 18% in 1992 (Gresham, et al., 2002). In France, approximately 20% of wild boars tested in one area were seropositive (Rossi, et al., 2002).

Clinical signs

Clinical signs of *B. suis* infection in pigs vary considerably in different herds and may be manifested as abortions, infertility, orchitis, posterior paralysis and lameness. Early abortions, with very little or no vaginal discharge, can occur and can easily be overlooked under field conditions as pigs often eat aborted foetuses. Infection in sucklings or weaner pigs usually appears as spondylitis associated with posterior paralysis. Infected pigs do not usually show persisting or undulating pyrexia. Clinical signs may be transient and death is a rare occurrence (MacMillan, 1999). Nonetheless mortality during the first month of life can sometimes occur. Most losses result from stillbirths and the death of weak piglets within the first few hours of birth.

In dogs infected with *B. suis* the clinical signs may be mild or non-existent, infections usually being sporadic and self-limiting. *Brucella suis* can also cause an acute infection in dogs, and pregnant bitches may abort. Brucellosis in horses can result in lethargy and generalised stiffness, but has a tendency for localisation in bursae, tendons, muscle and joints. Fistulous withers in horses may result from infection with *B. suis* (Blunt, et al. 1977). In cattle, *B. suis* infection does not appear to cause clinical disease, although the bacteria can localise in the mammary gland and be shed in the milk (Ewalt, et al., 1997).

Pathogenesis

The pathogenesis of disease due to *B. suis* biovars 1, 2, and 3 is similar. After exposure, there is a period where the organisms localise in the lymph nodes near the point of entry. In general, the onset of bacteraemia follows 1 to 7 weeks after infection. Bacteraemia persists an average of about 5 weeks and is generally continuous during that time. Nonetheless, bacteraemia in individual pigs has been observed to be as short as 1 week and intermittently as long as 34 months (MacMillan, 1999). Bacteria may be found in all body tissues, but, in chronically

infected pigs, tend to persist in the uterus, cervical lymph nodes, bone marrow and joints and, in boars, the testes.

Pathology

Gross pathologic changes produced by *B. suis* infection in pigs are quite variable. Chronic metritis manifested by nodular inflammatory thickening and abscessation of the uterine wall is characteristic. Lymph nodes may sometimes show diffuse granulomatous inflammation. Pronounced lymphadenopathy and splenic enlargement occur in some cases (Gay, et al. 2000). Abscesses may be found in affected organs, such as the testicles and prostate glands of boars, and the uterine mucosa of sows. Focal abscesses may be found infrequently in kidneys, spleen, brain, ovaries, adrenal glands, lungs and other tissues of infected pigs (MacMillan, 1999). Nodular splenitis, in the absence of other lesions, justifies a presumptive diagnosis of brucellosis in pigs, although it may occur infrequently.

Immunology

Bacteraemia generally precedes detectable antibody levels by as much as 6 to 8 weeks (MacMillan, 1999). Immune response resembles those against other intracellular bacteria and are both humoral (antibody-mediated) and cell-mediated. The latter is the principal component of defence against infection. It should be noted that boars are often permanently infected, despite the presence of antibodies.

An oral *B. suis* biovar 2 vaccine has been used extensively for immunising pigs in southern China and can protect pigs infected by most routes but not by natural mating (Xie, as quoted in (Corbel & MacMillan, 1998). Elsewhere, research suggests that *B. melitensis* Rev 1 vaccine may be effective in protecting pigs against *B. suis* infection (Cedro, et al., 1977). Similarly, *B. abortus* RB51 vaccine, a live rough vaccine, may be effective in pigs, preventing abortion due to infection with *B. suis* (Lord, et al., 1998).

Transmission via meat

Literature often states that the alimentary tract is an important and probably the most common route of infection in pigs (MacMillan, 1999; Gay, et al. 2000); however, there is a paucity of data to confirm that eating meat from infected pigs can cause disease in pigs. It is known that *B. suis* is transmitted via the oral route to pigs. Of two boars given *B. suis* cultures per os under experimental conditions, one contracted subclinical brucellosis and the other developed temporary swelling of the left testis. Also, four of 13 newborn pigs given *B. suis* cultures per os developed subclinical lesions from which the bacteria could be cultured (Thomsen, 1934).

It has been reported that pigs or other susceptible species including humans can become infected through the ingestion of carcasses contaminated with *B. suis* (Thomsen, 1934; Brazeau, et al., 1973; Neiland, 1975; Acha, et al. 1987). The bacteria can be isolated from infected carcasses by culture of lymph nodes, bone marrow and joint exudates. Experimentally the ID_{50} (infectious dose, causing infection in 50% of animals) of *B. suis* biovar 1 has been estimated to be around 500 colony forming units (CFU) in Indonesian pigs, although the route of infection was unable to be determined from the report (Sudibyo, 1998). Beagle dogs have readily been infected by $1.3x10^8$ CFU of *Brucella suis* biovar 4 administered on canned dog food (Neiland & Miller, 1981), however, biovar 4 may behave quite differently to biovars 1, 2 and 3. There is one report of *B. suis* infection in two dogs where the source of infection was possibly raw meat being fed to the dogs (Hellmann & Sprenger, 1978).

Brucellae have been shown to survive in a refrigerated guinea pig carcass for up to 44 days (Kuzdas and Morse, 1954, as summarised in a table by (Timoney, et al. 1988)) and for more than 15 days in refrigerated experimentally contaminated beef (Mitscherlich, et al. 1984). A data sheet produced by Health Canada states that brucellae may survive for up to 135 days in carcasses (Health Canada, 2001). Annex 6 of the US Department of Health and Human Services Food Code 2001 states that "Marginal refrigeration (during storage or distribution of food products in reduced oxygen packaging) … may still allow … *Brucella* spp to survive for long periods of time." Marginal refrigeration means temperatures of 5°C to 12°C.

Brucellae can also survive in salted meat for 65 days at 20°C (Prost, 1957, as summarised in a table by (Timoney, et al. 1988)). *Brucella suis* has survived in spleen and lymph node tissue held in meat-curing brine at -44°C for 40 days. It has been reported that brucellae are resistant to pickling and smoke curing and thus there is the possibility that meat products so prepared could cause human infection (Acha, et al. 1987) but this mode of transmission has never been verified. Thorough cooking of meat inactivates the bacteria (Corbel, 1997).

Release assessment

R1 — the likelihood that a source herd is infected

Prevalence is recognised as being higher in South East Asia and parts of South America, where there is very little or no regulatory activity to control or eradicate porcine brucellosis, than in Europe or North America. Although little data were found for between herd prevalence, the country prevalence has been reported as approximately 7% in the Philippines and Thailand to a high of 35% in Venezuela. Given this, the likelihood that slaughter-age pigs have been selected from an infected herd in a country where *B. suis* is endemic was considered to be 'low'.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

A serological survey in Venezuela showed the prevalence of *B. suis* infection on farms ranged from 5% up to 89% on one farm. In the United States of America, 66% of boars and breeding age sows were seropositive in one herd. Within herd prevalence has been reported to range from 11% to 58% in India, however, in market age pigs prevalence of infection was reported as 3.2%. In the late 1960s, in the United States of America, 4.6% of 2275 slaughter-age pigs were seropositive (White, et al., 1974). The figures for slaughter-age pigs reflect the fact that prevalence of infection is usually higher in sexually mature animals than in young pigs. In another study conducted in the United States of America, *B. suis* were isolated from 21% of pigs at slaughter that came from herds known to be seropositive. In view of these reports, it was considered that the likelihood of selecting an infected pig in an infected herd was 'low'.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

Clinical signs of brucellosis in finishing pigs are likely to be mild and non-specific. A few pigs may show some degree of lameness or posterior paralysis and severe cases may be rejected

from slaughter. Gross pathological changes may not be evident, especially in finisher pigs. However, older pigs may show multiple abscesses requiring condemnation of whole carcasses or affected parts of the carcass, such as liver, testes. On this basis, the sensitivity of the antemortem, slaughter and processing requirements in detecting and removing pigs infected with *B. suis* was considered to be 'very low'.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with *B. suis* and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a 'one minus the background rejection rate'. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered 'extremely low'. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

Brucella suis can be isolated from infected pigs by culture of lymph nodes, bone marrow and joint exudates. These tissues when attached to muscle are included in the definition of pig meat in this IRA. If an animal is bacteraemia at slaughter, muscle could also be infected. Bacteraemia persists on average for about 5 weeks but some individual cases may be intermittently bacteraemic for 34 months. In addition it is feasible that contamination of meat may occur within the abattoir, such as during deboning. This suggested that the likelihood that *B. suis* would be present in meat harvested from an infected pig was 'moderate'.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

As stated earlier, brucella organisms can survive in carcasses for up to 135 days. *Brucella suis* grows best between pH 6.6 and 7.4. For the purposes of this IRA, meat is not assumed to reach a pH lower than 6.2. Thus, it was considered that there was a 'high' likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

Brucellae are resistant to freezing and so can survive for prolonged periods. At 4°C the bacteria can survive for 180 days in blood. Given this, it was considered that there was a 'high' likelihood that meat infected at the completion of carcass maturation would remain infected during transport and storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a 'very low' likelihood that imported pig meat derived from a carcass would be infected with *B. suis*.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

An ID_{50} for *B. suis* has been estimated to be around 500 CFU in pigs, although the route of infection is unknown. There is no information on the number of CFU's found in infected tissues, such as lymph nodes. The Panel is unaware of documented evidence of pigs becoming infected via consumption of pig meat. Pigs can become infected through the ingestion of feed or water contaminated with infected discharges. On balance, the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

Brucella suis is readily destroyed when exposed to sunlight for several hours. For meat scraps covered by other refuse brucellae may be partially protected. In milk products the organism has survived for 16 days at 30°C and more than 50 days at 20°C to 25°C. In a moist environment it would appear that the virus is relatively stable but if exposed to sunlight viability decreases.

This information led the Panel to consider that the likelihood that *B. suis* would survive within meat scraps discarded in refuse for the period of time required for feral pigs to locate and subsequently scavenge the material was 'moderate'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = High
- Rural regions = Low
- Large towns = Extremely low

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be ‘high’.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a ‘very low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was ‘low’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a ‘high’ likelihood that *B. suis* would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;

- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'very low'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a 'high' likelihood that *B. suis* would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'high'.

Exposure assessment for other susceptible species

Dogs are carnivorous species that are susceptible to infection with *B. suis*. Wild dogs and dingoes may gain access to discarded pig meat scraps and domestic dogs may be fed pig meat scraps as part of their diet. The Panel was unable to find any reference to dingoes being infected with *B. suis* in Queensland. Nonetheless, it is unknown if dingoes have been tested for this agent.

Experimentally it has been shown that dogs can be infected orally with a large dose of *B. suis* biovar 4 ($1.3x10^8$ CFU). There is one report suggesting that natural infection of dogs with *B suis* may have occurred as a result of being fed raw contaminated meat (Hellmann & Sprenger, 1978). In other cases the infected dogs have been present on farms with the disease. Nonetheless it would appear from the literature that infection of dogs with *B. suis* is a sporadic event. Given this, it was considered that the annual likelihood of entry and exposure for other susceptible species was 'low'.

Consequence Assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the four exposure groups - that is, feral pigs, pigs in backyard enterprises pigs in small commercial piggeries and other susceptible species. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;

3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Brucella suis is commonly spread via ingestion and coitus. Transmission occurs principally as a result of eating food or pastures contaminated with discharges from infected pigs. Infected sows may abort or give birth to stillborn or weak and stunted piglets, many of which die a few days later. Other pigs in the group may eat the infected aborted foetuses and in turn become infected. Venereal spread is another important means of spread, as boars often become persistently infected, shedding the bacteria in their semen.

Feral pigs tend to maintain small discrete groups, having quite definite home ranges, although some mixing occurs in times of low feed or water availability and some populations are contiguous. Several cases of feral pigs establishing in new areas are because of deliberate introductions by recreational hunters (McGaw, et al. 1998). Deliberate movement of feral pigs, and contamination of common areas such as watering holes may assist in dissemination of the disease into the feral pig population, together with persistently infected boars. Nevertheless, it should be noted that *B. suis* in feral pigs in Australia has not become widespread throughout the feral pig population. The disease is confined to a few regions in central and south west Queensland. A 1985-87 survey in central Queensland showed 114 (26%) of 438 feral pigs sampled were seropositive for brucellosis. *Brucella suis* biovar 1 was isolated from 37% of 134 of these pigs (Cook & Kingston, 1988).

While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs; however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves. Nonetheless, in Queensland, where *B. suis* is present in feral pigs, spread to domestic pigs has not occurred for many years.

Brucella suis may also spread from feral pigs to humans or to domestic livestock such as cattle and horses as has been documented in Queensland (Cook & Kingston, 1988). Of 27 brucellosis cases reported in people in Australia in 2000, 26 were from central south west Queensland, where there is a relatively high frequency of infections among men who hunt and slaughter feral pigs (Cook & Noble, 1984; Robson, et al., 1993).

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: moderate

Scenario 3: very low

Scenario 4: extremely low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the ‘no outbreak’ scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

As discussed above, it is feasible that backyard pigs kept in rural areas may come in close contact with nocturnally foraging feral pigs and that transmission may result. It is also feasible that some mixing between pigs from an infected backyard herd and other domestic pigs may occur. For example, spread may occur when several local farms share an infected boar or where pigs are moved between backyard holdings for growing out or fattening.

Porcine brucellosis may not be diagnosed in backyard herds, particularly where the herd only consists of fattening pigs for private consumption, as clinical signs may be absent or mild. In small breeding herds disease may be investigated after a period of time due to reduced farrowing rates, dying piglets and failure to breed, although veterinary attention is not always sought. Where there are few movements of pigs from infected herds, with pigs consumed on the farm, the disease may die out without spreading. However, pig farmers are at risk of infection when slaughtering infected pigs. It may be that the disease is diagnosed initially in a human rather than pigs in a backyard holding.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: high

Scenario 2: low

Scenario 3: low

Scenario 4: very low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the ‘no outbreak’ scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;

3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

Brucella suis infection results in production losses through reduced farrowing rates, dying piglets and failure to breed. Such events in a small commercial piggery are more likely to be reported to a veterinarian. However, disease spread as a result of sale of infected breeders and/or boars at local and regional saleyards may occur. In addition, there is the potential for abattoir workers to become infected when processing infected pigs.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: very low

Scenario 3: moderate

Scenario 4: very low

Other susceptible species

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of other susceptible species:

1. Containment of the disease within the sub-population of directly exposed animals - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of domestic pigs - containment within a local population of backyard enterprises or small commercial piggeries - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs - exposure of medium-large commercial piggeries and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Dogs have been infected with *B. suis*. There is one report suggesting dogs may become infected as a result of eating infected meat, although it would appear that this is a rare event. The Panel is unaware of any reports of disease spreading between dogs or from dogs to other species. *Brucella suis* can also infect horses and cattle but is generally not pathogenic in these species. Horses and cattle usually become infected as a result of contact with pigs.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: high

Scenario 2: very low

Scenario 3: extremely low

Scenario 4: extremely low

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this scenario, *B. suis* would have established in the directly exposed animal, or group of animals, but would not have spread to other animals or to humans. This 'no outbreak' scenario would have resulted from low probability of contact between infected and susceptible animals or humans, rather than from human intervention. Indeed, because the clinical signs of disease can be mild, it was assumed that it would not have been identified.

The direct impact of B. suis infection

Animal life or health

Brucella suis infection in pigs may result in abortions, infertility, orchitis, posterior paralysis and lameness. In other cases clinical signs can be very mild. Horses may show bursitis, stiffness and lethargy. Pregnant bitches may abort. Given this, it was considered that the direct impact on animal health was unlikely to be discernible except at the local level. Thus, a rating of 'B' was given to this criterion.

Environment

Brucella suis is unlikely to affect native Australian species, with the possible exception of dingoes, although this does not seem to be the case in Queensland. Given this, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of B. suis infection

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this scenario, where the disease is contained within the initial exposure group with no secondary spread, it was considered unlikely that the primary case of *B. suis* would be diagnosed either in pigs or other susceptible species. Given this, it was considered unlikely that the indirect impact of new or modified control programs would be discernible at any level, and the rating assigned to this criterion was therefore 'A'.

Domestic trade or industry effects

As discussed above, it was considered unlikely that *B. suis* would be diagnosed in a single pig herd or another susceptible species. On this basis, the indirect impact on domestic trade and industry was considered unlikely to be discernible at any level, and thus the rating assigned to this criterion was 'A'.

International trade effects

As the disease is unlikely to be diagnosed if contained within the direct exposure group, it was considered that the indirect effects on international trade were unlikely to be discernible at any level, and a rating of 'A' was therefore assigned to this criterion.

Indirect impact on the environment

The indirect impact on the environment, such as reduced biodiversity, was considered unlikely to be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on communities

The indirect impact on local communities, such as reduced rural and regional economic viability, was considered unlikely to be discernible at any level, hence this criterion was rated 'A'.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, *B. suis* would have established in a broader population of feral pigs, and may have spread to feral pig hunters, or abattoir workers slaughtering infected animals. The disease would be contained through the diagnosis of illness in humans and the mounting of a control program.

The direct impact of B. suis infection

Animal life or health

In this scenario, there is secondary spread to a more general population of feral pigs. Currently in central and south west Queensland, several herds of feral pigs are infected with *B. suis*. However, its impact on animal life, health and welfare in these regions has not been discernible. Thus, the direct impact on animal health is not likely to differ from scenario 1 as discussed above, and a rating of 'B' was assigned to this criterion.

Environment

Brucella suis is unlikely to affect native Australian species, with the possible exception of dingoes, although this does not seem to be the case in Queensland. Given this, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of Brucella suis

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this outbreak scenario, spread to humans as well as feral pigs may occur. In light of this, it was considered that *B. suis* may be diagnosed due to human illness. Following diagnosis, some initial surveillance of the pig population may be undertaken, particularly if the person had no known contact with feral pigs in Queensland or if infected with a different biovar. If pigs in a domestic pig herd were positive for *B. suis*, they may be slaughtered. On the other hand, if the disease agent was restricted to feral pigs there may be no further surveillance. In Queensland, in areas where *B. suis* is endemic, feral pigs are not subject to regular surveillance and monitoring.

Taking the above factors into consideration, the indirect impact of new or modified control and surveillance programs was considered unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

Domestic trade or industry effects

There are currently several feral pig populations infected with *B. suis* in Australia, however, there is no discernible impact on domestic trade in meat or live animals. If a small commercial piggery was found to be infected the piggery is likely to be either destocked or a test and slaughter program introduced and movement restrictions on breeding animals. Herds in the area may need to be accredited or tested free of *B. suis* to trade in breeding pigs. Given this, the indirect impact on domestic trade was considered unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

International trade effects

The presence of porcine brucellosis in feral pigs in several areas of Queensland does not currently affect export of meat. Several countries currently require testing of live pigs for *B. suis* prior to export or donor boars prior to collection of semen. The OIE Code Chapter on porcine brucellosis provides guidelines for the importation of pigs for breeding and slaughter and porcine semen but not meat. Overall it was considered likely that the indirect effect on international trade was unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

Indirect impact on the environment

The indirect impact on the environment, such as reduced biodiversity, was considered unlikely to be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on communities

The indirect impact on local communities, such as reduced rural and regional economic viability, was considered unlikely to be discernible at any level, hence this criterion was rated 'A'.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, *B. suis* would have established in a local population of backyard piggeries or small commercial piggeries, and may have spread to abattoir workers and other susceptible species. The disease would be contained through the diagnosis of illness in humans or animals (i.e. pigs, horses, cattle, dogs), and the mounting of a control program.

The direct impact of B. suis infection

Animal life or health

The clinical signs of *B. suis* infection in pigs may result in abortions, infertility, orchitis, posterior paralysis and lameness. In other cases clinical signs can be very mild. In other species clinical signs may include bursitis or orchitis or in some cases clinical signs may be absent. Given this, the direct effect on animal health was considered unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

Environment

Brucella suis is unlikely to affect native Australian species, with the possible exception of dingoes, although this does not seem to be the case in Queensland. Given this, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of B. suis infection

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

Following the detection of *B. suis*, some surveillance of the domestic pig population based on tracing is likely to occur. Porcine brucellosis is not covered by the Emergency Animal Disease Response Agreement, and control costs may have to be borne by the producer, although State governments may consider compensation. Affected piggeries may be destocked or a slaughter and eradication program implemented. An accreditation program similar to that implemented in Queensland may apply. Domestic pigs being moved out of the *B. suis* endemic area of Queensland to other piggeries are required to undergo testing or come from an accredited free herd.

If infected pigs were sent for slaughter, there would need to be an eduction program for abattoir workers to cover occupation health and safety issues.

On this basis, the indirect impact of new or modified control programs was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

Domestic trade or industry effects

With a local outbreak of *B. suis* in small commercial piggeries or backyard enterprises, it is likely that pig herds in that and the surrounding area may need to test breeding pigs and semen donors prior to sale and semen collection respectively. A similar scheme to that which applies to pig herds in Queensland could be implemented.

There may be a general decrease in domestic pig meat consumption if additional human cases of brucellosis were diagnosed, particularly in abattoir workers. This could cause some disruption to domestic trade and associated industries.

Considering these issues, the indirect impact on domestic trade and industry was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

International trade effects

It is likely that breeding pigs and semen donors would need to be tested for *B. suis* before export. This already applies for several of our markets. Although the human health risk of brucellosis is predominantly associated with abattoir workers, not pig meat *per se*, several of Australia's major export markets for pig meat are very sensitive to human health issues. It is possible assurance may need to be provided to these markets that meat was sourced from herds free from *B. suis*. In this outbreak scenario, the disease has only spread to a local population of small commercial piggeries or backyard enterprises, nonetheless additional testing of domestic herds may be required to demonstrate freedom in larger commercial piggeries.

On this basis, the likely indirect effect on international trade was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

Indirect impact on the environment

The indirect impact on the environment, such as reduced biodiversity, was considered unlikely to be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on communities

The indirect impact on local communities, such as reduced rural and regional economic viability, was considered unlikely to be discernible at any level, hence this criterion was rated 'A'.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, *B. suis* would have established in a broader population of commercial piggeries (including medium-large piggeries), and may have spread to abattoir workers and other susceptible species. A control program would likely be mounted in response to the isolation of the agent from affected humans, or from the diagnosis of the disease in pigs or other animals.

The direct impact of B. suis infection

Animal life or health

Porcine brucellosis is a recognised production disease in commercial piggeries as it causes reduced farrowing rates, abortions, neonatal deaths, and failure to breed. Boars can be rendered infertile and artificial insemination with semen from infected boars and natural mating using infected boars can spread the disease. Given this, the direct effect on animal health was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

Environment

Brucella suis is unlikely to affect native Australian species, with the possible exception of dingoes, although this does not seem to be the case in Queensland. Given this, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of B. suis infection

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

A general outbreak of porcine brucellosis is likely to result in an eradication program. However, as previously stated, the Emergency Animal Disease Response Agreement does not include this disease, hence it is likely that producers would pay at least a proportion of the cost of any eradication and control program. The eradication and control program may include options such as (a) slaughter of the entire herd, cleaning and disinfecting the premises, and restocking with brucellosis-free stock; or (b) segregating the offspring and keeping them in isolation so that they form the nucleus of a free herd; or (c) regular testing of all pigs and disposal of reactors.

Overall, it was considered likely that the indirect impact of new or modified control programs would be unlikely to be discernible at the national level, but would have a minor impact at the State level. This resulted in a rating of 'D' for this criterion.

Domestic trade or industry effects

With further spread of *B. suis* to commercial piggeries trade in pigs and pig semen could be disrupted. Herds would likely need to be accredited free before animals or semen could be traded. If *B. suis* became widespread there would be ongoing costs to producers as a result of decreased pig production.

Hygienic procedures at abattoirs would need to be strengthened to minimise the potential for zoonotic spread during slaughter. If human cases of brucellosis occurred, pig meat consumption may decrease. A publicity campaign may need to be undertaken to reassure the public that cooked pig meat was safe to eat. If pig meat consumption remained depressed, associated industries such as processors, transport and stockfeed manufactures may also be affected.

Taking these issues into account, the indirect impact on domestic trade and industry was considered unlikely to be discernible at the national level, but would have a minor impact at the State level. This resulted in a rating of 'D' for this criterion.

International trade effects

As with scenario 3, it was considered that additional testing and certification requirements may be imposed by countries importing live pigs or pig semen. Although the OIE Code Chapter on porcine brucellosis does not provide recommendations for trade in pig meat, it is feasible that some markets may require additional measures. This could include certification that the meat was derived from pigs from herds free from *Brucella suis*. Additional testing of herds may be necessary to meet this requirement.

On this basis, the likely indirect effect on international trade was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

Indirect impact on the environment

The indirect impact on the environment, such as reduced biodiversity, was considered unlikely to be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on communities

The indirect impact on local communities, such as reduced rural and regional economic viability, was considered unlikely to be discernible at any level, hence this criterion was rated 'A'.

The overall impact of porcine brucellosis

When the direct and indirect impacts of *B. suis* infection were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences negligible

Scenario 3: Consequences very low

Scenario 4: Consequences low

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 75, Table 76, Table 77, and Table 78. It can be seen that the likely consequences associated with the exposure of feral pigs, backyard pigs, pigs in small commercial piggeries or other susceptible species to infected pig meat scraps were considered 'negligible', 'negligible', 'very low' and 'negligible' respectively.

Table 75 *B. suis*: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Moderate	Negligible	Negligible
Scenario 3	Very low	Very low	Negligible
Scenario 4	Extremely low	Low	Negligible
		Overall likely consequences	**Negligible**

Table 76 *B. suis*: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	High	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Low	Very low	Negligible
Scenario 4	Very low	Low	Negligible
		Overall likely consequences	**Negligible**

Table 77 *B. suis*: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Moderate	Very low	Very low
Scenario 4	Very low	Low	Negligible
		Overall likely consequences	**Very low**

Table 78 *B. suis*: summary of the consequences of exposure of other susceptible species

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	High	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Extremely low	Very low	Negligible
Scenario 4	Extremely low	Low	Negligible
		Overall likely consequences	**Negligible**

Human life or health

Separate to the above, is consideration of the consequences to human life or health. *B suis* is a zoonotic agent. Reporting of human brucellosis is mandatory in Australia. Each year, a number of human brucellosis cases due to infection with *B suis* are reported from Central West and South West Queensland. These cases generally occur in people involved in hunting feral pigs. Treatment consists of appropriate antibiotics and supportive treatment.

Should the disease establish in Australia in the domestic pig population, workers at abattoirs may be at risk of infection with *B. suis.* In 1992, an investigation of human cases of brucellosis at an abattoir processing pigs in the United States of America found that 19% of 154 workers in the kill division had been infected. Two people required hospitalisation. Employees were then equipped with rubber gloves and face shields to minimise infection (Centers for Disease Control and Prevention, 1994).

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with porcine brucellosis.

Table 79 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk meets Australia's ALOP (very low), risk management would not be required for porcine brucellosis.

Table 79 *B. suis*: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Very low	High	Negligible	Negligible
Backyard pigs	Very low	Very low	Negligible	Negligible
Small commercial piggeries	Very low	High	Very low	Very low
Other susceptible species	Very low	Low	Negligible	Negligible
			Overall annual risk	**Very low**

The Department of Health and Ageing has advised Biosecurity Australia that biosecurity measures for *B. suis* would be required to manage the risk to human life or health associated with the importation of pig meat. Appropriate measures for countries where *B. suis* is endemic, in the case of uncooked pig meat (not subject to further processing in Australia prior to retail sale), would be to require that the pigs from which the meat is derived be sourced from herds which have been tested negative, or are accredited free from *B. suis*.

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Porcine epidemic diarrhoea virus

Technical information

Background

Porcine epidemic diarrhoea (PED) is an infectious and contagious enteric disease of pigs, first reported in Belgium and the United Kingdom in 1978. Outbreaks have since been reported in many parts of Europe and in Asia. It has not been reported in Sweden, Northern Ireland, Hungary, the Americas, Australia or New Zealand. Infection with PED virus results in clinical signs similar to that of another porcine coronavirus, transmissible gastroenteritis (TGE), and careful identification of the virus or circulating antibodies is necessary to confirm the diagnosis.

Agent taxonomy

Porcine epidemic diarrhoea virus, an RNA virus, is a member of the family Coronaviridae, genus Coronavirus (Pensaert, 1999). Although antigenically distinct it is closely related to other porcine coronaviruses: transmissible gastroenteritis virus, haemagglutinating encephalomyelitis virus and porcine respiratory coronavirus. According to current nucleic acid detection techniques there are no indications that different strains of PED virus exist.

Agent properties

Porcine epidemic diarrhoea virus is difficult to culture. Cell culture adapted PED virus loses its infectivity when heated to at least 60°C for 30 minutes but is moderately stable at 50°C. The virus is stable between pH 5.0 and 9.0 at 4°C and between pH 6.5 and 7.5 at 37°C. Viral infectivity is not impaired by ultrasonication or by multiple freezing and thawing (Hofmann & Wyler, 1989).

Host range

Pigs are the only known vertebrate hosts.

Epidemiology

Porcine epidemic diarrhoea virus is transmitted by faeces from infected pigs. The faeco-oral route of transmission is the main, if not the only, route of infection. Outbreaks generally occur within 4 to 5 days following the introduction of infected pigs to the herd. The virus can also enter the herd indirectly by way of infected trucks, contaminated boots or fomites (Pensaert, 1989). Pigs are usually protected against reinfection 3 weeks after original infection (de Arriba, et al., 2002a) but not 5 months after exposure to virulent PED virus (Pensaert, 1999). It has been reported that the disease can occur all year round but is more prevalent during the cold season (Shibata, et al., 2000; Chae, et al., 2000).

In an outbreak in a 240-sow herd producing fattening pigs in 1989 in the Netherlands, clinical signs were most obvious in fattening and breeder pigs but were mild or not present in suckling pigs and young weaners. Infection and diarrhoea persisted in pigs 6 to 10 weeks old and in newly introduced gilts. The virus was detected in 90% of sampled pigs over 6 weeks old. No virus could be detected in the 3 to 6 week old pigs (Pijpers, et al., 1993).

In contrast to the present situation in Europe where outbreaks of diarrhoea due to PED virus infection occur mainly in feeder and fattening pigs and young breeding pigs, severe outbreaks in sucking pigs have been reported in Asia.

The between herd and within herd prevalence of PED virus infection, as determined by serological surveys, differ significantly between countries. In Britain, outbreaks of PED were common in finishing units during the early 1980's before dwindling to very occasional outbreaks in the 1990s. In a survey of 64 herds in East Anglia in 1996-97, only 9 (14%) herds contained seropositive finishing pigs. Of the 634 sera tested from these 64 herds only, 12 samples tested positive (Pritchard, et al., 1999). Whereas, in a region of Spain, where 25% of breeding sows were located, a survey found 442 of 803 herds (55.9%) sampled had seropositive sows. In Korea, PED mainly affected unweaned piglets and was diagnosed in 304 of 639 pig herds (48%) (Chae, et al., 2000).

In Switzerland, only 7 of 600 serum samples collected from sows and boars in the slaughterhouses of two cantons were seropositive to PED virus (Knuchel, et al., 1992). However, in another study conducted in Switzerland, 251 of 1024 pig sera (24.5%) collected from a slaughterhouse were seropositive to PED virus (Hofmann & Wyler, 1990).

High within herd seroprevalence of PED infection has been reported. Monthly testing of finisher pigs on a PED virus infected farm in Switzerland showed that by the age of 4 months, 27 of 30 (90%) had antibodies (Knuchel, et al., 1992). In Spain within herd seroprevalence of PED has been reported as ranging from 46% to 56.9% depending on the size of the farm. Small farms with less than 21 sows had the highest seroprevalence (Carvajal, et al., 1995a).

Clinical signs

The disease is characterised by anorexia, profuse watery yellow-green diarrhoea and high morbidity. Pigs are reluctant to stand, exhibit apparent abdominal pain and usually develop diarrhoea 2 to 4 days after infection, with diarrhoea lasting several days. Recovery occurs as a rule after 7 to 10 days in individual pigs. However, persistent diarrhoea has been reported in some herds. This may be due to the disease perpetuating in the herd through frequent introductions of susceptible pigs such as naïve gilts and weaners that have lost their maternal immunity. Growth may also be retarded in young animals. Low mortality is generally observed with the disease in Europe while high mortality is often seen in young piglets in Asia (Pensaert, 1999).

Pathogenesis

After ingestion, the virus passes through the stomach and infects the villous epithelial cells of the small intestines and the colon within 12 to 18 hours. Maximum infection of epithelial cells was reached between 24 and 36 hours post-inoculation. Viral replication results in degeneration of the villous enterocytes, causing villous atrophy within the jejunum and ileum (Pensaert, 1999). These pathogenic features are very similar to those of TGE, however, the events after TGE virus infection are of a more rapid and drastic nature (Pensaert, 1989).

Virus shedding in faeces, as determined by the detection antigen in experimentally infected pigs, started 1 to 3 days after infection and had an average duration of 5 to 6 days (de Arriba, et al., 2002b). However, PED virus antigens have been detected in faeces of experimentally infected pigs until 11 days after infection (Carvajal, et al., 1995b).

Pathology

Gross pathology is confined to the small intestine which is distended with yellow fluid (Pensaert, 1999).

Immunology

Serum antibodies against PED virus can be detected in some pigs as early as day 4 post-infection, in most pigs from day 7 and in all pigs from day 12. Antibodies persist for at least a year (de Arriba, et al., 2002a). However, these antibodies do not protect against reinfection, this is based on the presence of intestinal mucosal immunity, which is only of short duration (Pensaert, 1999).

Transmission via meat

No studies were identified in which PED virus was demonstrated in muscle tissue, muscle vasculature, adipose tissues, lymphatic system or the skeletal system. The virus has been identified only in the villous epithelial cells of the small intestine but not in the caecum or colon of naturally infected piglets. Moreover lung, tonsil and stomach specimens were negative for *in situ* hybridisation for PED virus nucleic acids (Kim & Chae, 2000). Experimentally an oral dose of 3 ml of tissue culture fluid containing 10^4 $TCID_{50}$/ml of PED virus has resulted in infection (Kim, et al., 2000).

Release assessment

R1 — the likelihood that a source herd is infected

While small surveys suggest few pig herds were infected in Britain, larger surveys in Spain and Korea showed 56% and 48% of herds respectively had been exposed to PED virus. Given this, the likelihood of selecting slaughter-age pigs from an infected source herd was considered to be ‘moderate’.

R2 — the likelihood that a slaughter-age pig is infected

The within herd seroprevalence of PED virus has been reported to range from 46% to 90%. In Switzerland, a survey of sows at slaughter found that 24.5% had been exposed to the virus. Nonetheless, infection in feeder pigs often occurs shortly after arrival at the fattening unit. Given that most pigs shed the virus in their faeces for a maximum of 11 days post-infection, and a carrier state has not been described, pigs are less likely to be infected by the time they reach slaughter age. Based on this information, it was considered that the likelihood of selecting an infected animal in an infected herd was ‘very low’.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

Slaughter-age pigs infected with PED virus may show signs of transient inappetence and loose faeces. Any excessively soiled pigs are likely to be either not passed for inspection or passed for slaughter subject to conditions that ensure they do not contaminate animals, carcases and carcase parts during the slaughter process. Once slaughtered, the pathology is unremarkable and consequently infected pigs are unlikely to be identified during post-mortem inspection. Hence the sensitivity of ante-mortem, slaughter and processing requirements in detecting and removing PED virus infected pigs was estimated to be ‘extremely low’.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with PED virus and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a ‘one minus the background rejection rate’. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered ‘extremely low’. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

Porcine epidemic diarrhoea virus has been identified only in the digestive tract, which is removed during the slaughter process. Contamination from faecal soiling is possible, however, the rectum is tied off before being removed to prevent soiling and washing of the carcass removes any accidental soiling. Given this, the likelihood that PED virus would be present in meat harvested for export that was derived from an infected carcass was considered to be ‘extremely low’.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

Given that the PED virus is stable between pH 5.0 and 9.0 at 4°C and between pH 6.5 and 7.5 at 37°C and that meat has been assumed to reach a pH of 6.2 during maturation, it was considered the there was a ‘high’ likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

Given that PED virus is stable between a wide pH range at 4°C and is not impaired by multiple freezing and thawing, it was considered that there was a ‘high’ likelihood that meat infected or

contaminated with PED virus at the completion of carcass maturation would remain infected during transport and storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was an 'extremely low' likelihood that imported pig meat derived from an individual carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was 'extremely low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

Porcine epidemic diarrhoea virus has only been found in the gastrointestinal tract, which is removed at slaughter. Viraemia does not seem to be a feature of this disease. It is possible that meat may be contaminated with faeces infected with PED virus at slaughter; however, most of this contamination will be removed during washing. As such, if meat is contaminated by this means, it is likely that little virus would be present.

It is known that PED virus is transmitted via the oral route and experimentally doses of at least 10^4 $TCID_{50}$/ml initiated infection. Epidemiological studies suggest the faeco-oral route is the only mode of transmission in the field.

In view of these facts, it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of PED virus to initiate infection was 'extremely low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

Porcine epidemic diarrhoea virus is moderately stable at temperatures up to 50°C, but is dependent on pH being stable between pH 6.5 and 7.5 at 37°C. As the pH of meat declines after slaughter and during putrefaction this will affect the viability of the virus. Coronaviruses are generally susceptible to sunlight. Given these facts, it was considered that the likelihood that PED virus would remain viable after exposure to sunlight, putrefaction and the environment in meat scraps discarded in refuse for the period of time for feral pigs to locate and subsequently scavenge the material was 'moderate'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered

that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = Extremely low
- Rural regions = Negligible
- Large towns = Negligible

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be ‘extremely low’.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was ‘extremely low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was ‘extremely low’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a 'high' likelihood that PED virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'negligible'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was 'extremely low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'extremely low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a 'high' likelihood that PED virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'extremely low'.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the three exposure groups - that is, feral pigs, pigs in backyard enterprises and pigs in small commercial piggeries. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and

4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Porcine epidemic diarrhoea spreads to susceptible pigs by ingestion of faeces from infected pigs. The disease spreads quickly within pens where pigs are intensively housed where profuse diarrhoea containing virus enhances spread of the contagious material throughout the piggery. However, the disease spreads more slowly where pigs are extensively housed and similarly is expected to spread slowly within a feral pig herd. The relatively short excretory period of virus in faeces may limit transmission between feral pig herds.

There are no definitive reports or evidence of PED in wild boars in Europe, although there is a report of 14 of 5000 wild boars in Germany seropositive to a porcine coronavirus (Dedek, et al., 1989). However, this may refer to TGE virus or PRCV.

Feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs; however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: high

Scenario 2: low

Scenario 3: very low

Scenario 4: very low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the ‘no outbreak’ scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

Porcine epidemic diarrhoea infection in backyard pigs may be evident as profuse diarrhoea lasting several days amongst older pigs with very low mortality rates or diarrhoea, dehydration and deaths amongst young piglets. An outbreak may be self-limiting and resolve within a couple of weeks, particularly if no naive pigs are introduced in this period. Spread of the diseases to other backyard herds or small commercial piggeries could occur via the movement of recently infected pigs or indirectly via faecal contamination of trucks, crates, feed or boots. Owners of infected backyard pigs are unlikely to seek veterinary attention and may pass the episode off as an outbreak of swine dysentery or colibacillosis. Even with veterinary attention, it may take some considerable time before the disease was diagnosed accurately, with endemic diseases needing to be ruled out. This could result in spread of the disease.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: very low

Scenario 3: low

Scenario 4: low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the ‘no outbreak’ scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between ‘small commercial piggeries’ and ‘backyard piggeries’ is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

An outbreak of PED in small commercial piggeries is more likely to result in veterinary attention, but it may be some time before the disease is accurately diagnosed, as endemic

diseases would need to be ruled out first. Moreover, there are no diagnostic tests for PED currently available in Australia, such that samples may need to be sent overseas. Due to the larger herd size and introductions and movements of pigs within the small commercial piggery, the outbreak may persist for several months or longer. This factor together with the relatively high level of movements of pigs, personnel and fomites, between piggeries may result in further spread of disease.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: low

Scenario 4: moderate

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this scenario, the disease is most likely to have established amongst the directly exposed animals, and to have run its course without identification. This is because the clinical symptoms of this disease are not distinct and may be confused with endemic diseases.

The direct impact of porcine epidemic diarrhoea

Animal life or health

Porcine epidemic diarrhoea virus may result in high mortalities in young piglets, however, if older pigs are infected profuse diarrhoea with low mortalities may be seen. As spread is limited to within the infected herd in this scenario, the likely impact of PED on animal health was considered unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

Environment

Because PED is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of porcine epidemic diarrhoea

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this outbreak scenario, the disease is contained within the directly exposed group of pigs. Thus, it was considered unlikely that the disease would be diagnosed in the primary herd.

Given this, it was considered unlikely that the indirect impact of new or modified control programs would be discernible at any level and the rating assigned to this criterion was therefore 'A'.

Domestic trade or industry effects

As discussed above, it was considered unlikely that PED would be diagnosed in a single herd. Hence the indirect impact of PED on domestic trade and industry was considered unlikely to be discernible at any level and a rating of 'A' was given to this criterion.

International trade effects

As it was unlikely that PED would be diagnosed under this scenario, it was considered that the indirect impact of PED on international trade was unlikely to be discernible at any level. Thus, a rating of 'A' was assigned to this criterion.

Indirect impact on the environment

Porcine epidemic diarrhoea in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, the disease is most likely to have established within a population of feral pigs, and to have run its course without identification. As explained above, this is because the clinical symptoms of this disease are not distinct.

The direct impact of porcine epidemic diarrhoea

Animal life or health

As with the first scenario, the direct impact on animal health was considered unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

Environment

Because PED is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of porcine epidemic diarrhoea

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this outbreak scenario, PED spreads to feral pigs, but not to domestic pigs. As the disease is unlikely to be diagnosed, the indirect impact of new or modified control programs was considered unlikely to be discernible at any level. This resulted in a rating of 'A' for this criterion.

Domestic trade or industry effects

It was considered likely that the disease may remain undiagnosed within feral pigs for a significant period of time. As such, it was considered that the indirect effects on domestic trade and industry were unlikely to be discernible at any level, hence this criterion was rated 'A'.

International trade effects

As described above, the indirect effect of PED on international trade was unlikely to be discernible at any level, hence this criterion was rated 'A'.

Indirect impact on the environment

Porcine epidemic diarrhoea in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, the disease has established within a local population of small commercial piggeries or backyard enterprises. It is likely that the disease would be diagnosed due to the increased piglet mortality and diarrhoea in older animals. Control measures such as quarantine and movement restrictions would likely be applied to limit spread.

The direct impact of porcine epidemic diarrhoea

Animal life or health

This scenario is characterised by spread of PED to a local population of domestic pigs in backyard enterprises or small commercial piggeries, but containment within this population. Porcine epidemic virus can cause high mortality in young piglets and profuse diarrhoea in older pigs with high morbidity. Given this, it was considered that the direct effect on animal health would be unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

Environment

Because PED is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of porcine epidemic diarrhoea

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this scenario, PED virus spreads to a local population of domestic pigs in backyard enterprises or small commercial piggeries. It was considered that PED would be diagnosed under this scenario.

Porcine epidemic diarrhoea is not included in Australia's Emergency Animal Disease Response Agreement and as such the cost of control measures and eradication would likely have to be met by industry. Should the disease occur in Australia, it may be considered as an emerging disease and the outbreak managed by adopting cost-effective strategies to control and, if feasible, eradicate the disease. In this limited outbreak, it may be possible to eradicate the disease with quarantine and movement controls similar to those proposed for the control of TGE.

Some surveillance of domestic pigs and possibly feral pigs may need to be undertaken to determine the extent of the outbreak.

When these issues were taken into account, the indirect impact of new or modified control programs was considered unlikely to be discernible at the national or State level, and of minor importance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

Domestic trade or industry effects

If movement controls are implemented, movement of pigs from infected herds may be restricted to those moving directly to slaughter. Thus, the movement of live pigs for breeding purposes or to saleyards may be prohibited. As this is likely to affect only those producers within the local area, the indirect impact of PED on domestic trade and industry was considered unlikely to be discernible at the national or State level and of minor significance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

International trade effects

The diagnosis of PED in domestic pigs may result in cessation of live pigs and semen to certain markets. Testing of pigs prior to export or semen donors may be an option. Australia exports only small numbers of breeding pigs and small quantities of semen. Porcine epidemic diarrhoea is not an OIE Listed disease. The detection of PED virus in Australia is unlikely to affect the export of meat. The disease is present in Japan, and Singapore does not have a pig industry and PED virus has no human health implications. Thus, the indirect effect of PED on international trade was considered unlikely to be discernible at any level. Overall, this resulted in a rating of 'A' for this criterion.

Indirect impact on the environment

Porcine epidemic diarrhoea in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, PED would have established in a broader population of commercial piggeries (including medium-large piggeries) and be identified. If the disease was not widespread in the Australian pig population, a control program may be implemented, alternatively, if widespread, control may be left to individual producers.

The direct impact of porcine epidemic diarrhoea

Animal life or health

In this scenario, where PED has spread to a more general population of domestic pigs including medium to large commercial piggeries, the direct impact on animal health of mortality amongst piglets or profuse diarrhoea amongst older pigs, was considered unlikely to be discernible at the national or State level, but would be of minor importance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

Environment

Because PED is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of porcine epidemic diarrhoea

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this outbreak scenario, PED virus has spread to a more general population of domestic pigs, including medium and large commercial piggeries. Surveillance may be undertaken to determine the extent of the spread of the virus and the feasibility of any control and eradication program. A similar control and eradication program to that recommended by AUSVETPLAN for TGE could be implemented, where stamping out is used sparingly and as many animals as possible are salvaged. However, industry may consider it to be uneconomic. The Panel is unaware of any attempts by a country with PED to eradicate the disease.

If an eradication and control program was not implemented, producers may be advised on biosecurity management practices for their premises. The disease can be managed within the herd by controlling movement of pigs through various units within the piggery to minimise the risk of transmission.

On this basis, it was considered that the indirect impact of new or modified control programs would be unlikely to be discernible at the national or State level, but of minor importance at the district or regional level, thus this criterion was rated as 'C'.

Domestic trade or industry effects

If quarantine and movement controls were implemented, this would mainly affect the sale of breeding pigs. The supply of pork to the domestic market should not be significantly disrupted as most pigs would still be able to be sent to slaughter.

However, productivity on infected piggeries would be reduced. Should PED virus infection result in high piglet mortality, losses may be similar to those for TGE, that is, where piglet mortality exceeds 50%, loss in net revenue is likely to exceed 70% in the first 6 months after the outbreak (Mullan, et al., 1994).

When all these issues were taken into account, the indirect impact of PED on domestic trade and industry was considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. This resulted in a rating of 'C' for this criterion.

International trade effects

As discussed in scenario 3, the diagnosis of PED in domestic pigs may result in cessation of pigs and semen to some markets but is unlikely to affect the export of meat. Thus the indirect effect of PED on international trade was considered unlikely to be discernible at any level. Overall, this resulted in a rating of 'A' for this criterion.

Indirect impact on the environment

Porcine epidemic diarrhoea in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

The overall impact of porcine epidemic diarrhoea

When the direct and indirect impacts of PED were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences negligible

Scenario 3: Consequences very low

Scenario 4: Consequences very low

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 80, Table 81, and Table 82. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small

commercial piggeries to infected pig meat scraps were considered ‘negligible’, ‘negligible’ and ‘very low’ respectively.

Table 80 PED: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	High	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Very low	Very low	Negligible
Scenario 4	Very low	Very low	Negligible
		Overall likely consequences	**Negligible**

Table 81 PED: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Low	Very low	Negligible
Scenario 4	Low	Very low	Negligible
		Overall likely consequences	**Negligible**

Table 82 PED: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Low	Very low	Negligible
Scenario 4	Moderate	Very low	Very low
		Overall likely consequences	**Very low**

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the ‘partial annual risk’ associated with each of the exposure groups;

- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with PED virus.

Table 83 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly less than Australia's ALOP (very low), risk management would not be required for PED virus.

Table 83 PED: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Extremely low	Extremely low	Negligible	Negligible
Backyard pigs	Extremely low	Negligible	Negligible	Negligible
Small commercial piggeries	Extremely low	Extremely low	Very low	Negligible
			Overall annual risk	**Negligible**

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Porcine respiratory coronavirus

Technical information

Background

Porcine respiratory coronavirus (PRCV) was first isolated in 1984 in Belgium but has now been reported throughout Europe (Pensaert, et al., 1993; Martin, et al., 1994; Flori, et al., 1995) and other countries including North America (Wesley, et al., 1990) and Asia (Laude, et al., 1993; Chae, et al., 2000). Its natural host range is restricted to pigs. It is a deletion mutant of transmissible gastroenteritis (TGE) virus and PRCV-infected pigs produce antibodies that neutralise TGE virus. In contrast to TGE virus, however, it is a respiratory virus of limited pathogenicity (Saif & Wesley, 1999).

Agent taxonomy

PRCV is an enveloped, single-stranded RNA virus of positive polarity. It is a member of the genus Coronavirus of the family Coronaviridae (Pensaert, 1989).

Agent properties

No reports or studies have been identified that address the biological characteristics of PRCV. Nonetheless various studies have determined the properties of TGE virus and these have been collated (Saif & Wesley, 1999). Transmissible gastroenteritis virus is stable when frozen but labile at room temperature and above. For example at 37ºC loss of infectivity was observed after 4 days. Transmissible gastroenteritis virus is also highly photosensitive. The virus is stable within a pH range of 3 to 9. Porcine epidemic virus, another coronavirus is relatively stable at 4ºC over a pH range from 5 to 9, whereas at 37ºC the virus was stable only between 6 and 8, being completely inactivated at a pH less than 4 and pH greater than 9 (Hofmann & Wyler, 1989).

Host range

The host range is limited to the pig.

Epidemiology

PRCV was first detected in Belgium in 1984 when routine surveillance for TGE virus resulted in 68% of sows registering neutralising antibodies, in contrast to the expected 12 to 14%. These sows had no clinical signs of TGE virus infection nor were they vaccinated against TGE (Pensaert, et al., 1986). Subsequently, it was determined that a deletion mutation of TGE virus had occurred, resulting in a coronavirus that shared complete cross-neutralisation with TGE virus, and thus could not be distinguished by classical seroneutralisation tests (Callebaut, et al., 1989). However, PRCV exhibited respiratory rather than enteric tropism, and caused little, if any, clinical disease. A coronavirus, similar in pathogenicity, tissue distribution and antigenicity, was reported in pigs in the United States of America in 1990 (Wesley, et al., 1990). The genomic differences between the European and American PRCV viruses are such that the two strains are thought to have arisen independently (Laude, et al., 1993).

Observational (Flori, et al., 1995) and experimental studies (Pensaert, et al., 1986; Pensaert & Cox, 1989; Cox, et al., 1990a) support a predominately aerogenous mode of transmission for

PRCV. The virus appears to be highly contagious and spreads rapidly between, and within, herds. Pigs of all ages may be infected but the duration of immunity is short. Periodic, seasonal re-infection of herds is reported (Pensaert, et al., 1993). The role, if any, of carrier animals in the reappearance of infection in a herd has not been defined. Pigs are usually infected between 5 and 10 weeks of age, although age of infection depends on factors including herd exposure to the virus, and the presence and degree of protection afforded by maternally-derived anti-PRCV antibodies (Pensaert & Cox, 1989).

PRCV is enzootic in many European countries and estimates of the proportion of herds infected in Belgium approached 100% (Pensaert & Cox, 1989). However, in areas that are less densely populated with pig farms, or in which the infection has only recently been introduced, the proportion of infected herds may be lower. The virus is thought to have been introduced into Denmark in 1984; a cross-sectional study performed in 1985 and 1986 showed evidence of PRCV infection in 61.5% of surveyed herds (Flori, et al., 1995). Similarly, PRCV was introduced into Spain in 1986; in 1991 the prevalence of PRCV infection in pig herds in the Catalunya region was between 75% and 91% (Martin, et al., 1994). Workers from the Netherlands (van Nieuwstadt & Boonstra, 1992) suggest that by 1990, most herds in the Netherlands were infected with PRCV. PRCV was first described in Korea in 1996. A serosurvey conducted in Korea in 1998 and 1999 showed that 61.3% of herds had evidence of PRCV infection (Chae, et al., 2000).

The prevalence of exposure to PRCV within an infected herd is typically very high. The Danish study (van Nieuwstadt & Boonstra, 1992) showed an overall mean within-herd prevalence of 90%, with 78.5% of the positive herds having 100% seropositive samples.

Clinical signs

Infection with PRCV is often subclinical and the presence of infection within a herd may only be suspected when serum neutralisation tests for TGE virus are positive in the absence of any signs of TGE virus infection (Pensaert, et al., 1986). However, signs of mild to (occasionally) moderate respiratory disease have been reported, particularly with North American strains under experimental, rather than field, conditions (Laude, et al., 1993; Paul, et al., 1994). The severity of disease manifested is likely affected by factors including age at infection, dose of virus, and environmental factors (Laude, et al., 1993).

Pathogenesis

Primary infection of the respiratory tract occurs after aerosol inoculation. The PRCV virus replicates to high titres in alveolar epithelial cells, epithelial cells of the nasal, tracheal, bronchial and bronchiolar pathways, and the alveolar macrophages (Pensaert & Cox, 1989). Viremia is detected by the second day after experimental infection via aerosol inoculation and persists for at least 6 days. Virus also can be isolated from the tonsils and mesenteric lymph nodes for at least 6 days post-inoculation. Experimentally PRCV has been detected in nasal secretions for 10 days post-exposure (Wesley, et al., 1990). Isolated intestinal epithelial cells contain viral antigen (Cox, et al., 1990a). These cells are likely exposed after the swallowing of respiratory secretions. Limited replication occurs in the small intestine (Pensaert & Cox, 1989). It is possible to infect pigs with PRCV via direct gastric inoculation but a high viral dose (10^7 $TCID_{50}$) was used (Cox, et al., 1990b). In this pig, virus was isolated in low titres from some faecal samples, but faecal excretion is not thought to be a major source of natural infection.

Pathology

Gross lesions after experimental infection are limited to the respiratory tract and are described as moderate-to-severe, tan-to-plum coloured mottled consolidated areas with irregular borders most commonly involving the cranial, middle and accessory lung lobes (Halbur, et al., 1993). Other workers have described consolidated areas as being greyish-plum coloured (Jabrane, et al., 1994). Enlarged tracheobronchial lymph nodes (7 to 11 days post-inoculation) have also been reported (O'Toole, et al., 1989).

Microscopic lesions, consistent with varying degrees of bronchointerstitial pneumonia, are described (O'Toole, et al., 1989; O'Toole, et al., 1989; Pensaert & Cox, 1989; Paul, et al., 1994; Paul, et al., 1994).

Immunology

Piglets acquire maternally-derived anti-PRCV antibodies from colostrum. These antibodies have a half-life of about 12 days but may be detected for up to 16 weeks (Callebaut, et al., 1989). Active infection has been shown to occur while maternal antibodies are still present. The age at which pigs are typically infected must depend, in part, on the concurrent herd infection status. One study followed 10 groups of pigs from birth, and determined that four groups had seroconverted within 1 month of weaning, an additional four groups by 3 months of age, and the remaining two groups by the end of the fattening period (5 months) (Pensaert, et al., 1986).

Infected pigs produce neutralising antibodies that can be detected 2 weeks after infection (Garwes, et al., 1988; Callebaut, et al., 1989).

Transmission via meat

No studies have been identified that address the possible transmission of PRCV via the ingestion of infected meat.

It is known that PRCV exhibits marked tropism for cells of the respiratory tract. The virus can survive passage through the intestinal tract and has been detected in small amounts in intestinal cells after (it is thought) the swallowing of respiratory secretions. Pigs were infected by direct gastric inoculation but very large quantities of the virus (10^7 $TCID_{50}$) were required (Cox, et al., 1990b).

Release assessment

R1 — the likelihood that a source herd is infected

The likelihood of selecting slaughter-age pigs from an infected source herd (in a country where PRCV is endemic) was considered to be 'high', as most studies report between herd prevalence figures in excess of 70%.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

In an infected herd, most pigs are likely to be infected with PRCV soon after weaning. High within-herd prevalence has been reported of up to 90%. Nonetheless viraemia is short and experimentally nasal shedding of PRCV has been reported to occur through 10 days post-exposure. A carrier state for PRCV has not been described. Based on this information, it was considered that the likelihood of selecting an infected animal in an infected herd was 'low'.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

Infection with PRCV is usually clinically inapparent, thus infected pigs are extremely unlikely to be condemned on the basis of ante-mortem inspections. The grossly visible pathological changes resulting from PRCV infections may result in condemnation of the lungs but would not result in condemnation of the carcass.

Given this, the likelihood that a PRCV infected pig would be identified via ante- and post-mortem inspection procedures is considered 'negligible'.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with PRCV and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a 'one minus the background rejection rate'. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered 'extremely low'. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

The virus exhibits marked tropism for the respiratory tract, which is removed after slaughter. Moreover, bleeding the carcass should remove, to a large extent, virus present in muscle due to viraemia. The likelihood that PRCV virus would be present in meat, harvested for export, that was derived from an infected carcass, was considered to be 'extremely low'.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

No information is available concerning the effect of pH changes on PRCV viability. However, it is known from experimental studies that although PRCV exhibits little to no replication in the intestine, the virus survives passage through the low pH environment of the stomach (Cox, et al., 1990a). In addition, the virus is very closely related to TGE virus. Transmissible gastroenteritis virus is stable between pH 3 and 9. As there is no information indicating that the stability of PRCV differs to TGE virus, it was considered that there was a 'high' likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

No information is available concerning the effect of temperature changes on PRCV viability. As stated above PRCV is very closely related to TGEV. It is known that TGE virus is stable for

prolonged periods when frozen, although infectivity decreased at 4°C following 180 days of storage. Thus it was considered that there was a 'high' likelihood that meat infected or contaminated with PRCV at the completion of carcass maturation would remain infected during transport and storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was an 'extremely low' likelihood that imported pig meat derived from an individual carcass would be infected with PRCV.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'extremely low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

PRCV has a tropism for tissues of the respiratory tract, which are removed at slaughter. Limited information is available on the oral infectious dose of PRCV. An experimental study demonstrated that pigs could be infected with PRCV via direct gastric inoculation using a high viral dose ($10^7 TCID_{50}$). If meat is infected this is likely due to contamination with blood when the pig is viraemic or associated lymphoid tissue. PRCV has been isolated from the inguinal lymph node of one pig 3 days post-inoculation at a titre $10^{2.2} TCID_{50}$/g but was unable to be isolated from the inguinal lymph nodes of other pigs on days 1, 2, 4, 5 and 6 post-inoculation nor from the cervical lymph nodes from days 1 to 6. Virus has been isolated from plasma samples of pigs euthanased from 2 days after inoculation at titres of $10^3 TCID_{50}$/g (Cox, et al., 1990b).

Given the above information, the likelihood that a waste unit from an infected pig would contain a sufficient dose of PRCV to initiate infection was considered 'extremely low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

This step describes the sensitivity of PRCV to ultraviolet light, to ambient temperatures ranging from 10°C to 35°C and to the putrefying effects of saprophytic organisms. Although specific information is unavailable for PRCV, data are available for TGE virus. Transmissible gastroenteritis virus is very susceptible to sunlight and is somewhat labile at room temperature and above. As PRCV is very closely related to TGE virus, having an overall nucleotide and amino acid sequence homology of 96%, the biological properties of TGE virus were considered applicable for PRCV.

Given this, the Panel considered that the likelihood that PRCV would remain viable after exposure to sunlight, putrefaction and the environment in meat scraps discarded in refuse for the period of time required for pigs to locate and subsequently scavenge the material was 'moderate'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = Extremely low
- Rural regions = Extremely low
- Large towns = Negligible

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be 'extremely low'.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was an 'extremely low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'extremely low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a 'high' likelihood that PRCV would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'negligible'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was an 'extremely low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was ‘extremely low’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was ‘high’ likelihood that PRCV would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be ‘extremely low’.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete ‘outbreak scenarios’ for each of the three exposure groups - that is, feral pigs, pigs in backyard enterprises and pigs in small commercial piggeries. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of ‘consequences’ for that scenario;

- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

PRCV is highly contagious and the virus spreads rapidly between, and within, herds via (predominantly) an aerogenous route. Infection with PRCV is often subclinical. However, signs of mild to (occasionally) moderate respiratory disease have been reported.

Feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs, however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: very low

Scenario 2: moderate

Scenario 3: low

Scenario 4: moderate

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;

3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

As discussed above, it is feasible that backyard pigs kept in rural areas may come in close contact with nocturnally foraging feral pigs, and that transmission of PRCV from one group to the other may result. It is also feasible that some mixing between pigs from an infected backyard herd and other domestic pigs may occur. For example, in the case of speciality breeds or unusual breeds live pigs or semen may be transferred from one herd to another for breeding purposes. Alternatively, pigs raised for personal consumption may be transferred between backyard holdings for growing out or fattening. However, often backyard pigs will be raised for consumption by that household.

It was stated above that were transmission to a piggery to occur, it is likely that the disease would be amplified and spread regionally by live pigs or other means to other piggeries. If large commercial piggeries were also situated within the region, it is conceivable that spread to these might occur, and that this would subsequently lead to a more general outbreak. Given this, it was considered unlikely that PRCV would be contained within a single directly exposed backyard pig herd, and unlikely that it would spread from an initially exposed backyard pig herd to a population of feral pigs, without involving other backyard or commercial pig herds.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: low

Scenario 4: moderate

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the ‘no outbreak’ scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and

4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios. An important consideration was the larger number of live pigs moved between small commercial piggeries than backyard enterprises.

As there may be no clinical evidence of disease or only very mild respiratory signs which may not be investigated, it is likely that the disease could be come widespread. The presence of PRCV infection within a herd may only be suspected when serum neutralisation tests for TGE virus (possibly required for export of live pigs or semen) are positive in the absence of any signs of infection, as the viruses cross-neutralise. Overseas, PRCV spread extensively in domestic pigs, and in areas with high pig densities, the virus has spread to pigs on neighbouring farms several kilometres away.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: very low

Scenario 2: very low

Scenario 3: low

Scenario 4: high

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this scenario, PRCV would have established in the directly exposed animal, or group of animals, but would not have spread to other animals or to humans. This 'no outbreak' scenario would have resulted from low probability of contact between infected and susceptible animals, rather than from human intervention. Additionally, because the disease is of low pathogenicity, and does not infect humans or animals other than domestic or wild pigs, it would not, under a 'no outbreak' scenario, have any discernible direct or indirect impacts.

On this basis, a rating of 'A' was assigned to each of the direct and indirect criteria.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, the disease is most likely to have established within a population of feral pigs, and to have run its course without identification. As explained above, this is because the clinical symptoms of this disease are very mild. Additionally, because the disease is not highly pathogenic, and does not infect humans or animals other than domestic or wild pigs, it would not, under this scenario, have any discernible direct or indirect impacts.

On this basis, a rating of 'A' was assigned to each of the direct and indirect criteria.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, the disease is most likely to have established within a local population of small commercial piggeries or backyard enterprises, and to have run its course without identification. Once again, this is because the clinical symptoms of this disease are not distinct.

The direct impact of PRCV infection

Animal life or health

The third scenario is characterised by spread of PRCV to a local population of domestic pigs, but containment within this population. Although PRCV spreads easily within a herd, infection is often subclinical. Clinical signs if present, may consist of mild to (occasionally) moderate signs of respiratory disease.

On the basis of this, the likely impact of PRCV in terms of *animal health* was considered unlikely to discernible except at the local level. This resulted in a rating of 'B' for this criterion.

Environment

Because PRCV is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of PRCV infection

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this outbreak scenario, although there is spread to a local population of pigs in backyard or small commercial enterprises, it is unlikely that PRCV would be diagnosed due to the subclinical nature of the disease. Given this, it was considered that the likely indirect impact of new or modified control programs would be undiscernible at any level, and the rating assigned to this criterion was therefore 'A'.

Domestic trade or industry effects

As described above, it was considered unlikely that PRCV would be detected in this outbreak scenario. On this basis, the indirect impact of PRCV on domestic trade and industry was

considered unlikely to be discernible at any level, and thus the rating assigned to this criterion was ‘A’.

International trade effects

As above, the indirect effect of PRCV on international trade was unlikely to be discernible at any level, hence this criterion was rated ‘A’.

Indirect impact on the environment

PRCV in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of ‘A’ was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of ‘A’ was thus assigned to this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, the disease would have been spread through pig movements within Australia to a more general population of domestic and feral pigs. Due to the mild or subclinical nature of this disease, it is unlikely that an eradication or control program would be implemented.

The direct impact of PRCV infection

Animal life or health

Infection with PRCV is often subclinical. When present, mild to (occasionally) moderate signs of respiratory disease may be observed.

On the basis of this, the likely impact of PRRS in terms of *animal health* was considered unlikely to be discernible at any level except the local level. This resulted in a rating of ‘B’ for this criterion.

Environment

Because PRCV is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of ‘A’ for this criterion.

The indirect impact of PRCV infection

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

If PRCV were to be identified in Australia, control and eradication programs are unlikely to be implemented due to the subclinical nature of PRCV infection and minimal affect the disease appears to have on pig health and production. In addition PRCV is likely to be widespread when diagnosed. PRCV is not listed under the Cost Sharing Agreement, hence there would be

no compensation should eradication be undertaken. On this basis, it was considered that the indirect impact of new or modified control programs would be unlikely to be discernible at all levels, giving the disease a rating of 'A' for this criterion.

Domestic trade or industry effects

The diagnosis of PRCV in Australia is unlikely to affect domestic trade in pig meat, live pigs or genetic material nor have an affect on the pig industry or associated industries. Other disease agents diagnosed in Australia, such as porcine circovirus and *Eperythrozoon suis*, where disease is mild or subclinical, have not resulted in discernible effects on industry.

When these issues were taken into account, the indirect impact of PRCV on domestic trade and industry was considered unlikely to be discernible at any level. This resulted in a rating of 'A' for this criterion.

International trade effects

The diagnosis of PRCV in domestic pigs in Australia is unlikely to affect international trade in either live pigs or semen, as PRCV is widespread within the world's pig-producing countries and is not regarded as a significant pathogen. Serological tests are available to differentiate infection with TGE virus and PRCV.

Thus, the indirect effect of PRCV on international trade was considered unlikely to be discernible at all levels, resulting in a rating of 'A' for this criterion.

Indirect impact on the environment

PRCV in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

The overall impact of PRCV

When the direct and indirect impacts of PRCV were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences negligible

Scenario 3: Consequences negligible

Scenario 4: Consequences negligible

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 84, Table 85 and Table 86. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were considered 'negligible', 'negligible' and 'negligible' respectively.

Table 84 PRCV: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Very low	Negligible	Negligible
Scenario 2	Moderate	Negligible	Negligible
Scenario 3	Low	Negligible	Negligible
Scenario 4	Moderate	Negligible	Negligible
		Overall likely consequences	**Negligible**

Table 85 PRCV: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Low	Negligible	Negligible
Scenario 4	Moderate	Negligible	Negligible
		Overall likely consequences	**Negligible**

Table 86 PRCV: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Very low	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Low	Negligible	Negligible
Scenario 4	High	Negligible	Negligible
		Overall likely consequences	**Negligible**

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with PRCV.

Table 87 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly less than Australia's ALOP (very low), risk management would not be required for PRCV.

Table 87 PRCV: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Extremely low	Extremely low	Negligible	Negligible
Backyard pigs	Extremely low	Negligible	Negligible	Negligible
Small commercial piggeries	Extremely low	Extremely low	Negligible	Negligible
			Overall annual risk	**Negligible**

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Porcine rubulavirus

Technical information

Background

Rubula, ‘rubalo’, ‘blue eye disease’ or ‘Mexican blue eye disease’ is a disease of pigs first reported in 1980 and characterised by central nervous system disorders, reproductive failure, neonatal mortality and corneal opacity. Although clinical disease occurs only in central Mexico, seropositive animals have occasionally been found in other parts of that country (Stephan, et al., 1988). It is not known how the virus entered the pig population but it appears that pigs had been infected with the virus for some years before it was first reported as antibodies were present in sera collected between 1972 and 1980 (Morilla, et al., 2002).

Agent taxonomy

Porcine rubulavirus, also called La-Piedad-Michoacan virus, is an enveloped single negative stranded sense RNA virus of genus Rubulavirus, subfamily Paramyxovirinae, family Paramyxoviridae, order Mononegavirales.

Agent properties

Little is known about the stability of the virus in the environment. Porcine rubulavirus is sensitive to lipid solvents and is inactivated at 56°C after 4 hours. No morphological, physicochemical, or serological differences have been observed among different strains (Stephan, et al., 1988; Stephano, 1999).

Host range

Pigs are the only known natural hosts for porcine rubulavirus, although the virus has infected mice, rats and chick embryos under experimental conditions. Experimentally rabbits, cats and peccaries produced antibodies to the virus but did not show clinical signs (Stephano, 1999). Vampire bats do not appear to be hosts (Solis-Hernandez, et al., 2002b).

Epidemiology

Natural infection with porcine rubulavirus is acquired by inhalation. Typically, naïve pig herds become infected when subclinically infected pigs are introduced. Three general patterns of herd infection have been observed:

- In the first and most common instance, sows were infected after coming into contact with introduced infected gilts or boars. The antibodies produced by the sows provided passive immunity for the piglets until they were 1 month old when they in turn became infected.
- In the second instance, on some farms, nearly 100% of pigs were seropositive.
- In the third instance, sows were infected and maternal antibodies were found in piglets until 3 months of age - after this, growers and fatteners remained seronegative, indicating infection was no longer active in these age groups (Morilla, et al., 2002).

Overall, the results suggested that sows are likely to be reservoirs of the virus, and that pigs during the growing period are amplifiers.

While the virus can be recovered from frozen semen of mature boars, transmission through semen has not been proven (Stephano, 1999; Solis-Hernandez, et al., 2002a). There is some evidence of persistence of porcine rubulavirus in the central nervous system of recovered pigs, through the detection of viral RNA at 53 days post-inoculation, although it is not known whether subsequent spread of virus to other animals can occur (Wiman, et al., 1998). By virus isolation techniques porcine rubulavirus has been detected in blood between 6 to 15 days post-inoculation (Hernandez, et al., 1998) and in tissues up to 11 days but not at 14 days post-inoculation (Allan, et al., 1996).

Clinical signs are most apparent in farms with poor disease or biosecurity management. The disease appears to be self-limiting in closed herds (Stephano, 1999) and elimination of porcine rubulavirus has been achieved in some herds by adopting sound biosecurity management practices. It has been observed that when clinical disease was present, other pathogens were often also present; particularly porcine reproductive and respiratory syndrome virus, Aujeszky's disease virus and swine influenza virus (Morilla, et al., 2002).

During the 1990s, antibodies to porcine rubulavirus were reported in 20 Mexican States (Morilla, et al., 2002). While there is no official control program against porcine rubulavirus, the ongoing classical swine fever eradication program has restricted pig movements between several States, thus restricting the potential spread of porcine rubulavirus. Clinical disease has been reported only in the seven States of central Mexico (Guanajuato, Hidalgo, Jalisco, Estado de Mexico, Michoacán, Querátero and Tlaxcala).

A national survey demonstrated 20.32% of pigs (4433 of 21818) in central Mexico were seropositive compared with 0.35% of pigs (20 of 5701) in the rest of Mexico (Morilla, et al., 2002). A similar trend was found in another survey where 11.02% of pigs (1037 of 9413) in central Mexico were seropositive compared with 1.08% of pigs (24 of 2221) elsewhere in Mexico (Mercado and others as quoted by (Morilla, et al., 2002)). Other prevalence data include 64% of pigs (120 of 188) from ten farms in central Mexico seropositive to the haemaglutination inhibition assay whereas none of 174 pigs from ten farms in north Mexico was positive (Carreon Napoles & Fuentes Rangel, 1991). Of 495 pigs from five farms in central Mexico, 288 (58.2%) were positive to the blocking ELISA (Gonzalez-Vega, et al., 2002). Within herd seroprevalence of porcine rubulavirus on some farms in central Mexico can be very high, up to nearly 100% (Morilla, et al., 2002).

Clinical signs

Clinical signs, if present, generally develop 5 to 6 days following infection. The signs can be variable, and appear to depend on the age of pigs, type of herd, production system, management or presence of other infections (Stephano, 2002). Clinical signs were observed in 30% of seropositive herds (Morilla, et al., 2002).

Clinical signs include fever, inappetence, corneal opacity with occasional conjunctivitis, nervous signs manifesting as fits and convulsions, adopting a dog sitting position, increased time of return to service in sows, increased weaning to mating intervals, stillbirths, mummified piglets, high mortality in piglets, and swollen testicles and loss of libido in boars. Between 29% and 73% of boars can become temporarily or permanently infertile or aspermic (Stephano, 1999).

Piglets are most susceptible to infection, with the virus producing acute meningoencephalitis in sucklings. During clinical outbreaks, 10% to 65% of litters can be affected. Within the affected litters, morbidity is between 20% and 50%, and mortality of the affected piglets is high. Most

sows with affected litters are clinically normal, but some become anorexic for 1 or 2 days prior to the appearance of clinical signs in their piglets. In pigs older than 30 days only 1% to 4% are affected as described above and mortality is low. However, on poorly managed piggeries with serious concurrent disease problems, mortality rates of 20% have been observed in grower pigs and corneal opacity reported in 30% of pigs (Stephan, et al., 1988).

Pathogenesis

Infection is initiated when the virus is adsorbed to a host cell with a specific saccharide structure. This structure is distributed widely in cells in the CNS and respiratory tract of newborn pigs. As the animal matures, the saccharide structure becomes restricted to the sexual organs and portions of the respiratory system in adults. Thus, infection in the very young animals affects mainly the respiratory system and the CNS, resulting in death, while infection in adults is selective for the reproductive organs (Espinosa, et al., 2002).

Pathology

Gross lesions are usually not apparent in infected pigs of slaughter-age. Corneal opacity generally occurs in only 1% to 4% of infected pigs. In boars, swollen testicles may be seen. Mild pneumonia may occasionally be seen (Stephano, 2002).

Immunology

Serum-neutralising antibody is detected as early as 1 week post-infection, although highest titres are not produced until after 4 weeks post-infection. Antibody inhibiting the viral haemagglutinating activity can be detected 2 weeks post-infection (Hernandez, et al., 1998). Antibody in naturally infected pigs usually persist throughout their lives (Stephano, 1999).

Transmission via meat

Porcine rubulavirus can be recovered from, or demonstrated in, the trachea, nasal mucosa, lungs, tonsils, and a range of central nervous system tissues for up to 11 days post-infection. Occasionally the virus was isolated from some lymph nodes including the parotid and retropharyngeal lymph node. The virus has not been found in spleen, liver, bone marrow or muscle even during viraemia (Allan, et al., 1996). The virus titres obtained from nasal mucosa were, in nearly all cases, less than $10^{2.0}$ $TCID_{50}$/0.1ml (Allan, et al., 1996). The highest titre of virus was found most consistently from the tonsil, with a maximum titre of $10^{2.5}$ $TCID_{50}$/0.1ml, and from the olfactory bulb, with a maximum titre of $10^{4.5}$ $TCID_{50}$/0.1ml (McNeilly, et al., 1997). Virus titres of up to $10^{1.0}$ $TCID_{50}$/0.1ml were found in the parotid lymph node.

It is not known if pigs can be infected orally with porcine rubulavirus. In the field transmission is considered to occur via inhalation. Under experimental conditions pigs have been infected with porcine rubulavirus by intranasal and intraconjunctival inoculation routes with $10^{7.0}$ $TCID_{50}$ (Allan, et al., 1996; McNeilly, et al., 1997). In another study pigs were intranasally inoculated with 5 ml of porcine rubulavirus of 10^{4} $TCID_{50}$/ml (Hernandez, et al., 1998).

Release assessment

R1 — the likelihood that a source herd is infected

Disease due to porcine rubulavirus occurs only in the intensive pig production areas of the central plateau area of Mexico, although antibodies to the virus have been reported in 20 States. Although between herd prevalence has not been reported, two extensive surveys in central Mexico where seroprevalence is the highest, have reported figures of 11% and 20% of pigs seropositive. On balance, it was considered that, in rubulavirus endemic areas the likelihood of selecting slaughter-age pigs from an infected herd was 'low'.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

Some pig farms have reported high seroprevalence of up to 100%. Epidemiological studies have indicated that generally pigs become infected soon after losing the protective effects of maternal antibodies at about 1 month of age. Viraemia is relatively short, being of 1 to 2 weeks. One study demonstrated viral RNA in the mid brain at 53 days post-infection, however, it is unknown if this could lead to spread of the virus to other pigs. Nonetheless it is reported that the disease is self limiting in closed herds.

Given the above, it was considered that the likelihood of selecting an infected animal in an infected herd was 'low'.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

Although clinical symptoms of infection with porcine rubulavirus can include fever, inappetence, corneal opacity with occasional conjunctivitis, nervous signs manifesting as fits and convulsions, these are most frequently seen in piglets. Slaughter-age pigs are generally asymptomatic. In addition, gross lesions are not often apparent.

In view of this, the sensitivity of ante-mortem or post-mortem inspection was considered 'extremely low'.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with porcine rubulavirus and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a 'one minus the background rejection rate'. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered 'extremely low'. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

Porcine rubulavirus has not been demonstrated in, or isolated from, muscle and bone marrow, even during viraemia. Virus has been recovered from tonsils, portions of the spinal cord and from some lymph nodes such as the parotid and retropharyngeal lymph nodes of infected pigs. Most tonsillar tissue will be removed at slaughter.

In view of this, the likelihood that porcine rubulavirus would be present in meat harvested for export from an infected pig was considered 'low'.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

There is no published information on the effect of pH on porcine rubulavirus. However, other paramyxoviruses, such as rinderpest virus or Newcastle disease virus, are not inactivated at the pH (approximately pH 6.2) that accompanies carcass maturation (Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 2000). On this basis, it was considered that there was a 'high' likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

There is no information on the effects of cold storage on porcine rubulavirus. However, paramyxoviruses are known to remain viable for long periods when kept at low temperatures. Rinderpest virus, for example, can survive in culture for at least 4 months at -20°C, 8 weeks at 4°C, and 1 week at 20°C to 25°C (Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 1996). In light of this, it was considered that there was a 'high' likelihood that meat infected at the completion of carcass maturation would remain infected during transport and cold storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a 'very low' likelihood that imported pig meat derived from a carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

There is no information on virus titres in carcasses. Porcine rubulavirus has not been isolated

from muscle or bone marrow. Virus titres in the parotid lymph node were low, generally less than $10^{1.0}$ $TCID_{50}$/0.1ml. It is unknown if pigs can be infected orally and no reported evidence that they can. Experimentally pigs have been infected using high doses of virus ($10^{7.0}$ $TCID_{50}$) by intranasal and intraconjunctival inoculation. Given that the bulk of the carcass consists of muscle and bone, the likelihood that a waste unit will contain a sufficient dose of the pathogenic agent to initiate infection was estimated to be 'extremely low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

This step describes the sensitivity of porcine rubulavirus to ultraviolet light, to ambient temperatures ranging from 10°C to 35°C and to the putrefying effects of saprophytic organisms, bearing in mind that some time may be required before meat scraps are located by scavenging feral pigs.

Although there is no information on the impact of these factors on the viability of porcine rubulavirus virus, other members of the paramyxovirus family are known to be readily inactivated by heating, ultraviolet light and desiccation (Bellini, et al., 1998). Given this, the likelihood that porcine rubulavirus would survive within meat scraps discarded in refuse for the period of time required for feral pigs to locate and subsequently scavenge the material was considered 'very low'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = Extremely low
- Rural regions = Extremely low
- Large towns = Negligible

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be 'extremely low'.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of porcine rubulavirus to initiate infection was 'extremely low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage. On balance, the likelihood that porcine rubulavirus would remain viable during the period prior to ingestion was considered 'low'.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'extremely low'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

It was shown in the exposure assessment for feral pigs that there is an 'extremely low' likelihood that a waste unit from an infected pig would contain a sufficient dose of porcine rubulavirus to initiate infection.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage. On balance, the likelihood that rubulavirus would remain viable during the period prior to ingestion was considered 'low'.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently (Table 7) and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'extremely low'.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the three exposure groups - that is, feral pigs, pigs in backyard enterprises and pigs in small commercial piggeries. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);

- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

There are no published reports of porcine rubulavirus in feral pigs (the so-called, Mexican hairless boars) in central Mexico, although they have been infected experimentally (Soto, et al., 2002; Ramírez, et al., 2002). This is likely to reflect the low probability of adequate contact between infective domestic pigs and feral pigs. However, factors determining the movement of this disease amongst porcine populations are not well understood - in particular, it is unclear why, with the movement of large numbers of pigs throughout Mexico prior to movement restrictions for classical swine fever, clinical disease has not been reported outside the Mexican central plateau.

In Australia, feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs. However, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: high

Scenario 2: low

Scenario 3: very low

Scenario 4: extremely low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for close contact between backyard pigs and either feral pigs or other domestic pigs, and by the likelihood that the disease would be identified early, and a control or eradication program initiated.

Although some pigs raised in backyards for domestic consumption may be transferred between farms, many will remain in a single pen and in relative isolation from other domestic or feral pigs. In addition it would appear that pigs only excrete virus for a short period, up to 2 weeks. For these reasons, there would be limited opportunity for spread of porcine rubulavirus by direct contact amongst backyard pig operations, and either commercial or feral pigs. Immediate diagnosis of index cases, if clinical signs are evident, is unlikely, as backyard pig producers are less likely to seek veterinary advice - indeed, the most likely event is for this self-limiting disease to establish and die out within the exposed backyard herd, without being recognised. Nonetheless if infected pigs are moved from a backyard premises to other premises spread of the disease could occur.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: high

Scenario 2: extremely low

Scenario 3: low

Scenario 4: low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;

2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for close contact between commercial pigs and either feral pigs or other domestic pigs, and by the likelihood that the disease would be identified early, and a control or eradication program initiated.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios. In particular, pig production at small commercial piggeries is generally more intensive, and includes a continual flow of pigs in and out of the facility. Most pigs from a small commercial piggery will go directly to slaughter, although some may be purchased as stores or breeders for other commercial piggeries or for backyard piggeries. It is also relevant that managers of small commercial piggeries are more likely to observe and report unusual illness to a veterinarian, and that exotic disease event are likely to be identified earlier than might be the case for backyard piggeries.

Aside form corneal opacity, the clinical symptoms of rubula are not generally distinct, and might initially be confused with endemic diseases. It is feasible, therefore, that the disease could run its course within an isolated commercial piggery, and die out without spread to other piggeries. Alternatively, the disease could have limited spread amongst piggeries that have received adult pigs (or possibly semen) from an infected piggery. However, because it is not known why this disease did not spread significantly beyond the Mexican central plateau during the period of high stock movements throughout Mexico, it is difficult to estimate likely spread in Australia with any precision.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: extremely low

Scenario 3: moderate

Scenario 4: low

Estimating the consequences associated with each outbreak scenario

For each exposure group, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the

assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed primary exposure group - no secondary spread

Under this scenario, the disease is most likely to have established amongst exposed animals, and to have run its course without identification. This is because the clinical symptoms of this disease are not distinct, and because it is often self-limiting within a discrete population. Additionally, because the disease is not highly pathogenic, and does not infect humans or animals other than domestic or wild pigs, it would not, under a 'no outbreak' scenario, have any discernible direct or indirect impacts.

On this basis, a rating of 'A' was assigned to each of the direct and indirect criteria.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, the disease is most likely to have established within a population of feral pigs, and to have run its course without identification. As explained above, this is because the clinical symptoms of this disease are not distinct, and because it is often self-limiting within a discrete population. Additionally, because the disease is not highly pathogenic, and does not infect humans or animals other than domestic or wild pigs, it would not, under this scenario, have any discernible direct or indirect impacts.

On this basis, a rating of 'A' was assigned to each of the direct and indirect criteria.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, the disease is most likely to have established within a local population of small commercial piggeries or backyard enterprises, and to have run its course without identification. Once again, this is because the clinical symptoms of this disease are not distinct, and because it is often self-limiting within a discrete population. Additionally, because the disease is not highly pathogenic, and does not infect humans or animals other than domestic or wild pigs, it would not, under this scenario, have any discernible direct or indirect impacts.

On this basis, a rating of 'A' was assigned to each of the direct and indirect criteria.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, the disease would have been spread through pig movements within Australia to a more general population of domestic and feral pigs. At this scale, the effects of the disease, and, in particular, increased piglet mortality, are likely to have been delineated from endemic diseases. This would have resulted in an eradication program based on testing and slaughter, and restrictions on the movement of animals from affected areas or piggeries.

The direct impact of porcine rubulavirus infection

Animal life or health

Porcine rubulavirus, if present in a larger commercial piggery, could have a significant effect on reproductive performance and piglet mortality, at least for some time. In those piggeries

where clinical disease was present, overall production would be reduced. Given this, the direct impact of rubulavirus on animal health was considered unlikely to be discernible at the national or State level, and of minor significance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

Environment

Because porcine rubulavirus is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of porcine rubulavirus infection

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

Porcine rubulavirus is not an OIE List A or B disease, and nor is it listed in the Australian Emergency Animal Disease Response Agreement. However, should it be detected in Australian pigs, it may be declared as an 'emerging disease', with some of the costs of any response program being shared between governments and industry.

If diagnosed, it is likely that porcine rubulavirus would be managed in a manner similar to Menangle virus, a closely related virus - i.e. eradication through movement restriction and the segregating of pigs into discrete age groups (Kirkland, et al., 2001). Pig movements would be traced forward and backwards, and all contact and at risk herds tested. If the virus was widespread within the pig industry, eradication may not be possible and its control may be managed by individual producers.

On balance, the indirect impact of control and eradication programs was considered unlikely to be discernible at the national or State level, but of minor significance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

Domestic trade or industry effects

The discovery of Menangle virus in three large intensive piggeries in Australia had no impact on domestic trade and a minor local impact on the pig industry - even though there was considerable scientific interest in the discovery. Given this, the indirect impact of porcine rubulavirus on domestic trade was considered unlikely to be discernible at any level, except locally. This gave the disease a rating of 'B' for this criterion.

International trade effects

As porcine rubulavirus is not an OIE List A or B disease, there are no official reporting obligations. However, if the disease were diagnosed, trading partners would be notified and be updated on disease control measures, including those affecting export of pigs and pig products. The presence of porcine rubulavirus in Mexico has not prevent export of pork to countries such as Japan and the United States of America. Given this, it was considered the indirect impact on international trade was unlikely to be discernible at any level and a rating of 'A' was given to this criterion.

Indirect impact on the environment

In this scenario, porcine rubulavirus is unlikely to lead to any indirect impacts on the environment such as affecting biodiversity, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

The overall impact of porcine rubulavirus

When the direct and indirect impacts of porcine rubulavirus were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences negligible

Scenario 3: Consequences negligible

Scenario 4: Consequences very low

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 88, Table 89 and Table 90. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were considered 'negligible', 'negligible' and 'negligible' respectively.

Table 88 Porcine rubulavirus: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	High	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Very low	Negligible	Negligible
Scenario 4	Extremely low	Very low	Negligible
		Overall likely consequences	**Negligible**

Table 89 Porcine rubulavirus: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	High	Negligible	Negligible
Scenario 2	Extremely low	Negligible	Negligible
Scenario 3	Low	Negligible	Negligible
Scenario 4	Low	Very low	Negligible
		Overall likely consequences	**Negligible**

Table 90 Porcine rubulavirus: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Extremely low	Negligible	Negligible
Scenario 3	Moderate	Negligible	Negligible
Scenario 4	Low	Very low	Negligible
		Overall likely consequences	**Negligible**

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;; and
- Combination of partial annual risks to give an estimate of 'overall annual risk'..

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with porcine rubulavirus.

Table 91 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly less than Australia's ALOP (very low), risk management would not be required for porcine rubulavirus.

Table 91 Porcine rubulavirus: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Very low	Extremely low	Negligible	Negligible
Backyard pigs	Very low	Extremely low	Negligible	Negligible
Small commercial piggeries	Very low	Extremely low	Negligible	Negligible
			Overall annual risk	**Negligible**

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Teschen disease (porcine teschovirus 1)

Technical information

Background

Teschen disease was first identified in Czechoslovakia in the 1930s. It is a disease of high morbidity and mortality and is characterised by nervous signs including progressive paralysis. However, of recent years the disease is reported only rarely, and its distribution appears limited to areas of central Europe and Africa (Derbyshire, 1989). Teschen disease is due to virulent strains of porcine teschovirus 1 (PTV-1) formerly classified as one of 13 serotypes of porcine enterovirus.

Less virulent strains of PTV-1, causing a sporadic and milder encephalomyelitis and known by a variety of names, including Talfan disease, porcine polioencephalomyelitis, enterovirus encephalomyelitis, or benign enzootic paresis, have been reported in several countries including England and Belgium (Harding, et al., 1957; Hoorens, et al., 1978). In most countries, including United States of America, Japan, Canada, Kenya, West Indies, Eire and Sweden, PTV-1 strains are usually avirulent and do not cause apparent disease (Huck, et al., 1962; Dunne, 1975)

Although Talfan disease has been diagnosed in, and PTV-1 has been isolated from, Australian pigs, Teschen disease has never been reported here (Hudson, 1962; Spradbrow, 1964; Forman, et al., 1982).

Agent taxonomy

Porcine teschovirus 1 is a member of the recently-defined teschovirus genus of the Picornaviridae family (Pringle, 1999).

Agent properties

The virus is a single-stranded, positive-sense, non-enveloped RNA virus. Previously, porcine enteroviruses were divided into at least 13 serotypes based on virus neutralisation tests and placed within the enterovirus genus of the Picornaviridae family (Kaku, et al., 1999). The serotypes were grouped into three categories (CPE I, II, III) according to their biological and biophysical properties, particularly the behaviour in cell culture (Knowles, et al., 1979). On this basis, the virus strains associated with pathogenic Teschen diseases were classified as porcine enterovirus 1 (PEV-1), in CPE group I. Within this classification, marked strain variations in pathogenicity exist, and many avirulent strains were included in this category. More recently, advances in molecular technology resulted in the reclassification of PEV-1 to PTV-1 as noted above. Again, strain variation in pathogenicity occurs within this grouping. Three main PTV-1 genotypes have been described, each of which contains neurotropic and non-neurotropic strains (Zell, et al., 2001). The genomes of several (but likely not all) strains of PTV-1 associated with Teschen disease have been fully or partially sequenced (Zell, et al., 2001), as have those of multiple avirulent or mildly virulent strains (Kaku, et al., 1999; Doherty, et al., 1999; Zell, et al., 2001; Kaku, et al., 2001).

As an enteric virus, PTV-1 is resistant to changes in pH and is reported (Derbyshire, 1989) to be stable within a pH range of 2.8 to 9.5 (this information was not referenced). The Teschen disease virus is reported to remain infective for 15 minutes at 60°C and for longer periods at 56°C (Derbyshire, 1989), but again no supporting data are provided. Little information is

available concerning the effect of decreasing temperatures on the virus, but other members of the family picornaviridae, such as the foot-and-mouth disease virus (genus apthovirus) and the swine vesicular disease virus (genus enterovirus), survive for prolonged periods when chilled or frozen (Cottral, 1969; Dawe, 1974). Overall, the virus is highly resistant to the environment and has been demonstrated to survive for more than 168 days in water at 15ºC (Ottis, 1976).

Host range

The host range is limited to the pig.

Epidemiology

Porcine teschoviruses (formerly porcine enteroviruses) are distributed widely throughout world pig populations (Derbyshire, 1999), including that of Australia (Forman, et al., 1982). However, based on the paucity of reports of clinical disease, Teschen disease virus appears to be restricted to areas of Africa and Eastern Europe (Office international des épizooties, 2003).

Outbreaks of Teschen disease are characterised by high morbidity and mortality in pigs of all ages (Derbyshire, 1989). Transmission is usually via the faeco-oral route although indirect transmission via fomites is considered likely due to the ability of the virus to persist in the environment (Derbyshire, 1989). The incubation period is around 7 to 8 days (Derbyshire, 1989), but is influenced by factors such as age and immunocompetency of the pigs, and degree of viral challenge.

Teschoviruses are usually introduced into piggeries by carrier pigs, especially by young gilts and boars, spreading the virus through contaminated faeces, and infecting other pigs by the faeco-oral route. Pigs excrete the virus for up to 8 weeks after infection (Derbyshire & Collins, 1972). Typically, in infected herds, once young pigs lose the protective effects of maternal antibodies at around 4 to 5 weeks of age, they become infected by the virus in the environment and shed the virus until 12 to 13 weeks of age. Pigs infected after weaning are capable of shedding the virus until they are about 6 months of age, while pigs that are infected as adults are seldom shedders of virus. Because the virus is relatively resistant, indirect transmission via fomites is highly likely. As the virus can be found in the semen of infected boars, venereal transmission is also a possibility (Derbyshire, 1999).

The virulent Teschen disease strains can cause polioencephalomyelitis of high morbidity and mortality (up to 70% to 90%) in pigs of all ages while Talfan disease produce polioencephalomyelitis of lower morbidity and mortality in young pigs only. In Belgium, 45 of 993 fattening pigs died in one outbreak of Talfan disease and 5 fattening pigs in a breeding herd of 60 sows died in another (Hoorens, et al., 1978).

Porcine teschoviruses are often reported to be ubiquitous wherever pigs are found, although as previously stated Teschen disease would appear to be rare. In a serosurvey where over 1000 pigs in south east England were tested, 69 of 72 herds (95.8%) were infected with PTV-1 of which nine herds were found to have all pigs tested seropositive. On average, 45.6% of porkers, 58.7% of baconers, and 63.7% of overweights tested were seropositive for PTV-1 (Huck, et al., 1962). In West Germany, 67.6% of 30 pigs tested were found to be infected with PTV-1. Also in Germany, a survey showed that of 224 pigs in 83 herds tested, 75% of adults and 60% of young pigs were seropositive to PTV-1 (Mayr, et al., 1971; Bibrack, et al., 1972). On a pig farm in Kansas, an untyped enterovirus was isolated from faeces of 54.6% of 282 pigs 5 to 6 weeks old and 73.9% of 219 pigs 9 to 10 weeks old (Beran, et al., 1958).

Porcine teschovirus 1 has been isolated from pigs in Australia. Eight of 13 serum samples collected from unselected pigs being slaughtered at a Victorian bacon factory were seropositive to PTV-1 while untyped enteroviruses were recovered from 18 of 33 faecal samples collected from baconers being slaughtered at a Brisbane abattoir (Hudson, 1962; Spradbrow, 1964).

There is no apparent seasonality in teschovirus infections.

Clinical signs

The incubation period for experimental exposure to teschoviruses is normally around 6 days but may vary from 4 to 28 days. The OIE Code states that the incubation period be 40 days. The incubation period for natural exposure to Teschen disease is not well documented but it generally takes 14 days for neurological signs to develop (Jones, 1975). Initial clinical signs of Teschen disease include diarrhoea, fever, anorexia and listlessness. Neurological signs follow soon after, and include locomotor ataxia, nystagmus and convulsions with loud squealing. Sometimes coma occurs. Paralysis usually ensues and the pig may adopt a dog-sitting posture or lie down in lateral recumbency, and stimulation by noise or touch may cause opisthotonus. Death due to asphyxiation arising from paralysis of the thoracic muscles commonly occurs within 3 to 4 days of onset of clinical signs (Watanabe, et al., 1971; Jones, 1975).

With milder forms of polioencephalomyelitis, as in Talfan disease, usually young piglets are the only animals affected and disease rarely progresses to complete paralysis. Nursing piglets and, on rare occasions, naïve older pigs may show mild nervous signs for up to 5 weeks after infection (Derbyshire & Collins, 1972).

Pathogenesis

Porcine teschoviruses generally infect pigs through the mouth and nose. Initial replication of the teschoviruses usually occurs in the tonsils and the intestinal tract, particularly the ileum and colon and their associated lymph nodes. However, it has not been clearly established which intestinal cells support the viral replication. Oral infection sometimes results in viraemia and the virus can be isolated from the faeces as early as 2 days post infection, and be consistently found in the faeces 5 to 6 days after exposure (Beran, et al., 1960). When viraemia occurs, as commonly happens with the virulent and sometimes the intermediate strains, the virus may be transiently found in a variety of tissues, including heart, lung, liver, kidney, spleen, adrenal and salivary glands (Beran, et al., 1960; Derbyshire, 1989). Evidence suggests the virus in the blood is the source of virus entering the central nervous system (CNS). Once within the CNS, the virus replicates in the neurones in ganglia and in capillary endothelial and glial cells, causing extensive neuronal damage. With avirulent strains, the virus usually does not replicate in extra-intestinal tissue and there is no viraemia, the virus replicating only in the intestinal tract (Derbyshire, 1989).

Pathology

With encephalomyelitis in Teschen disease or Talfan disease, there are usually no gross pathological lesions. There may be paralysed animals, some having evidence of muscle wasting in one or more limbs. The pathological changes are microscopically recognisable by the extensive neuronal damage the disease causes, however, it is not histologically possible to distinguish between Talfan disease and Teschen disease (Jones, 1975). In a study where pigs were experimentally infected with a Teschen disease strain, some post-mortem changes were noted. There was congestion of the small intestines and mesentery 3 to 4 days after infection,

spreading throughout the whole intestinal tract by day 10, as well as some slight brain oedema (Watanabe, et al., 1971).

Immunology

In young pigs with Teschen disease, virus neutralising antibodies first appear 4 to 6 days after infection and reach maximum titres in their terminal stages (Hájek, et al., 1971). In infected herds, piglets are usually immune against infection for the first 4 to 5 weeks due to passive immunity provided by antibodies in the milk of nursing sows (Dunne, 1975). As they lose their maternal immunity, these piglets become infected by the teschoviruses in the environment and develop neutralising antibodies 4 to 6 days later. The circulating antibodies can persist at fairly high levels in young pigs for at least 8 months and are probably maintained for life as most adult pigs have high antibody levels (Derbyshire & Collins, 1972; Derbyshire, 1999). Porcine teschovirus 1 strains causing mild disease can protect pigs against challenge with the virulent Teschen disease strains (Mayr, 1961; Huck, 1962).

Transmission via meat

The spread of Teschen disease has been linked to pig meat (European Commission Scientific Committee on Animal health and Animal Welfare, 1997). However, the reports appear to be anecdotal (Szent-Ivanyi, 1961). During viraemia, the virus may be transiently found in a variety of extra-intestinal tissues, including heart, lung, liver, kidney, spleen, adrenal, salivary glands and nervous tissues, particularly the brain and the upper spinal cord (Beran, et al., 1960; Derbyshire, 1989). Porcine teschovirus 3 can be consistently found in blood 5 to 6 days after oral exposure (Singh, et al., 1964) and can be detected in the blood of germfree pigs 2 days after oral infection but could not be isolated from extra-intestinal tissue 11 days after infection (Baba, et al., 1966). Concentration of virus recovered from extra-intestinal tissues of pigs that died of a porcine enterovirus infection ranged from $10^{1.5}$ to $10^{3.5}$ TCD_{50}/0.1 ml (Beran, et al., 1960).

The oral infectious dose is not known. Experimental infection of pigs with $10^{5.5}$ $TCID_{50}$ of an untyped enterovirus given orally did not result in disease but those given a high dose ($10^{8.2}$ $TCID_{50}$) orally developed clinical signs of alimentary infection (Beran, et al., 1960).

Release assessment

R1 — the likelihood that a source herd is infected

According to OIE Handistatus II, Teschen disease is reported only in Latvia, Ukraine, Uganda and Madagascar. All these countries vaccinate pigs against Teschen disease. Yet literature states porcine teschoviruses to be ubiquitous wherever pigs are raised. In countries where Teschen disease is endemic, sporadic outbreaks continue to occur (Derbyshire, 1999). It is likely less virulent and non-fatal strains of PTV-1 are also present, competing against the virulent strain in infecting pigs and providing some immunity against virulent infection, thus possibly masking the high morbidity and mortality characteristics of Teschen disease.

Given this, the likelihood that slaughter-age pigs have been selected from an infected herd where Teschen disease occurs, was considered ‘low’.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

Infection with Teschen disease results in high morbidity. Considering that, in herds infected with PTV-1, pigs generally become infected around 4 to 5 weeks of age and then excrete the virus for 8 weeks, most seropositive pigs upon reaching slaughter weight would no longer be infected. However, not all young pigs will become infected as soon as they lose their maternally derived immunity, some may become infected during the grower and fattening stage. On the basis of this information, it was considered that the likelihood of selecting an infected pig in an infected herd was 'low'.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

Initial clinical signs of Teschen disease include diarrhoea, fever, anorexia and listlessness. As these signs are generally mild and non-specific, it is likely that most of these animals will pass inspection. However, the neurological signs that follow would result in rejection of these pigs from slaughter, although it is considered unlikely that these animals would be presented. Gross pathological changes are not usually evident. Given this, the sensitivity of ante-mortem, slaughter and processing requirements in detecting and removing infected pigs was considered 'very low'.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with Teschen disease and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a 'one minus the background rejection rate'. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered 'extremely low'. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

While infected finisher pigs are known to excrete the virus for up to 8 weeks, the virus can be isolated from extra-intestinal tissues, namely tonsils, blood, and nervous tissues for up to 9 days, between 2 and 11 days after infection. In view of this, it is considered that the likelihood that the teschovirus would be present in the meat harvested from an infected pig was 'low'.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

As stated earlier, the porcine teschoviruses are relatively resistant to the environment, remaining stable between pH 3 to 9 for at least 4 hours. Thus, it was considered that there was a

'high' likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

Teschoviruses are typical of the picornaviruses, being relatively resistant to chilling or freezing. Infected tissues for teschovirus content have been stored at -20^0C until tested (Harding, et al., 1957; Baba, et al., 1966). Given this, it was considered that there was a 'high' likelihood that meat infected at the completion of carcass maturation would remain infected during transport and storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a 'very low' likelihood that imported pig meat derived from an individual carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

There are anecdotal reports of Teschen disease having been spread by pig meat or pig meat products. In the field transmission is considered to occur orally via a faecal-oral route. Most experimental infections involved intra-cranial inoculation, intra-nasal instillation or direct contact with infected pigs. Concentration of virus recovered from extra-intestinal tissues, namely tonsils, blood, and nervous tissues, ranged from $10^{1.5}$ to $10^{3.5}$ $TCID_{50}$/0.1 ml of tissue. In order for clinical signs to develop under experimental conditions, pigs were given $10^{8.2}$ $TCID_{50}$ of an enterovirus orally. A dose of $10^{5.5}$ TCD_{50} did not cause clinical signs. Hence, the likelihood that a waste unit from an infected pig would contain a sufficient dose of PTV-1 to initiate infection was considered 'very low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

Teschoviruses are readily destroyed when exposed to sunlight for several hours. Meat scraps may be covered by other refuse, thus partially protecting the virus from sunlight. The virus can survive at room temperature (12.5°C to 25°C) for a long period, being 50% inactivated by 90 days and still retaining some viability at 150 days. Thus, the likelihood that PTV-1 would survive within meat scraps discarded in refuse for the period of time required for feral pigs to locate and subsequently scavenge the material was estimated to be 'moderate'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = Low
- Rural regions = Very low
- Large towns = Extremely low

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be ‘low’.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a ‘very low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was ‘very low’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances uncooked pig meat may be discarded due to spoilage.

Overall, the Panel considered that there was a ‘high’ likelihood that PTV-1 would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be ‘extremely low’.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a ‘very low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected would contain a sufficient dose of the pathogenic agent to initiate infection was ‘very low’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in

small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances uncooked pig meat may be discarded due to spoilage.

Overall, the Panel considered that there was a 'high' likelihood that PTV-1 would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'low'.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the three exposure groups - that is, feral pigs, pigs in backyard enterprises and pigs in small commercial piggeries. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis, but for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;

2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

There are no reports of surveys for enterovirus infections in feral pigs. Porcine enterovirus serotype 8 antibodies were reported in 13 of 20 feral swine in Kansas and were similar to the prevalence found in commercial herds in the State (Gipson, et al., 1999). In light of this, it is feasible the prevalence of PTV-1 in Australian feral pigs is similar to that in commercial herds. The only report describing the prevalence of teschovirus in Australia is that where 8 of 13 baconer pigs slaughtered in a Victorian abattoir had antibodies to PTV-1 (Hudson, 1962). Although PTV-1 has been isolated from Australian pigs, Teschen disease has never been reported here. Sporadic outbreaks of less virulent teschovirus encephalomyelitis have been reported in commercial pigs but not in feral pigs. Should Teschen disease occur in Australia, it may not be accompanied by high morbidity and mortality because of widespread immunity against endemic strains of PTV-1 which may also be protective against Teschen disease. The pre-existing immunity may even cause the disease to die out. In view of these factors, it is considered that not only is Teschen disease not likely to be diagnosed in feral pigs, but it may not spread from the locally infected herd.

Feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. Although pigs can excrete the virus for up to 8 weeks in their faeces, pigs usually defaecate in areas away from their living quarters and other pigs are at less risk of becoming infected. As it is not likely the disease would be diagnosed in feral pigs, the disease is likely to spread slowly amongst the general feral pig population. Feral pigs are widespread in Australia and there is very limited close contact with domestic pigs, however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: moderate

Scenario 3: low

Scenario 4: very low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the ‘no outbreak’ scenario;

2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

As discussed earlier, Teschen disease spreads by movement of carrier pigs. It is feasible that backyard pigs kept in rural areas may come in close contact with nocturnally foraging feral pigs and that transmission may result. It is also feasible that some mixing between pigs from an infected backyard herd and other domestic pigs may occur. For example spread may occur when several local farms share an infected boar or where pigs are moved between backyard holdings for growing out or fattening.

Because of widespread immunity against PTV-1, Teschen disease may occur only sporadically and may not be diagnosed in backyard herds, particularly where the herd only consists of fattening pigs for private consumption. In small breeding herds disease may be investigated after a period of time due to symptoms of polioencephalomyelitis, but Teschen disease is likely to occur sporadically mainly in young pigs which have not had time to develop immunity against the endemic avirulent strains and consequently infection may be misdiagnosed as Talfan disease although veterinary attention is not always sought. Where there are few movements of pigs from infected herds, with pigs consumed on the farm, the disease may die out without spreading.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: very low

Scenario 3: moderate

Scenario 4: low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the ‘no outbreak’ scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;

3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

As discussed earlier, the pre-existing widespread immunity against PTV-1 is likely to result in Teschen disease occurring sporadically in young pigs if introduced. Thus, when veterinary attention is sought, the disease may not be accurately diagnosed, being misdiagnosed as Talfan or other causes of nervous conditions in pigs such as poisoning. Where there are no movements of pigs from infected herds to other herds, the disease may die out without spreading. However, sows and boars from small commercial herds are often sold to other herds via local and regional saleyards, facilitating disease spread.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: moderate

Scenario 4: low

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this scenario, the disease is most likely to have established amongst the directly exposed animals, and to have run its course without identification. This is because the clinical symptoms of this disease are not distinct and may be confused with endemic diseases.

The direct impact of Teschen disease

Animal life or health

Outbreaks of Teschen disease are normally characterised by high morbidity and mortality. In this scenario, the disease has only affected the directly exposed herd, hence the direct impact on animal health was considered unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

Environment

Because Teschen disease is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of Teschen disease

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

As discussed earlier, it is considered unlikely Teschen disease would be diagnosed in the directly exposed group. Should veterinary attention be sought, the cases occurring mainly in young pigs are likely to be diagnosed as porcine encephalomyelitis due to endemic strains of PTV-1. Given this, it was considered unlikely that the indirect impact of new or modified control programs would be discernible at any level, and a rating of 'A' was assigned to this criterion.

Domestic trade or industry effects

As discussed above, it was considered unlikely that Teschen disease would be diagnosed in a single herd. On this basis, the indirect impact of the virulent strain of PTV-1 on domestic trade and industry was considered unlikely to be discernible at any level, and a rating of 'A' was thus assigned to this criterion.

International trade effects

As the disease is unlikely to be diagnosed, it was considered that the indirect impact of Teschen disease on international trade was unlikely to be discernible at any level, and a rating of 'A' was therefore assigned to this criterion.

Indirect impact on the environment

In this scenario, Teschen disease is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, the disease is most likely to have established within a population of feral pigs, and to have run its course without identification.

The direct impact of Teschen disease

Animal life or health

In this scenario, there is secondary spread to a more general population of feral pigs. It is likely that any outbreak of Teschen disease amongst the general population of feral pigs may be sporadic, affecting mainly young pigs. Young feral pigs already have a poor survival rate and the increased losses are not likely to be noticeable. Hence, the direct impact on animal health is not likely to differ from scenario 1 as discussed above and a rating of 'B' was assigned to this criterion.

Environment

Because Teschen disease is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of Teschen disease

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

The consequences on this criterion would be similar to that described for the first scenario, resulting in a rating of 'A'.

Domestic trade or industry effects

As discussed above, it was considered unlikely that Teschen disease would be diagnosed in the general population of feral pigs. On this basis, the indirect impact of the virulent strain of PTV-1 on domestic trade and industry was considered unlikely to be discernible at any level, and thus the rating assigned to this criterion was 'A'.

International trade effects

As described above, the indirect effects of Teschen disease were considered unlikely to be discernible at any level, and a rating of 'A' was therefore assigned to this criterion.

Indirect impact on the environment

In this scenario, Teschen disease is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, Teschen disease has established within a local population of small commercial piggeries or backyard enterprises. The disease would be contained through the diagnosis of disease in pigs, and the mounting of an eradication program.

The direct impact of Teschen disease

Animal life or health

Teschen disease has been reported to cause deaths in 70% to 90% of young pigs. However, it is possible that if the local population of pigs in small commercial piggeries or backyard enterprises had previously been exposed to PTV-1 mortalities may be less. Given this, it was considered that the direct effect on animal health would be unlikely to be discernible at the national or State level, but of minor significance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

Environment

Because Teschen disease is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of Teschen disease

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

The occurrence of Teschen disease in several local herds of pigs are likely to be thoroughly investigated by veterinarians. Teschen disease is a Category 4 disease under the Emergency Animal Disease Response Agreement, that is, it is classified as being a mainly production loss disease with perhaps some international trade losses and local market disruptions but not of a magnitude to significantly affect the national economy. Under a Category 4 disease, costs of eradicating the disease are to be funded 20% by governments and 80% by industry. There is no AUSVETPLAN strategy for this disease, however, all infected and surrounding pig herds are likely to be quarantined, and infected herds slaughtered to eradicate the disease. Movement controls would be implemented. Extensive tracing would be undertaken and surveillance, although the presence of PTV-1 in Australia would make serosurveillance difficult.

On balance, the impact of new or modified control programs was considered unlikely to be discernible at the national or State level, and of minor significance at the district or regional level. Thus, a rating of 'C' was assigned to this criterion.

Domestic trade or industry effects

Movement restrictions are likely to be applied for infected and control areas. This could result in welfare issues associated with over-stocking and increased feed costs for those affected piggeries. There would be loss of income for producers whose herds are destroyed and those subjected to quarantine controls and, possibly, detrimental effects on the health and welfare of those producers and their families. If exports of pig meat ceased, that meat would enter the domestic market, resulting in an oversupply and price reduction.

Taking these factors into consideration, the indirect effect on domestic trade and industry was considered to be unlikely to be discernible at the national level and of minor impact at the State level. This resulted in a rating of 'D' for this criterion.

International trade effects

Enterovirus encephalomyelitis (previously Teschen/Talfan) is an OIE List B disease. The OIE International Animal Health Code provides guidelines for the import of pigs, pig semen and pig meat from infected countries. It should be noted that as PTV-1 is endemic throughout pig populations, this chapter of the Code should be revised. Nonetheless, it is possible that if an outbreak of enterovirus encephalomyelitis occurred in Australia, export of live pigs, pig semen and possibly fresh pig meat may cease until Australia could demonstrate freedom or has clearly defined a zone free from this disease. The OIE Code states that a country may be considered free from the disease 6 months after a stamping out policy with or without vaccination. An infected zone can be considered free 40 days after a stamping out policy. Trade in pig meat may be able to continue to Singapore, our major export market as they do not have a pig industy. The OIE Code recommends that pig meat from an infected country could be exported providing the entire consignment of meat comes from animals which have not been situated in an infected zone. As this outbreak is limited to a local area, this requirement could easily be met. Export of feral pig meat to Europe may cease. This may not only result in closure of export slaughterhouses, but also result in the loss of the feral pig harvesting industry and increased feral pigs numbers.

On this basis the likely impact of Teschen disease, was considered unlikely to be discernible at the national level, and of minor impact at the State level. This gave the disease a rating of 'D' for this criterion.

Indirect impact on the environment

In this scenario, Teschen disease is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, Teschen disease would have established in a broader population of commercial piggeries (including medium-large piggeries). An eradication program would have been mounted in response to the diagnosis of the disease in pigs.

The direct impact of Teschen disease

Animal life or health

In this scenario, Teschen disease has spread to a more general population of domestic pigs including medium to large commercial piggeries. As discussed earlier, Teschen disease has

been reported to cause deaths in 70% to 90% of pigs in infected herds. Depending on exposure of Australian pigs to the endemic strain of PTV-1 mortalities may not be as high.

On balance, the direct impact on animal health was considered unlikely to be discernible at the national or State level, but would be of minor significance at the district or regional level. This gave the disease a rating of 'D' for this criterion.

Environment

Because Teschen disease is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of Teschen disease

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

The response to a diagnosis of Teschen disease in the fourth scenario would be similar to that described for scenario 3, although vaccination may be considered an option as part of the control program. A rating of 'D' was thus assigned for this criterion.

Domestic trade or industry effects

The indirect effects on domestic trade and industry would be similar to those described for scenario 3 except over a wider area. Disruption to movement of pigs would occur for the duration of the outbreak. Associated industries such as transport, meat processors and stock feed manufacturers' may also be affected. Given this, the impact on domestic trade or industry was considered unlikely to be discernible at the national level and of minor significance at the State level. This resulted in a rating of 'D' for this criterion.

International trade effects

Were the fourth scenario to occur, the indirect effect on international trade would be as described above for scenario 3, of minor significance at the State level. This gave the disease a rating of 'D' for this criterion.

Indirect impact on the environment

If eradication of the disease is by slaughtering-out infected herds, rather than permitting those herds to be slaughtered at an abattoir, environmental problems may result from the disposal of pigs by burial. In view of this, the indirect impacts on the environment were considered unlikely to be discernible at national and State level, but of importance for the affected districts and regions. Thus, a rating of 'C' was assigned to this criterion.

Indirect impact on communities

Where the pig industry is significant to the local area, some aspects of that community may be affected. Some piggeries will suffer a loss of revenue due to the decreased production. This would likely have a flow on affect for that community. On balance, the indirect impact of Teschen disease on rural communities was considered unlikely to be discernible at the national or State level, but of minor importance to affected districts and regions. This resulted in a rating of 'C' for this criterion.

The overall impact of Teschen disease

When the direct and indirect impacts of Teschen disease were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences negligible

Scenario 3: Consequences low

Scenario 4: Consequences low

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 92, Table 93, and Table 94. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were considered 'very low', 'low' and 'low' respectively.

Table 92 Teschen disease: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Moderate	Negligible	Negligible
Scenario 3	Low	Low	Very low
Scenario 4	Very low	Low	Negligible
		Overall likely consequences	**Very low**

Table 93 Teschen disease: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Moderate	Low	Low
Scenario 4	Low	Low	Very low
		Overall likely consequences	**Low**

Table 94 Teschen disease: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Moderate	Low	Low
Scenario 4	Low	Low	Very low
		Overall likely consequences	**Low**

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with Teschen disease.

Table 95 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk meets Australia's ALOP (very low), risk management would not be required for Teschen disease.

Table 95 Teschen disease: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Very low	Low	Very low	Negligible
Backyard pigs	Very low	Extremely low	Low	Negligible
Small commercial piggeries	Very low	Low	Low	Very low
			Overall annual risk	**Very low**

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Rabies virus

Technical information

Background

Although rabies is a disease of public health significance, it is not, in the natural sense, a disease of humans. Rabies, most commonly acquired through the bite of a rabid animal, is an almost invariably fatal viral encephalitis, which may affect all warm-blooded animals. The almost 100% fatality rate of this infection in humans once symptoms develop, and its near global distribution (estimated 60,000 human fatalities per year worldwide), makes rabies one of the most significant zoonotic diseases (Rabnet, 2001). Some 90% of human cases of rabies and 99% of deaths due to rabies worldwide are caused by exposure to rabid dogs (Haupt, 1999). Endemic dog rabies is of major concern worldwide. Rabies control programs have reduced the number of dog rabies cases in many countries, including the United States of America, Canada and most European countries where a reservoir of rabies continues to exist in several wildlife species such as racoons, skunks, bats and foxes.

Agent taxonomy

Rabies virus is a bullet shaped RNA virus, one of the seven species belonging to the genus *Lyssavirus*, family *Rhabdoviridae*, order *Mononegavirales.* Several different rabies variants have been identified in terrestrial mammals and in insectivorous bats (Rupprecht, et al., 2002). Virus species assigned to the *Lyssavirus* genus are as follows: Australian bat lyssavirus, Duvenhage virus, European bat lyssavirus type 1, European bat lyssavirus type 2, Lagos bat virus, Mokola virus and rabies virus.

The OIE code exempts European bat lyssaviruses type 1 and 2 when setting the requirements for countries to declare themselves free from rabies. Australia is considered free of terrestrial (genotype 1) classic rabies, although a lyssavirus has been isolated from bats.

Agent properties

The rabies virus is well adapted to replication in mammalian neural tissue. It does not persist away from animals, being rapidly inactivated by exposure to ultraviolet radiation, direct sunlight and heat (Swanepoel, 1994; Geering, et al. 1995). Rabies virus survived for 144 hours when kept at 5°C, and for 24 to 48 hours at 20°C to 21°C. At 30°C, the virus remained active for over 20 hours without sunshine or less than 1.5 hours in sunshine (Matouch, et al., 1987). Temperatures above 55°C destroy the virus within minutes. Rabies is an enveloped virus and can be destroyed by formalin, strong acids and bases, several detergents and some disinfectants (Baer, 1990).

Host range

Almost all warm-blooded animal species are susceptible to rabies. The carnivores and the insectivorous bats are usually the most commonly infected species. The degree of susceptibility varies considerably depending on animal species, the age of animal (with younger animals usually being more susceptible), the viral variant involved, quantity of virus inoculated, and the site of infection. Human cases occur. Rabies is not commonly diagnosed in pigs (Swanepoel, 1994).

Epidemiology

Rabies almost always enters the victim through the bite of an infected host in the clinical or prodromal stages of disease. Uncommon routes of transmission include air-borne transmission, almost always in bat caves or laboratories where infectious aerosols are present, through contamination of mucous membranes (eyes, nose and mouth), ingestion of infected tissues (brain), transplacental infection and, in humans, corneal transplants (Afshar, 1979; Greene & Dreesen, 1998). Pigs would appear to be more resistant to infection with rabies virus than some other animal species (Morehouse, 1999).

Pigs become exposed to rabies virus by being bitten by infected animals such as dogs, skunks and vampire bats (Baer & Olson, 1972; Ito, et al., 2001; Mitmoonpitak, et al., 2002). In Brazil, humans have been bitten by rabid pigs and been given post-exposure prophylaxis (Nishioka, et al., 1994).

Porcine rabies appears to be relatively uncommon (Yates, et al., 1983). Historically, most documented cases of rabies in pigs involved herds of less than 100 animals (Merriman, 1966; Baer & Olson, 1972; Yates, et al., 1983). In one case, 17 of 90 weaned pigs died of rabies over a period of a month (Merriman, 1966). One report described rabies outbreaks in 15 pig herds in western Canada over a 14-year period (Yates, et al., 1983). A close interface between the abundant wild skunk population and backyard pig herds accounted for most of these cases. In 1986 in Canada, rabies was diagnosed in one pig in a barn of 785 pigs (Hazlett & Koller, 1986). A stray cat was suspected as the vector. Several other pigs died around the time rabies was detected. In the United States of America, three cases were reported in 1999 and none in 2000 (Krebs, et al., 2000; Krebs, et al., 2001). In recent years, the WHO Rabies Bulletin Europe reported around none to four pigs with rabies each quarter.

Clinical signs

Rabies can be present in two different forms, excitatory or paralytic. Both forms are preceded by a prodromal phase. In humans, it first presents as a malaise, fever or headache lasting for several days. Then subtle changes in temperament, such as depression, dementia or aggression manifests. The excitatory phase in animals is often referred to as furious rabies where the animal becomes viciously aggressive and the paralytic phase is often referred to as dumb rabies, where paralysis spreads from the initial site of infection to involve the entire nervous system.

In pigs, there would appear to be a wide variation in the incubation period ranging from 9 days up to 123 days (Morehouse, 1999). The typical course of the disease is one of sudden onset. Pigs usually exhibit the excitatory phase. Affected animals may become excited and attack other animals, people or objects around them. Sometimes the pig may become dull and uncoordinated. Other signs include nose twitching, rapid chewing movements, excessive salivation, clonic convulsions and paralysis (Radostits, et al. 2000). Death usually follows within 72 hours of onset of clinical signs.

Pathogenesis

The virus may enter the peripheral nerves directly at the site of infection and spread through the nerves to the spinal cord and the brain via retrograde axoplasmic flow. The virus can replicate in the spinal cord, brain and non-nervous tissues, such as muscles, at the site of infection before uptake into the peripheral nerves. Once in the brain, the virus rapidly disseminates, resulting in active cerebral infection, then spreads by passive centrifugal transfer back to the peripheral

nerves and invades highly innervated sites of various tissues, including the salivary glands (Morehouse, 1999; Jogai, et al., 2002). When cerebral infection occurs, the classic behavioural changes develop.

Pathology

Gross pathological changes are not seen with rabies. Histopathological changes vary from case to case. Sometimes only mild vasculitis is seen. Others may show extensive encephalitis, myelitis and/or meningitis. The negri body, inclusion bodies characteristic of rabies in humans and many animal species, is not always seen in pigs (Morehouse, 1999).

Immunology

The ability of an animal to develop neutralising antibodies depends on the dose of virus received, the route of infection, and the interval between infection and death (or, in some cases, recovery). Animals can develop neutralising antibodies 7 to 12 days after infection (Charlton, et al., 1987).

Transmission via meat

There have been few studies in which muscle tissue has been examined for the presence of rabies virus. In cattle, the virus was isolated from some salivary glands of cattle that died from vampire bat rabies but not from mammary glands, muscle, lung, kidney and liver (Delpietro, et al., 2001). Virus has been isolated from muscle following intramuscular inoculation in foxes and cats (Alexander, et al., 1981). As the main route of infection is through the bite of an infected animal, the initial location of the virus is often an animal's muscle before spreading to motor nerve endings and moving towards the spinal cord and the brain where further replication takes place.

Oral transmission of rabies has been occasionally reported. In one report a dog developed rabies 12 days after it was suspected to have eaten a pig carcass that had died of wolf rabies (Shah & Jaswal, 1976). This report could not rule out other means of transmission, nor is it known if the dog consumed the brain of the infected pig. In mice, ingestion of infected bovine brain tissues sometimes resulted in death due to rabies (Delpietro, et al., 1990).

Rabies virus does not survive long outside a living host. It has been reported that rabies virus remains viable in a carcass for less than 24 hours at 20°C (Greene & Dreesen, 1998). A carcass in a field during summer probably does not contain infectious virus for more than a few hours (Rupprecht, et al., 2001).

Release assessment

R1 — the likelihood that a source herd is infected

Most reports of rabies in pigs have been where rabid wild animal hosts, such as skunks, have had contact with backyard pigs or outdoor pigs. There are very few reports of rabies in pigs. In the United States of America, where there are nearly 60,000 pig farms, 3 cases were reported in 1999 and none in 2000. Considering the large number of pig farms and the small number of porcine rabies reported, the likelihood that slaughter-age pigs have been selected from an infected herd was considered to be 'extremely low'.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

Most documented cases of rabies have occurred as unusual events in herds of less than 100 pigs. In the United States of America, pig farms with less than 100 head account for less than 6% of the total pig population (USDA, 2002). Most reports on porcine rabies refer to infection in a single pig. Rabies is a notifiable disease in many countries, and overall very low numbers of cases have been reported, with the United States of America and Europe each reporting a few cases a year. In contrast, there was a report of 17 of 90 weaned pigs that died of rabies over a period of a month, but this occurred before 1966 when pig husbandry management systems were quite different. On the basis of this information, it was considered that the likelihood of selecting an infected pig in an infected herd was ‘very low’.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

Infected pigs can incubate the virus for several months, in some instances, before developing clinical signs. Pigs in the incubation phase would not be detected during the slaughter process. Clinical signs of either the excitatory or paralytic phase may be detected during ante-mortem inspection. Gross pathological lesions are not evident in pigs with rabies. Thus the sensitivity of ante-mortem, slaughter and processing procedures in detecting and removing infected pigs was considered ‘very low’.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with rabies virus and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a ‘one minus the background rejection rate’. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered ‘extremely low’. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

Rabies virus could not be isolated from muscle, lung, kidney, mammary glands or liver of cattle infected with vampire bat rabies, but was isolated from some salivary glands. Rabies virus is most commonly isolated from the brain of infected animals; however, brain is excluded in the definition of pig meat. As virus spreads centrifugally along in nerves late in infection, low levels of virus may be present in the nerves within muscle tissues. Given this, it was considered that the likelihood that rabies virus would be present in meat from an infected pig was ‘low’.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

Rabies virus does not survive well outside a living host and reportedly becomes rapidly inactivated in a carcass at room temperature. It was considered likely that during the process of carcass maturation that inactivation of virus, if present, would occur. On balance, it was considered that there was a 'low' likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

Rabies virus can survive for up to 6 days at 5°C and for several months in frozen carcasses. In view of this, it was considered that there was a 'high' likelihood that meat infected at the completion of carcass maturation would remain infected during transport and cold storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a 'negligible' likelihood that imported pig meat derived from an individual carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'negligible' likelihood that a waste unit will be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

Oral transmission of natural rabies virus would appear to be a very rare event and it is likely that a high dose of virus is required to initiate infection (Charlton & Casey, 1979a; Madhusudana & Tripathi, 1990). This has occurred experimentally where animals have been fed infected brain (Charlton & Casey, 1979a; Charlton & Casey, 1979b). Prolonged contact with buccal mucosa or accidental contact with nasal mucosa appears to be necessary for infection to occur (Charlton & Casey, 1979a; Charlton & Casey, 1979b). Given this, the likelihood that a waste unit from an infected pig would contain a sufficient dose of rabies virus to initiate infection was considered to be 'extremely low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

The likelihood that a pathogenic agent would remain viable after exposure to the environment depends on its inherent 'stability'. In particular, this likelihood will reflect the agent's sensitivity to ultraviolet light, to ambient temperatures between approximately 10°C and 35°C

and to the putrefying effects of saprophytic organisms. Rabies virus is rapidly inactivated by exposure to ultraviolet radiation, direct sunlight and heat. The virus survived for 24 to 48 hours at 20°C to 21°C, and for over 20 hours without sunshine at 30°C or less than 1.5 hours in sunshine.

In light of this information, it was considered that the likelihood that rabies virus would survive within meat scraps discarded in refuse for the period of time required for feral pigs to locate and subsequently scavenge the material was 'extremely low'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = Negligible
- Rural regions = Negligible
- Large towns = Negligible

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be 'negligible'.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'negligible' likelihood that a waste unit will be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'extremely low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be discarded due to spoilage. Overall, the Panel considered that there was an 'extremely low' likelihood that rabies virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'negligible'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'negligible' likelihood that a waste unit will be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'extremely low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. These pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be discarded due to spoilage. The virus is likely to be completely inactivated in spoiled meat and cooked meat. Overall, the Panel considered that the likelihood that rabies virus would remain viable during the period prior to ingestion was 'extremely low'.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'negligible'.

Exposure assessment for other susceptible species

Several carnivorous or omnivorous animal species such as rodent, dogs, cats, foxes and dingoes may have access to meat scraps in Australia. Rodents are not considered important in the epidemiology of rabies (Rupprecht, et al., 2002). Experimentally laboratory mice have been infected with rabies virus following ingestion of naturally infected bovine brain tissues (Delpietro, et al., 1990). Rabies virus replicates in the brain, and it was likely that the mice had been infected with a high infectious dose. There are very occasional reports of rabies in dogs linked to the consumption of infected carcasses (Shah & Jaswal, 1976). It is unclear in these reports if the brain was consumed. It would appear that infection via this route is a very rare event. Moreover, rabies virus is fairly fragile and does not persist in the environment (Rupprecht, et al., 2002).

Given this, it was considered that the annual likelihood of entry and exposure for other susceptible species was 'negligible'.

Conclusions

The annual likelihood of entry and exposure for each of the exposure groups was determined to be negligible. As such, further assessment was not conducted. No risk management measures would be required for rabies virus.

The Department of Health and Ageing has advised Biosecurity Australia that biosecurity measures for rabies virus would not be required to manage the risk to human life or health associated with the importation of pig meat.

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Mycobacterium bovis

Technical information

Background

Mycobacterium bovis is the most common cause of tuberculosis in cattle but can also cause disease in humans, pigs and numerous non-domesticated species.

Since 1971 bovine tuberculosis, caused by *M. bovis,* was subjected to an eradication campaign in Australia. The Brucellosis and Tuberculosis Eradication Campaign (BTEC) ended in 1997 (Lehane, 1996) when Australia attained Officially Free status in accordance with the recommendations of the OIE International Animal Health Code. BTEC was funded by both government and industry and was the most comprehensive and expensive animal disease eradication campaign undertaken in Australia. Currently, the Tuberculosis Freedom Assurance Program (TFAP), funded by Commonwealth and State governments and industry manages surveillance and eradication procedures to ensure Australia's continuing freedom from bovine tuberculosis.

Agent taxonomy

Domain	Bacteria		
Phylum	Actinobacteria		
Class	Actinobacteria	Subclass	Actinobacteridae
Order	Actinomycetales		
Family	Mycobacteriaceae		
Genus	*Mycobacterium*		

The genus *Mycobacterium* contains over 70 species, divided into a few obligate pathogens (*M. bovis, M. tuberculosis* and other slowly growing species) which may cause disease in animals and humans and a number of rapidly growing, non-pathogenic saprophytes. Slowly growing mycobacteria are further divided on pigmentation during growth. Recent biochemical and genetic studies have suggested the further subdivision of *M. bovis* to include a subspecies, *M. bovis* subsp. *caprae* (Niemann, et al., 2002).

Agent properties

The mycolic acid constituents of the bacterial cell wall render *Mycobacterium* spp. acid fast; previously stained cells resist decolourisation by acidified ethanol, an identifying characteristic of this genus. Because of high lipid content in the cell wall, mycobacteria are highly hydrophobic, and thus resistant to hydrophilic antibacterials (Quinn & Markey, 2001). The use of antibacterials against *M. bovis* is reviewed by Quinn and Markey (2001).

Mycobacteria are resistant to freezing and to desiccation. They survive well in aerosols and in water and are given added protection by proteinaceous material in sputum (Rubin, 1991). Their resistance to acid and alkaline environments is useful in prevention of overgrowth by other bacterial species in contaminated specimens for culture. However, they are susceptible to ultraviolet light in doses similar to that required to kill other bacteria. They are also susceptible to alcohols, chlorine, glutaraldehyde, iodophores, phenol, ethylene oxide and hydrogen

peroxide (Good & Shinnick, 1998). Merkal and Whipple (1980) examined the effects of formaldehyde vapour, Amphyl soap (active ingredient phenols) and ultraviolet irradiation in meat products containing 10^7 viable organisms per gram. They found that disinfectants, formaldehyde (10 g/m^3) and ultraviolet light (10 $\mu W/cm^2$) alone and in combination, were effective against unprotected *M. bovis* but there was a protective effect against individual treatments found in thick meat emulsion smears. Treatment of thick smears with 2% Amphyl, followed by formaldehyde vapour was effective. Benzalkonium chloride (0.3%) was not effective.

Mycobacteria are susceptible to heat and pasteurising procedures kill mycobacteria suspended in milk and meat products. Merkal and Whipple (1980) trialled heat inactivation of *M. bovis* in meat products (emulsions of beef, pork and pork fat in sausage casings) containing 10^7 viable organisms per gram. Above 57.5°C, numbers of viable *M. bovis* remaining was very small and above 60°C, no viable units remained by the time units tested reached designated temperatures. Effects of smoking at 47°C were not different from heat treatment at 47°C; *M. bovis* was not killed by either process. D values (decimal reduction time, or time for 1 log reduction in surviving bacterial numbers) were calculated by extrapolation for the product examined. Unfortunately neither raw data nor error values are given in the report.

Host range

Mycobacterium bovis causes tuberculosis in a wide variety of mammals. Bacille Calmette Guérin (BCG), developed as a vaccine for protection of humans against *M. tuberculosis*, is derived from *M. bovis*. All mammals appear to be susceptible, including cattle, humans, non-human primates, goats, sheep, horses, cats, dogs, pigs, buffalo, deer and bison. Isolations have been made from numerous wild animals (Office international des épizooties, 2000). Wildlife reservoirs exist in various parts of the world, notably badgers in the United Kingdom, brushtail possums and ferrets in New Zealand and cervidae in North America. Such reservoirs complicate control programs in livestock by providing a continuing source of infection. Despite high levels of infection in feral pigs exposed to tuberculous livestock, they do not appear to form such a reservoir (Corner, et al., 1981; McInerney, et al., 1995). Transmission between pigs probably only occurs rarely (Acha, et al. 1987) and the pig is not considered an important host of *M. bovis* (Francis, 1958).

Epidemiology

Unless identified by culture, reports of mycobacterial infection in pigs can be misleading: frequently they are not caused by *M. bovis*. However, where *M. bovis* is endemic and pigs are exposed, it is the major cause of tuberculosis in pigs. However, the majority of current reports of tuberculosis in pigs refer to infection with species within the *M. avium* complex (Thoen, 1999), consistent with low prevalences of *M. bovis* in cattle. Of specimens examined in the United Kingdom, the proportion of all mycobacterial isolates that were of *M. avium* increased from 44% to 92% between 1952 and 1966 during bovine tuberculosis eradication (Lesslie, et al., 1968).

Early in the 20th century infection of pigs with *M. bovis* was well recognised as a result of feeding skim milk from dairies with infected cattle and by feeding infected bovine offal (Albiston, 1965). In regions where the prevalence of bovine tuberculosis is high, isolation of *M. bovis* from tuberculous lesions in pigs can reach 80% (Acha, et al. 1987). In the Northern Territory of Australia, exposure of feral pigs to infected buffalo or to abattoir wastes resulted in high (31%) prevalence of infection of pigs with *M. bovis* (Letts, 1964). An age related increase

in prevalence (criterion: macroscopic lesion) from 16% at less than 12 months to 68% in pigs 5-6 years of age was reported (Corner, et al., 1981). The older animals also returned the lowest proportion of positive cultures, possibly indicating development of immunity with age.

Changes in exposure of pigs to *M. bovis* through management practices and through bovine tuberculosis eradication resulted in large reductions in prevalence in both domestic and feral pigs in Australia (Albiston, 1965; Buddle, 1985; McInerney, et al., 1995). Similar disease reductions were observed in the United States of America and the United Kingdom (Lesslie, et al., 1968; Thoen, 1999). In the United States of America, abattoir prevalence of *M. bovis* in pigs has approached zero in surveys done since 1970. A three year summary of isolations of mycobacteria from porcine tissues in the United States of America in 1972 revealed a prevalence of 1% *M. bovis* against 97% *M. avium* (Thoen, et al., 1975). In such situations, where bovine tuberculosis is under control, the relative prevalences of *M. bovis* and other mycobacterial infections are reversed (Lesslie, et al., 1968).

Prior to eradication of bovine tuberculosis in Australia, *M. bovis* was rarely reported in animals other than cattle, buffalo and pigs. Horses, dogs and sheep (Albiston, 1965) have been occasionally reported with infection and one goat (Cousins, et al., 1993) was diagnosed with bovine tuberculosis. Brushtail possums (*Trichosurus vulpecula*) and ferrets (*Mustela putorius furo*) are involved in the epidemiology of bovine tuberculosis in New Zealand but not in Australia.

Novel molecular techniques in strain identification of mycobacteria offer higher discrimination between isolates than were possible with serotyping and cultural techniques (Durr, et al., 2000). Identification of strain differences allows inferences to be made regarding origin of infection and this has been used as evidence of cattle to pig transmission of *M. bovis* (Serraino, et al., 1999) in northern Italy. While genotyping may identify like strains of bacteria, it does not necessarily infer the direction of transmission in a disease with a lengthy incubation. Serraino *et al*. (1999) consider that the distribution of lesions (lymph nodes in the head, in particular the submandibular lymph node) in pigs tested is consistent with infection by ingestion of discharges from infected cattle in pasture. In addition, they note that low prevalence of pulmonary and generalised infections limits the possibility of transmission from pigs.

Clinical signs

Clinical signs in tuberculosis infections generally depend on the route of infection because of the organs affected and severity of disease. Consumption of infected product (milk, offal) produces lesions in the digestive tract and associated lymph nodes, including those of the oropharynx. Aerosol infection produces lesions in the lung and associated lymph nodes, depending on dose and infectious particle size. More rarely, cutaneous infection introduced by trauma will cause local infection and again associated lymph nodes may eventually become involved.

Clinical signs in animals are rare, unless the disease is generalised and progressive. Lesions in cervical nodes are unlikely to be of a size that can be clinically apparent, and may only be obvious if ruptured. Infection of intestine and mesenteric lymph nodes may not be obvious ante-mortem even in progressive infections. Descriptions of clinical signs are inconsistent, however, a contemporary investigation in New Zealand describes two clinical cases, of 25 infected pigs in a herd of about 100 pigs, as being “thin” (McLaughlin, 1989).

Pathogenesis

After entry at the site of infection (usually respiratory or digestive system mucosa), mycobacteria may be detected and destroyed by activated macrophages (Roitt, 1997). Some virulent mycobacteria avoid destruction and escape into the cytoplasm of macrophages where they multiply and destroy the phagocyte (Clifton-Hadley, et al., 2001). Attraction of inflammatory cells results in development of T-cell immunity, immune system destruction of mycobacteria (van Crevel, et al., 2002) and reaction to delayed hypersensitivity testing. At this stage, infection may become dormant, and some mycobacteria survive in macrophages providing the stimulus for an ongoing chronic inflammatory response. Aggregation with other macrophages produces a granuloma, which when macroscopically visible, is known as a tubercle. Growth of a tubercle results in spread of bacteria and development of satellite tubercles, which may coalesce and/or spread along regional lymphatics to the lymph node which drains the region. This initial focus and the draining lymph node is known as the *primary focus* of the infection (Dungworth, 1993). Disease may later progress under conditions of failing immune surveillance (van Crevel, et al., 2002). Generalised secondary infection, in multiple organs, may result from haematogenous spread in macrophages, from blood vessel breakdown adjacent to an expanding lesion allowing entry of infectious material to the bloodstream or from further spread of the infection along lymphatics. The course of the disease is variable depending on the extent of dissemination. Clinical signs will be evident only when vital areas are affected or massive dissemination has occurred. Progression of disease results in weakness, debility and possibly death.

Apart from the physical growth of granulomas, the mechanisms of pathogenesis of mycobacterial infections are not well described.

Pathology

Tuberculosis in pigs usually involves the lymph nodes of the pharynx and/or mesentery, indicating infection by ingestion. Pulmonary involvement is less common: both Francis (1958, table, p185) and Corner, (1981) describe low proportions of lesions confined to the thoracic cavity. Extensive pulmonary involvement was reported in feral pigs with an estimated prevalence of *M. bovis* of 20% in an infected area in the United States of America (Essey, et al., 1981). The reason for this difference is uncertain but may be attributable to an alternate mode of infection.

Lesions caused by *M. bovis* vary in size from microscopic granulomas to involvement of the entire lymph node and other organs. They vary in consistency from soft to caseous, with varying calcification and in colour from white to yellowish. The extent of organ systems involvement is also variable, from just a primary focus - more common in pigs because of the comparatively young age at which most are slaughtered - to more rarely, generalised tuberculosis with diffuse involvement of multiple organ systems. Other bacteria are known to cause similar pathology, including a number of other mycobacteria - *M. tuberculosis, M. avium, M. avium* subsp. *paratuberculosis,* and saprophytic mycobacteria, as well as *Rhodococcus equi*. Parasitic granulomas may be similar in appearance. Lesions caused by each of these pathogens have typical characteristics, but the overlap in gross appearance is such that specimens require at least histopathological and bacteriological examination to reliably confirm a diagnosis.

Immunology

The immunology of *M. bovis* infections in pigs has not been specifically addressed in recent research. There are a number of similarities with other mycobacterial infections which may be drawn and the reader is referred to various reviews on the subject (van Crevel, et al., 2002; Pollock & Neill, 2002; Kaufman, 2003).

Current methods of diagnosis of disease in live pigs involve delayed hypersensitivity reactions with the use of purified protein derivative mammalian tuberculin. Because of the high incidence of *M. avium* in pigs comparative testing is often used, with tuberculin of bovine and avian origin of the same concentration injected at two sites usually on opposite sides of the animal. Other cell mediated immune response assays such as the γ interferon test have not been adapted for pigs. An ELISA is reported by the OIE (2000) for use in cattle and zoo animals but its use in pigs is not described.

Vaccination (in cattle) has been investigated by a number of workers, in particular the use of BCG and more recently protein vaccines and DNA plasmid vaccines (Skinner, et al., 2001). None has proved efficacious and are unlikely to have any effects on prevalence of *M. bovis* in pigs even where disease is endemic in cattle.

Transmission via meat

Research on transmission of *M. bovis* in meat began over 100 years ago. Chaussé reports on a series of trials in which muscle and visually clean lymph nodes from tuberculous cattle were fed to guinea pigs, pigs, piglets and other animals and concluded that oral infection was not possible (Chaussé, 1917). He also records parenteral inoculation of muscle tissue from 42 cattle and 18 pigs described as suffering from generalised tuberculosis into 209 guinea pigs, not one of which became infected with tuberculosis (Chaussé, 1917). In his work on infected bovine and porcine carcases, Chaussé concluded that contamination of meat by transfer from infected organs or lymph nodes was possible and that bacteraemia occurred in tuberculosis infections. However he concluded that mycobacteria did not appear to infect muscle, confirming earlier work of McFadyean (1890).

Infective doses of *M. bovis* for pigs, as a model for human infection, have been investigated for intratracheal (though not by aerosol), intravenous and tonsillar routes of infection (Bolin, et al., 1997). While the number of animals exposed was small, all recipients were infected by the intratracheal route with just 10^2 colony forming units (CFU) whilst only 50% of recipients were infected by the tonsillar route with 10^4 CFU.

Transmission of pathogenic mycobacteria to pigs has been extensively reviewed (Francis, 1958), and the oral susceptibility of pigs to *M. bovis* highlighted. Pigs are susceptible to infection from tuberculous milk and from grain contaminated by faecal material from infected cows (Schroeder & Mohler, 1906). The location of lesions in the digestive tract of pigs known to have consumed infectious material attests to ingestion as the method of disease transmission (Fichandler & Osborne, 1966; Corner, et al., 1981; Perez, et al., 2002).

While only small doses of bacteria are necessary to initiate infection, the viable bacterial content of lesions is not known but is expected to be variable. Francis (1958) discusses infective doses and notes that bacilli in culture appear to be some several hundred times less virulent than those from tuberculous tissue. In carcases, muscle tissue and blood are unlikely to contain infective material (M'Fadyean, 1892), which is limited to infected retained lymph nodes.

There is little recent work on transmission of *M. bovis* via meat but carcases from cattle with single lesions of tuberculosis have been entering the human food chain for many years. No risk associated with this practice has been identified.

Release assessment

R1 — the likelihood that a source herd is infected

Country prevalence varies with prevalence of *M. bovis* in cattle, and on exposure of pigs to infectious material by husbandry and nutrition practices. Current knowledge of the epidemiology of *M. bovis* infections in pigs suggests that an external source is required for infection in a herd to occur. Data on between herd prevalence of *M. bovis* in pig herds was not identified. In this IRA, it is assumed that *M. bovis* exists in the country of origin and that feeding practices do not preclude transmission to pigs. Overall prevalence of *M. bovis* in domestic pigs where *M. bovis* occurs varies from 0.1% (Lesslie, et al., 1968) to 6% (Perez, et al., 2002). Given this, the likelihood that slaughter-age pigs have been selected from an infected herd in a country where *M. bovis* is endemic was considered to be 'low'.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

Some surveys for mycobacteria have not identified the pathogen and may include granulomas caused by mycobacteria other than *M. bovis* or other bacterial or parasitic pathogens. An investigation into the prevalence of tuberculosis in feral pigs in New Zealand recorded a 31% prevalence of bovine tuberculosis but isolated *M. bovis* from only 16 pigs in 251 sampled (Wakelin & Churchman, 1991). Two surveys of feral pigs exposed to infected buffalo in the Northern Territory of Australia were reported as having prevalences of *M. bovis* infection of 16% and 19% (Letts, 1964; Corner, et al., 1981). Corner reported prevalence in pigs less than 12 months of age to be 16% (criterion: macroscopic lesion). In the United States of America, abattoir surveillance of domestic pigs showed prevalences from 6.6% reducing to zero in more recent years with eradication of bovine tuberculosis in cattle. Relative proportions of avian tuberculosis increased during the same period (Thoen, 1999). Given this range of prevalences, in both domestic and feral pigs, it was considered that the likelihood of selecting an infected pig in an infected herd was 'low'.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

Clinically affected animals are unlikely to pass ante-mortem inspection that complies with requirements described in the Australian Standard. While not specifically rejected for tuberculosis, it is expected that they would be rejected for reasons of body condition or similar findings. However, such condemnations will constitute a very small proportion of affected animals. Most animals infected with *M. bovis* do not show clinical signs of disease.

Abdominal and thoracic viscera and lymph nodes are not retained in carcasses dressed in accordance with the Australian Standard. Post-mortem appearance of tuberculous lesions is

specifically addressed in the Australian Standard, as are lesions which could be confused with tubercles. Additional procedures are required on detection or suspicion of tuberculosis. Necropsies on infected pig populations (Corner, et al., 1981) indicated that 85% showed macroscopic involvement of submandibular lymph nodes with only low proportions of pigs infected in other parts of the carcass. Infected material in such carcasses may not be rejected if they conformed to the Australian Standard.

The sensitivity of inspection procedures in cattle similar to those required by the current Australian Standard were examined (Corner, et al., 1990), and it was estimated that the probability of missing an animal with lesion(s) to be 47% (est. 95% CI of 22-74%).

In view of this, the sensitivity of ante-mortem, slaughter and processing procedures in detecting and removing infected pigs was considered 'moderate'.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with *M. bovis* and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a 'one minus the background rejection rate'. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered 'extremely low'. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

The majority of *M. bovis* infections in pigs occur in the submandibular and retropharyngeal lymph nodes draining the oropharyngeal area. Surveillance on natural infections in feral pigs showed that 85% of infected animals had lesions in those lymph nodes(Corner, et al., 1981). Buddle (1985) considered that about 80% of lesions were found in head, neck or abdominal areas but does not differentiate between these. Early studies indicated that mycobacteria do not appear to infect muscle, although muscle could be contaminated by contact with infected organs or lymph nodes or if the animal was bacteraemic at the time of slaughter.

Based on this information, it was considered that the likelihood that *M. bovis* would be present in meat harvested from an infected pig was 'low'.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

Mycobacterium bovis is resistant to alterations in pH and survives acidic environments well below pH 6.2. Thus, it was considered that there was a 'high' likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

Mycobacterium bovis is resistant to freezing and is considered likely to survive cold temperatures. Thus, it was considered that there was a 'high' likelihood that meat infected at the completion of carcass maturation would remain infected during transport and cold storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a 'very low' likelihood that imported pig meat derived from a carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

Accepted meat inspection procedures eliminate visibly affected carcass parts. The likelihood of carcass parts, including lymph nodes, containing infectious material is thus dependent on the ability of the meat inspection system to remove visibly affected material. Infectious material is not expected to be distributed evenly around a carcass, but restricted to lymph nodes, particularly in the head (submandibular, parotid, about 0.1% by weight of carcass). *Mycobacterium bovis* has rarely been detected in muscle tissue even in generalised infection. Other potentially infected materials, abdominal and thoracic viscera, thoracic and mesenteric nodes are not considered as 'pig meat' in this IRA.

Only small doses of bacteria would appear to be needed to initiate infection. It is known that pigs can become infected following ingestion of contaminated milk or offal. However, infection of pigs did not occur when fed muscle and visibly clean lymph nodes from tuberculosis cattle.

The likelihood that a waste unit would contain a sufficient dose of *M. bovis* to initiate infection, given that it was derived from an infected pig, was based on the source of the waste unit (head and neck region or the rest of the carcass).

Carcass region from which waste unit derived	Weighting factor	Likelihood that waste unit would contain a sufficient dose of *M. bovis* to initiate infection
Head and neck	10%	'Low'
Rest of the carcass	90%	'Extremely low'

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

Viability of *M. bovis* has been discussed under agent properties. While sensitive to ultraviolet light, it survives desiccation and post-mortem pH and temperature changes. Except on the surface of infected material, mycobacteria will be well protected from the effects of ultraviolet light. Thus, it was considered that the likelihood that *M. bovis* would survive within meat scraps

discarded in refuse for the period of time required for feral pigs to locate and subsequently scavenge the material was ‘high’.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be ‘very low’.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a ‘very low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit would contain a sufficient dose of *M. bovis* to initiate infection, given that it was derived from an infected pig, would be based on the source of the waste unit (head and neck region or the rest of the carcass), as follows:

Carcass region from which waste unit derived	Weighting factor	Likelihood that waste unit would contain a sufficient dose of *M. bovis* to initiate infection
Head and neck	10%	'Low'
Rest of the carcass	90%	'Extremely low'

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a 'high' likelihood that *M.* bovis would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'extremely low'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'very low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit would contain a sufficient dose of *M. bovis* to initiate infection, given that it was derived from an infected pig, would be based on the source of the waste unit (head and neck region or the rest of the carcass), as follows:

Carcass region from which waste unit derived	Weighting factor	Likelihood that waste unit would contain a sufficient dose of *M. bovis* to initiate infection
Head and neck	10%	'Low'
Rest of the carcass	90%	'Extremely low'

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a 'high' likelihood that *M. bovis* would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'extremely low'.

Exposure assessment for other susceptible species

M. bovis is infectious for a variety of animals some of which are known to be able to pass infection back to cattle, the primary host. Only carnivorous (eg dogs, cats) or omnivorous animals are likely to become infected, by ingestion of pig meat wastes. Such animals are unlikely to excrete *M. bovis* in a form infectious to the primary host and the disease may even remain undiagnosed in these species. The annual likelihood of entry and exposure for other susceptible species was considered 'very low'.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the four exposure groups - that is, feral pigs, pigs in backyard enterprises pigs in small commercial piggeries and other susceptible species. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs, however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

Infection of feral pigs with *M. bovis* has been reported in Australia when bovine tuberculosis was endemic in cattle and buffalo in the Northern Territory. The role of feral pigs as "spill-over" hosts was suggested well before eradication of the disease in cattle and buffalo in that region (Corner, et al., 1981) and ultimately proven during the final stages of eradication (McInerney, et al., 1995). The brushtail possum (*Trichosurus vulpecula*), a known vector in

New Zealand, is native to southern Australia but has never been reported as being involved in the epidemiology of tuberculosis in Australia. Wildlife cycles in brushtail possums, badgers (*Meles meles*) and various cervidae are well documented and 40 other wildlife species are reported as harbouring infection (de Lisle, et al., 2001). Humans are reported to have transmitted infections of *M. bovis* to cattle. Given the modes by which disease is transmitted to pigs, any of these animals could form a source of infection for pigs although spread from pigs to these species in the first instance was considered unlikely.

Secondary spread to other pigs, wild animals and humans or back to susceptible livestock did not occur at a sustainable level if at all when feral pigs were known to be infected in the Northern Territory of Australia. Given this, it was considered likely that the disease may die out within a feral pig herd or spread to other feral pigs, probably by ingestion of contaminated carcasses and then die out.

Depending on the region of occurrence of an outbreak in feral pigs, secondary spread to native animals or even backyard domestic pigs, probably by ingestion of contaminated carcasses, should not be ruled out. Spread into the more general commercial pig population, was considered negligible as swill feeding is illegal and large commercial piggeries are well aware of this ban.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: high

Scenario 2: low

Scenario 3: very low

Scenario 4: negligible

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the ‘no outbreak’ scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The role of pigs as a spill-over host for *M. bovis* and the usual modes of transmission for pigs means that exposure of other groups of pigs or domesticated animals will be limited to

exposure to possibly infected material derived from carcasses. Such an occurrence was considered similar to that discussed above for feral pigs. Trade in backyard pigs may increase the likelihood of transmission from one domestic backyard herd to another. Spread to humans is considered unlikely given the visible appearance of lesions, a high likelihood of detection of lesioned material in abattoirs, the low likelihood of presence of *M. bovis* in muscle tissue and its susceptibility to normal cooking temperatures. Aerosolisation of infected product is also considered unlikely. It is possible that spread may occur from backyard pigs to cattle in close contact, if generalised tuberculosis occurs in pigs such that faeces are contaminated. Spread from infected cattle to other cattle may then occur.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: low

Scenario 3: low

Scenario 4: very low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios. Trade in breeding stock may increase likelihood of transmission to other herds, but management differences may reduce the possibility of exposure to infected carcass material. If cattle were in close contact with infected pigs, spread to these may occur, with movement and mixing of cattle resulting in further spread in the cattle population.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: low

Scenario 3: low

Scenario 4: very low

Other susceptible species

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of other susceptible species:

1. Containment of the disease within the sub-population of directly exposed animals - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of domestic pigs - containment within a local population of backyard enterprises or small commercial piggeries - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs - exposure of medium-large commercial piggeries and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

As outlined in the exposure assessment for other susceptible species, this group includes only those carnivores and omnivores for which infection by ingestion is likely to occur. These animals may be dead-end hosts with no further transmission. In Australia, prior to the eradication of bovine tuberculosis these animals did not appear to be involved in spread of the pathogen. It is feasible that if a feral pig scavenged a dead infected animal spread to feral pigs may occur.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: high

Scenario 2: low

Scenario 3: very low

Scenario 4: very low

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed primary exposure group - no secondary spread

Under this scenario, the disease is most likely to have established amongst exposed animals, and to have run its course without identification. This is because infection may be asymptomatic in pigs and other animals, and because it may be self-limiting within a discrete population. Under a 'no outbreak' scenario, the disease would not have any discernible direct or indirect impacts.

On this basis, a rating of 'A' was assigned to each of the direct and indirect criteria.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, the disease is most likely to have established within a population of feral pigs. The disease may be identified during post-mortem examination of feral pigs intended for the export market. However, as the disease has not spread to the cattle population, it was considered no additional control measures would be implemented. Natural attrition would be used to achieve eradication. Given this, it would not, under this scenario, have any discernible direct or indirect impacts, and a rating of 'A' was assigned to each of the direct and indirect criteria.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries.

Under this scenario, the disease has established within a local population of small commercial piggeries or backyard enterprises and has spread to local cattle and possibly humans. It was considered likely that the disease would be diagnosed at post-mortem examination during the slaughter process. The disease would be contained through the mounting of an eradication program.

The direct impact of M. bovis infection

Animal life or health

Mycobacterium bovis rarely causes clinical signs in domestic animals; most infections are asymptomatic. Given this, it was considered that the direct effect on animal health would be unlikely to be discernible at any level. This gave the disease a rating of 'A' for this criterion.

Environment

Mycobacterium bovis has not produced discernible environmental effects during 200 years' presence in Australia prior to eradication. This resulted in a rating of 'A' for this criterion.

The indirect impact of M. bovis infection

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

Spread of *M. bovis* from pigs or other species into cattle in a local area in Australia, would represent a serious setback for Australian industry. Eradication plans are already in place for *M. bovis* in Australia (Tuberculosis Freedom Assurance Program) and these would result in movement controls, disease investigation, surveillance, traceback and traceforward from known

infected and in-contact herds, herd depopulation and compensation for slaughtered livestock. Costs will be dependent on the extent of the outbreak and such programs will have effects which would have repercussions at State or Territory level. Given this, the indirect impact of new or modified control programs was considered unlikely to be discernible at the national level, and of minor importance at the State level. This gave the disease a rating of 'D' for this criterion.

Domestic trade or industry effects

Livestock movement controls imposed will depend on the extent of the outbreak and the stage at which it is discovered. Recent isolated detection of disease has resulted in restrictions on sale and movement of cattle and thus limitations on earning potential for affected premises and areas. The extent of such controls would depend on stage and size of the outbreak at detection but could involve controls at a State or Territory level. On balance, the indirect impact of *M. bovis* on domestic trade and industry was considered unlikely to be discernible at the national level and of minor significance at the State level. This gave the disease a rating of 'D' for this criterion.

International trade effects

Eradication of bovine tuberculosis in Australia has not resulted in reduced testing requirements or improved access to international markets for cattle or beef. It was considered unlikely that the occurrence of *M. bovis* in pigs or cattle in a local area in Australia would have any discernible effect on international trade. Thus, this criterion was rated as 'A'.

Indirect impact on the environment

Mycobacterium bovis in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries).

Under this scenario, *M. bovis* would have established in a broader population of commercial piggeries (including medium-large piggeries), and the cattle population in several areas of Australia. An eradication program would have been mounted in response to the isolation of the agent from affected animals.

The direct impact of M. bovis infection

Animal life or health

Mycobacterium bovis rarely causes clinical signs in domestic animals; most infections are asymptomatic. Given this, it was considered that the direct effect on animal health would be unlikely to be discernible at any level. This gave the disease a rating of 'A' for this criterion.

Environment

Mycobacterium bovis has not produced discernible environmental effects during 200 years' presence in Australia prior to eradication. This resulted in a rating of 'A' for this criterion.

The indirect impact of M. bovis

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

As described for scenario 3, eradication plans would be implemented. As this outbreak involves cattle and pig herds in several areas of Australia the control program will be more extensive to that described above. Costs will be dependent on the extent of the outbreak and such programs will have effects which would have repercussions at State or Territory level. In light of this, the indirect impact of new or modified control programs was considered to be of minor significance at the national level, and a rating of 'E' was assigned for this criterion.

Domestic trade or industry effects

Livestock movement controls imposed will depend on the extent of the outbreak and the stage at which it is discovered. Recent isolated detection of disease has resulted in restrictions on sale and movement of cattle and thus limitations on earning potential for affected premises and areas. The extent of such controls would depend on stage and size of the outbreak at detection. In this outbreak where cattle and pig herds in several areas of Australia are affected, it was considered that the indirect impact of *M. bovis* on domestic trade and industry would be of minor significance at the national level. This gave the disease a rating of 'E' for this criterion.

International trade effects

Eradication of bovine tuberculosis in Australia has not resulted in reduced testing requirements or improved access to international markets for cattle or beef. It is unlikely that the occurrence of *M. bovis* in pigs or cattle in Australia would have any discernible impact on international trade. Thus, this criterion was rated as 'A'.

Indirect impact on the environment

Mycobacterium bovis in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

Where the pig or cattle industry is significant to the local area, some aspects of that community may be affected. Some cattle properties will suffer a loss of revenue due to the control measures implemented. This would likely have a flow on affect for that community. On balance, the indirect impact of *M. bovis* on rural communities was considered unlikely to be discernible at the national or State level, but of minor significance to affected local districts or regions. This resulted in a rating of 'C' for this criterion.

The overall impact of *M. bovis*

When the direct and indirect impacts of *M. bovis* were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences negligible

Scenario 3: Consequences low

Scenario 4: Consequences moderate

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 96, Table 97, Table 98, and Table 99. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were considered 'negligible', 'very low' and 'very low' respectively. The likely consequences associated with the exposure of other susceptible species to infected pig meat scraps were considered 'very low'.

Table 96 *M. bovis*: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	High	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Very Low	Low	Negligible
Scenario 4	Negligible	Moderate	Negligible
		Overall likely consequences	**Negligible**

Table 97 *M. bovis*: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Low	Low	Very low
Scenario 4	Very low	Moderate	Very low
		Overall likely consequences	**Very low**

Table 98 *M. bovis*: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Low	Low	Very low
Scenario 4	Very low	Moderate	Very low
		Overall likely consequences	**Very low**

Table 99 *M. bovis*: summary of the consequences of exposure of other susceptible species

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	High	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Very low	Low	Negligible
Scenario 4	Very low	Moderate	Very low
		Overall likely consequences	**Very low**

Human life or health

Respiratory infection in humans with *M. bovis* is indistinguishable from that caused by *M. tuberculosis* except by culture and identification of the causative organism. Depending on the mode of transmission and age of the patient, certain clinical signs may predominate - hence, children infected orally with *M. bovis* (usually ingestion of infected untreated bovine milk) acquire lesions in the tonsil or digestive tract. These may later present clinically as cervical or mesenteric lymphadenopathy (Grange, 1998). Treatment of *M. bovis* infection in humans may be complicated by its innate resistance to pyrazinamide, but multi-drug resistance has not been reported for *M. bovis* isolated from human infections in Australia since 1994.

Infections with *M. bovis* in humans are reported by the Australian Mycobacterium Laboratory Reference network. Thirty-eight cases of *M. bovis* from about 5100 isolations of mycobacteria in 7900 cases of tuberculosis in humans have been reported in the period 1994 to 2000 in Australia (Communicable Diseases Network Australia, 2002). Not all reports of tuberculosis in humans result in mycobacterial isolation and identification, but these figures give an approximate incidence for human infections with *M bovis* in Australia of 0.05 cases per 100,000 population.

Mycobacterium bovis is not considered likely to represent a hazard to human health in Australia unless disease prevalence in cattle approaches early 20th century levels and pasteurisation of milk and milk products is abandoned.

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with *M. bovis*.

Table 100 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly lower than Australia's ALOP (very low), risk management would not be required for *Mycobacterium bovis*.

Table 100 *M. bovis*: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Very low	Very low	Negligible	Negligible
Backyard pigs	Very low	Extremely low	Very low	Negligible
Small commercial piggeries	Very low	Extremely low	Very low	Negligible
Other susceptible species	Very low	Very low	Very low	Negligible
			Overall annual risk	Negligible

The Department of Health and Ageing has advised Biosecurity Australia that biosecurity measures for *M. bovis* would not be required to manage the risk to human life or health associated with the importation of pig meat.

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Haemorrhagic septicaemia

Technical information

Background

Haemorrhagic septicaemia is a specific form of acute, highly fatal, septicaemic pneumonic pasteurellosis, primarily of water buffalo and cattle, with a high mortality rate in clinically affected animals. It is caused by certain strains of *Pasteurella multocida*. Presently, two serotypes are recognised - the Asian serotype and the African serotype. Pigs may be infected and are capable of spreading the infection to cattle. In endemic areas, a small percentage of cattle carry the organism in the tonsils and upper respiratory tract. The organism is normally spread by direct contact or contaminated feed.

The disease is present in southern and South-East Asia, the Middle East, and most of Africa. The disease is also reported occasionally in Europe and South and Central America. A few outbreaks have also been reported in the United States of America, but these have been confined mainly to bison in national parks, the last outbreak being in the 1960s (Carter, 1998).

The disease is not the same as acute septicaemic pasteurellosis of pigs which is caused by different serotypes of *Pasteurella multocida* and which has occasionally occurred in Australia (Mackie, 1996).

Agent taxonomy

Pasteurella multocida is a small, facultative anaerobic, gram-negative, non-spore-forming coccobacillus with bipolar staining. It often exists as a commensal in the upper respiratory tract of animals. Three serotyping methods are available - the ‘capsular’ typing method using an indirect haemagglutination (IHA) test, ‘somatic’ typing using agglutination, and the agar gel immunodiffusion (AGID) test. The Asian strains capable of causing haemorrhagic septicaemia belong to capsular type B only, while the African strains are types B and E. Using the somatic typing methods, all strains belong to type 6 by the agglutination tests and type 2 by the AGID test. (Office international des épizooties, 2000).

Agent properties

The causal agent typically does not survive for more than 2 to 3 weeks in the soil or on pastures. The viability of toxigenic *Pasteurella multocida* (capsular group D) in aerosols declined to 2 to 8% of its initial value after 45 minutes depending on the relative humidity. Toxigenic *Pasteurella multocida* became nonculturable 1 to 14 days after inoculation in water and artificial seawater, depending on storage and constituents in the liquid (Thomson, et al., 1992).

Host range

Cattle and water buffaloes are the principal hosts of haemorrhagic septicaemia, and it is widely considered that buffaloes are the more susceptible. The disease has been confirmed in American bison in 1912, 1922, and 1965. Although outbreaks of haemorrhagic septicaemia have been reported in sheep and swine, it is not a frequent or significant disease. Cases have been reported in deer, elephants and yaks (Carter, 1998). Sporadic outbreaks of haemorrhagic septicaemia have been reported amongst pigs in Sri Lanka caused by the Asian serotype, and

typical haemorrhagic septicaemia has been reproduced in cattle using this isolate. There have been similar reports from Thailand, Vietnam, Malaysia and India (De Alwis, 1992). Not all cases of acute septicaemic pasteurellosis in pigs are haemorrhagic septicaemia (Townsend, et al., 1998).

Although systemic pasteurellosis has been reported in pigs in Australia, these reports differ from those in Asia as they involve *Pasteurella multocida* capsular type A (Blackall, et al., 2000).

Epidemiology

The disease is spread by direct and indirect contact (fomites). The source of the infection is infected animals or ('active' as distinct from 'latent') carriers. Cattle or buffalo artificially inoculated subcutaneously with lethal doses (approximately 20,000 bacilli) show clinical signs within a few hours and succumb within 18 to 30 hours (Carter, 1998).

The agent of haemorrhagic septicaemia in pigs has been experimentally transmitted to cattle but no study to date has demonstrated any epidemiological relationship between porcine and bovine capsular type B:2 isolates (Townsend, et al., 1998).

In endemic areas, the causative organism can frequently be isolated from the tonsils of healthy animals. It is present in the crypts of the tonsillar tissue, not the tonsillar tissue itself (Horadagoda & Belak, 1990). A transient carrier state exists when the organism is present in the nasopharynx of clinically normal cattle and buffaloes (De Alwis, 1992). An outbreak can begin when a 'latent carrier' becomes active and sheds virulent organisms infecting in-contact susceptible animals (Carter, 1998).

There are no reports indicating whether the carrier state exists in pigs. In Vietnam, a survey of 36 tonsils from healthy pigs at slaughter revealed 16 positive for *Pasteurella multocida* but none of these strains were capsular type B or toxigenic type D strains (Townsend, et al., 2000). Similarly 43 of 150 healthy pigs in Poland yielded *Pasteurella multocida* (Golebiowski, 1974). However, *Pasteurella multocida* capsular type B is commonly associated with "acute septicaemic pasteurellosis" in pigs in Asia. In Malaysia, of 20 strains of *Pasteurella multocida* isolated from swine specimens sent to the laboratory, 7 were capsular type B (Chandrasekaran & Yeap, 1982).

Clinical signs

Clinical symptoms and pathology of haemorrhagic septicaemia in pigs are characteristic of those observed in cattle and buffaloes (Townsend, et al., 1998)

The majority of cases in cattle and buffalo are acute or peracute with death occurring from 6 to 24 hours after the first recognized signs. In a few outbreaks, animals may survive as long as 72 hours. Dullness, reluctance to move, and elevated temperature are the first signs. Following these signs, salivation and nasal discharge appear, and oedematous swellings are seen in the pharyngeal region and then spread to the ventral cervical region and brisket. Visible mucous membranes are congested and respiratory distress is soon followed by collapse and death. Recovery, particularly in buffaloes, is rare. Chronic manifestations of haemorrhagic septicaemia do not appear to occur.

Pathogenesis

Bacterial endotoxin has a pivotal role in the pathogenesis of haemorrhagic septicaemia. The buffalo is more susceptible than cattle to the effects of *Pasteurella multocida* (Horadagoda, et al., 2001; Horadagoda, et al., 2002).

Pathology

Widely distributed haemorrhages, oedema, and general hyperaemia are the most obvious tissue changes observed in infected animals. In almost all cases there is an oedematous swelling of the head, neck, and brisket region. Incision of the oedematous swellings reveals a coagulated serofibrinous mass with straw-coloured or blood-stained fluid. This oedema, which distends tissue spaces, is also found in the musculature. There are subserosal petechial haemorrhages throughout the animal and blood-tinged fluid is frequently found in the thoracic and abdominal cavities. Petechiae may be found scattered throughout some tissues and lymph nodes, particularly the pharyngeal and cervical nodes, which are also swollen and often haemorrhagic. In the thoracic cavity, varying degrees of lung involvement are evident, ranging from generalised congestion to extensive consolidation with thickening of the interlobular septae, giving rise to a lobulated appearance. Marked pleurisy and pericarditis may be seen with thickening of the pericardium and a collection of serosanguinous fluid in the pleural cavity and pericardial sac (De Alwis, 1993b; Carter, 1998).

Immunology

In endemic areas, naturally acquired immunity in some animals was the result of ‘arrested infection’. It is surmised that morbidity and mortality due to haemorrhagic septicaemia in a given animal population is largely dependent on the proportion of immune and non-immune animals and therefore the phenomenon of naturally acquired immunity is responsible for the different patterns of morbidity and mortality in endemic and non-endemic areas (De Alwis, 1992).

Transmission via meat

There are no reports of *Pasteurella multocida* being transmitted via meat. Transmission is via aerosols from infected animals. Fomites such as feed and water have been quoted as means of transmission of haemorrhagic septicaemia (Geering, et al. 1995) and other strains of *Pasteurella multocida* (Thomson, et al., 1992) and certainly *Pasteurella multocida* can survive for months in water (Bendheim & Even, 1975) but there are no detailed reports of natural transmission other than directly from infected animals.

Release assessment

R1 — the likelihood that a source herd is infected

In a study in Sri Lanka, in areas endemic for haemorrhagic septicaemia, 38% of cattle herds and 48% of buffalo herds experienced outbreaks of the disease over a two year period (De Alwis & Vipulasiri, 1980). Pigs in contact or in close proximity to these herds could be occasionally infected although outbreaks in pigs in Sri Lanka have been described as rare and sporadic (De Alwis, 1993a).

Given this, the likelihood of selecting slaughter-age pigs from an infected source herd was considered to be ‘very low’.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

There are no reports of the age distribution of haemorrhagic septicaemia so it is assumed to affect all ages of susceptible pigs. If the herd was infected, there would be a number of clinically affected animals, not yet infected animals and recovering (possibly shedding) animals. The proportion of animals in these categories would depend on the nature of the outbreak, the stage of the outbreak at which animals were collected for slaughter and the immune status of the animals in the herd. Based on this information, it was considered that the likelihood of selecting an infected animal in an infected herd was ‘low’.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

An acutely affected pig would be expected to show post-mortem signs akin to those described above for cattle and buffaloes. Under the Australian Standard, such an animal would not be passed as fit for human consumption. Animals with bacteraemia would thus be unlikely to pass inspection. On the other hand, symptomless recovering animals could pass inspection. Hence, the sensitivity of ante-mortem, slaughter and processing requirements in detecting and removing *Pasteurella multocida* infected pigs was considered to be ‘low’.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with *Pasteurella multocida* and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a ‘one minus the background rejection rate’. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered ‘extremely low’. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

The most likely location of *Pasteurella multocida* organisms in infected animals would be the tonsils, which are discarded at slaughter. A much lesser likelihood is that the animal in question had bacteria in the blood or lymph nodes, e.g. in the case where it was bacteraemic but managed to pass inspection at slaughter.

Given this, the likelihood that *Pasteurella multocida* would be present in meat harvested for export that was derived from an infected carcass was considered to be ‘low’.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

There are no reports of the pH resistance of *Pasteurella multocida* or the effect on it of post-mortem acidification of the carcass. Experiments on phages of *Pasteurella multocida* and reports on vaccines indirectly suggest that the organism happily survives at the range of pH values which would be expected in a carcass. There is also a known but poorly understood lethal effect from serum on some strains of *Pasteurella multocida* (Diallo & Frost, 2000) but the extent of this effect on any organisms which might be present in muscle is unknown.

For the purposes of this analysis, it was considered the there was a 'high' likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

In survival experiments, *Pasteurella multocida* has been shown to survive better at warmer temperatures. For example, toxigenic *Pasteurella multocida* stored at 4°C was detected for up to 14 days whereas at 15 and 37°C it was detected for more than 49 days (Thomson, et al., 1992). Many authors mention without references that the agent survives no more than a few days in the environment.

Based on this information, it was considered that there was a 'high' likelihood that meat infected or contaminated with *Pasteurella multocida* at the completion of carcass maturation would remain infected during transport and storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was an 'extremely low' likelihood that imported pig meat derived from an individual carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'extremely low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

No reports are evident on the question of the infectious dose of *Pasteurella multocida* causing haemorrhagic septicaemia. Nor are there reports on the route of entry although it is generally assumed to be by the respiratory/nasal/conjunctival route through droplet infection.

The relative efficiency of infection via inhaled droplets as compared to oral ingestion of the agent is not clear but it is assumed the latter route is not as efficient as the former.

On balance, it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of *Pasteurella multocida* to initiate infection was 'low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

In the laboratory, *Pasteurella multocida* is very susceptible to being overgrown by contaminant organisms (De Alwis, 1992). The competitor organisms in a waste unit at ambient temperatures would be expected quickly to outgrow any pasteurellae present. Given this, it was considered that the likelihood that *Pasteurella multocida* would remain viable after exposure to sunlight, putrefaction and the environment in meat scraps discarded in refuse for the period of time for feral pigs to locate and subsequently scavenge the material was 'very low'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = Very low
- Rural regions = Extremely low

- Large towns = Negligible

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be ‘very low’.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a ‘extremely low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of *Pasteurella multocida* to initiate infection was ‘low’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a ‘low’ likelihood that *Pasteurella multocida* would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be ‘extremely low’.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'extremely low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of *Pasteurella multocida* to initiate infection was 'low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a 'low' likelihood that *Pasteurella multocida* would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'very low'.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the three exposure groups - that is, feral pigs, pigs in backyard enterprises and pigs in small commercial piggeries. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

An occurrence of haemorrhagic septicaemia in a susceptible population of pigs would likely be a fairly acute outbreak with significant mortalities and morbidity. Pigs infected with *Pasteurella multocida*, may not move the distances that healthy pigs move, and indeed might not move very much at all whilst infectious. Moreover, feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. It is thus, feasible that the disease could die out in the initially exposed herd of feral pigs.

While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs, however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

The reports on haemorrhagic septicaemia in pigs in parts of Asia suggest that the causative agent is identical to that which causes haemorrhagic septicaemia in cattle and that it is possible the organism may be transmitted between pigs and cattle (Verma, 1988) but epidemiological evidence of transmission between pigs and cattle is lacking (Townsend, et al., 1998).

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: high

Scenario 2: low

Scenario 3: very low

Scenario 4: very low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

During an occurrence of haemorrhagic septicaemia amongst backyard pigs, there would probably be noticeable mortalities and morbidity. However, there is a lesser likelihood that this event would be properly investigated and correctly diagnosed than in a small commercial piggery. Nonetheless pigs in backyard enterprises are often grown and slaughtered on the same premises, which may limit spread of the disease.

The likelihood of spread from a backyard herd to feral pigs would depend on the location of the backyard pig herd, its proximity to feral pigs, the frequency of contact between the domestic and feral pigs, the duration of excretion of *Pasteurella multocida*, and seasonal factors such as the availability of feed.

Spread between pigs and cattle or pigs and buffaloes is not part of the record of the disease in Asia, yet one would expect that reports of just such transmission would be part of the picture if it were common. For instance, to quote one report: "... it has been postulated, without substantial evidence, that haemorrhagic septicaemia in cattle and pig in India may be due to the same serotype." (Verma, 1988). In either cattle or pigs, fairly close contact between animals is required for transmission of haemorrhagic septicaemia.

The expected high level of mortalities in affected herds would suggest that if spread to a number of herds occurred this would lead to rapid identification of the disease and institution of control measures before the disease became widespread.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: low

Scenario 3: low

Scenario 4: very low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species.

As was the case for the backyard pigs, the likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact with either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

A disease which causes significant mortalities is likely to be investigated promptly in a small commercial piggery. However, as the disease can spread via fomites and there is more frequent movement of animals from a small commercial piggery, it is feasible that spread to other piggeries, in particular those in the local area, could occur. Spread to cattle may also occur if located in close proximity to infected animals.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: very low

Scenario 3: moderate

Scenario 4: very low

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each

exposure group follow a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this scenario, haemorrhagic septicaemia would have established in the directly exposed animal, or group of animals, but would not have spread to other pigs or cattle. In the case of a feral pig herd or backyard pig enterprise being infected, this 'no outbreak' scenario would have resulted from low probability of contact between infected and susceptible animals, rather than from human intervention. Indeed, because feral pigs are not closely observed and disease may not be investigated in a backyard enterprise, it was assumed that it would not have been identified in these exposure groups. In the case of a small commercial piggery, due to closer observation, it was assumed that the disease would have been identified and contained due to implementation of a control and eradication program.

The direct impact of haemorrhagic septicaemia

Animal life or health

In the sporadic outbreaks of haemorrhagic septicaemia in pigs in Asia, there have been significant mortalities. For example, an outbreak described in India resulted in mortality of 40% of the herd (Verma, 1988). As spread is limited to within the infected herd in this scenario, the likely impact of *Pasteurella multocida* on animal health was considered unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

Environment

Because haemorrhagic septicaemia is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of haemorrhagic septicaemia

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

This criterion was not rated identically for each exposure group.

It is likely that if haemorrhagic septicaemia were contained within one herd of feral pigs or a single backyard enterprise, it would not be diagnosed within either of these herds. However, if the primary outbreak involved a small commercial piggery it was considered that pigs showing clinical signs would be investigated. If haemorrhagic septicaemia were diagnosed in the directly exposed herd, a control and eradication strategy would probably be instituted. Whilst under AUSVETPLAN, there is no response plan manual for haemorrhagic septicaemia, the disease is included as a Category 4 disease (funded 20% by governments and 80% by the applicable industry(s)) in the Emergency Animal Disease Response Agreement[64]. These diseases are those that could be classified as being mainly production loss diseases. It is likely that control of the disease if diagnosed would be achieved via stamping out infected herds.

[64] http://www.aahc.com.au/eadp/response.htm

Any diagnosis of haemorrhagic septicaemia would likely be followed by surveillance over a wider area to determine whether the infection had spread.

Taking the above factors into account it was considered that the indirect impact of new eradication programs was unlikely to be discernible at any level when the direct exposure group was a feral pig herd or a backyard pig enterprise. This resulted in a rating of 'A' for this criterion. Whereas when the direct exposure group was a small commercial piggery it was considered that the indirect impact of new eradication programs was unlikely to be discernible at the national or State level, and of minor significance at the district or regional level. Thus, a rating of 'C' was assigned to this criterion.

Domestic trade or industry effects

This criterion was not rated identically for each exposure group.

As discussed above, it was considered unlikely that haemorrhagic septicaemia would be diagnosed in either a single feral pig herd or one backyard enterprise. On this basis, the indirect impact of this disease on domestic trade and industry was considered unlikely to be discernible at any level for these two exposure groups, and thus the rating assigned to this criterion was 'A'.

In the case of haemorrhagic septicaemia being diagnosed in a small commercial piggery, it is likely that all animals on the infected premises would be slaughtered. There would be quarantine and movement controls on animals in restricted and control areas surrounding the infected premises. In this scenario, the disease would be eradicated promptly. After consideration of these issues, the indirect impact of haemorrhagic septicaemia on domestic trade and industry when the direct exposure group was a small commercial piggery was considered unlikely to be discernible at that national or State level, and of minor significance at the district or regional level, and a rating of 'C' was assigned to this criterion.

International trade effects

This criterion was not rated identically for each exposure group.

As the disease is unlikely to be diagnosed in a single feral pig herd or backyard enterprise the indirect effects of haemorrhagic septicaemia on international trade for these exposure groups was unlikely to be discernible at any level, and a rating of 'A' was therefore assigned to this criterion.

It is likely that some overseas markets for Australian cattle and pigs would be affected by any outbreak of haemorrhagic septicaemia in pigs in Australia. The OIE Code recommends that cattle exported from an infected country be subject to quarantine for 3 months and testing on four occasions during the last month of quarantine. If these recommendations were applied it would not be feasible to export feeder cattle. Nonetheless the restrictions on trade may be limited in duration and would probably no longer apply after a period of surveillance to demonstrate country freedom. The OIE Code recommends that a country shall be considered free from haemorrhagic septicaemia 6 months after the slaughter of the last affected animal where a stamping-out policy is applied. As this is a very limited outbreak involving one premises, Australia may be able to negotiate with trading partners the recognition of free zones. The OIE Code does not recommend any measures with regard to the importation of meat from a country infected with haemorrhagic septicaemia.

On this basis, the likely impact of haemorrhagic septicaemia, when the direct exposure group was a small commercial piggery, was considered unlikely to be discernible at the national level and of minor significance at the State level. This gave the disease a rating of 'D ' for this criterion.

Indirect impact on the environment

Haemorrhagic septicaemia in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, haemorrhagic septicaemia would have established in a broader population of feral pigs. In the case of spread from a feral pig herd or backyard pig enterprise, the disease would have been contained due to low probability of contact between infected and susceptible animals, rather than from human intervention. Indeed, because feral pigs are not closely observed and disease may not be investigated in a backyard enterprise, it was assumed that it would not have been identified in these exposure groups and feral pigs. In the case of spread from a small commercial piggery to feral pigs, it was assumed that the disease would have been identified in the small commercial piggery and contained due to implementation of a control and eradication program.

The direct impact of haemorrhagic septicaemia

Animal life or health

As with the first scenario, the direct impact on animal health was considered unlikely to be discernible except at the local level. This resulted in a rating of 'B' for this criterion.

Environment

Because haemorrhagic septicaemia is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of haemorrhagic septicaemia

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

This criterion was not rated identically for each exposure group.

The comments for scenario 1 would also apply for this scenario. As such, it was considered that the indirect impact of new eradication programs was unlikely to be discernible at any level when the direct exposure group was a feral pig herd or a backyard pig enterprise. This resulted in a rating of 'A' for this criterion. Whereas when the direct exposure group was a small commercial piggery, it was considered that the indirect impact of new eradication programs

was unlikely to be discernible at the national or State level, and of minor significance at the district or regional level. Thus, a rating of 'C' was assigned to this criterion.

Domestic trade or industry effects

This criterion was not rated identically for each exposure group.

As discussed above, it was considered unlikely that haemorrhagic septicaemia would be diagnosed in either a single feral pig herd or one backyard enterprise. On this basis, the indirect impact of this disease on domestic trade and industry was considered unlikely to be discernible at any level for these two exposure groups, and thus the rating assigned to this criterion was 'A'.

The comments for scenario 1 would apply in the case of the direct exposure group being a small commercial piggery and the indirect impact of haemorrhagic septicaemia on domestic trade and industry was considered unlikely to be discernible at the national or State level, and of minor significance at the district or regional level, and a rating of 'C' was assigned to this criterion.

International trade effects

This criterion was not rated identically for each exposure group.

The comments for scenario 1 would also apply here. As the disease is unlikely to be diagnosed following spread to feral pigs from either a feral pig herd or backyard enterprise the indirect effects of haemorrhagic septicaemia on international trade for these exposure groups was unlikely to be discernible at any level, and a rating of 'A' was therefore assigned to this criterion. Whereas in the case where spread to feral pigs occurred from a small commercial piggery it was considered that the indirect effect on international trade would be unlikely to be discernible at the national level, and of minor importance at the State level. This gave the disease a rating of 'D' for this criterion.

Indirect impact on the environment

Haemorrhagic septicaemia in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, haemorrhagic septicaemia would have established in a local population of backyard piggeries or small commercial piggeries and cattle. The disease would be contained through the diagnosis of disease in pigs and cattle, and the mounting of an eradication program.

The direct impact of haemorrhagic septicaemia

Animal life or health

In this scenario, the disease would have spread to a local population of pigs in backyard enterprises or small commercial piggeries and to cattle or buffaloes. Mortalities would be high in affected herds.

Hence, the direct impact on animal health was, under this scenario, considered unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. Overall, this resulted in a rating of 'C' for this criterion.

Environment

Because haemorrhagic septicaemia is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of haemorrhagic septicaemia

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

The comments for scenario 1 above are also pertinent here. In this scenario, it is assumed that the outbreak would be identified and that counter measures as described above would be applied. As both cattle and pigs are now involved in the outbreak, the measures would be more widespread with greater tracing and surveillance required.

Overall, the indirect impacts of control and eradication programs were considered unlikely to be discernible at the national level, but would be of minor significance at the State level. This resulted in a rating of 'D' for this criterion.

Domestic trade or industry effects

In this scenario, there would be disruption to trade. At a district level, disruption to movement of cattle and pigs would occur for the duration of the outbreak. This would have a significant impact on affected producers. There could also be interstate movement restrictions until the extent of the outbreak was fully investigated.

Given this, the impact on domestic trade or industry was considered to be unlikely to be discernible at the national level, but would be of minor significance at the State level. This resulted in a rating of 'D' for this criterion.

International trade effects

The comments for scenario 1 above are also pertinent here. It is likely that some overseas markets for live cattle and pigs would be affected by any outbreak of haemorrhagic septicaemia in Australia. However, this would probably no longer apply after a period of surveillance to demonstrate country freedom. Zoning may be applicable with a local outbreak of the disease.

On this basis, the likely impact of haemorrhagic septicaemia was considered unlikely to be discernible at the national level, and of minor significance at the State level. This gave the disease a rating of 'D' for this criterion.

Indirect impact on the environment

An outbreak of haemorrhagic septicaemia as described by this scenario is likely to have indirect environmental impacts resulting mainly from the disposal of animal carcasses. Additional impacts could arise from the widespread use of disinfectants to decontaminate infected properties.

Given this, the indirect impact on the environment was considered unlikely to be discernible at the national and State levels, and of minor importance at the district or regional level. This resulted in a rating of 'C' for this criterion.

Indirect impact on communities

In this outbreak scenario, it was considered unlikely that this disease would have a measurable impact on communities such as reducing rural and regional economic viability, and a rating of 'A' was thus assigned to this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, haemorrhagic septicaemia would have established in a broader population of commercial piggeries (including medium-large piggeries) and cattle. An eradication program would have been mounted in response to the diagnosis of the disease in pigs and cattle.

The direct impact of haemorrhagic septicaemia

Animal life or health

In this scenario, there would be a greater impact on animals and it would occur over a wider area than that described for scenario 3. There would be high morbidity and mortality in affected pig and cattle herds. Given this, it was considered that the direct impact on animal health would be of minor significance at the national level. This resulted in a ranking of 'E' for this criterion.

Environment

Because haemorrhagic septicaemia is not known to affect native Australian species, its direct impact on the environment would not be discernible at any level. This resulted in a rating of 'A' for this criterion.

The indirect impact of haemorrhagic septicaemia

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

The comments for scenario 1 above are again pertinent here. In this scenario, the outbreak would be identified and control measures as described above would be applied. These measures would be expected to be applied over a wider geographic area and would be prolonged. Overall the indirect impact of control and eradication programs was considered to be of minor significance at the National level. This resulted in a rating of 'E' for this criterion.

Domestic trade or industry effects

The indirect effects on domestic trade and industry would be similar to those described for scenario 3 except over a wider area. Disruption to movement of cattle and pigs would occur for the duration of the outbreak. Associated industries such as transport, meat processors and stock feed manufacturers' may also be affected. Given this, the impact on domestic trade or industry was considered to be of minor significance at the national level. This resulted in a rating of 'E' for this criterion.

International trade effects

With a more general outbreak of haemorrhagic septicaemia involving commercial pig and cattle herds, exports of these animals could cease for a prolonged period. As discussed above, the OIE Code recommends that a country be considered free 6 months after the slaughter of the last affected animals if a stamping-out policy is practised. Given this, the indirect effect on international trade was considered to be of minor significance at the national level, and a rating of 'E' was assigned for this criterion.

Indirect impact on the environment

An outbreak of haemorrhagic septicaemia in a general population of pigs and cattle, as described by this scenario, is likely to have indirect environmental impacts resulting mainly from the disposal of animal carcasses. Additional impacts could arise from the widespread use of disinfectants to decontaminate infected properties.

Overall, it was considered that the indirect impact on the environment would be of minor importance at the national level. This resulted in a rating of 'E' for this criterion.

Indirect impact on communities

An outbreak of haemorrhagic septicaemia would affect the rural and regional economic viability including such things as businesses reliant on livestock revenue, employment, local governments together with social costs to individuals and communities. It is evident that those communities that are highly dependent on livestock industries would be affected with associated job losses and social consequences.

Considering these factors, the indirect impact of haemorrhagic septicaemia on rural communities was considered unlikely to be discernible at the national level, but would be of minor significance at the State level. Overall, this resulted in a rating of 'D' for this criterion.

The overall impact of haemorrhagic septicaemia

When the direct and indirect impacts of haemorrhagic septicaemia were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible (feral pigs, backyard pigs); low (small commercial piggeries)

Scenario 2: Consequences negligible (feral pigs, backyard pigs); low (small commercial piggeries)

Scenario 3: Consequences low

Scenario 4: Consequences moderate

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 101, Table 102, and Table 103. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were considered 'very low', 'very low' and 'low' respectively.

Table 101 Haemorrhagic septicaemia: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	High	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Very low	Low	Negligible
Scenario 4	Very low	Moderate	Very low
		Overall likely consequences	**Very low**

Table 102 Haemorrhagic septicaemia: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Low	Low	Very low
Scenario 4	Very low	Moderate	Very low
		Overall likely consequences	**Very low**

Table 103 Haemorrhagic septicaemia: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Low	Low
Scenario 2	Very low	Low	Negligible
Scenario 3	Moderate	Low	Low
Scenario 4	Very low	Moderate	Very low
		Overall likely consequences	**Low**

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with haemorrhagic septicaemia.

Table 104 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly less than Australia's ALOP (very low), risk management would not be required for haemorrhagic septicaemia.

Table 104 Haemorrhagic septicaemia: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Extremely low	Very low	Very low	Negligible
Backyard pigs	Extremely low	Extremely low	Very low	Negligible
Small commercial piggeries	Extremely low	Very low	Low	Negligible
			Overall annual risk	**Negligible**

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Japanese encephalitis virus

Technical information

Background

Japanese encephalitis (JE) virus has historically been recognised as an important zoonosis in Asia and more recently in the Australasian region, India and Pakistan. It is responsible for about 15,000 deaths and 50,000 cases of encephalitis in humans annually (Mackenzie, et al., 1998). Seasonal occurrence of an encephalitic disease in humans distinct from encephalitis lethargica "Type A" was recognised in humans in Japan as early as 1924. It was termed "Type B" encephalitis to differentiate it from "Type A" (Burke & Leake, 1988). The "B" has been dropped from the description of the human disease and it is now recognised that humans are not important in JE virus ecology.

Agent taxonomy

Order	Mononegavirales
Family	Flaviviridae
Genus	*Flavivirus*

The genus *Flavivirus*, of which yellow fever virus (L. flavus = yellow) is the type species, has over 50 species, divided into 12 serologically related groups (International Committee on the Taxonomy of Viruses, 2002). The JE virus group contains 8 antigenically related viruses. Infections with different viruses, within the JE virus group, may be difficult to distinguish serologically. Many of the flaviviruses have an arthropod vector.

Agent properties

Flaviviruses are single stranded positive sense RNA viruses with a genome of about 11,000 base pairs, spherical in shape, 40 to 60 nm diameter with a lipid envelope. Most have arthropod vectors but are rarely mechanically transmitted. Flaviviruses are inactivated by ultraviolet light, gamma irradiation, disinfectants (eg formaldehyde, glutaraldehyde, hydrogen peroxide, chlorine, alcohol and iodine) (Monath & Heinz, 1996).

Japanese encephalitis virus is unstable in the environment and susceptible to detergents, organic solvents, and proteolytic and lipolytic enzymes. At 50°C, 50% of its infectivity is lost in 10 minutes and it is inactivated after 30 minutes at 56°C (Monath & Heinz, 1996). Japanese encephalitis virus has optimal stability at pH 8.0 and is labile below pH 6.0 (Parsonson, 1997; Joo & Chu, 1999). Nawa (1996) investigated exposure of extracellular JE virus to decreased pH and found a 10^4 reduction in infective titres at pH 6.0. He surmised that conformational changes in the virus envelope protein at acidic pH led to loss in haemagglutinin activity and thus to decreased infectivity. The sensitivity of flaviviruses to low pH, bile, proteolytic and lipolytic enzymes renders oral infection unlikely (Monath & Heinz, 1996).

Host range

Japanese encephalitis virus affects a multitude of both homeothermic and poikilothermic animals, though the latter do not appear to be important in transmission cycles (Burke & Leake, 1988). Differing syndromes are seen in different species: encephalitis in humans and horses,

abortion and stillbirth in pigs. Other animals (eg sheep, goats, dogs, rodents, bats) may become infected, rarely show clinical signs but develop varying levels of viraemia and antibody (Mackenzie, et al., 1998). Clinical signs are not seen in infected ardeid birds (Note: the family Ardeidae includes herons, bitterns and egrets), some of which are considered important in natural transmission cycles.

Virus survival depends also on an ability to infect and multiply in vector mosquitoes. A number of *Culex*, *Aedes* and *Anopheles* spp. mosquitoes have been implicated in transmission (Ellis, et al., 2000). Vector efficiencies vary both between and within species of mosquitoes (Hardy, 1988), and a recent phylogenetic analysis has placed JE virus in a "Culex clade" of encephalitic viruses based on NS5 and E sequences(Gaunt, et al., 2001). Suitable vectors for JE virus exist in Australia; *C. annulirostris* was implicated as the vector in outbreaks in the Torres Strait in 1995.

Epidemiology

Japanese encephalitis virus occurs as far west as India and Sri Lanka; China to Japan and Korea in the north east; and South East Asia to the Philippines and Torres Strait in the east and south east. The distribution appears to be expanding as evidenced by incursions into Pakistan in 1992 and Torres Strait in 1995 and subsequent years. Five genotypes have been described with differing geographic distributions. Differences in antigenicity and receptor binding have been related to dispersal of 'newer' genotypes (Solomon, et al., 2003).

Maintenance of JE virus involves cycles between vectors and various hosts each of which may differ in importance depending on the region. Only pigs and birds develop viraemia sufficient to infect significant numbers of mosquitoes (Burke & Leake, 1988). Mackenzie et al (1998) reviewed the possible role of bats in virus maintenance and transmission and recommended research on the part that bats might play in natural transmission cycles.

High proportions (up to 100%) of slaughter-age pigs show positive serology to JE virus in infected areas (Tsai, 1998). The age structure of commercial pig populations ensures a continual supply of large numbers of susceptible pigs. Such situations can lead to infection of high proportions of vectors and spill-over infection into human and other animal populations. Although pigs develop high viraemias (4 days duration, 10^6 suckling mouse intra-cerebral LD_{50}, $SMICLD_{50}$) (Burke & Leake, 1988), they are not essential for virus transmission and JE occurs on islands without pigs and in areas where there are few pigs (Rosen, 1986; Mackenzie, et al., 1998). The appearance of JE epidemics in Japan have been attributed to changes in pig management in the latter 19th century (Rosen, 1986). Human agricultural and animal husbandry practices have resulted in alterations to JE virus ecology, with changes to distribution of both hosts and vectors in many parts of Asia and South East Asia, resulting in altered distribution of the virus.

Japanese encephalitis is essentially a disease of humans and horses, though the ratio of infection to clinical cases is very low for both species. In contrast, mortality:morbidity rates are high, up to 1:2 for horses and 1:4 in humans (Calisher & Walton, 1996; Mackenzie, et al., 1998). A horse to horse infection cycle with the mosquito vector *Culex tritaeniorhynchus* has been shown possible experimentally with viraemic titres in the index horse as low as $10^{1.2}$ $SMICLD_{50}$ per 0.03ml of serum (Gould, et al., 1964). Under natural conditions, humans and horses are effectively dead-end hosts for the virus as they do not develop virus titres capable of infecting large numbers of mosquitoes. Age structures and life expectancy in horse and human

populations mean that, even in endemic areas, susceptible individuals of these species are present in low proportions, presenting further barriers to virus dispersal.

While the potential for mechanical transmission by vectors exists the most important mode of transmission of arboviruses is biological transmission. The virus multiplies in the female arthropod vector during a "lag phase" dependent on virus dose, ambient temperature and vector species. After ingestion the virus particle penetrates the cells of the mesenteron and subsequently enters the haemocele to eventually enter and replicate in other organs including salivary glands and reproductive organs (Burke & Leake, 1988). Virus in salivary glands is then available to infect another host during the following blood meal by the female mosquito. Competent vectors exist widely in Australia. *Culex annulirostris* is a known vector implicated in transmission in the outbreaks of JE in the Torres Strait in 1995 and 1998. *C. annulirostris* is prevalent in the Murray-Darling drainage basin in Queensland, New South Wales, Victoria and South Australia, and in many parts of coastal northern and eastern Australia.

The role that wildlife might play in JE cycles in Australia remains unknown. Limited studies at the Australian Animal Health Laboratories have shown low viraemias after parenteral inoculation in the eastern grey kangaroo (*Macropus giganteus*), tammar wallaby (*Macropus eugenii*) and the agile wallaby (*Macropus agilis*). Brush tailed possums (*Trichosuris vulpecula*) developed viraemias up to $10^{3.}$ PFU/ml; rabbits (*Oryctolagus cuniculus*) failed to show detectable viraemia but developed antibody detectable by c-ELISA[65]. Mackenzie and Ritchie (2001) caution that mosquito infection may occur even with low levels of viraemia.

The epidemic and seasonal nature of JE outbreaks in temperate zones indicates that an arthropod vector is the major form of transmission in animals. Oral transmission to scavenging carnivores would produce non-seasonal outbreaks and is not considered to be important. A continuous host vector transmission cycle is perpetuated in tropical and some subtropical areas but there has been some doubt about modes of virus survival in temperate areas during winter. Reintroduction by migrating birds or infected mosquitoes has been proposed; the latter is thought to have been the method of introduction of the virus to Cape York Peninsula, with conditions conducive for vector incursion occurring almost annually (Ritchie & Rochester, 2001). The annual regularity with which JE appears in parts of the northern hemisphere does not support reintroduction by arthropods. Japanese encephalitis virus strains from northern temperate areas are genetically distinct from tropical isolates, suggesting local maintenance cycles (Chen, et al., 1990). Rosen (1986) considered whether the virus may overwinter in reptiles, but concluded that this was unlikely. Transovarial transmission has been shown experimentally (Rosen, et al., 1978) and Rosen (1986) reports isolation of JE from adult mosquitoes raised from field collected larvae, but its importance in natural cycles has not been elucidated.

Clinical signs

That high numbers of pigs show serological evidence of infection without clinical signs suggests a disease of minimal clinical importance in pigs. Clinical signs of infection with JE virus in pigs are limited to production of stillborn and mummified foetuses and occasional neurological signs in piglets. In addition, subcutaneous oedema and hydrocephalus in some stillborn piglets have been seen in experimental infections. Boars may suffer orchitis and infertility (Joo & Chu, 1999) but these signs are not pathognomonic as there are many porcine diseases involving reproductive disorders. Japanese encephalitis virus infections will increase

[65] D. Middleton, pers comm., 2003

time to first farrowing and cause economic losses in gilts, but could remain undiagnosed in the absence of veterinary investigation or human disease.

Human infections with JE virus are generally subclinical but the small proportion showing clinical signs have a poor prognosis. Clinical signs in humans range from an influenza-like illness to fatal meningoencephalomyelitis. Some 30% die and another 35% are left with varying neurological sequelae (Solomon, et al., 2000). There are reports of virus isolation from human foetuses after abortion following infection of pregnant women during the first and second trimesters (Tsai and Yu (1994), cited by Mackenzie, et al., 1998).

In horses, three syndromes have been described, in increasing order of severity: transient, lethargic and hyperexcitable (Ellis, et al., 2000). The latter syndrome commonly progresses to collapse and death. Case fatality rates from 5 to 15% are reported (Ellis, et al., 2000) but Calisher and Walton (1996) cite case fatality rates exceeding 50% for horses in Japan between 1947 and 1965.

Pathogenesis

Virus multiplication in pigs has not been extensively studied, though transplacental infection has been reported in pigs and mice (Joo & Chu, 1999). A number of models for infection, from laboratory mice to primates, have been used. Caution should be used when extrapolating between species because of the considerable species differences in observed clinical signs. For example, birds are not reported to show clinical signs, however, pigs, despite developing a high viraemia, rarely if ever suffer neurological signs. Viraemia in pigs lasts 12 hours to a few days (Joo & Chu, 1999). Horses develop fatal encephalomyelitis with low virus titres but cattle show evidence of infection by seroconversion yet are not reported to show clinical signs or develop high viraemias. Genetically determined resistance to JE virus infection has been described in mice.

In animals in which virus multiplication has been studied, replication occurs initially in local lymph nodes following the bite of an infectious mosquito, followed by a transient viraemia (Mackenzie, et al., 1998). Further replication in vascular tissues (spleen, liver, muscle) then augments the initial viraemia (Johnson, 1987; Joo & Chu, 1999). Interstitial myocarditis has been reported in humans and horses (Monath & Heinz, 1996). The mechanism of virus entry into the nervous system has not been definitively described. Studies in rats, which are resistant to infection with JE virus, have demonstrated virus tropism for developing neurones (Kimura-Kuroda, (1993) cited by Mackenzie, et al., 1998). The virus is also selectively neurotropic in mosquitoes, with temperature dependent sequential infection of fat tissues, nervous tissues (including retinae and ganglia) and then salivary glands, oviduct and ovaries (Leake & Johnson, 1987).

Infection and virus multiplication in vectors are well documented. No adverse effects have been reported in vectors (Burke & Leake, 1988).

Pathology

Gross lesions are not found in pigs infected after birth, but central nervous system (CNS) histopathology is evident in pigs up to 6 months of age, where a nonsuppurative encephalitis with neuronal degeneration is seen in both cerebrum and cerebellum. Testes may show oedema and inflammatory changes with haemorrhage in the interstitial tissues and degenerative changes of the seminiferous epithelium. Aborted/stillborn foeti show gross lesions involving fluid

retention in body cavities, congestion of lymph nodes, varying degrees of hypoplasia of CNS tissues and focal necrosis of liver and spleen (Joo & Chu, 1999).

In humans, histopathological lesions are described by Mackenzie et al (1998) as mostly confined to the neurones and blood vessels of the CNS. Invasion of neurones by virus particles is followed by perivascular cuffing and infiltration of inflammatory cells. Johnson (1987) describes inflammation, neuronophagia and the formation of glial nodules, as depending on the acuteness of the infection. A similar pattern of inflammation and neuronal destruction is described in horses (Ellis, et al., 2000).

Immunology

Monoclonal antibody and cross neutralisation studies have indicated a number of strains of virus, though strain variation is minor (Burke & Leake, 1988). Strain variations reflect differences in pathogenicity, virulence, heat stability, haemagglutinin and haemolytic activity (Burke & Leake, 1988) but do not appear to have affected protection by the vaccine strain in use since initial isolation in 1935. Close antigenic relationships with Kunjin, Murray Valley Encephalitis and other flaviviruses presented difficulties in analysis of serological results from surveillance and sentinel herd testing following the JE virus incursions into Torres Strait and the Northern Peninsula area of Cape York of Australia. Neutralisation tests are the most definitive but still require antibody titre comparison to determine the virus involved. This problem even occurs with sentinel pigs with a naïve immune status to flaviviruses.

Transmission via meat

Japanese encephalitis virus is not known to be infectious orally. Japanese encephalitis virus infection by ingestion has not been investigated experimentally but is not considered likely to be successful due to the acid pH lability of the virus and its susceptibility to the action of bile and proteolytic, and lipolytic, enzymes (Monath & Heinz, 1996). However, the tick borne encephalitides (TBE: Russian spring summer encephalitis and Central European encephalitis, both flaviviruses) are transmissible to humans by ingestion of unpasteurised milk and cheese from infected small ruminants (Monath & Heinz, 1996). Despite similar conformational changes in the E protein on exposure of JE virus to acid pH, significant resistance on exposure to acid pH is reported for TBE viruses (Monath & Heinz, 1996).

Aerosol infection has been used experimentally in mice, hamsters, guinea pigs, rats and squirrel monkeys (Larson, et al., 1980). The mice and hamsters were susceptible to this route of infection at doses down to $10^{1.2}$ plaque forming units (pfu) in weanling mice, but guinea pigs and rats were uniformly resistant. Squirrel monkeys were infected at high doses (10^{6} pfu). Aerosol susceptibility of pigs is not known. Larson et al (1980) determined JE virus survival in aerosols generated from virus suspended in nutrient medium at 24°C and found half lives of 26.5 min, 20.9 min and 17.3 min at 30%, 55%, and 80% relative humidities respectively. The same study noted the possibility of high titres in the upper respiratory tract of mice being able to generate infectious aerosols.

Peroral infection of lizards feeding on mosquitoes has been demonstrated experimentally. A single infected mosquito was enough to initiate infection in the Japanese skink, *Eumeces latiscutatus* (Oya, et al., 1983). It was not reported whether the mosquitoes were alive or dead when force fed to the lizards.

The potential for transmission in pig meat, either orally to another (carnivorous) host or to a mosquito vector is unknown. Virus titres as low as $10^{1.2}$ $SMICLD_{50}$/0.03mL in horses have

been used to infect *C. tritaeniorhynchus* (Gould, et al., 1964) but it is not known whether titres of this magnitude would remain in pig meat after slaughter and exposure, or whether mosquitoes might even feed on such material. Under experimental conditions, *in vitro* infection of mosquitoes is achieved by exposure to film covered blood reservoirs of known titre. Such conditions are forced by the lack of other energy or protein sources under experimental conditions. Nonetheless it was considered very unlikely that mosquitoes would become infected in the field via feeding on meat scraps.

Release assessment

R1 — the likelihood that a source herd is infected

In pig producing countries, and where JE infection is endemic, there are likely to be few barriers to prevent infection of pig herds with JE virus. The use of insect screening and site consideration for prevention of exposure to arthropods is not considered a usual husbandry practice. In temperate regions the winter period will significantly reduce exposure levels, because of reduced exposure to mosquitoes. Endemic JE in pig producing areas will result in up to 100% of pig populations showing serological evidence of infection (Scherer, et al., 1959). Given this, the likelihood that slaughter-age pigs have been selected from an infected herd in a country where JE is endemic was considered to be ‘high’.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

Within herd serological prevalences in pigs may approach 100%. While in piglets this may reflect maternal antibody, in endemic areas, infection is expected to be circulating and affecting young pigs as maternal antibody declines and they become susceptible. Viraemia lasts about four days in pigs.

On this basis, it was considered that the likelihood of selecting an infected pig in an infected herd was ‘very low’.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

Pigs of slaughter-age are not reported to show clinical signs of JE virus infection. Neither ante-mortem nor post-mortem inspection is expected to detect infected pigs. There are no processing procedures that would identify and reject infected pigs. Considering this, the sensitivity of ante-mortem, slaughter and processing procedures in detecting and removing infected pigs was considered ‘negligible’.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with JE virus and are *not* removed from the export chain because of other illnesses or abnormalities. In some

applications, this has been described as a ‘one minus the background rejection rate’. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered ‘extremely low’. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

Though virus replication in pigs has not been well studied, vascular tissues (spleen, liver, muscle) are considered replication sites in other species. Viraemic pigs have titres up to 10^6SMICLD$_{50}$/0.03 ml serum. Meat at slaughter is expected to contain varying amounts of blood despite ideal slaughter practices which aim for complete exsanguination. In view of this, the panel considered that the likelihood that JE virus would be present meat harvested from an infected pig at slaughter to be ‘moderate’.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

Japanese encephalitis virus is labile in even slightly acidic environments. While JE virus has not been isolated from carcasses, destruction rates are not known and the time taken for virus titres found in pigs to be reduced below infectious levels are unknown. Virus would be exposed to proteolytic enzymes and expected acidy changes to pH 6.2 and below in muscle cells undergoing necrosis. Virus free in serum of an animal viraemic at slaughter may be protected from some of these conditions by the buffering effects of electrolytes in serum. Thus, it was considered that there was a ‘very low’ likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

Cold temperatures *per se* are not expected to affect JE virus. Thus, it was considered that there was a ‘high’ likelihood that meat infected at the completion of carcass maturation would remain infected during transport and storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was an ‘extremely low’ likelihood that imported pig meat derived from a carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was an ‘extremely low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

Two methods of initiation of infection are addressed: infection via a mosquito vector and direct infection of pigs by ingestion.

Under experimental conditions, *in vitro* infection of mosquitoes is achieved by exposure to film covered blood reservoirs of known titre. Such conditions are forced by the lack of other energy or protein sources under experimental conditions. The likelihood of successful infection of vectors from waste material is influenced by virus titre, mosquito survival, ambient temperature, vector competence and other factors. Exposure of meat waste is considered highly unlikely to initiate viable infection in mosquito vectors.

Infected pigs at slaughter may show high virus titres in serum, up to 10^6 $SMICLD_{50}$/0.03mL; at some stages during infection, and muscle tissue may be involved in virus replication. If infected meat was ingested, adverse survival conditions for JE virus in a pigs gastrointestinal tract would mean that only well protected infected material which was able to cross the digestive mucosa could be considered infectious. Oral infection in mammals is not recorded, though the opportunity for this method of infection in pigs must have occurred. An oral infectious dose is not known for pigs.

On balance, the likelihood that a waste unit from an infected pig would contain a sufficient dose of JE virus to initiate infection was considered to be ‘very low’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

Japanese encephalitis virus is susceptible to desiccation and to ultraviolet light; bacterial degradation and putrefaction of meat are not expected to be conducive to virus survival. The virus is unstable in the environment and is labile at high temperatures.

On balance, it was considered that the likelihood that JE virus would survive within meat scraps discarded in refuse for the period of time required for feral pigs to locate and subsequently scavenge the material was ‘extremely low’.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low

- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = Extremely low
- Rural regions = Negligible
- Large towns = Negligible

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be ‘extremely low’.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was an ‘extremely low’ likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was considered to be ‘very low’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances meat may be being discarded due to spoilage.

Nonetheless, the Panel still considered that there was an ‘extremely low’ likelihood that JE virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'negligible'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was an 'extremely low' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected would contain a sufficient dose of the pathogenic agent to initiate infection orally was considered to be 'very low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances meat may be being discarded due to spoilage.

Nonetheless, the Panel still considered that there was an 'extremely low' likelihood that JE virus would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be ‘extremely low’.

Exposure assessment for other susceptible species

The possibility of infection of arthropod vectors from meat and subsequently transmitting infection to pigs has already been addressed. Exposure of susceptible carnivorous and omnivorous vertebrates other than pigs is considered in this section.

Wild and feral carnivores, both placental and marsupial, may be exposed to waste pork material. Avian reservoirs of JE virus (ardeid birds), apart from fishing habits, may forage and thus be exposed to waste material. Oral infectious doses are not known for any of these animals, if in fact oral transmission is possible. Bats are considered unlikely to forage on waste meat.

Reptiles may forage on waste meat material, and the Japanese skink is known to have been infected orally. A suitable dose of viable virus could initiate infection in a similar animal. The part this might play in the initiation of an outbreak is unknown, but considered highly unlikely.

On balance, it was considered that the overall annual likelihood of entry and exposure for other susceptible species was ‘extremely low’

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete ‘outbreak scenarios’ for each of the four exposure groups - that is, feral pigs, pigs in backyard enterprises pigs in small commercial piggeries and other susceptible species. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of ‘consequences’ for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or to other susceptible animals - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs, however, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

Infection of feral pigs with JE virus has previously been reported in Australia, and virus has been isolated from both humans and pigs in the Torres Strait and the Cape York Peninsula, in 1995 and 1998. Islands in the Torres Strait appear to be reinfected during monsoon conditions suitable for vector dispersal. Japanese encephalitis virus appears so far to have failed to establish either in the islands of Torres Strait or in the mainland Cape York Peninsula. Reasons for failure to establish on the islands may involve lack of suitable numbers of pigs or other susceptible animals to establish a continuous cycle and possible cross protection from other endemic flaviviruses. The reasons for failure to establish on the Cape York Peninsula are less clear, because detection of virus in the region of the Mitchell River places infection in areas of high feral pig densities. Possible reasons have included vector preferences, cross protection from other flaviviruses and lack of adequate numbers of susceptible hosts to maintain virus circulation.

Spread of JE virus from feral pigs is not expected to be by direct contact. Suitable vectors exist in many parts of Australia and spread after an initial outbreak, even from the index case, will depend on vectors becoming infected and finding susceptible hosts at a suitable time for virus transmission. Further spread would then depend on the development of viraemias in the newly infected animals , which are infective for vectors. Thus initial (feral) pig density, food availability and behaviour factors become important in assessing the likelihood of further spread. Vector factors (e.g. competence, abundance, mobility and host preferences) and virus factors (e.g. virulence, cross protection from other flaviviruses) will be critical to virus establishment. Establishment of a sylvatic cycle involving ardeid birds may be possible in the absence of suitable mammalian hosts. These factors make it difficult to predict the outcome of an incursion of JE virus.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: low

Scenario 3: moderate

Scenario 4: moderate

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for indirect spread to feral pigs other domestic pigs, other hosts or humans by arthropod vectors.

Abundance of arthropod vectors will depend on situation and climatic conditions, and the likelihood of spread to the availability of suitable, susceptible hosts for virus multiplication. Such conditions exist in a number of pig raising areas in Australia but may not exist in other areas. These factors have been discussed above. It is considered that the likelihood of spread to humans (in the presence of a suitable vector) could be increased in this situation, as could the possibilities of spread to ardeid birds.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: low

Scenario 3: moderate

Scenario 4: moderate

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the 'no outbreak' scenario;

2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The distinction between 'small commercial piggeries' and 'backyard piggeries' is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for indirect spread to feral pigs, other domestic pigs, other hosts or humans by arthropod vectors.

Abundance of arthropod vectors will depend on situation and climatic conditions, and the likelihood of spread to the availability of suitable, susceptible hosts for virus multiplication. Such conditions exist in a number of pig raising areas in Australia but may not exist in other areas. These factors have been discussed above. It is considered that the likelihood of spread to humans (in the presence of a suitable vector) could be increased in this situation, as could the possibilities of spread to ardeid birds.

Commercial piggeries, by the nature of their operation, are likely to provide a continuous pool of susceptible young pigs, such that viraemia could be maintained whenever vectors were available. Endemicity in suitable climatic areas is a possibility. For this reason it was considered that the likelihood of spread to a more general population of domestic pigs was higher.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: low

Scenario 3: low

Scenario 4: high

Other susceptible species

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of other susceptible species:

1. Containment of the disease within the sub-population of directly exposed animals - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;

3. Secondary spread (via feral pigs or other means) to a local population of domestic pigs - containment within a local population of backyard enterprises or small commercial piggeries - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs - exposure of medium-large commercial piggeries and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Ardeid birds are known to maintain virus infection cycles in the absence of pigs. Where any other susceptible species is present, and suitable vectors exist, there is potential for transfer to those susceptible animals. Other animals - mammals and reptiles that may be susceptible to infection - are considered to be "dead end" hosts, whether a proportion of them show signs of being infected or not.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: low

Scenario 3: low

Scenario 4: high

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this scenario, the disease is most likely to have established amongst exposed animals, and to have run its course without identification. This is because infection is asymptomatic in pigs, and because it may be self-limiting within a discrete population. Horses and humans (the only animals which are expected to show serious clinical signs) are not considered a primary exposure group. Under a 'no outbreak' scenario, the disease would not have any discernible direct or indirect impacts.

On this basis, a rating of 'A' was assigned to each of the direct and indirect criteria.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, JE virus would have established in a broader population of feral pigs, and spread to humans. The disease would be diagnosed through illness in humans. A surveillance program would be implemented. In this scenario, the disease does not spread to other susceptible animals such as horses or the commercial pig population.

The direct impact of Japanese encephalitis

Animal life or health

Japanese encephalitis virus infection in pigs is often subclinical. If clinical signs are present these may be limited to stillborn and mummified foetuses and occasional neurological signs in piglets. Hence the direct impact on animal health was considered unlikely to be discernible at any level. This resulted in a rating of 'A' for this criterion.

Environment

Environmental effects were not expected to be discernible at any level with spread to feral pigs, and the rating assigned to this criterion was therefore 'A'.

The indirect impact of Japanese encephalitis

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

After detection of JE virus infection in humans, it is likely that surveillance measures in animals will be implemented to determine the extent of disease spread. In the 1998 incursion of JE virus to the Cape York Peninsula, these were the only measures implemented, because disease was not detected in other domesticated animals. An incursion of JE would result in implementation of AUSVETPLAN (Agriculture and Resources Management Council of Australia and New Zealand (ARMCANZ), 1998) involving both State and Commonwealth. Hence the indirect impact of control and surveillance programs was considered unlikely to be discernible at the national level, but of minor significance at the State level. This gave the disease a rating of 'D' for this criterion.

Domestic trade or industry effects

Known establishment of disease in feral pigs without involvement of other susceptible animals will not incur movement controls for animals. Vaccination of horses in known infected areas is likely to be recommended but further procedures are unlikely. Given this consideration, the indirect effects on domestic trade and industry were considered unlikely to be discernible, except locally. This resulted in a rating of 'B' for this criterion.

International trade effects

Effects on international trade are difficult to predict. It is likely that horses from infected zones, if not the whole country, will require certified vaccination complying with manufacturer's requirements for export. Live pigs may require certification of freedom from infection and/or vaccination. Though they do not develop high viraemias and are not involved in disease transmission, trade in live cattle may be at risk. Australia's trading partners to the near north are endemic areas for JE virus, but near Eastern and North African markets may restrict trade until assurances of disease freedom can be given. While such restrictions on cattle exports would be viewed as scientifically unjustifiable, they could at least in the short term, do damage to live exports from affected parts of Australia.

On balance, the indirect effect of JE on international trade was considered unlikely to be discernible at the national level, but of minor significance at the State level. Overall, this resulted in a rating of 'D' for this criterion.

Indirect impact on the environment

Japanese encephalitis in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

Affected areas are likely to have clinical cases of JE within local communities. Human mortalities may ensue. In the 1995 and 1998 outbreaks on the Torres Strait Islands, vaccination of affected communities was implemented and backyard piggeries moved to suitable distances from human settlements. The AUSVETPLAN for JE recommends the use of insect repellents and vaccination for personnel with occupational risks of exposure.

On balance, the indirect impact of JE on communities was considered unlikely to be discernible at the national or State level, but of minor importance to the affected district or region. This resulted in a rating of 'C' for this criterion.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

Under this scenario, JE virus would have established in a local population of backyard piggeries or small commercial piggeries, and spread humans and other susceptible species, such as horses. The disease would be diagnosed through illness in humans or horses, and the strategies outlined in AUSVETPLAN report for JE invoked.

The direct impact of Japanese encephalitis

Animal life or health

Spread to a local population of backyard and commercial pigs may produce noticeable increases in time to first farrowing for gilts, depending on the nature of the operation, and stillbirths and mummified foetuses may be noted. Infected birds, some of which may form a virus reservoir, are not known to be clinically affected. Other affected animals are included in this scenario though only horses will be clinically affected. A proportion of clinically affected horses will die from the disease. Treatment for JE can only be supportive and euthanasia of severely affected horses should be considered.

Given this, it was considered that the direct effect on animal health would be unlikely to be discernible at the national or State level, but of minor importance at the district or regional level. This gave the disease a rating of 'C' for this criterion.

Environment

Environmental effects were not expected to be discernible at any level. Experimental infection of some marsupials did not result in any deaths, but the long term effects of an outbreak of JE on marsupials are not known. Thus, a rating of 'A' was assigned for this criterion.

The indirect impact of Japanese encephalitis

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

The implementation of AUSVETPLAN for JE at both State and Commonwealth level will result from detection of infection in domestic pigs. Vaccination, vector control and restriction on movements of possibly infected pigs are to be expected. Surveillance to define free zone may be implemented and these activities will have inherent expenses.

Hence, the indirect impact of control and surveillance programs was considered unlikely to be discernible at the national level, but of minor significance at the State level. This gave the disease a rating of 'D' for this criterion.

Domestic trade or industry effects

Establishment of disease in commercial pigs is likely to incur movement controls for potentially infected animals. Vaccination of breeding pigs may be required to lower virus load and ensure passage of maternal antibody to young pigs. Vaccination of horses in the known infected area is likely to be recommended but further procedures are unlikely. Zoning may be required. Given this, the indirect effects on domestic trade and industry were considered unlikely to be discernible at the national or State level, but of minor significance at the district or regional level. This resulted in a rating of 'C' for this criterion.

International trade effects

The impact of JE virus on international trade is likely to be similar under this scenario as was described for scenario 2 (see above). A rating of 'D' was assigned to this criterion.

Indirect impact on the environment

Japanese encephalitis in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

An affected area is likely to have human clinical cases of JE. The impact of JE virus on communities is likely to be similar under this scenario as was described for scenario 2 above. A rating of 'C' was assigned to this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, JE virus would have established in a broader population of commercial piggeries (including medium-large piggeries). Spread to humans and other susceptible animals such as horses would have occurred. The disease would have been diagnosed due to illness in humans or horses, and the strategies outlined in AUSVETPLAN report for JE invoked.

The direct impact of Japanese encephalitis

Animal life or health

Spread to large commercial piggeries may produce noticeable increases in time to first farrowing for gilts depending on the nature of the operation, and stillbirths and mummified foetuses may be noted. Infected birds, some of which may form a virus reservoir, are not known to be clinically affected. Other affected animals are included in this scenario though only horses will be clinically affected. A proportion of clinically affected horses will die from the disease. Treatment for JE can only be supportive and euthanasia of severely affected horses should be considered.

Due to the widespread nature of this outbreak, it was considered that the direct effect on animal health would be unlikely to be discernible at the national level, but of minor significance at the State level. This gave the disease a rating of 'D' for this criterion.

Environment

Environmental effects were not expected to be discernible. Experimental infection of some marsupials did not result in any deaths, but the long term effects of an outbreak of JE on marsupials are not known. Thus, a rating of 'A' was assigned for this criterion.

The indirect impact of Japanese encephalitis

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

The implementation of AUSVETPLAN for JE will involve the affected States and Commonwealth governments. Vaccination, vector control and restriction on movements of possibly infected pigs are to be expected. Vaccination of horses would be encouraged. Surveillance to define free zone would necessarily be implemented and these activities will have inherent expenses.

Because of the extent of this outbreak, the indirect impact of control and surveillance programs was considered to be of minor significance at the national level. This gave the disease a rating of 'E' for this criterion.

Domestic trade or industry effects

Establishment of disease in commercial pigs is likely to incur movement controls for potentially infected animals. With a widespread outbreak more animals will be affected by movement controls. Vaccination of breeding pigs may be required to lower virus load and ensure passage of maternal antibody to young pigs. Vaccination of horses in known infected areas is likely to be recommended but further procedures are unlikely. Zoning may be required.

Given this, the indirect effects on domestic trade and industry were considered unlikely to be discernible at the national level, but of minor significance at the State level. This resulted in a rating of 'D' for this criterion.

International trade effects

The impact of JE virus on international trade is likely to be similar under this scenario as was described for scenarios 2 and 3 (see above). A rating of 'D' was assigned to this criterion.

Indirect impact on the environment

Japanese encephalitis in this scenario is unlikely to lead to any indirect impacts on the environment, and a rating of 'A' was thus assigned to this criterion.

Indirect impact on communities

Affected areas are likely to have human clinical cases of JE. The impact of JE virus on communities is likely to be similar under this scenario as was described for Scenarios 2 and 3 above. A rating of 'C' was assigned to this criterion.

The overall impact of Japanese encephalitis

When the direct and indirect impacts of JE were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences low

Scenario 3: Consequences low

Scenario 4: Consequences moderate

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 105, Table 106, Table 107, and Table 108. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were all considered 'moderate'. The likely consequences associated with the exposure of other susceptible species to infected pig meat scraps were considered 'moderate'.

Table 105 JE: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Low	Low	Very low
Scenario 3	Moderate	Low	Moderate
Scenario 4	Moderate	Moderate	Moderate
		Overall likely consequences	**Moderate**

Table 106 JE: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Low	Low	Very low
Scenario 3	Moderate	Low	Low
Scenario 4	Moderate	Moderate	Moderate
		Overall likely consequences	**Moderate**

Table 107 JE: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Low	Low	Very low
Scenario 3	Low	Low	Very low
Scenario 4	High	Moderate	Moderate
		Overall likely consequences	**Moderate**

Table 108 JE: summary of the consequences of exposure of other susceptible species

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Low	Low	Very low
Scenario 3	Low	Low	Very low
Scenario 4	High	Moderate	Moderate
		Overall likely consequences	**Moderate**

Human life or health

Human infections with JE virus are generally subclinical but the small proportion showing clinical signs have a poor prognosis. Clinical signs in humans range from an influenza like-illness to fatal meningoencephalomyelitis. Some 30% die and another 35% are left with varying neurological sequelae (Solomon, et al., 2000). There are reports of virus isolation from human

foetuses after abortion following infection of pregnant women during the first and second trimesters (Tsai and Yu (1994), cited by Mackenzie, et al., 1998). Limited incursions of JE virus into the far northern Cape York Peninsula and the Torres Strait have produced a few cases of encephalitis in humans and two deaths.

Japanese encephalitis virus in populations of susceptible humans in Australia would cause encephalitis with mortalities and high proportions of neurological sequelae of varying degrees. Implementation of vaccination and vector control measures are expected to reduce the impact of an incursion. Vector control measures against *Aedes* spp. mosquitoes are already in place in a number of communities in attempts to reduce transmission of dengue virus, another flavivirus. Vaccination of people with occupational risk and of exposed communities may be undertaken. Commercially, vaccine for human use is available at around $270 for a 3 dose course. Widespread vaccination would entail considerable cost.

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with JE virus.

Table 109 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly less than Australia's ALOP (very low), risk management would not be required for JE virus.

Table 109 JE: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Extremely low	Extremely low	Moderate	Negligible
Backyard pigs	Extremely low	Negligible	Moderate	Negligible
Small commercial piggeries	Extremely low	Extremely low	Moderate	Negligible
Other susceptible species	Extremely low	Extremely low	Moderate	Negligible
			Overall annual risk	**Negligible**

The Department of Health and Ageing has advised Biosecurity Australia that biosecurity measures for JE virus would not be required to manage the risk to human life or health associated with the importation of pig meat. It should be noted that the annual likelihood of entry and exposure of JE virus via pig meat is extremely low or negligible.

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Surra

Technical information

Background

Surra has been recognised in India in camels and horses from ancient times. It was only in the late 19th century that the causative agent was discovered and described. Surra is believed to have originated in camels in tropical Africa and spread with infected animals into the middle East and India and from there into other Asian countries and South America (Hoare, 1972). Although known by various names around the world it is now accepted that the same organism causes all the described conditions in domestic animals and is referred to under the collective term of surra. Surra is primarily a disease of camels and horses but dogs may be affected. Camels develop a chronic wasting disease whilst the disease in horses and dogs is usually acute with high mortality (Losos, 1980). Infection causes mild disease in sheep and goats and is usually asymptomatic in cattle, buffalo and pigs.

Agent taxonomy

Surra is caused by a flagellate protozoan *Trypanosoma evansi* which is a member of the family Trypanosomatidae, subgenus Trypanozoon. Other species (*T. equinum, T. hippicum, T. venezuelense, T. cameli, T. soudanense, T. ninaekohlyakimov*), based primarily on geographical distribution or hosts are no longer recognised and considered synonymous with *T. evansi*.

Agent properties

Trypanosoma evansi is morphologically indistinguishable from the trypomastigote or slender form of *T. brucei* although pleomorphic forms occasionally are seen (Hoare, 1972). It is actively motile and generally about 24 µm long, has well-developed undulating membrane, a long free flagellum, a rounded or truncated narrow posterior end with the kinetoplast situated some distance from the tip and an elongated nucleus (Hoare, 1972). There is growing evidence to support that there is variation in pathogenicity between different isolates of *T. evansi*.

Multiplication is by binary fission and there is no cyclical development in an insect vector (Hoare, 1972). Hoare (1972) proposed that *T. evansi* originated from *T. brucei* that was carried out of the Tsetse zone in Africa in infected camels, thus forcing the development of a parasite adapted to mechanical transmission. Evidence for this theory is found in the observation that isolates of *T. brucei* that have been repeatedly syringe-passaged lose the ability to develop in tsetse flies (Losos, 1980). This lack of cyclical development is probably due to the absence of maxi-circle kinetoplast DNA (kDNA) the gene products of which are required by *T. brucei* for development in tsetse flies (Borst, et al., 1987).

The Trypanozoons (including *T. evansi* and *T. brucei*) survive freezing for many years in a suitable substrate. *Trypanosoma evansi* has been shown to survive long term (10 years) freezing in liquid nitrogen without loss of infectivity (Raether & Seidenath, 1977), and -70°C for 8 years (Hashemi-Fesharki, 1981). Experimentally there was no loss of infectivity of blood samples containing 25 *T. evansi* per ml of blood that were subject to cold storage (4°C) for up to 21 hours (Reid, et al., 2001a). *In vitro T. evansi* maintains infectivity at 5°C for 4 days but glucose was essential (Hogner, 1979). Other *in vitro* studies on the survivability on *T. evansi* in blood samples shows greater survival times at 4°C than at 20°C to 27°C or 37°C (Holland, et al., 2001).

Any samples, regardless of storage temperature, that were exposed to direct sunlight had undetectable levels of parasites after 30 minutes (Holland, et al., 2001). Similar findings have been shown for *T. brucei* with the infectivity and motility of trypanosomes being reduced at temperatures above 36°C (McOdimba, 1990). Reduced infectivity for mice was demonstrated after ultraviolet irradiation for 3 hours (Li, et al., 1996).

The optimum pH range for survival for *T. evansi* is reported to be 7.0 to 8.0 (Hogner, 1979). Similarly, further *in vitro* studies on the slender form of *T. brucei* have shown that it is unable to maintain a constant cytoplasmic pH in media outside of pH 7.0 to 7.5. Although tolerant to high pH of up to 9.5, at pH below 6.5 there was a deleterious effect on motility and metabolism rapidly resulting in death (Nolan & Voorheis, 2000). A high glucose requirement has long been recognised as essential for the survival of Trypanozoon trypanosomes (Hoare, 1972; Hogner, 1979; Michels, et al., 1997).

Host range

Surra has a wide host range potentially infecting all mammals. There is no evidence of natural infection in humans. The principal host species affected varies geographically but camels, horses and dogs are those most severely affected. Camels may be affected acutely or develop a chronic wasting disease and the disease in horses and dogs is usually acute with high mortality. Other animals are also susceptible resulting in chronic, often subclinical infections and reduced productivity in cattle, buffaloes, pigs and sheep. Infections have also been reported in many species of wild animals including deer (Singh, 1998; Reid, et al., 1999; Tuntasuvan, et al., 2000), capybara (Nunes, et al., 1993; Franke, et al., 1994; Arias, et al., 1997), coatis (Nunes, et al., 1993), vampire bats (also transmit surra) (Hoare, 1972), elephants (Losos, 1980), captive tigers (Reddy, et al., 1975; Dasgupta, et al., 1979; Rao, et al., 1995; Rao, et al., 1995; Upadhye & Dhoot, 2000) and leopards (Dasgupta, et al., 1979). Asymptomatic animals may provide a reservoir of infection. Numerous other species have been shown to be susceptible to experimental infections including bandicoot rats (Biswas, et al., 2001), guanaco (Kinne, et al., 2001) and various laboratory animals (rats, mice, guinea pigs, rabbits). Wallabies have also been shown to be susceptible to experimental infection with *T. evansi* (Reid, et al., 2001b).

Although *T. evansi* is considered less pathogenic to pigs than other livestock there have been reports in Asia of clinical disease resulting in mortalities (Kraneveld & Mansjoer, 1947; Sheng, et al., 1983; Gill, et al., 1987; Sirivan, et al., 1989; Arunasalam, et al., 1995). Experimentally infected pigs developed only an intermittent fever (Srivastava & Ahluwalia, 1972; Sheng, et al., 1983) and few trypanosomes could be detected in blood smears although blood samples were infective to rats (Srivastava & Ahluwalia, 1972). Similarly, no clinical signs were observed in another experimental study in pigs but an immunosuppressive effect was demonstrated (Holland, et al., 2003). No reports have been found of differences between breeds of pigs in their susceptibility to infection with *T. evansi* although Onah (1991) found differences in breed susceptibility between exotic and local African breeds to disease in pigs infected with *T. brucei* in Nigeria. Prevalence of infection in traditional free range was similar but clinical disease was only found in exotic breeds.

Epidemiology

Trypanosoma evansi is the most widely distributed of the pathogenic trypanosomes (Hoare, 1972). Infection with *T. evansi* occurs across North and West Africa, the northern part of Kenya and Sudan, the Middle East, southern states of the former USSR, southern China and most countries in South America (Losos, 1980). In south-east Asia *T. evansi* occurs throughout

Indonesia (except Papua), the Malaysian Peninsula, Thailand, Cambodia, Laos, the Philippines and Vietnam (Luckins, 1988).

Trypanosoma evansi infection is spread mechanically from animal to animal by infected blood on the mouthparts of biting flies, especially and most commonly tabanid flies of the genus *Tabanus* (Hoare, 1972). There are conflicting reports regarding the ability of *Stomoxys calcitrans* to transmit *T. evansi.* It is generally regarded in the literature that *S. calcitrans* is capable of transmission (Hoare, 1972; Gardiner & Mahmoud, 1992; Veer, et al., 2002). Infection, however, was not transmitted from goats after allowing unfed flies to partly feed from highly parasitaemic goats and complete their feed on recipient goats and camels (Ngeranwa & Kilalo, 1994).

Tabanids are efficient vectors because they are obligate blood feeders, they have large mouthparts which trap blood, and a painful bite which stimulates defensive behaviour in the host and re-initiation of feeding, possibly on a new host (Foil, 1989). The volume of blood trapped on the mouthparts of tabanids varies between species. Foil *et al*. (1987) showed that *Tabanus fuscicostatus* traps approximately 10 +/- 5 nl of blood. If 10% of residual blood is deposited in the next host when the tabanid resumes feeding then an infective titre must be at least 10^6 organisms per ml of host blood for transmission to occur via a single fly (Foil, 1989).

The efficiency of transmission of *T. evansi* by tabanids is affected by the proximity of host species, the length of time between feeding on an infected host and the re-initiation of feeding on an uninfected host. A feeding time of only 5 seconds is sufficient to acquire an infection from an infected host and *Tabanus* spp. are able to transmit infection for several hours after an infective feed (Luckins, 1988). Foil (1989) suggests that a distance of 200 metres between infected and susceptible horses is sufficient to reduce mechanical transmission of disease by tabanids.

It has been suggested that non-biting flies may transmit *T. evansi* from infected meat to susceptible animals through open wounds or mucous membranes (Weinman and Ristic (1968) cited by Ng and Vanselow (1978)) but this has not been substantiated.

In carnivores it is thought that oral transmission may be another route of infection of *T. evansi* (Hoare, 1972). The vampire bat (*Desmodus rotundus*) has also been implicated as both a reservoir and vector of *T. evansi* in South America (Hoare, 1972).

It is known that in naturally resistant reservoir hosts that trypanosomes of *T. brucei* may only persist in the blood in low numbers or survive in haemopoietic tissues, tissue fluids or cerebrospinal fluid (Hoare, 1972). As *T. evansi* originated from *T. brucei*, and causes similar clinical signs in vertebrate hosts, it is likely to behave in a similar way. An experimental infection of *T. evansi* in goats led to parasites being demonstrated in synovial, peritoneal and cerebrospinal fluid, and their presence in lymph nodes was also demonstrated indirectly after inoculation into mice (Ngeranwa, et al., 1993). The level of parasitaemias in pigs has not been accurately determined. In an experimental infection the parasitaemia in pigs only exceeded 20 trypanosomes per haematocrit tube on 11.5% of occasions samples were collected over 120 days post-infection (Reid, et al., 1999). This was estimated to be approximately 400 trypanosomes per ml of blood (Wernery, et al., 2001) assuming the approximate volume of the haematocrit tube to be 50 microlitres.

Prevalence of infection with *T. evansi* varies in different geographical areas and farming systems. Due to the fact that pigs are not often clinically affected with surra there is little information on the prevalence of the disease in these animals. A survey of livestock in Thailand

reports a prevalence of 4.6% in pigs (Tuntasuvan & Luckins, 1998). This is considerably lower than found in cattle and buffaloes in the same survey with 12.5% and 20% respectively. The prevalence reported in other susceptible animals in the same area may give some indication of potential levels of infection in pigs. There are no data on age prevalence in pigs, but an outbreak in Malaysia involved clinical signs in the breeding stock with no indication of younger animals affected (Arunasalam, et al., 1995). In other susceptible species such as camels it is widely reported that prevalence increases with the age of the animal, although there appears to be no age related differences in susceptibility to infection. In young animals there are conflicting reports with some reporting mortalities in young camel calves and others finding no evidence of infection. Camels kept by nomadic groups tend to have higher prevalence than those kept in pastoral herds (Elamin, et al., 1998). Husbandry systems were also found to influence the prevalence of *T. brucei* in pigs, with a far higher prevalence in traditionally kept free range pigs in Nigeria than those housed in an intensive piggery system (Onah, 1991). Herd prevalence is rarely reported and varies between regions as shown in a camel survey which found 55% of herds infected in one region and 68% in another (Diall, et al., 1993).

Clinical signs

Clinical signs exhibited by animals infected with *T. evansi* vary between host species and between individuals of the same species. Severe clinical disease is most common in horses, camels and dogs. Signs include fever, progressive anaemia, wasting and nervous signs (Gardiner & Mahmoud, 1992). Untreated infection in horses and dogs is usually fatal, with clinical disease lasting 30-90 days, but some horses recover and develop a chronic form of the disease with infrequent peaks of parasitaemia (Hoare, 1972; Gardiner & Mahmoud, 1992).

Acute clinical disease is occasionally seen in cattle, buffalo, sheep and pigs infected with *T. evansi* when they are placed under nutritional or work stress or if naïve animals are moved into an endemic area from an area where *T. evansi* is not present (Luckins, 1988).

Pigs are considered to have a low susceptibility to infection but clinical outbreaks have been reported in Asia. Symptoms in pigs in Indonesia were attributed to *T. evansi* and they responded to treatment (Kraneveld & Mansjoer, 1947). Mortalities in pigs on a farm in India were preceded by signs of fever, shivering, laboured breathing, necrosis of ear and tail tips, eyelid oedema and emaciation (Gill, et al., 1987). Although these pigs were thought to be more susceptible due to concurrent mange other reports suggest *T. evansi* may be pathogenic to pigs. In Malaysia an outbreak presented with clinical signs of pyrexia, anorexia, dullness, chronic emaciation, abortion and death (Arunasalam, et al., 1995). The pigs were also found to have a slightly lowered packed cell volume. In contrast, experimental infections in pigs have produced very mild if any symptoms. A prepatent period of 24 to 30 days was determined in pigs but the only symptom was an intermittent fever 11 to 19 days after inoculation (Srivastava & Ahluwalia, 1972). Parasites were found in pig blood much earlier (within 3 days of experimental infection) in another study and there were no observable signs of disease when pigs were infected with 5×10^8 organisms (Reid, et al., 1999). Other experimentally infected pigs (using 10^6 organisms) were serologically positive from 7 days after infection. No clinical signs were observed and there were no effects on production parameters (Holland, et al., 2003). However, all the experimental infections were carried out on young animals using non-pig isolates which may result in less pathogenic effects.

Domestic animals with mild or subclinical infections are believed to be the main reservoirs of infection, resulting in outbreaks when susceptible animals such as horses and camels are introduced to the area. Wild pigs have been demonstrated to be capable reservoirs in Indonesia,

although less than other animal species because of lower grade and more intermittent parasitaemias (Reid, et al., 1999).

Pathogenesis

The expression of disease due to trypanosomes varies with the strain of parasite and the susceptibility of the host. For example, in highly susceptible animals, such as horses, antibody production is insignificant, there is a constant high parasitaemia, the trypanosomes multiply rapidly and the host dies. In apparently resistant animals such as pigs the hosts defence mechanisms are sufficient to suppress the trypanosomes resulting in scanty numbers persisting in the circulation or localising in haemopoietic tissues or cerebrospinal fluid (Hoare, 1972).

There is no comprehensive review of the pathogenesis of *T. evansi* infection. Most knowledge has been inferred from studies involving infection with *T. brucei*. On the basis that clinical signs of surra in various species are comparable to those caused by *T. brucei*, it is feasible that the pathogenesis is also similar (Losos, 1980). Unlike other pathogenic trypanosomes, *T. brucei* and *T. evansi* are not confined to the plasma of the mammalian host and this has direct implications for the expression of disease due to these parasites. The trypanosomes that locate extravascularly may produce host reactions where they localise. The effects produced by extravascular parasites have been found to be indirect by producing damage in non-invaded tissues. For example, muscle damage in *T. evansi* infected horses has been shown to be due to an inflammatory myopathy rather that direct invasion of skeletal muscle (Quinones Mateu, et al., 1994). The extravascular location of parasites also explains the reason for less persistent and lower level parasitaemias. Parasites may often only be present in the circulation in the terminal stages of the infection. Tissue invasiveness of *T. evansi* has been demonstrated in goats with parasites detected in synovial, peritoneal and cerebrospinal fluids and in lymph nodes after mice inoculation (Ngeranwa, et al., 1993). Parasites have also been detected in bone marrow (Reid, et al., 2001b).

Pathology

Gross pathological changes seen in animals infected with *T. evansi* at post-mortem vary both between species and between individuals of the same species. In experimentally infected pigs there was no gross pathology other than appearing anaemic (Srivastava & Ahluwalia, 1972). Reduced packed cell volume is commonly reported as a prominent feature of trypanosomiasis with the development of anaemia. Eosinophilia and a slight leucopaenia were also found in a natural infection of *T. evansi* in pigs (Arunasalam, et al., 1995). Similarly with *T. brucei* experimentally infected pigs were asymptomatic and showed no differences in carcass quality from uninfected pigs (Ilemobade & Balogun, 1981). In clinical cases of surra reported in naturally infected pigs (Gill, et al., 1987; Arunasalam, et al., 1995) no post-mortem examinations were conducted.

Immunology

Antigenic variation developed by trypanosomes is a complex phenomenon which requires both host and parasite factors. Antigenic variation within a host is only observed in the presence of a competent immune response. Trypanosomes are able to change a variable surface glycoprotein (vsg) resulting in antigenic variants or Variable Antigenic Type (VAT). A population of trypanosomes within a host will comprise a mix of a major and several minor VATs which change as the host immune response eliminates the major VAT, allowing a new VAT to predominate. The total number of antigenic variants expressed by *T. evansi* is not known but

may have a limited antigenic diversity. This may explain the epidemiology of *T. evansi* infection when it is introduced into a non-endemic area where, initially, an epidemic of clinical disease with high mortality occurs followed rapidly by endemic stability (Hoare, 1972). Both humoral and cell-mediated immunity may be involved in the immunological response to *T. evansi.* The invasion of extravascular sites by *T. evansi* trypanosomes may make immunological mediated elimination more difficult for the host to achieve (Gardiner & Mahmoud, 1992).

The existence of several antigenic types in *T. evansi* has restricted the development of a vaccine.

Transmission via meat

Studies have shown that trypanosomes do not invade skeletal muscle (Horchner, et al., 1983; Quinones Mateu, et al., 1994) but they have been found in lymph nodes (Brown & Losos, 1977; Ngeranwa, et al., 1993), bone marrow (Reid, et al., 2001b) and cerebrospinal fluid (Horchner, et al., 1983; Ngeranwa, et al., 1993). Transmission to carnivores from the feeding of meat infected with *T. brucei* (Moloo, et al., 1973) and *T. evansi* (Raina, et al., 1985) and offal (Ray, et al., 1972) has been demonstrated experimentally. Experiments using laboratory animals demonstrated the survival of *T. evansi* trypanosomes for up to 12 hours in carcasses (Sarmah, 1998). Infection was transmitted experimentally to 60% of rats fed infected carcasses (Silva, et al., 1998). One hundred precent of rats in the same study became infected when directly inoculated orally. Reports of natural infections in wild (Wells, 1984) and captive (Wiesenhütter, 1975) carnivores from ingestion of infected meat provide circumstantial evidence but do not preclude the possible transmission by biting flies. Animals dying from surra have been shown to develop a very high parasitaemia (in excess of 10^8 organisms per ml) in the terminal stages (Horchner, et al., 1983).

Studies describing the oral infective dose are lacking. Experimental infections have been transmitted orally in dogs fed 200 ml of blood and 400 grams of meat from goats with a parasitaemia of 10^4 trypanosomes per ml of blood (Raina, et al., 1985). It is difficult to estimate the number of trypanosomes that may be ingested from infected meat when the level of parasitaemia may be scanty or not detectable. Levels of parasitaemias in pigs have been reported in experimental studies using *T. evansi* including a rapid rise peaking at ten days after inoculation and then progressively declining (Reid, et al., 1999). Another study reported barely detectable levels (Srivastava & Ahluwalia, 1972). The direct technique for detecting parasites in these studies (the haematocrit centrifugation technique) has been found to detect 80% of samples containing 250 trypanosomes per ml, 40% with 125 per ml and would not detect less than 31 *T. evansi* per ml (Reid, et al., 2001a). When parasites could not be detected animals were shown to be infected by inoculating blood samples into rodents.

Release assessment

R1 — the likelihood that a source herd is infected

Taking into account variations due to season, geography and the diagnostic test used, the overall seroprevalence in other species in endemic countries is generally less than 30% with up to 65% of herds of camels infected. There are no published reports indicating the herd prevalence of surra in pigs. The only indication in pigs is an overall prevalence of 4.6% in a survey in Thailand.

Given this, the likelihood that slaughter-age pigs have been selected from an infected herd in a country where surra is endemic was considered to be 'low'.

R2 — the likelihood that a slaughter-age pig is infected

There are no published reports of within herd prevalence for pigs. In the few reports of clinical outbreaks of surra it appears older pigs are clinically affected. About 18% of breeding animals were clinically affected in an outbreak in Malaysia. In other species it seems that although surra may be widespread in endemic areas the within herd prevalence has been about 10%. Young animals also tend to be less commonly infected, and it may be presumed that the same is likely for pigs, hence the prevalence is likely to be lower for slaughter-age pigs.

On the basis of this information, it was considered that the likelihood of selecting an infected pig in an infected herd was 'very low'.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

Pigs infected with *T. evansi* are usually asymptomatic. In outbreaks where pigs have shown clinical signs these include pyrexia, dullness, emaciation and death. Pigs showing marked clinical signs are unlikely to be presented for slaughter. In experimental infections in pigs there were no remarkable pathological changes that would be identified at post-mortem.

Considering these factors, the sensitivity of ante-mortem, slaughter and processing requirements in detecting and removing *T. evansi*-infected pigs was estimated to be 'negligible'.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with *T. evansi* and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a 'one minus the background rejection rate'. Because it is costly for pigs or carcasses to be rejected, the background rejection rate is considered 'extremely low'. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

Experimentally *T. evansi* has been shown to survive in tissues from a dead host (blood, liver, spleen) at room temperature for up to 12 hours. Trypanosomes may be present in extravascular sites including bone marrow, lymph vessels and nodes, cerebrospinal fluid and extravascular spaces in the brain. There is no evidence of trypanosomes being detected in skeletal muscle, so if present in muscle tissue this is likely to be due to infected blood or lymph perfusing the

muscle. In addition, *T. evansi* is highly dependent on glucose for survival and there would be insufficient glucose in meat to maintain the trypanosomes.

In view of this, it was considered that the likelihood that *T. evansi* would be present in meat harvested from an infected pig was 'low'.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

The optimum pH range for survival of *T. evansi in vitro* is 7.0 to 8.0. This has also shown to be the case with *T. brucei.* In addition, further research with *in vitro* cultures of the slender forms of *T. brucei* have shown they do not survive at pH less than 6.5.

Thus, it was considered that there was a 'negligible' likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

In vitro experiments have shown that *T. evansi* maintains infectivity at 5°C for 4 days and is readily maintained for many years by cryopreservation in suitable substrates. Given this, it was considered that there was a 'high' likelihood that meat infected with *T. evansi* at the completion of carcass maturation would remain infected during cold storage and transport.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a 'negligible' likelihood that imported pig meat derived from an individual carcass would be infected.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'negligible' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

Although the oral transmission of *T. evansi* has been demonstrated in dogs, evidence for the transmission of trypanosomes in pork or pork products has not been documented nor has oral transmission to pigs.

Trypanosoma evansi has been experimentally transmitted orally to dogs using 200 ml of blood and 400 grams of meat from goats with high parasitaemias of 10^4 trypanosomes per ml of blood. There may be more than 400 trypanosomes per ml of blood present in carcass tissues from acutely infected pigs, when the parasitaemia is likely to be at its highest (i.e. less than 4 weeks post-infection), but this is considerably less than that used to induce infection in dogs.

It is possible that even with a low level parasitaemia, trypanosomes may be present in higher numbers in other tissues including lymph nodes, bone marrow, synovial and cerebrospinal fluid.

In the light of this information, the likelihood that a waste unit from an infected pig would contain a sufficient dose of *T. evansi* trypanosomes to initiate infection was considered to be 'very low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

Trypanosomes have been shown experimentally to survive and remain infective at room temperature in rat carcases up to 10 hours after death. By 12 hours after death all trypanosomes were showing degenerative changes and infection could not be transmitted.

The effect of ultraviolet radiation on the survival of *T. evansi* trypanosomes is related to the length of exposure. Reduced infectivity for mice was demonstrated after ultraviolet irradiation for 3 hours and in blood samples kept in direct sunlight parasites were undetectable after 30 minutes.

In vitro studies on the survivability of *T. evansi* in blood samples showed greater survival times at 4°C than at 20°C to 27°C or 37°C. In addition, during putrefaction of meat trypanosomes will be deprived of a source of glucose which is essential for survival.

Given this, it was considered that the likelihood that *T. evansi* trypanosomes would survive within meat scraps discarded in refuse for the period of time required for feral pigs to locate and subsequently scavenge the material was 'extremely low'.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population

in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

- Remote regions = Negligible
- Rural regions = Negligible
- Large towns = Negligible

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs was found to be 'negligible'.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'negligible' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was 'very low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was an 'extremely low' likelihood that *T. evansi* trypanosomes would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs was found to be 'negligible'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that there was a 'negligible' likelihood that a waste unit would be infected.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected would contain a sufficient dose of the pathogenic agent to initiate infection was 'very low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was an 'extremely low' likelihood that *T. evansi* trypanosomes would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries was found to be 'negligible'.

Exposure assessment for other susceptible species

Surra has produced clinical disease in a wide variety of animals, and conceivably has the potential to infect all mammals. The major route of transmission is via biting flies. Other susceptible species that are carnivorous or omnivorous that may be potentially infected following the ingestion of contaminated meat waste could include dogs, dingoes, and rodents and cats. One study demonstrated that dogs could be infected after ingesting either blood or

goat meat containing over 10^6 *T. evansi* trypanosomes. As discussed above, the amount of trypanosomes present in carcass tissues from acutely infected pigs, when the parasitaemia is likely to be at its highest (i.e. less than 4 weeks post-infection) may exceed 400 trypanosomes per ml of blood but this is considerably less than that used to induce infection in dogs. Moreover, it is known that trypanosomes do not survive for very long outside a living host.

Given this, it was considered that the annual likelihood of entry and exposure for other susceptible species was 'extremely low'.

Consequence assessment

Consequence assessment was based on the identification and characterisation of discrete 'outbreak scenarios' for each of the four exposure groups - that is, feral pigs, pigs in backyard enterprises pigs in small commercial piggeries and other susceptible species. Outbreak scenarios for each of these groups were described in the Method for Import Risk Analysis but, for ease of reference, are reiterated in the discussion of each exposure group.

Having identified these scenarios, the assessment of consequences associated with each of the exposure groups was carried out according to the following steps as detailed in the Method for Import Risk Analysis:

- Estimation of the likelihood that each outbreak scenario would occur (the likelihood of establishment and spread);
- For each outbreak scenario, estimation of the consequences of a pathogenic agent according to the direct and indirect criteria;
- For each outbreak scenario, combination of consequences for individual criteria, to give an overall measure of 'consequences' for that scenario;
- Combination of the likelihood and consequences of each outbreak scenario, to give an estimate of likely consequences; and
- Combination of the likely consequences for each outbreak scenario to give an overall measure of the likely consequences associated with an exposure group.

Feral pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of feral pigs:

1. Containment of the disease within a directly exposed herd of feral pigs - no secondary spread - this option represents the 'no outbreak' scenario;
2. Secondary spread within a more general population of feral pigs - no spread to domestic pigs or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

Surra is usually spread to susceptible pigs by mechanical transmission by blood sucking flies. Tabanidae genera reported to transmit surra overseas are present in Australia including

Chrysops spp. and *Tabanus* spp. The stable fly (*Stomoxys calcitrans*) is widely distributed throughout Australia (Roberts, 1952).

Australian March flies (*Tabanus* spp.) occur in northern coastal areas. They require water or damp areas for laying eggs but may range several kilometres from their breeding habitat (Roberts, 1952). They are most active on warm sunny days and are especially abundant in swampy tropical and subtropical areas. Other species of Tabanidae in Australia such as *Scaptia* spp. are more common in southern Australia (Colless & McAlpine, 1970) but their potential for transmitting surra is unknown. *Stomoxys calcitrans* are most abundant in summer and autumn preferring strong light and therefore are rarely seen inside dark stables or buildings (Soulsby, 1982). They are swift fliers (but do not travel long distances) and will feed from all classes of mammals, particularly horses (Colless & McAlpine, 1970). Stable flies are most prevalent in coastal areas but may occur inland and larvae develop in manure or decaying vegetation (Colless & McAlpine, 1970).

Feral pigs tend to maintain small discrete groups, although some mixing occurs in times of low feed or water availability and some populations are contiguous. While feral pigs are widespread in Australia, there is very limited close contact with domestic pigs. However, there have been several reported cases of feral pigs gaining access to piggeries, in particular outdoor piggeries. Access may also occur where a pig producing enterprise is situated close to forests or reserves.

In order to become infected, pigs would need to come into proximity with other infected animals so that they were bitten by a vector after it had recently fed on an infected host. Native and wild animals in close proximity to feral pigs may become infected.

It was considered unlikely that the disease would be detected initially in feral pigs as infection is often subclinical. The disease would likely be detected after secondary spread to other susceptible species including wallabies, wild and domestic dogs, cats and horses where clinical signs of infection can be marked.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: low

Scenario 3: moderate

Scenario 4: very low

Backyard pigs

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of backyard pigs:

1. Containment of the disease within a directly exposed backyard herd - no secondary spread - this option represents the ‘no outbreak’ scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and

4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The likelihood assigned to each of these outbreak scenarios will be determined by the potential for; (a) close contact between backyard pigs and either feral pigs or other domestic pigs, and, (b) indirect spread to either of these groups by fomites or mechanical vectors. In the case of surra, spread is by biting flies to other animals within a short flying distance. Oral transmission to carnivores (and omnivores) feeding on infected carcases is theoretically possible. Direct transmission and spread by fomites or mechanical vectors other than biting flies does not occur.

If surra were introduced into backyard pigs it would likely go unnoticed due to the subclinical nature of the disease in pigs. As backyard pigs are unlikely to be kept in enclosed pens, the presence of blood sucking flies may result in transmission to other susceptible species in close proximity. This would probably include dogs, horses and other livestock species. Spread from backyard pigs to other groups of pigs is unlikely to occur via blood sucking flies as the distance would be too great. Transmission could occur if a live pig was transferred to another backyard herd. Transmission is less likely to occur to a commercial herd as vectors are rarely inside buildings. Feral pigs in close proximity to backyard pigs could become infected via a vector, but as feral pigs forage nocturnally they are unlikely to be bitten, as flies are active during the day.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: low

Scenario 2: very low

Scenario 3: moderate

Scenario 4: low

Small commercial piggeries

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of pigs raised in small commercial piggeries:

1. Containment of the disease within a directly exposed small commercial piggery - no secondary spread - this option represents the ‘no outbreak’ scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries - containment within this population - spread to other susceptible species - spread to humans if the agent is zoonotic; and
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries) - exposure of and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

The distinction between ‘small commercial piggeries’ and ‘backyard piggeries’ is to some extent arbitrary - indeed, the four outbreak scenarios described above are very similar to those outlined for the exposure of backyard pigs. However, managerial differences between the two classifications of pig-producing enterprise were considered important determinants of the likelihoods derived for the scenarios.

Surra has been found to be less prevalent in intensive pig herds, as opposed to free ranging unhoused pigs. There is likely to be less opportunity for transmission from a small commercial piggery to other susceptible species if the pigs are housed. Although infection in pigs is often asymptomatic, if clinical signs were present these are likely to be investigated. There may be greater likelihood of infected pigs from a small piggery being transferred to other small piggeries. Although spread is unlikely without vectors, iatrogenic spread may be possible when hypodermic needles are used for vaccination or treatment between numerous pigs without changing the needle.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: moderate

Scenario 2: very low

Scenario 3: low

Scenario 4: low

Other susceptible species

Estimating the likelihood of each outbreak scenario

Four outbreak scenarios were considered relevant to the exposure of other susceptible species:

1. Containment of the disease within the sub-population of directly exposed animals - no secondary spread - this option represents the ‘no outbreak’ scenario;
2. Secondary spread to feral pigs, but no subsequent spread to other domestic pig herds or other susceptible species - spread to humans if the agent is zoonotic;
3. Secondary spread (via feral pigs or other means) to a local population of domestic pigs - containment within a local population of backyard enterprises or small commercial piggeries - spread to other susceptible species - spread to humans if the agent is zoonotic;
4. Secondary spread (via feral pigs or other means) to a more general population of domestic pigs - exposure of medium-large commercial piggeries and/or spread within a pig-producing region - spread to other susceptible species - spread to humans if the agent is zoonotic.

If surra was introduced to wild or domestic carnivores there would be a greater likelihood of transmission because these species tend to develop more persistent and higher parasitaemias than pigs.

Spread may occur to other susceptible animals including horses and livestock that are in close proximity with domestic dogs. However, the disease may also be detected earlier, especially if in domestic animals, because of marked clinical signs and often death, resulting in veterinary attention and diagnosis. Wild carnivores may introduce the disease to other susceptible species such as wallabies, feral pigs and possibly horses and livestock via blood sucking flies.

Asymptomatic or chronically infected animals may act as a reservoir of infection for susceptible species.

On balance, the following likelihoods were assigned to the four scenarios.

Scenario 1: very low

Scenario 2: low

Scenario 3: moderate

Scenario 4: low

Estimating the consequences associated with each outbreak scenario

For each of the exposure groups, the first outbreak scenario denotes 'no further establishment or spread'. The remaining three outbreak scenarios describe 'secondary spread to other pigs, humans or to other susceptible species'. It can be seen that the scenarios described for each exposure group follows a similar pattern. Thus while the likelihoods associated with each will be different, their consequences will generally be the same. Any differences were noted in the assessment.

Outbreak scenario 1 — containment of the disease within a directly exposed group - no secondary spread

Under this scenario, surra would have established in the directly exposed animal, or group of animals, but would not have spread to other animals. This 'no outbreak' scenario would have resulted from low probability of spread between infected and susceptible animals due to distance or absence of vectors, rather than from human intervention. Indeed, because the disease may be of low pathogenicity for pigs, it was assumed that it would not have been identified and, it would not, under this scenario, have any discernible direct or indirect impacts.

On this basis, a rating of 'A' was assigned to each of the direct and indirect criteria.

Outbreak scenario 2 — secondary spread to feral pigs

Under this scenario, the disease is most likely to have established within a population of feral pigs, and to have run its course without identification. As explained above, this is because disease in pigs is often asymptomatic. Under this scenario, where the disease has not spread to other susceptible animals, it would not have any discernible direct or indirect impacts.

On this basis, a rating of 'A' was assigned to each of the direct and indirect criteria.

Outbreak scenario 3 — secondary spread (via feral pigs or other means) to a local population of backyard enterprises or small commercial piggeries

The third scenario is characterised by spread of surra to a local population of domestic pigs, but not to other pig populations, and spread to other susceptible species including cattle, horses and wildlife such as wallabies and dingoes. The disease would be diagnosed in other susceptible species such as horses that show marked clinical signs of infection and contained and eradicated using modified stamping out, quarantine and movement controls.

The direct impact of surra

Animal life or health

Although surra may be of low pathogenicity in pigs, an outbreak in a naïve population may result in clinical illness in breeding pigs such as anorexia, abortions, weight loss, possibly nervous signs and a general decrease in the efficiency of affected piggeries. Morbidity may be higher if there is iatrogenic spread through multiple use of hypodermic needles between pigs. Other susceptible animals such as dogs, cats and horses in the local area may also be clinically affected and there may be mortalities. The disease may also infect cattle, sheep and goats and, although clinical signs may not be evident, there may be losses in productivity.

Given this, it was considered that the direct effect on animal health would be unlikely to be discernible at the national level, but was of minor significance at the State level and hence this criterion was rated 'D'.

Environment

Wallabies have been shown to be highly susceptible to surra resulting in serious clinical signs and mortalities. Mortalities are also likely in dingoes and it is unknown if other native mammals may be susceptible and clinically affected. On this basis, the direct impact on the environment was considered unlikely to be discernible at the national level and of minor significance at the State level. Hence the rating assigned to this criterion was 'D'.

The indirect impact of surra

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

If identified in Australia, surra would require a control, eradication and compensation program. Although there is no specific AUSVETPLAN in place for surra, a draft is extant. The proposed policy is to eradicate surra where practicable using modified stamping out, quarantine and movement controls and associated activities.

The disease is listed as Category 4 under the Emergency Animal Disease Response Agreement. In this agreement the government and livestock industries have agreed to share costs based on the conduct of an agreed response plan for an outbreak of a disease that falls within one of four categories. Category 4 diseases are funded 20% by governments and the remaining 80% by the relevant industry. In this scenario, where surra has only limited spread within the domestic animal population, feral pig and wildlife population eradication would likely be attempted.

Under this outbreak scenario infected or suspect animals could be destroyed. However, this may not be appropriate due to the high monetary and genetic value of many horses and zoo animals and emotional distress arising from the companion animal bond between owners and their horses, dogs and cats. Chemotherapy may be an alternative option. It is likely that strict movement restrictions would be imposed to prevent spread of the disease by movement of infected animals. The draft AUSVETPLAN recommends movement restrictions from infected properties, movement control of susceptible species within and out of restricted and control areas, possible animal free buffer zones, vector control and surveillance of the wildlife, feral pig and domestic animal populations.

Overall, the indirect impact of new or modified control programs under this scenario was considered unlikely to be discernible at the national level, and of minor importance at the State level. This gave the disease a rating of 'D' for this criterion.

Domestic trade or industry effects

In this scenario, where surra spreads to a local population of other susceptible species including cattle and horses there may be disruption to livestock sales and events, horse racing and equestrian events and dog shows in that and the surrounding area.

When these issues were taken into account, the indirect impact of surra on domestic trade and industry was considered unlikely to be discernible at the national level, but of minor importance at the State level. This resulted in a rating of 'D' for this criterion.

International trade effects

With the involvement of horses under this scenario, export markets for these animals may be disrupted. Surra is listed as an OIE List B disease of horses, however, there is no Code Chapter for this disease, nor recommendations for regulating trade in animals or products. Some export markets for dogs, cats, breeding pigs and domestic livestock such as cattle, sheep, goats and camels may be disrupted although this is less likely. Export of beef, lamb and pork is unlikely to be affected. In light of this information, it was considered that the indirect effect of surra on international trade would be unlikely to be discernible at the national level, and of minor significance at the State level. This resulted in a rating of 'D' for this criterion.

Indirect effects on the environment

In this outbreak scenario surra spreads to local populations of native wildlife resulting in clinical disease and mortalities. Depending on the susceptibility of certain marsupial species to surra in the local area, and the population, some species may be threatened with extinction. Given this, the indirect effects of surra on the environment such as affecting biodiversity was considered unlikely to be discernable at a national level, and of minor significance at the State level and a rating of 'D' was thus assigned to this criterion.

Indirect impact on communities

One of the considerations within this criterion was the indirect impact of a disease on rural and regional economic viability. In this scenario, where a local population of domestic pigs and other susceptible species such as cattle and horses are infected with surra, it was considered that where these industries were important to the local economy or to individuals within a community, aspects of the rural community may be threatened. Given this, the indirect impact of surra on communities was considered unlikely to be discernible at the national or State level, and of minor significance at the district or regional level. This resulted in a rating of 'C' for this criterion.

Outbreak scenario 4 — secondary spread (via feral pigs or other means) to a more general population of domestic pigs (including medium-large commercial piggeries)

Under this scenario, surra would have spread to a broader population of commercial piggeries (including medium-large piggeries), and to other susceptible species such as cattle, horses, camels, goat and native animals including marsupials and dingoes. Spread to dogs and cats may

also have occurred. A control program would have been mounted in response to diagnosis of the disease in animals.

The direct impact of surra

Animal life or health

Under this scenario, where the disease has become widespread, the clinical signs would be as described for scenario 3 but with a greater number of animals affected and involvement of more species. Taking this into account the direct effects on animal health were considered to be of minor importance at the national level. This resulted in a rating of 'E' for this criterion.

Environment

Widespread disease in wildlife species and populations are likely. Clinical signs of disease may be severe in several marsupial species and dingoes and mortalities may be high. Hence the direct impact on the environment was considered likely to be of minor significance at the national level, and a rating of 'E' was assigned to this criterion.

The indirect impact of surra

New or modified eradication, control, surveillance/monitoring and compensation strategies/program

In this scenario, the disease is widespread, and as insect vectors are involved in transmission and the disease is present in the wildlife population, an eradication program is less likely to be effective. In these circumstances, the draft AUSVETPLAN recommends a control program to slow the spread of disease and reduce impacts on trade. Quarantine and movement controls would be maintained in the control and restricted areas. The movement of susceptible animals out of the infected area would only occur under conditions that require treatment before departure. Extensive tracing and surveillance would be required, together with ongoing surveillance to assist with zoning, particularly if the disease cannot be eradicated. If animal-free buffer zones are created then ongoing depopulation of wild animals will be necessary across a much broader area to ensure that normal home-range movements of wild animals does not include the buffer zone. There would be ongoing surveillance of susceptible species and surveys of feral and wild species. Surveillance of vectors for the detection of the parasite is appropriate using PCR tests to determine the extent of spread of the disease and identification suitable vectors. Isolation of stock from swampy areas may reduce the likelihood of vector transmission. Control of vectors by using insecticides on fly-breeding sites and the surrounding areas may also be warranted. Another option for the control of biting by tabanids is the daily application of a residual synthetic pyrethroid which acts as a repellent.

Given this, it was considered that the indirect impact of new or modified control programs would be of minor significance at the national level. This resulted in a rating of 'E' for this criterion.

Domestic trade or industry effects

As with outbreak scenario 3, there would be restrictions on movements of animals, livestock sales, holding of horse races, other equestrian events, greyhound racing and dog shows and events. Supporting industries such as stockfeed manufacturers, veterinarians and farriers could also be affected. Livestock would still be able to move for slaughter and trade in meat would

not be restricted in Australia. Establishment of the disease in areas of extensive livestock production would impact on the use of stockhorses to muster cattle, reduce productivity and increase property management costs.

Taking these issues into account, it was considered that the indirect impact of surra on domestic trade and industry would be of minor significance at the national level. This gave the disease a rating of 'E' for this criterion.

International trade effects

Major Australian markets for live horses such as New Zealand, the European Union, Japan, Singapore, Macau and Hong Kong are free of surra. Export trade and the conduct of, and participation in, international horse competitions would be disrupted until new conditions for trade were negotiated with trading partners. There may be restrictions on the importation of live cattle from Australia by current and potential importing countries although surra is endemic in most major market countries. The export of live dogs, cats, buffalo and camels may also be affected. Australia would possibly need to adopt zoning to assist in the international marketing of these animals.

Given this, it was considered that the indirect impact of surra on international trade would be significant nationally. This gave the disease a rating of 'F' for this criterion.

Indirect effects on the environment

In this outbreak scenario surra spreads to more general populations of native wildlife resulting in widespread clinical disease and mortalities. The impact is likely to be similar to that of the third scenario. The indirect effects of surra on the environment such as affecting biodiversity was considered likely to be of minor significance at the national level and a rating of 'E' was thus assigned to this criterion.

Indirect impact on communities

A widespread outbreak of surra involving horses with disruption of horse events would have social and economic consequences for the many thousands of people involved in horse riding and racing. Moreover horse racing contributes significantly to government revenue.

Where domestic pigs, cattle or other livestock are important to the local economy, these rural communities may suffer. Given this, the indirect impact of surra on rural communities was considered unlikely to be discernible nationally and of minor significance at the State level. This resulted in a rating of 'D' for this criterion.

The overall impact of surra

When the direct and indirect impacts of surra were combined using the decision rules described in the Method for Import Risk Analysis, the following results were obtained

Scenario 1: Consequences negligible

Scenario 2: Consequences negligible

Scenario 3: Consequences low

Scenario 4: Consequences high

Having obtained estimates of the likelihood of each scenario and its consequences for each exposure group, these were combined using the matrix in Table 9. The results of this procedure are summarised in Table 110, Table 111, Table 112, and Table 113. It can be seen that the overall likely consequences associated with the exposure of feral pigs, backyard pigs or pigs in small commercial piggeries to infected pig meat scraps were considered 'low', 'moderate' and 'moderate' respectively. The likely consequences associated with the exposure of other susceptible species to infected pig meat scraps were considered 'moderate'.

Table 110 Surra: summary of the consequences of exposure of feral pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Moderate	Low	Low
Scenario 4	Very Low	High	Low
		Overall likely consequences	**Low**

Table 111 Surra: summary of the consequences of exposure of backyard pigs

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Low	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Moderate	Low	Low
Scenario 4	Low	High	Moderate
		Overall likely consequences	**Moderate**

Table 112 Surra: summary of the consequences of exposure of small commercial piggeries

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Moderate	Negligible	Negligible
Scenario 2	Very low	Negligible	Negligible
Scenario 3	Low	Low	Very low
Scenario 4	Low	High	Moderate
		Overall likely consequences	**Moderate**

Table 113 Surra: summary of the consequences of exposure of other susceptible species

Scenario	Likelihood	Consequences	Likely consequences
Scenario 1	Very Low	Negligible	Negligible
Scenario 2	Low	Negligible	Negligible
Scenario 3	Moderate	Low	Low
Scenario 4	Low	High	Moderate
		Overall likely consequences	**Moderate**

Risk estimation

The (unrestricted) annual risk estimate was obtained in two stages:

- Estimation of the 'partial annual risk' associated with each of the exposure groups;
- Combination of partial annual risks to give an estimate of 'overall annual risk'.

Partial annual risks were obtained by combining the annual likelihood of entry and exposure with the corresponding estimate of 'likely consequences' for that exposure group using the rules shown in the risk estimation matrix (Table 10).

The decision rules described in the Method for Import Risk Analysis were then used to give an overall estimate of the unrestricted annual risk associated with surra.

Table 114 shows the results obtained for each of the exposure groups and for the overall unrestricted annual risk. Since the overall annual risk is clearly less than Australia's ALOP (very low), risk management would not be required for surra.

Table 114 Surra: components of the unrestricted risk estimate

Exposure group	Likelihood of entry	Annual likelihood of entry and exposure	Likely consequences	Annual risk
Feral pigs	Negligible	Negligible	Low	Negligible
Backyard pigs	Negligible	Negligible	Moderate	Negligible
Small commercial piggeries	Negligible	Negligible	Moderate	Negligible
Other susceptible species	Negligible	Extremely low	Moderate	Negligible
			Overall annual risk	**Negligible**

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Equine encephalomyelitis viruses

Technical information

Background

The equine encephalomyelitis viruses consist of three distinct but antigenically related viruses, Eastern equine encephalomyelitis (EEE) virus, Western equine encephalomyelitis (WEE) virus and Venezuelan equine encephalomyelitis (VEE) virus. These viruses are transmitted in nature by mosquitoes and are maintained in cycles with various vertebrate hosts, usually birds or forest dwelling rodents. They can cause severe, frequently fatal encephalitis in horses and humans, and occasionally pigs and some birds. The viruses are found only in the Americas with each virus infecting a different region. These viruses cause similar clinical syndromes in equines, but differ in the consequences of infections in humans.

As there are some differences between the viruses, each virus is discussed separately.

EEE virus

EEE occurs in eastern and north central United States of America, the bordering areas of Canada, the Caribbean and in parts of Central and South America. In southeastern United States of America, especially in Florida, disease activity in equines occurs annually (Walton, 1992). Neutralising antibodies to EEE virus have been detected in birds and mammals as far south as Argentina (Monath, et al., 1985).

WEE virus

WEE occurs in mainly in western United States of America. Outbreaks have occurred sporadically in central United States of America, western Canada and the northern half of South America. The virus has also been detected in mosquitoes and birds inhabiting fresh water swamps and waterways in central, north, and northwest Florida (Hoff, et al., 1978). Neutralising antibodies to WEE virus have also been detected in rodents and horses in Argentina (Monath, et al., 1985).

VEE virus

VEE is endemic in northern South America, Trinidad and Central America and epizootics occur in northern and western South America. Although VEE outbreaks occurred in the United States of America over 20 years ago and low virulence strains of VEE are endemic in southern Florida, the disease is considered to be exotic in the United States of America and Canada (Walton, 1992). Neutralising antibodies to VEE virus have been detected in horses, birds and wild mammals in Argentina (Monath, et al., 1985).

Agent taxonomy

All three viruses are enveloped, positive single-stranded RNA viruses, members of the genus Alphavirus of the family Togaviridae and are further subdivided by their serologic relationships.

EEE virus

The EEE virus complex consists of one virus having two antigenic variants, the North American variant, found in eastern and north central United States of America, the bordering areas of Canada and the Caribbean, and the South American variant, found in parts of Central

and South America. The former variant shows a high degree of genetic and antigenic homogeneity while the latter tend to be more heterogeneous (Smith, et al., 1997).

WEE virus

The WEE virus is one of six viruses within the WEE virus complex and several antigenic subtypes of the WEE virus itself have been identified. While the geographical distribution of different strains of WEE virus may overlap, it appears that the strains are maintained in specific geographic areas through local persistence in enzootic foci (Kramer & Fallah, 1999).

VEE virus

The VEE virus complex consists of one virus of six subtypes, each having its own ecological and epidemiological characteristics. Within subtype I, five variants have been identified of which two are epizootic strains, being highly pathogenic for humans and equines. The remaining variants and subtypes are the enzootic strains, capable of causing disease in humans but generally lacking virulence in equines. However, in Mexico, one strain, the I-E variant, can become pathogenic to horses under certain but undefined conditions (Walton, 1998).

Agent properties

EEE virus, WEE virus and VEE virus

The viruses are susceptible to organic solvents and detergents, sensitive to moist and dry heat and do not survive for long away from the host. They are stable if kept under *in vitro* conditions. Exposure to irradiation reduces viral infectivity (Office of Laboratory Security - Population and Public Health Branch, Health Canada, 2001).

EEE virus can survive freezing in brain tissue suspensions at -70°C without loss of infectivity for up to 20 months of storage. Infectivity for guinea pigs fell after storage in the freeze-dried state for 10 years (Polanco & Redondo, 1982).

There is no published information on pH stability. However, another alphavirus, Getah virus, is reported to be stable at pH 6 to pH 9 but unstable below pH 5 and above pH 10. It is inactivated by heating at 50°C and above, and by storage at 37°C for 4 days. It remains active at 10°C for 3 months, and longer at lower temperatures (Kamada, et al., 1982).

Host range

EEE virus

EEE virus normally cycles among passerine birds (that is, the perching songbirds) and the bird-feeding mosquito, *Culiseta melanura*. The virus replicates rapidly in nestlings which act as reservoirs for bridging vectors, such as *Coquillettidia perturbans* and some species of the genus *Aedes*, which are capable of feeding on both passerines and mammals. Horses, humans and wildlife not native to the geographical area can be infected and may suffer severe and sometimes fatal disease. Horses are usually dead-end hosts. Affected wildlife includes emus and captive-raised game birds, such as pheasants, chukars and quail (Wiersma, et al., 1998). Domestic poultry are susceptible to EEE virus, but clinical signs are seen only in chickens under 3 weeks old (Tully, et al., 1992). Clinical infection has been reported in pigs in Georgia, Wisconsin, New Jersey and Florida (Elvinger, et al., 1996).

WEE virus

WEE virus normally cycles among wild birds and mosquitoes. *Culex tarsalis* is the mosquito host of importance. Other mosquitoes, such as those of the *Aedes*, *Anopheles*, *Coquillettidia*, *Culex* and *Culiseta* species, also act as vectors, spreading the virus to mammals. Of the wild birds hosts, some passerines, particularly the house sparrow and the house finch, act as amplifying hosts. Some mammals, particularly the blacktail jackrabbits, are suspected of serving as hosts for infection of *Aedes melanimon* with WEE virus (Hardy, 1987). A number of wildlife species such as Richardson's ground squirrel, snowshoe hares, gartner snakes (*Thamnophis* sp) and leopard frogs (*Rana pipiens*), domestic poultry and livestock are incidental hosts and show inapparent infection. Horses, humans, emus, and captive-raised game birds are also incidental hosts, but WEE virus can cause severe and sometimes fatal disease in these species (Hardy, 1987). There is no report of natural disease in pigs although experimental disease can be produced (Liggett, 2002). WEE virus has been isolated from a spleen and liver suspension of a pig with classical swine fever and attempts to induce clinical disease experimentally were successful only in 3-day old piglets. Older pigs developed antibodies and the virus can be recovered from the brain of a clinically ill pig (Pursell, et al., 1972).

VEE virus

The enzootic or sylvatic strains of VEE virus normally cycle among forest dwelling rodents and mosquitoes. Horses and humans are only incidentally involved in this cycle and clinical disease can occur in humans with human deaths mostly in the young or aged. VEE epizootics occur sporadically and often several species of mosquitoes become involved in viral transmission to other mammals, particularly equines. During epizootic outbreaks, equines develop very high viraemia levels and are the only known animal species to act as efficient amplifiers of the epizootic VEE virus. Several laboratory animal species are susceptible to both enzootic and epizootic strains while several domestic animal species, including cattle, pigs, and dogs, can show serologic and virologic, but not clinical, evidence of VEE epizootics (Walton, 1998). Chickens are rarely infected by VEE virus (Scherer, et al., 1971).

Epidemiology

EEE virus

As stated earlier, EEE virus normally cycles among passerine birds and the bird-feeding mosquito, *Culiseta melanura*. It is not known how the virus is maintained in the mosquito-passerine bird cycle, especially in areas where winters are too cold for mosquito activity and result in migration of passerine birds. Although mosquitoes are known to remain infective for life, it is not certain if transovarial transmission occurs within mosquitoes (Morris & Srihongse, 1978).

In epizootic outbreaks, EEE virus is transmitted via the saliva of mosquitoes that had previously fed on the viraemic passerine birds. Viral transmission is dependent on warmer temperatures and on standing water being available for mosquito breeding. Thus in the warmer areas, cases may occur all year round while in the more temperate areas cases usually occur from mid-summer to late autumn (Elvinger, et al., 1996).

There is a close association between EEE in equines and man and the swamp breeding grounds of *Culiseta melanura*. Clinical infection in either vertebrate host seldom occurs beyond 8 km from these foci and prevalence of antibodies in birds decreases with distance from the swamp

(Emord & Morris, 1984; McLean, et al., 1985). Human disease generally follows equine infections by about 2 weeks (Walton, 1992).

Incubation period in humans and horses is 5 to 15 days and viraemia lasts from 2 to 4 days. Viraemia is low in equines, around 10^5 SMICLD[66]$_{50}$/ml of blood, and does not persist for more than 5 days. Latent infection has not been reported. By the time clinical encephalomyelitis is recognised, viraemia has ended (Walton, 1992).

Pigs are susceptible to oral inoculation of the EEE virus and can develop viraemia, antibodies and clinical disease. The virus can be shed in the faeces, be isolated from oropharyngeal swab specimens up to 4 days after infection, and be isolated from the tonsils of pigs up to 20 days after experimental infection (Elvinger & Baldwin, 1999). Thus contact transmission is possible and may enhance dissemination of the virus within a piggery. But pigs kept in direct contact with pigs experimentally inoculated with the virus showed no signs of infection and did not develop neutralising antibodies (Karstad & Hanson, 1959). Pigs experimentally infected with EEE virus can develop high titre viraemia, and consequently mechanic and biologic vector transmission may be possible (Elvinger, et al., 1994).

EEE causes an often fatal encephalitis in nursing piglets and subclinical infection in older pigs, especially where pigs have no shelter from above-average rainfall during the time of the outbreaks. Experimental inoculation of 10-day old piglets with 12.5 x 10^5 MCCID[67]$_{50}$/ml via the intracerebral and intraperitoneal routes resulted in severe central nervous system (CNS) disturbance while inoculation with 15 x 10^7 $MCCID_{50}$/ml via the intramuscular and intraperitoneal route did not result in CNS signs but resulted in antibody development (Pursell, et al., 1972).

Reports of clinical disease in pigs due to EEE virus infection are sporadic. Over 160 (80%) of 200 nursing pigs died in a piggery in Georgia during an outbreak in 1971 (Pursell, et al., 1972). Similarly, 280 (80%) of 350 piglets from 38 litters at a piggery in Georgia died during another EEE outbreak in 1991. Afterwards, 19 of 31 pigs (10/10 sows, 1/2 boars, 8/19 surviving pigs) were seropositive for EEE virus (Elvinger, et al., 1994). In Florida in 1994, 50 (56.6%) of 90 piglets from 10 litters developed CNS signs and 41 (45.6%) piglets died. All 10 sows and 9 of 10 surviving pigs had neutralising antibodies to EEE virus (Elvinger, et al., 1994; Jacoby, et al., 1995).

The number of pigs seropositive for EEE virus is far greater than that reported for clinical disease and EEE is probably under-diagnosed in pigs even though it can cause financial losses in some pig herds (Elvinger, et al., 1996). In Georgia, 11 (7.3%) of 151 samples collected in stockyards and 21 (2%) of 1064 samples collected from 45 piggeries were seropositive for EEE virus. Of the 45 piggeries, nine (20%) had one or more seropositive pigs. The higher prevalence found in pigs in stockyards as compared with the 45 piggeries probably reflects the diverse origins of the pigs in stockyards as the 45 piggeries were considered to have above average quality of management, having herds either certified brucellosis and pseudorabies (Aujeszky's disease) free or enrolled in a pseudorabies eradication program (Elvinger, et al., 1996).

A serosurvey also revealed antibody titres in feral swine on Ossabaw Island of Georgia, with 62 (16.5%) of 376 samples testing positive for EEE virus (Elvinger, et al., 1996).

[66] SMICLD = Suckling mouse intracranial median lethal doses
[67] MCCID = Median cell culture infective doses

WEE virus

WEE virus primarily cycles between passerine birds and mosquitoes, particularly *Culex tarsilis*. As with EEE virus, weather is an important influence of WEE virus transmission and vector abundance. It is not known how the virus is maintained in the mosquito-passerine bird cycle in areas where winters are too cold for mosquito activity and result in migration of passerine birds. Epizootics occur when swarms of mosquitoes that had opportunistically fed on the viraemic passerines infect horses and humans.

Incubation period in horses and humans is generally 5 to 15 days and viraemia lasts from 2 to 4 days. In clinical cases, fever starts during viraemia and encephalitis follows 4.5 to 5 days after infection when viraemia is ending, neutralising antibodies become detectable and temperature returns to normal (Walton, 1998).

Although WEE virus can be isolated from mosquitoes annually throughout the western United States of America, clinical disease in single animals and epizootics are much less frequent than those caused by EEE virus (Walton, 1992). In the United States of America, in 1996, 145 cases of EEE and 6 of WEE in horses were reported while in 1997, 114 of EEE and 9 of WEE in horses were reported. Similarly, in 1996 to 1997, 19 cases of human EEE were reported but no human cases for WEE, although enzootic activity of WEE was reported in 6 of 7 western States (Wiersma, et al., 1998). However, major epidemics have occurred, the worst of which occurred in the western United States of America and Canadian plains in 1941, resulting in 300,000 cases of encephalitis in equines and 3336 cases in humans (Nandalur & Urban, 2002). Organised mosquito abatement programs have resulted in the reduction of human cases since and only 639 confirmed human cases have been reported since 1964, but there are concerns that new epidemics are likely as population expands into endemic areas.

There is no published report of natural disease in pigs although experimental disease can be produced (Liggett, 2002). Antibody response has been reported in pigs exposed to the virus by feeding on inoculated feed (McNutt and Packer as reported in (Karstad & Hanson, 1959). Neutralising antibody was found in 33% (19 of 57) of pigs in Colorado (Winn, et al., 1958).

VEE virus

Enzootic VEE strains are typically found in tropical wet forests with high water table or open swampy areas where rain falls throughout the year and the virus continuously cycles among rodents and sometimes birds by the feeding of mosquitoes. In enzootic infections, VEE virus is normally transmitted via the saliva of mosquitoes that had previously fed on viraemic vertebrate hosts but in epizootic outbreaks, spread can also be by secretions or aerosols from highly viraemic animals, such as horses and laboratory animals. In laboratories, accidental exposure to aerosols of VEE virus has caused fatal infection in unvaccinated humans. In Mexico, a VEE outbreak caused by VEE virus subtype I-E resulted in 157 equine cases with a case fatality rate of 47.8%. No human VEE cases were confirmed although human seroprevalence in the region was high (Gonzalez-Salazar, et al., 2003).

In many cases, VEE epizootics terminate when sufficient susceptible equids are no longer available to serve as definitive hosts.

Incubation period in horses and humans is 0.5 to 2 days and may be as long as 5 days. Viraemia lasts for 2 to 4 days and does not persist for more than 5 days. Latent infection has not been reported. By the time clinical encephalomyelitis is recognised, viraemia generally has ended (Walton, 1992)

There is no report of natural disease in pigs (Dickerman, et al., 1973) although experimental disease can be produced (Liggett, 2002). However, enzootic VEE virus antibodies have been detected in 60.8% (31 of 54) of feral pigs in a wildlife refuge in southern Texas where a VEE epizootic had occurred (Smart, et al., 1975), in 1 of 25 pigs in Guatemala during an epizootic outbreak in 1969 (Scherer, et al., 1972), frequently in pigs in enzootic areas of southeastern Mexico (Scherer, et al., 1971) and high antibody titres in pigs in a VEE epizootic in Colombia in 1967 (Sanmartin, et al., 1973). There was speculation that pigs may be amplifiers of VEE virus and contribute to the cycling of the virus in nature (Dickerman, et al., 1973) although generally they are not considered to be amplifiers.

An examination of the blood meals of mosquitoes collected during an epidemic in Costa Rica in 1970 showed that 34% were from equines and 14% were from pigs (Martin, et al., 1972).

Clinical signs

EEE virus, WEE virus and VEE virus

Clinical signs have not been reported in pigs naturally infected with WEE or VEE viruses, only with the EEE virus in nursing piglets less than 2 to 3 weeks old. In pigs 2 to 3 months old, EEE virus given intravenously, intradermally, intracerebrally or intranasally resulted in development of neutralising antibodies but not clinical signs and viraemia could not be detected when blood was sampled every 48 hours (Karstad & Hanson, 1959).

Many EEE virus infections in piglets are subclinical and inapparent. Others are mild or severe and frequently fatal. For the first 4 to 5 days, clinical signs are nonspecific. Mild infections are characterised primarily by anorexia, high fever and depression while severe infections are characterised by anorexia, high fever, stupor, weakness, staggering, and blindness. Encephalitic signs, including behavioural and neurologic signs, may follow, becoming evident 4.5 to 5 days after infection when viraemia is ending, neutralising antibodies become detectable and body temperature drops to normal. Death frequently follows in severely affected pigs. Surviving piglets usually have retarded growth (Elvinger & Baldwin, 1999).

In humans infected with EEE, WEE or VEE virus, the clinical syndrome varies from a mild flu-like illness accompanied by frontal headaches to a severe encephalitic disease. Deaths have been reported mainly in children and the elderly (Walton, 1992).

In horses, the clinical syndrome for EEE, WEE and VEE are similar though they differ in severity, depending on the virus species and strain. There may be early anorexia and depression followed by transitory fever and nervous signs including hypersensitivity to sound and touch progressing to apparent blindness, severe mental depression, head pressing and, in the terminal stages, paralysis. (Walton, 1998).

Pathogenesis

EEE virus, WEE virus and VEE virus

After the animal is infected, the virus multiplies in muscles, enters the lymphatic system and localises in the lymph nodes where it replicates in both macrophages and neutrophils. Subsequent to replication, the virus is shed in small numbers. If most viruses are successfully cleared, no further clinical signs develop and initial viraemia passes. Neutralising antibodies will still be produced. If viral elimination was not completed, the remaining viruses infect the endothelial cells, particularly in highly vascular tissues of the liver and spleen, where they again replicate. The second viraemic period is often associated with circulating virus and

development of clinical signs. The virus may then invade the CNS and cause acute inflammatory disease of short duration involving brain, spinal cord and meninges (Bertone, 1998).

Pathology

EEE virus, WEE virus and VEE virus

Gross pathologic appearances of the CNS vary from no visible lesion to extensive necrosis and haemorrhages. Lesions in other tissues are too variable to be of any diagnostic significance.

Immunology

EEE virus, WEE virus and VEE virus

Alphaviruses are highly immunogenic and both cellular and humoral immune mechanisms contribute to recovery following natural or experimental infection. Experimental infection of pigs with EEE virus resulted in serum neutralising antibodies after 7 days.

There is evidence of familial relationship between EEE and WEE viruses. In horses and English sparrows previously infected with WEE virus, experimental infection with EEE virus did not produce CNS symptoms or death while experimental infection with EEE virus in horses and English sparrows not previously infected with WEE virus resulted in CNS symptoms and deaths in at least 50% of animals. Similarly, infection with WEE virus in EEE immune English sparrows did not result in CNS symptoms and death while infection with WEE virus in two non-EEE immune English sparrows resulted in death in one bird (Stamm & Kissling, 1957).

Transmission via meat

EEE virus

There is no published report of transmission via pig meat. During viraemia, high titres of EEE virus can be found in the blood of pigs and the virus can be isolated from the tonsils of pigs for up to 20 days after experimental infection (Elvinger & Baldwin, 1999). Viral titres were not reported. In general, mammals have been considered dead-end hosts due to low virus titres that are insufficient to infect vectors (Elvinger & Baldwin, 1999).

Pigs have reportedly been infected with EEE virus orally, but the dose was not specified (Elvinger & Baldwin, 1999).

WEE virus and VEE virus

There is no published report of transmission via pig meat.

Release assessment

R1 — the likelihood that a source herd is infected

EEE virus

Data on prevalence of pig herds infected with EEE are limited. Nine (20%) of 45 well-managed piggeries in Georgia had one or more pigs seropositive for EEE virus. This appears to be a conservative estimate considering that only 2% of the tested pigs were seropositive while at stockyards 7.3% of tested pigs were seropositive. EEE has been reported in pigs only in

Georgia, Wisconsin, New Jersey and Florida, which accounts for only 1.33 million pigs out of a total of 50.3 million pigs in the eastern States[68]. However, the domestic pig population in the EEE endemic area has apparently quadrupled in the past ten years with most of the population located in coastal and near coastal areas prone to flooding. There is concern that the expansion of the pig industry into the EEE endemic areas will increase the potential for the re-emergence of EEE in pigs (Liggett, 2002).

Given this, the likelihood that slaughter-age pigs have been selected from an infected herd in a country where EEE is endemic was considered to be 'low'.

WEE virus

There is no published report of prevalence of WEE virus or antibodies in pigs. Horses and sentinel chickens serve as sentinels for human cases in a given area. Public health authorities use sentinel chickens to monitor arbovirus activities in their region. In 2002, 50 local agencies in California maintained 207 sentinel chicken flocks, each flock containing 10 chickens. They reported 52 (0.025%) seroconversions to WEE (Coachella Valley Mosquito and Vector Control District, 2003). Of the 2.4 million horses in the western States of the United States of America[69], only 6 and 9 cases were reported in 1996 and 1997 respectively. In addition, 52589 birds were tested for WEE antibodies over ten years from 1987 to 1996. Passerines comprised 83% of the birds and only a negligible number tested positive (Gruwell, et al., 2000).

Given the prevalence of WEE infection in other animal species, the likelihood that slaughter-age pigs have been selected from an infected herd in a country where WEE is endemic was considered to be 'extremely low'.

VEE virus

There is no published report of prevalence of VEE virus or antibodies in domestic pigs. In enzootic areas, antibodies to VEE virus have been reported, in one case, in over 60% of feral pigs in an area where a VEE epizootic had occurred in southern Texas, and, in another, in pigs in south eastern Mexico. Other enzootic foci are found in several smaller Central American countries, particularly along the tropical Atlantic lowland coast and on the Pacific coastal lowlands (Scherer, et al., 1972). Generally, the geographical distribution of infected animals conforms with the known ecological distribution of the VEE virus.

Given the likely prevalence of pigs with VEE antibodies in enzootic foci of some Central American countries, the likelihood that slaughter-age pigs have been selected from an infected herd in a country where VEE is endemic was considered to be 'low'.

R2 — the likelihood that a slaughter-age pig from an infected herd is infected

EEE virus

Evidence suggests whenever a herd becomes infected with EEE virus, seroprevalence can be very high. However, the viraemic period is very short, less than 5 days, and outbreaks generally occur only during warm season when arthropods are active. EEE virus infection can occur year round in parts of south eastern United States of America especially Florida. Only those pigs that have been exposed to infection just prior to slaughter are likely to be viraemic

[68] Hogs and Pigs: Final estimates 1993-97, National Agricultural Statistics Services, United States Department of Agriculture

[69] US Equine Inventory, 1999, National Agricultural Statistics Services, United States Department of Agriculture.

Given this, it was considered that the likelihood of selecting an infected pig in an infected herd was ‘very low’.

WEE virus

There is no report of the within-herd prevalence of WEE in infected pig herds. Infection in pigs would appear to be extremely rare. In a study of wetland sites in California, in areas considered to be enzootic foci for WEE virus, 133 seroconversions to WEE were detected in 28 sentinel chicken flocks (Reisen, et al., 2000). Outbreaks of WEE had occurred in several turkey flocks in California in 1993 and 1994 and in all cases 100% of samples were seropositive for WEE virus (Cooper & Medina, 1999). However, the viraemic period is very short and outbreaks generally occur only during warm season when arthropods are active. Thus pigs that had reached slaughter-age before arthropods became active are most likely not to have been exposed to infection. Only those pigs exposed to infection in the few days prior to being slaughtered are likely to be infected.

Given this, it was considered that the likelihood of selecting an infected pig in an infected herd was ‘extremely low’.

VEE virus

As VEE virus infection generally occurs in warmer tropical areas, susceptible pigs in enzootic foci are likely to be at risk of infection with VEE virus for most of the year, particularly during the wet season. Thus, pigs are more likely to be infected early in life and be immune to the disease on reaching slaughter weight. Given this, it was considered that the likelihood of selecting an infected pig in an infected herd was ‘very low’.

R3 — the likelihood that the pathogenic agent will not be detected as a result of controls and procedures carried out in accordance with requirements dictated in the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption (the Australian Standard)

R3.1 — the sensitivity of ante-mortem, slaughter and processing requirements described in the Australian Standard

None of the three viruses cause clinical signs or pathologic lesions in slaughter-age pigs. Consequently infection is not likely to be detected during ante-mortem, slaughter and processing inspections. Hence the sensitivity of ante-mortem, slaughter and processing requirements in detecting and removing infected pigs was considered ‘negligible’.

R3.2 — the specificity of ante-mortem, slaughter and processing requirements described in the Australian Standard

This step describes the proportion of pigs or carcasses that are *not* infected with EEE, WEE or VEE virus and are *not* removed from the export chain because of other illnesses or abnormalities. In some applications, this has been described as a ‘one minus the background rejection rate’. Because slaughter-age pigs do not show symptoms of any of the three viruses, the background rejection rate is considered ‘extremely low’. This means that the specificity of ante-mortem and post-mortem inspection is thought to lie between 99.9% and approximately 100%.

R4 — the likelihood that the pathogenic agent will be present in the meat harvested for export

There is no report on the occurrence of any of the three viruses in pig meat. EEE virus can be isolated from the tonsils of pigs for up to 20 days after experimental infection; however, most tonsillar tissues are removed at slaughter. High titres of EEE virus have been isolated from the blood of experimentally infected pigs. Nonetheless it is generally accepted that mammals are considered dead-end hosts due to low EEE virus titres that are insufficient to infect vectors. Moreover, although some blood will be retained in pig carcasses after exsanguination at slaughter, the viraemic period is short, lasting 2 to 4 days.

As there is no evidence of differences in the distribution of the three viruses in pigs, it was considered that the distribution of these viruses in a pig carcass would be similar. These viruses can cause infection in humans, yet there is no published report of human infection amongst abattoir workers arising from the slaughter and processing of not only pigs, but also equines, cattle and poultry. Thus the likelihood that any of the three viruses would be present in the meat harvested for export was considered to be ‘very low’.

R5 — the likelihood that the pathogenic agent will not be destroyed by the post-mortem decrease in muscle pH that accompanies carcass maturation

There is no published information on the effects of any of the three viruses on pH. Alphaviruses are considered to be stable between pH 6.0 and pH 9.0 when kept in a suitable environment. The viruses cannot survive for long away from a living host. Thus they are likely to undergo some inactivation even at the pH (approximately 6.2) that accompanies carcass maturation. On this basis, it was considered that there was a ‘low’ likelihood that meat infected at the time of slaughter would remain so after the process of carcass maturation.

R6 — the likelihood that the pathogenic agent will not be destroyed during cold storage and transport

There is no published information on the effects of cold storage on any of the three viruses. Alphaviruses are considered to remain viable for long periods when kept at low temperatures. Getah virus, also an alphavirus, can remain viable for 4 days at 37°C and for 3 months when stored at 10°C (Kamada, et al., 1982). In light of this, it was considered that there was a ‘high’ likelihood that meat infected at the completion of carcass maturation would remain infected during transport and cold storage.

Conclusions — release assessment

When these likelihoods were inserted into the simulation model, it was concluded that, in the absence of risk management and without considerations regarding the exporting country, the likelihoods that imported pig meat derived from an individual carcass would be infected with EEE, WEE or VEE viruses were ‘extremely low’, ‘negligible’ and ‘extremely low’, respectively.

Exposure assessment

The exposure assessment for feral pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that the likelihoods that a waste unit would be infected with EEE, WEE or VEE virus were ‘extremely low’, ‘negligible’ and ‘extremely low’, respectively.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

There is no published information on the oral infective dose of any of the three viruses, or on the concentration of virus within infected tissues of a pork carcass.

While pigs are susceptible to oral inoculation of EEE virus under experimental conditions, and can develop high titre viraemia and excrete the virus in their faeces, there is no epidemiologic evidence of oral transmission occurring in pig herds. Spread within a pig herd is likely to be the result of mosquitoes feeding on viraemic pigs or viraemic birds within the area and then infecting susceptible pigs. Generally mammals are considered to have low virus titres that are insufficient to infect vectors and, as such any virus in meat from blood is likely to be at a very low titre. This would be the case for all three viruses.

Given the absence of reports of infection arising from the consumption of meat from the Americas, it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of EEE, WEE or VEE virus to initiate infection was ‘extremely low’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to scavenging

This step describes the sensitivity of any of the three viruses to ultraviolet light, to ambient temperatures ranging from 10°C to 35°C and to the putrefying effects of saprophytic organisms, bearing in mind that some time may be required before meat scraps are located by scavenging feral pigs. Although there is no published information on the impact of these factors on the viability of any of the three viruses, some alphaviruses can survive storage at 37°C for 4 days and longer at lower temperatures, but they cannot survive for long away from a living host or a suitable culture medium. Given this, it was considered that the likelihood of any of the three viruses surviving within meat scraps discarded in refuse for the period of time required for feral pigs to locate and subsequently scavenge the material was ‘very low’.

L4 — the likelihood that the waste unit would be accessible to a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very likely that refuse would be accessible to feral pigs in a remote region. In a rural region it was considered that there was a moderate likelihood but that it was very unlikely that refuse from large towns would be accessible to feral pigs.

- Remote regions = High
- Rural regions = Moderate
- Large towns = Very low

L5 — the likelihood that a waste unit would be located by a feral pig

It was stated in the Method for Import Risk Analysis that it was considered very unlikely that any individual pig meat waste unit would be located by a feral pig scavenging in a remote region, extremely unlikely in a rural region and negligible in a region with large towns.

- Remote regions = Very low
- Rural regions = Extremely low
- Large towns = Negligible

N — the number of waste units discarded each year

As discussed in the Method for Import Risk Analysis the number of waste units discarded each year was determined for remote regions, rural regions and large towns based on the population in each of these regions and the total number of pig meat waste units generated per year (see Table 4).

Conclusions — annual likelihood of entry and exposure for feral pigs

It was explained in the Method for Import Risk Analysis that a separate annual likelihood of exposing feral pigs will be derived for remote regions, rural regions and large towns, and these subsequently combined to give an overall exposure assessment for feral pigs. The derivation of each annual likelihood for each region was explained in Table 5.

The annual likelihood of entry and exposure for feral pigs for EEE, WEE or VEE virus was:

- Remote regions = Negligible
- Rural regions = Negligible
- Large towns = Negligible

When the annual likelihood for each region were combined the overall annual likelihood of entry and exposure for feral pigs for EEE, WEE or VEE virus was found to be ‘negligible’.

The exposure assessment for backyard pigs

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that the likelihoods that a waste unit would be infected with EEE, WEE or VEE virus were ‘extremely low’, ‘negligible’ and ‘extremely low’, respectively.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of EEE, WEE or VEE virus to initiate infection was ‘extremely low’.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by backyard pigs would be more protected from exposure

to the elements than that likely to be accessible to feral pigs. Backyard pigs are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a 'low' likelihood that any of the three viruses would remain viable during the period prior to ingestion.

N — the number of waste units fed to backyard pigs during a year

As discussed in the Method for Import Risk Analysis the number of waste units generated and fed to backyard pigs during a year (see Table 6) was the product of:

- The total number of waste units generated and discarded by a household;
- The proportion of the total household waste units generated by households that keep backyard pigs; and
- The proportion of backyard pig producers that may illegally feed waste to their pigs.

Conclusions — annual likelihood of entry and exposure for backyard pigs

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for backyard pigs for EEE, WEE or VEE virus was found to be 'negligible'.

Exposure assessment for small commercial piggeries

L1 — the likelihood that a waste unit is infected

The likelihood that a waste unit is infected is equivalent to the result of the release assessment. It was considered that the likelihoods that a waste unit would be infected with EEE, WEE or VEE virus were 'extremely low', 'negligible' and 'extremely low', respectively.

L2 — the likelihood that a waste unit would contain a sufficient dose of the pathogenic agent to initiate infection

As discussed above in the exposure assessment for feral pigs it was considered that the likelihood that a waste unit from an infected pig would contain a sufficient dose of EEE, WEE or VEE virus to initiate infection was 'extremely low'.

L3 — the likelihood that the pathogenic agent would remain viable during the period prior to ingestion

It was considered that waste ingested by pigs in small commercial piggeries would be more protected from exposure to the elements than that likely to be accessible to feral pigs. Pigs in small commercial piggeries are likely to be fed scraps collected on a daily basis although in some instances pig meat may be being discarded due to spoilage.

Overall, the Panel considered that there was a 'low' likelihood that any of the three viruses would remain viable during the period prior to ingestion.

N — the number of waste units fed to pigs in small commercial piggeries during a year

As stated in the Method for Import Risk Analysis the number of waste units fed to pigs in a

small commercial piggery included both household waste and waste from food service establishments. The number of household waste units and the number of units from food service establishments was estimated independently as described in Table 7, and subsequently summed.

Conclusions — annual likelihood of entry and exposure for small commercial piggeries

When these likelihoods were inserted into the simulation model, the overall annual likelihood of entry and exposure for pigs in small commercial piggeries for EEE, WEE or VEE virus was found to be ‘negligible’.

Exposure assessment for other susceptible species

EEE virus

Natural infections are usually acquired by mosquito bites. There is no evidence of oral infection occurring in rodents or other mammals; however, oral infection with EEE virus in birds has been reported, particularly in ratites, turkeys, crows and pheasants (Brown, et al., 1993). Emus are highly susceptible to EEE virus, and can develop high titre viraemia and excrete the virus in their faeces (Tully, et al., 1992). Emu to emu transmission has occurred under experimental conditions (Wiersma, et al., 1998). However, they do not eat meat. Some American avian species susceptible to EEE virus eat meat and scavenge sites containing dead animals and meat wastes yet there are no published reports of infection arising from consumption of meat. Taking into consideration the extremely low likelihood of a waste unit containing a sufficient dose of EEE virus to initiate infection and the very low likelihood of the virus remaining viable during the period prior to ingestion or scavenging (as described earlier), the annual likelihood of entry and exposure for EEE virus for other susceptible species was considered to be ‘negligible’.

WEE virus

There is no published report on oral infection of mammals or birds with WEE virus. Ratites, particularly emus, are also susceptible to the virus but the disease is not as severe as with EEE virus (Randolph, et al., 1994). Transmission is by mosquito vectors.

Taking into consideration the extremely low likelihood of a waste unit containing a sufficient dose of WEE virus to initiate infection and the very low likelihood of the virus remaining viable during the period prior to ingestion or scavenging (as described earlier), the annual likelihood of entry and exposure for WEE virus for other susceptible species was considered to be ‘negligible’.

VEE virus

There is no published report on oral infection of mammals or birds with VEE virus. The virus normally cycles within the rodent-mosquito cycle and it may be possible for Australian native rodents to be infected. Infection is usually by mosquito vectors. However, accidental aerosol infections of humans have occurred with epizootic strains. Thus it may be possible for oral infection to occur.

Taking into consideration the extremely low likelihood of a waste unit containing a sufficient dose of VEE virus to initiate infection and the very low likelihood of the virus remaining viable during the period prior to ingestion or scavenging (as described earlier), the annual likelihood

of entry and exposure for VEE virus for other susceptible species was considered to be ‘negligible’.

Conclusions

The annual likelihood of entry and exposure for each of the exposure groups for EEE, WEE and VEE virus was determined to be negligible. As such further assessment was not conducted. No risk management measures would be required for EEE, WEE or VEE virus.

The Department of Health and Ageing has advised Biosecurity Australia that biosecurity measures for EEE, WEE and VEE virus would not be required to manage the risk to human life or health associated with the importation of pig meat.

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RISK MANAGEMENT FOR QUARANTINE DISEASES

Australia has determined that to meet its conservative Appropriate Level of Protection (ALOP) biosecurity risk must be 'very low' or less before it will allow the importation of animal or plant commodities into Australia. In situations where the unrestricted risk estimation has confirmed that the biosecurity risk associated with importation is unacceptably high, risk management assessment is used to determine how the biosecurity risks may be mitigated. The risk management assessment aims to identify and evaluate measures that could be used to reduce the biosecurity risks associated with the importation of the commodity to acceptable levels (i.e. very low or negligible).

Measures to reduce the likelihood of disease entry, establishment and/or spread can either reduce the likelihood that imported product contains the causative agent or reduce the likelihood of its exposure to susceptible animals. For many diseases, the Panel was unable to identify feasible measures that would reduce the risk of exposure of susceptible hosts to infected imported products with the required high degree of confidence.

In this chapter having identified and evaluated the disease agents requiring risk management, the least trade restrictive risk management measures that could be applied are evaluated. These measures form the basis for the guidelines for the importation of pig meat, as appropriate.

In the risk assessment chapters, the Panel assessed the unrestricted risk estimate for each disease agent, to ascertain whether it exceeded Australia's ALOP ('very low'). In cases where the unrestricted risk was found to be 'very low' or 'negligible' it was concluded that no risk management measures were required in respect of that disease. The unrestricted risk estimate was estimated to be higher for the following diseases (Table 115) and thus it was concluded that risk management measures would be required.

Table 115 Disease agents requiring risk management

Disease Agent	Unrestricted annual risk
Foot-and-mouth disease virus	Extreme
African swine fever virus	Moderate
Classical swine fever virus	Moderate
Swine vesicular disease	Moderate
Rinderpest virus	Extreme
Aujeszky's disease virus	Low
Porcine reproductive and respiratory syndrome (PRRS) virus	Low
Trichinella spiralis	Low
Nipah virus	Low
PMWS	Low

The approach to risk management was to consider the practicable range of measures which might be applied including, where available, the recommendations in the international standard (OIE Terrestrial Animal Health Code) for trade in pig meat. Each of the selected possible measures was then given detailed consideration to evaluate its effect on the likelihoods of the disease agent entering and establishing in Australia at the appropriate step in the pathway. An important consideration when evaluating the effectiveness of risk management measures was the ability to confirm that the measure would be properly implemented and would deliver the desired effect.

Certain options clearly reduced the risk to a negligible level. An example was the requirement that the country or zone be free of the agent. In such cases there was no need for further consideration and that option was deemed acceptable, subject of course to the country's veterinary services having the capacity to determine disease freedom (see below). Another example was canned pig meat heated to an internal temperature of 100°C (i.e. shelf stable), which is currently permitted import into Australia.

In other cases, where a risk management measure might reduce the risk at a certain step but not eliminate it, further evaluation of the end result of that measure on the level of risk was required. In the case where further processing of pig meat (i.e. cooking, curing at specified times/temperatures) was examined as a risk management measure, an additional step (R7) was inserted into the release pathway. The likelihood assigned to R7 represents the probability that a pathogenic agent would not be destroyed by the specified processing.

The means by which the end result of the risk management measure was evaluated was to modify the parameters of the risk simulation model and to assess the effect of the change in terms of the overall annual risk. Where the effect was to reduce the overall annual risk to 'very low' or lower, the measure was deemed acceptable. Sometimes a combination of more than one risk management measure was an appropriate mix to meet Australia's ALOP.

In its consideration of risk management measures, the Panel was mindful that these should be the least trade restrictive measures which would meet Australia's ALOP. Attention was also given to normal commercial processing practices. An example was the consideration of perishable hams which require refrigeration and which typically are cooked to an internal temperature of around 69°C as against canned shelf stable hams which typically are cooked to above 100°C. Separate consideration was given to the effect of the lower cooking temperature requirement for perishable hams or uncanned hams on disease agents requiring risk management. In this way, the possibility of importation of several types of product could be encompassed.

In order for Australian authorities to be satisfied that a country or zone is free of a given disease, they must have a knowledge of the veterinary services of that country and be satisfied that those veterinary services have the capacity for disease control, monitoring and surveillance as appropriate for the disease. In some cases, it might be necessary for the disease to be subject to compulsory reporting or be the subject of consideration in disease investigation. Australia's "Guidelines for the approval of countries to export animals (including fish) and their products to Australia" have been published (ABPM 1999/41).

Correct identification of the origin of imported pig meat is central to the application of specific risk management measures. This entails correct identification of the animals of origin and their farm of origin, correct identification of animals eligible for export to Australia, and correct identification of the meat derived from these pigs during all stages including slaughter, chilling,

boning, processing and storage. It also entails correct segregation to ensure that the product maintains its status and is not contaminated by disease agents.

Risk management measures can either reduce the impact of a disease if it were to enter, establish and/or spread in Australia or alternatively reduce the likelihood of the disease agent's entry, establishment and/or spread. Australia has a long history of implementing measures to reduce the likelihood of susceptible host exposure, for example farmer awareness, controls on swill feeding of pigs and feral pig control programs. Other programs help to limit the impact of disease establishment, for example emergency control plans to limit spread and stamp out disease and access to emergency vaccine reserves. These programs were taken into consideration in making the unrestricted risk estimate, in particular in the consequence assessment.

The effectiveness of these programs is limited to the extent that it is not feasible or cost effective to control the actions of all people in Australia or negate all risk factors such as the presence of feral pigs. The programs that in are place seek to manage such risks and are targeted at the more significant risk factors; they also provide early advice of disease outbreaks. However, the consequences that would arise from the outbreak of diseases, such as foot-and-mouth disease, are serious both in terms of reduced production efficiency through increased costs and loss of access to export markets (access to many of Australia's export markets is based on our disease status, i.e. country disease freedom).

Australia is committed to exotic disease preparedness and will continue to investigate and develop emergency programs for the rapid identification, limiting the impact and stamping out of exotic diseases.

Foot-and-mouth disease virus

The international standard (OIE Terrestrial Animal Health Code) recommends that consideration be given to the risk of foot-and-mouth disease (FMD) when importing pig meat (Office international des épizooties, 2003c). *Inter alia*, the measures which the OIE recommends for the importation of pig meat can be summarised as follows:

- a requirement that the pigs have been kept since birth in an FMD free country or zone, have not been vaccinated, and have been slaughtered in an approved abattoir and subjected to ante-mortem and post-mortem inspections for FMD with favourable results;
- meat, may be imported from an FMD infected country or zone, if subjected to heat treatment in a hermetically sealed container to reach an internal core temperature of at least 70°C for a minimum of 30 minutes, or equivalent treatment which has been demonstrated to inactivate FMD virus;
- meat, deboned and defatted, may be imported from an FMD infected country or zone if it has been processed to destroy FMD virus by heating to a core temperature of at least 70°C for a minimum of 30 minutes or if it has been deboned and processed by drying after salting.

Other measures that could also be considered to reduce the likelihood of entry, establishment and/or spread of FMD virus via imported pig meat may include a requirement that the pigs from which the meat was derived were sourced from premises on which there had been no evidence of FMD within the 3 months prior to slaughter.

Country or zone freedom

Country or zone freedom from FMD to the satisfaction of Australian authorities would conform with Australia's ALOP (as discussed in the introduction to this Chapter, such approaches will be dealt with using existing mechanisms). Vaccination of animals may mask the symptoms of FMD in a herd and animals vaccinated against one serotype may be susceptible to infection with another. Hence, the Panel considered that to be considered free from FMD, a country or zone should not permit vaccination.

Heat processing to inactivate FMD virus

Canning - shelf stable

Australia currently accepts shelf stable canned pig meat from any source country subject to certain conditions. The Australian import conditions for canned meat include a requirement that all portions of the contents have been heated to at least 100°C. The Panel considered that FMD virus would be inactivated in canned pig meat heated to at least 100°C.

Pasteurised canned hams or cooked pig meat

Heat processing of pig meat from a FMD infected country or zone by canning and pasteurisation (i.e. to at least 69°C) or cooking would influence the additional likelihood step in the release pathway (R7). This step describes the likelihood that FMD virus would survive in pasteurised canned hams or cooked pig meat. Heat processing may also affect the exposure step L2, which describes the likelihood that a waste unit would contain a sufficient dose of FMD virus to initiate infection.

Several studies have examined the effect of heat treatment on FMD virus survival in meat or associated tissues. In one study, four pigs were experimentally inoculated with one of two strains of FMD virus and slaughtered at 72 hours. Lymph node, blood clot and bone marrow from these animals were heated to an internal temperature of 69°C for 0, 15, 30, 60 or 90 minutes. Foot-and- mouth disease virus could not be isolated from the heated lymph node samples for either strain of virus at 0 minutes when tested by inoculation into two naive pigs. The bone marrow and blood clot samples were unable to be tested (McKercher, et al., 1980). By contrast, FMD virus was detected (by bioassay in cattle but not by cell culture) in ground beef contaminated with FMD infected ground lymph node material processed to core temperatures of 63°C and 71.2°C. However, the virus was not detected in the same material when heated to 79.4°C (Blackwell, et al., 1988). A further study demonstrated that FMD virus was not inactivated in ground beef samples (including lymph nodes) when heated to 72°C for 30 minutes and 78°C for 10 minutes (Garcia-Vidal, et al, 1988). This data contrasts with the OIE recommendation that meat from FMD infected countries be processed at 70°C for 30 minutes for inactivation of the virus.

The Panel considered that there would be a reduction in the titre of FMD virus in pasteurised canned hams and cooked pig meat which have been heated to a minimum internal temperature of 69°C. Due to the conflicting results, the limited studies that have been undertaken and the small sample size in the studies, the Panel considered that further information was required in order to assess the likelihood of survival of FMD virus in pasteurised hams and cooked products.

Salting to inactivate FMD virus

Several studies have demonstrated that salting is not sufficient to inactivate FMD virus. Salting of casings did not inactivate the virus (Cottral, 1969; Bohm, & Krebs, 1974). Virus has been isolated from processed casings for up to 240 days (Heidelbaugh, & Graves, 1968). Foot-and-mouth disease virus has been isolated in salted bacon for 190 days (Dhennin, et al, 1980).

The Panel considered that salting would not reduce the likelihood of entry of FMD virus in pig meat products.

Curing to inactivate FMD virus

Parma, Iberian and Serrano hams

The survival of FMD virus in dry cured Parma hams has been studied in experiments in the United States of America and in Italy (McKercher, et al., 1987). Hams used in this experiment originated from experimentally inoculated pigs slaughtered 48 hours after inoculation. Samples of muscle, fat and bone marrow were negative on day 108 and day 136 of curing in the American study and day 170 and day 227 of curing in the Italian study. Samples were not tested at day 108 and 136 in the Italian study. Testing included animal inoculation of samples of muscle, bone marrow and fat.

The Panel considered that there would be a 'very low' likelihood that FMD virus would survive in dry cured Parma type hams according to the processes specified in the study above when cured for a minimum of 170 days.

The survival of FMD virus in "Serrano" hams and "Iberian" hams, loins and shoulders was studied in typical Spanish dry cured products (Mebus, et al., 1993a). Meat used in this experiment originated from experimentally inoculated pigs, 31 black Iberian pigs and 31 Spanish white pigs, slaughtered at the estimated peak of viraemia (2 days post-inoculation). At slaughter FMD virus titres varied from an average of $10^{0.2}$ PFU per gram in fat to $10^{4.37}$ in blood. Virus titres in muscle were very low averaging $10^{0.1}$ PFU per gram. Samples (muscle, bone marrow, fat and lymph node) of Iberian ham taken on days 160, 196 and 224 of curing which were negative on culture were confirmed negative by bioassay through intramuscular inoculation of naive pigs. Samples of Iberian shoulder and loin taken on days 112, 140 and 168 of curing and days 42, 56 and 79 respectively were negative by animal inoculation. Samples of Serrano ham taken on days 182, 196 and 210 of curing were negative by animal inoculation.

Due to the different times required for a negative sample to be obtained from different products, with no adequate explanation as to why these differed, the Panel considered a minimum curing time of 182 days for Iberian hams, shoulders, loins and Serrano hams. Given this, the Panel considered that there would be a 'very low' likelihood that FMD virus would survive in cured Serrano type hams and Iberian type hams, loins and shoulders when cured for a minimum of 182 days.

If cured hams (Parma, Iberian and Serrano) were imported the likelihood assigned to L2 may also be affected. Pigs are easily infected by FMD virus via the oral route. Swill feeding of infected meat (uncooked or inadequately cooked) is frequently linked to outbreaks of disease. Nonetheless the Panel considered that the virus titre in pig meat would be reduced following curing. Given this, it was considered that the likelihood assigned to L2 could be reduced from 'high' to 'low'.

Premises of origin free of FMD for past 3 months

A requirement that the premises from which pigs had been selected had been free of any evidence (clinical, serological, virological) of FMD within the 3 months prior to slaughter would influence the first step in the release pathway (R1). This step describes the likelihood of selecting slaughter-age pigs from an infected herd. The disease would need to be notifiable.

The period of 3 months was chosen by taking into account the likely epidemiological picture of FMD infection within a herd. New cases originating from within the herd would generally be expected to occur within 3 months of the last case.

If pigs were sourced from herds free from FMD for the 3 months prior to slaughter, the likelihood assigned to R1 could be reduced from 'moderate' to 'very low'.

Conclusions

The 'restricted risk' step estimates were as follows:

- the likelihood that FMD virus would survive in cured hams, loins, shoulders was estimated to be 'very low'; and
  - the likelihood that a waste unit (cured product) from an infected pig would contain a sufficient dose of FMD virus to initiate infection was estimated to be 'low';
- the likelihood of selecting slaughter-age pigs from a herd free from evidence of FMD infection for the past 3 months was estimated to be 'very low'.

Risk was estimated using the various combinations of mitigation measures discussed above and the results are summarised below (Table 116). Herd freedom, dry curing of hams either alone or in combination, would not reduce the likelihood of entry, establishment and/or spread to an acceptable level.

Table 116 Risk management measures for FMD virus

R1	R7	Restricted risk
Herd freedom	Cured hams (Parma, Iberian, Serrano)	
-	-	Extreme
+	-	Extreme
-	+	Extreme
+	+	Moderate

- measure/s not applied

+ measure/s applied

There are two alternative options for management of the risk posed by FMD virus, each of which would meet Australia's ALOP:

- the pigs from which the meat has been derived have been kept since birth in an FMD free country or zone to the satisfaction of Australian authorities, have not been vaccinated, and have been slaughtered in an approved abattoir and subjected to ante-mortem and post-mortem inspections for FMD with favourable results; or

- the pig meat has been canned such that all portions of the can contents have been heated to at least 100°C.

The Department of Health and Ageing has advised Biosecurity Australia that biosecurity measures for FMD virus would not be required to manage the risk to human life or health associated with the importation of pig meat.

African swine fever virus

The international standard (OIE Terrestrial Animal Health Code) recommends that consideration be given to the risk of African swine fever (ASF) when importing pig meat (Office international des épizooties, 2003a). *Inter alia*, the measures which the OIE recommends for the importation of pig meat include:

- a requirement that the pigs have been kept since birth in an ASF free country or zone, have been slaughtered in an approved abattoir situated in an ASF free country or zone and which only receives animals from an ASF free country or zone, and have been subjected to ante-mortem and post-mortem inspections for ASF with favourable results;
- pig meat may be imported from an infected country or zone if it has been processed to ensure the destruction of the ASF virus.

The OIE Code does not specify any particular process for destruction of ASF virus in pig meat.

Other measures that could also be considered to reduce the likelihood of entry, establishment and/or spread of ASF virus via imported pig meat may include a requirement that the pigs from which the meat was derived were sourced from premises on which there had been no evidence of ASF within the 3 months prior to slaughter.

Options

Country or zone freedom

Country or zone freedom from ASF to the satisfaction of Australian authorities would conform with Australia's ALOP (as discussed in the introduction to this Chapter, such approaches will be dealt with using existing mechanisms).

Heat processing to inactivate ASF virus

Canning - shelf stable

One study concluded that pig meat products reaching 69°C during processing were unlikely to contain residual ASF virus (McKercher, et al., 1980). Australia currently accepts shelf stable canned pig meat from any source country, subject to certain conditions. The Australian import conditions for canned meat include a requirement that all portions of the contents have been heated to at least 100°C. The Panel considered that ASF virus would be inactivated in canned pig meat heated to at least 100°C.

Pasteurised canned hams or cooked pig meat

Heat processing pig meat from an ASF infected country or zone by canning and pasteurisation (i.e. to at least 69°C) or cooking would influence the additional likelihood step in the release pathway (R7). This step describes the likelihood that ASF virus would survive in pasteurised

canned hams or cooked product. Heat processing may also affect the exposure step L2, which describes the likelihood that a waste unit would contain a sufficient dose of ASF virus to initiate infection.

The effect of heating perishable canned hams to an internal temperature of 69°C on the survivability of ASF virus was examined (McKercher, et al., 1978). The hams (0.8 kg) were prepared from four experimentally inoculated pigs slaughtered 48 hours post-inoculation. Virus could not be recovered from the hams after heating to 69°C. Neither could it be recovered from two naïve pigs inoculated with 1 gram samples of the product.

The effect of heating on ASF virus was also examined in tissues that were not canned. Lymph node, blood clot and bone marrow from two pigs experimentally inoculated with ASF virus, were heated to an internal temperature of 69°C for 2, 30 and 60 minutes. African swine fever virus could not be isolated from lymph nodes heated at this temperature for the three time periods nor following inoculation of each of two pigs with the treated samples. The blood and bone marrow samples heated for 0 and 15 minutes were also inoculated into each of two pigs, without causing disease (McKercher, et al., 1980).

Given the limited number of studies conducted under experimental conditions on the inactivation of ASF virus in pasteurised canned hams or cooked product, and the small numbers of pigs involved, the Panel considered that there would be a ‘low’ likelihood that ASF virus would survive in pasteurised canned hams or cooked deboned product heated to a minimum internal temperature of 69°C.

In considering pasteurised canned hams or cooked product the likelihood assigned to L2 may also be reduced. There are limited data on the oral infectious dose of ASF virus although it is known that swill feeding of infected meat (uncooked or inadequately cooked) is frequently linked to outbreaks of disease. Nonetheless the Panel considered that the virus titre in pig meat would be reduced following canning and pasteurisation or cooking to 69°C. Given this, it was considered that the likelihood assigned to L2 could be reduced from ‘high’ to ‘low’.

Curing to inactivate ASF virus

The curing of pig meat sourced from an ASF infected country or zone would influence the additional likelihood step in the release pathway (R7). This step describes the likelihood that ASF virus would survive in cured pig meat products. Curing may also affect the exposure step L2, which describes the likelihood that a waste unit would contain a sufficient dose of ASF virus to initiate infection.

Several studies have examined the effects of various methods of curing on the persistence of viruses such as ASF.

Pepperoni and salami sausages

In dried pepperoni and salami sausages cured in the presence of sodium nitrite and sodium nitrate, ASF virus was demonstrated at days 8 and 9 after slaughter but not on day 30 (McKercher, et al., 1978). The meat used in these products was from four viraemic pigs killed 48 hours after experimental inoculation with ASF virus. The viral titre of the meat from these pigs ranged from $10^{3.25}$ to $10^{3.75}$ HAD_{50} (50% haemadsorbing doses) per gram. These titres are considerably lower than those of other studies. The detection method included testing for haemadsorption on porcine buffy coat cultures of samples and also bioassay by way of intramuscular inoculation of naive pigs. The Panel concluded that the titre of ASF virus in pig

meat which has been subject to at least 30 days of curing in the presence of sodium nitrite and sodium nitrate, would be reduced. However, there was insufficient data, to assign a likelihood to R7. Further information would be required before such curing processes could be considered further.

Parma, Iberian and Serrano hams

The survival of ASF virus in dry cured "Parma" hams has been studied in experiments in the United States of America and in Italy (McKercher, et al., 1987). Hams used in this experiment originated from experimentally inoculated pigs slaughtered 5 days (estimated peak viraemia) after inoculation. The viral titre at the start of curing averaged 10^5 HAD_{50} per gram of tissue. In the American study involving 12 infected pigs, ASF virus became undetectable some time between day 291 and day 399 of curing when tested by animal inoculation using samples of muscle, bone marrow and fat and haemadsorption. Virus was not recovered at 399 days. In the Italian study involving eight infected pigs, ASF virus became undetectable sometime between day 180 and day 300 of curing when tested by animal inoculation and haemadsorption. Virus was not recovered at 300 days. No explanation was provided as to why the virus persisted for a longer period in the American study. The authors concluded that there would be little probability of the infectivity of the virus extending beyond the 365 day minimum curing process of the product.

The Panel considered that there would be a 'very low' likelihood that ASF virus would survive in dry cured Parma type hams according to the processes specified in the study above when cured for a minimum of 399 days.

The survival of ASF virus in "Serrano" hams and "Iberian" hams, loins and shoulders was studied in typical Spanish dry cured products (Mebus, et al., 1993a). Meat used in this experiment originated from experimentally inoculated pigs, 35 black Iberian pigs and 32 Spanish white pigs, slaughtered at the estimated peak of viraemia (5 days post-inoculation). At slaughter ASF virus titres varied from an average of $10^{4.9}$ HAD_{50} per gram in fat to $10^{9.7}$ in bone marrow. African swine fever virus became undetectable in the cured meat between days 112 and 140 days. Samples were still positive at 112 days. Detection in this experiment was by haemadsorption on porcine buffy coat cultures. Samples (muscle, bone marrow, fat and lymph node from hams) taken on days 140, 168 and 196 of curing which were negative on culture were confirmed negative by bioassay through intramuscular inoculation of naive pigs.

The Panel considered that there would be a 'very low' likelihood that ASF virus would survive in cured Serrano type hams and Iberian type hams, loins and shoulders, when cured for a minimum period of 140 days.

If cured hams (Parma, Iberian and Serrano) were imported the likelihood assigned to L2 may also be affected. There are limited data on the oral infectious dose of ASF virus although it is known that swill feeding of infected meat (uncooked or inadequately cooked) is frequently linked to outbreaks of disease. Nonetheless the Panel considered that the virus titre in pig meat would be reduced following curing. Given this, it was considered that the likelihood assigned to L2 could be reduced from 'high' to 'low'.

Premises of origin free of ASF for past 3 months

A requirement that the premises from which pigs had been selected had been free of any evidence (clinical, serological, virological) of ASF within the 3 months prior to slaughter would

influence the first step in the release pathway (R1). This step describes the likelihood of selecting slaughter-age pigs from an infected herd. The disease would need to be notifiable.

The period of 3 months was chosen by taking into account the likely epidemiological picture of ASF infection within a herd. New cases originating from within the herd would generally be expected to occur within 3 months of the last case. The virus can persist in tissues of recovered animals but the role of these carrier animals in transmission is not clear. Recovered pigs do not appear to shed the virus 1 month after infection nor is the virus transmitted by their secretions and excretions (McVicar, 1984). Other tissues are unlikely to sustain detectable infective levels of ASF virus for more than two months after infection (Mebus, 1988).

If pigs were sourced from herds free from ASF for the 3 months prior to slaughter, the likelihood assigned to R1 could be reduced from ‘low’ to ‘very low’.

Conclusions

The ‘restricted risk’ step estimates were as follows:

- the likelihood that ASF virus would survive in pasteurised canned hams or cooked pig meat heated to a minimum internal temperature of 69°C, was estimated to be ‘low’; and
  - the likelihood that a waste unit (pasteurised ham or cooked product) from an infected pig would contain a sufficient dose of ASF virus to initiate infection was estimated to be ‘low’;
- the likelihood that ASF virus would survive in cured hams, loins, shoulders was estimated to be ‘very low’; and
  - the likelihood that a waste unit (cured product) from an infected pig would contain a sufficient dose of ASF virus to initiate infection was estimated to be ‘low’;
- the likelihood of selecting slaughter-age pigs from a herd free from evidence of ASF infection for the past 3 months was estimated to be ‘very low’.

Risk was estimated using the various combinations of mitigation measures discussed above and the results are summarised in Table 117. Processing of pig meat by curing under specified conditions for Parma, Iberian and Serrano type hams together with certification that the pigs had been sourced from premises which had been free from evidence (clinical, serological, virological) of ASF infection for the 3 months prior to slaughter would reduce the risk of entry, establishment and/or spread to very low, which would meet Australia’s ALOP. Herd freedom, pasteurisation or cooking to 69°C either alone or in combination, and dry curing of hams alone, would not reduce the likelihood of entry, establishment and/or spread to an acceptable level.

Table 117 Risk management measures for ASF virus

R1 Herd freedom	R7 Pasteurised canned hams or cooked pig meat	R7 Cured hams (Parma, Iberian, Serrano)	Restricted risk
-	-	-	Moderate
+	-	-	Moderate
-	+	-	Moderate
+	+	-	Low
-	-	+	Low
+	-	+	Very low

- measure/s not applied

+ measure/s applied

Thus, there are several alternative options for management of the risk posed by ASF, each of which would meet Australia's ALOP:

- a requirement that the pigs from which the meat is derived have been kept since birth in a country or zone which is free of ASF to the satisfaction of Australian authorities; or
- a requirement that the pig meat had been processed by canning such that all portions of the contents have been heated to at least 100°C; or
- a requirement that the pig meat had been dry cured under specified conditions for Parma type hams (minimum curing 399 days), Iberian type hams, loins or shoulders, or Serrano type hams[70] (minimum curing 140 days), combined with certification that the pigs had been sourced from premises which had been free from evidence (clinical, serological, virological) of ASF infection for the past 3 months (the disease must be notifiable).

Classical swine fever virus

The international standard (OIE Terrestrial Animal Health Code) recommends that consideration be given to the risk of classical swine fever (CSF) when importing pig meat (Office international des épizooties, 2003b). The Code makes a distinction between domestic and wild pigs in terms of CSF infection and recognises the existence of countries or zones free of CSF in domestic pigs but with infection in wild pigs. Given this situation, the relevant OIE measures for fresh pig meat can be summarised as follows:

- a requirement that the pigs have been kept in a country or zone free of CSF since birth or for at least the past 3 months, and have been subjected to ante- and post-mortem inspections and have been found free of any sign suggestive of CSF;
- pig meat may be imported from an infected country or zone if it has been processed so as to ensure the destruction of the CSF virus.

[70] The processes used have been published in summarised form (McKercher, et al., 1985; Mebus, et al., 1993a) and full details are available through the bodies responsible for control of the production and certification of the product such as the Consorzio del Prosciutto di Parma, Fundacion del Jamon Serrano; Consorcio del J.Serrano.

The OIE Code recommends that for the inactivation of CSF virus in meat one of the following procedures be used:

- meat shall be heat treated in a hermetically sealed container with a Fo value of 3.00 or heat treated to a minimum temperature of 70°C;
- hams should be subjected to natural fermentation and maturation process for at least 190 days and loins for 140 days where the aw value is not more than 0.93 or the pH value is not more than 6.0;
- Italian style hams with bone-in should be cured with salt and dried for a minimum of 313 days;
- Spanish style pork meat with bone-in should be cured with salt and dried for a minimum of 252 days for Iberian hams, 140 days for Iberian shoulders, 126 days for Iberian loin and 140 days for Serrano hams.

Other measures that could also be considered to reduce the likelihood of entry, establishment and/or spread of CSF virus via imported pig meat may include a requirement that the pigs from which the meat was derived were sourced from premises on which there had been no evidence of CSF within the 3 months prior to slaughter.

Options

Country or zone freedom

Country or zone freedom from CSF to the satisfaction of Australian authorities would conform with Australia's ALOP (as discussed in the introduction to this Chapter, such approaches will be dealt with using existing mechanisms.

Heat processing to inactivate CSF virus

Canning - shelf stable

In one report, heating pig meat at 69°C inactivated CSF virus (McKercher, et al., 1978); in another it was inactivated after heating to 71°C for 1 minute (Stewart, et al., 1979). However, CSF virus in defibrinated blood was not inactivated when heated at 68°C for 30 minutes but was inactivated after 45 minutes and at 69°C for 30 minutes (Torrey & Prather, 1963). The virus was not inactivated in homogenates of spleen, tonsils, lymph nodes, muscle and viscera and blood at 80°C for 1 minute but was inactivated at 110°C for 30 seconds (Downing, et al., 1977). Australia currently accepts shelf stable canned pig meat from any source country, subject to certain conditions. The Australian import conditions for canned meat include a requirement that all portions of the contents have been heated to at least 100°C. The Panel considered that CSF virus would be inactivated in canned pig meat heated to at least 100°C.

Pasteurised canned hams or cooked pig meat

Heat processing of pig meat from a CSF infected country or zone by canning and pasteurisation or cooking (i.e. to at least 69°C) would influence the additional likelihood step in the release pathway (R7). This step describes the likelihood that CSF virus would survive in pasteurised canned hams or cooked pig meat. Heat processing may also affect the exposure step L2, which describes the likelihood that a waste unit would contain a sufficient dose of CSF virus to initiate infection.

In perishable canned hams heated to an internal temperature of 69°C and prepared from two experimentally inoculated animals, CSF virus could not be isolated from the processed meat (McKercher, et al., 1978). The assay used in this case included fluorescent antibody test of cell culture on PK15 cells and bioassay by intramuscular inoculation of two naive pigs. In this study the virus content of meat ranged from $10^{1.5}$ to $10^{1.87}$ plaque-forming units (pfu) per gram. In another study, the virus was destroyed in canned cured hams when an internal temperature of 65 deg C was sustained for 90 minutes (Stewart, et al., 1979). The peak internal temperature reached in these canned hams was 69°C. The assay used in this experiment included fluorescent antibody test of cell culture on PK15 cells and bioassay by intramuscular inoculation of naive pigs. The viral titre in these hams prior to heating varied widely from as little as 10^{1} to 10^{4} pfu/ml of suspension. The viability of CSF virus in cooked 0.45 kg canned hams was examined after heating in a water bath at 82°C for 50, 75 or 100 minutes (Helwig & Keast, 1966). Classical swine fever virus was inactivated in canned ham when it was estimated that centre of the product was maintained at a temperature of 65.5°C for 30 minutes.

The effect of heating has also been examined in tissues that were not canned. Lymph node, blood clot and bone marrow from two pigs experimentally inoculated with CSF virus, were heated to an internal temperature of 69°C for 0, 15, 30, 60 and 90 minutes. When tested by cell culture and by inoculation into naive pigs, CSF virus could be isolated when the samples had been reached 69°C for 0 minutes but not when held at that temperature for 15 or more minutes (McKercher, et al., 1980). In defibrinated blood CSF virus was not inactivated when heated at 68°C for 30 minutes but was inactivated after 45 minutes and at 69°C for 30 minutes (Torrey & Prather, 1963). Inactivation was determined by animal inoculation.

Given the limited number of studies conducted under experimental conditions on inactivation of CSF virus in pasteurised canned hams or cooked pig meat, the small number of pigs involved and the variation in the reported results, the Panel considered that there would be a ‘low’ likelihood that CSF virus would survive in pasteurised canned hams or cooked pig meat heated to a minimum internal temperature of 69°C for 15 minutes.

In considering pasteurised canned hams or cooked pig meat the likelihood assigned to L2 may also be affected. It has been demonstrated that the oral infectious dose of CSF virus is very low. An oral dose as little as 10 $TCID_{50}$ can cause fatal disease in pigs. Virus has been detected in muscle, lymph node and bone marrow at titres generally exceeding the oral infectious dose. It has been stated that only a few grams of infected tissue would be required to orally infect pigs. The Panel considered that the virus titre in pig meat would be reduced following canning and pasteurisation or cooking to 69°C for 15 minutes. Given this, it was considered that the likelihood assigned to L2 could be reduced from ‘high’ to ‘low’.

Curing to inactivate CSF virus

The curing of pig meat sourced from a CSF infected country or zone would influence the additional likelihood step in the release pathway (R7). This step describes the likelihood that CSF virus would survive in cured pig meat products. Curing may also affect the exposure step L2, which describes the likelihood that a waste unit would contain a sufficient dose of CSF virus to initiate infection.

There have been several studies on the effects of various methods of curing on the persistence of CSF virus.

Pepperoni and salami sausages

In dried pepperoni and salami sausages cured in the presence of sodium nitrite and sodium nitrate, CSF virus was demonstrated on day 22 after slaughter but not on day 104 (McKercher, et al., 1978). The meat used in these hams and sausages was from two viraemic pigs killed 5 days (peak body temperature) after experimental inoculation with CSF virus. The viral titre of the meat from these pigs ranged from $10^{1.5}$ to $10^{1.87}$ pfu/g. The detection method included virus isolation on PK-15 cell culture and examination using fluorescent antibody, virus isolation after passage on primary swine cell cultures and bioassay by way of intramuscular inoculation of two naive pigs.

The Panel concluded that the normal "traditional" curing time of the order of 2 to 4 weeks would not reduce the likelihood of entry of CSF virus in these types of product.

Parma, Iberian and Serrano hams

The survival of CSF virus in dry cured Parma hams has been studied in experiments in the United States of America and in Italy (McKercher, et al., 1987). Hams used in this experiment originated from experimentally inoculated pigs slaughtered 5 days (estimated peak viraemia) after inoculation. The viral titre at the start of this experiment ranged from $10^{1.5}$ pfu/g in fat to $10^{4.7}$ pfu/g in bone marrow. In the American study involving 12 infected pigs, CSF virus became undetectable some time between day 199 and day 313 of curing. Virus was not detected at 313 days. In the Italian study involving eight infected pigs, CSF virus became undetectable between day 112 and day 189 of curing. No explanation was provided as to why the virus persisted longer in the American study.

The Panel considered that there would be a 'very low' likelihood that CSF virus would survive in dry cured Parma type hams according to the processes specified in the study above when cured for a minimum of 313 days.

The survival of CSF virus in Serrano hams and Iberian hams, loins and shoulders was studied in typical Spanish dry cured products (Mebus, et al., 1993a). Meat used in this experiment originated from experimentally inoculated pigs, 32 black Iberian pigs and 32 Spanish white pigs, slaughtered at the estimated peak of viraemia (5 days post-inoculation for white pigs and 4 days post-inoculation for black pigs). At slaughter CSF virus titres varied from an average of $10^{0.8}$ pfu/g in fat to $10^{5.6}$ pfu/g in bone marrow. Virus titres in muscle were very low, an average of $10^{0.9}$ and $10^{1.1}$ pfu/g in Iberian back pigs and Spanish white pigs respectively. Classical swine fever virus became undetectable in the Iberian hams in pooled samples collected on days 252, 280 and 343 of curing and in Iberian shoulders and Serrano hams in pooled samples collected from day 140 up to day 196 of curing. The assay used here included a fluorescent antibody test on PK15 cells with passage of negative samples, and bioassay by intramuscular inoculation of naive pigs. Samples of muscle, bone marrow, fat and lymph node from the hams and samples muscle, fat and bone marrow from the shoulders which were negative on culture were confirmed negative by bioassay through intramuscular inoculation of naive pigs. No explanation was provided as to why Iberian hams remained positive for CSF virus almost twice as long as that of Iberian shoulders although it is noted that lymph node was not included in the pooled sample for Iberian shoulder. Other possible explanations include not all tissue samples were tested *in vitro* at predetermined intervals, there was a wide variation in virus titre in tissues of individual pigs and only three pooled samples were used for testing *in vivo*. For example, the CSF virus titre in lymph node and bone marrow of individual pigs ranged from just detectable to greater that 10^5 pfu/g and 10^6 pfu/g respectively.

Due to the different times required for a negative sample to be obtained from different products the Panel considered a minimum curing time of 252 days for Iberian hams, shoulders, loins and Serrano hams. Given this, the Panel considered that there would be a 'very low' likelihood that CSF virus would survive in cured Serrano type hams and Iberian type hams, loins and shoulders when cured for a minimum of 252 days.

If cured hams were imported the likelihood assigned to L2 may also be affected. It has been demonstrated that the oral infectious dose of CSF virus is very low. An oral dose as little as 10 $TCID_{50}$ can cause fatal disease in pigs. Virus has been detected in muscle, lymph node and bone marrow at titres generally exceeding the oral infectious dose. It has been stated that only a few grams of infected tissue would be required to orally infect pigs. The Panel considered that the virus titre in pig meat would be reduced following curing. Given this, it was considered that the likelihood assigned to L2 could be reduced from 'high' to 'low'.

Premises of origin free of CSF for past 3 months

A requirement that the premises from which pigs had been selected had been free of any evidence (clinical, serological, virological) of CSF within the 3 months prior to slaughter would influence the first step in the release pathway (R1). This step describes the likelihood of selecting slaughter-age pigs from an infected herd. The disease would need to be notifiable.

The period of 3 months was chosen by taking into account the likely epidemiological picture of CSF infection within a herd. New cases originating from within the herd would generally be expected to occur and be detected within 3 months of the last case.

If pigs were sourced from herds free from CSF for the 3 months prior to slaughter, the likelihood assigned to R1 could be reduced from 'low' to 'very low'.

Conclusions

The 'restricted risk' step estimates were as follows:

- the likelihood that CSF virus would survive in pasteurised canned hams or cooked pig meat heated to a minimum internal temperature of 69°C for 15 minutes, was estimated to be 'low'; and
  - the likelihood that a waste unit (pasteurised ham or cooked product) from an infected pig would contain a sufficient dose of CSF virus to initiate infection was estimated to be 'low';
- the likelihood that CSF virus would survive in cured hams, loins, shoulders was estimated to be 'very low'; and
  - the likelihood that a waste unit (cured product) from an infected pig would contain a sufficient dose of CSF virus to initiate infection was estimated to be 'low';
- the likelihood of selecting slaughter-age pigs from a herd free from evidence of CSF infection for the 3 months prior to slaughter was estimated to be 'very low'.

Risk was estimated using the various combinations of mitigation measures discussed above and the results are summarised in Table 118. Processing of pig meat by curing under specified conditions for Parma, Iberian and Serrano type hams together with certification that the pigs had been sourced from premises which had been free from evidence (clinical, serological, virological) of CSF infection for the past 3 months would reduce the risk of entry, establishment and/or spread to very low, which would meet Australia's ALOP. Herd freedom,

pasteurisation or cooking at 69°C for 15 minutes either alone, or in combination, and dry curing of hams alone would not reduce the likelihood of entry, establishment and/or spread to an acceptable level.

Table 118 Risk management measures for CSF virus

R1	R7	R7	Restricted risk
Herd freedom	Pasteurised canned hams or cooked pig meat	Cured hams (Parma, Iberian, Serrano)	
-	-	-	Moderate
+	-	-	Moderate
-	+	-	Moderate
+	+	-	Low
-	-	+	Low
+	-	+	Very low

- measure/s not applied

+ measure/s applied

Thus, there are several alternative options for management of the risk posed by CSF, each of which would meet Australia's ALOP:

- a requirement that the pigs from which the meat is derived have been kept since birth in a country or zone which is free of CSF to the satisfaction of Australian authorities; or
- a requirement that the pig meat had been processed by canning such that all portions of the contents have been heated to at least 100°C; or
- a requirement that the pig meat had been dry cured under specified conditions for Parma type hams (minimum curing 313 days), Iberian type hams, loins or shoulders, or Serrano type hams[71] (minimum curing 252 days), combined with certification that the pigs had been sourced from premises which had been free from evidence (clinical, serological, virological) of CSF infection for the past 3 months (the disease must be notifiable).

Rinderpest virus

The international standard (OIE Terrestrial Animal Health Code) recommends that consideration be given to the risk of rinderpest when importing pig meat (Office international des épizooties, 2003d). *Inter alia*, the measures which the OIE recommends for the importation of fresh pig meat include the following:

- a requirement that the meat came from animals which have been kept in a rinderpest free country or zone since birth or for at least 3 months prior to slaughter;
- pig meat may be imported from an infected country or zone if vaccination is carried out, the animals were vaccinated within 3 months, and there has been no rinderpest within 10 km of

[71] The processes used have been published in summarised form (McKercher, et al., 1985; Mebus, et al., 1993a) and full details are available through the bodies responsible for control of the production and certification of the product such as the Consorzio del Prosciutto di Parma, Fundacion del Jamon Serrano; Consorcio del J.Serrano.

the premises of origin during the past 30 days, and the meat is from deboned carcasses from which the major lymphatic glands have been removed.

Other measures that could also be considered to reduce the likelihood of entry, establishment and/or spread of rinderpest virus via imported pig meat may include a requirement that the meat of the pigs had been treated in such a way as to kill rinderpest virus.

Options

Country or zone freedom

Country or zone freedom from rinderpest to the satisfaction of Australian authorities would conform with Australia's ALOP (as discussed in the introduction to this Chapter, such approaches will be dealt with using existing mechanisms).

Heat processing to inactivate rinderpest virus

Canning - shelf stable

Australia currently accepts shelf stable canned pig meat from any source country, subject to certain conditions. The Australian import conditions for canned meat include a requirement that all portions of the contents have been heated to at least 100°C. The OIE reports that small amounts of virus resist heating at 56°C for 60 minutes or 60°C for 30 minutes. The Panel considered that rinderpest virus would be inactivated in canned pig meat heated to at least 100°C.

Pasteurised canned hams or cooked pig meat

Heat processing pig meat from a rinderpest infected country or zone by canning and pasteurisation (i.e. to at least 69°C) or cooking would influence the additional likelihood step in the release pathway (R7). This step describes the likelihood that rinderpest virus would survive in pasteurised canned hams or cooked pig meat. Heat processing may also affect the exposure step L2, which describes the likelihood that a waste unit would contain a sufficient dose of rinderpest virus to initiate infection.

There would appear to be limited studies undertaken on the effects of heat treatment on rinderpest virus. The Panel were unable to find any studies examining the survival of rinderpest virus in pasteurised canned hams or cooked pig meat. Hence, the Panel considered that further information was required in order to assess the likelihood of survival of rinderpest virus in such products.

Conclusions

There are two alternative options for management of the risk posed by rinderpest virus, each of which would meet Australia's ALOP:

- the pigs from which the meat is derived have been kept since birth in a country or zone which is free of rinderpest to the satisfaction of Australian authorities; or
- the pig meat has been canned such that all portions of the can contents have been heated to at least 100°C.

Swine vesicular disease virus

The international standard (OIE Terrestrial Animal Health Code) recommends that consideration be given to the risk of swine vesicular disease (SVD) when importing pig meat (Office international des épizooties, 2003e). The relevant measures which the OIE recommends for the importation of fresh pig meat can be summarised as follows:

- a requirement that the meat comes from animals which have been kept in an SVD free country or zone since birth or for at least the past 28 days and which have been slaughtered in an approved abattoir and have been subjected to ante-mortem and post-mortem inspections for SVD with favourable results;
- pig meat may be imported from an infected country or zone if the pigs have not been kept in an SVD infected zone, have been slaughtered at an approved abattoir not situated in an SVD infected zone and have been subjected to ante-mortem and post-mortem inspections for SVD with favourable results;
- pig meat products may also be imported from an infected country or zone if they have been processed to ensure the destruction of the SVD virus.

The OIE Code does not specify any particular process for destruction of SVD virus.

Other measures that could also be considered to reduce the likelihood of entry, establishment and/or spread of SVD virus via imported pig meat may include a requirement that the pigs from which the meat was derived were sourced from premises on which there had been no evidence of SVD within the 6 months prior to slaughter.

Options

Country or zone freedom

Country or zone freedom from SVD to the satisfaction of Australian authorities would conform with Australia's ALOP (as discussed in the introduction to this Chapter, such approaches will be dealt with using existing mechanisms).

Heat processing to inactivate SVD virus

Canning - shelf stable

One study demonstrated that SVD virus was inactivated in meat at temperatures above 69°C (McKercher, et al., 1980). Australia currently accepts shelf stable canned pig meat from any source country, subject to certain conditions. The Australian import conditions for canned meat include a requirement that all portions of the contents have been heated to at least 100°C. The Panel considered that SVD virus would be inactivated in canned pig meat heated to at least 100°C.

Pasteurised canned hams or cooked pig meat

Heat processing pig meat from a SVD infected country or zone by canning and pasteurisation (i.e. to at least 69°C) or cooking would influence the additional likelihood step in the release pathway (R7). This step describes the likelihood that SVD virus would survive in pasteurised canned hams or cooked pig meat. Heat processing may also affect the exposure step L2, which

describes the likelihood that a waste unit would contain a sufficient dose of SVD virus to initiate infection.

No virus was detectable in pasteurised canned hams prepared using meat from SVD infected pigs (McKercher, et al., 1974). The animals from which these hams were derived were infected with SVD and slaughtered at 48 or 72 hrs post-inoculation by which time they were showing severe clinical signs of SVD. The titre of virus in the meat varied from 10^3 to $10^{4.5}$ $TCID_{50}$ per gram. The canning process involved heating the products up to an internal temperature of 69°C over a 5 hour period. Detection methods involved virus isolation and bioassay by way of feeding of naive pigs. The paper does not provide full details of the method, numbers of animals or results.

The effect of heating on SVD virus was also examined in tissues that were not canned. Lymph node, blood clot and bone marrow from two animals experimentally inoculated with SVD virus were heated to an internal temperature of 69°C for 0 minutes. Swine vesicular disease virus could not be isolated from lymph node, bone marrow or blood clot when tested by intravenous inoculation into each of two naive pigs (McKercher, et al., 1980).

Given the limited number of studies conducted under experimental conditions on inactivation of SVD virus in pasteurised canned hams or cooked pig meat, and the small number of pigs involved, the Panel considered that there would be a 'low' likelihood that SVD virus would survive in pasteurised canned hams or cooked pig meat heated to a minimum internal temperature of 69°C.

In considering importation of pasteurised canned hams or cooked pig meat the likelihood assigned to L2 may also affected. Historically, outbreaks of SVD are associated with feeding of contaminated meat or meat products in swill. Pigs may be infected by contact with, or ingestion of, meat or meat products derived from pigs infected with SVD virus. In one experimental study, some pigs became infected when fed as little as 2 ounces (56.7 g) of infected meat in which the viral titres were between 10^3 and $10^{4.5}$ pfu/g (McKercher, et al., 1974). The Panel considered that the virus titre in pig meat would be reduced following canning and pasteurisation or cooking to 69°C. Given this, it was considered that the likelihood assigned to L2 could be reduced from 'high' to 'low'.

Curing to inactivate SVD virus

The curing of pig meat sourced from a SVD infected country or zone would influence the additional likelihood step in the release pathway (R7). This step describes the likelihood that SVD virus would survive in cured pig meat products. Curing may also affect the exposure step L2, which describes the likelihood that a waste unit would contain a sufficient dose of SVD virus to initiate infection.

There have been several studies on the effects of various methods of curing on the persistence of SVD virus.

Pepperoni and salami sausages

Swine vesicular disease virus survived in dried pepperoni and salami sausages for at least 200 days throughout the processing period (McKercher, et al., 1974). Similarly, other workers have shown the prolonged persistence of SVD virus in artificially-contaminated salami sausages for at least 42 days (Frescura, et al., 1976).

The Panel concluded that the normal "traditional" curing time of the order of 2 to 4 weeks would not reduce the likelihood of entry of SVD virus in these types of product.

Parma, Iberian and Serrano hams

The survival of SVD virus in dry cured Parma hams has been studied in experiments in the United States of America and in Italy using 24 experimentally inoculated pigs slaughtered 4 days post-inoculation (McKercher, et al., 1985). The viral titre at 3 days post-slaughter in this experiment ranged from 10^{1} pfu/g in fat to $10^{4.6}$ pfu/g in muscle. In the American study, SVD virus was detected in pooled samples of muscle, fat and bone marrow at 180 days curing but not at 300 and 360 days. In the Italian study, SVD virus was detected at 90 days curing but not at 182 and 310 days. The means of detection of SVD virus consisted of culture on IB-RS-2 cells and bioassay of a pooled sample by intravenous inoculation in naive pigs. It should be noted that the American study detected virus in muscle, bone marrow and fat at 3 days post-slaughter whereas in the Italian study virus was only detected in fat. This may explain the longer period required for inactivation in the American study. The authors of the study hypothesised that the difference may have been due to the different method of slaughter; the American pigs were anaesthetised and not bled whereas the Italian pigs were stunned by a captive-bolt stunner prior to bleeding.

The Panel considered that there would be a 'very low' likelihood that SVD virus would survive in dry cured Parma type hams according to the processes specified in the study above when cured for a minimum of 360 days.

The survival of SVD virus in Serrano hams and Iberian hams, loins and shoulders was studied in typical Spanish dry cured products (Mebus, et al., 1993b). Meat used in this experiment originated from experimentally inoculated pigs, 32 black Iberian pigs and 32 Spanish white pigs, slaughtered at 3 days post-inoculation based on knowledge of viral titre. Detection of virus at various times of curing was done by culture on IB-RS-2 cells and bioassay in naive pigs using a pool from the first three culture negative samples. At slaughter SVD virus titres varied from an average of $10^{0.1}$ pfu/g in fat to $10^{6.7}$ pfu/g in lymph node. In muscle, virus titres were very low averaging $10^{0.2}$ and $10^{0.3}$ pfu/g for Iberian black pigs and Spanish white pigs respectively. In many instances virus was not detected in muscle. In the muscle, fat and bone marrow of these products, SVD virus became undetectable after 84 days of curing on cell culture. In lymph node, however, the virus persisted for at least 470 days of curing in Iberian ham and Serrano ham. Samples from Iberian ham and Serrano ham collected on days 560, 574 and 589 of curing and days 539, 560 and 574 respectively were negative as determined by inoculation of naive pigs. Samples from Iberian shoulder and Iberian loin collected on days 196, 224 and 238 of curing and days 42, 56 and 70 respectively were negative as determined by inoculation of naive pigs. The paper reported commercial curing times for these products as follows:

- Iberian ham 365-730 days
- Serrano ham 180-365 days
- Iberian shoulder 240-420 days
- Iberian loin 90-130 days

The Panel considered that curing of dry cured Serrano type hams and Iberian type hams, loins and shoulders according to the processes specified, would reduce the titre of SVD virus in the product. However, due to the very low virus titres at slaughter in muscle and bone marrow, the extreme persistence of the virus in lymph node, and the wide variation in the time of

persistence of the virus in different products, the Panel considered that further information was required in order to assess the likelihood of survival of SVD in such cured hams.

Premises of origin free of SVD for 6 months pre and post-slaughter

A requirement that the herd from which pigs had been selected had been tested free from SVD within the 6 months prior to slaughter and within the 6 months following slaughter would influence the first step in the release pathway (R1). This step describes the likelihood of selecting slaughter-age pigs from an infected herd. The disease would need to be notifiable.

The period of 6 months was chosen by taking into account the likely epidemiological picture of SVD infection within a herd. New cases originating from within the herd would generally be expected to occur and be detected within 6 months of the last case. Clinical signs can be very mild making detection of the disease difficult, hence serological testing of the herd of origin pre and post-slaughter is required. The 2002 outbreak of SVD in Italy involved subclinical infection in all but one of 10,312 pigs (Brocchio, et al, 2002).

If pigs were sourced from herds tested serologically negative (virus neutralisation, ELISA) from SVD within the 6 months prior to slaughter and within the 6 months following slaughter, the likelihood assigned to R1 could be reduced from ‘moderate’ to ‘very low’.

Conclusions

The ‘restricted risk’ step estimates were as follows:

- the likelihood that SVD virus would survive in pasteurised canned hams or cooked pig meat heated to a minimum internal temperature of 69°C, was estimated to be ‘low’; and
    - the likelihood that a waste unit (pasteurised ham or cooked product) from an infected pig would contain a sufficient dose of SVD virus to initiate infection was estimated to be ‘low’;
- the likelihood that SVD virus would survive in cured Parma type hams was estimated to be ‘very low’; and
    - the likelihood that a waste unit (cured Parma type ham) from an infected pig would contain a sufficient dose of SVD virus to initiate infection was estimated to be ‘low’;
- the likelihood of selecting slaughter-age pigs from a herd tested serologically negative from SVD infection within the 6 months prior to slaughter and within the 6 months following slaughter was estimated to be ‘very low’.

Risk was estimated using the various combinations of mitigation measures discussed above and the results are summarised in Table 119. Herd freedom, pasteurisation, or cooking at 69°C either alone, or in combination, would not reduce the likelihood of entry, establishment and/or spread to an acceptable level.

Table 119 Risk management measures for SVD virus

R1 Herd freedom	R7 Pasteurised canned hams or cooked pig meat	R7 Cured Parma ham	Restricted risk
-	-	-	Moderate
+	-	-	Moderate
-	+	-	Moderate
+	+	-	Low
-	-	+	Moderate
+	-	+	Very low

- measure/s not applied

+ measure/s applied

Thus, there are several alternative options for management of the risk posed by SVD, each of which would meet Australia's ALOP:

- a requirement that the pigs from which the meat has been derived have been kept since birth in a country or zone which is free of SVD to the satisfaction of Australian authorities; or
- a requirement that the pig meat has been canned such that all portions of the can contents have been heated to at least 100°C; or
- a requirement that the pig meat has been dry cured under specified conditions for Parma type hams[72] (minimum curing 360 days), combined with certification that the pigs had been sourced from herds which had been tested negative for SVD within the 6 months prior to slaughter and within the 6 months following slaughter (the disease must be notifiable).

The Department of Health and Ageing has advised Biosecurity Australia that biosecurity measures for SVD virus would not be required to manage the risk to human life or health associated with the importation of pig meat.

Aujeszky's disease virus

The International standard (OIE Terrestrial Animal Health Code) recommends that consideration be given to the risk of Aujeszky's disease when importing "offal (head, and thoracic and abdominal viscera) of swine and products containing swine offal" (Office international des épizooties, 2003f). The relevant measures which the OIE recommends for the importation of offal from Aujeszky's disease infected countries can be summarised as follows:

- the products have been processed to ensure the destruction of the Aujeszky's disease virus.

The OIE Code does not specify any particular process for destruction of Aujeszky's disease virus.

[72] The processes used have been published in summarised form (McKercher, et al., 1985; Mebus, et al., 1993a) and full details are available through the bodies responsible for control of the production and certification of the product such as the Consorzio del Prosciutto di Parma.

In this IRA the definition of pig meat is limited to porcine muscle tissue, blood confined to muscle vasculature, bone and bone marrow, and any other tissues (for example, lymph nodes, skin, nerves) that may be considered inseparable from muscle. As such, pig meat products derived from offal, blood, bone or neurological tissue (such as brain, spinal cord) are not considered.

Measures that could be considered to reduce the likelihood of entry, establishment and/or spread of Aujeszky's disease virus via imported pig meat may include:

- a requirement that the pigs of origin had never been in an Aujeszky's disease infected country or zone since birth;
- a requirement that the pig carcass had been dressed in such a way as to remove high risk tissues at slaughter;
- reduction in the volume of pig meat waste discarded in Australia.

Options

Country or zone freedom

Country or zone freedom from Aujeszky's disease to the satisfaction of Australian authorities would conform with Australia's ALOP (as discussed in the introduction to this Chapter, such approaches will be dealt with using existing mechanisms).

Modified dressing of the carcass

Modified dressing of the carcass to remove certain tissues where the virus has an affinity would influence the fourth step in the release pathway (R4). This step describes the likelihood that the pathogenic agent would be present in meat harvested for export. Modified dressing of the carcass may also influence the exposure step L2, which describes the likelihood that a waste unit would contain a sufficient dose of Aujeszky's disease virus to initiate infection.

Aujeszky's disease virus has an affinity for certain tissues in the head region. Aujeszky's disease virus has been detected, with difficulty, in muscle tissues in very low titres from pigs slaughtered at peak pyrexia. Viral fragments have also been detected on occasions in bone marrow. As noted in the risk assessment chapter on Aujeszky's disease, the trigeminal nerve ganglia are a major site of latent Aujeszky's disease virus which has also been shown to persist in tonsils and olfactory bulbs. Based on this information the Panel considered the risk associated with a carcass on which the head and neck were retained as compared with one where the head and neck had been removed.

The Panel considered that if meat was derived from a carcass where the head and neck had been removed the likelihood assigned to R4 could be reduced from 'moderate' to 'low'.

Moreover, if meat is sourced from areas other than the head and neck, the amount of virus present in a waste unit may be reduced. As stated in the risk assessment, the likelihood that a waste unit would contain a sufficient dose of Aujeszky's disease virus to initiate infection (L2), given that it was derived from an infected pig, was based on the source of the waste unit (head and neck region or the rest of the carcass). A 'moderate' likelihood was assigned to a waste unit derived from the head and neck and a 'very low' likelihood assigned for waste unit derived from the rest of the body. Hence, if the head and neck were removed from the carcass the likelihood assigned to L2 would be 'very low'.

Reduction in the volume of waste discarded

If product was deboned and cooked or cured the volume of waste discarded by households and food service establishments would be significantly reduced. Smallgoods are generally bought in smaller quantities by households, and there is very little waste from these products. Deboning alone may not reduce the volume of waste significantly as it was considered that a significant portion of waste discarded would be uncooked meat that has spoiled. Cooked bone-in product alone also may not reduce the volume of waste significantly, as a significant portion of waste is bone. On balance, the Panel considered that if meat was deboned and processed either by cooking or curing the proportion of pig meat purchased by households and food service establishments that was discarded as waste would be reduced to one tenth of that estimated for the unrestricted risk (see Methods section, Table 4).

Conclusions

The 'restricted risk' steps estimates were as follows:

- the likelihood that Aujeszky's disease virus would be present in meat excluding the head and neck harvested for export was estimated to be 'low'; and
  - the likelihood that a waste unit (excluding the head and neck) from an infected pig would contain a sufficient dose of Aujeszky's disease virus to initiate infection was estimated to be 'very low';
- if the product was processed by cooking or curing and deboned, the proportion of waste discarded was estimated to be one tenth of that estimated for the unrestricted risk.

Risk was estimated using the mitigation measure discussed above and the results are summarised in Table 120. Modified dressing of the carcass i.e. removing the head and neck or deboning and processing of pig meat by cooking or curing would reduce the risk of entry, establishment and/or spread to very low or negligible respectively, which would meet Australia's ALOP.

Table 120 Risk management measures for Aujeszky's disease virus

R4	Reduction in the proportion of waste discarded	Restricted risk
Head and neck off	Cooked or cured product and deboned	
-	-	Low
+	-	Very low
-	+	Negligible
+	+	Negligible

- measure/s not applied

+ measure/s applied

Thus, there are three alternative options for management of the risk posed by Aujeszky's disease virus, each of which would meet Australia's ALOP:

- a requirement that the pigs from which the meat is derived have been kept since birth in a country or zone which is free of Aujeszky's disease to the satisfaction of Australian authorities; or
- a requirement that meat is not derived from the head or neck; or
- the meat has been deboned and the product has been cooked or cured.

Porcine reproductive and respiratory syndrome virus

Porcine reproductive and respiratory syndrome (PRRS) is an OIE List B disease, but there is no Code chapter for this disease and therefore no recommendations for risk management measures when importing pig meat.

The likelihood that PRRS virus could enter, become established and/or spread in Australia via imported pig meat could, in theory be reduced by the application of some or all of the following measures:

- a requirement that the pigs of origin had never resided in a PRRS infected country or zone since birth;
- a requirement that slaughter and processing ensured removal of organs and tissues which are sites of predilection for the virus;
- a requirement that the meat of the pigs had been treated in such a way as to inactivate PRRS virus.

Options

Country or zone freedom

Country or zone freedom from PRRS to the satisfaction of Australian authorities would conform with Australia's ALOP (as discussed in the introduction to this Chapter, such approaches will be dealt with using existing mechanisms). Since vaccinated pigs can shed PRRS virus, the Panel considered that to be considered free from PRRS, a country or zone should not permit vaccination.

Modified dressing of the carcass

Modified dressing of the carcass to remove certain tissues where the virus has an affinity would influence the fourth step in the release pathway (R4). This step describes the likelihood that the pathogenic agent would be present in meat harvested for export. Modified dressing of the carcass may also influence the exposure step L2, which describes the likelihood that a waste unit would contain a sufficient dose of PRRS virus to initiate infection.

The distribution of PRRS virus through the body and in the organs of slaughtered pigs has been the subject of several studies. PRRS virus has an affinity for lymphoid tissues and cells. The virus is present in significantly higher quantity in the lungs and lung lymph nodes, followed by the tonsils and spleen, than in other tissues (Yoon, et al., 1998). In persistently infected pigs the virus is isolated from the tonsils. It was considered that lymph nodes draining the pharynx were also likely to contain virus.

Very few studies have reported examination of bone marrow for PRRS virus. The virus could be isolated in low numbers from a pool of leg muscle and bone marrow (from the femur) for at least up to 4 weeks post slaughter when stored at 4°C (Frey, et al., 1995a; Frey, et al., 1995b).

In contrast, the virus was not isolated from the bone marrow of experimentally infected pigs (Bloemraad, et al., 1994; Duan, et al., 1997). As PRRS virus has an affinity for lymphoid cells it was considered that these were as likely to be in bone marrow as in blood perfusing muscle.

Based on this information, the Panel examined the effect of removing the head and neck and/or deboning on the above likelihoods. The Panel considered that removal of the head and neck, including any remaining tonsillar tissue, and lymph nodes draining the pharynx could reduce the likelihood assigned to R4 from 'moderate' to 'low'. It was considered that deboning of the carcass by itself would not significantly reduce the likelihood assigned to R4. Removal of the head and neck and deboning could reduce the likelihood assigned to R4 from 'moderate' to 'low'.

Moreover, if meat is sourced from areas other than the head and neck and/or bone is removed, the amount of virus present in a waste unit may be reduced. The minimum oral infectious dose of PRRS virus is unknown, however, it is known that 500 grams of muscle derived from a pig recently infected with PRRS virus provided a sufficient oral dose to infect a naïve pig. In this experiment, meat derived from some of the pigs contained virus at titres less than the limit of the detection of the assay ($10^{1.8}TCID_{50}$/gram tissue) (van der Linden, et al., 2003). One study reported that as few as 10 virions by intranasal inoculation were sufficient to achieve infection (Yoon, et al., 1998), indicating that the disease is highly infectious, at least by that route.

The Panel considered that the likelihood assigned to L2 could be reduced from 'high' to 'moderate' if meat was derived from areas other than the head or neck and/or if the carcass was deboned.

Heat processing to inactivate PRRS virus

Canning - shelf stable

Australia currently accepts shelf stable canned pig meat from any source country subject to certain conditions. The Australian import conditions for canned meat include a requirement that all portions of the contents have been heated to at least 100°C. Studies indicate that PRRS virus is a labile virus, being inactivated at 56°C for 60 minutes. The Panel considered that PRRS virus would be inactivated in canned pig meat heated to at least 100°C.

Pasteurised canned hams or cooked pig meat

Heat processing pig meat from a PRRS infected country or zone by canning and pasteurisation or cooking would influence the additional likelihood step in the release pathway (R7). This step describes the likelihood that PRRS virus would survive in pasteurised canned hams or cooked pig meat. Heat processing may also affect the exposure step L2, which describes the likelihood that a waste unit would contain a sufficient dose of PRRS virus to initiate infection.

Several studies have examined the effect of heating on PRRS virus. Inactivation at 56°C for 60 minutes decreased the titre of Lelystad virus by 3 log (Bloemraad, et al., 1994). Another study reported that PRRS virus was inactivated after heating at 56°C for 45 minutes (Benfield, et al., 1992). More recently, Biosecurity Australia has commissioned studies into the thermostability of PRRS virus. Preliminary results concur with those of Bloemraad and co-workers indicating that the virus is quite labile. The virus was undetectable at about 56°C for 60 minutes.

The currently approved heat treatments which Australian authorities require to manage the risk of PRRS virus in imported deboned pig meat are as follows:

56°C for 60 minutes or
57°C for 55 minutes or
58°C for 50 minutes or
59°C for 45 minutes or
60°C for 40 minutes or
61°C for 35 minutes or
62°C for 30 minutes or
63°C for 25 minutes or
64°C for 22 minutes or
65°C for 20 minutes or
66°C for 17 minutes or
67°C for 15 minutes or
68°C for 13 minutes or
69°C for 12 minutes or
70°C for 11 minutes

It is recognised that bone is a poorer conductor of heat than muscle. In requesting access for bone-in pig meat, the Danish Government recommended adjusting for a 4°C lower core temperature of bone marrow to take account of this. It is also known that some viruses are protected by bone marrow from the effects of heat (Blackwell, 1984), although no information is available on PRRS virus.

Given this, the Panel considered bone-in pig meat would need to be cooked for a longer period of time such that a core (bone) temperature of 70°C for 11 minutes or equivalent was reached. It was considered that there would be a 'very low' likelihood that PRRS virus would survive in bone-in or bone-out pig meat cooked to a core temperature of 70°C for 11 minutes.

In considering importation of cooked pig meat the likelihood assigned to L2 may also be affected. Although the minimum oral infectious dose is unknown, it is known that PRRS virus is very infectious at least intranasally. Overall, the Panel considered that the virus titre in pig meat would be reduced following cooking. Given this, it was considered that the likelihood assigned to L2 could be reduced from 'high' to 'low'.

Curing to inactivate PRRS virus

The curing of pig meat sourced from a PRRS infected country or zone would influence the additional likelihood step in the release pathway (R7). This step describes the likelihood that PRRS virus would survive in cured pig meat products. Curing may also affect the exposure step L2, which describes the likelihood that a waste unit would contain a sufficient dose of PRRS virus to initiate infection.

PRRS virus is slowly inactivated at temperatures above freezing. For instance, 72 hours after necropsy, the virus (US strain) could be isolated from 12/15 tissue samples (lung, spleen and thymus) and 5/5 serum samples which had been held at 4°C and from 1/15 tissue samples and 4/5 serum samples which had been held at 25°C. All these samples had yielded virus at necropsy. A calculation of the half life of Lelystad virus in culture medium at pH 7.5 (Bloemraad, et al., 1994) was as follows:

Temperature°C	Half life
4	139 hrs
21	20 hrs
37	3 hrs
56	6 minutes

In this experiment, a loss of 5 log of the initial titre was observed in culture medium at 4°C over 13 weeks. The maximal stability of the virus at 4°C was at pH 6.25.

Given that the minimum curing times stipulated in the section on CSF and ASF for Parma hams is 313 days and Iberian loins, shoulders and hams and Serrano hams is 140 days respectively, the Panel considered that there would be a 'very low' likelihood that PRRS virus would survive in these dry cured hams (bone-in or bone-out).

In considering importation of cured pig meat the likelihood assigned to L2 may also affected. Although the minimum oral infectious dose is unknown, it is known that PRRS virus is very infectious at least intranasally. Overall, the Panel considered that the virus titre in pig meat would be reduced following curing. Given this, it was considered that the likelihood assigned to L2 could be reduced from 'high' to 'low'.

Conclusions

The 'restricted risk' estimate steps were as follows:

- the likelihood that PRRS virus would be present in meat excluding the head and neck harvested for export was estimated to be 'low'; and
  - the likelihood that a waste unit (excluding the head and neck) from an infected pig would contain a sufficient dose of PRRS virus to initiate infection was estimated to 'moderate';
- the likelihood that PRRS virus would be present in meat excluding bone harvested for export was estimated to be 'moderate'; and
  - the likelihood that a waste unit (excluding bone) from an infected pig would contain a sufficient dose of PRRS virus to initiate infection was estimated to 'moderate';
- the likelihood that PRRS virus would be present in meat excluding the head, neck and bone harvested for export was estimated to be 'low'; and
  - the likelihood that a waste unit (excluding the head, neck and bone) from an infected pig would contain a sufficient dose of PRRS virus to initiate infection was estimated to 'moderate';
- the likelihood that PRRS virus would survive in bone-in or bone-out pig meat heated to a minimum internal temperature of 70°C for at least 11 minutes respectively was estimated to be 'very low'; and
  - the likelihood that a waste unit (cooked product) from an infected pig would contain a sufficient dose of PRRS virus to initiate infection was estimated to be 'low';
- the likelihood that PRRS virus would survive in cured (Parma, Iberian and Serrano) bone-in or bone-out hams was estimated to be 'very low'; and

- the likelihood that a waste unit (cured product) from an infected pig would contain a sufficient dose of PRRS virus to initiate infection was estimated to be 'low';

Risk was estimated using the various combinations of mitigation measures discussed above and the results are summarised in Table 121. Cooking or curing of bone-in or bone-out pig meat would reduce the risk of entry, establishment and/or spread to very low which would meet Australia's ALOP. Other measures (head and neck off) combined with cooking or curing at specified times/temperatures would also reduce the risk to an acceptable level. Modified dressing of the carcass (head or neck off and/or bone-out) alone would not reduce the likelihood of entry, establishment and/or spread to an acceptable level.

Table 121 Risk management measures for PRRS virus

R4 Head and neck off	**R4** Bone-out	**R7** Cooked (bone-in or bone-out)	**R7** Cured (bone-in or bone-out)	**Restricted risk**
-	-	-	-	Low
+	-	-	-	Low
-	+	-	-	Low
-	-	+	-	Very low
-	-	-	+	Very low
+	+	-	-	Low
+	-	+	-	Negligible
+	-	-	+	Negligible

- measure/s not applied

+ measure/s applied

Thus, there are thus several alternative options for management of the risk posed by PRRS virus, each of which would meet Australia's ALOP:

- a requirement that the pigs from which the meat is derived have been kept since birth in a country or zone which is free of PRRS and where vaccination is not permitted to the satisfaction of Australian authorities; or
- a requirement that the pig meat had been processed by canning such that all portions of the contents have been heated to at least 100°C; or
- a requirement that the pig meat had been dry cured under specified condition for Parma type hams (minimum curing 313 days), Iberian type hams, loins or shoulders or Serrano type hams (minimum curing 140 days); or
- a requirement that pig meat be heat processed such that all parts reach a temperature of at least 70°C for 11 minutes.

It is recommended that pig meat be processed off-shore or on-shore subject to processing (cooking/curing) in an urban area at the port of entry or in a rural area subject to appropriate security transport arrangements (eg. refrigerated container), under a compliance agreement with the Australian Quarantine and Inspection Service (AQIS). The Panel recognised that there may be a slight increase in risk with processing imported product on-shore in Australia, however, it

is considered that this is offset by the added confidence provided by AQIS audit of processors ensuring compliance with the conditions. It is considered that the assurance of AQIS audits of processing plants and compliance agreement arrangements covering the storage, transport and processing of imported product and management of waste provides an equivalent level of quarantine protection to that provided by processing off-shore. The Panel also noted the history of safely processing imported pig meat in Australia under quarantine control for the previous 11 years.

Biosecurity Australia has currently commissioned research into the thermostability of PRRS virus. Depending on the results of the study the time/temperature combinations specified for processing of pig meat from PRRS infected countries may change.

Trichinellosis (*Trichinella spiralis*)

The international standard (OIE Terrestrial Animal Health Code) recommends that consideration be given to the risk of *Trichinella spiralis* when importing pig meat (Office international des épizooties, 2003g). The relevant measures which the OIE recommends for the importation of fresh pig meat can be summarised as follows:

- a requirement that the meat comes from domestic swine which were born and bred in a country or zone free from trichinellosis in domestic swine; or
- a requirement that the pigs were subjected to a testing procedure for trichinellosis with negative results; or
- a requirement that the pig meat was processed to ensure the destruction of all the larvae of the parasite.

The international standard (OIE Terrestrial Animal Health Code) states that a country or zone may be considered free from trichinellosis in domestic swine when:

- trichinellosis is notifiable in the country; and there is in force an effective disease reporting system shown to be capable of capturing the occurrence of cases; AND EITHER:
  - it has been ascertained that Trichinella infection does not exist in the domestic swine population of the country or zone under consideration; this is established by the regular surveillance of the swine population using an approved testing procedure, which provided negative results when:

    a) within a 5-year period, a serological survey was conducted on a statistically based sample size from within the slaughter sow population sufficient to provide at least 95% confidence of detecting trichinellosis if it was present at a prevalence exceeding 0.02%, and during this 5-year period, continuous testing was conducted on a statistically based sample size from within the annual slaughter swine population sufficient to provide at least 95% confidence of detecting trichinellosis if it is present at a prevalence exceeding 0.01%, following which:

    b) a serological survey is carried out every third year on the slaughter sow population sufficient to provide at least 95% confidence of detecting trichinellosis if it is present at a prevalence exceeding 0.2%; during this time the number of samples in the slaughter swine population could be reduced to detect at the 0.5% level on an annual basis;

    OR
  - in the country or zone under consideration, the following conditions are met: Trichinellosis has not been reported in the domestic swine population for at least 5

years; wild susceptible species are subjected to a regular surveillance programme, and no clinical, serological or epidemiological evidence of trichinellosis has been found; the regular surveillance described in point b) above is carried out and should be concentrated where infestation was last identified, and/or where the feeding of swill to swine occurs; any suspicion of disease is followed at the field level by traceback, quarantine and laboratory testing; if trichinellosis is confirmed, the infected premises remains under official veterinary control and is subjected to disease control measures using a stamping-out policy and rodent control; all feeding of swill is officially regulated; and, any human outbreaks of trichinellosis are investigated to determine the animal source.

The OIE Code does not specify any particular process for destruction of *Trichinella spiralis*. However the International Commission on Trichinellosis has published methods involving cooking or freezing (ICT Standards for Control Guidelines Committee, 2000).

Options

Country or zone freedom in domestic pigs

The ongoing and extensive surveillance requirements for freedom from trichinellosis in domestic pigs according to the OIE Code provide a high level of assurance that domestic pigs are free and that should spill over from a wildlife reservoir occur this would be detected.

Country or zone freedom from *Trichinella spiralis* in domestic swine to the satisfaction of Australian authorities would conform with Australia's ALOP (as discussed in the introduction to this Chapter, such approaches will be dealt with using existing mechanisms).

Testing for *Trichinella spiralis*

Testing of pig meat for *Trichinella* larvae would influence the third step of the release pathway (R3.1). This step describes the sensitivity of ante-mortem, slaughter and processing requirements in detecting and removing infected pigs. Testing of pig meat may also affect the exposure step L2, which describes the likelihood that a waste unit would contain a sufficient dose of *Trichinella* larvae to initiate infection.

The OIE has published standard tests for *Trichinella* (Office international des épizooties, 2000). The OIE prescribed test for international trade is identification of the agent, either by trichinoscope or the digestion method. The digestion method has become the method of choice for routine slaughter inspection. The European Union recommends the use of digestion tests due to the enhanced sensitivity compared with trichinoscopy (Nockler, et al., 2000). Using 1 gram tissue samples the sensitivity of the digestion method is three larva per gram, whereas the sensitivity of the compression method is approximately five larvae per gram (Gamble, 1997a). Using a 5 gram sample the sensitivity of the digestion method is one larva per gram of tissue. An alternative method of testing pigs for *Trichinella* is the detection of antibodies to the parasites in serum using an ELISA. The ELISA is considered a very sensitive test for detecting infection in pigs (Gamble, 1997b). However, the ELISA may fail to detect infected pigs during both the early and very late stages of infection (Nockler, et al., 2000). In abattoir testing the ELISA has been reported as nearly 100% sensitive in detecting infected pigs with more than one larva per gram of tissue (Office international des épizooties, 2000).

Given this, the Panel considered that testing every pig or carcass at slaughter with either an ELISA test for antibodies to *Trichinella* or by digestion would increase the sensitivity of ante-

mortem, slaughter and processing requirements in detecting and removing pigs infested with *Trichinella* from 'negligible' to lie between '95% and 98%'.

In considering pig meat that has been tested for *Trichinella* larvae, the likelihood assigned to L2 may also be affected. The digestion method and ELISA have been reported to be sensitive methods for detecting low numbers of larvae (Office international des épizooties, 2000). Hence infested pigs that are not detected are likely to have a very low number of larvae per gram of tissue. As such, pigs in Australia may need to consume a large volume of waste. Nonetheless experimentally, infection of pigs has resulted from a dose of 10 larvae (Haralabidis, et al., 1989). Given the likely very low number of larvae per gram of pig meat, the composition of pig meat waste and the volume of waste consumed by a pig, it was considered that the likelihood assigned to L2 could be reduced from 'high' to 'very low'.

The Panel considered that the annual likelihood of entry and exposure for other susceptible species such as rodents could be reduced if pig meat was tested for *Trichinella* larvae prior to export from 'high' to 'very low'.

Processing to inactivate *Trichinella spiralis*

Canning - shelf stable

Australia currently accepts shelf stable canned pig meat from any source country subject to certain conditions. The Australian import conditions for canned meat include a requirement that all portions of the contents have been heated to at least 100°C. Studies indicate that *Trichinella* larvae are killed in less than a minute at 60°C. The Panel considered that *Trichinella spiralis* would be inactivated in canned pig meat heated to at least 100°C.

Cooking or freezing

Processing of pig meat sourced from a country where *Trichinella spiralis* is present in the domestic swine population would influence the additional likelihood step in the release pathway (R7). This step describes the likelihood that *Trichinella* larvae would survive in cooked or frozen pig meat. Cooking or freezing of meat may also affect the exposure step L2, which describes the likelihood that a waste unit would contain a sufficient dose of *Trichinella* larvae to initiate infection.

Trichinella spiralis does not survive in meat at temperatures more than about 60°C (Gamble, 1997a). A review of *Trichinella spiralis* quoted the cooking times required to kill the parasite as 47 minutes at 52°C, 6 minutes at 55°C and less than a minute at 60°C (Gamble, 1997a). These times and temperatures only apply when the product reaches and maintains temperatures evenly distributed throughout the meat. The following treatments, recommended by the International Commission on Trichinellosis and prescribed by the United States Department of Agriculture (USDA) to destroy *Trichinella spiralis*, reflect these data (Table 122).

Table 122 Inactivation of *Trichinella spiralis* in pig meat by heating

Core temperature (°C)	Minimum time (minutes)
50.0	570
51.1	270
52.2	120
53.4	60
54.5	30
55.6	15
56.7	6
57.8	3
58.9	2
60.0	1
61.1	1
62.2	Instant

The Panel considered that there would be a 'negligible' likelihood that *Trichinella* larvae would survive in cooked pig meat (heated as specified in Table 122).

Freezing of meat is known to kill *Trichinella spiralis* larvae. The following controlled freezing times are recommended by the International Commission on Trichinellosis and prescribed by the USDA to destroy *Trichinella spiralis* (Table 123).

Table 123 Inactivation of *Trichinella spiralis* in pig meat by freezing meat to a specified core temperature

Core temperature (°C)	Minimum time (hours)
-17.8	106
-20.6	82
-23.3	63
-26.1	48
-28.9	35
-31.7	22
-34.5	8
-37.2	0.5

Other requirements for inactivation of *Trichinella spiralis* by freezing have been based on the air temperature of the freezer and the thickness of the pieces/packages. On this basis, the following freezing times are recommended by the International Commission on Trichinellosis and prescribed by the USDA to destroy *Trichinella spiralis* (Table 124).

Table 124 Inactivation of *Trichinella spiralis* in pig meat by freezing - freezer temperature

Freezer temperature (°C)	Group 1 (<= 15 cm thickness) – days	Group 2 (> 15 but <= 68 cm thickness) - days
-15.0	20	30
-23.3	10	20
-28.9	6	12

Several countries have requirements that are broadly consistent with these figures. Current Australian requirements for crocodile meat are for -15°C for 20 days. Canadian requirements to inactivate *Trichinella* are for -25°C for 10 or 20 days (depending on the thickness of meat) (Canadian Food Inspection Agency, 2003), and the European Commission's requirements (Directive 77/96/EEC of 21 December 1976) are for -25°C for 10 or 20 days the former for meat thickness up to 25 cm and the latter up to 50 cm.

The Panel considered that there would be a 'negligible' likelihood that *Trichinella* larvae would survive in pig meat frozen in accordance with the conditions shown in the above tables (Table 123, Table 124).

In considering pig meat that has been cooked or frozen to destroy *Trichinella* larvae, the likelihood assigned to L2 could be reduced from 'high' to 'negligible'.

The Panel considered that the annual likelihood of entry and exposure for other susceptible species such as rodents could be reduced if pig meat was cooked or frozen to destroy *Trichinella* larvae from 'high' to 'negligible'.

Curing

Curing of pig meat sourced from a country where *Trichinella spiralis* is present in the domestic swine population would influence the additional likelihood step in the release pathway (R7). This step describes the likelihood that *Trichinella* larvae would survive in cured pig meat products. Curing of meat may also affect the exposure step L2, which describes the likelihood that a waste unit would contain a sufficient dose of *Trichinella* larvae to initiate infection.

The multitude of processes used to prepare cured pork products (salamis, hams, ready to eat products) complicates consideration of standard requirements for inactivation of *Trichinella* larvae (Gamble, 1997a). Hence, consideration will be given to the survival of larvae in dry cured Parma Iberian and Serrano type hams.

A study of the survival of *Trichinella spiralis* in dry cured pork products showed that the parasite was relatively quickly inactivated (Smith, et al., 1989). The muscle of 15 experimentally infected pigs carried 38 to 460 *Trichinella spiralis* larvae per gram with an average of 159 larvae per gram. In Genoa salami, which was made from the meat of these pigs

and cured in the presence of salt and sodium nitrate and nitrite, there was no survival in samples taken on eight occasions between 13 and 42 days post preparation. In wet cured "Proscuittini" cured in the presence of salt and sodium nitrate and nitrite, no live *Trichinella spiralis* were demonstrated from samples taken on seven occasions between days 27 and 69 post preparation. In "Proscuitto type I" which was dry cured in the presence of salt and sodium nitrate and nitrite, there was no survival in samples taken on six occasions between days 34 and 69 days post preparation. In "Proscuitto type II" which was dry cured in the presence of salt only (and not sodium nitrate or nitrite), there was a mean infection of 22.4 larvae per gram established for samples taken on day 34 post-preparation. No infection was demonstrated in 16 test rats fed material derived on days 55, 62 and 69 post-preparation.

In the above study, the concentration of salt used in salami preparation varied with levels of 2.0%, 2.75% and 3.3% being used without observable differences. However, other studies have shown that the concentration of salt was important in terms of larvicide activity. In one study, *Trichinella spiralis* larvae were completely destroyed at day 20 of curing in salami that had been subjected to high temperature fermentation with 3.33% NaCl, whereas in salami made with no salt viable larvae were found in 50% of the samples at day 5, 33% at day 10 and 25% at days 15 to 25 (Childers, et al., 1982). In another experiment, a 2.5% salt content was insufficient to kill larvae in smoked salted pork products but 5.5% killed the larvae in 72 days (Modic & Dovdevic, 1984).

The time estimated for destruction of larvae by curing at different temperatures and salt concentrations were reviewed (Kotula, 1983). At a curing temperature of 4°C it was estimated that 130 and 135 days were required for destruction of *Trichinella spiralis* larvae in hams and shoulders respectively. In another study, infective larvae were recovered from medium size cured hams (9.5 kg) held at 10°C for 90 days (Lin, et al., 1990). In cubes of pork containing 3.3, 2.5 or 2% salt infectivity was retained on average for 57, 85 and 107 days respectively (Kotula, 1983). It is for these reasons that the USDA requirements for minimum drying room times for processed sausages is modified such that at 2% salt content, the drying time must be extended by 40% beyond that for product with 3.3% salt content. These requirements stipulate a wide variety of conditions which are regarded as acceptable means of inactivating *Trichinella spiralis*.

Given that the minimum curing times stipulated in the section on CSF and ASF for Parma hams is 313 days and Iberian loins, shoulders and hams and Serrano hams is 140 days respectively, the Panel considered that there would be a 'negligible' likelihood that *Trichinella* larvae would survive in Parma hams and 'extremely low' likelihood for Iberian, loins, shoulders and hams and Serrano hams cured for 140 days.

However, given the shorter curing times used for many types of salami, the Panel concluded that these products would require individual assessments to confirm whether *Trichinella spiralis* would reliably be inactivated during the curing and fermentation process. Each process would need to be stipulated in terms of the concentrations of curing agents and salt, the holding times, curing times and temperatures, and the like. Experimental data demonstrating that the stated process inactivated *Trichinella spiralis* would likely be required.

In considering pig meat that has been dry cured for a minimum 313 days (Parma type ham), the likelihood assigned to L2 could be reduced from 'high' to 'negligible' and for pig meat that has been dry cured for a minimum of 140 days (Iberian and Serrano hams) from 'high' to 'extremely low'.

The Panel considered that the annual likelihood of entry and exposure for other susceptible species such as rodents would be reduced if pig meat was dry cured for 313 days (Parma type ham) to destroy *Trichinella* larvae from 'high' to 'negligible' and for pig meat dry cured for 140 days (Iberian and Serrano type hams) from 'high' to 'extremely low'.

Conclusions

The 'restricted risk' step estimates were as follows:

- the sensitivity of ante-mortem, slaughter and processing requirements if every carcass at slaughter was tested for *Trichinella* larvae by digestion or ELISA was estimated to lie between 95% and 98%; and
  - the likelihood that a waste unit (carcass tested negative) from an infected pig would contain a sufficient dose of *Trichinella* larvae to initiate infection was estimated to be 'very low'; and
  - the annual likelihood of entry and exposure for other susceptible species was estimated to be 'very low';
- the likelihood that *Trichinella* larvae would survive in cooked or frozen pig meat was estimated to be 'negligible'; and
  - the likelihood that a waste unit (cooked or frozen pig meat) from an infected pig would contain a sufficient dose of *Trichinella* larvae to initiate infection was estimated to be 'negligible'; and
  - the annual likelihood of entry and exposure for other susceptible species was estimated to be 'negligible';
- the likelihood that *Trichinella* larvae would survive in cured hams (Parma), was estimated to be 'negligible'; and
  - the likelihood that a waste unit (cured Parma ham) from an infected pig would contain a sufficient dose of *Trichinella* larvae to initiate infection was estimated to be 'negligible'; and
  - the annual likelihood of entry and exposure for other susceptible species was estimated to be 'negligible';
- the likelihood that *Trichinella* larvae would survive in cured hams (Iberian, Serrano) was estimated to be 'extremely low'; and
  - the likelihood that a waste unit (cured Iberian, Serrano ham) from an infected pig would contain a sufficient dose of *Trichinella* larvae to initiate infection was estimated to be 'extremely low'; and
  - the annual likelihood of entry and exposure for other susceptible species was estimated to be 'extremely low'.

Risk was estimated using the various combinations of mitigation measures discussed above and the results are summarised in Table 125. Testing each carcass for larvae, or processing of pig meat by cooking or freezing, or processing of pig meat by curing under specified conditions for Parma, Iberian and Serrano type hams would reduce the risk of entry, establishment and/or spread to very low or negligible, which would meet Australia's ALOP.

Table 125 Risk management measures for *Trichinella spiralis*

R3.1 Testing	R7 Cooking or freezing pig meat	R7 Cured Parma ham	R& Cured Iberian, Serrano ham	Restricted risk
-	-	-	-	Low
+	-	-	-	Very low
-	+	-	-	Negligible
-	-	+	-	Negligible
-	-	-	+	Negligible

- measure/s not applied

+ measure/s applied

Thus, there are several alternative options for management of the risk posed by *Trichinella spiralis*, each of which would meet Australia's ALOP:

- a requirement that the pigs from which the meat is derived have been kept since birth in a country or zone which is free of *Trichinella spiralis* to the satisfaction of Australian authorities; or
- a requirement that each of the pigs had been tested and found negative for the presence of *Trichinella spiralis* larvae by pepsin digestion or ELISA as described by the OIE (Office international des épizooties, 2000); or
- a requirement that the pig meat had been processed by canning such that all portions of the contents have been heated to at least 100°C; or
- a requirement that the pig meat had been cooked to one of the time/temperatures specified above (Table 122); or
- a requirement that the pig meat had been frozen at one of the time/temperatures specified above (Table 123, Table 124); or
- a requirement that the pig meat had been dry cured under specified condition for Parma type hams (minimum curing 313 days), Iberian type hams, loins, shoulders or Serrano type hams (minimum curing 140 days).

The Department of Health and Ageing has advised Biosecurity Australia that risk management measures would be required for *Trichinella spiralis* to address the risk to human health or life associated with the importation of pig meat. Appropriate measures would include testing, or herd or zone freedom, or processing to ensure destruction of larvae.

Nipah virus

Nipah virus is not listed by the OIE. As such the international standard (OIE Terrestrial Animal Health Code) does not include any recommendations in regard to the risk of Nipah virus disease when importing pig meat.

The likelihood that Nipah virus could enter, become established and/or spread in Australia via imported pig meat could, in theory be reduced by the application of some or all of the following measures:

- a requirement that the pigs of origin had never been in a Nipah virus infected country or zone since birth;
- a requirement that the pigs of origin had been kept since birth in a country or zone in which the domestic pig population is free from Nipah virus;
- a requirement that the meat of the pigs had been treated in such a way as to inactivate Nipah virus.

Options

Country or zone freedom

A requirement that the pigs have been kept since birth in a country or zone free from Nipah virus to the satisfaction of Australian authorities would conform with Australia's ALOP (as discussed in the introduction to this Chapter, such approaches will be dealt with using existing mechanisms).

Country or zone freedom in domestic pigs

A requirement that the domestic pig population was free from infection with Nipah virus as determined by surveillance which satisfied Australian authorities would conform with Australia's ALOP (as discussed in the introduction to this Chapter, such approaches will be dealt with using existing mechanisms).

Heat processing to inactivate Nipah virus

Canning - shelf stable

Australia currently accepts shelf stable canned pig meat from any source country subject to certain conditions. The Australian import conditions for canned meat include a requirement that all portions of the contents have been heated to at least 100°C. The Panel was unable to obtain data on the thermal inactivation of Nipah virus. However, an examination of data on closely related viruses may be of value. Nipah virus belongs to the genus Henipavirus, subfamily Paramyxovirinae, family Paramyxoviridae. This family includes several viruses that cause disease in animals i.e. Newcastle disease, rinderpest, canine distemper, none of which is particularly resistant to heat. Paramyxoviruses consist of a nucleocapsid surrounded by an envelope which is very fragile, rendering the virion vulnerable to destruction by storage or freezing (Fenner, et al. 1993). The Panel considered that Nipah virus would be inactivated in canned pig meat heated to at least 100°C.

Conclusions

There are several alternative options for management of the risk posed by Nipah virus, each of which would meet Australia's ALOP:

- the pigs from which the meat is derived have been kept since birth in a country or zone free from Nipah virus to the satisfaction of Australian authorities; or
- the pigs from which the meat is derived have been kept since birth in a country or zone in which the domestic pig population is free from Nipah virus to the satisfaction of Australian authorities; or

- the pig meat has been canned such that all portions of the can contents have been heated to at least 100°C.

The Department of Health and Ageing has advised Biosecurity Australia that risk management measures would be required for Nipah disease to address the risk to human life or health associated with the importation of pig meat. Appropriate measures for a country or zone which has reported Nipah virus would include that the pigs from which the pig meat was derived originate from a herd which has been tested negative for the disease agent or canning of pig meat such that all portions have been heated to at least 100°C (shelf stable).

Post-weaning multisystemic wasting syndrome

Post-weaning multisystemic wasting syndrome (PMWS) is not listed by the OIE. As such the international standard (OIE Terrestrial Animal Health Code) does not include any recommendations in regard to the risk of PMWS when importing pig meat.

The likelihood that PMWS could enter, become established and/or spread in Australia via imported pig meat could, in theory be reduced by the application of some or all of the following measures:

- a requirement that the pigs of origin had never been in a PMWS infected country or zone since birth;
- a requirement that slaughter and processing ensured removal of organs and tissues which are sites of predilection for the virus;
- reduction in the volume of pig meat waste discarded in Australia.

As porcine circovirus has been reported to be stable at a temperature 70°C for 15 minutes, the Panel did not examine the direct effect of processing (other than canning) on the destruction of this virus. Options were examined to identify the least trade restrictive measures which would reduce risks within Australia's ALOP.

Options

Country or zone freedom

A requirement that the pigs have been kept since birth in a country or zone free from PMWS to the satisfaction of Australian authorities would conform with Australia's ALOP (as discussed in the introduction to this Chapter, such approaches will be dealt with using existing mechanisms).

Canning – shelf stable

Australia currently accepts shelf stable canned pig meat from any source country subject to certain conditions. The Australian import conditions for canned meat include a requirement that all portions of the contents have been heated to at least 100°C. Porcine circovirus has been reported to be stable at 70°C for 15 minutes. The Panel considered that PCV2 would be inactivated in canned pig meat heated to at least 100°C.

Modified dressing of the carcass

Modified dressing of the carcass to remove certain tissues where the virus has an affinity would influence the fourth step in the release pathway (R4). This step describes the likelihood that the pathogenic agent would be present in meat harvested for export. Modified dressing of the

carcass may also influence the exposure step L2, which describes the likelihood that a waste unit would contain a sufficient dose of PCV2 to initiate infection.

Lymphoid tissues are the primary target tissues for PCV2, as for many other viruses. Viral antigen or nucleic acid is found in lymphoid tissues, including peripheral lymph nodes of clinically healthy and diseased pigs. Viral nucleic acid has been detected in bone marrow (Bolin, et al., 2001) and in serum for up to 16 weeks (Rodriguez-Arrioja, et al., 2002). Viral nucleic acid has also been detected in sera of slaughter-age pigs (Liu, et al., 2002). It is possible that these pigs may have been recently infected.

The Panel examined removal of major peripheral lymph nodes (i.e. removing the head and neck and removal of other major peripheral lymph nodes such as inguinal, popliteal, axillary etc) together with deboning the carcass on the above likelihoods. The Panel considered that removal of the head and neck, including any remaining tonsillar tissue, and lymph nodes draining the pharynx, other major peripheral lymph nodes and deboning could reduce the likelihood assigned to R4 from 'moderate' to 'low'.

Moreover, if meat is sourced from areas other than the head and neck and other major peripheral lymph nodes are removed together with bone, the amount of virus present in a waste unit would be reduced. It is known that the virus has a strong affinity for lymph nodes (Allan, et al., 1999; Rosell, et al., 1999). Virus titres of approximately 10^5 to 10^6 $TCID_{50}$/g of lymph node have been reported from clinically healthy pigs experimentally infected with PCV2 (Meehan, et al., 2001). Levels of virus in both serum and lymph nodes decrease with increasing time post-infection. In persistently infected pigs levels of virus in tissues are likely to be low.

Given the likely level of virus in muscle *per se*, the composition of pig meat waste (bone-out) and the volume of waste consumed by a pig, it was considered that the likelihood assigned to L2 could be reduced from 'moderate' to 'very low'.

Reduction in the volume of waste discarded

If product was deboned and cooked or cured the volume of waste discarded by households and food service establishments would be significantly reduced. Smallgoods are generally bought in smaller quantities by households, and there is very little waste from these products. Deboning alone may not reduce the volume of waste significantly as it was considered that a significant portion of waste discarded would be uncooked meat that has spoiled. Cooked bone-in product alone also may not reduce the volume of waste significantly, as a significant portion of waste is bone. On balance, the Panel considered that if meat was deboned and processed either by cooking or curing, the proportion of pig meat purchased by households and food service establishments that was discarded as waste would be reduced to one tenth of that estimated for the unrestricted risk (see Methods section, Table 4).

As discussed above, the direct effect of processing on PCV2 survival was not examined (other than canning), however, it was recognised that there may be some reduction in PCV2 titre after curing for long periods or cooking. It was also considered that if an unknown virus is required to trigger PCV2 for expression of PMWS, the Panel recognised that many other viruses are susceptible to heat leading to a reduction in virus titre or inactivation.

Conclusions

The 'restricted risk' estimate steps were as follows:

- the likelihood that the pathogenic agent would be present in meat excluding bone and major peripheral lymph nodes harvested for export was estimated to be 'low'; and
  - the likelihood that a waste unit (excluding bone and peripheral lymph nodes) from an infected pig would contain a sufficient dose of the pathogenic agent to initiate infection was estimated to 'very low';
- if product was processed by cooking or curing and deboned, the proportion of waste discarded was estimated to be one tenth of that estimated for the unrestricted risk.

Risk was estimated using the various combinations of mitigation measures discussed above and the results are summarised in Table 126. Deboning and processing of pig meat by cooking or curing and removal of peripheral major peripheral lymph nodes would reduce the risk of entry, establishment and/or spread to very low, which would meet Australia's ALOP. Modified dressing of the carcass (deboning, removal of major peripheral lymph nodes) or reducing the volume of waste discarded alone would not reduce the likelihood of entry, establishment and/or spread to an acceptable level.

Table 126 Risk management measures for PMWS

R4	Reduction in the proportion of waste discarded	Restricted risk
Deboned and peripheral lymph nodes removed	Cooked or cured product and deboned	
-	-	Low
+	-	Low
-	+	Low
+	+	Very low

- measure/s not applied

+ measure/s applied

Thus, there are several alternative options for management of the risk posed by PMWS, each of which would meet Australia's ALOP:

- the pigs from which the meat is derived have been kept since birth in a country or zone free from PMWS to the satisfaction of Australian authorities; or
- the pig meat has been canned such that all portions of the can contents have been heated to at least 100°C; or
- the meat has not been derived from the head or neck, major peripheral lymph nodes have been removed, meat has been deboned and the product has been cooked or cured.

As PCV2 has been shown to result in persistent infections, has been isolated from many tissues, is a hardy virus and is likely to be transmitted orally, the Panel considered that if an unknown disease agent was involved in PMWS, the risk management measures requiring removal of bone, major peripheral lymph nodes, head and neck and cooking or curing would act to reduce the risks associated with that agent. Biosecurity Australia and the PMWS technical working group will continue to monitor the situation in response to new information that becomes available on the disease.

It is recommended that pig meat be processed off-shore or on-shore subject to processing (cooking/curing) in an urban area at the port of entry or in a rural area subject to appropriate security transport arrangements (eg. refrigerated container), under a compliance agreement with the Australian Quarantine and Inspection Service (AQIS). The Panel recognised that there may be a slight increase in risk with processing imported product on-shore in Australia, however, it is considered that this is offset by the added confidence provided by AQIS audit of processors ensuring compliance with the conditions. It is considered that the assurance of AQIS audits of processing plants and compliance agreement arrangements covering the storage, transport and processing of imported product and management of waste provides an equivalent level of quarantine protection to that provided by processing off-shore. The Panel also noted the history of safely processing imported pig meat in Australia under quarantine control for the previous 11 years. As the risk management measure for PMWS relies partly on reducing the amount of waste discarded, removal of the head and neck, bone and major lymph nodes must occur prior to the export of pig meat for further processing (cooking/curing) in Australia. Removal of these materials prior to export will significantly reduce the amount of waste which would be associated with imported pig meat.

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QUARANTINE REQUIREMENTS FOR IMPORTATION OF PIG MEAT

This biosecurity policy is applicable to the importation of pig meat whether uncooked, cooked or cured.

1. DOCUMENTATION

1.1 A Permit to Import pig meat into Australia (the Permit includes an Approval Advice for the source establishment), must be obtained in writing from the Director of Animal and Plant Quarantine (Australia) (hereinafter called the Director) prior to export of the first consignment from the approved source establishment.

1.2 The application to import must specify the following:

- the name and address of the importer and exporter and the name and veterinary control number of the approved abattoir and, if applicable, approved cutting-up establishment, approved processing establishment and approved storage establishment in the source country;
- the cut or cuts (trade description) of the meat/product to be imported;
- the anticipated port or ports of entry of the pig meat

1.3 The application will be assessed on the above criteria as well as any other criterion which is considered relevant by the Director.

2. REQUIREMENTS

2.1 Each consignment must be accompanied by official certification in accordance with these requirements and will require, on arrival, a "Quarantine Entry" issued by the Australian Quarantine and Inspection Service (AQIS).

2.2 Quarantine entry barrier clearance of each consignment will remain subject to examination of accompanying documentation and sighting by a Quarantine Officer.

2.3 The product and consignment details must correspond exactly with documentation and the Permit to Import.

2.4 The pigs must be slaughtered and the meat prepared in establishments currently approved by the Director. The standard of construction and facilities of the slaughter establishments, the establishment where the meat was prepared and the establishment where it was stored must meet the Australian Standard for the Hygienic Production and Transportation of meat for Human Consumption, or any standards agreed by AQIS to be equivalent. AQIS may take into account existing approvals granted by the relevant overseas veterinary authorities.

2.5 While preparing product for Australia, establishments must conduct slaughter, preparation and storage of the meat in accordance with quality assurance principles such as the HACCP approach.

2.6 The pig meat for export to Australia must comply with AQIS quarantine requirements and other requirements including the Australia New Zealand Food Standards Code.

2.7 Public health requirements (see Annex 4 for requirements of the Department of Health and Ageing)

Imported pig meat must comply with the *Imported Food Control Act 1992* and the *Food Standards Code* developed under *Food Standards Australia New Zealand Act 1991*. Under this legislation, AQIS may inspect, sample, hold and test imported pig meat for microbial agents or residues of public health concern. Additional requirements regarding labelling, packaging and food composition standards must also be complied with. Information on the *Food Standards Code* may be obtained from the Food Standards Australia New Zealand (FSANZ).

2.8 The Quarantine Officer at the port of entry shall note the number of containers which have been off-loaded at the port of call, and their identifying marks and seal numbers.

3. CERTIFICATION

3.1 Each consignment must be accompanied by a Veterinary Certificate in accordance with the Office International des Epizooties (OIE) International Animal Health Code 'Model international veterinary certificate for meat of domestic animals' (Appendix 4.2.1. of the Code) signed by an Official Veterinarian. The certificate must provide details of:

- the packaging of the meat including details of the labelling,
- the addresses and veterinary approval numbers of establishments at which the animals from which the meat was derived were slaughtered, the cutting-up establishment at which it was prepared and the establishment at which it was stored prior to export,
- the names and addresses of the exporter and the consignee.

3.2 The Official Veterinarian of the source country must certify in English, under **IV. <u>Attestation of wholesomeness</u>**, that:

(i) The pigs from which the meat was derived have been continuously resident in the source country since birth and were slaughtered on ..... (dates).

(ii) The pigs from which the meat was derived passed ante- and post-mortem veterinary inspection under official veterinary supervision; the meat is considered to be fit for human consumption.

(iii) All of the following risk management measures apply:

a) The pigs from which the meat was derived have been kept since birth in a country or zone which is recognised by Australian authorities as free from foot and mouth disease and have not been vaccinated. **or**

The pig meat has been canned such that all portions of the can contents have been heated to at least 100°C.

b) The pigs from which the meat was derived have been kept since birth in a country or zone which is recognised by Australian authorities as free from African swine fever (ASF). **or**

The pig meat has been processed by canning such that all portions of the contents have been heated to at least 100°C. **or**

The pigs from which the meat was derived have been sourced from premises which have been free from evidence (clinical, serological, virological) of ASF infection for the 3 months prior to slaughter; **and**, the premises are located in an area where ASF is compulsorily notifiable; **and**, the pig meat has been dry cured[73] under specified conditions for the production of Parma type hams (minimum curing 399 days), Iberian type hams, loins or shoulders, or Serrano type hams (minimum curing 140 days).

c) The pigs from which the meat was derived have been kept since birth in a country or zone which is recognised by Australian authorities as free of classical swine fever (CSF). **or**

The pig meat has been processed by canning such that all portions of the contents have been heated to at least 100°C. **or**

The pigs from which the meat was derived have been sourced from premises which have been free from evidence (clinical, serological, virological) of CSF infection for the 3 months prior to slaughter; **and**, the premises are located in an area where CSF is compulsorily notifiable; **and**, the pig meat has been dry cured under specified conditions for the production of Parma type hams (minimum curing 313 days), Iberian type hams, loins or shoulders, or Serrano type hams (minimum curing 252 days).

d) The pigs from which the meat was derived have been kept since birth in a country or zone which is recognised by Australian authorities as free from rinderpest. **or**

The pig meat has been processed by canning such that all portions of the contents have been heated to at least 100°C.

e) The pigs from which the meat was derived have been kept since birth in a country or zone which is recognised by Australian authorities as free from swine vesicular disease (SVD). **or**

The pig meat has been processed by canning such that all portions of the contents have been heated to at least 100°C. **or**

The pigs from which the meat was derived were sourced from herds serologically tested negative for SVD using either virus neutralisation or ELISA as described in the OIE Manual of Diagnostic Tests and Vaccines within the 6 months prior to slaughter and within the 6 months following slaughter; **and**, the premises are located in an area where SVD is notifiable; **and**, the pig meat has been dry cured under specified conditions for the production of Parma type hams (minimum curing 360 days).

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[73] The full published specifications for Parma hams, Serrano hams, and Iberian hams, loins and shoulders, are available through the institutions responsible for certification of the respective products. They are not reproduced here but the relevant specifications will be part of Australia's certification requirements.

f) The pigs from which the meat was derived have been kept since birth in a country or zone which is recognised by Australian authorities as free from Aujeszky's disease. **or**

The meat is not derived from the head or neck[74]. **or**

The meat has been deboned[75] and the product is processed (cooked or cured)[76].

g) The pigs from which the meat was derived have been kept since birth in a country or zone which is recognised by Australian authorities as free from porcine reproductive and respiratory syndrome (PRRS) and where vaccination is not permitted. **or**

The pig meat has been processed by canning such that all portions of the contents have been heated to at least 100°C. **or**

The pig meat has been dry cured under specified conditions for Parma type hams (minimum curing 313 days), Iberian type hams, loins or shoulders or Serrano type hams (minimum curing 140 days). **or**

The pig meat has been heat processed such that all parts reach a core temperature of at least 70°C for 11 minutes, or equivalent according to the time/temperatures specified in Annex 1 below. **or**

The pigs from which the meat was derived are not from a country or zone recognised by Australia as free from PRRS and the meat has not been processed as specified above.
Note: In this case, the meat must be processed in Australia - see Section 4.

h) The pigs from which the meat was derived have been kept since birth in a country or zone which is recognised by Australian authorities as free from *Trichinella spiralis*. **or**

Appropriate samples from each pig from which the meat was derived have been tested and found negative for the presence of *Trichinella spiralis* larvae by pepsin digestion or ELISA as described in the OIE Manual of Diagnostic Tests and Vaccines. **or**

The pig meat has been processed by canning such that all portions of the contents have been heated to at least 100°C. **or**

The pig meat has been cooked to one of the time/temperatures specified below (Annex 2)[77]. **or**

The pig meat has been frozen according to one of the time/temperature conditions specified below (Annex 3). **or**

The pig meat has been dry cured under specified condition for Parma type hams (minimum curing 313 days), Iberian type hams, loins or shoulders or Serrano type hams (minimum curing 140 days)[78].

[74] Meat must not be derived cranial to the fourth cervical veterbrae.
[75] Deboning can occur after product has been cooked or cured.
[76] Processing could occur off-shore or on-shore.
[77] Processing could occur off-shore or on-shore.
[78] Processing could occur off-shore or on-shore.

i) The pigs from which the meat was derived have been kept since birth in a country or zone which is recognised by Australian authorities as free from Nipah virus. **or**

The pigs from which the meat was derived have been kept since birth in a country or zone in which the domestic pig population is recognised by Australian authorities as free of Nipah virus **or**

The pig meat has been canned such that all portions of the can contents have been heated to at least 100°C.

j) The pigs from which the meat was derived have been kept since birth in a country or zone which is recognised by Australian authorities as free from post-weaning multisystemic wasting syndrome (PMWS). **or**

The pig meat has been processed by canning such that all portions of the contents have been heated to at least 100°C. **or**

The meat has <u>not</u> been derived from the head or neck[79], major peripheral lymph nodes have been removed, meat has been deboned[80] and the product is processed (cooked or cured). **or**

The pigs from which the meat was derived are not from a country or zone recognised by Australia as free from PMWS and the meat has not been processed by cooking or curing as above[81].
Note: In this case, the meat must be processed in Australia - see Section 4.

k) The pigs from which the meat was derived have been kept since birth in a country or zone which is recognised by Australian authorities as free from vesicular exanthema virus.

(iv) The establishment where the pigs from which the meat was derived were slaughtered, the establishment where the meat was prepared and the establishment where it was stored, have current AQIS approval for facilities and hygienic operation;

Note: The name(s), address(es) and veterinary control number(s) of plant(s) must be specified;

(v) Officials of the *Veterinary Authority* of the source country were present in plants at all times when pigs were being slaughtered for export to Australia.

(vi) The establishment where the meat was prepared did not prepare or process pig meat not eligible for export to Australia while pig meat was being prepared for export to Australia.

(vii) The meat has been prepared for export and packed on ..... (dates), and the bags, wrappers or packing containers were clean and new.

(viii) The identification number of the slaughtering establishment and the establishment where the meat was prepared is readily visible on the meat or, where the meat is wrapped or packed, was marked on the package or wrapping containing the meat, in such a way that

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79 Meat must not be derived cranial to the fourth cervical vertebrae.

80 Deboning and removal of major peripheral lymph nodes can occur after product has been cooked or cured.

81 Meat must not be from the head or neck and must be deboned, and major peripheral lymph nodes removed prior to export to Australia for further processing.

the numbers cannot readily be removed without damaging the meat, package or wrapping.

(ix) The meat was not exposed to contamination prior to export.

(x) The meat is being transported to Australia in a clean packing container sealed with a seal bearing the number or mark ..... ; the container contains only meat eligible for entry into Australia.

4. POST-ENTRY CONTROL AND PROCESSING REQUIREMENTS

Pig meat from countries/zones not recognised as free from PRRS and not processed to ensure inactivation of PRRS virus, as specified in 3.2 (iii) g), may be imported subject to further processing in an establishment that has entered into a compliance agreement with AQIS under a quality assurance arrangement.

Pig meat from countries/zones not recognised as free from PMWS and not processed as specified in 3.2 (iii) j) (cooked or cured), may be imported subject to further processing in an establishment that has entered into a compliance agreement with AQIS under a quality assurance arrangement.

The following conditions apply.

4.1 A copy of the documentation must accompany each consignment of imported pig meat and its derivatives during transport to storage and processing establishments and until it has been adequately processed.

4.2 The pig meat and its derivatives must be securely transported from the nearest port of entry to the approved storage establishment(s) thence to the processing establishment(s) within the urban area and finally, with respect to inadequately processed surplus wastes and by-products, to the place(s) of disposal of quarantinable waste. Potentially suitable control systems may include leak-proof packing containers sealed with a numbered, tamper-proof seal at the point of origin for removal and retention at the point of destination. Alternatively, a system based on despatch and receival weights may be used to accurately account for control of the product. The transport of imported pig meat outside urban areas associated with the Australian port of entry will require appropriate security arrangements to prevent spillage (e.g. refrigerated container) and be transported by the most direct route.

After release from quarantine, the meat must be processed in accordance with the Compliance Agreement prior to distribution for retail sale or consumption. The compliance agreement also covers such thing as disposal of packaging, waste water and trimmings.

5. REVIEW

Conditions for importation may be reviewed if there are any changes in the source country's import policy or animal disease status or at any time at the discretion of the Director.

DAVID BANKS
General Manager
Animal Biosecurity

ANNEX 1

To conform with Australian requirements for inactivation of PRRS virus by heat processing, the minimum core temperatures shown in the following table must be maintained continuously for the minimum times stipulated.

Inactivation of PRRS virus in pig meat by heating

Minimum core temperature (°C)	Minimum time (minutes)
56	60
57	55
58	50
59	45
60	40
61	35
62	30
63	25
64	22
65	20
66	17
67	15
68	13
69	12
70	11

ANNEX 2

To conform with Australian requirements for inactivation of *Trichinella spiralis* in pig meat by heating, the minimum core temperatures shown in the following table must be maintained continuously for the minimum times stipulated.

Inactivation of *Trichinella spiralis* in pig meat by heating

Minimum core temperature (°C)	Minimum time (minutes)
50.0	570
51.1	270
52.2	120
53.4	60
54.5	30
55.6	15
56.7	6
57.8	3
58.9	2
60.0	1
61.1	1
62.2	Instant

ANNEX 3

To conform with Australian requirements for inactivation of *Trichinella spiralis* in pig meat by freezing, the maximum core temperatures shown in the following table must be maintained continuously for the minimum times stipulated.

Inactivation of *Trichinella spiralis* in pig meat by freezing meat to a specified core temperature

Maximum core temperature (°C)	Minimum time (hours)
-17.8	106
-20.6	82
-23.3	63
-26.1	48
-28.9	35
-31.7	22
-34.5	8
-37.2	0.5

Alternatively, the meat may be subjected to maximum freezer temperatures according to the table below. The initial meat temperature must not be above 5°C. Group 1 products are those whose minimum thickness is up to 15 cm. Group 2 products are those whose minimum thickness lies between 15 cm and 68 cm. In either case, the product must be packed such that there is free air access between layers.

Inactivation of *Trichinella spiralis* in pig meat by freezing - freezer temperature

Freezer temperature (°C)	Group 1 (<= 15 cm thickness) - days	Group 2 (> 15 but <= 68 cm thickness) – days
-15.0	20	30
-23.3	10	20
-28.9	6	12

ANNEX 4

The Department of Health and Ageing requires additional certification to address human health concerns.

1. In countries where Nipah virus has been reported:

   In the case of uncooked meat imported into Australia, the pigs from which the meat is derived originate from herds that have tested negative for Nipah virus.

2. In countries where *Brucella suis* is endemic:

   In the case of uncooked meat imported into Australia, the pigs from which the meat is derived originate from herds that have been tested negative or are accredited free from *B. suis*.

3. Processed (cooked, cured) pig meat must comply with the Food Standards Code including testing for *Salmonella*.

CONCLUSIONS

The findings of this *Final IRA Report* are based on a comprehensive analysis of relevant scientific literature and existing import requirements for importation of pig meat into Australia.

Biosecurity Australia considers that the risk management measures in this *Final IRA Report* will provide an appropriate level of protection against the disease agents identified in the risk assessment. Various risk management measures may be suitable to manage the risks associated with pig meat and Biosecurity Australia will consider other measures suggested by stakeholders that provide an equivalent level of protection.

In the course of preparing the *Final IRA Report*, Biosecurity Australia received submissions on scientific issues raised in the *Technical Issues Paper* and on the draft paper on method for the import risk analysis and the *Draft IRA Report*. A synopsis of submissions received in response to the *Technical Issues Paper*, *Draft Methods Paper* and the *Draft IRA Report* along with Biosecurity Australia's and the Panel's response are released concurrently with this *Final IRA Report*. Biosecurity Australia and the Panel considered all scientific issues raised in the submissions of stakeholders and incorporated the comments as appropriate.

FURTHER STEPS IN THE IMPORT RISK ANALYSIS PROCESS

The IRA process requires that the following steps be followed:

- Release of the *Final IRA Report*;
- Consideration of any appeals;
- When the above processes are complete, the Director of Animal and Plant Quarantine makes the final determination;

Stakeholders will be advised of any significant variations to this process.