Australian Government

Biosecurity Australia

Generic Import Risk Analysis Report for Chicken Meat

Final Report

Part C

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Foreword

This import risk analysis report is issued in four parts:

- Part A contains a brief summary of the import risk analysis (IRA).
- Part B contains background material, an explanation of the method used in the IRA, and a report of the Hazard identification and Hazard refinement steps.
- Part C contains the detail of the assessments for each of the identified hazards, together with the proposed risk management measures, and Health Certification requirements.
- Part D contains appendices with comments received from stakeholders in earlier stages of the risk analysis process, and further explanatory or background material.

This document is Part C

It contains detailed risk assessments for each of the identified hazards. It also contains a discussion of the risk management measures proposed to manage the identified risks, and a set of proposed quarantine conditions for the import of chicken meat.

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Glossary of Terms and Abbreviations

AAHL	Australian Animal Health Laboratory
AAS	Avian adenovirus splenomegaly
ABARE	Australian Bureau of Agricultural and Resource Economics
ABPM	Animal Biosecurity Policy Memorandum
ACMF	Australian Chicken Meat Federation
ACT	Australian Capital Territory
AECL	Australian Egg Corporation Limited
AEIA	Australian Egg Industry Association (now AECL)
AGID	Agar Gel Immunodiffusion Test
AI	Avian influenza
ALOP	Appropriate Level of Protection
AMPV	Avian metapneumovirus
APMV	Avian paramyxovirus
AQIS	Australian Quarantine and Inspection Service
AQPM	Animal Quarantine Policy Memorandum
AQRC	Australian Quarantine Review Committee
ARAZPA	Australian Regional Association of Zoological Parks and Aquaria
AUSVETPLAN	Australian Veterinary Emergency Plan
Biosecurity Australia	a prescribed agency within the Agriculture, Fisheries and Forestry portfolio
BAPM	Biosecurity Australia Policy Memorandum
BP	Backyard poultry (refers to the low biosecurity poultry exposure group)
CELO	Chicken Embryo Lethal Orphan (virus)
CSIRO	Commonwealth Scientific and Industrial Research Organisation
CP	Commercial poultry (refers to the medium biosecurity commercial poultry exposure group)
DAFF	Australian Government Department of Agriculture, Fisheries and Forestry
DEWHA	Australian Government Department of the Environment, Water, Heritage and the Arts – formerly Australian Government Department of the Environment and Water Resources (DEW) – formerly the Department of Environment and Heritage (DEH)
DIVA	Differentiating Infected from Vaccinated Animals
DoHA	Australian Government Department of Health and Ageing
EAD	Emergency animal disease
EDS	Egg Drop Syndrome
EEE/WEE/VEE	Eastern, Western and Venezuelan equine encephalomyelitis
EHEC	Enterohaemorrhagic *Escherichia coli*
EID	Egg infectious dose
EMS	Environmental Management Services

EPA	Environmental Protection Authority
EU	European Union
FAdV	Fowl adenovirus
FAO	Food and Agriculture Organization of the United Nations
FSANZ	Food Standards Australia New Zealand
FSC	Australia New Zealand Food Standards Code
Generic IRA	An import risk analysis relevant to all exporting countries. The generic IRA does not consider the disease status or data of individual countries, but is based on estimates of the most likely situation in an hypothetical infected country. Country-specific data may be considered at a later date should appropriate data from prospective exporting countries be supplied.
HA	Haemagglutinin
HEV	Haemorrhagic Enteritis virus
HI	Haemagglutination Inhibition
HP	Highly pathogenic
HPNAI	Highly Pathogenic Notifiable Avian Influenza
HPS	Hydropericardium syndrome
IBDV	Infectious Bursal Disease Virus
IBV	Infectious Bronchitis virus
ID	Infectious dose
IgG	Immunoglobulin G
IgM	Immunoglobulin M
ILT	Infectious Laryngotracheitis
ICPI	Intra-cerebral pathogenicity index
IVPI	Intravenous pathogenicity index
ICON	AQIS Import Conditions database
IPPC	International Plant Protection Convention
IRA	Import risk analysis
JE	Japanese encephalitis
LPNAI	Low pathogenicity notifiable avian influenza (H5 and H7 subtypes)
LPAI	Low pathogenicity avian influenza (subtypes other than H5 and H7)
MDCK	Madin-Darby Canine Kidney II (MDCK II) cells (cell-culture)
MOU	Memorandum of understanding
MSDV	Marble Spleen Disease virus (of pheasants)
NAHIS	National Animal Health Information System
NAI	Notifiable avian influenza
NAMAC	National Arbovirus and Malaria Advisory Committee – a subcommittee of Communicable Diseases Australia within the Health and Ageing Portfolio
NAQS	Northern Australia Quarantine Strategy
NAS	Non-avian species
NDV	Newcastle Disease virus
NSW	New South Wales

NT	Northern Territory
OIE	World Organization for Animal Health (formerly known as the Office International des Epizooties)
OIE Code	OIE Terrestrial Animal Health Code
PALEEES	Partial annual likelihood of entry, exposure, establishment and spread
PAREEES	Partial annual risk of entry, exposure, establishment and spread
PCR	Polymerase chain reaction
PLE	Partial likelihood of exposure
PLEEES	Partial likelihood of entry, exposure, establishment and spread
PLES	Partial likelihood of establishment and spread
PRNT	Plaque reduction neutralisation test
RT-PCR	Reverse transcriptase-polymerase chain reaction
SCARM	Standing Committee on Agriculture and Resource Management
SPA	Serum plate agglutination
SPF	Specific pathogen free
SPS	Sanitary and Phytosanitary
SPS Agreement	WTO Agreement on the Application of Sanitary and Phytosanitary Measures
TWG	Technical Working Group
USA	United States of America
USDA-FSIS	United States Department of Agriculture Food Safety and Inspection Service
WB	Wild Birds (refers to the wild birds exposure group)
WNV	West Nile Virus
WTO	World Trade Organization

Notifiable Avian Influenza Virus

Technical Information

Background

Avian influenza viruses circulate in wild and domestic birds. Most avian influenza (AI) viruses are of low pathogenicity (LP), producing either subclinical disease or mild respiratory or reproductive disease in domestic birds. Notifiable forms of AI viruses exist, however, which are either highly pathogenic (HP), or of low pathogenicity but with the potential to mutate to highly pathogenic forms. All reported outbreaks of highly pathogenic AI in poultry have been of the H5 or H7 subtypes, with the exception of two H10 isolates that fulfilled some criteria for classification as HP (Wood et al. 1996).

Highly pathogenic notifiable avian influenza (HPNAI), formerly known as fowl plague, is a highly contagious systemic disease of poultry that causes high mortality in domestic chickens. HPNAI viruses have been documented to arise from mutations in low pathogenicity notifiable AI viruses (LPNAI) viruses, with mutations probably occurring within domestic poultry populations (Swayne and Suarez 2000; Garcia et al. 1996).

Although outbreaks have been recorded in Australia in the past, Australia is currently free of HPNAI. The definition of notifiable avian influenza (NAI) viruses was recently reviewed by the OIE and is described below (World Organisation for Animal Health (OIE) 2007a).

Agent taxonomy

AI viruses (AIV) are single-stranded, enveloped RNA viruses of the *Orthomyxoviridae* family. The influenza viruses that constitute this family are classified into types A, B or C based on differences between their nucleoproteins and matrix protein antigens. All avian influenza viruses belong to type A (Easterday, Hinshaw, and Halvorson 1997).

Influenza A viruses are divided into subgroups according to serologic reactions to their haemagglutinin (HA) and neuraminidase (NA) surface antigens. Sixteen haemagglutinins (H1–H16) and nine neuraminidases (N1–N9) have been identified to date (Swayne and Halvorson 2003; Fouchier et al. 2005). The HA gene is the primary determinant of pathogenicity in chickens.

All reported outbreaks of highly pathogenic AI in poultry have been of the H5 or H7 subtypes, with the exception of two H10 isolates that fulfilled some criteria for classification as HP (Wood et al. 1996). Many H5 and H7 subtypes isolated from poultry have been of low or mild pathogenicity (Swayne and Suarez 2000; Swayne and Halvorson 2003). However, because of the risk of an H5 or H7 virus becoming virulent by mutation, all H5 and H7 viruses have been identified as NAI viruses (World Organisation for Animal Health (OIE) 2007a; Alexander 2004).

For the purposes of the OIE Terrestrial Animal Health Code (World Organisation for Animal Health (OIE) 2007a), ‘avian influenza in its notifiable form (NAI) is defined as an infection of

poultry caused by any influenza A virus of the H5 or H7 subtypes, or by any AI virus with an intravenous pathogenicity index (IVPI) greater than 1.2 (or as an alternative at least 75% mortality) as described below. NAI viruses can be divided into highly pathogenic notifiable avian influenza (HPNAI) and low pathogenicity notifiable avian influenza (LPNAI):

'a. HPNAI viruses have an IVPI in 6-week-old chickens greater than 1.2 or, as an alternative, cause at least 75% mortality in 4– to 8–week-old chickens infected intravenously. H5 and H7 viruses which do not have an IVPI of greater than 1.2 or cause less than 75% mortality in an intravenous lethality test should be sequenced to determine whether multiple basic amino acids are present at the cleavage site of the hemagglutinin molecule (HA0); if the amino acid motif is similar to that observed for other HPNAI isolates, the isolate being tested should be considered as HPNAI;

'b. LPNAI are all influenza viruses of H5 and H7 subtype that are not HPNAI viruses.'

Agent characteristics

Resistance to physical and chemical action is shown in Table 1 below.

Table 1. Resistance of agent to physical and chemical action.

Temperature:	Most strains inactivated by 56 °C for 3 hours; 60 °C for 30 min
pH:	Inactivated by acid pH; stable between pH 5 and pH 8
Chemicals:	Inactivated by oxidising agents, sodium dodecyl sulphate, lipid solvents, ß-propiolactone
Disinfectants:	Inactivated by formalin and iodine compounds
Survival:	Remains viable for long periods in tissues, faeces, and also in water

(World Organisation for Animal Health (OIE) 2002)

Infectivity of virus can be maintained for several weeks at 4 ºC in the laboratory, for 30–35 days at 4 ºC in faeces, and for seven days at 20 ºC in the field. The virus can be stored long term at –70 ºC (Swayne and Halvorson 2003).

Tissues of experimentally-infected chickens contained infective virus for surprisingly long periods of time at various temperatures. Skeletal muscle and some other organs remained infective at room temperature for 30–40 days, and bone marrow remained infective for 60 days at room temperature. Virus was found to persist in the bone marrow of unopened carcasses stored in soil for 105 days (Vrtiak and Kapitancik 1967). AI virus has been isolated from the surface, yolk and albumen of eggs during a naturally-occurring NAI (H5N2) outbreak (Cappucci et al. 1985).

Specific scientific data on the destruction of avian influenza viruses are not available for most virus strains and for the majority of poultry commodities (World Organisation for Animal Health (OIE) 2003). Experimentally, heat inactivation of AI virus appears to be dependent on the strain of the virus and the medium in which it is suspended during testing. Infectivity of most strains was lost after heating to 60 °C for five minutes, and 56 °C for 15–30 minutes; however, some strains required up to six hours at 56 °C for inactivation (Moses et al. 1948; Lang et al. 1968b; Lang et al. 1968a; Homme and Easterday 1970; Lu et al. 2003).

The European Commission requires that cooked imported chicken meat from Thailand reaches a core temperature of 70 °C, with no time specified, and Thailand certifies that chicken meat is cooked to 70 °C for one minute for export to the European Union (European Commission Health and Consumer Protection Directorate-General 2005). The World Health Organization INFOSAN web site states that 'Conventional cooking (temperatures at or above 70 °C in all parts of a food item) will inactivate the H5N1 virus. Properly cooked poultry meat is therefore safe to consume' (World Health Organization 2005).

Swayne (2006) developed a precise microassay technique for determining thermal inactivation of avian influenza virus in chicken meat samples. Part of the justification for the study was to overcome some of the factors that lead to the variation in inactivation data in the published literature, such as virus titre variations, uneven dispersion of the added virus in the medium, or inconsistent heating of samples. The study involved the use of meat samples from chickens experimentally infected with HPAI virus. Initial muscle titres varied between $10^{6.8}$ mean egg infectious doses (EID_{50})/g, and $10^{2.3}$ EID_{50}/g. The microassay technique gave precise, reproducible results in small samples of lean meat with no visible skin or fat, and showed that the titre of AI virus in the meat of experimentally-infected chickens is reduced after achieving a temperature of 60 °C, and that the virus is inactivated when an internal temperature of 70 °C is reached. In this experiment, no virus was recoverable after five seconds at 70 °C, even from the most heavily infected samples ($10^{6.8}$ EID_{50}/g). However, inactivation curves and D values for AI viruses were not published in this report (Swayne 2006).

Further quantitative measurements of heat inactivation of AI virus in chicken meat were published in 2007 (Thomas and Swayne 2007a). In that study, thigh and breast meat were harvested from four-week-old SPF chickens intranasally-infected with Korea/03 H5N1 virus, viral titres in the meat were recorded, and thermal inactivation was quantitatively measured over a range of temperatures from 57 °C to 61 °C. The calculations suggested that an 11-log reduction in Korea/03 H5N1 virus should take place within one second at 73.9 °C, but could take 5.5 seconds at 70 °C. 'The D values for 70 °C and 73.9 °C were predicted by extrapolation beyond the range of temperatures included in this study, and the true accuracy of the regression model in this range is unknown (Thomas and Swayne 2007a). In further unpublished research, the same authors have showed that D values for Newcastle disease (ND) virus were similar to those for AI virus under identical experimental conditions (Thomas and Swayne 2007b).

Alexander and Manvell (2004) investigated the heat inactivation of ND virus in homogenised chicken meat with a 15% fat and skin content, and calculated a D_{70} value of 82 seconds (Alexander and Manvell 2004). 'In the absence of similar data on AI viruses, Newcastle disease virus could be considered sufficiently similar to AI viruses that these figures could serve as a guide for estimating the efficacy of heat treatments at reducing the risk of infective poultry meat' (Capua and Alexander 2006).

The time required to inactivate AI virus at 70 °C in different commercial chicken meat products has not yet been determined (Swayne 2006).

Epidemiology

AI viruses are distributed worldwide in many species of domestic and wild birds, including chickens, turkeys, domestic and wild waterfowl and game birds, passerines, psittacines, raptors and ratites (Easterday, Hinshaw, and Halvorson 1997; Swayne and Halvorson 2003). Wild birds, particularly wild aquatic birds such as ducks, gulls and shorebirds, are believed to provide a reservoir of AI viruses, with asymptomatic enteric infections leading to faecal

shedding of virus. H5 and H7 subtypes are found sporadically in ducks, shorebirds and gulls, although other HA types are more commonly isolated from these groups of birds (Sharp et al. 1993; Fouchier et al. 2003; Munster et al. 2005).

Virus is circulated in waterfowl by the faecal-oral route, with the virus persisting in bodies of water for variable periods of time from 9 to over 100 days (Stallknecht et al. 1990; Brown et al. 2007). Domestic birds, including poultry, are infected through direct or indirect contact with wild birds, or through faecal contamination of water or feed supplies (Swayne and Suarez 2000), and spread of infection between farms can occur mechanically by people or fomites, or by aerosol.

There is accumulating evidence that HP viruses arise from LP H5 or H7 viruses infecting chickens and turkeys after spread from free-living birds (Horimoto et al. 1995; Capua and Marangon 2000; Banks et al. 2001; Alexander 2003; Lee et al. 2004; Munster et al. 2005). Experimentally, an avirulent AI virus derived from wild swans became highly pathogenic after 29 consecutive passages in chickens (Ito et al. 2001). H7N3 viruses, responsible for the 2002–03 avian influenza outbreak in domestic poultry in northern Italy, were shown by genetic sequencing to have been derived directly from viruses circulating in ducks at least a year earlier (Campitelli et al. 2004). H5 and H7 virus isolates recovered from wild birds in Europe over a four year period were closely related to those causing HPNAI and LPNAI outbreaks in Italy and the Netherlands between 1997 and 2003 (Munster et al. 2005). ‘It can only be assumed that all H5 and H7 viruses have this potential [to mutate] and mutation to virulence is a random event’ (Alexander 2003).

Until recently, HPNAI viruses had rarely been isolated from wild birds, even during outbreaks in domestic poultry (Morgan and Kelly 1990; Ellis et al. 2004a; Gilchrist 2005; Li et al. 2004; Tracey et al. 2004; Swayne and Suarez 2000; Sturm-Ramirez et al. 2004; FAO Technical Task Force on Avian Influenza 2005a; Capua et al. 2000). However, H5N1 viruses have been isolated from numerous orders and species of wild birds since 2003, including: Ciconiiformes (herons, egrets; storks); Phoenicopteriformes (flamingos); Podicipediformes (grebes); Anseriformes (ducks, swans, geese); Charadriformes (gulls); Falconiformes (falcons, hawk-eagle, eagles); Strigiformes (owls); Struthioniformes (emu); Galliformes (chukars, partridge, quail, chickens, turkeys, guineafowl; pheasant); Pelecaniformes (cormorants); Passeriformes (finches, crows, black drongo, mynahs, red-billed leiothrix, munia, oriole, Eurasian tree-sparrow, magpie, starling, robin), Gruiformes (common moorhen) and Columbiformes (pigeons, doves) (USGS National Wildlife Health Center 2005). Experimental studies show that there is significant species-related variation in susceptibility to infection, clinical disease, and antibody response to H5N1 virus infection in wild birds (Brown et al. 2006).

In 2005, H5N1 viruses were isolated from a sample of more than 6000 wild birds which died at Lake Qinghai in northwest China. Although this was a large mortality event, the dead birds represented a relatively small proportion of the migratory birds present at the lake. Viruses isolated from affected humans and birds in Turkey in January 2006 and Nigeria in February 2006 show genetic similarity to those isolated from migratory birds at Lake Qinghai in April 2006 (World Health Organization 2006), supporting speculation that the spread of H5N1 viruses from South East Asia into Eastern and Central Europe may have been associated with the migration of wild bird species (Gilbert et al. 2006). Domestic poultry may have become infected while sharing water-sources with wild birds (FAO Technical Task Force on Avian Influenza 2005b). Studies on genetic and antigenic lineages of H5N1 viruses in South East Asia have provided further evidence that migratory birds can disseminate the viruses over long distances (Chen et al. 2006). However, intercontinental spread of H5N1 has also been

associated with trade in poultry, poultry products and live birds (Ducatez et al. 2006; Feare 2007; Kilpatrick et al. 2006; DEFRA 2007).

In the absence of good biosecurity, non-migratory wild bird species may also represent a risk of transmitting H5N1 locally between poultry farms (FAO Technical Task Force on Avian Influenza 2005a). The likelihood of transmission of HPNAI virus by wild passerine birds was examined using an isolate from an Australian outbreak. In an experimental study using a Victorian strain of HPNAI virus (A/Chicken/Vic/1/85 (H7N7)), inoculated sparrows had a 30% mortality rate, with surviving birds shedding virus in the faeces, while inoculated starlings had 100% mortality (Nestorowicz et al. 1987). The authors concluded that infection in starlings would have been self-limiting, but sparrows may have been effective at transmitting the virus.

In an experimental study, Hong Kong H5N1 virus was inoculated into emus, geese, domestic ducks and pigeons. The geese, emus and ducks were susceptible to infection and it was thought that they could serve as transient hosts of the virus. Pigeons were resistant to infection and did not shed virus after inoculation (Perkins and Swayne 2002). However, H5N1 viruses have since been isolated from dead pigeons in several countries between 2004 and 2006, during outbreaks in poultry and wild birds (Swayne 2007). Hong Kong H5N1 virus underwent replication after experimental inoculation into pigs, rats and mice but was not transmitted to in-contact animals of the same species. However, in chickens the virus was highly contagious via the faecal-oral route, resulting in severe clinical signs and early mortality (Shortridge et al. 1998).

Avian influenza viruses have been shown in experimental studies to infect pigs, ferrets, rats, rabbits, guinea pigs, mice, cats, mink, non-human primates and humans (Swayne and Halvorson 2003). H5N1 virus has been isolated from sick and dead domestic cats and zoo felids which were thought to have consumed infected poultry or other birds (Keawcharoen et al. 2004; World Organisation for Animal Health (OIE) 2004; Thiry et al. 2007). Transmission of virus apparently occurred between cats experimentally inoculated with H5N1 virus, and cats kept in close contact with them in trials in the Netherlands (Kuiken et al. 2004; Rimmelzwaan et al. 2006). Systemic replication of H5N1 virus was demonstrated in cats experimentally infected by the different routes, which included intra-tracheal inoculation, the feeding of virus-infected chicks, and horizontal transmission (Rimmelzwaan et al. 2006). Fatal H5N1 infection has also been reported in captive civets in Vietnam (ProMED-mail 2005b), in a stone marten in Europe (ProMED-mail 2006) and in a dog in Thailand (Songserm et al. 2006).

There were 18 outbreaks of HPNAI reported in the English language scientific literature between 1955 and 2000 (Swayne and Suarez 2000), and, in 2003, outbreaks of H7N7 HPNAI were reported in the Netherlands, Belgium and Germany (Shane 2003). In 2003–06, outbreaks of H5N1 HPNAI of unprecedented magnitude were confirmed in 9 countries across East Asia (Japan, South Korea, Vietnam, Thailand, Cambodia, Laos, Indonesia, China and Malaysia). From July 2005 to May 2007, further outbreaks in poultry or wild birds were recorded in Russia, Mongolia, Kazakhstan, Ukraine, Turkey, India, Pakistan, Bangladesh, the Middle East, Europe, and Africa. Reports indicate that over 150 million birds have died or been culled in Asia in attempts to control the spread of the disease (FAO/OIE/WHO 2005a). HPNAI (H7) outbreaks in poultry were also reported in the United States (Texas), Canada, and Pakistan in 2004, and an outbreak of HP H5N2 infection occurred in ostriches in South Africa in 2005 (Alexander 2007). LPNAI viruses were isolated from poultry in the Netherlands (H7N3 in 2002; H7N7 in 2006), Italy (H7N3 in 2002–03; H5N2 in 2005), Denmark (H5N7 in 2003; H5N3 in 2006), France (H5N2 in 2003), Taiwan (H5N2 in 2004), Japan (H5N2 2005), the United States (2007), and the United Kingdom (H7N2 in 2007) (Alexander 2007; World

Organisation for Animal Health (OIE) 2006; World Organisation for Animal Health (OIE) 2007b).

Outbreaks of HPNAI caused by H7 subtype occurred in Australia in 1976, 1985, 1992, 1995 and 1997 (Swayne and Suarez 2000). In each instance, the virus was believed to have originated from wild water birds, with direct contact between birds and poultry, or faecal contamination of a water source, being implicated in the outbreak of disease in poultry (Morgan and Kelly 1990; Animal Health Australia 1995; Selleck et al. 1997; Animal Health Australia 1998). However, to date, there have been no reports of H7 virus being isolated from wild waterfowl in Australia (Arzey 2004; Tracey et al. 2004). In the 1985 outbreak in Victoria, chickens showed mild clinical signs (10–20% reduction in egg production and mild respiratory disease) for several weeks before a sudden increase in death rate occurred (30–80% in different sheds on the same farm). This suggests that the birds were initially infected with a LP virus that became HP during circulation in poultry. The 1997 outbreak in New South Wales was attributed to faecal contamination of river water by wild birds, with aerosol and mechanical spread occurring between the first infected farm and two other nearby farms. Mortality rate in affected sheds on the first farm varied from 40% to 100% (Animal Health Australia 1998). Australia is considered free of HPNAI, and there is no evidence of LPNAI in Australia's commercial poultry flock.

In Hong Kong in 1997, the H5N1 virus crossed the species barrier, with 18 people developing clinical symptoms of infection, of whom six died. In 2003, a Hong Kong resident died from complications after infection with H5N1 AI virus (World Health Organization 2003). Between 28 January 2004 and 16 May 2007, a total of 306 confirmed human cases of AI (H5N1) infection were reported in twelve countries, resulting in 185 deaths (World Health Organization 2007). Human cases in the 2003–2007 outbreaks have mostly been linked to direct contact with diseased or dead poultry in rural areas (ProMED-mail 2005a; FAO/OIE/WHO 2005b). Transmission to humans in the 1997 Hong Kong outbreak was thought to be caused by exposure to high levels of viral aerosols originating from respiratory secretions and faeces of poultry rather than the preparation or consumption of poultry meat products (Mounts et al. 1999).

A number of workers associated with the control of HPNAI outbreaks in the Netherlands (H7N7) in 2003 and in Canada (H7N3) in 2004 developed conjunctivitis (Koopmans et al. 2003; ProMED-mail 2004b). H7N7 virus was isolated from lung tissue of one veterinarian who died in the Netherlands (Koopmans et al. 2004). A further 88 people were confirmed by PCR to be infected with the virus, including 3 people with no known poultry contact. At least 50% of people exposed to infected poultry had detectable H7 antibodies, and the seroprevalence of H7 antibodies in people without contact with infected poultry, but with close household contact to an infected poultry worker, was 59%. This data suggests that person-to-person transmission of at least some strains of AI virus may occur (Bosman, Meijer, and Koopmans 2005).

Serological evidence of human infection or exposure to LPNAI was recorded during LPNAI H7N3 outbreaks in Italy in 2002–2003. All seropositive tests were in poultry workers having direct physical contact with chickens or turkeys in poultry housing (Puzelli et al. 2005).

Clinical signs

Natural infection by AI viruses results in a wide range of clinical outcomes which depend on virus strain, host species and environmental factors including concomitant infections with bacteria (Swayne and Halvorson 2003). The HP disease in chickens may vary from

characteristic disease with respiratory signs, excessive lacrimation, sinusitis, oedema of the head, cyanosis of the unfeathered skin and diarrhoea to one of sudden death with few overt signs (Alexander 2004). Wild birds affected in recent H5N1 outbreaks have shown signs of neurological disease and diarrhoea (Liu et al. 2005; Hulse-Post et al. 2005).

Highly pathogenic H5N1 virus has been isolated in China from domestic ducks exhibiting no clinical signs of infection (Li et al. 2004; Chen et al. 2004). In 2002, the biology of H5N1 virus in Asia appeared to change, with some ducks exhibiting clinical signs of infection, including neurological signs and mortality (Hulse-Post et al. 2005). Ducks that survived infection shed virus for a prolonged period (up to 17 days). However, recent studies show that some H5N1 virus strains are reverting to non-pathogenicity in ducks, while remaining highly pathogenic to chickens (Hulse-Post et al. 2005).

Other AI viruses, on their own, cause a much milder disease consisting of mild respiratory disease, depression and decreased egg production (Capua and Alexander 2004). Chickens infected with LPAI and LPNAI viruses may show no clinical signs of infection (Dunn et al. 2003).

Pathogenesis

The incubation period may vary from a few hours to three days in individual birds, or up to 14 days in a flock (Swayne and Halvorson 2003). The incubation period depends on the dose of the virus, the route of exposure and the species exposed, as well as ability of the virus to initiate clinical signs. For the purposes of the *Terrestrial Animal Health Code*, the OIE defines the incubation period for NAI as 21 days (World Organisation for Animal Health (OIE) 2007a).

HP strains of AI virus induce viraemia that may persist for several days, with virus remaining detectable by virus isolation from the tissues for 6 to 10 days (Becker and Uys 1967). Viral titres up to 8.0 $\log_{10}$ EID_{50}/g[1] were found in muscle of chickens experimentally inoculated with H5N1 virus (Thomas and Swayne 2007a). Birds other than ducks may shed AI virus from five days to four weeks after infection, while ducks may shed the virus up to 30 days post-infection (Swayne and Halvorson 2003). More recent evidence suggests that H5N1 strains are shed primarily from the upper respiratory tract of ducks (Hulse-Post et al. 2005; Sturm-Ramirez et al. 2004). Duration of shedding of LPNAI virus in experimentally inoculated chickens has varied with age, with five week old chickens shedding virus in the faeces for two weeks, compared with 23 week old birds, shedding virus for one week (Lu and Castro 2004). Meat chickens in a naturally occurring H7N2 LPNAI outbreak in the United States continued to shed virus 13 days after disease onset, as detected by virus isolation from tracheal and cloacal swabs (Lu et al. 2004).

Serological tests may be used to demonstrate the presence of antibodies to AI virus as early as 7–10 days after infection. However, there is considerable variation in the immune response among the various avian species (Swayne and Halvorson 2003).

Infection by HPNAI virus results in systemic replication of the virus and cell death in several organs, including viscera, brain and skin. Thrombocytopaenia occurs and is followed by terminal haemorrhagic diathesis. Non-HP strains replicate locally within the respiratory and alimentary tracts, but disease may be complicated by secondary viral or bacterial infections (Swayne and Suarez 2000). Occasionally, LPAI viruses spread systemically, replicating and

[1] EID – egg infectious dose

causing damage in kidney tubules, pancreas and other organs (Swayne and Halvorson 2003; Horimoto et al. 1995).

Pathology

The lesions produced in association with H5N1 HPAI infections vary, depending on host species, host age and strain of virus (Swayne 2007).

In peracute HPAI infections, death occurs within one to two days, often in the absence of detectable gross lesions. However, a range of congestive, haemorrhagic, transudative and necrotic changes has been described in outbreaks of HPNAI. Gross changes may include oedema of the head, with swollen and congested or haemorrhagic wattle and comb; haemorrhage and congestion may also be visible on the legs. Congestion, haemorrhage and necrotic foci may be present in a variety of internal organs. Histopathological findings may include lymphoid infiltrations and haemorrhages in the heart, spleen, lungs, brain, wattles, liver, kidney and, with some strains of virus, in skeletal muscle (Easterday, Hinshaw, and Halvorson 1997). During the changing of a mildly pathogenic to a HPNAI virus, gross lesions consistent with HPNAI may be seen in some birds, but the mortality rates may be low or similar to those seen with LPAI (Swayne and Halvorson 2003).

Following infection with LP strains, gross changes include inflammation of the respiratory tract, including infraorbital sinuses, trachea, lungs and airsacs. Inflammation may also be seen in the coelomic cavity, intestines and oviduct, and ‘egg peritonitis’ may be present (Swayne and Halvorson 2003). Pancreatic enlargement and haemorrhages and petechial haemorrhages on the heart and caecal tonsils have also been reported (Mutinelli et al. 2003). Pathological changes associated with LPNAI infection may be more severe in turkeys than in chickens (Mutinelli et al. 2003; Swayne and Halvorson 2003).

Immunology

Haemagglutinin (HA) is the major surface antigen that elicits antibody and cell-mediated immunity which protects against death and clinical signs. Protection following vaccination is subtype-specific (Swayne and Halvorson 2003).

The OIE states that the use of live conventional influenza vaccines against any subtype is not recommended (Alexander 2004). Experimental studies have shown that the use of inactivated whole AI virus vaccines, baculovirus-derived AI haemagglutinin vaccine or recombinant fowl poxvirus- or infectious laryngotracheitis-AI haemagglutinin vaccine can protect chickens against multiple strains of H5 subtype of AI viruses. For both NAI and LPAI, vaccination protects against clinical signs and death following challenge with NAI virus, reduces viral shedding and increases resistance to infection; however virus is still able to infect and replicate in clinically healthy, vaccinated birds (Alexander 2004). None of the vaccines eliminate virus shedding altogether; vaccines with <90% homology between the vaccine and challenge viruses did not cause consistent reductions in viral shedding from the respiratory tract (Swayne et al. 2000).

Use of commercially inactivated H5N2 vaccine in chickens was shown to interrupt virus transmission in an outbreak of H5N1 avian influenza in Hong Kong (Ellis et al. 2004b). In that report, inactivated H5N2 vaccine appeared to protect chickens from clinical signs of disease from day 18 after vaccination. Although sample sizes were relatively small, virus was not isolated from subsequent batches of vaccinated birds produced by affected farms. The authors

emphasised that avian influenza vaccination in Hong Kong is used to complement strict biosecurity measures and a comprehensive monitoring and surveillance program (Ellis et al. 2004b). The importance of biosecurity has been further emphasized by a study in Mexico, which demonstrated that Mexican lineage LP H5N2 viruses have undergone antigenic drift away from the vaccine strain (Lee, Senne, and Suarez 2004). LPNAI virus continues to circulate in Mexico despite nine years of use of inactivated and fowl pox-vectored vaccines (Lee, Senne, and Suarez 2004).

The role of vaccination in the control of HPNAI outbreaks remains controversial (Capua and Marangon 2004; FAO 2004). Use of vaccines to control NAI may jeopardise some export markets (ProMED-mail 2004c; Capua and Marangon 2003). Although OIE provisions do not preclude the use of vaccination as a tool in controlling outbreaks, in many countries the use of vaccines to control NAI is not permitted because they may interfere with stamping out control policies (Alexander 2004).

The differentiation of infected from vaccinated animal (DIVA) approach to vaccination is recommended by the FAO and OIE in situations where vaccination is used to control avian influenza in poultry. Some examples of DIVA strategies include: the use of an inactivated vaccine with a heterologous N determinant; the use of unvaccinated sentinel birds within vaccinated flocks, or the use of recombinant vaccines, allowing continued serological surveillance for infection with field virus in vaccinated flocks (Alexander 2004; FAO 2004; Capua and Marangon 2006; FAO 2004). Virological surveillance by antigen detection or PCR can also be used to detect infections in vaccinated flocks. For inactivated vaccines, a test that detects antibodies to the nonstructural virus protein has been described (Tumpey et al. 2005). This system is yet to be validated in the field (Alexander 2004).

Diagnosis

Diagnosis of NAI is based on isolation of virus from clinical swabs or tissue in embryonated chicken eggs (Swayne, Senne, and Beard 1998). Demonstration of HPNAI virus requires inoculation of susceptible chickens and production of ≥ 75% mortality, or demonstration of an IVPI greater than 1.2, or sequencing of the HA cleavage site to identify multiple basic amino acids. For H5 and H7 viruses of low pathogenicity in chickens, the amino acid sequence of the HA cleavage site must be determined. If similar to that observed for other HPNAI isolates, the virus is considered HPNAI. H5 and H7 viruses that do not meet these criteria are identified as LPNAI, while non-H5 and non-H7 isolates that are not virulent for chickens are identified as LPAI (Alexander 2004).

Alternatively, the presence of influenza virus can be confirmed by the use of reverse-transcription polymerase chain reaction (RT-PCR) using nucleoprotein-specific or matrix-specific conserved primers, and the presence of subtype H5 or H7 influenza virus can be confirmed by using H5- or H7-specific primers (Alexander 2004; Spackman et al. 2002; Suarez, Das, and Ellis 2007).

Two tests are used for detection of influenza A-specific antibodies: the agar gel immunodiffusion test (AGID) and the enzyme-linked immunosorbent assay (ELISA). Antibodies can be detected 7–10 days after infection (Swayne and Halvorson 2003). In acute outbreaks of HPNAI, chickens may die before the development of an antibody response (Forsyth, Grix, and Gibson 1993).

Transmission in chicken meat

Virus has been detected in blood, brain, heart, lung, liver, spleen, kidney and skeletal muscle of infected chickens for 5–6 days after experimental inoculation with HP strains of AI viruses (Becker and Uys 1967). Titres of virus in thigh meat of chickens experimentally infected with H5N1 virus were as high as 8.0 $\log_{10}$ EID_{50}/g of muscle (Thomas and Swayne 2007a). Histopathological changes, such as lymphoid infiltration, are detectable in skeletal muscle of birds affected with HPNAI (Easterday, Hinshaw, and Halvorson 1997), although grossly visible lesions may be absent. Virus was re-isolated from the skeletal muscle, bone marrow and other organs of inoculated chickens which died and were allowed to decompose at room temperature for more than 40 days (Vrtiak and Kapitancik 1967). Virus was also recovered for more than 280 days from chilled muscle and bone marrow of chickens inoculated and killed before exhibiting clinical signs of infection (Purchase 1931).

Early research (Purchase 1931) failed to demonstrate transmission of fowl plague virus in chicken meat. Healthy adult chickens did not show signs of disease when fed meat from infected chicken carcasses (Purchase 1931). However, the author acknowledged that the conditions in which the meat was stored were not ideal, and may have influenced the viability of the virus. Under appropriate conditions, AI viruses can remain viable for long periods in skeletal muscle (Vrtiak and Kapitancik 1967).

In a more recent study, 90% of specific pathogen free (SPF) chickens became infected and died after being fed the breast meat of chickens experimentally infected with one strain of HPNAI virus (Swayne and Beck 2005). HPNAI virus infection produced viraemia and systemic infection, with the virus being present in the whole body of the chicken. The work of Swayne and Beck (2005) did appear to suggest that the proper use of vaccines could prevent the presence of HPNAI virus in the meat of vaccinated chickens, but did not appear to take into account the possibility of carcass contamination during processing.

Virus could not be detected in the meat of birds experimentally infected with either of two strains of LPNAI virus (Swayne and Beck 2005). However, LPNAI virus was detected in rinse-water of the coelomic cavity of infected birds, indicating that residual virus may remain in the thoracic region after lung removal (Swayne and Beck 2005). In addition, LPNAI virus is detectable in the faeces of infected birds, and contamination of the carcass with virus during evisceration is likely. Cross-contamination of other carcasses is therefore likely during commercial processing of infected or contaminated carcasses. However, this factor has not been taken into account in some other published risk assessments to date (Zepeda and Salman 2007; Sabirovic, Hall, and Paterson 2005).

The number of LPNAI viruses that have been tested for ability to spread systemically, and therefore be present in muscle, is small. However, LPAI viruses (H10N7; H9N2) have been detected in the carcasses, including meat and bone marrow, of legally imported and smuggled poultry, indicating that LPAI viruses ‘may be introduced into a country through trade of carcasses regardless of the non-systemic nature of this disease’ (Beato et al. 2006; Mase et al. 2006; Kishida et al. 2004).

Highly pathogenic H5N1 avian influenza viruses have been isolated from duck meat imported into South Korea from China (Tumpey et al. 2002) and into Japan from China (Mase et al. 2005). When inoculated intravenously and intranasally into chickens, these isolates caused 100% mortality. In one study, the virus could be re-isolated from multiple organs, including

breast muscle of inoculated chickens, in which viral titres measured up to 5.5 $\log_{10}$ EID_{50}/g ($10^{5.5}$ EID_{50}/g) of tissue (Tumpey et al. 2002).

Tissue experimentally seeded with NAI virus was found to be non-infectious after 10 days of composting (Senne, Panigrahy, and Morgan 1994).

Quarantine significance

NAI is notifiable to the OIE and trade in potentially affected animals or animal products is subject to international controls.

HPNAI is notifiable in all Australian States and Territories. Australia's policy in the event of an outbreak is eradication as detailed in AUSVETPLAN (Animal Health Australia 2006a). The categorisation of HPNAI and LPNAI under the Emergency Animal Disease Response Agreement has been reviewed (Animal Health Australia 2006b). HPNAI is included in category 2 of the Emergency Animal Disease Response Agreement and LPNAI is included in category 3. Diseases in category 2 are emergency animal diseases that 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. This category includes diseases that may have slightly lower national socio-economic consequences, but also have significant public health and/or environmental consequences. Costs associated with category 2 diseases are funded 80% by government and 20% by industry. Diseases in category 3 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. Category 3 diseases have minimal or no effects on human health or the environment and costs are funded 50% by government and 50% by industry (Animal Health Australia 2006b).

There have been five outbreaks of HPNAI reported in Australia, the most recent of which occurred in 1997. This outbreak cost A$4.445 million and involved the destruction of 310,565 chickens, 1.23 million fertile chicken eggs, 261 emu chicks and 147 emu eggs. The flow-on effects to industry were considerable (Selleck et al. 2003). Australia is free of HPNAI, and there is no evidence of LPNAI in the Australian commercial poultry flock. A pilot serological survey for the presence of H5 and H7 antibodies in commercial chicken farms in Australia was conducted in 2006, funded jointly by the Rural Industries Research Development Corporation (RIRDC), the Australian Egg Corporation Limited (AECL) and the Australian Government Department of Agriculture Fisheries and Forestry. This survey showed no evidence of exposure of commercial chicken flocks to NAI viruses (Dubs 2007).

Eradication of HPNAI in Hong Kong in 1997 and the United States in 1983–85 cost US$12 million and US$63 million in government funds, and involved the depopulation of 1.4 million and 17 million birds respectively (Swayne and Suarez 2000). The outbreak in United States is estimated to have cost an additional US$350 million in increased consumer costs (Galyon and Roth 2003), and resulted in a more than 30% increase in retail egg prices (Animal and Plant Health Inspection Service 1995).

In 1999–2000 HPNAI in Italy resulted in the deaths of more than 13 million birds, and interrupted activities of establishments such as hatcheries, feed mills, abattoirs, and processing plants. Disruptions to the marketing system caused unemployment and heavy economic loss both to the poultry industry and the community (Capua et al. 1999). Recent outbreaks of HPNAI in the Netherlands, Belgium and Germany resulted in the culling of over 25 million

commercial birds, disruptions to exports of breeding stock, hatching eggs and chicks, and disruptions to the supply of commercial meat and eggs for the European Union and export markets (Shane 2003). The costs to the Netherlands Government for control of the 2003 HPNAI outbreak were reported to be €270 million, and costs related to industry and trade disruption were estimated at €500 million (Weijtens 2006). HPNAI outbreaks in Asia and Europe in 2003–06 continue to have significant effects on domestic and international trade at the time of writing, with domestic sales of poultry meat declining sharply in many affected countries. Over 150 million birds have been destroyed/culled in Asia alone. It is estimated that total poultry farm losses in Asia in 2004 were in excess of $10 billion (FAO Newsroom 2005).

Outbreaks of LPNAI in poultry can also be costly in terms of production losses, costs of eradication and loss of export markets. An outbreak of LPNAI (H7N2) in Virginia in 2002 is estimated to have cost the poultry industry approximately US$130 million, and resulted in the depopulation of 4.7 million birds (Akey 2003). The United States has experienced trade embargos associated with the presence of LPNAI viruses in the north-eastern United States (Myers, Rhorer, and Clifford 2003).

In some circumstances, some strains of NAI may be transmitted from infected poultry to humans, causing illness and death (World Health Organization 2003; Koopmans et al. 2004; World Health Organization 2004b; Perdue and Swayne 2005). The risk of transmission from poultry to humans is greatest when infected birds have close contact with family members (e.g. entering the family home), multiple species of animals are farmed in the same location, untreated chicken faeces are used as fertilizer, sick or dead birds are inappropriately disposed of, and birds or their meat are marketed in unregulated live bird markets (FAO/OIE/WHO 2005b). The husbandry and marketing systems that have contributed to human infections in the outbreaks in Asia do not occur in Australia, which has a lower likelihood of widespread human contact with infected poultry.

It is expected that personnel working with HPNAI-infected flocks under conditions such as occurred in the Netherlands would use appropriate protective equipment and take recommended precautions against infection (World Health Organization 2004a). Nevertheless, it is acknowledged that compliance with preventive measures amongst poultry workers during culling operations is highly variable, and that transmission of virus to humans in such circumstances may occur (Bosman, Meijer, and Koopmans 2005).

Risk Assessment: Highly Pathogenic Notifiable Avian Influenza Virus

Release assessment

Rel_1: Selection of source flock (between flock prevalence)

For an unrestricted risk estimate, it was assumed that the prevalence is at the highest sustainable level in an endemically infected country or zone (see Method for Risk Assessment). While specific prevalence data is not available, the IRA team considered that outbreaks of HPNAI in domestic chickens in endemically infected countries would not be uncommon events, based on information about outbreaks of H5N1 HPNAI in Asia; therefore, in countries with ongoing outbreaks of disease, the likelihood that a source flock will be infected with HPNAI was assessed as ***moderate***.

Rel_2: Infection detected through flock surveillance and the flock withdrawn from slaughter

In a country where the disease is endemic, if HPNAI virus is introduced to a flock late in the growth cycle, clinical signs may not be evident or the infection recognised before the flock is sent to slaughter. Similarly, a partially immune flock may contain infected birds that are not recognised when the flock is sent to slaughter. The likelihood that an infected flock will be detected through routine flock surveillance and withheld from slaughter was assessed as ***very low*** by the IRA team.

Rel_3: Selection of an infected chicken from an infected flock (within-flock prevalence)

HPNAI is a highly contagious disease, and in an outbreak, multiple birds within a shed are likely to be affected. When an affected flock is sent to slaughter, the likelihood that a selected individual chicken will be infected was assessed as ***high*** by the IRA team.

Rel_4: Background cross-contamination rate

This likelihood represents the background cross-contamination rate during slaughter and processing and applies to cross-contamination with material, infectious or otherwise, from other carcasses, including those tissues or materials in which the disease agent tends to localise. HPNAI is a systemic infection, with virus present in the skin and subcutaneous tissues, skeletal muscle, internal organs and digestive tract. The IRA team considered that if a carcass infected with HPNAI virus were to be processed in accordance with usual practice, the likelihood of a carcass being contaminated with potentially contaminated material from other birds would be ***moderate***.

Rel_5: Likelihood that an uninfected carcass will be contaminated with the disease agent during slaughter and processing

As discussed in the Method for Risk Assessment (page 58, Part B), Rel_4 was used to calculate Rel_{5a} (the likelihood that an uninfected carcass from an infected flock will become contaminated during slaughter and processing) and Rel_{5b} (the likelihood that an uninfected carcass from an uninfected flock will become contaminated during slaughter and processing).

For HPNAI, Rel_{5a} was calculated as ***moderate***, and Rel_{5b} was calculated as ***low***.

Rel_6: The likelihood that the carcass of a bird that was *infected* before slaughter will be removed as a result of inspections before or during processing

Carcasses affected by obvious congestion or necrosis of muscle, skin or internal organs would, most likely, be detected during processing. However, birds in the acute viraemic stages of infection may show no gross lesions. The IRA team considered that the rejection rate of infected carcasses would be greater than the background rejection rate but still very low. The likelihood that a contaminated/infected carcass will be removed during processing inspections was therefore assessed as ***very low.***

Rel_7: The likelihood that the carcass of a bird that was *not infected* before slaughter will be removed as a result of inspections before or during processing

The likelihood that an uncontaminated/uninfected carcass will be removed during processing inspections was considered to be equal to the background rejection rate of 0.75% as described in the Method section (page 59, Part B).

Rel_8: Inactivation of the agent during further processing, storage, handling and transport

HPNAI virus is inactivated at pH <5. However, the pH of chilled or frozen chicken meat is unlikely to fall below 5.5 (Lyon, Hamm, and Thomson 1985). Given that the carcasses for importation will be chilled or frozen, and that the virus can persist for weeks or months at low temperatures, the likelihood of inactivation of the virus during further processing, storage, handling and transport was assessed as ***extremely low*** by the IRA team.

Conclusions – Release assessment

After inserting the above estimates into the simulation model, and using the outputs from the model as a guide, the IRA team concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a ***moderate*** likelihood that imported chicken meat would be infected or contaminated with HPNAI virus.

Exposure assessment

Exposure Group 1: Wild birds

Of the steps identified as determinants in the infection of wild birds by exposure to scraps from an imported contaminated chicken carcass, $WB_{agentsurvival}$ and $WB_{infectivedose}$ are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Methods section (pages 67-86, Part B).

$WB_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by a wild bird

HPNAI virus, protected within chicken meat scraps, is likely to survive in the environment under ambient temperatures of 10 ºC to 35 ºC for several days, giving ample time for wild birds to locate and scavenge the material. This likelihood was assessed as ***high*** by the IRA team**.**

$WB_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a wild bird is sufficient to produce infection

Until relatively recently, infection and establishment of HPNAI in wild birds had rarely been reported, although wild birds do form a reservoir of AI viruses of low pathogenicity, and are frequently believed to be the source of infection with LPNAI viruses in poultry that later mutate to HPNAI virus causing outbreaks of disease. However, some strains of HPNAI have recently been reported to occur in birds known to frequent refuse dumps. Many species of wild birds have been reported to be infected with H5N1 virus, including gulls, which frequent refuse dumps and may have access to discarded chicken meat scraps.

The IRA team recognised that most strains of HPNAI virus cause little or no clinical disease in wild birds, and that the current H5N1 outbreaks are exceptional, suggesting that this likelihood might be very low, or lower. Nevertheless, given the recent spate of reports of HPNAI virus infections in wild birds, the IRA team considered that the likelihood that HPNAI virus would infect a wild bird consuming contaminated meat scraps was ***low***.

Exposure Group 2: Low biosecurity poultry

Of the steps identified as determinants in the infection of low biosecurity poultry by exposure to material from an imported contaminated chicken carcass, $BP_{agentsurvival}$, $BP_{infectivedose}$, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Methods section (pages 67-86, Part B).

$BP_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by low biosecurity poultry

The time between feeding of scraps and consumption by low biosecurity poultry is likely to be very short, so environmental degradation of the disease agent will be minimal. The likelihood that the agent will remain viable was assessed as ***certain (=1)*** by the IRA team.

$BP_{infectivedose}$: The likelihood that an amount of the contaminated chicken waste eaten by a low biosecurity bird is sufficient to produce infection

Transmission of HPNAI to chickens by feeding of infected meat has been documented, and experimental transmission of virus has been demonstrated via the oral route. Four of 5 chickens inoculated orally with $10^{6.6}$ EID_{50} A/Tern/South Africa/61 virus were infected and died 23 to 25 days after inoculation (Becker and Uys 1967). In a more recent study, 90% of specific pathogen free (SPF) chickens became infected and died after being fed the breast meat of chickens experimentally infected with a strain of HPNAI virus (Swayne and Beck 2005). HPNAI virus infection produced viraemia and systemic infection, with the virus being present in the whole body of the chicken. Virus has been isolated from skin, muscle and internal organs for 5–6 days after experimental infection of chickens with HPNAI, and has been shown to persist in carcasses for 30 days or longer at room temperature. The titre of virus in the muscle of chickens inoculated with H5N1 virus reached $10^{8.0}$ EID_{50}/g (Thomas and Swayne 2007a). Given that a chicken can consume up to 150g of feed per day, the IRA team considered that it is highly likely that a sufficient dose of virus would be available to initiate infection. The likelihood that an amount of scrap from an infected chicken carcass would contain a sufficient quantity of virus to infect low biosecurity poultry was assessed as ***high***.

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

The likelihood that HPNAI virus would survive the rendering process was negligible. For HPNAI virus, the IRA team considered that the likelihood that the product will be re-contaminated post-processing was negligible. Therefore, the likelihood that poultry feed derived from an imported contaminated carcass would be contaminated with HPNAI virus was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

Given that HPNAI virus would be destroyed by rendering, and that feed derived from contaminated carcasses would be diluted with feed from non-risk material, the likelihood that the amount of final poultry ration eaten by a bird would contain an oral infectious dose of virus was considered to be ***negligible***.

Exposure Group 3: Medium biosecurity commercial poultry

Of the steps identified as determinants in the infection of medium biosecurity commercial poultry by exposure to material from an imported contaminated chicken carcass, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Methods section (pages 67-86, Part B).

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with HPNAI virus was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that the amount of final poultry ration would contain an oral infectious dose of HPNAI virus was considered to be ***negligible***.

Exposure Group 4: Non-avian species

As discussed above, $NAS_{agentsurvival}$ was considered to be equal to $WB_{agentsurvival}$.

HPNAI has been shown to cross the species barrier but only in exceptional circumstances. Cats and zoo felids have become infected after being fed the carcasses of H5N1-infected birds, and some showed clinical signs of disease (ProMED-mail 2004a; Keawcharoen et al. 2004). However, it was assessed that scraps from imported chicken carcasses of birds not exhibiting clinical signs of HPNAI would present a lower risk to domestic cats than the whole carcasses of infected birds that led to clinical disease in zoo felids and a small number of domestic cats overseas. Whole birds fed to zoo species would be sourced locally; however, it is possible that imported whole carcasses or cuts could be fed to zoo carnivores. Nevertheless, the IRA team considered that the likelihood that meat from an imported infected chicken carcass would contain a sufficient quantity of virus to infect non-avian species ($NAS_{infectivedose}$) was **extremely low**.

Conclusions – Exposure assessment

Using the simulation model as a guide, the partial likelihood of exposure for each of the exposure groups was determined, taking the above estimates of the exposure variables into account. A summary of the outcomes determined by the IRA team is set out in Table 2.

Table 2. Partial likelihoods of exposure (PLE)

Exposure group	PLE
Wild birds	Extremely low
Low biosecurity poultry	Very low
Medium biosecurity poultry	Negligible
Non-avian species	Extremely low

Consequence assessment

Establishment and spread was considered in the context of the outbreak scenarios described in the Method for Risk Assessment.

Estimating the likelihood of each outbreak scenario

The partial likelihood of establishment and spread (PLES) of HPNAI for the different exposure groups is described below. Four outbreak scenarios were considered relevant:

1. Disease agent does not establish or is not recognised within the directly exposed population

2. Disease agent establishes within the directly exposed population, is identified and is eliminated by human efforts or by natural means

3. Disease agent establishes in the directly exposed population, spreads within a district/region, including into other exposure groups if applicable, and is eliminated by human action or by natural means

4. Disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia.

Wild birds

Outbreaks of disease due to HPNAI in wild birds have previously been considered to be uncommon. Moreover, HPNAI viruses are generally well adapted to a particular host species, and transmission occurs most frequently and readily between individuals of the same species. However, infection of numerous species of wild birds with H5N1 virus has been reported in 2005–07. The IRA team considered that, with strains of HPNAI virus other than H5N1, the most likely outcome of infection of a wild bird resulting from scavenging chicken meat scraps would be a single or a few isolated occurrences of infection in wild birds, with the virus being unable to establish ongoing infection in the population. However, wild birds, including gulls and other scavenging species, are susceptible to infection with H5N1 viruses. Infection of wild birds is not likely to be recognised quickly unless there is a large mortality event that attracts investigation. Therefore, amplification of the virus and transmission to other wild birds and other exposure groups may occur. If HPNAI were diagnosed in wild birds, it is unlikely that attempts would be made to eradicate the infection. Overall, after considering HPNAI of H5N1 and other subtypes, the IRA team considered that the likelihood of scenario 1 was **low,** while the likelihood of scenario 2 was **moderate**.

Spread of HPNAI virus from scavenging wild birds to poultry is considered a rare event since HPNAI infections in poultry usually occur after mutation of LPNAI viruses within the poultry

population, rather than via direct spread of HPNAI virus from wild birds. However, direct spread of H5N1 from wild birds to poultry appears to have occurred in a number of countries in 2005–07. The H5N1 strains of virus were used as the model virus for this risk assessment.

If infection were to spread to low or medium biosecurity commercial poultry, there would be a greater likelihood of recognition and implementation of control programs. Therefore, the likelihood of the disease becoming widespread was considered to be ***low***, with a ***very low*** likelihood that it would become endemic (Table 3).

Table 3. Estimated partial likelihood of establishment and spread (PLES) values for HPNAI in wild birds

Outbreak scenario 1:	$PLES_{(wb)}1$	**Low**
Outbreak scenario 2:	$PLES_{(wb)}2$	**Moderate**
Outbreak scenario 3:	$PLES_{(wb)}3$	**Low**
Outbreak scenario 4:	$PLES_{(wb)}4$	**Very low**

Low biosecurity poultry

Although this exposure group includes commercial free-range poultry and ratites, the IRA team considered that the sub-set of this group most likely to be exposed to scraps of imported chicken meat was small flocks of backyard poultry, because these are more likely to be directly fed table scraps. HPNAI virus is highly infectious in poultry, and infection will generally result in high morbidity and mortality rates in the infected flock. In backyard poultry, the mortality rate may be so high as to preclude efficient spread of the disease. The IRA team considered that infected backyard chickens were likely to die before the disease was diagnosed or had an opportunity to spread, and so the virus was considered unlikely to establish ongoing infection in the population. If the disease were to establish in the flock, the level of expertise in disease recognition is likely to be low, and disease investigation may not occur.

In backyard poultry flocks, which generally have relatively few birds, there is less opportunity for the generation of high levels of environmental contamination, than might occur with an outbreak of infectious disease in a large commercial flock, so there is less chance of spread to other flocks or other exposure groups. HPNAI viruses could multiply extensively in backyard ducks, but transmission to such relatively uncommonly kept species was considered unlikely.

The IRA team considered that the most likely outcome of exposure of low biosecurity poultry would be sudden high mortality in a small backyard flock, which might go undiagnosed. However, if the disease was recognised, eradication would be relatively quick due to the small number of birds affected. Therefore, the IRA team considered that Scenarios 1 and 2 were approximately equally likely, while Scenarios 3 and 4 were relatively unlikely to occur, given Australia's history of swift eradication of previous HPNAI outbreaks. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 4).

Medium biosecurity commercial poultry

The IRA team considered that the most feasible route for exposure of medium biosecurity commercial poultry to imported carcass components would be through poultry feed containing

inadequately rendered processing waste. The likelihood that notifiable avian influenza viruses would remain viable following rendering of contaminated imported carcasses and parts was assessed as negligible (page 15, Part C). Nevertheless, the IRA team estimated the PLES values, based on their assessment of the likely outcomes in the improbable event that exposure of medium biosecurity commercial poultry did occur via this route.

Table 4. Estimated partial likelihood of establishment and spread (PLES) values for HPNAI in low biosecurity poultry

Outbreak scenario 1:	$PLES_{(bp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(bp)}2$	**Moderate**
Outbreak scenario 3:	$PLES_{(bp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(bp)}4$	**Extremely low**

HPNAI virus is highly infectious, and spread within an exposed flock of medium biosecurity commercial poultry will be rapid due to the large number of susceptible birds kept in close contact, leading to a high level of environmental contamination. However, higher levels of management expertise should ensure that an outbreak would be recognised sooner than in backyard poultry, and higher levels of biosecurity should ensure that spread from the affected flock is also less likely. In view of these factors, outbreak scenario 2 (a recognised outbreak contained within the directly exposed population) was considered the most likely. However, movement of affected birds or contaminated fomites may occur before the infection is recognised, leading to infection becoming more widespread before it is eradicated (scenario 3). In Australia, HPNAI outbreaks have historically been controlled while remaining localised, so scenario 4 was considered extremely unlikely.

The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 5).

Table 5. Estimated partial likelihood of establishment and spread (PLES) values for HPNAI in medium biosecurity commercial poultry

Outbreak scenario 1:	$PLES_{(cp)}1$	**Very low**
Outbreak scenario 2:	$PLES_{(cp)}2$	**High**
Outbreak scenario 3:	$PLES_{(cp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(cp)}4$	**Extremely low**

Non-avian species

Until recently, HPNAI infection of non-avian species was thought to occur under exceptional circumstances of genetic reassortment combined with close physical contact between species. Reassortment between human and avian influenza viruses has not been reported during the 2003–07 HPNAI outbreaks in Asia and Europe, despite the confirmed infection of over 300 people. However, in 2004, domestic cats and zoo felids were found to be susceptible to infection with H5N1 HPNAI virus after consumption of infected poultry and in 2006, a small number of domestic cats and a stone marten in Europe became infected, presumably after

consuming infected wild birds. Transmission from infected cats to other non-avian species has not been demonstrated, although transmission to other cats has been shown to occur in experimentally infected cats kept in close contact (Kuiken et al. 2004). Outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely for this exposure group. Because transmission of NAI virus between affected non-avian species has not been documented in naturally-occurring infections, the likelihood of the other outbreak scenarios occurring was considered extremely low. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 6).

Table 6. Estimated partial likelihood of establishment and spread (PLES) values for HPNAI in non-avian species

Outbreak scenario 1:	$PLES_{(nas)}1$	**High**
Outbreak scenario 2:	$PLES_{(nas)}2$	**Extremely low**
Outbreak scenario 3:	$PLES_{(nas)}3$	**Extremely low**
Outbreak scenario 4:	$PLES_{(nas)}4$	**Extremely low**

Conclusion – Likelihood assessments

The estimates for the likelihood of release, the partial likelihoods of exposure for each of the exposure groups, and the partial likelihood of establishment and spread for each of the outbreaks scenarios were combined with the expected volume of trade using the simulation model. This allowed the calculation of partial annual likelihood of entry, exposure, establishment and spread for each of the outbreak scenarios. The results of this calculation are shown in Table 7.

Estimating the impacts associated with each outbreak scenario

For each outbreak scenario the direct and indirect impacts of HPNAI were estimated at the national, State or Territory, district/region and local levels, as described in the Methods section (pages 90-95, Part B).

Outbreak Scenario 1

By definition, outbreak scenario 1 means that the disease agent does not establish in the initially exposed population, or does not cause sufficient disease to lead to investigation and recognition of the infection. Therefore the impacts will be ***unlikely to be discernible*** for all exposure groups and all criteria.

Table 7. Partial annual likelihood of entry, exposure, establishment and spread (PALEEES) for the outbreak scenarios

Exposure group	Outbreak scenario	PALEEES
Wild birds	Scenario 1	High
	Scenario 2	High
Low biosecurity poultry	Scenario 1	High
	Scenario 2	High
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Extremely low
Non-avian species	Scenario 1	High
	Scenario 2	Very low
Total all exposure groups	Scenario 3	High
Total all exposure groups	Scenario 4	High

Outbreak Scenario 2

The impacts of this outbreak scenario (disease agent establishes within the directly exposed population, and is identified and eliminated) will differ between exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

Since wild birds and susceptible non-avian species do not play a significant part in production, direct economic loss from death of wild birds or non-avian species, if it were to occur, is not measurable. Other impacts from the death of wild birds will be considered under direct criterion 2 and indirect criterion 4. The direct impacts of disease on wild birds were assessed by the IRA team as ***unlikely to be discernible*** at all levels. In countries where non-avian species have been affected by HPNAI virus, the effects were limited to the deaths of individual felids that consumed infected avian carcasses. The impacts of such an event occurring in Australia are considered by the IRA team to be ***unlikely to be discernible***.

An outbreak of HPNAI, contained within a local population of poultry, will result in losses to individual owners. Significant mortalities, decreased egg production and meat production may occur. The impacts of a disease outbreak in a local population of low biosecurity poultry were assessed as ***unlikely to be discernible*** at national, State/Territory, or district/region levels but ***minor*** at the local level, especially if commercial free-range poultry were involved. Impacts of an outbreak of HPNAI contained within a local population of medium biosecurity commercial poultry were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory, or district/region levels but ***minor*** at the local level. Costs related to the deaths of birds are

accounted for under this criterion, but costs arising from the implementation and administration of the eradication program will be accounted for under indirect criterion 1.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

LPAI viruses are widespread in wild birds, but HPNAI viruses have rarely been isolated from these species, even during outbreaks in domestic poultry. H5N1 viruses are the exception, with deaths in many species of wild birds being associated with H5N1 infection in 2005–07. If an outbreak of HPNAI in wild birds was to result in illness and deaths, the direct impacts on the environment were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory, or district/region levels but may be ***minor*** at the local level.

The impacts on the environment of an outbreak of HPNAI contained within a local population of low biosecurity poultry or medium biosecurity commercial poultry were assessed as ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak of HPNAI contained within a local population of non-avian species on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

If an outbreak of HPNAI were diagnosed in wild birds, there would be an increase in surveillance and monitoring of the local wild bird population, and surveillance of commercial and low biosecurity poultry. A diagnosed HPNAI infection in non-avian species would also lead to increased surveillance and monitoring of the local wild bird population, as non-avian species are most likely to be infected by consuming infected birds. Impacts at national, State/Territory, or district/region levels were assessed by the IRA team as ***unlikely to be discernible***, while impacts at the local level were assessed as ***minor***.

The planned Australian response to a diagnosis of HPNAI in poultry flocks is detailed in AUSVETPLAN (Animal Health Australia 2006a). Affected flocks would be destroyed, quarantine and movement controls instigated, and increased surveillance and monitoring of the low and medium biosecurity poultry populations implemented. Control programs could lead to disruption in breeding and production programs. Impacts of a localised outbreak of HPNAI diagnosed in low biosecurity poultry, especially if involving a commercial free-range flock, were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels. Impacts at the district/region level were assessed as ***minor***. Similarly, impacts of an outbreak diagnosed in medium biosecurity commercial poultry were assessed as ***unlikely to be discernible*** at national and State/Territory levels, but ***minor*** at the district/region level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

An outbreak of HPNAI in any exposure group will have impacts on domestic trade. In many cases, sales of poultry meat have declined sharply in response to H5N1 outbreaks overseas, even in countries with outbreaks confined to wild birds. The impact of a localised outbreak of HPNAI in wild birds or non-avian species may occur as a result of public over-reaction to the

possible human health risk. Sales of poultry meat would be expected to decline nationally, with an effect on the entire poultry industry. It is considered that this effect would be of short duration and was assessed by the IRA team as ***unlikely to be discernible*** at national level, but ***minor*** at State/Territory level.

The impacts of an outbreak of HPNAI in a local population of low biosecurity poultry or medium biosecurity commercial poultry will result in financial losses in the domestic poultry industry, and in associated sales and service industries. Sales of poultry meat would be expected to decline nationally, with an effect on the entire poultry industry. These impacts were assessed by the IRA team as ***minor*** at national level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

HPNAI is an OIE-listed disease, and international markets for poultry and poultry products, and other live birds or avian products are likely to be adversely affected if an outbreak was diagnosed in Australia. However Australia's exports are relatively small. The impacts of an outbreak of HPNAI within the wild bird, low biosecurity poultry or medium biosecurity commercial poultry populations or in non-avian species would be similar. Impacts were assessed by the IRA team as ***significant*** at district/region level and ***minor*** at State/Territory level, because of at least temporary loss of markets, but recognising the relatively small percentage of annual poultry production which is exported.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of a disease outbreak in the local wild bird population were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, and ***minor*** at the local level.

The impact of an outbreak of HPNAI confined to low biosecurity poultry or medium biosecurity commercial poultry or non-avian species were assessed by the IRA team as ***unlikely to be discernible*** at any level.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Restrictions on movement of birds, eggs, poultry products and people may lead to temporary community disruption. Community activities such as shows or pigeon races would be suspended, and destruction of pet birds in affected areas could result in significant levels of community concern and individual emotional distress. Community concern about the possible transmission of the virus to people could be considerable. The impacts of a disease outbreak in the local wild bird population, low biosecurity poultry or medium biosecurity commercial poultry populations or in non-avian species on this criterion would be similar and were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, but ***minor*** at the district/region level.

Outbreak Scenario 3

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a district/region, and is eliminated) will be the same for all exposure groups, no matter which exposure group has been directly exposed to HPNAI virus in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

Australia has adopted a stamping out policy in relation to outbreaks of HPNAI, and all birds on infected properties would be destroyed. Large numbers of birds may be involved on commercial properties, and the birds may represent the owner's sole source of income. The direct costs of dead birds will be larger for commercial enterprises than for low biosecurity or aviary birds, but with prompt eradication, the costs will be limited to the local area. While the impacts at national and State/Territory levels were assessed by the IRA team as ***unlikely to be discernible***, there may be ***minor*** impacts at the district/region level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

Some strains of HPNAI (e.g. H5N1) may cause illness and deaths in wild birds. However, the number of birds affected is expected to be relatively small, as illustrated by the small proportions of wild bird populations found to be clinically affected in H5N1 outbreaks in Asia. The impacts of an outbreak of HPNAI on the environment were assessed by the IRA team as ***unlikely to be discernible*** at national and State/territory levels but, if wild birds are infected, are considered to be ***minor*** at the district/region level.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

Spread of HPNAI to a local population of poultry or caged birds would require implementation of eradication, surveillance and monitoring programs. The disease is subject to the cost-sharing agreement between governments and industry. While the impact on the national economy was assessed by the IRA team as ***unlikely to be discernible***, impacts at the State/Territory level were assessed as ***minor***.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Depending on the extent of the outbreak, financial losses in the domestic poultry industry, and in associated sales and service industries could be considerable. Sales of poultry meat would be expected to decline nationally, with an effect on the entire poultry industry. Movement controls associated with control and eradication programs would interfere with trading patterns. Impacts were assessed by the IRA team as ***minor*** at the national level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

International markets for poultry and poultry products are likely to be adversely affected if an outbreak of HPNAI was diagnosed in Australia. However, Australia's exports of poultry and poultry products are relatively small. The IRA team considered that an outbreak diagnosed within the wild bird and local poultry population is likely to have ***significant*** impacts at district/region level and ***minor*** at State/Territory level, because of at least temporary loss of markets, but recognising the relatively small percentage of annual poultry production which is exported.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The IRA team considered that a localised outbreak of HPNAI would have ***no discernible impacts*** on the environment at the national or State/Territory levels, but is likely to have ***minor*** impacts at the district/region level.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Movement restrictions, suspension of community activities, destruction of pet birds, or infection of domestic pets in affected areas could result in significant levels of community concern and individual emotional distress. The impacts of control measures would be more widespread than in scenario 2, given that scenario 3 involves spread of the outbreak beyond a single commercial establishment or a local bird population. There would be a heightened level of community awareness of the outbreak, increased media reporting, and increased concern about the transmission of the virus to humans, especially if the outbreak appeared to be spreading rapidly. Such concern may result in some media overstatement and public over-reaction to the human health risks associated with the outbreak, especially if the strain of virus involved were H5N1. In addition to the likely reduction in poultry consumption, accounted for above under 'effects on domestic trade', there may be increases in worker absenteeism and community concern about respiratory illnesses, especially in communities associated with poultry-producing areas or in regions with increased wild bird mortality. There may be an impact on demand for and availability of medical services locally, due to increased demand for consultation and diagnostic testing. The impacts of a disease outbreak on communities were assessed by the IRA team as ***unlikely to be discernible*** at national level, but ***minor*** at the State/Territory level.

Outbreak Scenario 4

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a State/Territory, and becomes endemic in Australia) will be the same for all exposure groups, no matter which exposure group has been directly exposed to HPNAI virus in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

If the disease spreads widely through medium biosecurity commercial poultry and low biosecurity poultry, losses of birds and production may be severe, and on a wider scale than in scenarios 2 and 3. The direct impacts on bird life and health were assessed by the IRA team as ***minor*** at the national level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

With the exception of H5N1 viruses in 2005–07, HPNAI viruses have rarely been isolated from wild birds, even during outbreaks in domestic poultry. The environmental impact of a more general outbreak of HPNAI was assessed by the IRA team as ***unlikely to be discernible*** at the national and State/Territory levels, but may be ***minor*** at the district/region level.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

If HPNAI spreads more widely, there would be significant efforts at the State/Territory, district/region and local levels to eradicate the disease, with intensive monitoring and surveillance programs nationally. Serious consideration would be given to the use of vaccines to control disease outbreaks. The impact at the national level was assessed by the IRA team as ***minor***.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Financial losses in the domestic poultry industry, and in associated sales and service industries could be considerable. Sales of poultry meat would be expected to decline nationally, as has been the case in overseas countries where H5N1 HPAI has occurred, with an effect on the entire poultry industry. Movement controls associated with control and eradication programs would also interfere with trading patterns. The impact of a more general outbreak of HPNAI was assessed by the IRA team as ***minor*** at the national level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

International markets for poultry and poultry products are likely to be adversely affected if an outbreak of HPNAI was diagnosed in Australia. However, Australia's exports of poultry and poultry products are relatively small. Impacts of a more general outbreak were assessed by the IRA team as ***minor*** at State/Territory level, because of at least temporary loss of markets, but recognising the relatively small percentage of annual poultry production which is exported.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The IRA team considered that a generalised outbreak of HPNAI would have ***no discernible impacts*** on the environment at the national or State/Territory levels, but may have ***minor*** impacts at the district/region level.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Movement restrictions, suspension of community activities, destruction of commercial and pet birds, and infection of domestic pets in affected areas could result in significant levels of community concern and emotional distress. The impacts of control measures would be conspicuous over wide areas of the affected States or Territories, given that scenario 4 involves generalised outbreaks resulting in the disease agent becoming endemic in Australia. There would be a heightened level of community awareness of the outbreaks, increased media reporting, and amplified concern about the transmission of the virus to humans, especially if the outbreak appeared to be spreading rapidly. Such concern may result in some media overstatement and public over-reaction to the human health risks associated with the outbreak. This would particularly be so if the strain of virus involved were H5N1. In addition to the likely reduction in poultry consumption, accounted for above under 'effects on domestic trade', there may be increases in worker absenteeism and community concern about respiratory illnesses, especially in communities associated with poultry-producing areas or in regions with increased wild bird mortality. There may be an impact on demand for medical services generally, due to increased demand for consultation and diagnostic testing. The Australian Government may consider implementing the national Influenza Pandemic Prevention and Preparedness Action Plan in the event of any human infections with avian influenza virus (especially H5N1). The impacts on Australian communities of endemic HPAI were assessed by the IRA team as ***significant*** at the national level.

Conclusions – impact assessment

The above estimates for each of the impact criteria were entered into the simulation model, and the overall impact of each outbreak scenario was estimated. Results are shown in Table 8.

Partial annual risk estimate for each outbreak scenario

The partial annual likelihood of entry, exposure, establishment and spread were combined with the impacts of the relevant outbreak scenarios to provide an estimate of the partial annual risk associated with each outbreak scenario. The outcomes of this process are shown in Table 9.

Unrestricted risk estimate

The overall risk associated with the import of whole chicken carcasses was assessed as ***high*** for HPNAI viruses. This unrestricted risk estimate exceeds Australia's ALOP, and therefore, risk management is deemed necessary.

Table 8. Impacts of each outbreak scenario

Exposure group	Outbreak scenario	Impact
Wild birds	Scenario 1	Negligible
	Scenario 2	Low
Low biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Moderate
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Moderate
Non-avian species	Scenario 1	Negligible
	Scenario 2	Low
	Total Scenario 3	Moderate
	Total Scenario 4	High

Table 9. Partial annual risk (PAR) of each outbreak scenario

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Low	Low
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Moderate	Moderate
Medium biosecurity poultry	Scenario 1	Negligible	Negligible	Negligible
	Scenario 2	Extremely low	Moderate	Negligible
Non-avian species	Scenario 1	High	Negligible	Negligible
	Scenario 2	Very low	Low	Negligible
	Total Scenario 3	High	Moderate	Moderate
	Total Scenario 4	High	High	High

Direct impact on human life or health

Under some circumstances, AI viruses may cause illness and death in humans. Between 28 January 2004 and 16 May 2007, a total of 306 confirmed human cases of AI (H5N1) infection were reported in twelve countries resulting in 185 deaths *(World Health Organization 2007).* The majority of those deaths were in people who had been closely associated with village or backyard poultry kept under conditions of minimal biosecurity, and with the birds or their products being consumed locally (FAO/OIE/WHO 2005b). No sustained transmission of HPNAI infection between humans has yet been proven, although possible transmission of H5N1 virus may have occurred between family members in close contact in Thailand

(ProMED-mail 2004d), and evidence for person to person transmission was found during outbreaks of H7N7 avian influenza in poultry in the Netherlands in 2003 (Koopmans et al. 2004). Infection of humans with LPNAI virus has also been documented (Perdue and Swayne 2005; Health Protection Agency (United Kingdom) 2007). If outbreaks of avian influenza were to occur in Australia, personnel culling infected poultry will be required to use protective equipment, and may be offered antiviral medication.

Risk Assessment: Low Pathogenicity notifiable Avian Influenza Virus (H5 and H7)

Release assessment

Rel_1: Selection of source flock (between flock prevalence)

For an unrestricted risk estimate, it was assumed that the prevalence is at the highest sustainable level in an endemically infected country or zone (see Method for Risk Assessment). The IRA team considered that outbreaks of LPNAI in domestic chickens in endemically infected countries would not be uncommon events. However, such infections would be less likely to spread extensively in a short period of time; therefore, in countries with ongoing outbreaks of infection, the likelihood that a source flock will be infected with LPNAI was assessed as ***low*** by the IRA team.

Rel_2: Infection detected through flock surveillance and the flock withdrawn from slaughter

In a country where infection is endemic, if LPNAI virus is introduced to a flock late in the growth cycle, clinical signs may not be evident or the infection recognised before the flock is sent to slaughter. Similarly, a partially immune flock may contain infected birds that are not recognised when the flock is sent to slaughter. The likelihood that an infected flock will be detected through routine flock surveillance, and withheld from slaughter was assessed as ***very low*** by the IRA team.

Rel_3: Selection of an infected chicken from an infected flock (within-flock prevalence)

Spread of LPNAI virus within a flock is unlikely to be as rapid as for HPNAI virus, and recovered birds will develop immunity and cease shedding virus. When an affected flock is sent to slaughter, the likelihood that a selected individual chicken will be infected was assessed as ***moderate*** by the IRA team.

Rel_4: Background cross-contamination rate

This likelihood represents the background cross-contamination rate during slaughter and processing and applies to cross-contamination with material, infectious or otherwise, from other carcasses, including those tissues or materials in which the disease agent tends to localise. LPNAI infection tends to be localised to the respiratory and gastrointestinal systems, and possibly the reproductive tract. The IRA team considered that if a carcass infected with LPNAI virus were to be processed in accordance with usual practice, the likelihood of a carcass being contaminated with potentially contaminated material from other birds was ***low***.

Rel_5: Likelihood that an uninfected carcass will be contaminated with the disease agent during slaughter and processing

As discussed in the Method for Risk Assessment (page 58, Part B), Rel_4 was used to calculate Rel_{5a} (the likelihood that an uninfected carcass from an infected flock will become

contaminated during slaughter and processing) and Rel_{5b} (the likelihood that an uninfected carcass from an uninfected flock will become contaminated during slaughter and processing).

For LPNAI, Rel_{5a} was calculated as ***low***, and Rel_{5b} was calculated as ***very low***.

Rel_6: The likelihood that the carcass of a bird that was *infected* before slaughter will be removed as a result of inspections before or during processing

Carcasses showing obvious signs of airsacculitis or pneumonia may be removed during processing inspections. The IRA team considered that the rejection rate of infected carcasses would be greater than the background rejection rate but still very low. The likelihood that a contaminated/infected carcass will be removed during processing inspections was therefore assessed as ***very low*** by the IRA team**.**

Rel_7: The likelihood that the carcass of a bird that was *not infected* before slaughter will be removed as a result of inspections before or during processing

The likelihood that an uncontaminated/uninfected carcass will be removed during processing inspections was considered to be equal to the background rejection rate of 0.75% as described in the Method section (page 59, Part B).

Rel_8: Inactivation of the agent during further processing, storage, handling and transport

While AI virus is inactivated at pH <5, the pH of chilled or frozen chicken meat is unlikely to fall below 5.5 (Lyon, Hamm, and Thomson 1985). Given that the carcasses for importation will be chilled or frozen, and that the virus can persist for weeks at low temperatures, the likelihood of inactivation of the virus during further processing, storage, handling and transport was assessed as ***extremely low*** by the IRA team.

Conclusions – Release assessment

After inserting the above estimates into the simulation model, and using the outputs from the model as a guide, the IRA team concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a ***low*** likelihood that imported chicken meat would be infected or contaminated with LPNAI virus.

Exposure assessment

Exposure Group 1: Wild birds

Of the steps identified as determinants in the infection of wild birds by exposure to scraps from an imported contaminated chicken carcass, $WB_{agentsurvival}$ and $WB_{infectivedose}$ are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Methods section (pages 67-86, Part B).

$WB_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by a wild bird

In contrast to HPNAI virus, which may be present within skeletal muscle and skin, LPNAI virus is likely to be present only on the surface of chicken carcasses. At ambient temperatures of 10 ºC to 35 ºC the likelihood that LPNAI virus would remain viable on chicken meat scraps was assessed as ***low*** by the IRA team**.**

$WB_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a wild bird is sufficient to produce infection

LPNAI viruses of the H5 and H7 subtypes have not frequently been reported to occur in birds known to visit refuse dumps. However, wild birds do form a reservoir of AI viruses of low pathogenicity, and are frequently believed to be the source of infection with LPAI virus, which later mutates to HPNAI virus causing outbreaks of disease in poultry. Gulls, which frequent refuse dumps, could be susceptible to infection with some strains of LPNAI virus.

The IRA team considered that there was an ***extremely low*** likelihood that LPNAI virus would infect a wild bird consuming the contaminated meat scraps.

Exposure Group 2: Low biosecurity poultry

Of the steps identified as determinants in the infection of low biosecurity poultry by exposure to material from an imported contaminated chicken carcass, $BP_{agentsurvival}$, $BP_{infectivedose}$, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Methods section (pages 67-86, Part B).

$BP_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by low biosecurity poultry

The time between feeding of scraps and consumption by low biosecurity poultry is likely to be very short, so environmental degradation of the disease agent will be minimal. The likelihood that the agent will remain viable was assessed as ***certain (=1)*** by the IRA team.

$BP_{infectivedose}$: The likelihood that an amount of the contaminated chicken waste eaten by a low biosecurity bird is sufficient to produce infection

Transmission of LPNAI to chickens by feeding of infected meat has not been documented although experimental transmission of AI virus has been demonstrated via the oral route. LPNAI virus would be present on the carcass as surface contamination only. Given that a chicken can consume up to 150g of feed per day, the IRA team considered that it is moderately likely that a sufficient dose of virus would be available to initiate infection. The likelihood that an amount of waste from an infected chicken carcass would contain a sufficient quantity of virus to infect low biosecurity poultry was assessed as ***moderate*** by the IRA team.

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

The likelihood that AI virus would survive the rendering process was negligible. For LPNAI virus, the IRA team considered that the likelihood that the product will be re-contaminated

post-processing was negligible. Therefore, the likelihood that poultry feed derived from an imported contaminated carcass would be contaminated with LPNAI virus was estimated to be ***negligible*** by the IRA team.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

Given that LPNAI virus would be destroyed by rendering, and that feed derived from contaminated carcasses would be diluted with feed from non-risk material, the likelihood that the amount of final poultry ration eaten by a bird would contain an oral infectious dose of virus was considered to be ***negligible*** by the IRA team.

Exposure Group 3: Medium biosecurity commercial poultry

Of the steps identified as determinants in the infection of medium biosecurity commercial poultry by exposure to material from an imported contaminated chicken carcass, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Methods section (pages 67-86, Part B).

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with LPNAI virus was estimated to be ***negligible*** by the IRA team.

INFECTDOSEINFEED: The likelihood that the amount of contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that the amount of final poultry ration would contain an oral infectious dose of LPNAI virus was considered by the IRA team to be ***negligible***.

Exposure Group 4: Non-avian species

As discussed above, $NAS_{agentsurvival}$ was considered to be equal to $WB_{agentsurvival}$. Infection of non-avian species with LPNAI viruses has not been widely reported. The likelihood that meat from an infected chicken carcass would contain a sufficient quantity of virus to infect non-avian species ($NAS_{infectivedose}$) was assessed as ***extremely low*** under Australian conditions.

Conclusions – Exposure assessment

Using the simulation model as a guide, the partial likelihood of exposure for each of the exposure groups was determined, taking the above estimates of the exposure variables into account. A summary of the outcomes determined by the IRA team is set out in Table 10.

Table 10. Partial likelihoods of exposure (PLE)

Exposure group	PLE
Wild birds	Negligible
Low biosecurity poultry	Very low
Medium biosecurity poultry	Negligible
Non-avian species	Negligible

Consequence assessment

Establishment and spread was considered in the context of the outbreak scenarios, described in the Method for Risk Assessment.

Estimating the likelihood of each outbreak scenario

The partial likelihood of establishment and spread (PLES) of LPNAI for the different exposure groups is described below. Four outbreak scenarios were considered relevant:

1. Disease agent does not establish or is not recognised within the directly exposed population

2. Disease agent establishes within the directly exposed population, is identified and is eliminated by human efforts or by natural means

3. Disease agent establishes in the directly exposed population, spreads within a district/region, including into other exposure groups if applicable, and is eliminated by human action or by natural means

4. Disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia.

Wild birds

There are few reports of H5 and H7 LPNAI infections in scavenging bird species such as gulls. In most cases, avian species infected with LPNAI viruses show no obvious clinical signs of disease. The IRA team considered that the most likely outcome of infection of a wild bird resulting from scavenging chicken meat scraps would be a single or a few isolated occurrences of infection in wild birds.

Spread of LPNAI virus from scavenging wild birds to poultry may occur in the absence of suitable biosecurity. The wild birds considered likely to be the source of LPNAI virus for poultry are waterbirds, especially ducks, although these are considered relatively unlikely to gain access to discarded chicken meat scraps, compared with more common scavenging species. In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. If LPNAI were to spread to commercial poultry it would be subject to eradication according to the AUSVETPLAN (Animal Health Australia 2006a). The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 11).

Table 11. Estimated partial likelihood of establishment and spread (PLES) values for LPNAI in wild birds

Outbreak scenario 1:	$PLES_{(wb)}1$	**High**
Outbreak scenario 2:	$PLES_{(wb)}2$	**Extremely low**
Outbreak scenario 3:	$PLES_{(wb)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(wb)}4$	**Extremely low**

Low biosecurity poultry

Although this exposure group includes commercial free-range poultry and ratites, the IRA team considered that the sub-set of this group most likely to be exposed to scraps of imported chicken meat was small flocks of backyard poultry, because these are more likely to be directly fed table scraps. The most likely outcome of infection would be a single or a few isolated occurrences of infection, resulting in minimal clinical signs, limited to signs of respiratory disease, a drop in egg production, and low mortality. If the disease were to establish in the flock, the level of expertise in disease recognition is likely to be low, and the limited clinical signs mean that identification of the virus is unlikely. Therefore, since the disease is unlikely to be self-limiting and less likely to cause serious disease so that disease investigation and subsequent diagnosis and eradication efforts are also less likely, there is a greater likelihood that LPNAI would establish and spread more widely to other low biosecurity flocks and potentially to medium biosecurity commercial flocks before it is recognised.

Mechanical transmission of the virus by persons or fomites, and transmission by movement of birds may facilitate spread of the virus beyond the initially infected flock before it is recognised, and eradication measures are implemented. LPNAI viruses could multiply extensively in backyard ducks.

The IRA team therefore considered that the most likely outcome was that the disease was not recognised at all (Scenario 1), but that if it did establish it was most likely to be identified once it had spread to commercial poultry, where LPNAI infection may be recognised as such, or mutation to HPNAI may occur and lead to disease investigation, diagnosis and eradication attempts. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 12).

Table 12. Estimated partial likelihood of establishment and spread (PLES) values for LPNAI in low biosecurity poultry

Outbreak scenario 1:	$PLES_{(bp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(bp)}2$	**Very low**
Outbreak scenario 3:	$PLES_{(bp)}3$	**Moderate**
Outbreak scenario 4:	$PLES_{(bp)}4$	**Extremely low**

Medium biosecurity commercial poultry

The IRA team considered that the most feasible route for exposure of medium biosecurity commercial poultry to imported carcass components would be through poultry feed containing inadequately rendered processing waste. The likelihood that notifiable avian influenza viruses would remain viable following rendering of contaminated imported carcasses and parts was assessed as negligible (page 33, Part C). Nevertheless, the IRA team estimated the PLES values, based on their assessment of the likely outcomes in the improbable event that exposure of medium biosecurity commercial poultry did occur via this route.

Infection of commercial chickens or turkeys with LPNAI virus will generally be associated with clinical signs of respiratory disease and/or drops in egg production. Investigation of such disease syndromes will lead to isolation of virus and diagnosis of H5 or H7 infection. Higher levels of management expertise should ensure that an outbreak would be recognised sooner than in backyard poultry, and higher levels of biosecurity should ensure that spread from the affected flock is also less likely. If LPNAI were recognised in commercial poultry, it would be subject to eradication according to the AUSVETPLAN (Animal Health Australia 2006a). In view of these factors, outbreak scenario 2 (a recognised outbreak contained within the directly exposed population) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 13).

Table 13. Estimated partial likelihood of establishment and spread (PLES) values for LPNAI in medium biosecurity commercial poultry

Outbreak scenario 1:	$PLES_{(cp)}1$	**Very low**
Outbreak scenario 2:	$PLES_{(cp)}2$	**High**
Outbreak scenario 3:	$PLES_{(cp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(cp)}4$	**Extremely low**

Non-avian species

Infection of non-avian species with LPNAI viruses has not been widely reported. Outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely for this exposure group. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 14).

Table 14. Estimated partial likelihood of establishment and spread (PLES) values for LPNAI in non-avian species

Outbreak scenario 1:	$PLES_{(nas)}1$	**High**
Outbreak scenario 2:	$PLES_{(nas)}2$	**Extremely low**
Outbreak scenario 3:	$PLES_{(nas)}3$	**Extremely low**
Outbreak scenario 4:	$PLES_{(nas)}4$	**Extremely low**

Conclusion – Likelihood assessments

The estimates for the likelihood of release, the partial likelihoods of exposure for each of the exposure groups, and the partial likelihood of establishment and spread for each of the outbreaks scenarios were combined with the expected volume of trade using the simulation model. This allowed the calculation of partial annual likelihood of entry, exposure, establishment and spread for each of the outbreak scenarios. The results of this calculation are shown in Table 15.

Table 15. Partial annual likelihood of entry, exposure, establishment and spread (PALEES) for the outbreak scenarios

Exposure group	Outbreak scenario	PALEEES
Wild birds	Scenario 1	High
	Scenario 2	Very low
Low biosecurity poultry	Scenario 1	High
	Scenario 2	High
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	High
	Scenario 2	Very low
Total all exposure groups	Scenario 3	High
Total all exposure groups	Scenario 4	High

Estimating the impacts associated with each outbreak scenario

For each outbreak scenario the direct and indirect impacts of LPNAI were estimated at the national, State or Territory, district/region and local levels, as described in the Methods section (page 90–95, Part B).

Outbreak Scenario 1

By definition, outbreak scenario 1 means that the disease agent does not establish in the initially exposed population, or does not cause sufficient disease to lead to investigation and recognition of the infection. Therefore the impacts will be ***unlikely to be discernible*** for all exposure groups and all criteria.

Outbreak Scenario 2

The impacts of this outbreak scenario (disease agent establishes within the directly exposed population, and is identified and eliminated) will differ between exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

LPNAI infection does not usually lead to clinical signs or death in wild birds. Since wild birds do not play a significant part in production, direct economic loss from death of wild birds, were it to occur, is not measurable. Other impacts from the death of wild birds will be considered under direct criterion 2 and indirect criterion 4. The direct impacts of disease on wild birds were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

An outbreak of LPNAI, contained within a local population of poultry, will result in losses to individual owners. There may be significant morbidity due to respiratory disease; decreased egg production and meat production may occur, and there may be moderate increases in mortalities in some flocks. The impacts of a disease outbreak in a local population of low biosecurity poultry were assessed by the IRA team as ***unlikely to be discernible*** at all levels. Impacts of an outbreak of LPNAI contained within a local population of medium biosecurity commercial poultry were assessed as ***unlikely to be discernible*** at national, State/Territory, or district/region levels but ***minor*** at the local level. With LPNAI infection, the potential exists for HPNAI viruses to emerge by mutation, with a subsequent increase in morbidity and mortality. Costs related to the deaths of birds are accounted for under this criterion, but costs arising from the implementation and administration of the eradication program will be accounted for under indirect criterion 1.

Clinical signs of LPNAI infection in non-avian species have not been reported. The direct impacts of infection on non-avian species were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

While mildly pathogenic AI viruses are widespread in wild birds, H5 and H7 subtypes are only sporadically isolated from these species. If an outbreak of LPNAI in wild birds was to result in illness and deaths, the IRA team considered that these would be sporadic in nature and few in number, and therefore the impacts on the environment were assessed as ***unlikely to be discernible*** at all levels.

The impacts on the environment of an outbreak of LPNAI contained within a local population of low biosecurity poultry or medium biosecurity commercial poultry were assessed by the IRA team as ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak of LPNAI contained within a local population of non-avian species on this criterion were assessed as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

If an outbreak of LPNAI were diagnosed in wild birds or non-avian species, there would be an increase in surveillance and monitoring of the wild bird population, and surveillance of commercial and low biosecurity poultry. Impacts at national, State/Territory, or district/region

levels were assessed by the IRA team as ***unlikely to be discernible***, while impacts at the local level were assessed as ***minor***.

The Australian response to a diagnosis of LPNAI in poultry flocks is detailed in the AUSVETPLAN (Animal Health Australia 2006a). Affected flocks would be destroyed, quarantine and movement controls instigated, and increased surveillance and monitoring of the low and medium biosecurity poultry populations implemented. Control programs could lead to disruption in breeding and production programs. Impacts of an outbreak of LPNAI diagnosed in low biosecurity poultry, especially if involving a commercial free-range flock, were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels. Impacts at the district/region level were assessed as ***minor***. Similarly, impacts of an outbreak diagnosed in medium biosecurity commercial poultry were assessed as ***unlikely to be discernible*** at national and State/Territory levels, but ***minor*** at the district/region level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

The impacts of an outbreak of LPNAI in the local wild bird population or in non-avian species on domestic trade and industry were assessed by the IRA team as ***unlikely to be discernible*** at any level.

The impacts of an outbreak of LPNAI in a local population of low biosecurity poultry will depend on the extent of the outbreak. Financial losses in the domestic poultry industry, and in associated sales and service industries could be considerable. These impacts were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, but ***minor*** at district/region level, especially if commercial free-range poultry were involved. Impacts of an outbreak of LPNAI in a local population of medium biosecurity commercial poultry were assessed as ***unlikely to be discernible*** at national and State/Territory levels, but ***minor*** at the district/region level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

International markets for poultry and poultry products, and other live birds or avian products are likely to be adversely affected if an outbreak of LPNAI was diagnosed in Australia. However Australia's exports are relatively small. The impacts of an outbreak of LPNAI within the wild bird, low biosecurity poultry or medium biosecurity commercial poultry populations or in non-avian species would be similar. Impacts were assessed by the IRA team as ***significant*** at district/region level and ***minor*** at State/Territory level because of at least temporary loss of markets, but recognising the relatively small percentage of annual poultry production which is exported.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of a disease outbreak in the local wild bird population were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, and ***minor*** at the local level.

The impact of an outbreak of LPNAI confined to non-avian species, low biosecurity poultry or medium biosecurity commercial poultry were assessed by the IRA team as ***unlikely to be discernible*** at any level.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Restrictions on movement of birds, eggs, poultry products and people may lead to temporary community disruption. Community activities such as shows, or pigeon races would be suspended, and destruction of pet birds in affected areas could result in significant levels of community concern and individual emotional distress. There may be community concern about the possible transmission of the virus to people. The impacts of a disease outbreak in the local wild bird population, low biosecurity poultry, medium biosecurity commercial poultry or non-avian species on this criterion would be similar and were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, but ***minor*** at the district/region level.

Outbreak Scenario 3

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a district/region, and is eliminated) will be the same for all exposure groups, no matter which exposure group has been directly exposed to LPNAI virus in chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

In accordance with the AUSVETPLAN (Animal Health Australia 2006a), all birds on infected properties would be destroyed or scheduled for early processing. Large numbers of birds may be involved on commercial properties, and the birds may represent the owner's sole source of income. The direct costs of dead birds will be larger for commercial enterprises than for low biosecurity or aviary birds, but with prompt eradication, the costs will be limited to the local area. While the impacts at national and State/Territory levels were assessed by the IRA team as ***unlikely to be discernible***, there may be ***minor*** impacts at the district/region level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

The impacts of an outbreak of LPNAI on the environment were assessed by the IRA team as ***unlikely to be discernible*** at national and State/territory levels but, if wild birds are infected, may be ***minor*** at the district/region level.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

Spread of LPNAI to a local population of poultry or caged birds would require implementation of eradication, surveillance and monitoring programs. While the impact on the national economy was assessed by the IRA team as ***unlikely to be discernible***, impacts at the State/Territory level were assessed as ***minor***.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Depending on the extent of the outbreak, financial losses in the domestic poultry industry, and in associated sales and service industries could be considerable. Movement controls associated with control and eradication programs would also interfere with trading patterns. Impacts were assessed by the IRA team as ***unlikely to be discernible*** nationally, but ***minor*** at the State/Territory level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

International markets for poultry and poultry products are likely to be adversely affected if an outbreak of LPNAI was diagnosed in Australia. However, Australia's exports of poultry and poultry products are relatively small. Impacts were assessed by the IRA team as ***minor*** at State/Territory level, because of at least temporary loss of markets, but recognising the relatively small percentage of annual poultry production which is exported.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The IRA team considered that a localised outbreak of LPNAI would have ***no discernible impacts*** on the environment at the national or State/Territory levels, but is likely to have ***minor*** impacts at the district/region level.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Movement restrictions and suspension of community activities in affected areas could result in significant levels of community concern and individual emotional distress. There may also be community concern about the transmission of the virus to humans. The impacts of a disease outbreak on communities were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, and ***minor*** at the district/region level.

Outbreak Scenario 4

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a State/Territory, and becomes endemic in Australia) will be the same for all exposure groups, no matter which exposure group has been

directly exposed to LPNAI virus in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

If the disease spreads widely through medium biosecurity commercial poultry and low biosecurity poultry, losses of birds and production may be severe. The direct impacts on bird life and health were assessed by the IRA team as ***minor*** at the national level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

The environmental impact of a more general outbreak of LPNAI was assessed by the IRA team as ***unlikely to be discernible*** at the national and State/Territory levels, but may be ***minor*** at the district/region level.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

If LPNAI spreads more widely, there would be significant efforts at the State/Territory, district/region and local levels to eradicate the disease, with intensive monitoring and surveillance programs. Serious consideration would be given to the use of vaccines to control disease outbreaks. The impact at the national level was assessed by the IRA team as ***minor***.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Financial losses in the domestic poultry industry, and in associated sales and service industries could be considerable. Movement restrictions associated with control and eradication programs may also interfere with trading patterns. The impact of a more general outbreak of LPNAI was assessed by the IRA team as ***minor*** at the national level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

International markets for poultry and poultry products are likely to be adversely affected if an outbreak of LPNAI was diagnosed in Australia. However, Australia's exports of poultry and poultry products are relatively small. Impacts were assessed by the IRA team as ***minor*** at State/Territory level, because of at least temporary loss of markets, but recognising the relatively small percentage of annual poultry production which is exported.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The IRA team considered that a generalised outbreak of LPNAI would have ***no discernible impacts*** on the environment at the national or State/Territory levels, but may have ***minor*** impacts at the district/region level.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

For reasons described under scenario 2, and because of the widespread nature of the outbreak, the impact of a general outbreak of LPNAI on communities was assessed by the IRA team as ***unlikely to be discernible*** at the national level, but ***minor*** at the State/Territory level.

Conclusions – impact assessment

The above estimates of impacts for each of the impact criteria were entered into the simulation model, and the overall impact of each outbreak scenario was estimated. Results are shown in Table 16.

Table 16. Impacts of each outbreak scenario

Exposure group	Outbreak scenario	Impact
Wild birds	Scenario 1	Negligible
	Scenario 2	Low
Low biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Low
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Low
Non-avian species	Scenario 1	Negligible
	Scenario 2	Low
	Total Scenario 3	Low
	Total Scenario 4	Moderate

Partial annual risk estimate for each outbreak scenario

The partial annual likelihood of entry, exposure, establishment and spread were combined with the impacts of the relevant outbreak scenarios to provide an estimate of the partial annual risk associated with each outbreak scenario. The outcomes of this process are shown in Table 17.

Table 17. Partial annual risk (PAR) of each outbreak scenario

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	High	Negligible	Negligible
	Scenario 2	Very low	Low	Negligible
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Low	Low
Medium biosecurity poultry	Scenario 1	Negligible	Negligible	Negligible
	Scenario 2	Negligible	Low	Negligible
Non-avian species	Scenario 1	High	Negligible	Negligible
	Scenario 2	Very low	Low	Negligible
	Total Scenario 3	High	Low	Low
	Total Scenario 4	High	Moderate	Moderate

Unrestricted risk estimate

The overall risk associated with the import of whole chicken carcasses was assessed as ***moderate*** for LPNAI virus. This unrestricted risk estimate exceeds Australia's ALOP, and therefore, risk management is deemed necessary.

Direct impact on human life or health

Under some circumstances, AI viruses may cause illness and death in humans. Infection of humans with LPNAI virus has been documented (Perdue and Swayne 2005; Health Protection Agency (United Kingdom) 2007). If outbreaks of avian influenza were to occur in Australia, personnel culling infected poultry will be required to use protective equipment, and may be offered antiviral medication.

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Technical Information

Background

Strains of Newcastle disease (ND) virus vary greatly in their virulence and tissue tropism and, in susceptible birds, infection induces a wide range of clinical signs and pathological lesions. Based on the severity of the disease produced in infected chickens, ND virus (NDV) strains are broadly classified as velogenic (highly virulent), mesogenic (moderately virulent) and lentogenic (mildly virulent) (Alexander 2003).

The natural hosts of NDV are domestic poultry, including chickens, turkeys, ducks, geese, pigeons, quail, pheasants, guinea fowl and ostriches and many species of captive caged birds and wild birds (Alexander 2000). Susceptibility varies between species, with chickens the most likely to show clinical ND, and water birds the least likely to be affected clinically (Kaleta and Baldauf 1988). Infection with NDV has also been reported in reptiles such as crocodiles, snakes and lizards (Kouwenhoven 1993). Human infections with NDV are generally mild and conjunctivitis has been reported in workers in laboratories and poultry processing plants (Chang 1981).

ND is an OIE-listed disease. Although outbreaks have been recorded in Australia in the past, Australia is currently considered free of virulent ND, with vaccination, in accordance with the OIE definition of a ND-free country.

Agent taxonomy

NDV is a member of the genus *Avulavirus* of the family *Paramyxoviridae* (Buchen-Osmond 2002). Using standard serological tests nine distinct serogroups of avian paramyxoviruses have been defined. These are designated avian paramyxovirus-1 (APMV-1) to APMV-9. NDV has been designated APMV-1 (Alexander 2003) and has minor antigenic relationships to some other sero-groups, notably APMV-3 (Alexander 1993; Box, Holmes, and Webb 1988). Using conventional serological tests, APMV-1 is regarded as an homologous group, however, testing using monoclonal antibody panels has shown that strains and isolates may be further divided into groups (Alexander et al. 1997).

For the purposes of trade, control measures and policies, the OIE adopts the following criteria for classifying ND (Alexander 2004):

‘Newcastle disease is defined as an infection of birds caused by a virus of avian paramyxovirus serotype 1 (APMV-1) that meets one of the following criteria for virulence:

The virus has an intracerebral pathogenicity index (ICPI) in day-old chicks *(Gallus gallus)* of 0.7 or greater.

or

Multiple basic amino acids have been demonstrated in the virus (either directly or by deduction) at the C-terminus of the F2 protein and phenylalanine at residue 117, which is the N-terminus of the F1 protein. The term “multiple basic amino acids” refers to at least three arginine or lysine residues between residues 113 and 116. Failure to demonstrate the

characteristic pattern of amino acid residues as described above would require characterisation of the isolated virus by an ICPI test.

In this definition, amino acid residues are numbered from the N-terminus of the amino acid sequence deduced from the nucleotide sequence of the F0 gene, 113-116 corresponds to residues -4 to -1 from the cleavage site.'

Agent characteristics

Strains of NDV vary in their sensitivity to heat inactivation. The degree of inactivation depends also on the initial titre of the virus and the nature of the suspending medium. NDV may survive for months at 8 °C and for several days at 37 °C. Viral infectivity is destroyed at 56 °C in 5 minutes to 6 hours (Beard and Hanson 1984), and 70 °C for 50 seconds (Foster and Thompson 1957). All of the NDV strains tested were inactivated at 100 °C within 60 seconds (Beard and Hanson 1984). Pasteurisation did not eliminate NDV from egg, serum or viral diagnostic agents (King 1991) and NDV could be recovered from liquid egg heated at 64.4 °C for 200 seconds (Gough 1973).

The variable thermostability of NDV is illustrated by the following studies. NDV survival in faeces varies from 8 to 22 days in summer and 26 to 50 days in winter (Frolich, Cortez de Jackel, and Selhorst 1992). At 40 °C to 43 °C and 20–30% humidity, NDV has been reported to remain infective in carcasses for 4 weeks, in water for 5 weeks and for 8 weeks in soil, faeces and poultry feed (Saber et al. 1978). At 37 °C, haemagglutination titres remained unchanged for 392 days (Kreimer 1983). Heating of water up to 80 °C was unreliable for disinfecting virus contaminated water (Mahnel 1977).

In fermented edible waste, the inactivation of NDV appears to be temperature dependent, with more rapid inactivation occurring at higher temperatures (Gilbert et al. 1983). Depending on experimental protocol, NDV has been reported to:

- remain viable for 4 days at 20 °C, 2 days at 30 °C and 1 day at 40 °C (Shotts, Wooley, and Dickens 1984)
- remain viable for 96 hours at 5 °C, 10 °C, 20 °C and 30 °C (Wooley et al. 1981)
- be inactivated within 6–8 days by yeast fermentation and *Lactobacillus* fermentation (Gilbert et al. 1983).

There are conflicting reports on the effectiveness of pasteurisation. One report states that standard pasteurisation is not reliable to inactivate NDV (King 1991) whereas another trial using NDV-inoculated milk demonstrated virus inactivation at 65 °C within 20–40 seconds.

Various strains of NDV have also been reported to survive 56 °C for greater than 1 hour but to be inactivated within 2 hours (Khadzhiev and Hadjiev 1974). Infectivity was halved and haemagglutination titre greatly reduced at 56 °C for 30 minutes and total inactivation at 60 °C for 30 minutes (Kreimer 1983). A 2 log reduction in titre was reported at 56 °C in 60 minutes (Lomniczi 1991).The haemagglutination activity of two strains of NDV (F and CDF-66 strains) was greatly reduced at 56 °C for 30–60 minutes, but this time and temperature had only marginal effect on haemagglutination activity of the K strain (Samuel et al. 1979). The ICPI of the Australian V4 strain was not reduced after heating at 56 °C for 30 minutes and the haemagglutinins of the V4 and various other lentogenic Australian isolates were stable at 56 °C for longer than 60 minutes (Kim and Spradbrow 1978). The above data demonstrates the variability of NDV's thermostability, especially at temperatures of 56 °C or lower.

Inactivation studies on NDV (strain Herts 33/56) in an homogenate composed of chicken muscle with 15% w/w skin and fat were undertaken in 1997 by the Veterinary Laboratories Agency (VLA) in the United Kingdom on behalf of AQIS (Alexander 1997a), and later published (Alexander and Manvell 2004). The D_{10} values (i.e. time required to reduce the infectivity by 90% (i.e. 1 $\log_{10}$) at a specific temperature) calculated from the thermophilic phase of their inactivation curves are as follows (Table 18):

Table 18. D_{10} values for Newcastle disease virus

Temperature	D_{10} (in seconds)
65 °C	120
70 °C	82
74 °C	40
80 °C	29

D_{10} values provide the average titre reduction calculated over a specific time frame. However, they may not adequately reflect the tailing effect frequently encountered with virus inactivation at relatively low temperatures possibly due to factors such as thermophilic populations of the virus, homogeneity and heat penetrability of the medium. In the VLA trial, at 60 °C, the titre was reduced from 6.7 log to 2.7 log within 2 minutes but there was no significant reduction after that. At 65 °C, the titre was rapidly reduced from 7.1 log to about 1.9 log in 1 minute (in allantoic fluid) and 2.5 log in 4 minutes (in meat homogenate) after which the rate of reduction was greatly reduced. A tailing effect was less obvious at 70 °C and above.

Calculations from this experimental work (Alexander 1997a; Alexander and Manvell 2004), have shown that a process equivalent to 8.2 minutes at 70 °C would result in a 6 log reduction of ND virus in homogenized chicken meat with a 15% skin and fat content.

Thermal inactivation studies of NDV in lean infected chicken meat have recently been conducted using a microassay technique (Thomas and Swayne 2007). Quantitative measurements were made of heat sensitivity of two strains of NDV in samples of breast and thigh meat, with all visible skin and fat removed. In this study, a D value of 0.51 seconds at 70 C was estimated. The authors concluded that 'the current USDA-FSIS[1] time-temperature guidelines for cooking chicken meat are sufficient for reducing NDV in chicken muscle tissue to levels that do not represent a health hazard to susceptible poultry' (Thomas and Swayne 2007). The USDA-FSIS cooking guidelines vary with the fat content of the chicken meat. The guidelines were developed to ensure a 7 log reduction in Salmonella in poultry meat for food safety purposes, and were not developed specifically for controlling viruses of quarantine concern.

There are a number of possible explanations for the differences in D values estimated by Alexander and Manvell (2004) and Thomas and Swayne (2007). These are explored in more detail in the chapter on Risk Management.

[1] USDA-FSIS: United States Department of Agriculture-Food Safety and Inspection Service. Guidelines for cooking poultry meat are available at:
http://www.fsis.usda.gov/OPPDE/rdad/FSISNotices/RTE_Poultry_Tables.pdf

Resistance to other physical and chemical agents is as follows (Table 19) (World Organisation for Animal Health (OIE) 2002b; Beard and Hanson 1984; Lancaster 1963a; Lancaster 1963b).

Table 19. Resistance of agent to physical and chemical action

pH:	Readily inactivated by high and low pH (<pH 2 and > pH 11)
Chemicals:	Inactivated by lipid solvents (chloroform, ether, alcohol)
Disinfectants:	Inactivated by a wide range of chemicals, including chlorine-based disinfectants, lipid solvents, formalin, propiolactone and phenol.
Survival:	Inactivated by direct sunlight within 30 minutes, but in cool weather can retain infectivity in faeces and contaminated poultry sheds for up to 21 days. Survives frozen in bone marrow and muscle > 300 days; at 5 °C for >134 days Survives in lake water for up to 19 days; in soil for up to 22 days.

Epidemiology

NDV has a worldwide distribution. However, the assessment of the prevalence of ND is difficult given the widespread use of live ND vaccines, problems with the diagnosis and reporting of ND and the presence of strains of low virulence to chickens in some countries (Alexander 2000).

ND is highly contagious and transmission of virus is frequently by direct contact with diseased or carrier birds. Virus is excreted from the respiratory tract and in the faeces for at least one day before clinical signs become apparent (Sinha, Hanson, and Brandly 1954) and birds can be infected both by inhalation of aerosols or by ingestion. Infection from fomites such as chicken crates, egg flats, contaminated feed, trucks, dust, humans, other animals, feathers and clothing is important in the spread of the disease in an outbreak (Lancaster and Alexander 1975; Alexander 2000). There are conflicting reports of the susceptibility of mammals including cats, foxes, dogs, pigs and rats to infection with NDV but it is probable they act as mechanical carriers only (Lancaster 1963a; Asplin 1949; Lancaster 1963b). Windborne transmission of NDV has been postulated and virus has been recovered from the air up to 64 metres from infected premises (Hugh-Jones et al. 1973). Contaminated vaccines and contaminated water have also been implicated in spread of virus during epizootics (Alexander 2003).

Infection with NDV has been reported in 241 species of birds. Susceptibility varies between species and many may be infected with NDV without showing signs of disease (Kaleta and Baldauf 1988). Psittacine birds may excrete virus in their faeces for more than a year after recovery from clinical disease (Kouwenhoven 1993; Erickson et al. 1977). Wild birds may serve as a significant reservoir of NDV and may introduce the virus to poultry. They may also be responsible for transmitting infection within an area, following the infection of poultry (Alexander 2000; Gilchrist 2005). Several species of introduced and native wild birds found in Australia, have been shown to be susceptible to infection with NDV (Gilchrist 1993) and viruses of low virulence have been isolated from wild birds in Western Australia (Alexander, Mackenzie, and Russell 1986) and Victoria (Peroulis-Kourtis et al. 2002; Peroulis and O'Riley 2004). Sampling of wild bird populations suggests that the virulent NDVs that have been isolated from poultry are antigenically distinct from the avirulent ND strains circulating in Australian wild birds (Gould et al. 2001; Peroulis and O'Riley 2004).

In the 1980s, severe disease resembling neurotropic ND occurred in pigeons in Europe, Japan, Israel and the United States. The disease was caused by a variant of APMV-1 and this virus was later implicated in outbreaks of ND in poultry given feed that had been contaminated by infected pigeons (Alexander et al. 1985; Alexander, Parsons, and Marshall 1984).

Outbreaks of virulent ND occurred in Australia in 1930, 1932, 1998, 1999, 2000 (Westbury 2001), and 2002. Early outbreaks of ND in Australia were believed to be caused by the feeding of wastes containing viscera of infected birds to backyard flocks (Geering, Forman, and Nunn 1995), while recent outbreaks were due to virulent endemic strains of NDV closely related to strains of low virulence that were known to be circulating in poultry for some time (Gould et al. 2001; Westbury 2001). On all occasions, the disease was successfully eradicated by slaughter.

Avirulent and lentogenic strains of NDV have been isolated in a number of countries (McNulty et al. 1988; Durham et al. 1980; Westbury 1981) and some are used as the basis of live ND vaccines for poultry. Avirulent strains of NDV have been endemic in poultry flocks in Australia for some time (Westbury 1981), but the emergence of strains of low virulence associated with mild disease (termed late respiratory syndrome) in the 1990s (Hooper et al. 1999) and, more recently virulent strains, suggests that there has been a gradual evolution in Australian poultry strains of NDV (Westbury 2001). The reason for this change is unknown but selection pressures such as changes in the environment (for instance, intensification of poultry industries) or host characteristics (‘improved’ breeds, altered disease or immune status) may have favoured a shift in the predominant virus type in the population (Westbury 2001).

Clinical signs

Clinical signs alone do not present a reliable basis for diagnosis of ND. Gastrointestinal, respiratory and/or neurological signs have been described, but the clinical presentation of infection with NDV is very variable. Factors such as the virulence and tissue tropism of the virus, the species and age of bird, pre-existing immunity, environmental stress and concurrent diseases influence the outcome of infection with NDV (Alexander 2000).

The mortality rate in susceptible flocks varies between negligible (lentogenic ND) and 100% (velogenic ND) (Alexander 2003). In some circumstances infection with the extremely virulent viruses may result in sudden high mortality with few clinical signs. Immune birds infected with virulent strains of NDV may show greatly diminished clinical signs (Allan, Lancaster, and Toth 1978; Parede and Young 1990).

In laying flocks there may be a decrease in egg production, with depigmentation and loss of shell and albumen quality (Monlux 1972). In recent outbreaks of ND in Australia, a reduction in egg production was the main clinical sign reported in infected layers (Murray 2002a; Murray 2002b).

Pathogenesis

In natural infections, NDV enters through the respiratory or intestinal tracts and replicates locally. The incubation period depends on the dose of the virus, route of exposure and the species exposed. In the chicken, the incubation period ranges from two to 15 days (generally 5–6 days) (Alexander 2003). For the purposes of the *Terrestrial Animal Health Code*, the OIE defines the incubation period for ND as 21 days (World Organisation for Animal Health (OIE) 2007).

Within 22–48 hours of infection, virulent NDV may be found in practically all tissues of the body (Asdell and Hanson 1960; Hofstad 1951; Alexander 1997b). Levels of immunity and the age of the bird may influence the distribution and duration of virus persistence in various tissues; however, NDV may still be present in tissues of infected, immune birds up to 19 days after exposure to virulent virus (Parede and Young 1990). Large amounts of virus are excreted in the faeces during the course of infection, and virus is also excreted in the expired air of infected chickens. A long term carrier state has been postulated for both lentogenic and velogenic NDV (Heuschele and Easterday 1970).

Pathology

The gross lesions of ND reflect the clinical presentation. Congestion and haemorrhage in various organs and tissues are the main findings. Lesions are minimal in lentogenic infections.

Histological lesions in the brain are of value in diagnosis. Moderate to severe non-suppurative encephalitis, perivascular cuffing and neuronal necrosis in the brain stem, cerebellum and/or optic tectum are considered a strong indication of infection with virulent neurotropic NDV (Reece et al. 2000). Necrosis of the endothelial lining of blood vessels, thrombosis, oedema and haemorrhages may be seen in all organs. Oedema and cellular infiltration of the submucosa of the nasal tract and trachea, and of the lungs and airsacs may be present (Alexander 2000; Cheville et al. 1972; Brown, King, and Seal 1999).

Immunology

Live virus and inactivated vaccines are used in the control of ND. The OIE recommends that live vaccines should not have an ICPI exceeding 0.7, and that vaccine master seed virus strains not have an ICPI exceeding 0.4 (Alexander 2004). Commercial live virus vaccines are based on avirulent or lentogenic viruses, such as V4, Hitchner-B_1, La Sota and F; or mesogenic viruses, such as Roakin, Mukteswar and Komarov. The use of mesogenic vaccines to control ND presents a problem since all have two pairs of basic amino acids at the F0 cleavage site and ICPI values of around 1.4. Therefore, infections of birds with these viruses fall within the OIE definition of ND. However, these vaccines are primarily used in countries where ND is endemic (Alexander 2004).

Vaccine virus may be excreted from the cloaca of vaccinated birds for 15-16 days (van Eck, van Wiltenburg, and Jaspers 1991; Tanwani, Sinha, and Malik 1981). Although vaccination protects birds from the clinical effects of virulent ND, it does not always prevent infection with virulent NDV. Virulent virus may still replicate, be excreted and be present in tissues and organs of some clinically healthy vaccinated birds (Asplin 1952). In one study, vaccinated birds continued to excrete virulent NDV for at least 14 days after infection (Westbury, Parsons, and Allan 1984), while in another, virus was isolated from the caecal contents of vaccinated birds up to 19 days after challenge with virulent NDV (Parede and Young 1990; Utterback and Schwartz 1973). Control birds, placed in contact with birds vaccinated five weeks earlier with an oral dose of V4 vaccine, developed antibodies within ten days, indicating that the vaccinated birds, or their environment, contained viable NDV (Spradbrow, Samuel, and Ibrahim 1988).

Diagnosis

Diagnosis of ND relies on isolation and characterisation of NDV. Virus present in suspensions prepared from tracheal and cloacal swabs (or faeces) obtained from live birds, or of faeces and pooled organ samples taken from dead birds, will replicate in the allantoic cavity, yolk sac,

amnion or ectodermal layer of the chorio-allantoic membrane of 9 to 11-day-old embryonating chicken eggs (Alexander 1998). The significance of an isolation of NDV is determined by pathogenicity testing of the isolate (Alexander 2004). *In vivo* pathogenicity tests include the mean death time of embryos injected with the minimum lethal dose of virus, the ICPI or the intravenous pathogenicity index (IVPI). NDV will also replicate in a variety of cell types of mammalian and avian origin including chicken embryo fibroblasts and chicken kidney cells (Alexander 1998).

The detection of NDV by reverse-transcription polymerase chain reaction (RT-PCR) may be more sensitive than detection by virus isolation late in the course of infection when antibody titres are rising (Jorgensen et al. 2006).

In vitro procedures are also used for the determination of pathogenicity. RT-PCR and sequencing of the cleavage site gives a clear indication of the virulence of the virus (Jorgensen et al. 2006). RT-PCR may be carried out on isolated virus or on tissues and faeces from infected birds (Alexander 1998). Panels of mouse monoclonal antibodies have also been used to establish antigenic profiles of NDV isolates (Alexander et al. 1997).

A number of serological tests are available for the diagnosis of ND. At present, the haemagglutination inhibition (HI) test is used most widely (Alexander 2000). APMV-1 may show some antigenic cross-reactions in HI tests with APMV-3 and APMV-7 (Alexander 1997b), but these can be resolved by the use of suitable antigen and antiserum controls.

Transmission in chicken meat

Trade in frozen poultry carcasses is considered to be a source of international spread of NDV. The first recorded outbreak of velogenic ND in Australia in 1930 was believed to be due to the feeding of ships' garbage containing viscera of infected birds to backyard poultry flocks (Geering, Forman, and Nunn 1995). In Great Britain, untreated waste containing poultry offal from imported frozen chicken carcasses, fed to backyard poultry, was responsible for outbreaks of ND (Gordon, Reid, and Asplin 1948; Reid 1961; Asplin 1952). In Switzerland, outbreaks of ND in backyard poultry could be traced to the feeding of offal from frozen poultry and to exposure to thaw fluid from the carcasses (Grausgruber 1973; Grausgruber and Möslinger 1965).

Virus has been demonstrated in the organs, muscles and faeces of vaccinated birds up to six days after challenge with virulent NDV (Guittet et al. 1993). In birds experimentally infected with Herts 33/56 NDV, virus titres in muscle and faeces were about 10^4 EID_{50}/g, and the oral infectious dose of NDV in 3-week-old chickens was 10^4 EID_{50} (Alexander, Manvell, and Parsons 2006; Alexander and Manvell 2004; Alexander 1997a; Alexander 1997c). Viral titres in the meat of 4-week-old SPF chickens, infected with a California strain of virulent Newcastle disease (CA/02), were as high as $10^{6.8}$ EID_{50}/g (Thomas and Swayne 2007).

Since NDV is relatively stable at pH values between 3 and 11, it is unlikely to be affected by pH changes that accompany *rigor mortis*. NDV has been isolated from poultry carcasses frozen for two years, and may survive at –14°C to –20°C on poultry packaging materials for as long as nine months (Michalov and Vrtiak 1966).

Quarantine significance

ND is an OIE-listed disease, and trade in potentially affected animals or animal products is subject to international controls.

ND is notifiable in all Australian States and Territories. Australia's policy in the event of an outbreak is eradication and is detailed in AUSVETPLAN (Animal Health Australia 2004). The Emergency Animal Disease Response Agreement includes ND in category 3. Diseases in this category are considered to be 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. Category 3 diseases cause minimal or no effect on human health or the environment and costs are funded 50% by government and 50% by industry (Animal Health Australia 2006).

A series of outbreaks of ND of endemic origin occurred in Australia between 1998 and 2002 (World Organisation for Animal Health (OIE) 2002a). Following the outbreaks in 2002, a National Management Plan for ND was developed and instigated (Animal Health Australia 2005). The basis for the Plan is compulsory national vaccination of all commercial poultry flocks with structured surveillance and standard operating procedures and programs for virulent NDV. Under the Standard Operating Procedures, meat chickens are vaccinated with V4 at between 1 and 14 days of age. The Plan aims to maintain Australia's virulent ND-free status.

Direct costs of the outbreak at Mangrove Mountain in 1999 to government and industry were A$26.4 million and involved destruction of 1.9 million birds on commercial poultry farms and backyard properties, as well as pet birds. The estimated indirect cost of the outbreak to the poultry industry was A$200 million (NSW Agriculture 2002). Other outbreaks at Blacktown, New South Wales (1998), and Tamworth, New South Wales (1999–2000), cost A$2.8 million and A$0.45 million respectively. Limited outbreaks in Meredith, Victoria, and Horsley Park, New South Wales in 2002, cost industry and government $1.9 million and $0.4 million respectively in shared costs under the Emergency Animal Disease Response Agreement (M. Willoughby, Animal Health Australia, pers. comm. January 2006). Overseas outbreaks of ND have also been costly to eradicate. For example, control of an outbreak of ND in Southern California in 2003 cost over US$100 million and resulted in the slaughter of more than 3 million birds (Shane 2003).

Risk Assessment

Release Assessment

Rel_1: Selection of source flock (between flock prevalence)

For an unrestricted risk estimate, it was assumed that the prevalence is at the highest sustainable level in an endemically infected country or zone (see Method for Risk Assessment). ND is endemic in many countries and most countries rearing poultry commercially rely on vaccination to control the disease. Infected flocks may not necessarily be clinically affected due to the protective effect of vaccination. The likelihood that a source flock would be infected with virulent Newcastle disease virus was assessed by the IRA team as ***moderate***.

Rel_2: Infection detected through flock surveillance and the flock withdrawn from slaughter

In an infected flock, clinical signs may not occur or be recognised by the producer. Where birds are immune to ND due to infection with endemic viruses of low virulence, or due to

vaccination, infection with virulent NDV may be masked. The IRA team considered that the likelihood that an infected flock will be detected through routine flock surveillance, and would subsequently be withheld from slaughter, is ***extremely low***.

Rel_3: Selection of an infected chicken from an infected flock (within flock prevalence)

NDV is readily transmissible, especially among birds kept on litter in large groups. Therefore, many birds within a shed are likely to be affected. Given that birds would be sent to slaughter on a shed or flock basis, if an infected flock is showing clinical signs it is likely that an entire flock would be withheld from slaughter. However, the IRA team assessed that if an infected flock were sent to slaughter, the likelihood that a selected individual chicken will be infected is ***moderate***.

Rel_4: Background cross-contamination rate

This likelihood represents the background cross-contamination rate during slaughter and processing and applies to cross-contamination with material from other carcasses, including those tissue or materials in which the disease agent tends to localise. ND is a systemic infection, with virus present in the skeletal muscle, most tissues and internal organs. The IRA team considered that the likelihood of a carcass being contaminated with potentially infective material from other birds is ***moderate***.

Rel_5: Likelihood that an uninfected carcass will be contaminated with disease agent during slaughter and processing

As discussed in the Method for risk assessment (page 58, Part B), Rel_4 was used to calculate Rel_{5a} (the likelihood that an uninfected carcass from an infected flock will become contaminated during slaughter and processing) and Rel_{5b} (the likelihood that an uninfected carcass from an uninfected flock will become contaminated during slaughter and processing).

For ND, Rel_{5a} was calculated as ***low***, and Rel_{5b} was calculated as ***low***.

Rel_6: The likelihood that the carcass of a bird that was *infected* before slaughter will be removed as a result of inspections before or during processing

In a country with endemic ND, flocks with variable levels of immunity and birds at various stages of the disease will be presented for slaughter. Carcasses showing obvious congestion or haemorrhage would, most likely, be detected and removed during processing. On the other hand, birds in the acute viraemic stages of infection may show no gross lesions. Birds recovering from infection while still shedding virus, and infected birds with partial immunity (e.g. maternally-derived antibodies) may show no clinical manifestations of disease. The IRA team considered that the rejection rate for carcasses would be greater than the background rejection rate but still very low. The likelihood that a contaminated/infected carcass will be removed during processing inspections was therefore assessed as ***very low***.

Rel_7: The likelihood that the carcass of a bird that was *not infected* before slaughter will be removed as a result of inspections before or during processing

The likelihood that an uncontaminated/uninfected carcass will be removed during processing inspections was considered to be equal to the background rejection rate of 0.75% as described in the Method section (page 59, Part B).

Rel_8: Inactivation of the agent during further processing, storage, handling and transport

While NDV is inactivated by pH <2, the pH of chilled or frozen chicken meat is unlikely to fall below 5.5 (Lyon, Hamm, and Thomson 1985). Given that the carcasses for importation will be chilled or frozen, and that the virus can persist for weeks or months at low temperatures, the IRA team considered that the likelihood of inactivation of the virus during further processing, storage, handling and transport is ***extremely low***.

Conclusions – Release assessment

After inserting the above estimates into the simulation model, and using the outputs from the model as a guide, the IRA team concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a ***moderate*** likelihood that imported chicken meat would be infected or contaminated with NDV.

Exposure assessment

Exposure Group 1: Wild birds

Of the steps identified as determinants in the infection of wild birds by exposure to scraps from an imported contaminated chicken carcass, $WB_{agentsurvival}$ and $WB_{infectivedose}$ are pathogen-dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$WB_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by a wild bird

ND virus, protected within chicken meat scraps, is likely to survive in the environment under ambient temperatures of 10 °C to 35 ºC for several days, giving ample time for wild birds to locate and scavenge the material. This likelihood was assessed as ***high*** by the IRA team**.**

$WB_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a wild bird is sufficient to produce infection

NDV infection has been reported in 241 species of birds. Wild birds may act as carriers of NDV, serve as a significant reservoir of viruses of low pathogenicity for chickens, and are frequently believed to be the source of infection during outbreaks of disease in poultry. It is reasonable to assume that birds which frequent refuse dumps would be susceptible to infection with some strains of ND.

Following infection, there is systemic replication of NDV, with persistent viraemia and widespread tissue distribution. Virus has been isolated from muscle and internal organs for up to 19 days after experimental infection of chickens with NDV. The titre of ND in the muscle of

chickens inoculated intranasally with NDV was around $10^{6.8}$ EID_{50}/g (Thomas and Swayne 2007), and the oral infectious dose of NDV in 3-week-old chicks was 10^4 EID_{50} (Alexander, Manvell, and Parsons 2006). Given that a silver gull can consume up to 54g of garbage per day, the likelihood that the level of pathogen present in scraps eaten by wild birds would be sufficient to cause infection was considered ***moderate*** by the IRA team.

Exposure Group 2: Low biosecurity poultry

Of the steps identified as determinants in the infection of low biosecurity poultry by exposure to material from an imported contaminated chicken carcass, $BP_{agentsurvival}$, $BP_{infectivedose}$, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$BP_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by low biosecurity poultry

The time between feeding of scraps and consumption by low biosecurity poultry is likely to be very short, so environmental degradation of the disease agent will be minimal. The likelihood that the agent will remain viable was assessed as ***certain (=1)*** by the IRA team.

$BP_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a low biosecurity bird is sufficient to produce infection

Several outbreaks of ND in backyard chickens due to feeding of wastes from infected birds have been reported (Geering, Forman, and Nunn 1995; Grausgruber 1973; Grausgruber and Möslinger 1965). The titre of ND in the muscle of chickens inoculated intranasally with NDV was around $10^{6.8}$ EID_{50}/g (Thomas and Swayne 2007), and the oral infectious dose of NDV in 3-week-old chicks was 10^4 EID_{50} (Alexander, Manvell, and Parsons 2006). Given that a chicken can consume up to 150g of feed per day, the likelihood that a sufficient dose of virus would be available to initiate infection was considered ***moderate*** by the IRA team.

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

The likelihood that NDV would survive the rendering process was negligible. For NDV the IRA team considered that the likelihood that the product will be re-contaminated post-processing was negligible. Therefore the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with NDV was estimated by the IRA team to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

Given that NDV would be destroyed by rendering, and that even rendered waste contaminated with viable virus would be diluted with non-risk material during the production of poultry feed, the likelihood that the amount of final poultry ration eaten by a bird would contain an oral infectious dose of virus was considered by the IRA team to be ***negligible***.

Exposure Group 3: Medium biosecurity commercial poultry

Of the steps identified as determinants in the infection of medium biosecurity commercial poultry by exposure to material from an imported contaminated chicken carcass, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that poultry feed derived from the imported contaminated carcass, would be contaminated with NDV was estimated by the IRA team to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that the amount of the final poultry ration consumed contained an oral infectious dose of NDV was considered by the IRA team to be ***negligible***.

Exposure Group 4: Non-avian species

As discussed above, $NAS_{agentsurvival}$ was considered to be equal to $WB_{agentsurvival}$.

While cats, foxes, dogs, pigs and rats have been reported to be susceptible to infection with NDV, the IRA team considered it to be most likely that they act as mechanical carriers only.

The likelihood that an amount of scrap from an infected chicken carcass would contain a sufficient quantity of NDV to infect non-avian species ($NAS_{infectivedose}$) was assessed by the IRA team as ***negligible.***

Conclusions – Exposure assessment

Using the simulation model as a guide, the partial likelihood of exposure for each of the exposure groups was determined, taking the above estimates of the exposure variables into account. A summary of the outcomes determined by the IRA team is set out in Table 20.

Table 20. Partial likelihoods of exposure (PLE)

Exposure group	PLE
Wild birds	Very low
Low biosecurity poultry	Very low
Medium biosecurity poultry	Negligible
Non-avian species	Negligible

Consequence assessment

Establishment and spread was considered in the context of the outbreak scenarios described in the Method of Risk Assessment.

Estimating the likelihood of each outbreak scenario

The partial likelihood of establishment and spread (PLES) of NDV for the different exposure groups is described below. Four outbreak scenarios were considered relevant:

1. Disease agent does not establish or is not recognised within the directly exposed population

2. Disease agent establishes within the directly exposed population, is identified and is eliminated by human efforts or by natural means

3. Disease agent establishes in the directly exposed population, spreads within a district/region, including to other exposure groups if applicable, and is eliminated by human action or by natural means

4. Disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia.

Wild birds

Many species of birds found in Australia, including both native and introduced wild species, are susceptible to infection with NDV. There are few reported outbreaks of ND in wild birds worldwide.

NDVs isolated from wild birds in Australia during the 1998–2000 ND outbreaks were genetically different from NDV isolated from poultry (Gould et al. 2001). This suggested that poultry adapted strains were less likely to easily infect wild birds. The IRA team concluded that the most likely outcome of infection of wild birds with NDV derived from scraps of imported chicken meat was that the disease would be self limiting, and that therefore scenario 1 would be most likely in the Australian context. This likelihood was ranked as ***high*** by the IRA team.

The IRA team considered that, if a disease outbreak were restricted to wild birds, it was highly unlikely that the disease would be diagnosed quickly. There is some passive and targeted but no widespread active surveillance of wild birds, and there have been few ND outbreaks reported. The IRA team therefore considered that there was a ***very low*** likelihood of scenario 2 occurring.

However, the IRA team recognised that there was a residual ***low*** likelihood that the disease could spread to other exposure groups. The IRA team considered that the most likely exposure group to be affected by spread from wild birds was low biosecurity poultry. However, the IRA team considered that diagnosis would follow relatively quickly if spread to commercial poultry occurred. Once diagnosed, experience in Australia has shown that implementation of AUSVETPLAN recommendations is effective in eradicating virulent Newcastle disease from poultry in this country. This leads to the conclusion that Scenario 3 is more likely than Scenario 4. The IRA team therefore assigned the following likelihoods to the four outbreak scenarios (Table 21).

Table 21. Estimated partial likelihood of establishment and spread (PLES) values for ND in wild birds

Outbreak scenario 1:	$PLES_{(wb)}1$	**High**
Outbreak scenario 2:	$PLES_{(wb)}2$	**Very low**
Outbreak scenario 3:	$PLES_{(wb)}3$	**Low**
Outbreak scenario 4:	$PLES_{(wb)}4$	**Very low**

Low biosecurity poultry

Although this exposure group includes commercial free-range poultry and ratites, the IRA team considered that the sub-set of this group most likely to be exposed to scraps of imported chicken meat was small flocks of backyard poultry, because these are more likely to be directly fed table scraps. NDV is highly infectious in poultry, and infection will generally result in high morbidity and mortality rates in naive flocks. In backyard poultry, the mortality rate may be so high as to preclude efficient spread of the disease. In such flocks, there is less opportunity for the generation of high levels of environmental contamination than might occur with an outbreak of infectious disease in a large commercial flock. Infected backyard poultry were considered likely to die before the disease was diagnosed, or had an opportunity to spread, and so the virus was considered unlikely to be able to establish ongoing infection in the broader poultry population. The level of expertise in disease recognition in backyard poultry is likely to be low. If, however, the disease was recognised, immediate steps would be taken to control the disease in accordance with AUSVETPLAN. The IRA team therefore concluded that it was equally likely that either Scenario 1 or Scenario 2 would result following exposure of low biosecurity poultry to scraps of imported poultry meat contaminated with Newcastle disease virus.

Mechanical transmission of the virus by contaminated persons or fomites and transmission by movement of birds may facilitate spread of Newcastle disease virus beyond the initially infected flock, before it is recognised and eradication measures implemented. The IRA team considered that, if disease did spread from an infected low biosecurity flock, the most likely exposure group to be affected would be wild birds, which are likely to be closely associated with low biosecurity poultry. If medium biosecurity commercial poultry were to become infected, diagnosis was likely to follow soon after, and eradication programs would be implemented. Experience suggests that eradication of virulent Newcastle disease would follow relatively quickly. Therefore the IRA team considered that, given that disease had spread from low biosecurity poultry to other exposure groups, the next most likely outcome was Scenario 3, followed by Scenario 4. After consideration of all the relevant material, the IRA team assigned the following likelihoods to the four outbreak scenarios (Table 22).

Medium biosecurity commercial poultry

The IRA team considered that the most feasible route for exposure of medium biosecurity commercial poultry to imported carcass components would be through poultry feed containing inadequately rendered processing waste. The likelihood that Newcastle disease virus would remain viable following rendering of contaminated imported carcasses and parts was assessed as negligible (page 66, Part C). Nevertheless, the IRA team estimated the PLES values, based

on their assessment of the likely outcomes in the improbable event that exposure of medium biosecurity commercial poultry did occur via this route.

Table 22. Estimated partial likelihood of establishment and spread (PLES) values for ND in low biosecurity poultry

Outbreak scenario 1:	$PLES_{(bp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(bp)}2$	**Moderate**
Outbreak scenario 3:	$PLES_{(bp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(bp)}4$	**Very low**

Spread of NDV within an exposed flock of medium biosecurity commercial poultry will be rapid due to the large number of susceptible birds kept in close contact, leading to a high level of environmental contamination. However, higher levels of management expertise should ensure that an outbreak would be recognised sooner than in backyard poultry. Widespread vaccination in the Australian poultry population could delay recognition of infection but it was considered likely that identification would be made while still a local infection. Following diagnosis of infection, the emergency animal disease response procedures in AUSVETPLAN would be initiated and, as stated above, experience suggests that eradication of the disease would follow.

In view of these factors, outbreak scenario 2 (disease agent establishes within the directly exposed population, and is identified and eliminated) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 23).

Table 23. Estimated partial likelihood of establishment and spread (PLES) values for ND in medium biosecurity commercial poultry

Outbreak scenario 1:	$PLES_{(cp)}1$	**Very low**
Outbreak scenario 2:	$PLES_{(cp\ )}2$	**High**
Outbreak scenario 3:	$PLES_{(cp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(cp)}4$	**Very low**

Non-Avian Species

There are conflicting reports of the susceptibility of non-avian species to infection with NDV, but it is probable they act as mechanical carriers only. Non-avian species have not been recognised as epidemiologically significant in the spread of ND in documented outbreaks. Outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. The IRA team considered that there was a negligible likelihood that scenario 2, 3 or 4 would eventuate. Therefore, since by definition scenario 1 had no adverse consequences, this exposure group was not considered further in this risk assessment.

Conclusion – Likelihood assessments

The estimates for the likelihood of release, the partial likelihoods of exposure for each of the exposure groups, and the partial likelihood of establishment and spread for each of the outbreaks scenarios were combined with the expected volume of trade using the simulation model. This allowed the calculation of partial annual likelihood of entry, exposure, establishment and spread for each of the outbreak scenarios. The results of this calculation are shown in Table 24.

Table 24. Partial annual likelihood of entry, exposure, establishment and spread (PALEES) for the outbreak scenarios

Exposure group	Outbreak scenario	PALEEES
Wild birds	Scenario 1	High
	Scenario 2	High
Low biosecurity poultry	Scenario 1	High
	Scenario 2	High
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Extremely low
Non-avian species	Scenario 1	Low
	Scenario 2	Negligible
Total all exposure groups	Scenario 3	High
Total all exposure groups	Scenario 4	High

Estimating the impacts associated with each outbreak scenario

For each outbreak scenario the direct and indirect impacts of ND were estimated at the national, State or Territory, district/region and local levels, as described in the Methods section (page 90-95, Part B).

The likelihood of NDV affecting non-avian species (exposure group 4) was considered to be negligible. Therefore, the impacts of ND occurring in this exposure group were not considered further.

Outbreak Scenario 1

By definition, outbreak scenario 1 means that the disease agent does not establish in the initially exposed population, or does not cause sufficient disease to lead to investigation and recognition of the infection. Therefore the impacts will be ***unlikely to be discernible*** for all exposure groups and all criteria.

Outbreak Scenario 2

The impacts of this outbreak scenario (disease agent establishes within the directly exposed population, and is identified and eliminated) will differ between exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

Members of the families *Laridae* and *Corvidae*, to which scavenging species such as gulls, crows and magpies belong, have been shown to be susceptible to infection with NDV. However these, and other wild birds that might come into contact with them, are not production species. Direct economic loss from death of wild birds, were it to occur, is not measurable. Other impacts from the death of wild birds will be considered under direct criterion 2 and indirect criterion 4. The direct impacts of disease on wild birds are ***unlikely to be discernible*** at all levels.

An outbreak of ND, contained within a local population of poultry, will result in losses to individual owners. Significant mortalities, decreased egg production and meat production may occur. The impacts of a disease outbreak in a local population of low biosecurity poultry were assessed as ***unlikely to be discernible*** at national, State/Territory, or district/region level, since the majority of these effects would be felt in smaller backyard flocks. Impacts could be as large as ***minor*** at the local level, especially if large numbers of commercial free-range poultry were present in the area. Impacts of an outbreak of ND contained within a local population of medium biosecurity commercial poultry were assessed as ***unlikely to be discernible*** at national, State/Territory, or district/region levels but would be ***minor*** at the local level. Costs related to the deaths of birds are accounted for under this criterion, but costs arising from the implementation and administration of the eradication program will be accounted for under indirect criterion 1.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

The IRA team considered that the impacts on the environment of an outbreak of ND, resulting in the death of a local population of wild birds, are ***unlikely to be discernible*** at national, State/Territory, or district/region levels but may be ***minor*** at the local level.

The IRA team considered that the impacts of an outbreak of ND contained within a local population of low biosecurity poultry on this criterion are ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak of ND contained within a local population of medium biosecurity commercial poultry on the environment are considered ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

If an outbreak of ND were diagnosed in wild birds, there would be an increase in surveillance and monitoring of the wild bird population, and surveillance of low and medium biosecurity poultry. Impacts were assessed as ***unlikely to be discernible*** at national, State/Territory, or district/region levels but costs of surveillance may lead to ***minor*** impacts at the local level.

The response to diagnosis of virulent ND in poultry flocks is detailed in AUSVETPLAN (Animal Health Australia 2004). Affected flocks would be destroyed, quarantine and movement

controls instigated, and increased surveillance and monitoring of the low and medium biosecurity poultry populations implemented. Control programs could also lead to disruption in breeding and production programs. The impacts of a recognised outbreak of ND in low biosecurity poultry, especially if involving a commercial free-range flock, were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels but ***minor*** at the district/region level. Similarly, the impacts of an outbreak diagnosed in medium biosecurity commercial poultry would be ***unlikely to be discernible*** at national and State/Territory levels but ***minor*** at the district/region level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

The domestic trade and industry impacts of an outbreak of ND in the local wild bird population are ***unlikely to be discernible*** at all levels.

Depending on the extent of the outbreak, financial losses in the domestic poultry industry, and in associated sales and service industries could be considerable. Even a relatively small outbreak could result in disruption of the industry and its normal marketing patterns. The IRA team considered that the impacts of a disease outbreak in a local population of low biosecurity poultry are ***unlikely to be discernible*** at national and State/Territory levels, but are ***minor*** at the district/region level, especially if free-range commercial poultry were involved. Impacts of an outbreak of ND in a local population of medium biosecurity commercial poultry would be ***unlikely to be discernible*** at national and State/Territory levels, but ***minor*** at the district/region level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

ND is an OIE Listed disease, and international markets for poultry and poultry products are likely to be adversely affected if an outbreak were diagnosed in Australia. However, the Australian export market in poultry and poultry products is small. The initial impacts of an outbreak of ND within any of the exposure groups (wild bird, low biosecurity poultry or medium biosecurity commercial poultry) on international trade would be similar. The impacts would persist until acceptance of zoning or compartmentalisation arrangements were negotiated with trading partners. Impacts were assessed by the IRA team as ***significant*** at district/region level, and ***minor*** at State/Territory level, because of at least temporary loss of markets, but recognising the relatively small percentage of annual poultry production which is exported.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

There are no reports of ND outbreaks in wild birds affecting biodiversity and endangered species. The IRA team considered that the indirect impacts of a disease outbreak in the local wild bird population on the environment are ***unlikely to be discernible*** at national, State/Territory and district/region levels, and ***minor*** at the local level.

The indirect impact of an outbreak of ND confined to low biosecurity poultry on the environment were assessed by the IRA team as ***unlikely to be discernible*** at any level. Similarly, the impact of an outbreak of ND confined to medium biosecurity commercial poultry

would be ***unlikely to be discernible*** at any level. While there is potential for adverse environmental effects from the disposal of large numbers of birds and their wastes, these effects are minimised by disposal carried out according to Environment Protection Authority (EPA) requirements. The costs of conforming to such requirements are accounted for under Indirect criterion 1.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Depending on the extent of the outbreak, restrictions on movement of birds, eggs, poultry products and people may lead to temporary community disruption. Community activities such as shows or pigeon races would be suspended, and destruction of pet birds in affected areas could result in significant levels of community concern and individual emotional distress. The impacts of a disease outbreak in wild birds are ***unlikely to be discernible*** at any level. The IRA team considered that the impacts of an outbreak in low biosecurity poultry or medium biosecurity commercial poultry on this criterion are ***unlikely to be discernible*** at national and State/Territory levels, but ***minor*** at the district/region level.

Outbreak Scenario 3

The consequences of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a district/region, and is eliminated) will be the same for all exposure groups, no matter which exposure group has been directly exposed to NDV in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

Australia has adopted a stamping out policy in relation to outbreaks of ND, and all birds on infected properties would be destroyed. Large numbers of birds may be involved on commercial properties, and the birds may represent the owner's sole source of income. If breeding flocks are infected, valuable genetic material may be lost. The direct costs of dead birds will be larger for commercial enterprises than for backyard or aviary birds, but with prompt eradication, the costs will be limited to the local area.

In addition to the death of birds due to disease or culling, the health of birds may be affected by movement restrictions leading to delayed marketing, with birds possibly outgrowing their accommodation and ready marketability. The IRA team considered that while the impacts at national and State/Territory levels are ***unlikely to be discernible***, there may be ***minor*** impacts at the district/region level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

The impacts of a contained outbreak of ND on the environment were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, but ***minor*** at the district/region level if the outbreak causes clinical signs or death in wild birds.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

An outbreak of ND in poultry or other birds would require implementation of eradication, surveillance and monitoring programs, which would be more extensive than those described under scenario 2. The IRA team considered that while the impact on the national economy is ***unlikely to be discernible***, there may be ***minor*** impacts at the State/Territory level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Depending on the extent of the outbreak, financial losses in the domestic poultry industry and in associated sales and service industries could be considerable. Movement restrictions associated with control and eradication programs may also interfere with marketing and trading patterns. The IRA team considered that while the impact on the national economy is ***unlikely to be discernible***, there may be ***minor*** impacts at the State/Territory level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

International markets for poultry and poultry products are likely to be adversely affected if an outbreak of ND was diagnosed in Australia. However, Australia's exports of poultry and poultry products are relatively small. Impacts were assessed by the IRA team as ***significant*** at district/region level and ***minor*** at State/Territory level, because of at least temporary loss of markets, but recognising the relatively small percentage of annual poultry production which is exported.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

Disposal of dead birds, their products and wastes during an outbreak of ND may lead to impacts on the environment, within the confines of EPA requirements. Impacts at the national or State/Territory levels were assessed by the IRA team as ***unlikely to be discernible***, while impacts at the district/region level were assessed as ***minor***.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Movement restrictions, suspension of community activities, and destruction of pet birds in affected areas could result in significant levels of community concern and individual emotional distress. Disruption of the flow of products and services, and decreased production may cause some job losses on farms and in associated industries. Prolonged loss or reduction of income for contract growers and their suppliers and associated industries may result until farms can be re-stocked following stamping out. The IRA team considered that the impacts of a disease outbreak on communities are ***unlikely to be discernible*** at national and State/Territory levels, and ***minor*** at the district/region level.

Outbreak Scenario 4

The consequences of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a State/Territory, and becomes endemic in Australia) will be the same for all exposure groups, no matter which exposure group has been directly exposed to NDV in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

If the disease spreads more widely through medium biosecurity commercial poultry and low biosecurity poultry, there will be significant losses of birds and production. The impacts will be on a larger scale than described under scenario 3, and were therefore assessed by the IRA team as ***minor*** at the national level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

The IRA team considered that the impacts of a more general outbreak of ND on the environment are ***unlikely to be discernible*** at the national level, but may be ***minor*** at the State/Territory level if large numbers of wild birds show clinical effects of disease.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

If ND spreads more widely, there would be significant efforts at the State/Territory, district/region and local levels to eradicate the disease, with intensive monitoring and surveillance programs in poultry and wild birds. There would be continuing efforts at surveillance even after stamping out was achieved to try to re-establish regional/country freedom for trade purposes. The costs of eradication, control, surveillance and compensation programs would be greater than in scenario 3. The IRA team considered that the impact at the national level was ***minor***.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Financial losses in the domestic poultry industry, and in associated sales and service industries could be considerable. Movement controls associated with control and eradication programs may also interfere with marketing and trading patterns on a larger scale than described in scenario 3. Impacts at the national level were assessed as ***minor*** by the IRA team.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

International markets for poultry and poultry products are likely to be adversely affected if an outbreak of ND were diagnosed in Australia. Impacts were assessed by the IRA team as ***significant*** at district/region level and ***minor*** at State/Territory level, because of at least temporary loss of markets, but recognising the relatively small percentage of annual poultry production that is exported.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

A generalised outbreak of ND will have ***no discernible impacts*** on this criterion at the national or State/Territory levels, but is likely to have ***minor*** impacts at the district/region level, if wild birds are clinically affected.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Movement restrictions, suspension of community activities such as shows and pigeon racing, and destruction of pet birds in affected areas could result in significant levels of community concern and individual emotional distress. Disruption of the flow of products and services, and decreased production may cause job losses on farms and in associated industries. Prolonged loss or reduction of income for contract growers and their suppliers and associated industries may result until farms can be re-stocked following stamping out. The IRA team considered that the impacts on communities are ***unlikely to be discernible*** at the national level, but ***minor*** at the State/Territory level.

Conclusions – impact assessment

The above estimates for each of the impact criteria were entered into the simulation model, and the overall impact of each outbreak scenario was estimated. Results are shown in Table 25.

Partial annual risk estimate for each outbreak scenario

The partial annual likelihood of entry, exposure, establishment and spread were combined with the impacts of the relevant outbreak scenarios to provide an estimate of the partial annual risk associated with each outbreak scenario. The outcomes of this process are shown in Table 26.

Unrestricted risk estimate

The overall risk associated with the import of whole chicken carcasses was assessed as ***moderate*** for NDV. This unrestricted risk estimate exceeds Australia's ALOP, and therefore, risk management is deemed necessary.

Direct impact on human life or health

Human infections with NDV are generally mild. Conjunctivitis has been reported in workers in laboratories and poultry processing plants.

Table 25. Impacts of each outbreak scenario

Exposure group	Outbreak	Impact
Wild birds	Scenario 1	Negligible
	Scenario 2	Low
Low biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Low
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Low
Non-avian species	Scenario 1	Negligible
	Scenario 2	Negligible
	Total Scenario 3	Low
	Total Scenario 4	Moderate

Table 26. Partial annual risk (PAR) of each outbreak scenario

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Low	Low
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Low	Low
Medium biosecurity poultry	Scenario 1	Negligible	Negligible	Negligible
	Scenario 2	E. Low	Low	Negligible
Non-avian species	Scenario 1	Low	Negligible	Negligible
	Scenario 2	Negligible	Negligible	Negligible
	Total Scenario 3	High	Low	Low
	Total Scenario 4	High	Moderate	Moderate

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Avian Infectious Bronchitis Virus

Technical Information

Background

Avian infectious bronchitis (IB) is an acute, contagious viral disease of chickens, which results in high morbidity and variable mortality in affected flocks. The disease can be manifested as a respiratory syndrome, characterised by coughing, sneezing and tracheal rales; a nephritic syndrome, in which the kidneys are the primary target organs, or as a reproductive syndrome characterised by a drop in egg production and quality (Cavanagh and Naqi 2003). IB virus (IBV) strains tend to differ between geographic regions, and emergence of variant strains is relatively common. Therefore, although IBV is endemic in Australia, if some exotic strains were introduced, currently available vaccination programs may be inadequate to prevent disease (Ignjatovic and Sapats 2000). IB is an OIE-listed disease.

Agent taxonomy

IB is caused by a virus of the genus *Coronavirus* and is a member of the family *Coronaviridae*. Numerous serotypes and strains exist, which differ in virulence, tropism and antigenicity.

Agent characteristics

Most strains of IBV are sensitive to heat at 56 ºC for 15 minutes, although resistance to thermal inactivation varies with the strain (McMartin 1993) and with the medium in which the virus is suspended (Hofstad 1956). The virus will survive in allantoic fluid for years at –30 ºC (Cavanagh and Naqi 2003).

IBV is considered to be susceptible to most common disinfectants (Cavanagh and Naqi 2003). It was inactivated in water containing copper at 0.2mg/L and 5ppm of free chloride ions, and increasing temperature over the range 20 ºC to 35 ºC adversely affected survival (Jordan and Nassar 1973). The presence of organic matter is protective against disinfectants, prolonging virus survival time (Jordan and Nassar 1973; McMartin 1993).

Resistance to pH varied between viral strains, but the virus has been shown to survive in the pH range 2 to 9, with optimum stability at pH 7.8 (McMartin 1993). Ten strains of virus, tested in culture medium, were found to be sensitive to direct irradiation with ultraviolet light (Otsuki, Yamamoto, and Tsubokura 1979).

IBV was stable in edible fermented food waste (pH 4-5) for 72 hours at 5 ºC to 10 ºC, but was inactivated within 24 hours at 20 ºC to 30 ºC (Wooley et al. 1981).

Epidemiology

Chickens and pheasants are the only known natural hosts of IBV, and the only species expected to show clinical signs in response to infection (Ignjatovic and Sapats 2000; Spackman and Cameron 1983; Gough et al. 1996). Coronaviruses isolated from pheasants have close gene sequence identity with IBV, but have not been shown to cause clinical disease in chickens, and some authors are of the opinion that they could be considered separate species (Cavanagh 2001). An IBV of the same serotype as a commonly used vaccine strain has been isolated from

tracheal mucosae and cloacal swabs of racing pigeons in Victoria. Specific Pathogen Free (SPF) chickens inoculated with the isolate exhibited marked respiratory rales, indicating that the virus isolated from the cloacal swab of the pigeons could be transmitted to chickens (Barr et al. 1988). Antibodies to IBV have been detected in turkeys, quails and rockhopper penguins (Ignjatovic and Sapats 2000), but the significance of this finding is unknown, as attempts to experimentally infect birds other than chickens and pheasants have been unsuccessful (McMartin 1993).

IBV is present in all countries in which there is an intensive poultry industry, with between- and within-flock prevalence approaching 100% (Ignjatovic and Sapats 2000). However, each geographic region (Europe, the United States, Asia, Australia) has a group of unique IBVs, which can be distinguished by their nucleotide sequences, and which are more closely related to each other than to strains from other parts of the world (Ignjatovic and Sapats 2000). Genetic recombination is common amongst IBV strains, and new serotypes are emerging all the time (Zanella et al. 2000; Cavanagh and Naqi 2003; Liu and Kong 2004; Lee and Jackwood 2001; Nix et al. 2000; El Houadfi et al. 1986).

The virus is present in respiratory secretions and in faeces, and transmission is principally horizontal through aerosol, direct contact with infected faeces, or by mechanical transmission. New antigenic strains have been introduced to poultry producing regions by live-haul trucks used to transport chickens (Erbeck and McMurray 1998). Experimentally infected birds may shed virus in the faeces for over 20 weeks, and the virus may persist in the trachea, liver, spleen, kidney and bursa for up to 29 days (Alexander and Gough 1977). Faecal shedding can be intermittent, and experimentally infected hens, which had ceased to shed virus, resumed faecal shedding at the point of lay (19 weeks) and continued to do so 28 weeks after inoculation (Jones and Ambali 1987).

Infection spreads rapidly among chickens in a flock, and between sheds on a farm (McMartin 1993). Windborne transmission between farms 1200 yards apart has been reported (Cumming 1970). While vertical transmission has not been conclusively proven to occur in the field, it was suspected in outbreaks of disease in newborn chicks and dead-in-shell embryos (McFerran et al. 1971). Vertical transmission of IBV was demonstrated to occur in experimentally inoculated hens in two studies (Cook 1971). Vectors do not appear to be involved in the spread of IBV (Ignjatovic and Sapats 2000).

Chickens of all ages appear to be susceptible to infection, with disease being most severe in young chicks. Morbidity approaches 100%, with mortalities of 20-30% occurring in the presence of concurrent infections (McMartin 1993). In general, mortality is low, but can be up to 25% in young chicks with IB nephritis (Meulemans et al. 1987). Economic losses from IBV are predominantly from production deficits. Respiratory disease leads to poor feed conversion rates and weight gains, as well as increased carcass condemnation rates, usually due to secondary infections. Layers and breeders may fail to reach their full potential because of delayed maturity, drops in egg production and failure to fully recover egg production after infection (Ignjatovic and Sapats 2000).

The incubation period of the virus is very short (18-36 hours) with naturally-occurring spread requiring about 36 hours or more (Cavanagh and Naqi 2003). However, for the purposes of international trade, the OIE defines the incubation period for IB as 50 days (World Organisation for Animal Health (OIE) 2005).

Clinical signs

Clinical signs will depend on age of affected birds, route of exposure, immune status and virulence of the virus (McMartin 1993). Respiratory signs are most prominent in chicks from two to six weeks of age, but also occur in older birds. Respiratory distress, coughing, sneezing, and rales, with or without nasal exudate, are accompanied by weakness, depression, lowered feed consumption and reduced weight gain. In uncomplicated infections, respiratory signs may resolve within seven days; however, secondary infections with *E.coli* and other pathogens may accentuate and prolong the respiratory disease for several weeks (Ignjatovic and Sapats 2000).

Birds acquiring the nephritic form of the disease may show transient respiratory signs, followed by depression, ruffled feathers, increased water intake and wet droppings, with death occurring four to five days after infection (Ignjatovic and Sapats 2000). Mortalities of up to 25% have been reported (Meulemans and van den Berg 1998).

IB in young chicks can cause permanent damage to the oviduct, with subsequent permanently reduced egg production and quality. Presence of maternal antibody may help prevent damage to the oviduct during early infections (Cavanagh and Naqi 2003). In adult hens, mild respiratory signs may be followed by a drop in egg production, the severity of which varies with the stage of lay and strain of virus (Cavanagh and Naqi 2003). Reductions in egg production of between 3% and 50% have been reported (Ignjatovic and Sapats 2000). There may also be declines in egg quality, including reduction in size of the egg, thin-shelled or misshapen eggs and thin albumen. Production may return to normal within 6–8 weeks, or may remain below expected levels.

IBV has been implicated in the formation of epididymal stones in roosters, resulting in reduced sperm production and fertility (Boltz, Nakai, and Bahr 2004).

Pathogenesis

The respiratory system is the main site of virus replication following inhalation, while the digestive system is another site of primary multiplication (McMartin 1993). The virus replicates in tracheal ciliated epithelial cells causing gross lesions within 18–24 hours (Purcell and McFerran 1972). The virus spreads within the respiratory tract, and high virus titres are measurable for five to seven days. From the respiratory system, the virus becomes blood-borne, and can then be isolated from various organs, including the oviduct, kidney and bursa of Fabricius where multiplication of virus can occur (McMartin 1993). The virus multiplies in the epithelial cells of the oviduct (McMartin 1993) and the tubular epithelial cells of the kidney (Meulemans and van den Berg 1998). Serotypes differ in their ability to cause damage to target organs, with some strains causing no pathological changes while others cause severe and permanent damage to oviduct epithelium, resulting in obstructive lesions (Crinion and Hofstad 1972).

The role of the digestive system for viral entry is uncertain (McMartin 1993). After administration of vaccine virus via the water, multiplication of virus took place in trachea (Hofstad and Yoder 1966). Vaccination with live virus at a dose of 10^6 EID_{50} delivered via drinking water resulted in lower serum antibody levels than the same dose delivered via the ocular route. Lower doses of intra-ocular vaccine failed to elicit a systemic antibody response, suggesting that lower doses in the drinking water also would have been ineffective (Toro et al. 1997).

Virus has been detected at all levels of the gut after experimental inoculation of day-old chicks, and was consistently found in duodenum and ileum up to 28 days post-infection (Jones and Ambali 1987). Excretion of virus in the faeces is erratic and may be prolonged (Jones and Ambali 1987), suggesting that virus replication occurs in the intestinal tract (Alexander and Gough 1977).

Pathology

Respiratory pathology includes mucosal thickening and exudate in the nasal passages, airways and airsacs, however, affected birds can die without visible lesions (McMartin 1993). If secondary infections occur, the lesions may be more severe, and accompanied by pericarditis and perihepatitis (Ignjatovic and Sapats 2000). The Delaware 072 serotype can cause severe airsacculitis in the absence of secondary infections (Ignjatovic and Sapats 2000).

Gross lesions are not usually found in the oviduct in acute cases of IB infection, but reduction in the length of the oviduct and ovarian regression or cystic lesions may be seen in chronic cases (McMartin 1993).

Birds with acute nephritic disease may have pale, swollen kidneys, with whitish fluid (urate deposits) in the ureters, and the carcass may be dehydrated (Chong and Apostolov 1982). These birds may have little or no visible pathology of the respiratory tract. Older birds (14–25 weeks) dying of chronic kidney disease may have reduced kidney size, urolithiasis, distended ureters and visceral deposits of urates (Brown et al. 1987). Persistent virus has been detected in the kidneys of chickens with experimentally-induced chronic active interstitial nephritis due to IBV (Albassam, Winterfeld, and Thacker 1986).

One strain of IBV has produced unusual lesions of the pectoral muscles in vaccinated meat chicken breeders (Gough et al. 1992).

Immunology

Immunity to IBV is complicated by the existence of multiple serotypes with variable virulence and tissue tropism (Cavanagh and Naqi 2003). Coronaviruses are known to have a high frequency of recombination, which can play a role in the generation of new strains. The use of several serotypes of live attenuated IBV strains could result in the induction of new variants (Kusters et al. 1990; Lee and Jackwood 2001). Although vaccination is routinely used in the intensive poultry industry, outbreaks of IBV continue to occur in vaccinated flocks in Australia and overseas (Ignjatovic and Sapats 2000; Liu and Kong 2004). Current Australian vaccination procedures do not guarantee protection from emerging field strains (Endo-Munoz and Faragher 1989), and would be unlikely to be protective against overseas strains (Ignjatovic and Sapats 2000).

Both attenuated live and oil emulsion inactivated vaccines are currently available overseas, with the strain of virus used being dependent on the strains prevalent in the region. Live vaccines may confer a better local immunity to the respiratory tract and may protect against a wider antigenic spectrum of field strains (Gough and Alexander 2004). The use of some live vaccines carries the risk of residual pathogenicity associated with vaccine back-passage in flocks (Gough and Alexander 2004). Inactivated vaccines are generally used in layers and breeders after priming with live vaccine, to confer longer lasting immunity. The high levels of antibodies help to protect against spread of virus to internal organs and subsequent drops in egg

production, protection which is not always conferred by live vaccines (Ignjatovic and Sapats 2000). Currently, only attenuated live vaccines are registered for use in Australia.

Vaccine virus has been isolated from trachea, liver, spleen, kidneys and bursa for at least two weeks, and from faeces for up to 14 weeks following vaccination (Alexander and Gough 1977).

Diagnosis

Diagnosis is based on a combination of history, clinical signs, post-mortem findings, isolation of IBV or demonstration of IBV antigen or RNA, and evidence of seroconversion or rising IBV/antibody titres (Ignjatovic and Sapats 2000; Cavanagh and Naqi 2003).Virus isolation is attempted from the trachea in acute infections, and from the caecal tonsils or faeces if infection has been present for a week or more. ELISA to detect IBV antigen is the most rapid test to confirm the presence of IBV; PCR can also be used to test field samples. Strain differentiation is most accurately determined by nucleotide sequencing of the S1 glycoprotein, but can also be carried out using virus isolation in tracheal organ cultures or embryonating eggs (Ignjatovic and Sapats 2000). Diagnosis is complicated by the presence of vaccine and field strains occurring simultaneously in many field samples (Ignjatovic and Sapats 2000).

Transmission in chicken meat

Cooked chicken meat for human consumption, which has undergone treatment at temperatures above 56 ºC for 30–45 minutes, is thought to represent a low risk for introducing IBV into importing countries (Ignjatovic and Sapats 2000). In an experimental study, IBV was isolated from the rectum, kidney and trachea of SPF chickens infected 10 days previously, killed, and kept in cold storage for 24 hours (Ganapathy, Cargill, and Jones 2005). Because vaccine and field strains can persist in organs such as airsacs and kidneys for more than two weeks, in the bursa for over 4 weeks and faeces for at least 20 weeks (Alexander and Gough 1977), there is potential for carcasses to be infected or contaminated at the time of slaughter and processing, and for such infections to be clinically undetected. Condemnation rates of 3–8% have been estimated, following an outbreak of IB (Ignjatovic and Sapats 2000).

Quarantine significance

IB is an OIE-listed disease.

Some strains of IB are endemic in Australia. The disease is not notifiable in any State or Territory of Australia, and is not subject to official controls within Australia. IB is not included in the Emergency Animal Disease Response Agreement.

An incursion of an exotic strain of IBV would probably necessitate the introduction of new vaccines, and increase the pool of genetically different viruses circulating in an area (Ignjatovic and Sapats 2000). Costs of new vaccines would be reflected ultimately in higher management costs.

Risk Assessment

Release assessment

Rel_1: Selection of source flock (between flock prevalence)

For an unrestricted risk estimate, it was assumed that the prevalence is at the highest sustainable level in an endemically infected country or zone (see Method for Risk Assessment). While between flock prevalence approaches 100% across the poultry industry, including layer flocks and meat chickens, it does not necessarily follow that all meat chicken flocks will be infected by the time of slaughter, particularly as vaccination is widely practiced. Meat chickens are slaughtered at approx 6 weeks of age, and therefore some flocks of meat chickens will reach slaughter weight before becoming infected. The likelihood that a particular source flock will be infected at the time of slaughter was assessed by the IRA team as ***moderate***.

Rel_2: Infection detected through flock surveillance and the flock withdrawn from slaughter

Clinical signs associated with IB will depend on several factors, described above. Respiratory signs may resolve within seven days (Ignjatovic and Sapats 2000), however, apparently recovered chickens may still carry the virus in airsacs, kidneys and digestive tract (Alexander and Gough 1977). The IRA team considered that the likelihood that an infected flock will be detected through routine flock surveillance, and the flock withheld from slaughter, is ***extremely low***.

Rel_3: Selection of an infected chicken from an infected flock (within-flock prevalence)

Within flock prevalence approaches 100% (Ignjatovic and Sapats 2000); therefore the likelihood that a selected individual chicken will be infected was assessed as ***high*** by the IRA team.

Rel_4: Background cross-contamination rate

This likelihood represents the background cross-contamination rate during slaughter and processing and applies to cross-contamination with material, infectious or otherwise, from other carcasses, including those tissues or materials in which the disease agent tends to localise.

IBV may persist in internal organs, including the respiratory and digestive tracts. However, the virus is sensitive to free chloride ions in water (Jordan and Nassar 1973), and surface contamination may, therefore, be reduced during washing and chilling of the carcass. The IRA team considered that the likelihood of a carcass being contaminated with potentially contaminated material from other birds is ***moderate***.

Rel_5: Likelihood that an uninfected carcass will be contaminated with the disease agent during slaughter and processing

As discussed in the Method for Risk Assessment (page 58, Part B), Rel_4 was used to calculate Rel_{5a} (the likelihood that an uninfected carcass from an infected flock will become

contaminated during slaughter and processing) and Rel_{5b} (the likelihood that an uninfected carcass from an uninfected flock will become contaminated during slaughter and processing).

For IB, Rel_{5a} was calculated as ***moderate***, and Rel_{5b} was calculated as ***low***.

Rel_6: The likelihood that the carcass of a bird that was *infected* prior to slaughter will be removed as a result of inspections before or during processing

Carcasses affected by obvious airsacculitis and muscle pathology would most likely be detected during processing. Condemnation rates of 3–8% due to airsacculitis have been reported (Ignjatovic and Sapats 2000); however, birds infected with IBV may not show gross pathological lesions that would prompt removal from the processing line. The IRA team considered that the likelihood that a contaminated/infected carcass would be removed during processing inspections was ***very low.***

Rel_7: The likelihood that the carcass of a bird that was *not infected* prior to slaughter will be removed as a result of inspections before or during processing

The likelihood that an uncontaminated/uninfected carcass will be removed during processing inspections was considered to be equal to the background rejection rate of 0.75% as described in the Method section (page 59, Part B).

Rel_8: Inactivation of the agent during further processing, storage, handling and transport

Given that the carcasses for importation will be chilled or frozen, and that the virus can persist for weeks to months at low temperatures, the likelihood of inactivation of the virus during further processing, storage, handling and transport, was assessed as ***very low*** by the IRA team.

Conclusions – Release assessment

After inserting the above estimates into the simulation model, and using the outputs from the model as a guide, the IRA team concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a ***moderate*** likelihood that imported chicken meat would be infected or contaminated with IBV.

Exposure assessment

Exposure Group 1: Wild birds

Of the steps identified as determinants in the infection of wild birds by exposure to scraps from an imported contaminated chicken carcass, $WB_{agentsurvival}$ and $WB_{infectivedose}$ are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$WB_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period prior to consumption by a wild bird

IBV is sensitive to ultraviolet light and is inactivated within 24 hours at 20 ºC to 30 ºC. Chicken meat scraps which would be available to wild birds would be likely to be exposed to UV in sunlight, and to ambient temperatures of 10 ºC to 35 ºC. The IRT team considered that the likelihood that IBV in chicken meat scraps remains viable for long enough to be accessed by a susceptible wild bird was ***low*.**

$WB_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a wild bird is sufficient to produce infection

IBV has not been reported to occur in birds known to frequent refuse dumps, although it has been isolated from pigeons in association with an infected flock. The oral dose of IBV sufficient to initiate infection in wild birds is not known; however, the lack of reported infections in birds other than chickens and pheasants suggests that infection of wild birds with IBV is a rare event.

The IRA team considered that there was a ***negligible*** likelihood that IBV would infect a wild bird consuming the contaminated meat scraps.

Exposure Group 2: Low biosecurity poultry

Of the steps identified as determinants in the infection of low biosecurity poultry by exposure to material from an imported contaminated chicken carcass, $BP_{agentsurvival}$, $BP_{infectivedose}$, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$BP_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period prior to consumption by low biosecurity poultry

The time between feeding of scraps and consumption by low biosecurity poultry is likely to be very short, so environmental degradation of the disease agent will be minimal. The IRA team considered that the likelihood that the agent would remain viable was ***certain (=1)***.

$BP_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a low biosecurity bird is sufficient to produce infection

Transmission of IB to chickens by feeding of infected meat has not been documented. However, infection with vaccine virus has been demonstrated following administration via the drinking water. Following infection, there is replication of virus in the respiratory and probably the intestinal tracts, with widespread distribution of virus in internal organs. The titre of virus in the muscle of viraemic chickens is not known; there is potential, however, for virus to be present in remnants of airsacs or kidneys in whole carcasses. Infection is most severe in younger birds, but chickens of all ages are susceptible to IBV. After considering the lack of documented evidence of transmission through meat, the evidence for oral transmission, and the high likelihood that carcass meat will contain the virus, the IRA team considered that the likelihood that low biosecurity poultry would be infected with IBV as a result of consuming the contaminated imported chicken meat scraps would be ***moderate***.

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

The likelihood that IBV would survive the rendering process was negligible. For IBV the IRA team considered that the likelihood that the product will be re-contaminated post processing was negligible. Therefore the likelihood that poultry feed would be contaminated with IBV was estimated to be ***negligible*** by the IRA team.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

Given that IBV would be destroyed by rendering, and that feed derived from contaminated carcasses would be diluted with feed from non-risk material, the likelihood that the amount of final poultry ration eaten by a bird would contain an oral infectious dose of virus was considered to be ***negligible*** by the IRA team.

Exposure Group 3: Medium biosecurity commercial poultry

Of the steps identified as determinants in the infection of medium biosecurity commercial poultry by exposure to material from an imported contaminated chicken carcass, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that poultry feed would be contaminated with IBV was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As discussed above (low biosecurity poultry), the likelihood of the amount of final poultry ration consumed containing an oral infectious dose of IBV was considered to be ***negligible***.

Exposure Group 4: Non-avian species

IB has not been reported in non-avian species. Therefore this group was not considered further. $NAS_{infectivedose}$ was set to a value of zero.

Conclusions – Exposure assessment

Using the simulation model as a guide, the partial likelihood of exposure for each of the exposure groups was determined, taking the above estimates of the exposure variables into account. A summary of the outcomes determined by the IRA team is set out in Table 27.

Table 27. Partial likelihoods of exposure (PLE)

Exposure group	PLE
Wild birds	Negligible
Low biosecurity poultry	Very low
Medium biosecurity poultry	Negligible
Non-avian species	Zero

Consequence assessment

Establishment and spread was considered in the context of the outbreak scenarios, described in the Method for Risk Assessment.

Estimating the likelihood of each outbreak scenario

The partial likelihood of establishment and spread (PLES) of IBV for the different exposure groups is described below. Four outbreak scenarios were considered relevant:

1. Disease agent does not establish or is not recognised within the directly exposed population
2. Disease agent establishes within the directly exposed population, is identified and is eliminated by human efforts or by natural means
3. Disease agent establishes in the directly exposed population, spreads within a district/region, including into other exposure groups if applicable, and is eliminated by human action or by natural means
4. Disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia.

Wild birds

Outbreaks of IB in birds other than poultry have not been reported, except for one incident in racing pigeons. Infection of wild birds with IBV, with subsequent spread to poultry, has not been reported. The IRA team considered that the most likely outcome of exposure of a wild bird, resulting from scavenging imported contaminated chicken meat scraps would be a single, or at most a few, isolated occurrences of infection in wild birds, with the virus being unable to establish ongoing infection in the population. In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 28).

Table 28. Estimated partial likelihood of establishment and spread (PLES) values for IBV in wild birds

Outbreak scenario 1:	$PLES_{(wb)}1$	**High**
Outbreak scenario 2:	$PLES_{(wb)}2$	**Extremely low**
Outbreak scenario 3:	$PLES_{(wb)}3$	**Extremely low**
Outbreak scenario 4:	$PLES_{(wb)}4$	**Extremely low**

Low biosecurity poultry

IB is an acute, contagious disease of chickens and pheasants, resulting in high morbidity and variable mortality. Clinical signs are variable. Although this exposure group includes commercial free-range poultry and ratites, the IRA team considered that the sub-set of this group most likely to be exposed to scraps of imported chicken meat was small flocks of backyard poultry, because these are more likely to be directly fed table scraps. In such flocks, there is less opportunity for the generation of high levels of environmental contamination, than might occur with an outbreak of infectious disease in a large commercial flock. In addition, the relative instability of the virus under ambient conditions means that environmental contamination from an infected backyard flock is limited. This would help to limit the likelihood of windborne transmission from low biosecurity flocks. The most likely outcome of infection would be a single or a few isolated occurrences of infection.

If the disease were to establish in a low biosecurity backyard flock, the level of expertise in disease recognition is likely to be low. This would tend to decrease the likelihood of detection in the low biosecurity flock. In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely, followed by outbreak scenario 2.

Mechanical transmission of the virus by contaminated persons or fomites, and transmission by movement of birds may facilitate spread of the virus beyond the initially infected flock before it is recognised. If the infection had spread to commercial poultry, the IRA team considered that diagnosis would follow relatively rapidly, if the exotic agent was of a type which could ‘break through’ existing vaccination regimes. However, the disease is not notifiable and not subject to the emergency animal disease response agreement, so no nationally coordinated eradication measures are likely to be implemented. It is likely that exotic strains would slowly spread through the national commercial poultry flock, particularly if available vaccines were not fully effective at preventing infection. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 29).

Table 29. Estimated partial likelihood of establishment and spread (PLES) values for IBV in low biosecurity poultry

Outbreak scenario 1:	$PLES_{(bp)}1$	**High**
Outbreak scenario 2:	$PLES_{(bp)}2$	**Low**
Outbreak scenario 3:	$PLES_{(bp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(bp)}4$	**Very low**

Medium biosecurity commercial poultry

The IRA team considered that the most feasible route for exposure of medium biosecurity commercial poultry to imported carcass components would be through poultry feed containing inadequately rendered processing waste. The likelihood that avian infectious bronchitis virus would remain viable following rendering of contaminated imported carcasses and parts was assessed as negligible (page 93). Nevertheless, the IRA team estimated the PLES values, based on their assessment of the likely outcomes in the improbable event that exposure of medium biosecurity commercial poultry did occur via this route.

Poultry are highly susceptible to IB, and spread of exotic IBV within exposed flocks will be rapid. The IRA team considered that there was an extremely low likelihood that IB would not establish, following exposure of susceptible birds to an introduced exotic strain. Antibody to endemic strains of IBV in Australian poultry flocks (whether following natural infection or vaccination) may confer some degree of protection against exotic strains of the virus, making it less likely that an outbreak of exotic IB would be recognised early. Since the disease is not notifiable, nor included in the Emergency Animal Disease Response Agreement, it is unlikely that immediate action would be taken to eradicate it. Transmission of IBV is principally horizontal through aerosol, direct contact with infected faeces, or by mechanical transmission, and windborne transmission between farms has been reported. In view of these factors, outbreak scenario 4 (disease becomes endemic) was considered the most likely. If the disease were to become established in medium biosecurity commercial poultry, the IRA team considered that the disease would be likely to spread to other susceptible exposure groups, and that it would be highly unlikely to be eradicated once such spread had occurred. In this case, only other poultry would be affected. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 30).

Table 30. Estimated partial likelihood of establishment and spread (PLES) values for IBV in medium biosecurity commercial poultry

Outbreak scenario 1:	$PLES_{(cp)}1$	**Extremely low**
Outbreak scenario 2:	$PLES_{(cp)}2$	**Low**
Outbreak scenario 3:	$PLES_{(cp)}3$	**Low**
Outbreak scenario 4:	$PLES_{(cp)}4$	**Moderate**

Non-Avian Species

As stated above, this exposure group was not considered further in relation to this disease.

Conclusion – Likelihood assessments

The estimates for the likelihood of release, the partial likelihoods of exposure for each of the exposure groups, and the partial likelihood of establishment and spread for each of the outbreaks scenarios were combined with the expected volume of trade using the simulation model. This allowed the calculation of partial annual likelihood of entry, exposure, establishment and spread for each of the outbreak scenarios. The results of this calculation are shown in Table 31.

Table 31. Partial annual likelihood of entry, exposure, establishment and spread (PALEEES) for the outbreak scenarios

Exposure group	Outbreak scenario	PALEEES
Wild birds	Scenario 1	Very low
	Scenario 2	Extremely low
Low biosecurity poultry	Scenario 1	High
	Scenario 2	High
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
Total all exposure groups	Scenario 3	High
Total all exposure groups	Scenario 4	High

Estimating the impacts associated with each outbreak scenario

For each outbreak scenario the direct and indirect impacts of IB were estimated at the national, State or Territory, district/region and local levels, as described in the Methods section (page 90-95, Part B).

The likelihood of IBV affecting non-avian species (exposure group 4) was considered to be remote. Therefore, the impacts of IB occurring in this exposure group were not considered further.

Outbreak Scenario 1

By definition, outbreak scenario 1 means that the disease agent does not establish in the initially exposed population, or does not cause sufficient disease to lead to investigation and recognition of the infection. Therefore the impacts will be ***unlikely to be discernible*** for all exposure groups and all criteria.

Outbreak Scenario 2

The impacts of this outbreak scenario (disease agent establishes within the directly exposed population, and is identified and eliminated) will differ between exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

Chickens and pheasants are the only known natural hosts of IBV, and the only species expected to show clinical signs in response to infection. Direct economic loss from death of wild birds, were it to occur, is not measurable. Other impacts from the death of wild birds will be

considered under direct criterion 2 and indirect criterion 4. The direct impacts of disease on wild birds were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

An outbreak of IB contained within the low biosecurity poultry population may result in losses to individual owners, but the impacts of such losses on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Economic losses from IB in medium biosecurity commercial poultry are predominantly from production deficits such as poor feed conversion rates and weight gains, increased carcass condemnation rates, and drops in egg production. Mortalities of 20–30% have been reported. The impacts of a recognised outbreak, contained within a single shed or a local area of commercial poultry, were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory, and district/region levels, but ***minor*** at the local level due to the loss of birds and production on the affected farms.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

Since chickens and pheasants are the only species commonly infected with IBV, the impacts of an outbreak of IB in any of the exposure groups was considered to be limited to commercial birds. Wild birds, and in particular native species, are not expected to be affected. Impacts on the environment were assessed by the IRA team as ***unlikely to be discernible*** at any level.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

The impact of an outbreak of IB in wild birds, with no subsequent spread, was assessed as ***unlikely to be discernible*** at any level. Similarly, the impacts of an outbreak in low biosecurity poultry were assessed by the IRA team as ***unlikely to be discernible*** at any level.

It is expected that an outbreak of IB in commercial poultry would result in increased surveillance and monitoring of the poultry population and increased costs of vaccine production and vaccination. Impacts in medium biosecurity commercial poultry were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, but ***minor*** at the district/region level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Domestic trade and industry impacts of an outbreak of IB in wild birds were assessed as ***unlikely to be discernible*** at all levels. The impacts of an outbreak in low biosecurity poultry were assessed by the IRA team as ***unlikely to be discernible*** at any level.

An outbreak of IB in commercial poultry may lead to temporary marketing and movement restrictions, while the disease outbreak is being investigated and vaccination is being arranged. Impacts in medium biosecurity commercial poultry were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory, and district/region levels, but ***minor*** at the local level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

Some strains of IBV are endemic in Australia, and an outbreak of IB is unlikely to have any impacts on international trade. The impacts of an outbreak of IB in any exposure group on international trade markets were assessed by the IRA team as ***unlikely to be discernible*** at any level.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

Because of the species specificity of IBV (discussed above) the impacts of an outbreak of IB in any exposure group on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at any level.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures.

The impacts of a disease outbreak in wild birds on this criterion were assessed as ***unlikely to be discernible*** at any level. Similarly the impacts of an outbreak in low biosecurity poultry were assessed by the IRA team as ***unlikely to be discernible*** at any level.

Restrictions on movement of birds, eggs, poultry products and people may lead to temporary community disruption. Community activities such as shows or pigeon races may be suspended. Impacts of an outbreak in medium biosecurity commercial poultry were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory, and district/region levels, but ***minor*** at the local level.

Outbreak Scenario 3

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a district/region, and is eliminated) will be the same for all susceptible exposure groups, no matter which exposure group has been directly exposed to IBV in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

Poor feed conversion rates and weight gains, increased carcass condemnation rates, drops in egg production, and mortalities may result in economic losses to some producers. Costs were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, and ***minor*** at the district/region level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

As discussed above, the impacts of an outbreak of IB on the environment were assessed as ***unlikely to be discernible*** at any level.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

Spread of an exotic strain of IB to other exposure groups would require increased surveillance and monitoring programs, and may require the development of new vaccines. The IRA team considered that while the impacts on the national and State/Territory economies were assessed as ***unlikely to be discernible***, there may be ***minor*** impacts at the district/region level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Spread of an exotic strain of IBV may initially lead to marketing and movement restrictions while the disease is being investigated and vaccination is being arranged. The impacts on the national, State/Territory and district/region economies were assessed by the IRA team as ***unlikely to be discernible***. Impacts at the local level were assessed as ***minor***.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

IB is present in all countries where poultry are intensively raised, although the serotypes may differ. The impacts of an outbreak of IB on international trade markets were assessed by the IRA team as ***unlikely to be discernible*** at any level.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

As discussed above, IB causes clinical disease only in chickens and pheasants, and is not expected to cause infection in native species. It is not expected to cause problems in native species nor to impact on biodiversity. The impacts of an outbreak of IB on the environment were assessed by the IRA team as ***unlikely to be discernible*** at any level.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Restrictions on movement of birds, eggs, poultry products and people, and suspension of community activities such as shows or pigeon races may lead to temporary community disruption. The impact of an outbreak of IB on communities was assessed by the IRA team as ***unlikely to be discernible*** at the national, State/Territory and district/region levels, and ***minor*** at the local level.

Outbreak Scenario 4

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a State/Territory, and becomes endemic in Australia) will be the same for all susceptible exposure groups, no matter which exposure group has been directly exposed to IBV in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

If the disease spreads more widely through medium biosecurity commercial poultry and low biosecurity poultry, there will be significant losses of birds and production. At the national level, the impacts were assessed as ***unlikely to be discernible*** by the IRA team. Impacts were assessed as ***minor*** at the district/region level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

IBV does not affect wild birds or native animals. The impacts of a more general outbreak of IB on the environment were assessed by the IRA team as ***unlikely to be discernible*** at any level.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

General spread of an exotic strain of IBV within poultry is likely to lead to increased costs of diagnosis and surveillance, and may require the development of new vaccines. The impact on the national economy was assessed by the IRA team as ***unlikely to be discernible***. Impacts at the district/region level were assessed as ***minor***.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

While the disease is being investigated and vaccination is being arranged, there may be marketing and movement restrictions. The IRA team considered that impacts of a more general outbreak of IB on domestic trade and industry are ***unlikely to be discernible*** at national and State/Territory levels, and ***minor*** at the district/region level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

As discussed above, the IRA team considered that an outbreak of IB is ***unlikely to have any discernible*** impact on international trade markets at any level.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

As discussed above, the IRA team considered that the impacts of an outbreak of IB on the environment were assessed as ***unlikely to be discernible*** at any level.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

As discussed above, the IRA team considered that the impact of a general outbreak of IB on communities was assessed as ***unlikely to be discernible*** at national, State/Territory, and district/region levels, but ***minor*** at the local level.

Conclusions – impact assessment

The above estimates for each of the impact criteria were entered into the simulation model, and the overall impact of each outbreak scenario was estimated. Results are shown in Table 32.

Table 32. Impacts of each outbreak scenario

Exposure group	Outbreak scenario	Impact
Wild birds	Scenario 1	Negligible
	Scenario 2	Negligible
Low biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Very low
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
	Scenario 3	Very low
	Scenario 4	Very low

Partial annual risk estimate for each outbreak scenario

The partial annual likelihood of entry, exposure, establishment and spread were combined with the impacts of the relevant outbreak scenarios to provide an estimate of the partial annual risk associated with each outbreak scenario. The outcomes of this process are shown in Table 33.

Unrestricted risk estimate

The partial annual risk of each outbreak scenario was combined to provide an estimate of overall annual risk. The overall risk associated with the import of whole chicken carcasses was assessed as ***very low*** for avian IBV. As the unrestricted risk estimate meets Australia's ALOP, no risk management was considered necessary.

Direct impact on human life or health

IBV is not known to affect humans and is not considered to be a threat to public health.

Table 33. Partial annual risk (PAR) of each outbreak scenario

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	Very low	Negligible	Negligible
	Scenario 2	E. low	Negligible	Negligible
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Medium biosecurity poultry	Scenario 1	Negligible	Negligible	Negligible
	Scenario 2	Negligible	Very low	Negligible
Non-avian species	Scenario 1	Zero	Zero	Zero
	Scenario 2	Zero	Zero	Zero
	Total Scenario 3	High	Very low	Very Low
	Total Scenario 4	High	Very low	Very Low

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Very Virulent and Exotic Antigenic Variant Strains of Infectious Bursal Disease Virus

Technical Information

Background

Infectious bursal disease (IBD) is an acute, contagious viral infection, which causes immunosuppression in young chicks, and disease and mortality in 3 to 6 week old chickens (van den Berg et al. 2000; Lukert and Saif 2003). The IBD virus (IBDV) infects actively dividing B lymphocytes within the bursa of Fabricius, leading to immunosuppression of varying duration and severity and increased susceptibility to secondary viral and bacterial infections. IBD was first described in the area of Gumboro in the United States, and 'Gumboro disease' is sometimes used as a synonym for IBD (Lukert and Saif 2003).

There are two serotypes of the virus: IBDV serotype 1 and IBDV serotype 2. While antibodies to both serotypes have been found in chickens, serotype 2 may be more commonly isolated from turkeys. IBDV serotype 1 is an important pathogen of chickens. No clinical disease has been reported in chickens or turkeys as a result of infection with IBDV serotype 2 (Lukert and Saif 2003). Serotype 1 IBD viruses can be classified in a number of ways, based on phenotypic traits (such as antigenicity and pathogenicity) and genetic molecular traits (nucleotide sequence of the gene coding for the viral protein VP2) (Lukert and Saif 2003).

Based on their phenotypic traits, serotype 1 IBD viruses can be conveniently classified as attenuated (vaccine strains), classic (standard), antigenic variant, and very virulent strains (vvIBDV, sometimes known as hypervirulent) (van den Berg et al. 2000). This classification is also supported by the genetic traits – that is, VP2 sequence differences. Both classic and antigenic variant strains exist in Australia, but these can be genetically differentiated from overseas classic, variant and very virulent strains (Sapats and Ignjatovic 2000; Ignjatovic and Sapats 2002).

For the purposes of this risk assessment, exotic antigenic variant strains are defined as variant strains that are antigenically and genetically different from those that exist in Australia, and include United States variant strains. This risk assessment is concerned with serotype 1 IBD viruses that are exotic to Australia, including the very virulent IBDV strains and IBDV strains that are antigenically and genetically different from Australian strains.

Infectious bursal disease is an OIE-listed disease.

Agent taxonomy

Infectious bursal disease is caused by a double stranded RNA virus of the family *Birnaviridae*. Within serotype 1, IBDV strains can be further differentiated into classic and variant types, based on their antigenic or serotypic properties.

In addition to serotyping, IBD viruses can be classified according to genetic molecular types. By combining nucleotide sequencing with phylogenetic analysis of the hypervariable region of the VP2 protein of IBDV, it is possible to differentiate serotype 1 IBD viruses into vvIBDV, United States variants, Australian variants, Australian classic strains and classic strains from

other countries (Cao et al. 1998; Hamoud and Villegas 2006; Ignjatovic et al. 2004; Jackwood et al. 2006; Sapats and Ignjatovic 2000). RT-PCR has also been developed using probes binding to the VP4 sequence, which is able to differentiate classic, variant, and very virulent strains of IBDV (Peters, Lin, and Wu 2005).

Classic strains of IBDV can be antigenically differentiated from vvIBDV, United States variants and Australian variants using either chicken recombinant antibodies or monoclonal antibodies (Eterradossi et al. 1997; Eterradossi et al. 1998; Sapats and Ignjatovic 2000; Sapats et al. 2005; Sapats et al. 2006; Sapats et al. 2005; Sapats et al. 2006). It is not possible to antigenically differentiate classic strains from vaccine strains, unless the vaccine strains are based on vvIBDV or variant strains. While the classic and very virulent strains are similar antigenically, although not identical, they differ markedly in virulence.

IBD viruses can also be classified by pathogenicity testing. Very virulent IBD viruses induce mortalities, while the majority of classic and variant strains induce bursal regression but few mortalities (van den Berg et al. 2000). Vaccine strains cause less bursal regression and the time to restoration is shorter than for field strains (Giambrone and Clay 1986; Mazariegos, Lukert, and Brown 1990).

Agent characteristics

IBDV is very stable and persists in poultry houses even after cleaning and disinfection (Lukert and Saif 2003). The virus has been shown to remain infectious for 122 days in a chicken house and 52 days in feed, water and faeces (Benton, Cover, and Rosenberger 1967). The virus is inactivated above pH 12, but is resistant to low pH (Lukert and Saif 2003). IBDV is resistant to many classes of disinfectants. One study concluded that only chlorine- and aldehyde-containing disinfectants were effective against IBDV, and aldehydes required particular temperatures for greatest effect (Meulemans and Halen 1982). In another study, an iodine-complex disinfectant inhibited viral infectivity (Benton et al. 1967). Invert soaps with 0.05 % sodium hydroxide at room temperature or above, and at pH 12 or above, either inactivated or had a strong inhibitory effect on the virus in cell culture (Shirai et al. 1994).

In addition to its resistance to chemical disinfection, IBDV is remarkably heat-resistant. In a study conducted in 1988 to assess heat inactivation of a field strain of IBDV (52/70) in peptone broth, approximate times to reduce the infectivity by 1 $\log_{10}$ were 18.8 minutes at 70 °C, 11.4 minutes at 75 °C and 3.0 minutes at 80 °C (Alexander and Chettle 1998). Unpublished work conducted in 1997 at the Quality Control Unit, Central Veterinary Laboratory, Alderstone, United Kingdom, showed that a mix of bursal homogenate (23%), skin and fat (4%), muscle tissue (23%) and peptone broth (50%) contained no viable IBDV only after cooking at 80 °C for at least 120 minutes (Quality Control Unit 1997b).

Epidemiology

IBDV occurs in all major poultry producing areas of the world, with the exception of New Zealand, where an attenuated (vaccine) strain was first detected in domestic poultry in 1993 (Chai et al. 2001). The New Zealand poultry industry has taken action to eradicate the vaccine virus from poultry flocks and results of monitoring indicate success (Bingham, Christensen, and Stanislawek 2006; Gerber 2006). Different antigenic and pathogenic viral types exist in different geographic locations (van den Berg et al. 2000; Lukert and Saif 2003). Both classic and variant strains of IBDV have been demonstrated to be present in Australian poultry (Sapats and Ignjatovic 2000; Ignjatovic and Sapats 2002). Strains of the classic type that exist in

Australia are genetically different from IBD viruses isolated overseas (Sapats and Ignjatovic 2000; Ignjatovic and Sapats 2002). Antigenic variant viruses identified in Australia are also genetically different from IBD viruses isolated overseas and are only distantly related to antigenic variants described in North America and elsewhere (Sapats and Ignjatovic 2000), and vaccines currently used in Australia do not protect against United States variant strains of IBDV strains (Ignjatovic, Sapats, and Gould 2001). Some variant-like IBD viruses recently described in Asia, Europe, and Central and South America are genetically similar to variant strains from the United States (Cao et al. 1998; Hamoud and Villegas 2006; Jackwood et al. 2006). Very virulent strains are exotic to Australia, but are found in Europe, Asia, Africa, the Middle East and South America (Chettle, Stuart, and & Wyeth 1989; Cao et al. 1998; Di Fabio et al. 1999; van den Berg, Gonze, and Meulemans 1991; Hernandez et al. 2006; Ikuta et al. 2001; Lin et al. 1993; Kasanga et al. 2007; Mardassi et al. 2004; Meir, Jackwood, and Weisman 2001; Parede et al. 2003).

IBDV serotype 1 only causes clinical disease in chickens. A serotype 1 IBDV has been isolated from the faeces of clinically healthy adult ducks, but the significance of the isolate is uncertain (McFerran et al. 1980). Guinea fowl showed clinical signs and developed antibodies to IBDV following intra-ocular inoculation with IBD bursal extract and signs of disease were observed in in-contact chickens (Adewuyi, Durojaiye, and Adene 1989). However, in a more recent study, pheasants, partridges and guinea fowl failed to excrete virus after experimental infection with IBDV, while quail shed virus via the faeces for several days after inoculation, without showing clinical signs (van den Berg et al. 2001). The authors concluded that persistence of IBDV in wild birds is unlikely to occur and that the virus is highly host-specific for chickens. Antibodies to serotype 1 IBDV have been found in turkeys, pheasants, ostriches, guinea fowl, ducks, geese and wild birds, including gulls, crows, pigeons and penguins (Gardner et al. 1997; van den Berg et al. 2000; Gauthier-Clerc et al. 2002; Gilchrist 2005). IBDV has not been *confirmed* to cause disease in wild birds.

IBDV is a highly contagious virus, which is able to persist in the environment between outbreaks. The main route of transmission is the faecal-oral route and the virus can survive for prolonged periods in faeces and bedding (Benton, Cover, and Rosenberger 1967). Mechanical transmission of virus can occur via faecal contamination of fomites (van den Berg et al. 2000). The lesser mealworm (larva of the darkling beetle) is common in poultry houses worldwide and can serve as a reservoir for the virus (McAllister et al. 1995). IBDV has been isolated from mosquitos and rats but there is no evidence they act as vectors (Lukert and Saif 2003). vvIBDV was recovered from the faeces of a dog for two days after it was fed on the spleen, liver, bursa of Fabricius and intestines of birds which died from acute infection with vvIBDV (Pages-Mante et al. 2004). However, it is considered that the findings represent simple mechanical passage of the virus through the dog's digestive tract. While there is no clear evidence of wild birds acting as reservoirs or vectors of IBDV, the possibility can not be ruled out (Gilchrist 2005; van den Berg et al. 2000).

Chickens are most susceptible to infection during active bursal development, with clinical signs being most common between three and six weeks of age. Chickens infected before three weeks of age, and not protected by maternal antibodies, develop immunosuppression which can lead to secondary viral and bacterial infections and reduce the efficacy of vaccination. Clinical signs are occasionally seen in birds up to the age of 15–20 weeks (Ley et al. 1979; Okoye and Uzoukwu 1981). Age-resistance to the development of clinical signs of IBD has been documented, and is believed to be related to changes in the bursa as chickens mature (van den

Berg et al. 2000). In susceptible birds, the incubation period may be as short as two to three days.

The prevalence of IBDV infection in slaughter-age chickens is unknown (Mandeville, Cook, and Jackwood 2000). However, in a 2002 outbreak in Spain, 10 of 18 vvIBD viruses were isolated from slaughter-aged broilers (between 35 and 49 days old), and at least some of these were from vaccinated flocks (Dolz et al. 2005). Variant IBDV was isolated from 39-43 day old layer pullets that had been vaccinated with a classic vaccine strain at 28 and 35 days of age, after having high levels of maternal antibodies at hatch (Ismail et al. 1990). These reports indicate that infection of chicks with field strains of IBD virus can occur after waning of maternal antibody levels. The virus persists in the bursa and lymphoid tissues, including caecal tonsils, for 4-6 weeks after infection (Elankumaran, Heckert, and Moura 2002), and therefore, the virus may be present in, and being shed by, slaughter-age chickens. Subclinical infection with classic strains of IBDV has been reported in over 90% of unvaccinated poultry flocks in the United Kingdom (Edwards 1981), over 80% of meat chicken flocks in Belgium (van den Berg, Gonze, and Meulemans 1991) and in 77% of layer flocks tested in Ohio (Jackwood and Saif 1983). In infected flocks, seroconversion is up to 100% with mortality rates of 1–2% (van den Berg et al. 2000).

Of samples obtained from 20 meat chicken farms in Victoria, New South Wales, Queensland, South Australia and Western Australia, samples from 14 farms (70%) were positive for IBDV using an antigen capture ELISA. Sequencing of the viruses from 10 farms determined their genetic similarity to Australian vaccine or classic strains, while viruses from four farms (from Victoria and South Australia) were determined to be Australian variant strains (Ignjatovic and Sapats 2002). An outbreak of IBD in meat chickens in New South Wales in 1999, causing 2–2.5% mortality in affected sheds, was found to be due to a classic IBDV of Australian origin, and was controlled by vaccination (Sergeant 1999; Ignjatovic et al. 2004). Subsequent testing of this isolate in SPF chickens revealed no mortalities. Variant strains remain largely restricted to Victoria and South Australia, with little if any genetic change over the last ten years (S. Sapats, Australian Animal Health Laboratory, Geelong, Victoria, Australia, pers. comm. March 2007).

The prevalence of disease associated with very virulent strains of IBDV in most countries where vaccination for vvIBDV is practiced has not been documented. This is largely for the reason that there has been no reliable way to identify the highly virulent strain of IBDV, except through experimental infection in chickens (Lin et al. 1993; Jackwood and Sommer 2005). Recently, however, the nucleotide sequence of the VP2 gene of vvIBDV has been characterised, facilitating recognition of new incursions of vvIBDV into regions not previously affected (Ignjatovic, Sapats, and Gould 2001; Jackwood and Sommer 2005). In addition, data have been published suggesting that both RNA segments of the virus co-evolve and that vvIBDV may be identified by analysis of both genomic segments (Le Nouen et al. 2006).

In flocks affected by vvIBDV, mortality rates vary from 25% in meat chickens, to 60% in flocks of laying breeds, and up to 100% in SPF chickens (van den Berg, Gonze, and Meulemans 1991; van den Berg et al. 2000).

Antigenic variants are endemic in poultry in the United States (Rosales et al. 1989; Jackwood and Nielsen 1997; Jackwood and Sommer-Wagner 2005; Hamoud and Villegas 2006; Jackwood and Nielsen 1997). Antigenic variant-like strains have also been described in Canada, Central and South America, Asia and Europe, and some of these strains bear close

genetic resemblance to the United States variant strains (Cao et al. 1998; Hamoud and Villegas 2006; Ikuta et al. 2001; Jackwood et al. 2006).

Clinical signs

Disease severity depends on the age and breed of the affected birds, the degree of passive immunity and the virulence of the strain of virus (van den Berg et al. 2000). After a short incubation period, clinical signs include watery diarrhoea, ruffled feathers and prostration, with mortality reaching an early peak (three to four days post-infection) before subsiding. Initial infection on a farm is generally acute, with very high mortality rates if a virulent strain is involved (van den Berg et al. 2000). Infection by viral strains of low pathogenicity, or occurring while maternal antibodies are present, may be inapparent.

Three main clinical forms of IBD are described, in association with different viral strains:

a) Classic (standard) virulent strains of IBDV are associated with disease that is most often subclinical. It occurs after a decline in passive immunity, and mortality specifically due to IBDV infection is relatively low

b) The United States variant strains principally cause immunosuppression, leading to an increased susceptibility to secondary infections

c) The very virulent strains of IBDV are associated with acute clinical disease and high mortality rates (van den Berg et al. 2000).

Pathogenesis

Within four hours of oral inoculation, the virus is found within the lymphoid tissue of the digestive tract, where it replicates before entering the circulation via the hepatic portal vein. A primary viraemia is followed by invasion and replication in the bursa of Fabricius; a second viraemia, and massive infection of lymphoid organs (van den Berg et al. 2000). In chicks less than three weeks old, unless protected by maternal antibody, atrophy of the bursa leads to immunosuppression and predisposes to secondary infections, in the absence of specific clinical signs of IBD. Chickens affected after three weeks and generally before six weeks of age, show typical clinical signs of IBD in addition to evidence of immunosuppression.

Maternal antibodies derived from immunisation with vaccines based on classic strains does not protect against vvIBD unless present at high levels, and may be poorly protective against some variant strains (Ignjatovic, Sapats, and Gould 2001; Lukert and Saif 2003; Jackwood and Sommer-Wagner 2005). In one study, variant strains were shown to persist longer in tissues in the face of maternal immunity, with caecal tonsils, bone marrow and bursa supporting virus replication for up to four weeks after experimental inoculation (Elankumaran, Heckert, and Moura 2002). An earlier study had suggested that a United States variant strain persisted for a shorter duration in the bursa in the presence of maternal antibodies (Abdel-Alim and Saif 2001).

Pathology

Grossly visible lesions are mostly seen in the bursa of Fabricius, which becomes inflamed and often haemorrhagic within three to four days of infection. Within five days, the swollen bursa returns to normal size and then begins to atrophy (Lukert and Saif 2003). Haemorrhages in the pectoral and thigh muscles may also be observed (Lukert and Saif 2003). Some United States

variant strains appear to cause rapid atrophy of the bursa without obvious inflammation (van den Berg et al. 2000). Very virulent strains cause more severe lesions in non-bursal lymphoid organs but similar changes in the bursa of Fabricius (van den Berg et al. 2000; Lukert and Saif 2003).

Virus has been isolated from the thymus, spleen, caecal tonsils and bone marrow, four weeks after experimental inoculation of an antigenic variant strain of IBDV into one-day-old chicks, and from the bursa six weeks after inoculation (Elankumaran, Heckert, and Moura 2002).

Immunology

Maternal antibodies can protect chicks against early infections with classic and some variant strains of IBDV, with resultant protection against the immunosuppressive effects of the virus. However, maternal antibody does not protect chicks from disease associated with very virulent strains, unless present at high levels, and may be poorly protective against some variant strains (Ignjatovic, Sapats, and Gould 2001; Lukert and Saif 2003; Jackwood and Sommer-Wagner 2005). Vaccination of parent birds with inactivated oil emulsion vaccines, following initial vaccination with live virus vaccines, appears to afford longer-lasting passive immunity to progeny than vaccination with attenuated live vaccines (Lukert and Saif 2003).

Because maternal antibody levels are rarely uniform in young flocks, spread of field strains of IBDV through a flock may occur over days or weeks (Wyeth and Chettle 1990). Variant strains of virus may persist in the face of maternal immunity, leading to clinical disease or immunosuppression late in the grow-out period for meat chickens (Elankumaran, Heckert, and Moura 2002). Furthermore, the presence of maternal antibodies may delay the onset of susceptibility to classic IBDV strains until beyond 40 days of age (Wyeth and Cullen 1979), with an increased likelihood of infection being maintained until slaughter age.

Active immunity is acquired either by vaccination or through exposure to field strains of the virus, and to date, it is not possible to differentiate vaccinated from naturally infected birds. In young birds, the vaccine can be administered orally while, in adult birds, injection of an inactivated vaccine is required, as oral vaccines appear to be ineffective in stimulating an antibody response in older birds (Hitchner 1976).

Available vaccines are either live or inactivated. Live IBDV vaccines are produced from fully or partially attenuated strains of the virus, and depending on the degree of virus attenuation, vaccines can be ‘mild’, ‘mild-intermediate’, ‘intermediate’, or ‘intermediate-plus’ (‘hot’) (Lukert and Saif 2003). Vaccine virus RNA has been detected in the caecal tonsils and bursa of orally inoculated chickens for 22 days, the duration of the experimental study (Barlic-Maganja, Zorman-Rojs, and Grom 2002). During an investigation of an outbreak of vvIBDV in Spain, an intermediate virulence vaccine strain was detected by RT-PCR 36 days after vaccination (Dolz et al. 2005). The authors speculated that the vaccine strain may have been recirculating in the birds during the growing period.

Mild or intermediate vaccines are used in young breeder chickens to produce a primary response, followed by the use of inactivated vaccine near to the point of lay. For best effect, live vaccine must be administered after maternally derived antibodies have waned, and is generally given between four and eight weeks of age (van den Berg et al. 2000).

Inactivated vaccines are used in breeding hens that have previously been primed by live vaccine, to produce high, long-lasting and uniform levels of antibodies. The usual program is to give one or more live vaccines followed by an inactivated vaccine at 16–20 weeks.

Revaccination of breeders may be required if antibody profiling indicates that flock titres have declined (Lukert and Saif 2003).

Technology has been developed to deliver live vaccine into eggs during the incubation period. Intermediate live IBD vaccines administered *in ovo* can cause significant bursal lesions in hatched chicks at 7–21 days of age, unless protected by maternal antibodies, but can protect against challenge with classic and variant forms of the disease at three weeks of age (Giambrone, Dormitorio, and Brown 2001). Live vaccines can be combined with IBDV antibody and injected *in ovo* at 18 days of incubation. The eggs hatch and the vaccine virus is released from the immune complex at about seven days of age. The complex of IBDV and antibody improves the safety of the vaccine to the embryo, while also being effective in the presence of maternal antibody (Jeurissen et al. 1998; Baines et al. 1999). *In ovo* vaccination of meat chickens with a combination of variant and classic strains is commonly practised in the United States (Professor J. Giambrone, Auburn University, United States, pers. comm. October 2004 and April 2007). In one report, vaccine virus was detectable in lymphoid tissue of hatched chicks at day 15 but not day 21 after *in ovo* vaccination (Corley, Giambrone, and Dormitorio 2001).

DNA vaccines containing IBDV gene have recently been trialled experimentally in the United States (Wu, Chang, and Lin 2001). A subunit vaccine, based on an avian herpesvirus vector expressing VP2 antigens, has also been released and is commercially available (Tsukamoto et al. 2002).

Vaccination against very virulent IBDV

In countries where vvIBDV is present, intermediate and intermediate-plus vaccines are used to protect meat chickens and commercial layer replacements. They are also used in young parent chickens if there is a high risk of natural infection with vvIBDV. Second and third applications are usually administered, especially if there is high risk of exposure to virulent disease (van den Berg et al. 2000).

Recent studies have shown that none of the tested IBDV vaccines fully protected against experimental vvIBDV challenge (Rautenschlein et al. 2005; Kabell et al. 2005). In one study, although none of the challenged birds died, vvIBDV continued to replicate in the bursa and caecal tonsils. IBDV antigen was detected in 100% of bursae 21 days after birds were vaccinated with an intermediate-plus vaccine (Rautenschlein et al. 2005). The authors speculated that in maternal antibody-positive birds, IBDV vaccines may complex with antibodies, with the virus being released as the maternal antibody levels wane. This is a similar mechanism of action to that described for *in-ovo* vaccination using combined vaccine and IBDV antibody.

In another study using vaccinated and challenged SPF chickens with no maternal antibody, vvIBDV RNA was detected in the bone marrow of a 44 day-old chicken vaccinated at one week of age, and challenged with vvIBDV at 28 days (Kabell et al. 2005). The challenge strain was also detected in the bursa and thymus of 31 to 44 day-old chickens vaccinated at 3 weeks of age, and challenged with vvIBDV a week later. The authors of this study concluded that surviving field virus in a broiler house may infect, replicate and be excreted from vaccinated chickens (Kabell et al. 2005).

Vaccination against exotic antigenic variant strains

Vaccines protective against classic strains of IBDV may not protect chickens against disease associated with some variant strains (Ismail et al. 1990; Ignjatovic, Sapats, and Gould 2001; Lukert and Saif 2003). In the United States, bivalent vaccines, containing classic and variant strains, have been developed to overcome this problem. However, the emergence of new antigenic variants has recently been reported in the United States, against which commercially available vaccines are not protective (Jackwood and Sommer-Wagner 2005). *In-ovo* vaccination of meat chickens against IBDV (including variant strains) is common practise in the United States, with about 25% of birds then receiving a second dose of vaccine (Professor J. Giambrone, Auburn University, United States, pers. comm. October 2004). While some bivalent vaccines from the United States will protect Australian chickens against challenge with Australian classic and variant strains, currently available Australian vaccines confer no protection against United States variant strains (Ignjatovic, Sapats, and Gould 2001).

Vaccination regimes in use in Australia

In general, Australian meat chicken breeders and layer breeders are vaccinated at eight to twelve weeks of age with an intermediate classic-strain live vaccine, and receive a booster with inactivated classic-strain vaccine at around 18 weeks (Dubs 2007). High levels of maternal antibody to classic strains will protect broiler chickens against Australian variant strains (Ignjatovic, Sapats, and Gould 2001). Vaccination of meat chickens is rarely practiced in Australia. Vaccination of meat chickens was practiced by one smaller company for a period of 12 months from January 2006 in an effort to establish if poorer than expected performance results could be attributed to infection with IBDV. This company has since ceased vaccination of meat chickens against IBDV (Dubs 2007). Australian commercial layers are generally not vaccinated against IBD (Grimes 2001).

Diagnosis

Clinical diagnosis in the acute form of IBD is based on clinical signs and post-mortem examination findings of typical bursal lesions. In subclinical forms, histological examination of the bursa is required for diagnosis, as bursal atrophy may also occur in other diseases, such as Marek’s disease (van den Berg et al. 2000).

Some serological tests do not distinguish between IBDV serotypes 1 and 2, nor between vaccine and field strains of virus, and serology is, therefore, not used extensively for diagnosis in IBDV endemic areas.

Diagnosis is achieved by virus isolation, preferably from the bursa of Fabricius within three days after the appearance of clinical signs. Viral antigens in the bursa can be demonstrated by direct and indirect immunofluorescence or by immunoperoxidase staining of thin sections of bursal tissue. The use of monoclonal antibodies can enhance the specificity of the test. Other tests, such as AGID and agglutination tests can also be used to demonstrate the presence of viral antigens, but are relatively insensitive. Polymerase chain reaction (PCR), DNA probes and nucleotide sequencing have also been used to demonstrate and characterise the presence of IBDV (Brown, Green, and Skinner 1994). RT-PCR has been developed using probes binding to the VP4 sequence, which is able to differentiate classic, variant, and very virulent strains of IBDV (Peters, Lin, and Wu 2005). However, the exact genetic elements needed for expression of the very virulent phenotype have not yet been determined (Jackwood and Sommer 2005; van den Berg et al. 2004).

Methods used in Australia to differentiate between IBDV strain types include:

i) Molecular approaches: Using nucleotide sequencing combined with phylogenetic analysis of the hypervariable region of the VP2 protein of IBDV, it is possible to differentiate vvIBDV, United States antigenic variants, Australian variants, Australian classic and classic strains from other countries in Europe, Asia and the United States. Sequences for the hypervariable region of VP2 for many vaccine strains are available and these can be differentiated from classical strains. PCR and antigen ELISA can be used for rapid diagnosis of vvIBDV, followed by conventional PCR with nucleotide sequencing and pathogenicity testing (Ignjatovic 2004).

ii) Antigenic differentiation, using either chicken recombinant antibodies or monoclonal antibodies to differentiate vvIBDV, United States antigenic variants and Australian variants from classic IBDV (Sapats and Ignjatovic 2000; Sapats et al. 2005; Sapats et al. 2006).

iii) Pathogenicity testing, bursal regression and examination of histopathological lesions.

iv) Cross-protection studies, including serum neutralisation tests in tissue culture, vaccination/challenge trials in SPF birds, and challenge experiments using young commercial meat chickens with high levels of maternal antibody to classic IBDV strains. These methods are useful to confirm antigenic variant strains (S. Sapats, Australian Animal Health Laboratory (AAHL), Geelong, Victoria, Australia; pers. comm. February 2007).

Transmission in chicken meat

Contaminated meat may be produced by the slaughter of viraemic, asymptomatic chickens, or by the slaughter of convalescent chickens which still carry the virus in the digestive tract or other tissues (van den Berg et al. 2000). Virus has been detected at low titres in the muscle for at least four days after experimental inoculation of chickens (Quality Control Unit 1997a) and may persist in bone marrow and other tissues for up to four weeks after infection (Elankumaran, Heckert, and Moura 2002). Virus in the digestive tract can serve as a source of cross-contamination of carcasses in the slaughter line (van den Berg et al. 2000). Vaccine virus was detected in the bursa of SPF chickens for 14 days after administration of a low oral dose ($10^{2.5}$ EID_{50} per bird) (Abdel-Alim and Saif 2001), and virus RNA was detected in the caecal tonsils and bursa of chickens for at least 22 days after inoculation (Barlic-Maganja, Zorman-Rojs, and Grom 2002).

In one trial, chicken products (drumsticks and patties) were inoculated with five strains of IBDV and then subjected to cooking. The meat products were cooked either by frying in hot oil or cooking in a flame grill. Following cooking to an internal temperature of 71 ºC and 74 ºC respectively, meat contained viable virus detected in cell culture. The authors concluded that temperatures and times required to totally inactivate IBDV in meat would compromise texture and palatability of the poultry products, and that poultry offal processing may not adequately neutralise IBDV (Mandeville, Cook, and Jackwood 2000). There were no appreciable differences in the heat lability of the five strains tested.

Research undertaken by AAHL on behalf of the Australian Government Department of Agriculture, Fisheries and Forestry, concluded that vvIBDV (strain CS88) can be ‘transmitted with ease from both muscle and bursa of infected non-immune chickens to day old chicks by a natural route of infection’ (Australian Animal Health Laboratory 2002). This report further concluded that, while vaccination using standard European vaccination regimes was sufficient to prevent overt illness, it ‘did not inhibit chickens from developing IBD infection when challenged with vvIBDV. Sufficient virus was present in both bursae and muscles to infect

susceptible day-old-chicks via a natural route' (Australian Animal Health Laboratory 2002). A copy of the unpublished report of this experimental work is available from Biosecurity Australia on request.

A second experiment was conducted using variant E IBDV in place of the very virulent CS88 strain. In the first part of this experiment, unvaccinated chickens were challenged with variant E IBDV. Bursa and muscle samples harvested from these chickens 72–80 hours later were subsequently fed to naïve three- to seven-week-old chicks. Transmission of variant E IBDV was seen in all 50 chicks fed bursa, and in at least 32 of 50 chicks fed muscle. In another part of the experiment chickens, bred from vaccinated hens and vaccinated against Variant E IBDV at either 10 or 20 days of age, were challenged with variant E IBDV. Bursa and muscle samples were again collected 72–80 hours post challenge and fed to groups of naïve chicks. There was transmission of variant E IBDV to all 50 chicks fed bursa from chickens vaccinated at 10 days of age, and to all 50 chicks fed bursa from chickens vaccinated at 20 days of age. However, transmission of variant E IBDV was not demonstrated in any of 100 chicks fed muscle from vaccinated chickens (Australian Animal Health Laboratory 2004). A copy of the unpublished report of this experimental work is available from Biosecurity Australia on request.

The results of this research suggest that vaccination at 10–20 days of age will prevent chickens developing sufficient virus in muscle to infect naïve chicks. Not all chickens are vaccinated however, so it is probable that some chickens infected with variant IBDV will have sufficient virus in their muscle tissues to cause infection in naive birds. The experiments demonstrated that the bursa did become infected, and presumably there was infection of other gastrointestinal lymphoid tissues following oral exposure. Under commercial slaughter conditions, the IRA team considered it highly likely that there would be cross-contamination of carcasses from gastrointestinal contents and from direct contact of bursa with the carcass during processing, so that meat and scraps from them would contain variant IBDV capable of infecting other poultry.

Quarantine significance

IBD is an OIE-listed disease. Very virulent IBD is a notifiable disease in Australia.

Classic and Australian variant strains are present in Australian poultry. The Australian Emergency Animal Disease Cost-sharing Agreement includes vvIBD in Category 4 (Animal Health Australia 2006). Diseases in this category are considered to be mainly associated with production losses. Although there may be international trade losses and local market disruptions these would not be of a magnitude that would be expected to significantly affect the national economy. Costs associated with eradication of outbreaks of diseases included in Category 4 of the Emergency Animal Disease Cost-Sharing Agreement are funded 20% by government and 80% by industry.

Potential losses to the Australian poultry industry of an outbreak of vvIBD, including eradication costs and disruption of production, have been estimated at $100 million, although the actual costs may be lower due to existing vaccination programs (Agriculture and Resource Management Council of Australia and New Zealand In preparation). If vvIBDV or exotic antigenic variant IBD became endemic in Australia, new vaccination strategies would be required to control the disease. Meat chickens and commercial layers would require vaccination, as maternal antibody would not be fully protective against infection in three to six week old chickens that are most susceptible to clinical disease. Estimated annual requirements for vaccination are outlined in Table 34.

If exotic antigenic variant strains of IBDV were introduced into Australia, alterations in the vaccination program for breeders and layers would be required. Since vaccines currently used in Australia do not protect against exotic antigenic variant strains of IBDV, suitable vaccines would also need to be imported or locally developed, and registered in Australia.

Table 34 shows an example of vaccination programs as they currently exist for the Australian endemic strains of IBDV, and in the event of incursions of vvIBDV, and exotic antigenic variant strains of IBDV. It can be seen that, because of the need to vaccinate meat chickens, the costs associated with control of IBD would be considerably greater than in the present situation, especially if incursions of both very virulent and variant IBDV were to occur.

Table 34. Estimated annual requirements for vaccination against IBDV

	Current Australian regime	vvIBDV	Exotic antigenic variant IBDV
Breeders (meat chickens and layers) 5.5 million	5.5 million doses live 5.5 million doses inactivated	11 million doses live[1] 11 million doses inactivated[1]	11 million doses live 11 million doses inactivated
Meat chickens 400 million	NIL	400 million doses live	400 million doses live
Layers 12 million	NIL	24 million doses live	24 million doses live

[1] Assumes a second live and a second inactivated vaccine is necessary in breeders in order to maintain acceptable antibody profile

Risk Assessment (Very Virulent IBDV)

Release assessment

Rel_1: Selection of source flock (between flock prevalence)

For an unrestricted risk estimate, it was assumed that the prevalence is at the highest sustainable level in an endemically infected country or zone (see Method for Risk Assessment). IBDV infection is widespread wherever chickens are raised. While infection is most common in chickens less than six weeks of age, spread of infection through the flock may occur over days or weeks due to variations in waning levels of maternal antibodies (Wyeth and Chettle 1990) and the persistence of field strains in poultry houses. The prevalence of infection with IBDV in slaughter-age chickens is unknown. Nevertheless, virus can persist in body tissues, including the bursa and bone marrow for several weeks (Barlic-Maganja, Zorman-Rojs, and Grom 2002; Elankumaran, Heckert, and Moura 2002). The IRA team concluded that the likelihood that a source flock will be infected with vvIBDV at the time of slaughter was ***moderate***.

Rel_2: Infection detected through flock surveillance and the flock withdrawn from slaughter

Clinical signs of vvIBDV infection may not be obvious to the producer, since vaccination is known to prevent disease expression, but not to prevent infection. A vaccinated flock surviving infection with IBDV would most likely be sent to slaughter at the appropriate time. The likelihood that an infected flock will be withheld from slaughter was assessed as ***extremely low*** by the IRA team.

Rel_3: Selection of an infected chicken from an infected flock (within-flock prevalence)

IBD is a highly contagious disease and, in an outbreak, most birds within a shed are likely to be infected. The virus persists in some tissues, including the intestinal tract, for several weeks. The IRA team concluded that, if an infected flock were sent to slaughter, the likelihood that a selected individual chicken will be infected was ***moderate***.

Rel_4: Background cross-contamination rate

This likelihood represents the background cross-contamination rate during slaughter and processing and applies to cross-contamination with material, infectious or otherwise, from other carcasses, including those tissues or materials in which the disease agent tends to localise. IBDV persists in the digestive tract and bursa of Fabricius of infected chickens (Barlic-Maganja, Zorman-Rojs, and Grom 2002). The IRA team considered that the likelihood of a carcass being contaminated with potentially infective material from other birds, especially from the digestive tract or the bursa, was ***moderate***.

Rel_5: Likelihood that an uninfected carcass will be contaminated with the disease agent during slaughter and processing

As discussed in the Method for Risk Assessment (page 58, Part B), Rel_4 was used to calculate Rel_{5a} (the likelihood that an uninfected carcass from an infected flock will become contaminated during slaughter and processing) and Rel_{5b} (the likelihood that an uninfected carcass from an uninfected flock will become contaminated during slaughter and processing).

For vvIBD, Rel_{5a} was calculated as ***low***, and Rel_{5b} was calculated as ***low***.

Rel_6: The likelihood that the carcass of a bird that was *infected* before slaughter will be removed as a result of inspections before or during processing

Carcasses affected by obvious haemorrhages in muscle would most likely be detected during processing. However, birds infected with IBDV may show no gross pathological lesions that would prompt removal from the processing line. The IRA team considered that the rejection rate of infected carcasses would be greater than the background rejection rate but still very low. The likelihood that a contaminated/infected carcass will be removed during processing inspections was therefore assessed as ***very low*** by the IRA team.

Rel_7: The likelihood that the carcass of a bird that was *not infected* before slaughter will be removed as a result of inspections before or during processing

The likelihood that an uncontaminated/uninfected carcass will be removed during processing inspections was considered to be equal to the background rejection rate of 0.75% as described in the Method section (page 59, Part B).

Rel_8: Inactivation of the agent during further processing, storage, handling and transport

IBDV is resistant to inactivation by heat, changes in pH, and alterations in temperature. Given that the carcasses for importation will be chilled or frozen, and that the virus can persist for weeks or months at low temperatures, the likelihood of inactivation of the virus during further processing, storage, handling and transport was assessed as ***negligible*** by the IRA team.

Conclusions – Release assessment

After inserting the above estimates into the simulation model, and using the outputs from the model as a guide, the IRA team concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a ***moderate*** likelihood that imported chicken meat would be infected or contaminated with vvIBDV.

Exposure assessment

Exposure Group 1: Wild birds

Of the steps identified as determinants in the infection of wild birds by exposure to scraps from an imported contaminated chicken carcass, $WB_{agentsurvival}$ and $WB_{infectivedose}$ are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$WB_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by a wild bird

IBDV is very stable in the environment, tolerating variations in pH and temperature. The IRA team considered that the likelihood that the virus will remain viable in the chicken meat scraps is ***high***.

$WB_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a wild bird is sufficient to produce infection

While the establishment of IBDV infection has not been reported in wild birds, wild birds have developed antibody following exposure to the virus, presumably due to transient infection. Taking account of this information, the IRA team considered that there was an ***extremely low*** likelihood that very virulent IBDV would infect a wild bird consuming the contaminated meat scraps.

Exposure Group 2: Low biosecurity poultry

Of the steps identified as determinants in the infection of low biosecurity poultry by exposure to material from an imported contaminated chicken carcass, $BP_{agentsurvival}$, $BP_{infectivedose}$,

FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$BP_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by low biosecurity poultry

The time between feeding of scraps and consumption by low biosecurity poultry is likely to be very short, so environmental degradation of the disease agent will be minimal. The IRA team considered that the likelihood that the agent will remain viable is ***certain (=1)***.

$BP_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a low biosecurity bird is sufficient to produce infection

Following infection, there is systemic replication of vvIBDV, with viraemia and widespread tissue distribution. Virus has been isolated from muscle for at least four days, from caecal tonsils, spleen, thymus and bone marrow for 4 weeks, and from bursa for six weeks after experimental infection of one-day-old commercial meat chickens (Elankumaran, Heckert, and Moura 2002). Vaccine virus at an oral dose of $10^{2.5}$ EID_{50} per bird can be detected in the bursa 14 days after administration, therefore suggesting that a relatively low oral dose is sufficient to produce infection in young chickens.

Transmission of vvIBDV to chickens by feeding of infected meat has been documented in SPF chickens (Australian Animal Health Laboratory 2002), and experimental transmission of virus has been demonstrated via the oral route. However, chickens over the age of 15–20 weeks, which make up the majority of backyard flocks, are resistant to clinical infection with IBDV (van den Berg et al. 2000). Disease is less likely to occur in flocks without young birds.

When these factors are combined it was considered that there was a high likelihood that vvIBDV would infect chickens of susceptible age that consumed meat scraps, but that such flocks would be in the minority. The overall likelihood that low biosecurity poultry would be infected with vvIBDV as a result of consuming the contaminated chicken meat scraps was therefore considered by the IRA team to be ***low***.

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

The likelihood that IBDV would survive the rendering process was negligible. The IRA team considered that the likelihood the product would be re-contaminated with IBDV post-processing was negligible. Therefore the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with IBDV was estimated by the IRA team to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

Given that IBDV would be destroyed by rendering, and that feed derived from contaminated carcasses would be diluted with feed from non-risk material, the likelihood that the amount of final poultry ration eaten by a bird would contain an oral infectious dose of virus was considered by the IRA team to be ***negligible***.

Exposure Group 3: Medium biosecurity commercial poultry

Of the steps identified as determinants in the infection of medium biosecurity commercial poultry by exposure to material from an imported contaminated chicken carcass, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with IBDV was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As discussed above (low biosecurity poultry), the likelihood of the amount of final poultry ration consumed containing an oral infectious dose of IBDV was considered to be ***negligible***.

Exposure Group 4: Non-avian species

As discussed above, $NAS_{agentsurvival}$ was considered to be equal to $WB_{agentsurvival}$.

Although virus was detected for two days in the faeces of an experimentally exposed dog, there is no evidence that IBDV occurs in non-avian species in nature. Therefore, this exposure group was not considered further in relation to this disease. $NAS_{infectivedose}$ was set to a value of zero.

Conclusions – Exposure assessment

Using the simulation model as a guide, the partial likelihood of exposure for each of the exposure groups was determined, taking the above estimates of the exposure variables into account. A summary of the outcomes determined by the IRA team is set out in Table 35.

Table 35. Partial likelihoods of exposure (PLE)

Exposure group	PLE
Wild birds	Extremely low
Low biosecurity poultry	Very low
Medium biosecurity poultry	Negligible
Non-avian species	Zero

Consequence assessment

Establishment and spread was considered in the context of the outbreak scenarios, described in the Method for Risk Assessment.

Estimating the likelihood of each outbreak scenario

The partial likelihood of establishment and spread (PLES) of vvIBDV for the different exposure groups is described below. Four outbreak scenarios were considered relevant:

1. Disease agent does not establish or is not recognised within the directly exposed population

2. Disease agent establishes within the directly exposed population, is identified and is eliminated by human efforts or by natural means

3. Disease agent establishes in the directly exposed population, spreads within a district/region, including into other exposure groups if applicable, and is eliminated by human action or by natural means

4. Disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia.

Wild birds

Infection and establishment of IBDV in wild birds has not been reported, although wild birds may become transiently infected and do develop antibodies following exposure to the virus. The IRA team considered that the most likely outcome of infection of a wild bird, resulting from scavenging imported contaminated chicken meat scraps, would be a single or a few isolated occurrences of infection in wild birds, with the virus being unable to establish ongoing infection in the population. Infection of wild birds with IBDV, with subsequent spread to poultry, has not been reported, and it was considered an extremely unlikely event. In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 36).

Table 36. Estimated partial likelihood of establishment and spread (PLES) values for vvIBDV in wild birds

Outbreak scenario 1:	$PLES_{(wb)}1$	**High**
Outbreak scenario 2:	$PLES_{(wb)}2$	**Extremely low**
Outbreak scenario 3:	$PLES_{(wb)}3$	**Extremely low**
Outbreak scenario 4:	$PLES_{(wb)}4$	**Extremely low**

Low biosecurity poultry

Although this exposure group includes commercial free-range poultry and ratites, the IRA team considered that the sub-set of this group most likely to be exposed to scraps of imported chicken meat was small flocks of backyard poultry, because these are more likely to be directly fed table scraps. In such flocks, there is less opportunity for the generation of high levels of environmental contamination than might occur with an outbreak of infectious disease in a large commercial flock. Moreover, most backyard poultry flocks consist of adult layers or spent hens, with only a small proportion of flocks containing breeding birds and chicks, or pullets under the age of 15–20 weeks. It is the latter flocks that would be most susceptible to infection with IBDV with obvious clinical signs, while infection of flocks of older birds, if it occurred at all,

would most likely be subclinical. The most likely outcome of infection would be a single or a few isolated occurrences of infection, which were considered unlikely to be noticed.

If the disease did establish in a backyard flock, the level of expertise in disease recognition is likely to be low. Transmission of the virus by movement of infected birds or contaminated persons or fomites may facilitate spread of the virus beyond the initially infected flock before it is recognised and eradication measures are implemented. If this disease established in the Australian poultry population, it would be difficult to eradicate because of its high level of resistance to environmental factors. Therefore, in the long term it was considered likely that the disease would become endemic, if it did establish in this country.

In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 37).

Table 37. Estimated partial likelihood of establishment and spread (PLES) values for vvIBDV in low biosecurity poultry

Outbreak scenario 1:	$PLES_{(bp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(bp)}2$	**Low**
Outbreak scenario 3:	$PLES_{(bp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(bp)}4$	**Low**

Medium biosecurity commercial poultry

The IRA team considered that the most feasible route for exposure of medium biosecurity commercial poultry to imported carcass components would be through poultry feed containing inadequately rendered processing waste. The likelihood that infectious bursal disease virus would remain viable following rendering of contaminated imported carcasses and parts was assessed as negligible. Nevertheless, the IRA team estimated the PLES values, based on their assessment of the likely outcomes in the improbable event that exposure of medium biosecurity commercial poultry did occur via this route.

IBDV is a highly infectious and resistant virus, and spread within an exposed flock of medium biosecurity commercial poultry would be rapid. The manifestations of IBDV can be very variable, particularly in vaccinated flocks, and while higher levels of management expertise and formal flock monitoring should ensure that an outbreak would be recognised sooner than in backyard poultry, immunity to Australian field strains might be expected to mask the presence of the infection for some time. Scenario 1 was therefore considered an extremely unlikely outcome. Eradication would be made more difficult by the fact that IBDV is so resistant to environmental factors. Only one country, New Zealand, has reported the eradication of IBDV infection in the national poultry flock. The particular circumstances that contributed to the ability of the New Zealand poultry industry to achieve this result included the early detection of the infection, aided by absence of vaccination against IBD, and a restructuring in the industry which led to the closure of a significant number of the infected farms. Because of the widespread use of vaccination of breeder birds to provide maternal antibody, early detection of an incursion of exotic IBDV is less likely in Australia than was the case in New Zealand, and attempts to eradicate the disease by farm closures are less likely. In view of these factors,

outbreak scenario 2 (a recognised outbreak in the directly exposed population followed by eradication by human or natural means) was considered less likely. Once established in the Australian poultry population, the environmental resistance of the organism would make eradication very difficult. The IRA team concluded that the most likely outcome following introduction of vvIBDV to a population of medium biosecurity commercial poultry would be that the organism would become endemic. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 38).

Table 38. Estimated partial likelihood of establishment and spread (PLES) values for vvIBDV in medium biosecurity commercial poultry

Outbreak scenario 1:	$PLES_{(cp)}1$	**Extremely low**
Outbreak scenario 2:	$PLES_{(cp)}2$	**Low**
Outbreak scenario 3:	$PLES_{(cp)}3$	**Low**
Outbreak scenario 4:	$PLES_{(cp)}4$	**Moderate**

Non-Avian Species

As stated above, this exposure group was not considered further in relation to this disease.

Conclusion – Likelihood assessments

The estimates for the likelihood of release, the partial likelihoods of exposure for each of the exposure groups, and the partial likelihood of establishment and spread for each of the outbreaks scenarios were combined with the expected volume of trade using the simulation model. This allowed the calculation of partial annual likelihood of entry, exposure, establishment and spread for each of the outbreak scenarios. The results of this calculation are shown in Table 39.

Estimating the impacts associated with each outbreak scenario

For each outbreak scenario the direct and indirect impacts of IBD were estimated at the national, State or Territory, district/region and local levels, as described in the Methods section (pages 90-95, Part B).

The likelihood of IBDV affecting non-avian species (exposure group 4) was considered to be remote. Therefore, the impacts of IBDV occurring in this exposure group were not considered further.

Outbreak Scenario 1

By definition, outbreak scenario 1 means that the disease agent does not establish in the initially exposed population, or does not cause sufficient disease to lead to investigation and recognition of the infection. Therefore the impacts will be ***unlikely to be discernible*** for all exposure groups and all criteria.

Table 39. Partial annual likelihood of entry, exposure, establishment and spread (PALEEES) for the outbreak scenarios

Exposure group	Outbreak scenario	PALEEES
Wild birds	Scenario 1	High
	Scenario 2	Very low
Low biosecurity poultry	Scenario 1	High
	Scenario 2	High
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
Total all exposure groups	Scenario 3	High
Total all exposure groups	Scenario 4	High

Outbreak Scenario 2

The impacts of this outbreak scenario (disease agent establishes within the directly exposed population, and is identified and eliminated) will differ between exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

Since wild birds do not play a significant part in production, direct economic loss from death of wild birds is not measurable. Other impacts from the death of wild birds will be considered under criterion 2: the environment. The direct impacts of disease on wild birds were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

An outbreak of vvIBD contained within the low biosecurity poultry population will result in losses to individual owners, especially if commercial free-range poultry are involved. The impacts of such losses on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels but ***minor*** at the local level.

Initial infection with vvIBDV in a poultry flock is generally acute with high mortality rates. Loss of birds and production within a single shed of medium biosecurity commercial poultry on the affected farm were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels but ***minor*** at the local level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

Although antibodies to IBDV have been demonstrated in gulls and crows, clinical disease has not been demonstrated in these species. The impacts of an outbreak of IBD in wild birds on the

environment, were it to occur, were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

The impacts of an outbreak of vvIBD in low biosecurity poultry on the environment were assessed by the IRA team as ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak in medium biosecurity commercial poultry were assessed as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

Diagnosis of vvIBD in wild birds, with no subsequent spread, would result in increased surveillance and monitoring of the wild bird population, and surveillance of commercial and low biosecurity poultry. The impacts were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level.

Diagnosis of vvIBD in a poultry flock would result in destruction of affected flocks and increased surveillance and monitoring of the poultry population. Impacts in the low biosecurity poultry population were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels but ***minor*** at the local level. Impacts in medium biosecurity commercial poultry were assessed as ***unlikely to be discernible*** at national and State/Territory levels but ***minor*** at the district/region level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

The impacts of a disease outbreak in the local wild bird population were assessed as ***unlikely to be discernible*** at all levels.

Movement controls to reduce spread of disease and delays in supply and demand for product and day old chicks could lead to considerable disruption to normal industry programs. Domestic trade and industry impacts in low biosecurity poultry were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels and ***minor*** at the district/region level, especially if commercial free-range poultry were involved. Impacts in medium biosecurity commercial poultry were assessed as ***unlikely to be discernible*** at national and State/Territory levels and ***minor*** at the district/region level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

The impacts of an identified outbreak of vvIBD in the local wild bird population on international trade were assessed as ***unlikely to be discernible*** at the national and State/Territory levels, but ***minor*** at the district/region level.

vvIBD is an OIE-listed disease, and international markets for poultry and poultry products are likely to be adversely affected if an outbreak were diagnosed in Australia. However, the Australian export market for poultry and poultry products is currently relatively small, and the number of countries which do not have vvIBDV is also relatively small, so the impacts of an outbreak are not likely to be as great as for other diseases such as avian influenza or Newcastle

disease. Impacts in low biosecurity poultry and medium biosecurity commercial poultry were assessed by the IRA team as ***unlikely to be discernible*** at the national and State/Territory levels and ***minor*** at the district/region level.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

vvIBD is unlikely to affect native birds, and indirect impacts on the environment are unlikely to be significant. The impacts of an outbreak of vvIBD in all susceptible exposure groups on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impacts of a disease outbreak in the local wild bird population were assessed as ***unlikely to be discernible***.

Movement restrictions and declaration of quarantine areas will impact local communities. The impacts of a disease outbreak in low biosecurity poultry were assessed by the IRA team as ***unlikely to be discernible*** at the national and State/Territory levels, and ***minor*** at the district/region level. Similarly, the impacts of a disease outbreak in the medium biosecurity commercial poultry population were assessed as ***unlikely to be discernible*** at the national and State/Territory levels, and ***minor*** at the district/region level.

Outbreak Scenario 3

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads within a district/region, including to other exposure groups if applicable, and is eliminated) will be the same for all exposure groups within each impact level, no matter which exposure group has been directly exposed to exotic antigenic variant IBDV in imported chicken meat. This is because, by definition, the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

IBD is an OIE-listed disease, and the very virulent form is listed as a Category 4 disease under the Emergency Animal Disease Response Agreement. It is, therefore, classified as being mainly a disease associated with production losses. While there may be international trade losses and local market disruptions, they would not be expected to significantly affect the national economy. It is expected that a local outbreak of disease may be subject to eradication measures, but if the disease became more widespread, it would most likely be controlled by vaccination and increased biosecurity measures. Costs arising from the implementation and administration of the eradication program will be accounted for under indirect criterion 1. Large numbers of birds may be involved on commercial properties, and the birds may represent the owner’s sole source of income. The direct costs of dead birds will be larger for commercial enterprises than for backyard or aviary birds, but with prompt eradication, the costs will be limited to the local area. In addition to bird deaths, the health of birds may be affected by movement restrictions

leading to delayed marketing. The impacts at the national and State/Territory levels were assessed by the IRA team as ***unlikely to be discernible***. There may be ***minor*** impacts at the district/region level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

The impact of vvIBDV on the environment was assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

Spread of vvIBDV to a local population of poultry or caged birds would require implementation of eradication, surveillance and monitoring programs. The IRA team considered that while the impact on the national economy was ***unlikely to be discernible***, there may be ***minor*** impacts at the State/Territory level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Movement controls to reduce spread of disease and delays in supply and demand for product and day old chicks could lead to considerable disruption to normal industry programs. If flocks in a declared quarantine area are not depopulated, the costs of keeping the birds beyond normal market age could be substantial. The impacts nationally were assessed by the IRA team as ***unlikely to be discernible,*** but movement and marketing restrictions will initially lead to ***minor*** impacts on domestic trade and industry at the State/Territory level until effective vaccination regimes are implemented.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

vvIBD is an OIE-listed disease, and international markets for poultry and poultry products are likely to be adversely affected if an outbreak were diagnosed in Australia. However, the Australian export market for poultry and poultry products is currently relatively small. The impacts on international trade markets were assessed by the IRA team as ***minor*** at the district/region level.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of an outbreak of vvIBD on the environment were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Movement restrictions and declaration of quarantine areas will impact local communities. The impact of a localised outbreak of vvIBD on communities was assessed by the IRA team as ***unlikely to be discernible*** at the national and State/Territory levels, but is likely to be ***minor*** at the district/region level.

Outbreak Scenario 4

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia) will be the same for all exposure groups within each impact level, no matter which exposure group has been directly exposed to exotic antigenic variant IBDV in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

If the disease spreads more widely through medium biosecurity commercial poultry and low biosecurity poultry, there will be significant losses of birds and production, and the effects will be more widespread than described under outbreak scenario 3. At the national level, the impacts were assessed by the IRA team as ***unlikely to be discernible***. Impacts were assessed as ***minor*** at the State/Territory level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

The impacts of a general outbreak of vvIBD on the environment were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

Diagnosis of vvIBD in poultry flocks would result in destruction of affected flocks and increased surveillance and monitoring of the poultry population in both affected and apparently unaffected areas. There will be additional costs of disposal of contaminated products and packaging and of extensive decontamination. Intensive vaccination programs may be instituted in an effort to control the disease. If vvIBDV spreads to a more general population of poultry, the impact at the national level was assessed by the IRA team as ***minor***.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Movement controls to reduce spread of disease and delays in supply and demand for product and day old chicks could lead to considerable disruption to normal industry programs. If flocks in a declared quarantine area are not depopulated, the costs of keeping the birds beyond normal market age could be substantial. The IRA team considered that movement and marketing restrictions will initially lead to ***minor*** impacts on domestic trade and industry at the national level until effective vaccination regimes are implemented.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

Impacts of a more general outbreak of vvIBD on international trade were assessed by the IRA team as ***unlikely to be discernible*** at the national and State/Territory levels, and ***minor*** at the district/region level.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impact of an outbreak of vvIBD on the environment was assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impact of a general outbreak of vvIBD on communities was assessed by the IRA team as ***unlikely to be discernible*** at the national and State/Territory levels, but is likely to be ***minor*** at the district/region level.

Conclusions – impact assessment

The above estimates for each of the impact criteria were entered into the simulation model, and the overall impact of each outbreak scenario was estimated. Results are shown in Table 40.

Partial annual risk estimate for each outbreak scenario

The partial annual likelihood of entry, exposure, establishment and spread were combined with the impacts of the relevant outbreak scenarios to provide an estimate of the partial annual risk associated with each outbreak scenario. The outcomes of this process are shown in Table 41.

Unrestricted risk estimate

The overall risk associated with the import of whole chicken carcasses was assessed as ***moderate*** for vvIBDV. This unrestricted risk estimate exceeds Australia's ALOP, and therefore risk management was deemed necessary.

Direct impact on human life or health

IBDV is not known to affect humans and is not considered to be a threat to public health.

Table 40. Impacts of each outbreak scenario

Exposure group	Outbreak scenario	Impact
Wild birds	Scenario 1	Negligible
	Scenario 2	Very low
Low biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Very low
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Very low
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
	Total Scenario 3	Low
	Total Scenario 4	Moderate

Table 41. Partial annual risk (PAR) of each outbreak scenario

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	High	Negligible	Negligible
	Scenario 2	Very low	Very low	Negligible
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Very low	Very Low
Medium biosecurity poultry	Scenario 1	Negligible	Negligible	Negligible
	Scenario 2	Negligible	Very low	Negligible
Non-avian species	Scenario 1	Zero	Zero	Zero
	Scenario 2	Zero	Zero	Zero
	Total Scenario 3	High	Low	Low
	Total Scenario 4	High	Moderate	Moderate

Risk Assessment (Exotic Antigenic Variant Strains of IBDV)

For the purposes of this risk assessment, exotic variant strains are defined as variant strains that are antigenically and genetically different from those that exist in Australia, and include United States variant strains. Due to the similarities between vvIBDV and exotic antigenic variant IBDV, only those variables that are expected to differ from the previous assessment will be specifically addressed here.

Release assessment

Rel_1: Selection of source flock (between flock prevalence)

With similar reasoning as for vvIBDV, the IRA team assessed Rel_1 as ***moderate***.

Rel_2: Infection detected through flock surveillance and the flock withdrawn from slaughter

As for vvIBDV (i.e. ***extremely low***).

Rel_3: Selection of an infected chicken from an infected flock (within-flock prevalence)

IBD is a highly contagious disease and, in an outbreak, most birds within a shed are likely to be infected. The virus persists in some tissues, including the intestinal tract, for several weeks. The IRA team considered that the likelihood of selection of an infected chicken from a flock infected with exotic antigenic variant IBDV was ***moderate***.

Rel_4: Background cross-contamination rate

Variant IBDV persists in the digestive tract and bursa of Fabricius of infected chickens (Elankumaran, Heckert, and Moura 2002). The IRA team assessed that the likelihood of a carcass being contaminated with potentially infective material from other birds, especially from the digestive tract or the bursa, was ***moderate***.

Rel_5: Likelihood that an uninfected carcass will be contaminated with disease agent during slaughter and processing

As discussed in the Method for Risk Assessment (page 58, Part B), Rel_4 was used to calculate Rel_{5a} (the likelihood that an uninfected carcass from an infected flock will become contaminated during slaughter and processing) and Rel_{5b} (the likelihood that an uninfected carcass from an uninfected flock will become contaminated during slaughter and processing).

For exotic antigenic variant IBD, Rel_{5a} was calculated as ***low***, and Rel_{5b} was calculated as ***low***.

Rel_6: The likelihood that the carcass of a bird that was *infected* before slaughter will be removed as a result of inspections before or during processing

No obvious post-mortem lesions are likely to be present during slaughter and processing. The IRA team considered that the rejection rate of infected carcasses would be equal to the background rejection rate of 0.75%.

Rel_7: The likelihood that the carcass of a bird that was *not infected* before slaughter will be removed as a result of inspections before or during processing

The likelihood that an uncontaminated/uninfected carcass will be removed during processing inspections was considered to be equal to the background rejection rate of 0.75% as described in the Method section (page 59, Part B).

Rel_8: Inactivation of the agent during further processing, storage, handling and transport

As discussed above (for vvIBDV) the IRA team considered that Rel_8 is ***negligible***.

Conclusions – Release assessment

After inserting the above estimates into the simulation model, and using the outputs from the model as a guide, the IRA team concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a ***moderate*** likelihood that imported chicken meat would be infected or contaminated with exotic antigenic variant IBD virus.

Exposure assessment

Exposure Group 1: Wild birds

Of the steps identified as determinants in the infection of wild birds by exposure to scraps from an imported contaminated chicken carcass, $WB_{agentsurvival}$ and $WB_{infectivedose}$ are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (page 67-86, Part B).

$WB_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by a wild bird

As discussed above (for vvIBDV) the IRA team considered that $WB_{agentsurvival}$ is ***high***.

$WB_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a wild bird is sufficient to produce infection

While the establishment of IBDV infection has not been reported in wild birds, wild birds have developed antibody following exposure to the virus, presumably due to transient infection. Taking account of this information, the IRA team considered that there was an ***extremely low*** likelihood that exotic antigenic variant IBDV would infect a wild bird consuming the contaminated meat scraps.

Exposure Group 2: Low biosecurity poultry

Of the steps identified as determinants in the infection of low biosecurity poultry by exposure to material from an imported contaminated chicken carcass, $BP_{agentsurvival}$, $BP_{infectivedose}$, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$BP_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by low biosecurity poultry

As for vvIBDV (i.e. ***certain***).

$BP_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a low biosecurity bird is sufficient dose to produce infection

Transmission of variant E IBDV to susceptible chickens was demonstrated by the feeding of bursal tissue but not muscle from vaccinated challenged SPF chickens (Australian Animal Health Laboratory 2004). The presence of infectious virus in the bursa was considered by the IRA team as indicative of a gastrointestinal infection that had a high likelihood of contaminating carcasses during commercial processing. It was therefore assumed that there is a high likelihood that exotic antigenic variant IBDV would infect chickens of susceptible age that consumed meat scraps contaminated during processing. However, chickens over the age of 15–20 weeks, which make up the majority of backyard flocks, are much less likely to become infected with exotic antigenic variant IBDV; clinical disease would occur only in those flocks with young birds.

In combining these factors, the IRA team considered that the likelihood of infecting susceptible flocks from the consumption of contaminated meat scraps was high, but that such flocks would be in the minority. The overall likelihood that low biosecurity poultry would be infected with exotic antigenic variant IBDV as a result of consuming the contaminated chicken meat scraps was therefore considered to be ***low***.

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As for vvIBDV (i.e. ***negligible***).

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As for vvIBDV (i.e. ***negligible***).

Exposure Group 3: Medium biosecurity commercial poultry

Of the steps identified as determinants in the infection of medium biosecurity commercial poultry by exposure to material from an imported contaminated chicken carcass, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As discussed above (Exposure Group 2: Low biosecurity poultry), the IRA team considered that the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with exotic antigenic variant IBDV was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As discussed above (low biosecurity poultry), the likelihood of the final poultry ration containing an oral infectious dose of exotic antigenic variant IBDV was considered by the IRA team to be ***negligible***.

Exposure Group 4: Non-avian species

As discussed above, $NAS_{agentsurvival}$ was considered to be equal to $WB_{agentsurvival}$.

Although virus was detected for two days in the faeces of an experimentally exposed dog, there is no evidence that IBDV occurs in non-avian species in nature. Therefore, this exposure group was not considered further in relation to this disease. $NAS_{infectivedose}$ was set to a value of zero.

Conclusions – Exposure assessment

Using the simulation model as a guide, the partial likelihood of exposure for each of the exposure groups was determined, taking the above estimates of the exposure variables into account. A summary of the outcomes determined by the IRA team is set out in Table 42.

Table 42. Partial likelihoods of exposure (PLE)

Exposure group	PLE
Wild birds	Extremely low
Low biosecurity poultry	Very low
Medium biosecurity poultry	Negligible
Non-avian species	Zero

Consequence assessment

Establishment and spread was considered in the context of the outbreak scenarios, described in the Method for Risk Assessment.

Estimating the likelihood of each outbreak scenario

The partial likelihood of establishment and spread (PLES) of exotic antigenic variant strains of IBDV for the different exposure groups is described below. Four outbreak scenarios were considered relevant:

1. Disease agent does not establish or is not recognised within the directly exposed population

2. Disease agent establishes within the directly exposed population, is identified and is eliminated by human efforts or by natural means

3. Disease agent establishes in the directly exposed population, spreads within a district/region, including into other exposure groups if applicable, and is eliminated by human action or by natural means

4. Disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia.

Wild birds

For reasons discussed previously (see vvIBDV), outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 43).

Table 43. Estimated partial likelihood of establishment and spread (PLES) values for exotic antigenic variant strains of IBDV in wild birds

Outbreak scenario 1:	$PLES_{(wb)}1$	**High**
Outbreak scenario 2:	$PLES_{(wb)}2$	**Extremely low**
Outbreak scenario 3:	$PLES_{(wb)}3$	**Extremely low**
Outbreak scenario 4:	$PLES_{(wb)}4$	**Extremely low**

Low biosecurity poultry

For reasons discussed previously (see vvIBDV), the IRA team considered that the most likely outcome of infection in a backyard poultry flock would be a single or a few isolated occurrences of infection.

The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 44).

Table 44. Estimated partial likelihood of establishment and spread (PLES) values for exotic antigenic variant strains of IBDV in low biosecurity poultry

Outbreak scenario 1:	$PLES_{(bp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(bp)}2$	**Low**
Outbreak scenario 3:	$PLES_{(bp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(bp)}4$	**Low**

Medium biosecurity commercial poultry

For reasons discussed previously (see vvIBDV), outbreak scenario 4 (the organism becomes endemic) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 45).

Table 45. Estimated partial likelihood of establishment and spread (PLES) values for exotic antigenic variant strains of IBDV in medium biosecurity commercial poultry

Outbreak scenario 1:	$PLES_{(cp)}1$	**Extremely low**
Outbreak scenario 2:	$PLES_{(cp)}2$	**Low**
Outbreak scenario 3:	$PLES_{(cp)}3$	**Low**
Outbreak scenario 4:	$PLES_{(cp)}4$	**Moderate**

Non-Avian Species

As stated above, this exposure group was not considered further in relation to this disease.

Conclusion – Likelihood assessments

The estimates for the likelihood of release, the partial likelihoods of exposure for each of the exposure groups, and the partial likelihood of establishment and spread for each of the outbreaks scenarios were combined with the expected volume of trade using the simulation model. This allowed the calculation of partial annual likelihood of entry, exposure, establishment and spread for each of the outbreak scenarios. The results of this calculation are shown in Table 46.

Table 46. Partial annual likelihood of entry, exposure, establishment and spread (PALEEES) for the outbreak scenarios

Exposure group	Outbreak scenario	PALEEES
Wild birds	Scenario 1	High
	Scenario 2	Very low
Low biosecurity poultry	Scenario 1	High
	Scenario 2	High
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
Total all exposure groups	Scenario 3	High
Total all exposure groups	Scenario 4	High

Estimating the impacts associated with each outbreak scenario

For each outbreak scenario the direct and indirect impacts of exotic antigenic variant IBD were estimated at the national, State or Territory, district/region and local levels, as described in the Methods section (pages 90-95, Part B).

The IRA team considered that the likelihood of exotic antigenic variant strains of IBDV affecting non-avian species (exposure group 4) was remote. Therefore, the impacts of exotic antigenic variant strains of IBDV occurring in this exposure group were not considered further.

Outbreak Scenario 1

By definition, this outbreak scenario means that the disease agent does not establish in the initially exposed population, or does not cause sufficient disease to lead to investigation and recognition of the infection. Therefore the impacts will be ***unlikely to be discernible*** for all exposure groups and all criteria.

Outbreak Scenario 2

The impacts of this outbreak scenario (disease agent establishes within the directly exposed population, and is identified and eliminated) will differ between exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

Since wild birds do not play a significant part in production, direct economic loss from death of wild birds is not measurable. Other impacts from the death of wild birds will be considered under criterion 2: the environment. The direct impacts of disease on wild birds were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

The specific mortality rate due to exotic antigenic variant strain of IBDV is reported to be less than 5%. However, the IRA team considered that losses due to decreased feed conversion efficiency, weight loss, secondary infections, impaired response to subsequent vaccinations, and reduced egg production and hatchability in breeder flocks may be substantial. An outbreak of exotic antigenic variant IBD contained within the low biosecurity poultry population may result in losses to individual owners, especially if commercial free-range poultry are involved. The impacts of such losses on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at the national, State/Territory and district/region levels but ***minor*** at the local level.

Loss of birds and production within a single shed of medium biosecurity commercial poultry on the affected farm were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels but ***minor*** at the local level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

Although antibodies to IBDV have been demonstrated in gulls and crows, clinical disease has not been demonstrated in these species. The impacts of an outbreak of IBD in wild birds on the environment, if it occurred, were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

The impacts of an outbreak of exotic antigenic variant IBD in low biosecurity poultry on the environment were assessed by the IRA team as ***unlikely to be discernible*** at all levels. Similarly the impacts of an outbreak in or medium biosecurity commercial poultry were assessed as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

Diagnosis of exotic antigenic variant IBD in wild birds, with no subsequent spread, would lead to increased surveillance of the wild bird population, and surveillance and monitoring of commercial and low biosecurity poultry. The impacts were assessed by the IRA team as ***unlikely to be discernible*** at the national, State/Territory and district/region levels, but ***minor*** at the local level.

Diagnosis of exotic antigenic variant IBD in a poultry flock would result *in attempts at eradication of the virus* and increased surveillance and monitoring of the poultry population. Impacts in the low biosecurity poultry population were assessed by the IRA team as ***unlikely to be discernible*** at the national, State/Territory and district/region levels but ***minor*** at the local level. Impacts in medium biosecurity commercial poultry were assessed as ***unlikely to be discernible*** at the national and State/Territory levels but ***minor*** at the district/region level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

The impacts of a disease outbreak in the local wild bird population were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Domestic trade and industry impacts in low biosecurity poultry were assessed by the IRA team as ***unlikely to be discernible*** at the national and State/Territory levels and ***minor*** at the district/region level, especially if commercial free-range poultry were involved. Impacts in medium biosecurity commercial poultry were assessed as ***unlikely to be discernible*** at the national and State/Territory levels and ***minor*** at the district/region level, for similar reasons to those described under vvIBDV.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

The impacts of an identified outbreak of exotic antigenic variant IBD in any exposure group on international trade were assessed by the IRA team as ***unlikely to be discernible*** at the national and State/Territory levels, but ***minor*** at the district/region level, for similar reasons to those described under vvIBDV.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of an outbreak of exotic antigenic variant IBD in all exposure groups on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impacts of a disease outbreak in the local wild bird population were assessed as ***unlikely to be discernible***.

The impacts of a disease outbreak in low biosecurity poultry were assessed by the IRA team as ***unlikely to be discernible*** at the national and State/Territory levels, and ***minor*** at the district/region level. Similarly, the impacts of a disease outbreak in the medium biosecurity commercial poultry population were assessed as ***unlikely to be discernible*** at the national and State/Territory levels, and ***minor*** at the district/region level, for similar reasons to those described under vvIBDV.

Outbreak Scenario 3

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a district/region, and is eliminated) will be the same for all exposure groups, no matter which exposure group has been directly exposed to exotic antigenic variant IBDV in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

It is expected that a local outbreak of disease due to exotic antigenic variant IBDV would be subject to eradication measures, but if the disease became more widespread, it would most likely be controlled by increased biosecurity measures and vaccination. Costs arising from the implementation and administration of the eradication program will be accounted for under indirect criterion 1. Large numbers of birds may be involved on commercial properties, and the birds may represent the owner's major source of income. The direct costs of sick and dead birds will be larger for commercial enterprises than for backyard or aviary birds, but with prompt eradication, the costs will be limited to the local area. The impacts at the national and State/Territory levels were assessed by the IRA team as ***unlikely to be discernible***. There may be ***minor*** impacts at the district/region level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

The impact of exotic antigenic variant IBDV on the environment was assessed by the IRA team as ***unlikely to be discernible*** at all levels, since variant IBDV is not known to cause adverse effects on wild birds.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

Spread of exotic antigenic variant IBDV to a local population of poultry or caged birds would require implementation of eradication, surveillance and monitoring programs. While the impact

on the national and State/Territory economy was assessed as ***unlikely to be discernible***, there may be ***minor*** impacts at the district/region level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Movement controls to reduce spread of disease and delays in supply and demand for product and day old chicks could lead to considerable disruption to normal industry programs. The impacts at the national and State/Territory levels were assessed by the IRA team as ***unlikely to be discernible***. Movement restrictions associated with control and eradication programs will initially lead to ***minor*** impacts on domestic trade and industry at the district/region level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

If exotic antigenic variant IBDV were diagnosed in Australian poultry flocks, international markets for poultry and poultry products may be adversely affected. However, the Australian export market for poultry and poultry products is relatively small. The impacts on international trade markets were assessed by the IRA team as ***minor*** at the *local* level.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of an outbreak of exotic antigenic variant IBD with spread to commercial poultry were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impact of a localised outbreak of exotic antigenic variant IBD on communities was assessed by the IRA team as ***unlikely to be discernible*** at the national and State/Territory levels, but is likely to be ***minor*** at the district/region level for similar reasons as described in vvIBDV.

Outbreak Scenario 4

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a State/Territory, and becomes endemic in Australia) will be the same for all exposure groups, no matter which exposure group has been directly exposed to exotic antigenic variant IBDV in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

If exotic antigenic variant IBDV spreads more widely through medium biosecurity commercial poultry and low biosecurity poultry, significant production losses could occur. The effects will

be more widespread than described under scenario 3. At the national level, the impacts were assessed as ***unlikely to be discernible***. Impacts were assessed by the IRA team as ***minor*** at the State/Territory level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

The impacts of a general outbreak of exotic antigenic variant IBD on the environment were assessed by the IRA team as ***unlikely to be discernible*** at all levels, since variant IBDV is not known to cause adverse effects on wild birds.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

If exotic antigenic variant IBDV spreads more widely through medium biosecurity commercial poultry and low biosecurity poultry, changes to the vaccination program for breeders and layers would be required. Since vaccines currently used in Australia do not protect against exotic antigenic variant strains of IBDV, control by vaccination will be delayed until suitable vaccines are imported or locally developed and registered for use in Australia. The impact at the national level was assessed as ***unlikely to be discernible***. Impacts were assessed since variant IBDV is not known to cause adverse effects on wild birds as ***minor*** at the State/Territory level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Impacts of a more general outbreak of exotic antigenic variant IBD on domestic trade and industry were assessed since variant IBDV is not known to cause adverse effects on wild birds as ***unlikely to be discernible*** at the national level. Impacts at the State/Territory level were assessed as ***minor***, for reasons described under scenarios 2 and 3.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

Impacts of a more general outbreak of exotic antigenic variant IBD on international trade were assessed *by the IRA team* as ***unlikely to be discernible*** at the national and State/Territory levels, and ***minor*** at the district/region level.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impact of an outbreak of exotic antigenic variant IBD with spread to commercial poultry was assessed *by the IRA team* as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impact of a general outbreak of exotic antigenic variant IBD on communities was assessed by the IRA team as ***unlikely to be discernible*** at the national and State/Territory levels, but is likely to be ***minor*** at the district/region level.

Conclusions – impact assessment

The above estimates for each of the impact criteria were entered into the simulation model, and the overall impact of each outbreak scenario was estimated. Results are shown in Table 47.

Table 47. Impacts of each outbreak scenario

Exposure group	Outbreak scenario	Impact
Wild birds	Scenario 1	Negligible
	Scenario 2	Very low
Low biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Very low
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Very low
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
	Total Scenario 3	Very low
	Total Scenario 4	Low

Partial annual risk estimate for each outbreak scenario

The partial annual likelihood of entry, exposure, establishment and spread were combined with the impacts of the relevant outbreak scenarios to provide an estimate of the partial annual risk associated with each outbreak scenario. The outcomes of this process are shown in Table 48.

Unrestricted risk estimate

The overall risk associated with the import of whole chicken carcasses was assessed as ***low*** for exotic antigenic variant strains of infectious bursal disease virus. This unrestricted risk estimate exceeds Australia's ALOP, and therefore risk management was deemed necessary.

Direct impact on human life or health

IBDV is not known to affect humans and is not considered to be a threat to public health.

Table 48. Partial annual risk of each outbreak scenario

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	High	Negligible	Negligible
	Scenario 2	Very low	Very low	Negligible
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Very low	Very Low
Medium biosecurity poultry	Scenario 1	Negligible	Negligible	Negligible
	Scenario 2	Negligible	Very low	Negligible
Non-avian species	Scenario 1	Zero	Zero	Zero
	Scenario 2	Zero	Zero	Zero
	Total Scenario 3	High	Very low	Very Low
	Total Scenario 4	High	Low	Low

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Avian Salmonellosis

Salmonellae are Gram negative, non-sporogenic, facultative anaerobic bacteria belonging to the family *Enterobacteriaceae*. The genus *Salmonella* is divided into two species (*Salmonella enterica* and *Salmonella bongori*), several subspecies, and more than 2,400 serotypes (Gast 2003b). Some serovars are highly host-adapted, while others are considered pathogenic to a wide range of animals and birds. Infections in poultry can be divided into three categories:

1. Infections with non-motile serotypes *S.* Pullorum and *S.* Gallinarum

   These serotypes, which cause pullorum disease and fowl typhoid respectively, are generally host-specific for avian species, and are highly pathogenic for chickens and turkeys (Gast 2003b). Both serotypes are responsible for serious economic losses in poultry but are of little public health significance. Both *S.* Gallinarum and *S.* Pullorum are OIE-listed disease agents and have been eradicated from Australian commercial poultry flocks. Although listed separately in the Hazard List, these diseases will be discussed together because of their similarity in terms of epidemiology and management.

2. Infections with motile serotypes, including *S.* Enteritidis and *S.* Typhimurium

   These are not host-specific, and are found almost ubiquitously in domestic and wild animals, and humans (Gast 2003b). Infection in poultry is common but seldom causes acute systemic disease except in susceptible young birds (Gast 2003b). Contamination of poultry products (meat or eggs) may occur. These serotypes are primarily of concern as causes of food-borne disease in humans (Gast 2003b). Outbreaks may cause severe economic losses to poultry producers as a result of regulatory action, market restrictions and decreased consumption of poultry products. Multi-drug resistant (MDR) strains of *S.* Typhimurium such as DT104 are not present in livestock in Australia (Joint Expert Technical Advisory Committee on Antibiotic Resistance 1999). *S.* Enteritidis has rarely been isolated from poultry in Australia, and is subject to regulatory control. Isolations of *S.* Enteritidis from commercial poultry in Queensland in 2005 were investigated and the affected flocks are subject to ongoing control measures and intensive monitoring (K Bell, Safe Food, Queensland, Australia, pers. comm. May 2006). *S.* Enteritidis and MDR *S.* Typhimurium will be discussed together because of their similarity in terms of epidemiology.

3. Infection with other motile serotypes

   Arizonosis, caused by the poultry-adapted serotype of *S.* Arizonae is of particular significance in turkeys, and is exotic to Australia (Davos 2001).

Agent characteristics

In general, salmonellae are relatively resistant to environmental conditions and survive well in the presence of moisture. Inactivation occurs quickly on exposure to sunlight (Shivaprasad 2003), although in the natural environment, the organisms may be protected by organic material such as faeces and litter. In poultry sheds, for example, salmonellae have been shown to survive and multiply. *Salmonellae* survive for 140 days in liquid faeces and at least 180 days in sewage sludge. They grow at temperatures between 5 °C and 43 °C, and in pH range 4 to 9 (Gast 1997). *Salmonellae* survive freezing for at least 29 days in chicken meat and at least 390 days

on turkey skin at –20 °C, although there is a reduction in the number of viable cells (Mitscherlich and Marth 1984).

Salmonellae are readily inactivated by disinfectants but the presence of solids, the pH, and exposure time modify the sensitivity of the organisms. Hydrogen peroxide, acetic acid, lactic acid, and potassium sorbate, chlorine and trisodium phosphate have been used to reduce *Salmonella* levels on chicken carcasses, while chlorine, trisodium phosphate, ozone and formaldehyde are used to reduce levels in buildings (Gast 1997). Chlorine is rapidly deactivated on contact with the skin and therefore is not very effective against organisms on the surface of carcasses.

Salmonellae are generally quite susceptible to destruction by heat, although this is influenced by the fat and moisture content of the medium, the stage of growth of the organism, and the method and duration of heat application. Experimental studies on the time and temperature required for inactivation of *Salmonella* in chicken meat have produced varying results (Murphy et al. 2002; Murphy et al. 2004; Murphy et al. 2004; Schnepf and Barbeau 1989; Veeramuthu et al. 1998). A minimum internal temperature of 74 °C will reduce viable *Salmonella* in chicken meat by at least 7 logs, according to a draft report by the United States National Advisory Committee on Microbiological Criteria for Foods (National Advisory Committee on Microbiological Criteria for Foods 2006). Resistance to heat is increased by prior exposure to alkaline conditions, and decreased by prior refrigeration (Gast 1997). *Salmonellae* show a relatively high resistance to drying, smoking and salting (World Health Organization 1988). Extrapolating from the available literature, and for the purposes of this risk analysis, it will be assumed that heating of chicken meat to a core temperature of 70 °C for a minimum of 2.5 minutes will result in a 6 log reduction in *Salmonella* species of concern.

Gamma irradiation has been used to inactivate *Salmonella* in eggs (Schaffner et al. 1989; Matic et al. 1990), although a combination of heat and irradiation is reported to be more successful (Schaffner et al. 1989). Ultraviolet radiation and ultrasonic wave treatment have also been used to inactivate salmonellae in egg products (Gast 1997).

Salmonella Pullorum and *Salmonella* Gallinarum

Technical Information

Background

Pullorum disease and fowl typhoid are septicaemic bacterial diseases of chickens, turkeys and pheasants. Pullorum disease is caused by *Salmonella* Pullorum, and fowl typhoid is caused by *Salmonella* Gallinarum (Shivaprasad 2000). Although listed separately in the Hazard List, these diseases are commonly discussed together in standard texts, because of their similarity in terms of epidemiology and management (Shivaprasad 2000; Davies 2004). These two *Salmonella* species are distinguished from the remainder of the salmonellae, in that they are host-adapted and highly pathogenic for avian species, but are considered to pose little zoonotic risk (Davies 2004).

Both pullorum disease and fowl typhoid are OIE-listed diseases, and it is generally recommended that export of poultry and poultry products be made only from flocks known to be serologically negative for these diseases (Shivaprasad 2000). The OIE records that these two diseases have been eradicated from Australian commercial flocks (World Organisation for Animal Health (OIE) 2005). Australia undertook a successful campaign to eradicate Pullorum disease from commercial flocks, commencing in the 1930s (Beveridge and Hart 1985). Commercial breeding flocks are tested to ensure continued freedom from Pullorum disease. The Australian Salmonella Reference Laboratory has not recorded isolation of *S.* Pullorum from any Australian source since 1992 (D. Davos, Australian Salmonella Reference Centre, pers. comm. 2007). Fowl typhoid was last reported in Australia in 1952 (Seddon 1953; Beveridge and Hart 1985; Animal Health Australia 2007).

Agent taxonomy

The organisms are Gram-negative, non-sporogenic, non-motile, facultative anaerobic bacteria of the family *Enterobacteriaceae*, genus *Salmonella*, species *enterica,* subspecies *enterica* and serovar Gallinarum–Pullorum (Shivaprasad 2003). The bacteria belong to serogroup D according to the Kauffmann-White scheme (Shivaprasad 1997). These organisms are isolated primarily from chickens and other birds.

Agent characteristics

Both *S.* Pullorum and *S.* Gallinarum can survive in a favourable environment for a limited time, but are susceptible to heat, chemicals and adverse environmental conditions (Shivaprasad 2000). *S.* Gallinarum was killed within 10 minutes at 60 °C, within a few minutes in direct exposure to sunlight, and in 3 minutes by 1:1000 phenol (Shivaprasad 2003). *S.* Gallinarum can survive in faeces from infected birds for up to 37 days when kept indoors and 31 days in the open (Smith 1955). The organism has survived and retained pathogenicity for more than 148 days in frozen liver, for two weeks in liver stored at 7 °C and for 93 days in frozen water despite being thawed out twice in that period. When subject to daily freezing and thawing the organism remained viable in water for up to 43 days (Orr and Moore 1953). *S.* Gallinarum was

resistant to high concentrations of hypochlorite in the presence of organic material (Berchieri and Barrow 1995).

Epidemiology

Chickens are the natural hosts for both *S.* Pullorum and *S*. Gallinarum. Naturally-occurring outbreaks have occurred in turkeys, pheasants, guinea fowl, quail, sparrows, and parrots (Shivaprasad 2003). Pullorum disease has been described in canaries and bullfinches, and fowl typhoid in ring doves, ostriches and peafowl (Shivaprasad 2003). Ducks, geese and pigeons appear to be resistant to *S.* Gallinarum, and ducks appear to be resistant to experimental infection with *S*. Pullorum (Shivaprasad 2003). Pullorum disease is becoming a significant disease of pheasant chicks in the United Kingdom (Pennycott and Duncan 1999).

Pullorum disease has been described as a naturally-occurring or experimental infection in several species of mammals, including wild rats (Badi, Iliadis, and Sarris 1992; Shivaprasad 2003). Experimentally infected rats shed *S*. Gallinarum in the faeces for at least 121 days without showing clinical signs of infection (Badi, Iliadis, and Sarris 1992). Human salmonellosis caused by *S.* Pullorum has been reported occasionally, with the disease generally being transient and recovery prompt (Shivaprasad 2003).

Pullorum disease and fowl typhoid are widely distributed throughout the world. The diseases have been eradicated from commercial poultry via a test-and-slaughter method of disease control in the United States, Canada, Australia, Japan and most countries in Western Europe (Shivaprasad 2000; Rabsch et al. 2000; Audisio and Terzolo 2002). The last reported outbreak of fowl typhoid in Australia was in 1952 (Animal Health Australia 2001; Hart 1985). Compulsory monitoring for pullorum disease commenced in Australia in the 1930s and the disease has not been seen in commercial flocks for many years (Hart 1985). The last outbreak of pullorum disease in commercial poultry in the United States was in 1991 (Johnson, Davis, and Goldsmith 1992), and fowl typhoid has not been reported in the United States since 1980 (Shivaprasad 2000). An outbreak of fowl typhoid occurred in commercial poultry in Denmark in 1992 (Christensen et al. 1994). In Mexico, Central and South America, Africa and the Indian subcontinent the diseases are considered endemic, while the frequency of disease in many other parts of the world, including Eastern Europe, Russia, the People’s Republic of China and South-East Asia is unknown (Shivaprasad 2000). The last reported occurrence of pullorum disease in New Zealand was in 1985, and there have been no reports of fowl typhoid occurring in that country (World Organisation for Animal Health (OIE) 2005).

While commercial flocks may be free of pullorum disease and fowl typhoid, the same is not necessarily true of smaller, non-commercial flocks. Pullorum disease has been reported in small (backyard) poultry flocks in the United Kingdom and the United States (Curtis and Boachie 1982; Erbeck, McLaughlin, and Singh 1993; Shivaprasad 2003) and in pheasants in the United Kingdom (Pennycott and Duncan 1999).

The major route of transmission of *S*. Pullorum and *S*. Gallinarum is vertically via transovarial transmission, the organism being present in up to 33% of eggs laid by an infected hen (Shivaprasad 2003). Bacteria can localise in the ovules before ovulation, or can contaminate the ovum following ovulation, with the former mode considered to be the more important (Shivaprasad 2003). Transmission via the egg can also occur through shell penetration but has been reported to be of minor importance (Williams, Dillard, and Hall 1968). A carrier state exists, with infected birds capable of infecting the next generation through vertical transmission, and other birds through faecal shedding of organisms (Shivaprasad 2003).

Horizontal transmission between chicks or pullets is an important means of dissemination of both organisms. Fluff from infected chicks is heavily contaminated, and bacteria may be disseminated throughout the incubator or brooder leading to lateral transmission between chicks (Wray and Davies 2001). Faeces from infected birds are an important source of infection, while other routes of infection include fomites (contaminated feed, water, litter, attendants), cannibalism and egg eating (Shivaprasad 2003). Wild birds, other animals and flies may spread the organisms mechanically (Shivaprasad 2000), although the importance of wild birds in the spread of these non-motile *Salmonellae* has been questioned (Wilson and MacDonald 1967).

Pullorum disease is principally a disease of chicks and poults, mortality being highest within the first two to three weeks of life (Shivaprasad 2003). Mortality and wasting of adult chickens can be caused by pullorum disease (Erbeck, McLaughlin, and Singh 1993). Fowl typhoid, frequently referred to as a disease of growing or adult birds, can cause clinical signs in young chickens that are indistinguishable from those seen in pullorum disease (Wray and Davies 2000). Mortality rates in chicks of up to 26% have been recorded during the first month of life in association with fowl typhoid (Shivaprasad 1997). Mortality due to pullorum disease and fowl typhoid often begins in the hatchery, but with fowl typhoid the mortalities may persist to laying age (Shivaprasad 1997).

The incubation period of *S*. Pullorum varies with the route of infection and the age and condition of the host. With egg-transmission, moribund and dead chicks may be found in the incubator shortly after hatching, although in some cases signs of disease are not seen for 5–10 days after hatching. In adult birds, body temperature may increase within 2–3 days of exposure, followed by clinical signs and possibly death within four to 10 days (Shivaprasad 2003). Clinical signs of fowl typhoid were seen at three to six days after oral inoculation in one study (Mdegela et al. 2002), but not until eight days post-inoculation in another (Jones et al. 2001).

It is difficult to determine, from the literature, the flock prevalence of pullorum disease and fowl typhoid in countries where disease occurs in commercial flocks. Of 150 meat-chicken-breeder flocks in Pakistan tested by a rapid agglutination test using stained pullorum antigen, 75% of flocks were positive. The prevalence of carriers on these farms was 4.63% (Javed and Hameed 1989). However, a number of other *Salmonella* species may cross-react with pullorum antigen, leading to false positive results and an overestimate of the true prevalence of disease (see sections on immunology and diagnosis below) (Waltman and Horne 1993). Within-flock seroprevalence varied from 16-92% in three commercial farms in Zambia (Hasegawa et al. 1999).

While these diseases have been eradicated from commercial poultry in many countries, they may still be present in backyard or small poultry flocks (Curtis and Boachie 1982; Erbeck, McLaughlin, and Singh 1993; Shivaprasad 2003). Serosurveys disclosed pullorum reactors in 2.4% and <4% of wild turkeys in two separate areas of the United States (Hensley and Cain 1979; Charlton 2000).

Clinical Signs

Chicks and poults show signs of depression, droopy wings, huddling, diarrhoea, ruffled feathers and somnolence, and may show laboured breathing. Deaths may be observed in the incubator, or may be delayed for five to ten days, with the peak of mortality occurring in birds two to three weeks of age. Other clinical signs include blindness and swelling of joints. Survivors may show poor weight gain and poor feathering (Shivaprasad 2003).

In growing and mature birds, decline in feed consumption and other non-specific clinical signs may be seen, including ruffled feathers and a droopy appearance. In acute infections, depression, anorexia, diarrhoea, dehydration and loss of weight may occur, with decreased egg production, fertility and hatchability in laying flocks. Atypical clinical signs occurred in two backyard flocks infected with pullorum disease, with sudden deaths of adult birds occurring in one flock, and chronic wasting with high mortality in another, in the absence of clinical disease in chicks (Erbeck, McLaughlin, and Singh 1993). In some infected flocks, clinical signs may be inapparent (Shivaprasad 2000). Carriers persist following infection, even in the absence of obvious clinical disease.

Mortality rates in chicks vary from 0–100% with pullorum disease, and from 10–93% with fowl typhoid (Shivaprasad 2003). Mortality is less common in mature birds, although growing and mature turkeys appear to be more susceptible than chickens to fowl typhoid.

Pathogenesis

The pathogenesis of pullorum disease and fowl typhoid is not completely understood (Shivaprasad 2000). Host-specificity of *S.* Pullorum and *S.* Gallinarum has been related to lack of adherence factors for some epithelial cell types, with adherence being a necessary pre-requisite to invasion (Wilson et al. 2000). Both organisms replicate mainly in cells of the reticuloendothelial system, rather than the intestinal tract, despite the fact that infection is via the faecal-oral route (Berchieri et al. 2001). *S.* Typhimurium and *S.* Enteritidis infection induces a strong inflammatory response in the intestine, which may limit the spread of these salmonellae largely to the gut. *S.* Gallinarum, however, does not induce an inflammatory response and may not be limited by the immune system to the intestinal tract, leading to severe systemic disease (Kaiser et al. 2000). *S.* Pullorum preferentially targets the bursa of Fabricius before eliciting intestinal inflammation (Henderson, Bounous, and Lee 1999). *S.* Gallinarum appears to invade the caecal tonsils and Peyer's patches during the early stages of fowl typhoid before leading to septicaemia (Jones et al. 2001). Oral infection leads to septicaemia, and bacteria can be isolated from multiple organs, including skeletal muscle, within a week of experimental oral inoculation with *S.* Pullorum (Wigley et al. 2001). At five weeks post-inoculation, organisms could still be recovered from spleen, heart, airsacs and bone marrow of some birds. *S.* Pullorum persisted in the spleen and reproductive tract of some chickens for 42 weeks (Wigley et al. 2001).

Pathology

In peracute cases of pullorum disease and fowl typhoid, there may be no gross lesions on post-mortem examination. Enlarged and congested liver, spleen and kidney may be seen in acute cases in young chicks, with fibrinous exudate in the pericardium and peritoneum. White nodules may be present in the heart, liver, pancreas and gizzard, and there may be caseous cores in the caecal lumen. Some birds exhibit swollen joints with increased volumes of creamy synovial fluid. While similar lesions can be present in adult birds, post-mortem findings may be minimal, with lesions confined to the reproductive tract (Shivaprasad 2000). Turkeys show similar lesions to chickens, but may, in addition, show intestinal ulceration and ascites.

Bobwhite quail infected with *S.* Pullorum show similar gross post-mortem lesions to chickens (Buchholz and Fairbrother 1992). Distended yolk sacs, nodules of the lungs, caecum and rectum, and caseous cores in the caeca were the most common post-mortem findings in

pheasants infected with *S*. Pullorum (Pennycott and Duncan 1999). The causative bacteria could be isolated from most internal organs.

Immunology

Chicks infected orally at four days of age may not produce agglutinating antibodies until 20–40 days of age, with maximum antibody production occurring at 100 days. Adult birds, however, produce antibodies within three to ten days after infection (Shivaprasad 2003). Some genetic types of *S*. Pullorum appear to induce a poor humoral response, leading to low levels of seroconversion (Dodson et al. 1999). Diagnostic tests for flock infection rely on the presence of agglutinating antibodies; it is therefore possible that an infected flock could show negative test results immediately following infection, and repeated tests are necessary to prove freedom from infection (Shivaprasad 2000).

Because pullorum disease has been eradicated from commercial flocks in many developed countries, there is little incentive for vaccine production against this disease (Shivaprasad 2003). Attenuated live vaccines against *S*. Gallinarum are available, and may be used in developing countries to help control fowl typhoid. The vaccine most widely used is made from the rough 9R strain (Davies 2004). The vaccine organisms can survive many months in inoculated birds and can be transmitted via the egg (Davies 2004), although this was not demonstrated in a clinical trial using the vaccine in commercial layers in the Netherlands (Feberwee et al. 2001). Vaccination may reduce flock losses but will not prevent infection with field strains of *S*. Gallinarum (Davies 2004). A 9R vaccine strain of *S*. Gallinarum is being evaluated in the Netherlands as a method of controlling *S*. Enteritidis in egg-producing flocks (Feberwee et al. 2001).

Diagnosis

Definitive diagnosis of pullorum disease or fowl typhoid requires the isolation and identification of the causative organism. In recently infected chicks and poults, the organisms can be isolated from most body tissues, although the liver, spleen, yolk sac, and caeca are the preferred organs for culture (Shivaprasad 2000). In adult birds with lesions of the reproductive tract, the ovaries and testes can be cultured.

Serological tests used to detect pullorum disease and fowl typhoid include the macroscopic tube agglutination test, rapid serum test, stained antigen whole blood test and micro agglutination test using tetrazolium-stained antigens (Shivaprasad 2000). These tests can cross-react with the sera from birds infected with other salmonellae, including *S*. Enteritidis, *S*. Typhimurium, and *S*. Heidelberg, and positive results should be followed by bacterial culture of reactor birds. Non-Gallinarum and non-Pullorum reactors may vary from a few birds to 40% of a flock (Shivaprasad 2000).

Other tests used for diagnosis include ELISA and dot immunobinding assay for detecting serological responses.

Transmission in chicken meat

Because pullorum disease and fowl typhoid are septicaemic diseases, organisms may localise in a wide range of body tissues. *S*. Pullorum was isolated from all examined body tissues, including skeletal muscle and bone marrow, one week after oral inoculation, and from bone marrow, heart, liver and spleen 5 weeks after inoculation (Wigley et al. 2001). The organisms

may persist in internal organs, such as the reproductive tract and spleen for prolonged periods (Shivaprasad 2000; Wigley et al. 2001; Orr and Moore 1953). In chronically affected birds, organisms may be isolated from reproductive tissue, peritoneum, liver, intestines, synovial tissue and other sites (Shivaprasad 2000). Contamination of the carcass from the alimentary tract may also occur during processing. *S*. Pullorum/Gallinarum have been isolated from the meat of poultry carcasses at processing plants (Zagaevskii 1980). *S*. Gallinarum can survive for up to six months on frozen poultry carcasses (Georgiev, Zahariev, and Kaloyanov 1978). Thus there is potential for transmission of *S*. Pullorum and *S*. Gallinarum through exposure to carcasses of poultry from infected flocks.

Quarantine significance

Both pullorum disease and fowl typhoid are OIE-listed diseases.

Pullorum disease and fowl typhoid have been eradicated from Australian commercial poultry. Pullorum has not been recently reported from any Australian source, and fowl typhoid was last reported in Australia in 1952. Fowl typhoid is notifiable in Victoria and New South Wales, and pullorum disease is notifiable in Victoria, New South Wales, South Australia, Tasmania and Western Australia. Fowl typhoid and pullorum disease are not included in the Emergency Animal Disease Response Agreement.

Despite the eradication of pullorum disease from commercial flocks in the United States, an outbreak did occur in 1990–91, which eventually involved 19 breeder flocks and more than 260 grower facilities. Exact costs of the outbreak are not available, but control involved the eradication and replacement of a grandparent line, parent flocks and growing birds in several states (Shivaprasad 2000). Because the disease is transmitted both horizontally and vertically, investigation of such an outbreak can be extremely complex (Johnson, Davis, and Goldsmith 1992), and the outbreak can spread widely before it is brought under control. Although pullorum disease and fowl typhoid are of no public health significance, disease in commercial flocks can lead to restricted marketing and trade opportunities.

Risk Assessment

Release assessment

Rel_1: Selection of source flock (between flock prevalence)

Pullorum disease and fowl typhoid are subject to official control programs in many countries that have intensive poultry industries. In those countries in which the disease is endemic, the prevalence of infection is difficult to estimate. For an unrestricted risk estimate, it was assumed that the prevalence is at the highest sustainable level in an endemically infected country or zone (see Method for Risk Assessment). Up to 75% of flocks may be infected in some countries (Javed and Hameed 1989), therefore, the likelihood that a source flock will be infected with pullorum disease or fowl typhoid at the time of slaughter was estimated by the IRA team to be ***high***.

Rel_2: Infection detected through flock surveillance and the flock withdrawn from slaughter

Clinical signs of pullorum disease or fowl typhoid infection may be inapparent (Shivaprasad 2000), and carriers may persist after clinical signs and mortalities have ceased. A flock surviving an outbreak of pullorum disease or fowl typhoid would most likely be sent to slaughter at the appropriate time. The likelihood that an infected flock will be withheld from slaughter was assessed by the IRA team as ***extremely low***.

Rel_3: Selection of an infected chicken from an infected flock (within-flock prevalence)

Pullorum disease and fowl typhoid are contagious diseases and, in an outbreak, many birds within a shed are likely to be infected. Organisms persist in several tissues, including liver, heart, spleen and bone marrow, for at least five weeks after infection (Wigley et al. 2001). If an infected flock was sent to slaughter, the likelihood that a selected individual chicken will be infected was assessed by the IRA team as ***moderate***.

Rel_4: Background cross-contamination rate

This likelihood represents the background cross-contamination rate during slaughter and processing and applies to cross-contamination with material, infectious or otherwise, from other carcasses, including those tissues or materials in which the disease agent tends to localise.

S. Gallinarum and *S*. Pullorum persist in the liver, spleen and other organs of infected chickens, and have been detected on the carcasses of chickens at processing plants. The organisms are resistant to the effects of chlorine in the presence of organic matter. The IRA team considered the likelihood of a carcass being contaminated with potentially contaminated material from other birds, especially from the digestive tract, is ***moderate***.

Rel_5: Likelihood that an uninfected carcass will be contaminated with the disease agent during slaughter and processing

As discussed in the Method for Risk Assessment (page 58, Part B), Rel_4 was used to calculate Rel_{5a} (the likelihood that an uninfected carcass from an infected flock will become contaminated during slaughter and processing) and Rel_{5b} (the likelihood that an uninfected carcass from an uninfected flock will become contaminated during slaughter and processing).

For *S*. Pullorum, Rel_{5a} was calculated as ***low***, and Rel_{5b} was calculated as ***low***.

For *S*. Gallinarum, Rel_{5a} was calculated as ***low***, and Rel_{5b} was calculated as ***low***.

Rel_6: The likelihood that the carcass of a bird that was *infected* before slaughter will be removed as a result of inspections before or during processing

Carcasses with obvious pathology would most likely be detected and removed during processing. However, birds infected with these bacteria may show no gross pathological lesions that would prompt removal from the processing line. The likelihood that a contaminated/infected carcass will be removed during processing inspections was assessed by the IRA team as ***very low***.

Rel_7: The likelihood that the carcass of a bird that was *not infected* before slaughter will be removed as a result of inspections before or during processing

The likelihood that an uncontaminated/uninfected carcass will be removed during processing inspections was considered to be equal to the background rejection rate of 0.75% as described in the Method section (page 59, Part B).

Rel_8: Inactivation of the agent during further processing, storage, handling and transport

S. Pullorum and *S*. Gallinarum can persist for weeks or months in carcasses at low temperatures. The IRA team concluded that the likelihood of inactivation of the bacteria during further processing, storage, handling and transport is ***extremely low***.

Conclusions – Release assessment

After inserting the above estimates into the simulation model, and using the outputs from the model as a guide, the IRA team concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a ***moderate*** likelihood that imported chicken meat would be infected or contaminated with *S*. Pullorum, or *S*. Gallinarum.

Exposure assessment

Exposure Group 1: Wild birds

Of the steps identified as determinants in the infection of wild birds by exposure to scraps from an imported contaminated chicken carcass, $WB_{agentsurvival}$ and $WB_{infectivedose}$ are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$WB_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by a wild bird

S. Pullorum and *S*. Gallinarum, protected within chicken meat scraps, are likely to survive in the environment under ambient temperatures of 10 ºC to 35 ºC for several days, giving ample time for wild birds to locate and scavenge the material. Under appropriate conditions, these organisms may multiply to some extent in chicken meat. In contrast to other Salmonellae, such as S. Typhimurium and S. Enteritidis, however, there is no evidence that S. Pullorum and S. Gallinarum multiply to any significant extent in the environment. The IRA team considered the likelihood that this organism will remain viable until wild birds locate and scavenge the material as **moderate**.

$WB_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a wild bird is sufficient to produce infection

The role of wild birds in the dissemination of these organisms is unclear. However, wild birds have been implicated by several authors in transmission of pullorum disease and fowl typhoid, particularly in poultry kept outdoors. While clinical disease may not occur in wild birds, they may act as mechanical vectors, shedding the bacteria through their faeces. Bacterial contamination of carcasses is most likely to result from faecal contamination during processing;

therefore, the dose of bacteria present on the carcass is likely to be relatively low. However, organisms may be sequestered in the heart, liver and gizzard, and if these organs were available to wild birds, the risk of infection associated with their consumption would be higher than for other carcass scraps. This risk assessment considers the importation of whole carcasses, including heart, gizzard and liver; therefore $WB_{infectivedose}$ was estimated on the assumption that these organs may be discarded in refuse.

The IRA team considered that there was a ***low*** likelihood that *S.* Pullorum or *S.* Gallinarum would infect a wild bird consuming the contaminated meat scraps.

Exposure Group 2: Low biosecurity poultry

Of the steps identified as determinants in the infection of low biosecurity poultry by exposure to material from an imported contaminated chicken carcass, $BP_{agentsurvival}$, $BP_{infectivedose}$, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$BP_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by low biosecurity poultry

The time between feeding of scraps and consumption by low biosecurity poultry is likely to be very short, so environmental degradation of the disease agent will be minimal. The likelihood that the agent will remain viable was assessed by the IRA team as ***certain (=1)***.

$BP_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a low biosecurity bird is sufficient to produce infection

Transmission of these organisms occurs via the faecal-oral route, and birds of all ages can be affected. The ID_{50} for *S.* Pullorum and *S.* Gallinarum have been documented, but the load of bacteria present in chicken meat has not. Therefore, it is difficult to determine if a sufficient dose of bacteria would be present on a contaminated/infected carcass. However, bacteria may be sequestered in the heart, liver and gizzard, and if these organs were available to low biosecurity poultry, the risk of infection associated with their consumption would be higher than for other carcass scraps. This risk assessment considers the importation of whole carcasses, including heart, gizzard and liver; therefore $BP_{infectivedose}$ was estimated on the assumption that these organs may be fed to low biosecurity poultry.

Given that a chicken can consume up to 150g of feed per day, and that chickens are the primary host for these organisms, and are therefore likely to be more susceptible to infection than are individuals of other species, the IRA team considered that it was ***moderately*** likely that the oral infectious dose for pullorum disease/fowl typhoid would be exceeded.

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

The likelihood that the agent would survive the rendering process was negligible. For *S.* Pullorum or *S.* Gallinarum the IRA team assessed the likelihood that the product will be re-contaminated post-processing as negligible. Therefore, the likelihood that poultry feed derived

from the imported contaminated carcass would be contaminated with *S*. Pullorum or *S*. Gallinarum was estimated by the IRA team to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

Given that salmonellae would be destroyed by rendering, and that feed derived from contaminated carcasses would be diluted with non-risk material, the likelihood that the amount of final poultry ration would contain an oral infectious dose of bacteria was considered by the IRA team to be ***negligible***.

Exposure Group 3: Medium biosecurity commercial poultry

Of the steps identified as determinants in the infection of medium biosecurity commercial poultry by exposure to material from an imported contaminated chicken carcass, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with *S*. Pullorum or *S*. Gallinarum was estimated by the IRA team to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that the amount of final poultry ration consumed would contain an oral infectious dose of *S*. Pullorum or *S*. Gallinarum was considered by the IRA team to be ***negligible***.

Exposure Group 4: Non-avian species

As discussed above, $NAS_{agentsurvival}$ was considered to be equal to $WB_{agentsurvival}$.

Pullorum disease has been described as a naturally occurring or experimental infection in some mammals, including rats. However, it is generally considered that these organisms are host-specific, and infection of non-avian species is usually considered to be transitory.

The role of non-avian species in the dissemination of these organisms is unclear, but they may act as mechanical vectors, shedding the bacteria through their faeces.

The likelihood that an amount of scrap from an infected chicken carcass would contain a sufficient quantity of the agent to infect non-avian species ($NAS_{infectivedose}$) was assessed by the IRA team as ***low*** under Australian conditions.

Conclusions – Exposure assessment

The partial likelihood of exposure for each of the exposure groups was calculated after inserting the estimates of the distribution variables, exposure group-dependent variables and pathogen-

dependent variables into the simulation model. A summary of the outputs from the simulation model is shown in Table 49.

Table 49. Partial likelihoods of exposure (PLE)

Exposure group	PLE (Pullorum)	PLE (Gallinarum)
Wild birds	Extremely low	Extremely low
Low biosecurity poultry	Very low	Very low
Medium biosecurity poultry	Negligible	Negligible
Non-avian species	Extremely low	Extremely low

Consequence assessment

Establishment and spread was considered in the context of the outbreak scenarios, described in the Method for Risk Assessment. Because the dynamics of *S*. Gallinarum and *S*. Pullorum infection may differ within flocks of birds, the partial likelihood of establishment and spread (PLES) of pullorum disease and fowl typhoid were considered separately, as described below.

Estimating the likelihood of each outbreak scenario

The partial likelihood of establishment and spread (PLES) of *S*. Pullorum or *S*. Gallinarum for the different exposure groups is described below. Four outbreak scenarios were considered relevant:

1. Disease agent does not establish or is not recognised within the directly exposed population
2. Disease agent establishes within the directly exposed population, is identified and is eliminated by human efforts or by natural means
3. Disease agent establishes in the directly exposed population, spreads within a district/region, including into other exposure groups if applicable, and is eliminated by human action or by natural means
4. Disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia.

Wild birds

Chickens are the natural hosts for *S*. Pullorum and *S*. Gallinarum, and outbreaks in other avian species are uncommon. Outbreaks of pullorum disease and fowl typhoid have not been reported in wild birds known to scavenge at rubbish dumps, although they may act as mechanical carriers of these bacteria. The IRA team considered that the most likely outcome of infection of a wild bird resulting from scavenging chicken meat scraps would be a single or a few isolated occurrences of infection in wild birds, with the organism being unable to establish ongoing infection in the population. Infection of wild birds with *S*. Pullorum or *S*. Gallinarum, with subsequent spread to poultry, was considered highly unlikely. In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 50).

Table 50. Estimated partial likelihood of establishment and spread (PLES) values for *S*. Pullorum and *S*. Gallinarum in wild birds

		***S*. Pullorum**	***S*. Gallinarum**
Outbreak scenario 1:	$PLES_{(wb)}1$	**High**	**High**
Outbreak scenario 2:	$PLES_{(wb)}2$	**Extremely low**	**Extremely low**
Outbreak scenario 3:	$PLES_{(wb)}3$	**Extremely low**	**Extremely low**
Outbreak scenario 4:	$PLES_{(wb)}4$	**Extremely low**	**Extremely low**

Low biosecurity poultry

Chickens are the natural hosts for both *S.* Pullorum and *S*. Gallinarum, but naturally occurring outbreaks have also been reported in turkeys, pheasants, guinea fowl and quail. Clinical signs of pullorum disease are usually observed in young chicks, while clinical signs of fowl typhoid are usually observed in older poultry. Although this exposure group includes commercial free-range poultry and ratites, the IRA team considered that the sub-set of this group most likely to be exposed to scraps of imported chicken meat was small flocks of backyard poultry, because these are more likely to be directly fed table scraps. In such flocks, there is less opportunity for the generation of high levels of environmental contamination, than might occur with an outbreak of infectious disease in a large commercial flock. The IRA team considered that the most likely outcome of infection with either *S.* Pullorum or *S*. Gallinarum would be a single or a few isolated occurrences of infection.

If the disease were to establish in the flock, the level of expertise in disease recognition is likely to be low. Mechanical transmission of the organisms by contaminated persons or fomites, and transmission by movement of birds may facilitate spread of *S.* Pullorum or *S*. Gallinarum beyond the initially infected flock before it is recognised, and eradication measures are implemented. However, such spread is likely to be limited to other low biosecurity poultry, rather than to medium biosecurity commercial poultry, since breeder flocks, which are generally kept under high biosecurity conditions, would need to become infected for *S.* Pullorum infection to establish in the broader population. *S*. Gallinarum is more likely than *S.* Pullorum to be transmitted horizontally outside of the hatchery, and therefore, there is a greater likelihood of an outbreak of fowl typhoid spreading to involve commercial poultry flocks.

In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely, with outbreak scenario 2 the next most likely outcome. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 51).

Medium biosecurity commercial poultry

The IRA team considered that the most feasible route for exposure of medium biosecurity commercial poultry to imported carcass components would be through poultry feed containing inadequately rendered processing waste. The likelihood that pathogens would remain viable following rendering of contaminated imported carcasses and parts was assessed as negligible (page 162, Part C). Nevertheless, assessment of PLES was based on the assumption that medium biosecurity commercial poultry had been exposed to poultry feed contaminated with the disease agent.

Table 51. Estimated partial likelihood of establishment and spread (PLES) values for *S*. Pullorum and *S*. Gallinarum in low biosecurity poultry

		***S*. Pullorum**	***S*. Gallinarum**
Outbreak scenario 1:	$PLES_{(bp)}1$	**High**	**High**
Outbreak scenario 2:	$PLES_{(bp)}2$	**Low**	**Low**
Outbreak scenario 3:	$PLES_{(bp)}3$	**Extremely low**	**Very low**
Outbreak scenario 4:	$PLES_{(bp)}4$	**Negligible**	**Extremely low**

S. Pullorum and *S*. Gallinarum are host-adapted organisms, and highly pathogenic for chickens and turkeys. While Pullorum disease is primarily a disease of newly hatched chicks, with few clinical signs occurring in older birds, fowl typhoid may cause clinical signs in older birds. The major route of transmission of *S*. Pullorum and *S*. Gallinarum is vertically via transovarial transmission and by definition, breeding birds are not considered part of this exposure group. Therefore, infection with *S.* Pullorum would be unlikely to be diagnosed in this exposure group, although *S.* Gallinarum could be expected to be more readily detected. The IRA team considered that the most likely outcome of infection with *S.* Pullorum would be a single or a few isolated occurrences of infection. Clinical signs of fowl typhoid caused by *S*. Gallinarum may be non-specific or inapparent, complicating disease investigation, and it is likely that the outbreak would spread before it is brought under control. However, fowl typhoid is unlikely to become endemic in commercial poultry because it would be detected and eradicated. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 52).

Table 52. Estimated partial likelihood of establishment and spread (PLES) values for *S*. Pullorum and *S*. Gallinarum in medium biosecurity commercial poultry

		***S*. Pullorum**	***S*. Gallinarum**
Outbreak scenario 1:	$PLES_{(cp)}1$	**High**	**Very Low**
Outbreak scenario 2:	$PLES_{(cp)}2$	**Very low**	**Low**
Outbreak scenario 3:	$PLES_{(cp)}3$	**Negligible**	**Moderate**
Outbreak scenario 4:	$PLES_{(cp)}4$	**Negligible**	**Extremely low**

Non-Avian Species

Pullorum disease has been described as a naturally occurring or experimental infection in some mammals, including rats. However, it is generally accepted that *S.* Pullorum and *S*. Gallinarum are avian-specific (Shivaprasad 2003), and infection of non-avian species is therefore likely to be transitory. Therefore, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 53).

Table 53. Estimated partial likelihood of establishment and spread (PLES) values for *S*. Pullorum and *S*. Gallinarum in non-avian species

		***S*. Pullorum**	***S*. Gallinarum**
Outbreak scenario 1:	$PLES_{(nas)}1$	**High**	**High**
Outbreak scenario 2:	$PLES_{(nas)}2$	**Negligible**	**Negligible**
Outbreak scenario 3:	$PLES_{(nas)}3$	**Negligible**	**Negligible**
Outbreak scenario 4:	$PLES_{(nas)}4$	**Negligible**	**Negligible**

Conclusion – Likelihood assessments

The estimates for the likelihood of release, the partial likelihoods of exposure for each of the exposure groups, and the partial likelihood of establishment and spread for each of the outbreaks scenarios were combined with the expected volume of trade using the simulation model. This allowed the calculation of partial annual likelihood of entry, exposure, establishment and spread for each of the outbreak scenarios. The results of this calculation are shown in Table 54.

Table 54. Partial annual likelihood of entry, exposure, establishment and spread (PALEEES) for the outbreak scenarios

Exposure group	Outbreak scenario	PALEEES (Pullorum)	PALEEES (Gallinarum)
Wild birds	Scenario 1	High	High
	Scenario 2	High	High
Low biosecurity poultry	Scenario 1	High	High
	Scenario 2	High	High
Medium biosecurity poultry	Scenario 1	Extremely low	Negligible
	Scenario 2	Negligible	Negligible
Non-avian species	Scenario 1	High	High
	Scenario 2	Very low	Very low
Total all exposure groups	Scenario 3	High	High
Total all exposure groups	Scenario 4	High	High

Estimating the impacts associated with each outbreak scenario

For each outbreak scenario the direct and indirect impacts of *S.* Pullorum and *S.* Gallinarum infection were estimated at the national, state or territory, district/region and local level, as described in the Methods section (page 90-95, Part B). The IRA team considered that the impacts of fowl typhoid and pullorum disease would be similar in each of the exposure groups.

Outbreak Scenario 1

By definition, outbreak scenario 1 means that the disease agent does not establish in the initially exposed population, or does not cause sufficient disease to lead to investigation and recognition of the infection. Therefore the impacts will be ***unlikely to be discernible*** for all exposure groups and all criteria.

Outbreak Scenario 2

The impacts of this outbreak scenario (disease agent establishes within the directly exposed population, and is identified and eliminated) will differ between exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

S. Pullorum and *S.* Gallinarum are host-adapted and highly pathogenic for chickens and turkeys. Naturally occurring outbreaks have been reported in turkeys, pheasants, guinea fowl, quail, sparrows, and parrots. Pullorum disease has been described in canaries and bullfinches, and fowl typhoid in ringdoves, ostriches and peafowl (Shivaprasad 2003). Since wild birds do not play a significant part in production, direct economic loss from death of wild birds, were it to occur, is not measurable. Other impacts from the death of wild birds will be considered under direct criterion 2 and indirect criterion 4. The direct impacts of disease on wild birds were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

An outbreak of pullorum disease or fowl typhoid contained within the low biosecurity poultry population may result in some losses to individual owners, but the impacts of such losses on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

An outbreak of pullorum disease or fowl typhoid in medium biosecurity commercial poultry flocks could result in loss of birds, poor weight gain, and decreased egg production, fertility and hatchability in laying birds. The impacts on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level.

The direct impacts of infection on non-avian species were assessed as ***unlikely to be discernible*** at any level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

An outbreak of disease in wild birds is likely to be transient, with few detectable impacts on the wild bird population. The impacts were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory, district/region or local levels.

The impacts of an outbreak of pullorum disease or fowl typhoid in low biosecurity poultry on this criterion were assessed as ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak of pullorum disease or fowl typhoid in medium biosecurity commercial poultry on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

An outbreak of infection in non-avian species is likely to be transient, with few detectable impacts of disease on the exposed population. The impacts on this criterion were assessed as ***unlikely to be discernible*** at national, State/Territory, district/region or local levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

The impact of a diagnosed outbreak in wild birds, with no subsequent spread, was assessed by the IRA team as ***unlikely to be discernible*** at any level.

Diagnosis of pullorum disease or fowl typhoid in poultry would result in increased surveillance and monitoring of poultry populations. The impacts of an outbreak in low biosecurity poultry on this criterion were assessed as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level. Impacts in the medium biosecurity commercial poultry population were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels**,** and ***minor*** at the local level.

The impact of a recognised outbreak in non-avian species, with no subsequent spread, was assessed by the IRA team as ***unlikely to be discernible*** at any level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

The impacts of a disease outbreak in the local wild bird population were assessed by the IRA team as ***unlikely to be discernible***.

If pullorum disease or fowl typhoid were diagnosed in poultry, it is likely that movement and marketing controls would be imposed. The IRA team considered that impacts of movement restrictions in low biosecurity poultry were ***unlikely to be discernible*** at national, State/Territory, district/region and local levels. Impacts on medium biosecurity commercial poultry were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, and ***minor*** at the local level.

The IRA team considered that impacts of a disease outbreak in the local population of non-avian species were ***unlikely to be discernible***.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

The impacts of outbreaks in wild birds on international trade were assessed by the IRA team as ***unlikely to be discernible*** at any level. Similarly, the impacts of an outbreak of pullorum disease or fowl typhoid in low biosecurity poultry, medium biosecurity commercial poultry and non-avian species on international trade were assessed as ***unlikely to be discernible*** at any level.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The IRA team considered that impacts of an outbreak of pullorum disease or fowl typhoid were ***unlikely to be discernible*** for all exposure groups at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impacts of outbreaks in wild birds on communities were assessed by the IRA team as ***unlikely to be discernible*** at any level. Similarly, the impacts of an outbreak in low biosecurity poultry on communities were assessed as ***unlikely to be discernible*** at any level.

There may be temporary restrictions imposed on movement of birds, eggs, poultry products and people if an outbreak of pullorum disease or fowl typhoid were diagnosed in a flock of medium biosecurity commercial poultry. The IRA team considered that these impacts were ***unlikely to be discernible*** at national, State/Territory and district/region levels, and ***minor*** at the local level.

The impacts of outbreaks in non-avian species on international trade were assessed as ***unlikely to be discernible*** at any level.

Outbreak Scenario 3

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a district/region, and is eliminated) will be the same for all exposure groups, no matter which exposure group has been directly exposed to the agent in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

Increased mortalities in chicks and poults, decreased feed consumption and loss of weight in growing birds, and decreased egg production, fertility and hatchability in infected laying flocks will result in production losses. The IRA team considered that infected meat chicken flocks and spent hens would be sent to slaughter at the normal time. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, but ***minor*** at the district/region level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

A contained outbreak in commercial or low biosecurity poultry was assessed by the IRA team as ***unlikely*** to have ***discernible*** impacts on the environment at any level.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

Spread of pullorum disease or fowl typhoid to a local population of poultry or caged birds would lead to an increase in surveillance and monitoring programs. The IRA team considered that impacts on the national and State/Territory economies were ***unlikely to be discernible***. Impacts were assessed as ***minor*** at the district/region level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Movement and marketing controls placed on affected farms may lead to impacts on domestic trade and local industry. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, and ***minor*** at the district/region level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

The IRA team considered that the impacts of a local or general outbreak of pullorum disease on international trade would be ***unlikely to be discernible*** at national, State/Territory and district/region levels, and ***minor*** at the local level.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

Impacts for all exposure groups were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Temporary restrictions imposed on movement of birds, eggs, poultry products and people may lead to community disruption. The IRA team considered that impacts were ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level.

Outbreak Scenario 4

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a State/Territory, and becomes endemic in Australia) will be the same for all exposure groups, no matter which exposure group has been directly exposed to the agent in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

If the disease spreads more widely through medium biosecurity commercial poultry and low biosecurity poultry, and particularly if hatcheries become infected, there may be significant losses of birds and production. At the national and State/Territory levels, the impacts were assessed by the IRA team as ***unlikely to be discernible***. Impacts were assessed as ***minor*** at the district/region level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

The IRA team considered that a more general outbreak of pullorum disease or fowl typhoid is ***unlikely*** to have ***discernible*** impacts on the environment at any level.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

If pullorum disease or fowl typhoid spread to a more general population of poultry, there will be more intensive monitoring and surveillance programs. Eradication and control schemes would be undertaken in affected breeder flocks. The IRA team considered that impacts at the national level were ***unlikely to be discernible***. Impacts were assessed as ***minor*** at the State/Territory level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

The impacts of a more general outbreak of pullorum disease or fowl typhoid on domestic trade and industry were assessed by the IRA team as ***unlikely to be discernible*** at the national and State/Territory levels but ***minor*** at the district level, due to disruption in the supply of hatching eggs, and the impact of adverse publicity on the market.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

Impacts of a general outbreak of pullorum disease or fowl typhoid on international trade were assessed as ***unlikely to be discernible*** at national, State/Territory and district/region levels, and ***minor*** at the local level.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impact of a general outbreak of pullorum disease/fowl typhoid on the environment was assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impact of a general outbreak of pullorum disease/fowl typhoid on communities was assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level.

Conclusions – impact assessment

The above estimates for each of the impact criteria were entered into the simulation model, and the overall impact of each outbreak scenario was estimated. Results are shown in Table 55.

Table 55. Impacts of each outbreak scenario

Exposure group	Outbreak scenario	Impact (Pullorum)	Impact (Gallinarum)
Wild birds	Scenario 1	Negligible	Negligible
	Scenario 2	Negligible	Negligible
Low biosecurity poultry	Scenario 1	Negligible	Negligible
	Scenario 2	Negligible	Negligible
Medium biosecurity poultry	Scenario 1	Negligible	Negligible
	Scenario 2	Negligible	Negligible
Non-avian species	Scenario 1	Negligible	Negligible
	Scenario 2	Negligible	Negligible
	Total Scenario 3	Very low	Very low
	Total Scenario 4	Low	Low

Partial annual risk estimate

The partial annual likelihood of entry, exposure, establishment and spread were combined with the impacts of the relevant outbreak scenarios to provide an estimate of the partial annual risk associated with each outbreak scenario. The outcomes of this process are shown in Table 56 for *Salmonella* Pullorum, and in Table 57 for *Salmonella* Gallinarum.

Unrestricted risk estimate

The overall risk associated with the import of whole chicken carcasses was assessed as ***low*** for both *S*. Pullorum and *S*. Gallinarum. This unrestricted risk estimate exceeds Australia's ALOP, and therefore, risk management was deemed necessary.

Direct impact on human life or health

Pullorum disease and fowl typhoid are not considered to be significant threats to public health.

Table 56. Partial annual risk (PAR) of each outbreak scenario (Pullorum)

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Medium biosecurity poultry	Scenario 1	E. low	Negligible	Negligible
	Scenario 2	Negligible	Negligible	Negligible
Non-avian species	Scenario 1	High	Negligible	Negligible
	Scenario 2	Very low	Negligible	Negligible
	Total Scenario 3	High	Very low	Very Low
	Total Scenario 4	High	Low	Low

Table 57. Partial annual risk (PAR) of each outbreak scenario (Gallinarum)

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Medium biosecurity poultry	Scenario 1	Negligible	Negligible	Negligible
	Scenario 2	Negligible	Negligible	Negligible
Non-avian species	Scenario 1	High	Negligible	Negligible
	Scenario 2	Very low	Negligible	Negligible
	Total Scenario 3	High	Very low	Very Low
	Total Scenario 4	High	Low	Low

Salmonella Enteritidis & Multi-drug Resistant *Salmonella* Typhimurium

Technical Information

Background

S. Enteritidis and *S.* Typhimurium are typically non-host-specific bacterial pathogens, principally of concern as a major cause of food-borne salmonellosis in humans. In poultry, strains of these two *Salmonella* serovars cause systemic infection, leading to contamination of meat and eggs. *S.* Enteritidis and *S.* Typhimurium seldom cause clinical disease, except in susceptible young birds (Gast 2003a). The serovars are distributed virtually worldwide in a range of species (Barrow 2000) but significant subtypes and particular strains of concern are not endemic in Australia.

S. Enteritidis phage type 4 (PT 4), phage type 8 (PT 8) and phage type 13a (PT 13a) are generally recognised as the most important of the 50 or so phage types of *S.* Enteritidis. In some countries, invasive strains of PT4 have replaced all other phage types in the poultry population (Wilks, Parkinson, and Young 2000). In 1994, *S.* Enteritidis PT 4 was isolated from samples taken from a commercial layer flock in Australia. However, the isolates were thought to be the result of laboratory contamination since isolation could not be repeated on re-sampling of the same shed (Davos 2002). An *S.* Enteritidis outbreak involving a commercial meat-chicken company was investigated in Queensland in 2005. Control measures, including culling, stringent disinfection procedures and on-going monitoring, were put in place by Queensland State Government authorities (K Bell, Safe Food Queensland, Australia, pers. comm. May 2006). No further isolations of S. Enteritidis have been made since July 2006 (Dubs 2007).

S. Typhimurium multi-drug resistant (MDR) strains are characteristically resistant to at least five antibiotics, including ampicillin, chloramphenicol, streptomycin, sulfonamides and tetracycline. MDR *S.* Typhimurium isolates are best characterised by *S.* Typhimurium definitive type 104 (DT104), which carries all five antibiotic resistances on a chromosomally-coded 43kb genomic island (SGI1), which carries the ACSSuT genes, coding for the multiresistance phenotype (Boyd et al. 2001). The presence of this stable genomic island ensures the continued presence of multiple antibiotic resistance even in the absence of selection pressure (Boyd et al. 2001; World Health Organization 2005). These resistance genes can also be transferred to other *Salmonella* strains via bacteriophage transduction or bacterial conjugation. *S.* Typhimurium DT104 and related strains can be readily identified by evaluation of antibiotic resistance and by multiplex PCR amplification of parts of the 43 kb SGI1 genomic island (Chiu et al. 2006). MDR strains of *S.* Typhimurium have not been isolated from livestock in Australia (Joint Expert Technical Advisory Committee on Antibiotic Resistance 1999).

S. Enteritidis PT 4 and *S.* Typhimurium DT 104 have been isolated from cases of human Salmonellosis in Australia, but are usually associated with overseas travel (Davos 2002; O'Grady et al. 2001). However, the source of a recent outbreak of MDR *S.* Typhimurium DT 104 was imported contaminated food (O'Grady et al. 2001). Introduction of these pathogens would have a significant impact on the Australian poultry industry through their effect on public health, animal health and trade (Crerar, Nicholls, and Barton 1999).

Salmonellosis, due to *S*. Enteritidis or *S*. Typhimurium infection, is an OIE-listed disease.

This risk assessment is concerned with all strains of *Salmonella* Enteritidis and multi-drug resistant strains of *Salmonella* Typhimurium.

Agent taxonomy

S. Enteritidis and *S*. Typhimurium are Gram-negative, non-sporogenic, motile, facultative anaerobic bacteria of the family *Enterobacteriaceae*, *Salmonella* species *enterica*, subspecies *enterica*, serovars Enteritidis and Typhimurium. Serovars are further subdivided into phage types based on their susceptibility to a set of bacteriophages (Barrow 2000).

Epidemiology

In general, *S*. Enteritidis and *S*. Typhimurium do not cause severe systemic disease in physiologically and immunologically normal, healthy adult chickens (Barrow 2000). Clinical disease usually occurs only in very young birds, and birds infected within 24 hours of hatching may develop persistent infection and shed organisms for up to 28 weeks.

In wild birds, outbreaks of salmonellosis have been associated with overcrowding at food sources and scavenging on refuse tips or human sewage, or have occurred in birds with concurrent disease (Friend 1999; Alley et al. 2002). *S*. Typhimurium outbreaks are occasionally reported in garden birds, such as sparrows and finches, that visit feeding stations in winter (Pennycott, Park, and Mather 2006). Strains of *S*. Typhimurium associated with these outbreaks are typically ‘wild bird’ strains, with different strains of *S*. Typhimurium being associated with different categories of wild birds (Pennycott, Park, and Mather 2006). These wild bird strains do not pose a major threat to chickens, turkeys and other livestock (Pennycott, Park, and Mather 2006). Wild birds do occasionally acquire *Salmonella* species of importance to livestock, such as *S*. Enteritidis and MDR strains of *S*. Typhimurium (Davies 2001; Pennycott, Park, and Mather 2006). A *S*. Typhimurium DT 160 outbreak resulted in mortalities in sparrows, other passerine birds, and a small number of psittacine birds in New Zealand, and occurred in parallel with a concurrent increase in cases of human salmonellosis in 2000 (Alley et al. 2002; Thornley et al. 2003). The source of the outbreak was not documented. In an experimental study, infection of house sparrows with *S*. Typhimurium DT160 was found to be transient unless high infective doses were administered (Connolly et al. 2006). Wild birds infected with *S*. Enteritidis and *S*. Typhimurium may show no clinical signs of disease and appear healthy (Friend 1999; Tizard, Fish, and Harmeson 1979).

Transmission is generally by mouth, but infection may also occur by the respiratory or conjunctival route, via the egg (shell contamination and transovarian) and via the cloaca. The organisms multiply in the intestine, invade the wall of the gut and are shed in relatively large numbers in the faeces. The duration of faecal excretion is affected by the external temperature, the use of antibiotics and growth promoters, and concomitant infections with other agents such as IBDV and *Eimeria* (Barrow 2000).

Both horizontal and transovarian transmission occur and may produce highly persistent infection in flocks. In the hatchery, chicks may be infected through the ingestion of contaminated fluff, shell and dust, usually leading to cross contamination of other birds. In older birds, infection occurs through direct contact with clinically ill or asymptomatic carriers, consumption of contaminated feed or water, or from the environment (feed, rodents, birds,

insects, water and litter) (White, Baker, and James 1997) or clothing and footwear of animal attendants. Replacement birds may also introduce new infections to a flock (Barrow 2000).

S. Enteritidis and *S*. Typhimurium are able to colonise many types of livestock, but show a preference for particular hosts. *S*. Enteritidis predominates in poultry, especially chickens, while *S*. Typhimurium is ubiquitous and has a number of species as maintenance hosts (Thorns 2000).

Rodents, birds and cats may act as reservoirs of infection of *S*. Enteritidis and *S*. Typhimurium. Rats infected with *S*. Enteritidis have been shown to excrete the organism in their faeces for 34 days, where it may survive for at least 148 days (Henzler and Opitz 1999). Infected mice may shed an average of 2.3×10^5 organisms in one faecal pellet, sufficient to infect adult hens. Mice and chicks may be infected with fewer than 15 *S*. Enteritidis bacteria (Henzler and Opitz 1999). Domestic pets may also be infected with *S*. Typhimurium DT 104, and cats have been shown to carry the organisms for up to 14 weeks (Wall et al. 1995).

There is little data on the prevalence of *S*. Enteritidis and MDR *S*. Typhimurium in flocks. Studies of the between flock prevalence and within flock prevalence of *Salmonella* spp. in meat chicken flocks report rates of 0.7% to 76.9% and 2% to 92% respectively (Kelly, Anderson, and Snary 2000). Small sample sizes, differences in sampling methods and variation due to differences in production methods, climatic conditions and other factors make interpretation and comparison of these reports difficult. Data from the United States suggests a 10.5% flock prevalence rate of *S*. Enteritidis infection in commercial layers (Kinde et al. 2004). Prevalence data in humans suggest that the prevalence of MDR strains of *S*. Typhimurium is increasing in many countries, including the United States, Canada, Europe, and Asia (World Health Organization 2005; Glynn et al. 1998; Chiu et al. 2006).

Clinical signs

The clinical signs are not pathognomonic and are similar to those of pullorum disease. Enteritis is the most common clinical sign in infected chicks and poults, but a wide range of clinical signs may be observed. Affected birds may show anorexia, adypsia, depression, drowsiness, and enteritis (vent pasting). The mortality rates in chicks and poults vary from 10% to 80%, but differ with strain and serotype. Death occurs four to ten days after infection. Stunting is observed in convalescent birds (Barrow 2000). There is considerable variation in the severity of the disease produced. Older birds may show little or no clinical disease.

Pathogenesis

Following infection of chicks by the oral route there is massive bacterial multiplication in the gut, invasion of the gut wall, dissemination to organs such as the liver, spleen, lung and ovary, and multiplication of *S*. Enteritidis and *S*. Typhimurium in the cells of the reticuloendothelial system. Large numbers of organisms are shed in the faeces for several weeks (Barrow 2000). Between 53% and 75% of birds, experimentally infected with *S*. Typhimurium at one, three or six weeks of age, were positive by culture of cloacal swabs until eight to nine weeks of age (Beal et al. 2004).

In older birds, infection may cause little or no clinical disease due to inhibition of the organism’s multiplication by the normal intestinal flora, but invasion of the intestinal wall occurs and bacteria reach the liver and spleen. The main sites of localisation are the caeca and crop (Barrow 2000).

In hens in lay, the organisms may localise in the reproductive tract, especially the ovary and oviduct. This occurs particularly with *S*. Enteritidis and may result in transmission of the organism through the egg. Egg contamination also occurs by surface contamination of the shell with faeces and penetration of the shell of freshly laid eggs (Barrow 2000). Experimental studies have shown that *S*. Typhimurium is capable of colonising reproductive tissues and developing eggs of mature laying hens at a similar rate to that of *S*. Enteritidis (Keller et al. 1997). Vertical transmission of both *S*. Enteritidis and *S*. Typhimurium from breeders to meat chickens was demonstrated in an integrated broiler production system in the United States (Liljebjelke et al. 2005).

Pathology

Lesions are not pathognomonic and include persistent yolk sacs, congestion and necrosis of the liver and spleen, coagulated yolks, and cores of necrotic material in the caeca. Birds infected with S. Enteritidis may show pericarditis and polyserositis. Arthritis may follow (Barrow 2000).

Immunology

S. Enteritidis and *S*. Typhimurium can elicit strong antibody responses from infected poultry (Gast 2003a). Antibody levels rise rapidly after infection and may persist for many weeks. A number of serological tests based on agglutination or complement fixation have been developed for the diagnosis of salmonella infections in animals, but have relatively poor specificity. ELISA tests are also available and are in routine use for detecting antibodies to *Salmonellae*. These are especially useful for large scale screening of poultry flocks (Barrow 2000). However, results may be difficult to interpret where birds have been vaccinated for pullorum disease. All tests are subject to false positive reactions, and all positive serological tests should be confirmed by culture of samples from the suspect bird (Waltman, Gast, and Mallinson 1998).

Both inactivated and live vaccines against *S*. Enteritidis and *S*. Typhimurium have been developed for use in poultry (Barrow 2000). In general, inactivated whole cell vaccines have produced poor and inconsistent protection against challenge with salmonellae, in both vaccinated flocks and their progeny (Wilks, Parkinson, and Young 2000). Live vaccines developed using molecular biological techniques are now available and have been shown to protect poultry against infection (Barrow 2000; Wilks, Parkinson, and Young 2000). Live attenuated *S*. Enteritidis and *S*. Typhimurium vaccines may be used to cross-protect chickens against colonisation with other *Salmonella* species (Barrow 2000; Curtiss and Hassan 1996; Hassan and Curtiss 1997; Cerquetti and Gherardi 2000; Dueger et al. 2003).

Diagnosis

Clinical infections in flocks are diagnosed principally by the isolation of the organisms from samples of internal organs such as liver, spleen, heart, synoviae, eye and brain, or from heart blood, ovary or yolk sac. Drag swabs are a widely accepted method of sampling for Salmonellae on poultry farms (Kinde et al. 2004; Mallinson et al. 1989; Kingston 1981). Samples of litter, dust, feed, or hatch residues from the hatchery or poultry house, or pooled egg samples may also be used to survey and monitor flocks (Gast 2003a). *Salmonellae* are excreted intermittently, often in low numbers so that cloacal swabs are an unreliable means of detection of the organisms.

Transmission in chicken meat

The authors of a recent study concluded that infection of meat chickens with *S*. Typhimurium at any age will result in the presence of *Salmonella* in the intestines at slaughter, thereby increasing the risk of carcass contamination (Beal et al. 2004). Surveys have shown that a significant proportion of chicken carcasses and cuts are contaminated with *Salmonella* spp., however, there is limited data on the numbers of organisms within or on contaminated carcasses (Kelly, Anderson, and Snary 2000). The principal source of *Salmonella* spp. on the dressed chicken carcass is faeces, and extensive faecal cross-contamination occurs during shed depopulation, transport and slaughter of the birds. This results in contamination of equipment and the carcass during processing.

If a processing plant is inadequately cleaned and disinfected on a regular basis, there may be significant contamination of carcasses during processing. A study of four slaughter-houses in Belgium showed that *Salmonella* spp. persist on plucking and processing equipment, even after cleaning. Flocks that were not infected before slaughter became contaminated with *Salmonellae* when processed before infected flocks, and *Salmonella*-positive flocks were contaminated with additional species during processing (De Zutter, Herman, and Heyndrickx 2005). Infection of less than 5% of a flock may lead to contamination of 50% to 100% of carcasses at the retail outlet (Barrow 2000).

S. Enteritidis and *S*. Typhimurium may also be sequestered in organs such as liver, heart and gizzard of infected birds.

Since *S*. Enteritidis and *S*. Typhimurium are the predominant serotypes associated with poultry in many countries, it is reasonable to assume that meat derived from infected flocks, or from uninfected flocks processed following infected flocks and before thorough cleaning and disinfection of the plant, will be contaminated with these serotypes. Although carcasses are frequently contaminated, the numbers of organisms per carcass are generally low, and not all *S*. Typhimurium contaminants will be MDR types. Mean levels of 4 to 1400 *Salmonellae* per 100g chicken skin have been reported (Mulder, Notermans, and Kampelmacher 1977; Uyttendaele, de Troy, and Debevere 1999).

Quarantine significance

S. Enteritidis and *S*. Typhimurium *are OIE-listed disease agents* (World Organisation for Animal Health (OIE) 2006).

S. Enteritidis is notifiable in all Australian States and Territories, and any isolations are subject to official investigation and control. In New South Wales, the SE (*S*. Enteritidis) Monitoring and Accreditation Scheme monitors *S*. Enteritidis infection in layer and breeder flocks. *S*. Enteritidis is not currently included in the Australian Emergency Animal Disease Response Agreement. However it has been proposed that it be included in Category 1 or 2 because of implications for public health and high cost to community (Sergeant et al. 2003).

Outbreaks may have significant effects on public health, animal health and trade. In countries where *S*. Enteritidis is endemic, it is responsible for around 75% of all human cases of salmonellosis. If *S*. Enteritidis were to become established in the Australian egg industry, costs of treatment of human infections, loss of productivity, and industry losses due to reduced egg consumption and control or eradication costs could be considerable (Sergeant et al. 2003).

MDR strains of *S*. Typhimurium are of great public health significance, and introduction of these strains into Australian poultry would have both human and animal health consequences, and require increased surveillance and control programs in the poultry industry.

Risk Assessment

Release Assessment

Rel_1: Selection of source flock (between flock prevalence)

In some countries, *S*. Enteritidis or *S*. Typhimurium in the intensive poultry industries are included in official control programs. In those countries in which the disease is endemic, the prevalence of infection is difficult to estimate, since the reported prevalence rates for *Salmonella* infections in poultry flocks vary widely. For an unrestricted risk estimate, it was assumed that the prevalence is at the highest sustainable level in an endemically infected country or zone (see Method of Risk Assessment). The IRA team assumed that the prevalence rate for *S*. Enteritidis and MDR *S*. Typhimurium would be lower than the overall prevalence rate for *S.* Pullorum or *S.* Gallinarum, which are specifically chicken adapted strains. The likelihood that a source flock will be infected with *S*. Enteritidis or MDR *S*. Typhimurium at the time of slaughter was therefore estimated by the IRA team to be ***moderate***.

Rel_2: Infection detected through flock surveillance and the flock withdrawn from slaughter

Clinical signs due to *S*. Enteritidis and *S*. Typhimurium infection usually occur only in very young birds. In birds of slaughter age infection will most likely be inapparent. The likelihood that an infected flock will be detected through routine flock surveillance, and the flock withheld from slaughter, was assessed by the IRA team as ***extremely low***.

Rel_3: Selection of an infected chicken from an infected flock (within flock prevalence)

S. Enteritidis and MDR *S*. Typhimurium are very contagious, and in an outbreak, many birds within a shed are likely to be infected. If an infected flock were sent to slaughter, the IRA team considered that the likelihood that a selected individual chicken will be infected was ***moderate***.

Rel_4: Background cross contamination rate

This likelihood represents the background cross-contamination rate during slaughter and processing and applies to cross-contamination with material, infectious or otherwise, from other carcasses, including those tissues or materials in which the disease agent tends to localise.

S. Enteritidis and MDR *S*. Typhimurium persist in the liver, spleen and other organs of infected chickens, and contamination of chicken carcasses at processing plants is common. The organisms are resistant to the effects of chlorine in the presence of organic matter. The IRA team considered that the likelihood of a carcass being contaminated with potentially contaminated material from other birds, especially from the digestive tract, was ***moderate***.

Rel_5: Likelihood that an uninfected carcass will be contaminated with the disease agent during slaughter and processing

As discussed in the Method for Risk Assessment (page 58, Part B), Rel_4 was used to calculate Rel_{5a} (the likelihood that an uninfected carcass from an infected flock will become contaminated during slaughter and processing) and Rel_{5b} (the likelihood that an uninfected carcass from an uninfected flock will become contaminated during slaughter and processing).

For *S*. Enteritidis and MDR *S*. Typhimurium, Rel_{5a} was calculated as ***low***, and Rel_{5b} was calculated as ***low***.

Rel_6: The likelihood that the carcass of a bird that was *infected* before slaughter will be removed as a result of inspections before or during processing

No obvious post-mortem lesions are likely to be present during slaughter and processing. The IRA team considered that the rejection rate of infected carcasses would be equal to the background rejection rate of 0.75%.

Rel_7: The likelihood that the carcass of a bird that was *not infected* before slaughter will be removed as a result of inspections before or during processing

The likelihood that an uncontaminated/uninfected carcass will be removed during processing inspections was considered to be equal to the background rejection rate of 0.75% as described in the Method section (page 59, Part B).

Rel_8: Inactivation of the agent during further processing, storage, handling and transport

The carcasses for importation will be chilled or frozen, and *S*. Enteritidis and MDR *S*. Typhimurium can persist for weeks or months in carcasses at low temperatures. The IRA team concluded that the likelihood of inactivation of the bacteria during further processing, storage, handling and transport is ***extremely low***.

Conclusions – Release assessment

After inserting the above estimates into the simulation model, and using the outputs from the model as a guide, the IRA team concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a ***moderate*** likelihood that imported chicken meat would be infected or contaminated with *S*. Enteritidis or MDR *S*. Typhimurium.

Exposure assessment

Exposure Group 1: Wild birds

Of the steps identified as determinants in the infection of wild birds by exposure to scraps from an imported contaminated chicken carcass, $WB_{agentsurvival}$ and $WB_{infectivedose}$ are pathogen-dependent. All other determinants are pathogen-independent and are discussed in detail in the Method section (pages 67-86, Part B).

$WB_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by a wild bird

S. Enteritidis or MDR *S.* Typhimurium, protected within chicken meat scraps, is likely to survive in the environment under ambient temperatures of 10 °C to 35 ºC for several days. Under appropriate conditions, these organisms may multiply in chicken meat. The IRA team considered that the likelihood that the disease agent will remain viable until wild birds locate and scavenge the material was ***high***.

$WB_{infectivedose}$: The likelihood that the amount of contaminated chicken waste eaten by a wild bird is sufficient to produce infection

Bacterial contamination of carcasses is most likely to result from faecal contamination during processing, and the dose of bacteria present on the carcass is likely to be relatively low. However, organisms may be sequestered in the heart, liver and gizzard, and if these organs were available to wild birds, the risk of infection associated with their consumption would be higher than for other carcass scraps. This risk assessment considers the importation of whole carcasses, including heart, gizzard and liver; therefore $WB_{infectivedose}$ was estimated on the assumption that these organs may be discarded in refuse.

Infections with *S.* Enteritidis and *S.* Typhimurium have been reported in a range of avian species, and there are reports of infection of wild birds known to frequent refuse tips (Friend 1999), and, in the case of MDR *S.* Typhimurium, in wild birds on farms with infected cattle (Davies 2001). The IRA team considered that there was a ***low*** likelihood that *S.* Enteritidis or MDR *S.* Typhimurium would infect a wild bird consuming contaminated meat scraps.

Exposure Group 2: Low biosecurity poultry

Of the steps identified as determinants in the infection of low biosecurity poultry by exposure to material from an imported contaminated chicken carcass, $BP_{agentsurvival}$, $BP_{infectivedose}$, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen-dependent. All other determinants are pathogen-independent and are discussed in detail in the Method section (pages 67-86, Part B).

$BP_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by low biosecurity poultry

The time between feeding of scraps and consumption by low biosecurity poultry is likely to be very short, so environmental degradation of the disease agent will be minimal. The likelihood that the agent will remain viable was assessed by the IRA team as ***certain (=1)***.

$BP_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a low biosecurity bird is sufficient to produce infection

Transmission of these organisms to chickens has been demonstrated via the oral route. Chicken carcasses may be contaminated with *S.* Enteritidis or MDR *S.* Typhimurium, and these organisms may multiply if the meat is kept in favourable conditions. Bacteria may be sequestered in the heart, liver and gizzard, and if these organs were available to low biosecurity poultry, the risk of infection associated with their consumption would be higher than for other carcass scraps. This risk assessment considers the importation of whole carcasses, including

heart, gizzard and liver. Therefore, $BP_{infectivedose}$ was estimated on the assumption that these organs may be fed to low biosecurity poultry.

The overall likelihood that low biosecurity poultry would be infected with *S*. Enteritidis or MDR *S*. Typhimurium as a result of consuming the contaminated chicken meat scraps was therefore considered by the IRA team to be ***high***.

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

The likelihood that these organisms would survive the rendering process was negligible. The post-processing contamination of animal sources of protein is considered to be the most important single factor responsible for the presence of *Salmonella* spp. in rendered feeds. However, while it is recognised that overall salmonella contamination rates in animal feeds may reach 40%, the likelihood of contamination with these particular serotypes is negligible since they are not present in Australian poultry, and the vast majority of material in the rendering plant will be from Australian sources. Any contamination with exotic agents from imported material was considered by the IRA team to be negligible when compared with the competing agents derived from domestically produced material. For *S.* Enteritidis or MDR *S.* Typhimurium, the IRA team assessed this likelihood as negligible. Therefore, the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with *S.* Enteritidis or MDR *S*. Typhimurium was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

Given that *S.* Enteritidis or MDR *S*. Typhimurium would be destroyed by rendering, and that feed derived from contaminated carcasses would be diluted with feed from non-risk material, the likelihood that the amount of final poultry ration eaten by a bird would contain an oral infectious dose of *S.* Enteritidis or MDR *S*. Typhimurium was considered by the IRA team to be ***negligible***.

Exposure Group 3: Medium biosecurity commercial poultry

Of the steps identified as determinants in the infection of medium biosecurity commercial poultry by exposure to material from an imported contaminated chicken carcass, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with *S.* Enteritidis or MDR *S*. Typhimurium was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that the amount of the final poultry ration consumed would contain an oral infectious dose of *S.* Enteritidis or MDR *S.* Typhimurium was considered to be ***negligible***.

Exposure Group 4: Non-avian species

As discussed above, $NAS_{agentsurvival}$ was considered to be equal to $WB_{agentsurvival}$.

S. Enteritidis and MDR *S.* Typhimurium are able to colonise many types of livestock, and rodents, birds and cats may act as reservoirs of infection.

The IRA team considered that rodents, foxes, feral pigs and dogs would be the major non-avian species likely to scavenge refuse and ingest discarded waste at a refuse dump, while zoo animals could be exposed if fed imported uncooked chicken meat. Infection with MDR *S.* Typhimurium is likely to be a major concern in rodents and zoo animals, while infection with either *S.* Enteritidis or MDR *S.* Typhimurium is likely to be a concern in feral pigs. The likelihood that an amount of scrap from an infected chicken carcass would contain a sufficient quantity of the agent to infect non-avian species ($NAS_{infectivedose}$) was assessed as ***high***.

Conclusions – Exposure assessment

The partial likelihood of exposure for each of the exposure groups was calculated after inserting the estimates of the distribution variables, exposure group-dependent variables and pathogen-dependent variables into the simulation model. A summary of the outputs from the simulation model is shown in Table 58.

Table 58. Partial likelihoods of exposure (PLE)

Exposure group	PLE
Wild birds	Extremely low
Low biosecurity poultry	Very low
Medium biosecurity poultry	Negligible
Non-avian species	Very low

Consequence assessment

Establishment and spread was considered in the context of the outbreak scenarios, described in the Method for Risk Assessment.

Estimating the likelihood of each outbreak scenario

The partial likelihood of establishment and spread (PLES) of *S.* Enteritidis and MDR *S.* Typhimurium for each of the exposure groups is described below. Four outbreak scenarios were considered relevant:

1. Disease agent does not establish or is not recognised within the directly exposed population

2. Disease agent establishes within the directly exposed population, is identified and is eliminated by human efforts or by natural means
3. Disease agent establishes in the directly exposed population, spreads within a district/region, including into other exposure groups if applicable, and is eliminated by human action or by natural means
4. Disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia.

Wild birds

Wild birds may act as reservoirs of infection of *S*. Enteritidis and MDR *S*. Typhimurium and outbreaks of *S*. Typhimurium infection have been reported in wild birds. However, such infections are not common. The IRA team considered that the most likely outcome of infection of a wild bird resulting from scavenging chicken meat scraps would be a single or a few isolated occurrences of infection in wild birds. Reported outbreaks of salmonellosis in wild birds are not common, and have generally been associated with overcrowding or concurrent disease. However, occasional outbreaks have been associated with scavenging on refuse tips (Alley et al. 2002). Infection of wild birds with *S*. Enteritidis and MDR *S*. Typhimurium, with subsequent spread to poultry, could occur, although this was considered to be unlikely. If these infections became established in commercial poultry, it was considered likely that action would be taken to control them. In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 59).

Table 59. Estimated partial likelihood of establishment and spread (PLES) values for *S*. Enteritidis and MDR *S*. Typhimurium in wild birds

Outbreak scenario 1:	$PLES_{(wb)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(wb)}2$	**Low**
Outbreak scenario 3:	$PLES_{(wb)}3$	**Low**
Outbreak scenario 4:	$PLES_{(wb)}4$	**Extremely low**

Low biosecurity poultry

Although this exposure group includes commercial free-range poultry and ratites, the IRA team considered that the sub-set of this group most likely to be exposed to scraps of imported chicken meat was small flocks of backyard poultry, because these are more likely to be directly fed table scraps. In such flocks, there is less opportunity for the generation of high levels of environmental contamination, than might occur with an outbreak of infectious disease in a large commercial flock. The IRA team considered that the most likely outcome of infection of a backyard bird would be a single or a few isolated occurrences of infection in backyard birds. If the disease were to establish in the flock, mechanical transmission of the organism by contaminated persons or fomites, and transmission by movement of birds may facilitate spread of the organism beyond the initially infected flock before it is recognised and eradication measures are implemented. *S*. Enteritidis and MDR *S*. Typhimurium seldom cause clinical disease, except in susceptible young birds. Recognition in a low biosecurity flock could be

delayed due to the lack of clinical signs in older birds, so it would be unlikely to be recognised unless the owners of the infected flock, or other people in contact, became ill. However, if the infection spread to commercial poultry, the likelihood of detection would increase because of the increased level of in-house monitoring of commercial poultry and the increased likelihood of infection in humans. As stated above, it is likely that actions to control the infection would follow. Rodents could also be infected and spread infection among low biosecurity poultry in a local area. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 60).

Table 60. Estimated partial likelihood of establishment and spread (PLES) values for *S*. Enteritidis and MDR *S*. Typhimurium in low biosecurity poultry

Outbreak scenario 1:	$PLES_{(bp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(bp)}2$	**Very low**
Outbreak scenario 3:	$PLES_{(bp)}3$	**Low**
Outbreak scenario 4:	$PLES_{(bp)}4$	**Very low**

Medium biosecurity commercial poultry

The IRA team considered that the most feasible route for exposure of medium biosecurity commercial poultry to imported carcass components would be through poultry feed containing inadequately rendered processing waste. The likelihood that pathogens would remain viable following rendering of contaminated imported carcasses and parts was assessed as negligible (page 182, Part C). Nevertheless, assessment of PLES was based on the assumption that medium biosecurity commercial poultry had been exposed to poultry feed contaminated with the disease agent.

In general, *S*. Enteritidis and MDR *S*. Typhimurium do not cause severe systemic disease in physiologically and immunologically normal, healthy adult chickens. Clinical disease usually occurs only in very young birds and the clinical signs are similar to those of pullorum disease. It is unlikely that flock infection due to *S*. Enteritidis and MDR *S*. Typhimurium would be recognised in medium biosecurity commercial poultry, unless an outbreak occurred in humans, or unless monitoring programs detected their presence. In view of these factors, outbreak scenarios 3 and 4 are considered the most likely.

However, once an outbreak of disease in humans had taken place, it was considered that trace back activities would quickly identify the source, and action to control the infection would follow. It was therefore considered that Scenario 3 was the most likely outcome, and it was less likely that the disease would become endemic.

The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 61).

Table 61. Estimated partial likelihood of establishment and spread (PLES) values for *S*. Enteritidis and MDR *S*. Typhimurium in medium biosecurity commercial poultry

Outbreak scenario 1:	$PLES_{(cp)}1$	**Very low**
Outbreak scenario 2:	$PLES_{(cp)}2$	**Very low**
Outbreak scenario 3:	$PLES_{(cp)}3$	**Moderate**
Outbreak scenario 4:	$PLES_{(cp)}4$	**Low**

Non-Avian Species

Rodents, birds and cats may act as reservoirs of infection of *S*. Enteritidis and MDR *S*. Typhimurium. Infection of non-avian species, for example rodents, with *S*. Enteritidis and *S*. Typhimurium, with subsequent spread to poultry, has been reported. However, it was considered highly unlikely that infection of non-avian species would be diagnosed unless people contracted the infection from their pets or from zoo animals and became ill. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 62).

Table 62. Estimated partial likelihood of establishment and spread (PLES) values for *S*. Enteritidis and MDR *S*. Typhimurium in non-avian species

Outbreak scenario 1:	$PLES_{(nas)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(nas)}2$	**Low**
Outbreak scenario 3:	$PLES_{(nas)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(nas)}4$	**Very low**

Conclusion – Likelihood assessments

The estimates for the likelihood of release, the partial likelihoods of exposure for each of the exposure groups, and the partial likelihood of establishment and spread for each of the outbreaks scenarios were combined with the expected volume of trade using the simulation model. This allowed the calculation of partial annual likelihood of entry, exposure, establishment and spread for each of the outbreak scenarios. The results of this calculation are shown in Table 63.

Estimating the impacts associated with each outbreak scenario

For each outbreak scenario the direct and indirect impacts of *S*. Enteritidis or MDR *S*. Typhimurium infection were estimated at the national, state or territory, district/region and local level, as described in the Methods section (page 90-95, Part B).

Table 63. Partial annual likelihood of entry, exposure, establishment and spread (PALEEES) for the outbreak scenarios

Exposure group	Outbreak scenario	PALEEES
Wild birds	Scenario 1	High
	Scenario 2	High
Low biosecurity poultry	Scenario 1	High
	Scenario 2	High
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	High
	Scenario 2	High
Total all exposure groups	Scenario 3	High
Total all exposure groups	Scenario 4	High

Outbreak Scenario 1

By definition, outbreak scenario 1 means that the disease agent does not establish in the initially exposed population, or does not cause sufficient disease to lead to investigation and recognition of the infection. Therefore the impacts will be ***unlikely to be discernible*** for all exposure groups and all criteria.

Outbreak Scenario 2

The impacts of this outbreak scenario (disease agent establishes within the directly exposed population, and is identified and eliminated) will differ between exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

Several species of introduced and native wild birds, found in Australia, have been shown to be susceptible to infection with *S*. Enteritidis or *S*. Typhimurium, and most Australian psittacine birds would be susceptible to infection with these species. However, since wild birds do not play a significant part in production, direct economic loss from death of wild birds, were it to occur, is not measurable. Other impacts from the death of wild birds will be considered under direct criterion 2 and indirect criterion 4. The direct impacts of disease on wild birds were assessed by the IRA team as ***unlikely to be discernible*** at any level.

S. Enteritidis and MDR *S*. Typhimurium seldom cause clinical disease, except in susceptible young birds. Older birds may show little or no clinical disease. The mortality rates in chicks and poults vary from 10% to 80% (Barrow 2000). An outbreak of *S*. Enteritidis or MDR *S*. Typhimurium, contained within a local population of poultry, may result in losses to individual owners.

The impacts of a disease outbreak in a local population of low biosecurity poultry were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory, or district/region levels but ***minor*** at the local level, especially if free-range commercial poultry were involved. Impacts of an outbreak of *S*. Enteritidis and MDR *S*. Typhimurium contained within a local population of medium biosecurity commercial poultry were assessed as ***unlikely to be discernible*** at national, State/Territory, or district/region levels but ***minor*** at the local level. Costs related to the deaths of birds are accounted for under this criterion, but costs arising from the implementation and administration of the eradication program will be accounted for under indirect criterion 1.

The direct impacts of disease on non-avian species were assessed as ***unlikely to be discernible*** at any level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

Several species of introduced and native wild birds, found in Australia, have been shown to be susceptible to infection with *S*. Enteritidis and *S*. Typhimurium. However, outbreaks of Salmonellosis in wild birds are uncommon and are generally associated with specific predisposing factors. The IRA team considered that an outbreak of *S*. Enteritidis or MDR *S*. Typhimurium is likely to result in minimal deaths of wild birds, and the impacts were assessed as ***unlikely to be discernible*** at all levels.

The IRA team considered that the impacts of an outbreak of *S*. Enteritidis or MDR *S*. Typhimurium contained within a local population of low biosecurity poultry on this criterion were ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak of *S*. Enteritidis or MDR *S*. Typhimurium contained within a local population of medium biosecurity commercial poultry on this criterion were assessed as ***unlikely to be discernible*** at all levels.

The impacts of infection in non-avian species were assessed as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

If an outbreak of *S*. Enteritidis or MDR *S*. Typhimurium were diagnosed in wild birds, there would be an increase in surveillance and monitoring of the wild bird population, and surveillance of low biosecurity and medium biosecurity commercial poultry. Impacts at the local level were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory, or district/region levels but ***minor*** at the local level.

The impacts of a recognised outbreak of *S*. Enteritidis or MDR *S*. Typhimurium in low biosecurity poultry, especially if involving a free-range flock, were assessed as ***unlikely to be discernible*** at national and State/Territory or district/region levels but ***minor*** at the local level. Affected flocks would be destroyed, quarantine and movement controls instigated, and increased surveillance and monitoring of the low biosecurity poultry and medium biosecurity commercial poultry populations implemented. Control programs could also lead to disruption in breeding and production programs.

These measures would also be applied if an outbreak was diagnosed in medium biosecurity commercial poultry. Accordingly, the impacts of an outbreak diagnosed in medium biosecurity commercial poultry were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory or district/region levels, and ***minor*** at the local level. The impact of a recognised outbreak in non-avian species, with no subsequent spread, was assessed as ***unlikely to be discernible*** at any level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

The domestic trade and industry impacts of an outbreak of *S*. Enteritidis or MDR *S*. Typhimurium in the local wild bird population were assessed by the IRA team as ***unlikely to be discernible***.

Similarly, the impacts of a disease outbreak in a local population of low biosecurity poultry were assessed as ***unlikely to be discernible*** at all levels.

Depending on the extent of the outbreak, financial losses in the domestic poultry industry, especially the egg industry, and in associated sales and service industries could be considerable. Impacts of an outbreak of *S*. Enteritidis or MDR *S*. Typhimurium in a local population of medium biosecurity commercial poultry were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level.

The impact of a recognised outbreak in non-avian species, with no subsequent spread, was assessed as ***unlikely to be discernible*** at any level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

The impacts of an outbreak of *S*. Enteritidis or MDR *S*. Typhimurium within wild birds, low biosecurity poultry, medium biosecurity commercial poultry or non-avian species on international trade were assessed as ***unlikely to be discernible*** at any level.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts on the environment of an outbreak of *S*. Enteritidis or MDR *S*. Typhimurium within wild birds, low biosecurity poultry, medium biosecurity commercial poultry or non-avian species were assessed by the IRA team as ***unlikely to be discernible*** at any level.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Depending on the extent of the outbreak, restrictions on movement of birds, eggs, and poultry products may lead to temporary community disruption. There could be community concern about the possible transmission of the organism to people. However, these impacts are most likely to be felt if the outbreak affects medium biosecurity commercial poultry. The IRA team considered that the impacts of a disease outbreak in medium biosecurity commercial poultry on this criterion were ***unlikely to be discernible*** at national, state and district/region levels, but

minor at the local level, while the impacts of an outbreak in wild birds, low biosecurity poultry or non-avian species on this criterion were assessed as ***unlikely to be discernible*** at any level.

Outbreak Scenario 3

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a district/region, and is eliminated) will be the same for all exposure groups, no matter which exposure group has been directly exposed to *S*. Enteritidis or MDR *S*. Typhimurium in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

S. Enteritidis and MDR *S*. Typhimurium seldom cause clinical disease in chickens, except in young chicks. Older birds may show little or no clinical disease. The mortality rates in chicks and poults vary from 10% to 80%. While the impacts at national, State/Territory and district/region levels were assessed by the IRA team as ***unlikely to be discernible***, there will be ***minor*** impacts at the local level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

The impacts of a contained outbreak on the environment were assessed by the IRA team as ***unlikely to be discernible*** at all levels for reasons stated previously under outbreak scenario 2.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

If *S*. Enteritidis or MDR *S*. Typhimurium were to spread to poultry or caged birds, eradication, surveillance and monitoring programs would be required, particularly in commercial poultry. While the impact on the national economy was assessed by the IRA team as ***unlikely to be discernible***, the team considered that there may be ***minor*** impacts at the State/Territory level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Movement controls associated with control and eradication programs would adversely affect domestic trade. There would be alterations in marketing arrangements for affected flocks, and perhaps increased testing of unaffected flocks, leading to disruption of normal marketing patterns. Adverse publicity may affect the sales of meat and eggs. Impacts were assessed by the IRA team as ***unlikely to be discernible*** nationally, but ***minor*** at the State/Territory level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

The IRA team considered that a limited outbreak of *S*. Enteritidis or MDR *S*. Typhimurium was likely to have ***no discernible impacts*** on this criterion at national or State/Territory levels, but ***minor*** impacts at the district/region level.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of a disease outbreak on the environment were considered by the IRA team to be ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Depending on the extent of the outbreak, restrictions on movement of birds, eggs, and poultry products may lead to temporary community disruption. There could be community concern about the possible transmission of the organism to people. The impacts of this outbreak scenario on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at national, state and district/region levels, but ***minor*** at the local level.

Outbreak Scenario 4

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a State/Territory, and becomes endemic in Australia) will be the same for all exposure groups, no matter which exposure group has been directly exposed to *S*. Enteritidis or MDR *S*. Typhimurium in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production effects) of production, domestic or feral animals

S. Enteritidis and MDR *S*. Typhimurium seldom cause clinical disease, except in susceptible young birds. Older birds may show little or no clinical disease. The mortality rates in chicks and poults vary from 10% to 80%. If the disease spreads more widely through low biosecurity poultry and medium biosecurity commercial poultry, there may be significant losses of young birds. The impacts were assessed by the IRA team as ***unlikely to be discernible*** at the national, State/Territory and district/region levels, but ***minor*** at the local level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

The impacts of a more general outbreak of *S*. Enteritidis or MDR *S*. Typhimurium on the environment were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

If *S.* Enteritidis spreads to a more general population of poultry, there will be some effort at the State/Territory, district/region and local levels to eradicate the disease, with monitoring and surveillance programs. This may also be the case for MDR *S.* Typhimurium. The impact was assessed by the IRA team as ***unlikely to be discernible*** at the national level, but ***minor*** at the State/Territory level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Domestic trade and industry impacts would be more widespread than those described under scenario 3. The IRA team considered that a more general outbreak was likely to have ***minor*** impacts on domestic trade and industry nationally.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

Impacts of a more general outbreak of *S.* Enteritidis or MDR *S.* Typhimurium on international trade were assessed by the IRA team as ***unlikely to be discernible*** at the national and State/Territory levels, and ***minor*** at the district/region level.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The IRA team considered that a generalised outbreak of *S.* Enteritidis or MDR *S.* Typhimurium was likely to have ***no discernible impacts*** on this criterion at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Disruption of the flow of products and services, and decreased production may cause some job losses on farms and in associated industries. Prolonged loss or reduction of income for contract growers and their suppliers and associated industries may result, until farms can be re-stocked following attempts to control the infection. The impact of a general outbreak of *S.* Enteritidis or MDR *S.* Typhimurium on communities was assessed by the IRA team as ***unlikely to be discernible*** at the national and State/Territory levels, but ***minor*** at the district/region level.

Conclusions – impact assessment

The above estimates for each of the impact criteria were entered into the simulation model, and the overall impact of each outbreak scenario was estimated. Results are shown in Table 64.

Table 64. Impacts of each outbreak scenario

Exposure group	Outbreak scenario	Impact
Wild birds	Scenario 1	Negligible
	Scenario 2	Negligible
Low biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Negligible
	Scenario 2	Negligible
	Total Scenario 3	Low
	Total Scenario 4	Moderate

Partial annual risk estimate for each outbreak scenario

The partial annual likelihood of entry, exposure, establishment and spread were combined with the impacts of the relevant outbreak scenarios to provide an estimate of the partial annual risk associated with each outbreak scenario. The outcomes of this process are shown in Table 65.

Unrestricted risk estimate

The overall risk associated with the import of whole chicken carcasses was assessed as ***moderate*** for MDR *S*. Typhimurium and *S*. Enteritidis. This unrestricted risk estimate exceeds Australia's ALOP, and therefore, risk management is deemed necessary.

Direct impact on human life or health

S. Enteritidis and MDR *S*. Typhimurium are major causes of food-borne salmonellosis in humans. The cost of treatment and loss of productivity as a result of human cases of infection with *Salmonella* Enteritidis or MDR *Salmonella* Typhimurium in Australia could be considerable (Sergeant et al. 2003).

Table 65. Partial annual risk (PAR) of each outbreak scenario

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Medium biosecurity poultry	Scenario 1	Negligible	Negligible	Negligible
	Scenario 2	Negligible	Negligible	Negligible
Non-avian species	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
	Total Scenario 3	High	Low	Low
	Total Scenario 4	High	Moderate	Moderate

Salmonella Arizonae

Technical Information

Background

The name *Salmonella* Arizonae is used to designate a group of bacteria comprising some 415 different antigenic types (Davos 2001). The nomenclature and classification of this group has been somewhat controversial, and *S.* Arizonae is used in this document in place of the previous names *Salmonella arizona, Arizona arizonae* or *Arizona hinshawii*, and *S. enterica* subspecies IIIa or *S. enterica* subspecies *arizonae*. Although infections have been reported in sheep, cattle, reptiles and birds, the organism is most commonly isolated from reptiles and turkeys (Shivaprasad 1997).

Arizonosis in turkeys is an acute systemic disease and may cause significant economic losses due to reduced egg production and hatchability, and morbidity and mortality in poults. Although reports of arizonosis in chickens are few, evidence suggests that serious disease could result if the organism becomes established in chickens (Silva, Hipolito, and Grecchi 1980). Historically, isolates from chickens and turkeys in the United States and United Kingdom were of two serovars, 18:Z_4,Z_{32} (original designation 7a,7b:1,7,8:-) and 18:Z_4,Z_{23} (original designation 7a,7b:1,2,6:-) (Hall and Rowe 1992; Silva, Hipolito, and Grecchi 1980). Reports suggest that a change has occurred in the relative proportion of the two serovars, and that isolation of 18:Z_4,Z_{23} is now rare (Shivaprasad et al. 1997). Serovar 18:Z_4,Z_{32} has not been isolated in Australia (Davos 2001).

Human infections have been reported in association with handling of reptiles or ingestion of rattlesnake capsules (containing uncooked rattlesnake meat), and, although serious, are uncommon (Shivaprasad et al. 1997).

Arizonosis is not an OIE-listed disease.

Agent taxonomy

Salmonellae belonging to the group *S.* Arizonae are Gram-negative, motile, non-sporogenic bacilli belonging to the family *Enterobacteriaceae*. The group comprises some 415 different antigenic types of which serovars 18:Z_4,Z_{32} and 18:Z_4,Z_{23} have been isolated from turkeys and chickens. The organism is a facultative anaerobe.

Agent characteristics

S. Arizonae is readily destroyed by heat and by common disinfectants (Shivaprasad et al. 1997). The organism can survive in contaminated water for 5 months, contaminated feed for 17 months, soil for six to seven months and on tools, utensils and in the poultry house for five to 25 weeks. On eggshell, *S.* Arizonae can survive up to 12 weeks at 20 °C, and at least 25 weeks on egg cartons (Geissler and Youssef 1981; Shivaprasad et al. 1997).

Epidemiology

S. Arizonae is non-host specific and occurs worldwide. Among poultry, *S.* Arizonae is most frequently isolated from turkeys, but chickens are affected both experimentally and naturally. Wild birds and reptiles are potential sources of infection in poultry houses.

Infected adult birds may carry the organism in their intestine or reproductive tract for prolonged periods and thus spread the disease (Crespo et al. 2004). *S.* Arizonae has been isolated from soil, feed, litter or eggshell (Geissler and Youssef 1981) and may be transmitted in contaminated feed and water. The organism may also be spread in the incubator and brooder by direct contact.

There are few reports of the prevalence of avian arizonosis. The organism has been eliminated from turkey flocks in the United Kingdom by a rigorous program of slaughter of the original breeding flock, selection, management, screening and culling of progeny flocks (Shivaprasad et al. 1997). However, a recent report from the United States indicates that this organism persists in some integrated turkey operations (Crespo et al. 2004).

Clinical signs

Clinical signs of arizonosis are not specific, and are rarely seen in adult birds. Infected chicks and poults may show listlessness, diarrhoea, leg paralysis, twisted necks and vent pasting. Blindness and nervous signs may also be observed (Silva, Hipolito, and Grecchi 1980). Mortality of up to 50% has been reported in infected poults. Chicks and poults are most susceptible within a few days of hatching, and mortalities may continue for three to four weeks.

Pathogenesis

Arizonosis has been transmitted experimentally by subcutaneous inoculation, contact, mouth and eye-drop (Silva, Hipolito, and Grecchi 1980). Infection results in septicaemia, especially in young fowl. *S.* Arizonae localises in the wall of the intestine and is excreted in the faeces. The organism has been isolated in the faeces of experimentally infected chickens up to 49 days after infection (Youssef and Geissler 1979).

Although recovery of *S.* Arizonae from the ovary of infected birds suggests that transovarian transmission may occur, this is not common. It has been shown that *S.* Arizonae is able to penetrate the shell and shell membrane of intact eggs contaminating the contents of chicken and turkey eggs (Williams and Dillard 1968). *S.* Arizonae has also been isolated from semen of turkeys, although it is probable that this was due to faecal contamination (Perek, Elian, and Heller 1969).

Pathology

In affected poults and chicks, lesions observed post mortem are those of a generalised septicaemia. The liver is enlarged and mottled, the heart enlarged, and retained yolks and caseous exudate may be present in the abdominal cavity. Exudate may be present on the meninges and in the vitreous humor of the eye.

Immunology

Several serological tests including the rapid serum plate test, somatic tube agglutination test, rapid whole blood and microagglutination tests have been developed to detect infection in

turkeys. However, these are not entirely effective (Shivaprasad et al. 1997). Antibody titres do not persist for long periods and may not be detectable in birds with subclinical infections. Adult carrier birds may lack detectable antibody 12 to 14 weeks after exposure and there is an antibody negative phase in infected turkey hens from 16 to 20 weeks of age, the time at which most breeder flocks are tested. An ELISA has been developed which is reported to be sensitive and specific for the detection of infection in breeding turkey flocks (Nagaraja et al. 1986).

Vaccination using bacterins (inactivated vaccines) is available and is reported to decrease shedding of the organisms in the faeces and protect hens from systemic infection (Shivaprasad et al. 1997).

Diagnosis

High mortality in turkey poults in association with neurological signs and blindness is suggestive of avian arizonosis. Confirmation of the diagnosis depends on isolation and identification of *S.* Arizonae from infected birds. Samples of most organs plus eye, heart blood or unabsorbed yolk sac are suitable. Culture of shells and shell membrane of turkey egg rather than yolk material allows rapid detection of contaminated eggs (Greenfield, Bigland, and Dukes 1971).

Transmission in chicken meat

Since infection with *S.* Arizonae results in septicaemia, organisms may localise in a range of body tissues, including muscle. In addition, infected adult poultry may become intestinal carriers and excrete bacteria for prolonged periods, leading to carcass contamination during processing. *S.* Arizonae has been demonstrated in chicken meat samples overseas (Bidarte 1990; Izat, Kopek, and McGinnis 1991).

Quarantine significance

S. Arizonae is not an OIE-listed disease agent.

Serovar 18:Z_4,Z_{32} has not been isolated in Australia (Davos 2001), however, *S.* Arizonae is not notifiable in any state or territory of Australia, nor subject to official controls. *S.* Arizonae is not included in the Emergency Animal Disease Response Agreement. Therefore, it is considered to be of relatively minor concern, and is unlikely to have consequences for the poultry industry that are discernible beyond the local level.

Risk Assessment

Release Assessment

Rel_1: Selection of source flock (between flock prevalence)

Arizonosis in domestic poultry is primarily a disease of turkeys. Although *S.* Arizonae occurs worldwide and infects a number of species, reports of arizonosis in domestic chickens are few. The likelihood that a source flock of chickens will be infected with *S.* Arizonae was estimated by the IRA team to be ***extremely low***.

Rel_2: Infection detected through flock surveillance and the flock withdrawn from slaughter

Clinical signs of arizonosis in affected chickens are not specific. The likelihood that an infected flock will be detected through routine flock surveillance, and the flock withheld from slaughter was assessed as ***extremely low***.

Rel_3: Selection of an infected chicken from an infected flock (within flock prevalence)

Reports of arizonosis in domestic chickens are few and within flock prevalence of infection is unknown. However, the organisms are not as well adapted to growing in chickens as in other hosts. In the opinion of the IRA team, the likelihood that a selected individual chicken will be infected was assessed as ***low***.

Rel_4: Background cross-contamination rate

This likelihood represents the background cross-contamination rate during slaughter and processing and applies to cross-contamination with material, infectious or otherwise, from other carcasses, including those tissues or materials in which the disease agent tends to localise. *S.* Arizonae localises in the wall of the intestine of infected poultry and may be excreted for prolonged periods, leading to carcass contamination during processing. The IRA team considered that the likelihood of a carcass being contaminated with potentially contaminated material from other birds, especially from the digestive tract, is ***moderate***.

Rel_5: Likelihood that an uninfected carcass will be contaminated with the disease agent during slaughter and processing

As discussed in the Method for Risk Assessment (page 58, Part B), Rel_4 was used to calculate Rel_{5a} (the likelihood that an uninfected carcass from an infected flock will become contaminated during slaughter and processing) and Rel_{5b} (the likelihood that an uninfected carcass from an uninfected flock will become contaminated during slaughter and processing).

For *S.* Arizonae, Rel_{5a} was calculated as ***low***, and Rel_{5b} was calculated as ***extremely low***.

Rel_6: The likelihood that the carcass of a bird that was *infected* before slaughter will be removed as a result of inspections before or during processing

No obvious post-mortem lesions are likely to be present during slaughter and processing. The IRA team considered that the rejection rate of infected carcasses would be equal to the background infection rate of 0.75%.

Rel_7: The likelihood that the carcass of a bird that was *not infected* before slaughter will be removed as a result of inspections before or during processing

The likelihood that an uncontaminated/uninfected carcass will be removed during processing inspections was considered to be equal to the background rejection rate of 0.75% as described in the Method section (page 59, Part B).

Rel_8: Inactivation of the agent during further processing, storage, handling and transport

Given that the carcasses for importation will be chilled or frozen, and that salmonellae can persist for weeks or months in carcasses at low temperatures, the likelihood of inactivation of *S.* Arizonae during further processing, storage, handling and transport was assessed as ***extremely low***.

Conclusions – Release assessment

After inserting the above estimates into the simulation model, and using the outputs from the model as a guide, the IRA team concluded that, in the absence of risk management and without considerations regarding the exporting country, there was an ***extremely low*** likelihood that imported chicken meat would be infected or contaminated with *S.* Arizonae.

Exposure assessment

Exposure Group 1: Wild birds

Of the steps identified as determinants in the infection of wild birds by exposure to scraps from an imported contaminated chicken carcass, $WB_{agentsurvival}$ and $WB_{infectivedose}$ are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$WB_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by a wild bird

S. Arizonae, protected within chicken meat scraps, is likely to survive in the environment under ambient temperatures of 10 ºC to 35 ºC for several days, giving ample time for wild birds to locate and scavenge the material. Under appropriate conditions, these organisms may multiply in chicken meat. The IRA team considered that the likelihood that the disease agent will remain viable until wild birds locate and scavenge the material was ***high***.

$WB_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a wild bird is sufficient to produce infection

Although infections have been reported in a range of species, *S.* Arizonae is most commonly associated with reptiles and amphibians and there are no reports of infection of wild birds known to frequent refuse tips. Bacterial contamination of carcasses is most likely to result from faecal contamination during processing; therefore, the dose of bacteria present on the carcass is likely to be relatively low. However, organisms may be sequestered in the heart, liver and gizzard, and if these organs were available to wild birds, the risk of infection associated with their consumption would be higher than for other carcass scraps. This risk assessment considers the importation of whole carcasses, including heart, gizzard and liver, therefore $WB_{infectivedose}$ was estimated on the assumption that these organs may be discarded in refuse.

The IRA team considered that there was a ***low*** likelihood that *S.* Arizonae would infect a wild bird consuming the contaminated meat scraps.

Exposure Group 2: Low biosecurity poultry

Of the steps identified as determinants in the infection of low biosecurity poultry by exposure to material from an imported contaminated chicken carcass, $BP_{agentsurvival}$, $BP_{infectivedose}$, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$BP_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by low biosecurity poultry

The time between feeding of scraps and consumption by low biosecurity poultry is likely to be very short, so environmental degradation of the disease agent will be minimal. The likelihood that the agent will remain viable was assessed by the IRA team as ***certain (=1)***.

$BP_{infectivedose}$: The likelihood that the amount of contaminated chicken waste eaten by a low biosecurity bird is sufficient to produce infection

Transmission of *S*. Arizonae to chickens by feeding of infected meat has not been documented. However, experimental transmission of the organism has been demonstrated via the oral route (Silva, Hipolito, and Grecchi 1980). Bacteria may be sequestered in the heart, liver and gizzard, and if these organs were available to low biosecurity poultry, the risk of infection associated with their consumption would be higher than for other carcass scraps. This risk assessment considers the importation of whole carcasses, including heart, gizzard and liver, therefore $BP_{infectivedose}$ was estimated on the assumption that these organs may be fed to low biosecurity poultry.

The overall likelihood that low biosecurity poultry would be infected with *S*. Arizonae as a result of consuming the contaminated chicken meat scraps was considered by the IRA team to be ***moderate,*** given that infection is less likely to establish in older backyard poultry.

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

The likelihood that the agent would survive the rendering process was negligible.

The post-processing contamination of animal sources of protein is considered to be the most important single factor responsible for the presence of *Salmonella* spp. in rendered feeds. However, while it is recognised that overall salmonella contamination rates in animal feeds may reach 40%, the likelihood of contamination with these particular serotypes is negligible since they are not present in Australian poultry, and the vast majority of material in the rendering plant will be from Australian sources. Any contamination with exotic agents from imported material was considered by the IRA team to be negligible when compared with the competing agents derived from domestically produced material. For *S*. Arizonae, the IRA team assessed this likelihood as negligible. Therefore the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with *S*. Arizonae was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

Given that *S.* Arizonae would be destroyed by rendering, and that feed derived from contaminated carcasses would be diluted with feed from non-risk material, the likelihood of the amount of the final poultry ration containing an oral infectious dose of *S.* Arizonae was considered by the IRA team to be ***negligible***.

Exposure Group 3: Medium biosecurity commercial poultry

Of the steps identified as determinants in the infection of medium biosecurity commercial poultry by exposure to material from an imported contaminated chicken carcass, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that poultry feed would be contaminated with *S.* Arizonae was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood of the amount of the final poultry ration containing an oral infectious dose of *S.* Arizonae was considered to be ***negligible***.

Exposure Group 4: Non-avian species

As discussed above, $NAS_{agentsurvival}$ was considered to be equal to $WB_{agentsurvival}$.

Although infections have been reported in a range of species, the major hosts of *S.* Arizonae are reptiles and amphibians. Some serotypes are host-adapted, and mammals and reptiles are not commonly infected with serotypes responsible for outbreaks of arizonosis in poultry.

The likelihood that an meat from an infected chicken carcass would contain a sufficient quantity of the agent to infect non-avian species ($NAS_{infectivedose}$) was assessed by the IRA team as ***very low***.

Conclusions – Exposure assessment

The partial likelihood of exposure for each of the exposure groups was calculated after inserting the estimates of the distribution variables, exposure group-dependent variables and pathogen-dependent variables into the simulation model. A summary of the outputs from the simulation model is shown in Table 66.

Table 66. Partial likelihoods of exposure (PLE)

Exposure group	PLE
Wild birds	Extremely low
Low biosecurity poultry	Very low
Medium biosecurity poultry	Negligible
Non-avian species	Extremely low

Consequence assessment

Establishment and spread was considered in the context of the outbreak scenarios, described in the Method of Risk Assessment.

Estimating the likelihood of each outbreak scenario

The partial likelihood of establishment and spread (PLES) of *S.* Arizonae for the different exposure groups is described below. Four outbreak scenarios were considered relevant:

1. Disease agent does not establish or is not recognised within the directly exposed population
2. Disease agent establishes within the directly exposed population, is identified and is eliminated by human efforts or by natural means
3. Disease agent establishes in the directly exposed population, spreads within a district/region, including into other exposure groups if applicable, and is eliminated by human action or by natural means
4. Disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia.

Wild birds

There are no reports of *S.* Arizonae infection of wild birds known to scavenge at refuse tips. The major hosts of *S.* Arizonae are reptiles and amphibians, and these species are not commonly infected with host-adapted serotypes responsible for outbreaks of arizonosis in poultry. The IRA team considered that the most likely outcome of infection of a wild bird resulting from scavenging imported contaminated chicken meat scraps would be a single or a few isolated occurrences of infection in wild birds, with *S.* Arizonae being unable to establish ongoing infection in the population. In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 67).

Table 67. Estimated partial likelihood of establishment and spread (PLES) values for *S*. Arizonae in wild birds

Outbreak scenario 1:	$PLES_{(wb)}1$	**High**
Outbreak scenario 2:	$PLES_{(wb)}2$	**Extremely low**
Outbreak scenario 3:	$PLES_{(wb)}3$	**Extremely low**
Outbreak scenario 4:	$PLES_{(wb)}4$	**Extremely low**

Low biosecurity poultry

Reports of infection of chickens with the poultry-adapted serotype of *S.* Arizonae are few, but infection is of particular significance in turkeys. Although this exposure group includes commercial free-range poultry and ratites, the IRA team considered that the sub-set of this group most likely to be exposed to scraps of imported chicken meat was small flocks of backyard poultry, because these are more likely to be directly fed table scraps. Moreover, turkeys represent a small proportion of the backyard poultry kept by households. In such flocks, there is less opportunity for the generation of high levels of environmental contamination than might occur with an outbreak of infectious disease in a large commercial flock. The most likely outcome of infection would be a single or a few isolated occurrences of infection.

Clinical signs of arizonosis are not specific, and are rarely seen in adult birds. Therefore, if the disease was to establish in the flock, it is unlikely that it would be recognised. Mechanical transmission of the organism by contaminated persons or fomites, and transmission by movement of birds may facilitate spread of the organism beyond the initially infected flock before it is recognised, and control measures are implemented. The disease is not notifiable, nor is it subject to the Emergency Animal Disease Response Agreement, so it is likely that control measures will be limited to those taken by the individual farmer. In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. Outbreaks of arizonosis in chickens are not known to have spread widely. The IRA team therefore assigned the following likelihoods to the four outbreak scenarios (Table 68).

Table 68. Estimated partial likelihood of establishment and spread (PLES) values for *S*. Arizonae in low biosecurity poultry

Outbreak scenario 1:	$PLES_{(bp)}1$	**High**
Outbreak scenario 2:	$PLES_{(bp)}2$	**Low**
Outbreak scenario 3:	$PLES_{(bp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(bp)}4$	**Very low**

Medium biosecurity commercial poultry

The IRA team considered that the most feasible route for exposure of medium biosecurity commercial poultry to imported carcass components would be through poultry feed containing inadequately rendered processing waste. The likelihood that pathogens would remain viable following rendering of contaminated imported carcasses and parts was assessed as negligible.

Nevertheless, assessment of PLES was based on the assumption that medium biosecurity commercial poultry had been exposed to poultry feed contaminated with the disease agent.

Infection with the poultry-adapted serotype of *S.* Arizonae occurs more frequently in turkeys than in chickens. Clinical signs are rarely seen in adult birds, and are not specific. However, neurological signs, blindness and mortality of up to 50% have been reported in infected poults, and mortalities may continue for three to four weeks. The most likely outcome of infection would be a single or a few isolated occurrences of infection.

If the disease were to establish in a chicken flock, the chances of early recognition and eradication are low, although slightly higher in turkeys. *S.* Arizonae may be transmitted in contaminated feed and water, and may also be spread in the incubator and brooder by direct contact. Breeding flocks were considered much less likely to become infected due to the higher levels of biosecurity involved in their management, (to the extent that breeding birds are not commercial flocks as defined in the Methods), so spread via contact in incubators should be of relatively little concern. In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. Outbreaks of arizonosis in chickens are not known to have spread widely. The IRA team therefore assigned the following likelihoods to the four outbreak scenarios (Table 69).

Table 69. Estimated partial likelihood of establishment and spread (PLES) values for *S.* Arizonae in medium biosecurity commercial poultry

Outbreak scenario 1:	$PLES_{(cp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(cp)}2$	**Very low**
Outbreak scenario 3:	$PLES_{(cp)}3$	**Low**
Outbreak scenario 4:	$PLES_{(cp)}4$	**Very low**

Non-Avian Species

Mammals and reptiles are not commonly infected with serotypes responsible for outbreaks of arizonosis in poultry. In addition, infection of non-avian species with *S.* Arizonae, with subsequent spread to poultry, is unlikely to occur. Therefore, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 70).

Table 70. Estimated partial likelihood of establishment and spread (PLES) values for *S.* Arizonae in non-avian species

Outbreak scenario 1:	$PLES_{(nas)}1$	**High**
Outbreak scenario 2:	$PLES_{(nas)}2$	**Extremely low**
Outbreak scenario 3:	$PLES_{(nas)}3$	**Extremely low**
Outbreak scenario 4:	$PLES_{(nas)}4$	**Extremely low**

Conclusion – Likelihood assessments

The estimates for the likelihood of release, the partial likelihoods of exposure for each of the exposure groups, and the partial likelihood of establishment and spread for each of the outbreaks scenarios were combined with the expected volume of trade using the simulation model. This allowed the calculation of partial annual likelihood of entry, exposure, establishment and spread for each of the outbreak scenarios. The results of this calculation are shown in Table 71.

Table 71. Partial annual likelihood of entry, exposure, establishment and spread (PALEEES) for the outbreak scenarios

Exposure group	Outbreak scenario	PALEEES
Wild birds	Scenario 1	High
	Scenario 2	Very low
Low biosecurity poultry	Scenario 1	High
	Scenario 2	High
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	High
	Scenario 2	Extremely low
Total all exposure groups	Scenario 3	High
Total all exposure groups	Scenario 4	High

Summary: Impact assessment

If *S*. Arizonae was introduced to Australia in chicken meat, the most significant impacts of infection would be on the health and production of domestic turkeys. The impacts of infection in wild birds (exposure group 1), low biosecurity poultry (exposure group 3) and medium biosecurity commercial poultry (exposure group 4) would only be discernible if secondary spread to a local or more general population of commercial or backyard turkeys occurred. The IRA team considered that the impacts at the national, State/Territory and district/region levels were ***unlikely to be discernible***. Impacts at the local level were assessed as being no greater than ***minor***. Therefore, the overall impacts for each outbreak scenario were assessed as ***negligible.***

Conclusions – impact assessment

As discussed above, all outbreak scenarios were assessed as having negligible impact. Results are shown in Table 72.

Partial annual risk estimate for each outbreak scenario

The partial annual likelihood of entry, exposure, establishment and spread were combined with the impacts of the relevant outbreak scenarios to provide an estimate of the partial annual risk associated with each outbreak scenario. The outcomes of this process are shown in Table 73.

Table 72. Impacts of each outbreak scenario

Exposure group	Outbreak scenario	Impact
Wild birds	Scenario 1	Negligible
	Scenario 2	Negligible
Low biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Negligible
	Scenario 2	Negligible
	Total Scenario 3	Negligible
	Total Scenario 4	Negligible

Table 73. Partial annual risk (PAR) of each outbreak scenario

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	High	Negligible	Negligible
	Scenario 2	Very low	Negligible	Negligible
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Medium biosecurity poultry	Scenario 1	Negligible	Negligible	Negligible
	Scenario 2	Negligible	Negligible	Negligible
Non-avian species	Scenario 1	Moderate	Negligible	Negligible
	Scenario 2	E. Low	Negligible	Negligible
	Total Scenario 3	High	Negligible	Negligible
	Total Scenario 4	High	Negligible	Negligible

Unrestricted risk estimate

The overall risk associated with the import of whole chicken carcasses for *S.* Arizonae was assessed as ***negligible***. As the unrestricted risk estimate meets Australia's ALOP, no risk management was considered necessary.

Direct Impact on human Life or Health

S. Arizonae has been demonstrated in chicken meat samples overseas and can lead to food-borne illness in humans.

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Technical Information

Background

Infectious coryza is an acute to subacute respiratory disease of chickens caused by *Haemophilus paragallinarum*. Clinical signs are generally mild, and the main impact of the disease is a reduction in egg production in laying flocks and poor performance in growing chickens (Blackall and Yamamoto 1998). Infectious coryza occurs worldwide, but the distribution of serovars varies from country to country (Sandoval, Terzolo, and Blackall 1994; Poernomo et al. 2000; Bragg, Coetzee, and Verschoor 1996). Page serovar B has not been isolated in Australia, but strains of serovars A and C are endemic (Blackall et al. 1990) and controlled through the use of antibiotics and commercial vaccines. If serovar B was to enter Australia, new vaccination strategies would be required (Zhang et al. 2003). Infectious coryza is of no public health significance.

Infectious coryza due to *H. paragallinarum* infection is not an OIE-listed disease.

Agent taxonomy

H. paragallinarum is a fastidious, slow growing, Gram-negative, non-motile bacterium. Two different but complementary schemes, the Page and the Kume scheme, are used to serotype strains of *H. paragallinarum*. The most widely recognised and applied is the Page scheme that recognises three serovars, A, B and C. The Kume serogroups correspond to the Page serovars, and represent three distinct immunovars. There is no significant vaccine cross-protection between the Kume serogroups, and variable cross-protection within serogroups (Soriano et al. 2004).Variant or unusual serovars have been reported recently in Argentina and Brazil, and may have implications for use of vaccines. DNA fingerprinting by restriction endonuclease analysis has also been used to classify strains (Blackall and Matsumoto 2003).

Agent characteristics

H. paragallinarum is a fragile organism and is unlikely to persist long outside the normal niche for the organism, the upper respiratory tract of the chicken (Blackall 2002). At 45 to 55 °C, *H. paragallinarum* is killed in two to ten minutes. Exudate or tissue kept at 37 °C remains infective for 24 to 48 hours, and for several days if kept at 4 °C. At 22 °C, exudate in saline is viable for at least 24 hours. In embryonic fluid, the organism is inactivated by 0.25% formalin within 24 hours at 6 °C, but survives several days in 1:10000 thiomersal (Blackall and Matsumoto 2003).

Epidemiology

Infectious coryza occurs worldwide, but the distribution of serovars varies from country to country. Page serovars A and C are present in many countries, including Australia (Blackall et al. 1990). Kume serovar A-4 and Kume serovar C-4 have only been reported in Australia, and in fact Kume serovar A-4 is the only form of Kume serogroup A present in Australia (Soriano et al. 2004). Serovar B has not been recognised in Australia, but is present in Argentina (Sandoval, Terzolo, and Blackall 1994), Brazil (Blackall et al. 1994), Germany (Blackall and

Matsumoto 2003), Indonesia (Poernomo et al. 2000), Mexico (Soriano et al. 2001), the Philippines (Nagaoka et al. 1994), South Africa (Bragg, Coetzee, and Verschoor 1996), Spain (Blackall and Matsumoto 2003), the United States (Page 1962) and China (Zhang et al. 2003; Page, Rosenwald, and Price 1963). In South Africa, there is evidence of a shift in the prevalence of serovars and infectious coryza has become an important and widespread disease despite the extensive use of commercial vaccines (Bragg, Coetzee, and Verschoor 1996).

Chickens are the natural hosts of *H. paragallinarum*. Birds of all ages are susceptible, but disease is generally less severe in juvenile birds. In mature birds the incubation period is shortened and the course of the disease longer. Turkeys, pigeons, sparrows, ducks, crows, rabbits, guinea pigs and mice are reported to be refractory to infection (Blackall and Matsumoto 2003).

Chronically infected or healthy carrier birds are the main reservoir of infection. Transmission may occur by droplet inhalation, contact, ingestion and via drinking water. *H. paragallinarum* is not egg transmitted (either transovarially or by contamination of the shell).

Clinical signs

Serous to mucoid nasal discharge, facial oedema, sinusitis, conjunctivitis, anorexia and diarrhoea are the most commonly reported clinical signs in infected chickens. Decreased feed and water consumption leads to decreased egg production in layers, and growth retardation in growing birds, resulting in an increased culling rate. Swollen wattles, rales and lower respiratory tract infections have also been reported. In the absence of concurrent infection, the duration of the disease may be two to three weeks. The morbidity and mortality vary with the virulence of organism, breed and age of bird, and other complicating factors such as housing, parasitism, nutrition and presence of other diseases (Blackall and Matsumoto 2003).

Infectious coryza is sometimes complicated by infections with other pathogens such as *Mycoplasma gallisepticum*, *M. synoviae*, *Pasteurella* spp., *Salmonella* spp., and infectious bronchitis virus, and has been associated with arthritis in meat chicken flocks and septicaemia in layer flocks (Sandoval, Terzolo, and Blackall 1994).

Pathogenesis

Birds may be infected experimentally by intranasal or intrasinus inoculation of infectious exudate or culture, as well as by contact, through the drinking water and by aerosol. Signs develop rapidly, within 24 to 48 hours after experimental inoculation, or 24 to 72 hours after contact (Rimler 1979).

Pathology

Lesions in uncomplicated infections are generally confined to the upper respiratory tract where catarrhal inflammation of the mucous membranes of the nasal passages and sinuses has been described. Pneumonia and airsacculitis are sometimes present. In meat chickens up to 70% condemnation due to airsacculitis has been reported (Blackall and Matsumoto 2003).

Immunology

It is widely accepted that Page serovars represent distinct immunovars since inactivated vaccines based on any one serovar provide no protection against the other two. A range of

serological tests for the detection of antibodies to *H. paragallinarum* have been described, but only haemagglutination-inhibition tests are in widespread use (Blackall and Yamamoto 1998).

Inactivated vaccines based on killed *H. paragallinarum* are commercially available, but are only effective against the serovar included in the vaccine. Cross-protection within Kume serogroups is incomplete, and if Kume serovar A-2 were to enter the Australian poultry flock, there may be lowered vaccine efficacy in the field, because all Australian-based infectious coryza vaccines are based on Kume serovar A-4 (Soriano et al. 2004). Antigenic variation has been reported within Page serovar B and only partial cross-protection occurs within various strains of Page serovar B. Strains of serovar B isolated in Argentina are reported to be genetically distinct from all other strains, regardless of serovar. Therefore, control of serovar B by vaccination is reported to be more complex than for the other two serovars (Blackall and Matsumoto 2003).

Diagnosis

The clinical signs and lesions of infectious coryza are not pathognomonic. Diagnosis is based on the history of rapidly spreading, acute coryza in the flock, and the isolation of catalase negative, Gram-negative organisms that exhibit a specific growth pattern (satellitic). For isolation of *H. paragallinarum*, swabs should be taken from the sinus, airsac and trachea and grown on blood or chocolate agar at 37 °C in 5% carbon dioxide, anaerobically or under reduced oxygen tension. *H. paragallinarum* may also be propagated in five- to seven-day old chicken embryos by yolk sac inoculation, or by inoculation of the sample into the infraorbital sinuses of susceptible chickens (Blackall and Yamamoto 1998). However, *H. paragallinarum* is a slow-growing, fastidious organism and is frequently overgrown by other faster growing commensal organisms (Blackall 1999).

A PCR assay has been developed and can be applied to suspect colonies or directly to samples from the sinus of chickens as a molecular diagnostic test (Blackall and Yamamoto 1998).

Transmission in chicken meat

H. paragallinarum is primarily a pathogen of respiratory tissues, which are generally removed from all chicken carcasses at slaughter. Despite the removal of these tissues, remnants may remain, so carcass contamination is considered a likely outcome. However, because of the fragility of the organism, the IRA team considered that any contamination would be unlikely to survive on the carcass during slaughter, processing, storage, and transport, and therefore that the likelihood of introduction of *H. paragallinarum* in imported chicken meat is negligible.

Quarantine significance

H. paragallinarum is not an OIE-listed disease agent.

Some strains of *H. paragallinarum* are endemic in Australian poultry. *H. paragallinarum* infection is not notifiable in any State or Territory in Australia, and is not subject to official controls. *H. paragallinarum* is not included in the Emergency Animal Disease Response Agreement.

Because the organism is primarily a pathogen of respiratory tissues which are removed at slaughter, and the fragility of the organism means that it is unlikely to survive for long in or on

contaminated carcases, and because of the low quarantine significance, no further risk assessment was considered necessary for *H. paragallinarum*.

Direct impact on human life or health

H. paragallinarum is not known to affect humans and is not considered to be a threat to public health.

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Avian Adenoviruses

Avian adenoviruses are non-enveloped, double-stranded DNA viruses, varying in size from 74-80nm (McNulty and Smyth 2001). There are four genera in the family *Adenoviridae*: *Mastadenovirus, Aviadenovirus, Atadenovirus* and *Siadenovirus* (Buchen-Osmond 2002).

Recently reclassified, avian adenoviruses were traditionally categorised into three groups. Group 1 consisted of 12 serotypes, including quail bronchitis virus (Fowl adenovirus 1 or FAdV-1), hydropericardium syndrome (FAdV-4), and inclusion body hepatitis virus (FAdV-8 and other serotypes). The main differences between hydropericardium syndrome and inclusion body hepatitis are that the mortality rate and incidence of hydropericardium are higher in the former disease (McFerran and Adair 2003). Inclusion body hepatitis is endemic in Australia, but hydropericardium syndrome is not.

Group 2 adenoviruses included marble spleen disease (MSD) virus of pheasants, haemorrhagic enteritis virus (HEV) of turkeys, and avian adenovirus group 2 splenomegaly (AAS). Group 3 avian adenoviruses included the egg drop syndrome (EDS) virus. Since reclassification, Group 1 viruses have remained in the genus *Aviadenovirus*, Group 2 viruses have been reclassified into the genus *Siadenovirus*, and EDS has been reclassified as an *Atadenovirus* (Buchen-Osmond 2002). As many standard texts still refer to the previously existing classification, it will be retained for use in this document.

Group 1 adenoviruses can establish latent infections, and affected birds may become life-long carriers. Transmission is vertical, through transovarial transmission, and horizontal, via faecal shedding and to a lesser extent, respiratory secretions (McFerran and Smyth 2000). EDS virus multiplies in the reproductive tract and is vertically transmitted, but there is no evidence that Group 2 avian adenoviruses can be transmitted vertically (McFerran and Smyth 2000).

Group 1 Fowl Adenovirus, Serotype 1

Technical Information

Background

Fowl Adenovirus, serotype 1 (FAdV-1), also known as Quail Bronchitis Virus, is a highly contagious respiratory viral pathogen of quail. The virus causes high morbidity and mortality in young bobwhite quail (*Colinus virginianus*), various clinical signs in Japanese quail (*Coturnix coturnix japonica*) but few or no clinical signs in chickens, turkeys and other game birds. Although the virus is of no economic importance to the chicken industry, chickens may serve as vectors for FAdV-1. Introduction of this exotic disease to Australian quail could have adverse effects on the quail industry.

FAdV-1 infection is not an OIE-listed disease.

Agent taxonomy

Quail bronchitis is caused by a Group 1, serotype 1 adenovirus of the genus *Aviadenovirus*. Chicken embryo lethal orphan virus (CELO) and FAdV-1 are indistinguishable by conventional techniques (Reed and Jack 2003). Both FAdV-1 and CELO virus antiserum were capable of neutralising another group 1, serotype 1 adenovirus (Indiana C) that was isolated from chickens experiencing a problem in eggshell quality (Winterfield, Fadly, and Gallina 1973). The relationship between these three isolates is not clear from current literature.

Agent characteristics

Avian adenoviruses, in general, exhibit resistance to lipid solvents such as ether and chloroform, and 50% alcohol, but are inactivated by 1:1000 concentration of formaldehyde. They are resistant to variations in pH between 3 and 9 (McFerran and Adair 2003). Avian adenoviruses are more heat resistant than adenoviruses in general but few details are available on the specific characteristics of FAdV-1. FAdV-1 survived heat treatment at 56 ºC for 90 minutes, in a trial to differentiate FAdV-1 from infectious bronchitis virus using heat stability as the criterion (Du Bose, Grumbles, and Flowers 1960). In wet faeces at 20 ºC, the titre of CELO virus decreased by only 1 $\log_{10}$ after 60 days; infectivity was reduced more rapidly in dry faeces (Monreal 1992).

FAdV-1 has been successfully stored for four years at –20 ºC and –50 ºC (Du Bose 1971).

Epidemiology

Quail species, including bobwhite and Japanese quail, are the main host species that develop disease from infection with FAdV-1. Chickens and turkeys can be experimentally infected but develop few or only mild clinical signs (Olson 1950). Naturally-occurring infections in chickens are suggested by serological conversion (Du Bose 1971). Natural infection has not been reported in non-avian species; however, experimental inoculation of hamsters may be associated with tumour development at the site of injection (Monreal 1992).

Avian adenoviruses are distributed throughout the world. FAdV-1 has been confirmed in the United States (Reed and Jack 2003), Singapore (Chew-Lim 1980), Italy and India (Chandra and Kumar 1998).

Vertical transmission is an important route of transmission of avian adenoviruses in general, but there is no information available on whether FAdV-1 is transmitted vertically in quails or chickens (McFerran and Smyth 2000). Horizontal transmission probably occurs via aerosol, by mechanical means, and possibly via the faecal-oral route (Reed and Jack 2003). Faecal-oral and mechanical transmission have been documented for other Group 1 avian adenoviruses (McFerran 1997).

FAdV-1 results in clinical disease in bobwhite quail less than six weeks of age, with most severe outbreaks occurring when the birds are aged one to three weeks (McFerran and Smyth 2000; Reed and Jack 2003). Disease in Japanese quail, however, has been reported in older (five-month-old) laying birds, with a high morbidity and low mortality (Chew-Lim 1980). Antibodies to FAdV-1 have been detected in wild bobwhite quail, and the virus was isolated from the caecum of two bobwhite quail aged seven to nine months old (King, Pursglove, and Davidson 1981).

An incubation period of between two and seven days has been demonstrated experimentally (Du Bose and Grumbles 1959; Jack and Reed 1989). Morbidity rates generally reach 100% in quail less than three weeks old. Mortality rates in bobwhite quail under four weeks of age have reached 100% but typically range from 10–80% (Du Bose 1971; Jack and Reed 1989). In older birds, mortality rates are usually less than 10%, with many birds remaining asymptomatic. A seroprevalence of 23% was reported in wild bobwhite quail (King, Pursglove, and Davidson 1981). The importance of quail bronchitis in wild quail, and the susceptibility of Australian native quail are unknown.

The prevalence of FAdV-1 infection in commercial meat chickens is not known; however, naturally-occurring infection in slaughter-age meat chickens has not been reported.

Clinical Signs

Affected bobwhite quail develop sudden onset, rapid spread of respiratory distress and high mortality. Clinical signs include rales, coughing, depression, neurological signs and sometimes nasal-ocular discharge (McFerran and Adair 1977; Reed and Jack 2003). Adult *Coturnix* quail showed unspecified respiratory and neurological signs and a 10–15% drop in egg production, with the appearance of soft-shelled and unpigmented eggs. Mortality was low (less than 1%) and the quail recovered within four weeks, with egg production returning to normal by eight weeks (Chew-Lim 1980).

Experimental infection of five-day-old chickens resulted in mild respiratory disease characterised by mild rales and slight accumulation of mucus in the trachea in one bird (Olson 1950). Indiana C virus was isolated from mature White Leghorn chickens in a flock exhibiting alterations in eggshell quality (Winterfield, Fadly, and Gallina 1973).

Pathogenesis

After experimental tracheal inoculation of bobwhite quail, the virus was isolated from lung tissue as early as two hours post-inoculation, from caecal tonsil and bursa of Fabricius at four hours, and from the spleen and liver at 8 hours (Jack, Reed, and Burnstein 1994). Virus could be isolated from lung, spleen, caecal tonsil and bursa at titres of 10^5-10^7 EID_{50}/g of tissue for

eight days post-inoculation. This suggests an early viraemia that results in infection of various organs.

The virus has been isolated from the aqueous humour and trachea of quail 48 days after they were placed with inoculated cage-mates (Du Bose and Grumbles 1959).

The pathogenesis of FAdV-1 infection in chickens has not been reported in detail.

Pathology

Mucoid tracheitis, sinusitis, airsacculitis and severe pulmonary congestion have been reported in birds succumbing to quail bronchitis. Pin-point necrotic foci may be present in the liver, and, histologically there may be lymphoid necrosis in the bursa and inclusion bodies in tracheal or bronchial epithelium (Jack and Reed 1990).

Japanese quail affected at five months of age with FAdV-1 showed post-mortem evidence of spleen enlargement, congested lungs and tracheas, with bile stasis and egg peritonitis in some cases (Chew-Lim 1980).

Chickens experimentally infected with a related Group 1, serotype 1 adenovirus, designated Indiana-C virus, showed respiratory clinical signs, and post-mortem evidence of hepatitis, with virus being isolated from trachea, liver, spleen, kidney and bursa (Winterfield, Fadly, and Gallina 1973).

Immunology

Young quail chicks may be protected from infection by maternal antibody. Survivors of natural and experimental infection remain refractory to infection for at least six months (Reed and Jack 2003). Indiana C virus had been used to vaccinate quails during quail bronchitis outbreaks; however, more recent studies showed that Indiana C virus was pathogenic for quail, resulting in clinical signs and post-mortem lesions similar to those caused by FAdV-1 (Jack and Reed 1994). There is currently no commercially available vaccine against FAdV-1.

Diagnosis

A presumptive diagnosis of quail bronchitis may be made in young quail exhibiting a sudden onset of respiratory disease accompanied by a high mortality. Older birds may show less severe signs and lower mortality, and there may be neurological signs and a drop in egg production in commercial quail layers. Isolation of FAdV-1 in embryonating chicken eggs is necessary for diagnosis (Du Bose 1971; Reed and Jack 2003). Neutralisation of the virus by specific FAdV-1 or CELO virus antiserum confirms identification of the virus (Reed and Jack 1997).

Transmission in chicken meat

CELO virus has been reisolated from the blood, muscle, lungs, liver, spleen, intestines and aqueous humour of inoculated chickens (Du Bose 1971). Indiana C virus has been isolated from the tracheas, livers, spleens, kidneys and bursae of chickens 10 days after experimental inoculation, indicating a systemic infection (Winterfield, Fadly, and Gallina 1973). However, naturally-occurring infection associated with FAdV-1 has not been documented in slaughter-age commercial meat chickens, although there is a report of infection in mature layers (Winterfield, Fadly, and Gallina 1973). On balance, the IRA team considered that it was unlikely that FadV-1 would be present in the meat of commercial broiler birds.

Quarantine significance

FAdV-1 infection is not an OIE-listed disease. It is not notifiable in any State or Territory in Australia, and is not subject to official controls. FAdV-1 is not included in the Emergency Animal Disease Response Agreement. The agent causes disease only in quail. While it is acknowledged that introduction of FAdV-1 could have adverse effects on the Australian quail industry, the IRA team considered that the overall impacts of this disease would be minor.

Experimental infection of chickens with FAdV-1 has been demonstrated, and seroconversion has been demonstrated in some chicken flocks, but naturally occurring infection in slaughter age chickens has not been reported. Therefore, the IRA team considered that the likelihood of release of FAdV-1 into Australia as a result of the importation of chicken meat was negligible. After considering the negligible likelihood of introduction of this virus in chicken meat, and the minor economic impact that would result, the IRA team considered that no further risk assessment for FAdV-1 was necessary.

Group 1 Fowl Adenovirus, Serotype 4

Technical Information

Background

Group 1 Fowl Adenovirus (FAdV), serotype 4 is the causative agent of the disease variously known as hydropericardium syndrome (HPS), Angara disease, hydropericardium-hepatitis syndrome, infectious hydropericardium and inclusion body hepatitis/hydropericardium syndrome. The disease was first reported in the Angara Goth region of Pakistan in 1988, and resulted in over 100 million deaths in meat chickens, with mortality rates up to 75% in individual flocks (Toro et al. 1999). Hydropericardium syndrome has since been reported in other countries in Asia, the Middle East, Russia, Central and South America (Ganesh and Raghavan 2000; McFerran and Smyth 2000), often in association with other viruses such as IBDV, Marek's Disease or Chicken Anaemia Virus. There are reports of inclusion body hepatitis in Australia, but these are caused by serotypes other than Serotype 4.

FAdV-4 infection is not an OIE-listed disease.

Agent taxonomy

The causative organism is a Group 1 *Fowl Adenovirus* (FAdV), serotype 4 (Ganesh and Raghavan 2000).

Agent characteristics

In general, avian adenoviruses are resistant to lipid solvents, more resistant than other adenoviruses to heat inactivation, and are resistant to variations in pH between 3 and 9. They are inactivated by 1:1000 concentrations of formaldehyde (McFerran and Adair 2003).

FAdV-4 in liver suspensions withstands heating at 50 ºC for 30 minutes. Heating of infected liver homogenate at 60 ºC for 1 hour, 80 ºC for 10 minutes and 100 ºC for 5 minutes appeared to inactivate the virus, as shown by loss of infectivity when inoculated into chickens (Afzal, Muneer, and Stein 1991). The high level of heating required to inactivate this virus is greater than would normally be expected to kill other adenoviruses. In the laboratory, virus infectivity is preserved during storage at –20 ºC (Anjum 1990). The sensitivity of different isolates of FAdV-4 to pH 3 to 10 is variable (Ganesh and Raghavan 2000). Little is known about the resistance of the virus to environmental conditions.

Epidemiology

FAdV-4 is associated with HPS primarily in chickens (Chandra, Shukla, and Kumar 2000), although the disease has also been reported in a flock of Japanese quail (*Coturnix coturnix japonica*) (Roy et al. 2004). A hydropericardium syndrome causing mortality in a flock of pigeons was also described in Pakistan in 1995 (Naeem and Akram 1995). Although no attempt was made to isolate a virus, liver homogenates from affected pigeons caused HPS when inoculated into chickens and the authors presumed that FAdV-4 was the cause (Naeem and Akram 1995).

Hydropericardium syndrome was first reported in Pakistan and subsequently has been diagnosed in Iraq, Kuwait, India, Russia, Japan, Mexico, Peru, Ecuador, Chile (Ganesh and Raghavan 2000) and Slovakia (Balamurugan and Kataria 2004; McFerran and Smyth 2000). The disease has not been reported in Australia, New Zealand, or the United States.

Transmission occurs by the vertical and horizontal routes. Vertical transmission was demonstrated in 35-week-old layer breeders, which transmitted the virus to their progeny as early as five days after experimental inoculation with FAdV-4. FAdV-4 was isolated from the livers of progeny chicks, some of which died from HPS-like disease and had typical post-mortem lesions of HPS, while others were killed and examined at 30 days of age (Toro et al. 2001). Virus may remain latent in progeny chicks until point of lay or can be shed under conditions of immunosuppression or stress (Shane 1996). Non-fertile eggs and hatchery waste can be infected (McFerran and Smyth 2000). Viral replication occurs in the intestine, and the faeces are a source of infection. Chicks may not excrete virus until three weeks of age because of the effect of maternal antibody, after which virus shedding may continue for up to 14 weeks. Horizontal transmission occurs by mechanical means, including faecally contaminated litter, clothing, and equipment. Chickens orally inoculated with liver suspensions from naturally-infected chickens developed HPS (Abdul-Aziz and Hasan 1995). In one experimental study, orally-inoculated chickens excreted the virus earlier than birds inoculated by intramuscular injection, resulting in early transmission of the disease to in-contact uninoculated chickens. Field observations are characterised by a sudden onset of disease, suggesting that natural infection may occur by the oral route with a very short incubation period (Abdul-Aziz and Hasan 1995). Aerosol transmission may occur but only over short distances, and transmission has occurred via vaccines prepared in infected eggs (Shane 1996). It is postulated that the initial massive spread of the virus in Pakistan may have been due to use of a contaminated vaccine (McFerran and Adair 2003).

The disease is most commonly reported in meat chickens between 3 and 6 weeks of age, and mortality is highest in this age group. However, disease has been reported in layers and in meat chicken- and layer-breeders, in birds aged from 10–32 weeks (Asrani et al. 1997; Ganesh et al. 2002). Variable incubation periods have been reported, with disease and death occurring within two to five days of experimental inoculation in some reports, and from 5–18 days in others (Ganesh and Raghavan 2000). Mortality rates up to 80% have been reported in three- to five-week-old chicks in naturally-occurring outbreaks in India and Pakistan, with lower mortality rates (5–8%) in older birds (Ganesh and Raghavan 2000) and in other countries (Kumar et al. 1997). Mortality rates were lower in outbreaks of HPS in Chile, varying between 7% and 25% (Toro et al. 1999). Mortality rates may be higher when the flock is affected by concurrent disease (Shane 2001; Toro et al. 1999). Mortality in an affected flock of five to six week old quail was 3–4% (Roy et al. 2004).

A between-flock prevalence of 46.6% was reported in 131 flocks examined in Pakistan (Akhtar, Zahid, and Khan 1992). The prevalence and severity of the disease is thought to be related to the density of the poultry production, the level of biosecurity in the region and the presence of concurrent immuno-suppressive disease such as infectious bursal disease, Marek's disease, and chicken infectious anaemia, and the presence of other endemic diseases, such as Newcastle disease, and avian influenza (Shane and Jaffery 1997). In an outbreak situation, the within-flock prevalence is high, with mortalities approaching 80% in some circumstances.

Clinical Signs

Clinical signs may be absent in acute outbreaks of disease, although depression, ruffled feathers, huddling and yellow mucoid faeces may precede death.

Pathogenesis

FAdV-4 has a predilection for lymphoid tissues, and infection leads to lymphoid depletion in the bursa of Fabricius, spleen and thymus with subsequent immunosuppression (Mazaheri et al. 1998; Ganesh and Raghavan 2000). The pathogenesis is often complicated by the presence of concurrent infection with other disease agents, with one of the most common associations being with Chicken Anaemia Virus (CAV) (Voss et al. 1996). In birds with dual HPS/CAV infections, profound anaemia is a common clinicopathological finding. However, anaemia and leucopaenia have been reported in birds infected with FAdV-4 alone (Shane and Jaffery 1997). Hypoalbuminaemia has been attributed to reduced albumin synthesis associated with the severe hepatitis, and may contribute to the development of pericardial effusion.

Pathology

The most prominent lesion at post-mortem examination is the presence of excessive watery or jelly-like fluid in the pericardial sac (hydropericardium). The liver and kidneys appear enlarged and pale, and in some reports, there is splenic enlargement and congestion and oedema of the lungs (Asrani et al. 1997). Bursal atrophy may or may not be present (Asrani et al. 1997), and haemorrhages may be present in the breast and other muscles (Toro et al. 1999).

Histopathology of the liver typically shows the presence of multifocal necrosis and intra-nuclear inclusion bodies (Ganesh and Raghavan 2000). The spleen, bursa, and thymus show lymphoid depletion, and oedema and inflammatory cell infiltrates can be demonstrated in the myocardium (Akhtar 1994; Asrani et al. 1997).

Immunology

The original formalin-inactivated vaccines prepared from liver homogenates of infected birds were found to reduce mortality rates in HPS-endemic areas (Kumar et al. 1997; Ganesh and Raghavan 2000). Oil-emulsified vaccines were later demonstrated to induce greater antibody titres than the formalinised vaccine (Chandra, Shukla, and Kumar 2000). More recently, vaccines have been developed using purified virus propagated in chicken liver cells or embryonated eggs (Chandra, Shukla, and Kumar 2000).

Vaccines in current use are manufactured and distributed on a local level, and there is considerable variability in the safety and efficacy of currently available vaccines (Ganesh and Raghavan 2000). Vaccinated birds undergo seroconversion by about day 7 post-vaccination, with seroconversion correlating well with protection against disease. Mortality due to HPS is reduced by vaccination, from 31% in unvaccinated flocks to 4% in vaccinated flocks (Shane 1996).

Birds infected with natural disease show reduced serological response to vaccination against Newcastle disease because of immunosuppression. Birds that recover from infection are immune to subsequent attacks of HPS (Chandra, Shukla, and Kumar 2000).

Diagnosis

Sudden death from the third week of age, and post-mortem findings such as hydropericardium and hepatitis, with intranuclear inclusion bodies are considered pathognomonic for HPS (Ganesh and Raghavan 2000). Diagnosis can be confirmed by virus isolation and serum neutralisation tests with inoculation of chick liver tissue cell culture considered more sensitive than the inoculation of embryonated eggs (Ganesh and Raghavan 2000). A PCR has recently been developed to assist in the detection of FAdV-4 in infected tissues (Ganesh, Suryanarayana, and Raghavan 2002).

Transmission in chicken meat

Virus has been isolated from the liver of vertically-infected chicks at 30 days of age (Toro et al. 2001), and in horizontally-infected chicks at 14 days post-inoculation (Mazaheri et al. 1998) at the end of experimental trials. Studies have not been done to determine if virus persists in the liver beyond the duration of these experimental trials. Faecal shedding of virus may persist for up to 14 weeks after infection (Shane 1996) and the infection can become latent, with resumption of shedding during periods of stress. McFerran and Smyth concluded that the risk of transmission of virus in poultry meat appears to be small, despite the fact that during viraemia, it is likely that virus is present in all tissues of the carcass and that potential exists for carcass contamination with infected faeces during processing (McFerran and Smyth 2000). This conclusion took into account the fact that flocks which are infected with significant adenoviruses would show signs of disease, and should therefore be rejected from slaughter for human consumption, and that viruses will not multiply in carcass meat, unlike bacteria. The IRA team considered the view of McFerran and Smyth, but also took into account the effect of vaccination on disease expression, and considered that infected flocks which had been vaccinated might escape detection, and therefore would not be rejected from slaughter.

Quarantine significance

FAdV-4 is not an OIE-listed disease agent. HPS is not notifiable in any State or Territory of Australia, and is not subject to official controls. HPS is not included in the Emergency Animal Disease Response Agreement. Given overseas experience of factors contributing to severe disease, and husbandry differences between Australia and overseas countries where severe disease has been reported, it is considered unlikely that this disease would appear in Australia in the form that was reported in Pakistan and elsewhere.

Therefore it is considered to be of relatively minor concern, and unlikely to have consequences for the poultry industry that are discernible beyond the district/region level. However, given that chickens are the natural hosts of FAdV-4, and that transmission in or on the carcass is possible, a detailed risk assessment was carried out.

Risk Assessment

Release Assessment

Rel_1: Selection of source flock (between flock prevalence)

For an unrestricted risk estimate, it was assumed that the prevalence is at the highest sustainable level in an endemically infected country or zone (see Method for Risk Assessment). Given that seroprevalence approaches 50% in some countries, the IRA team considered that the likelihood that a source flock will be infected with HPS at the time of slaughter was ***moderate***.

Rel_2: Infection detected through flock surveillance and the flock withdrawn from slaughter

In an outbreak of HPS, increased mortality in the flock would be obvious to the producer. However, clinical signs may be absent before the onset of mortalities, and the incubation period can be up to 18 days. The virus may cause minor losses in vaccinated flocks, and the likelihood that an infected flock will be detected through routine flock surveillance in an HPS endemic area, and the flock withheld from slaughter, was assessed by the IRA team as ***very low***.

Rel_3: Selection of an infected chicken from an infected flock (within-flock prevalence)

HPS is a highly contagious disease, and in an outbreak, multiple birds within a shed are likely to be affected. Given that birds would be sent to slaughter on a shed or flock basis, it is likely that a flock with an increased mortality rate would be withheld from slaughter, although in an area where disease was endemic, vaccination of flocks would reduce the likelihood of disease detection. However, the IRA team considered that if an infected flock were sent to slaughter, the likelihood that a selected individual chicken will be infected was ***high***.

Rel_4: Background cross-contamination rate

This likelihood represents the background cross-contamination rate during slaughter and processing and applies to cross-contamination with material, infectious or otherwise, from other carcasses, including those tissues or materials in which the disease agent tends to localise.

HPS is a systemic infection of slaughter-age birds, with virus present in multiple organs and in the faeces. The IRA team considered that the likelihood of a carcass being contaminated with potentially contaminated material from other birds, especially the digestive tract, is ***high***.

Rel_5: Likelihood that an uninfected carcass will be contaminated with the disease agent during slaughter and processing

As discussed in the Method for Risk Assessment (page 58, Part B), Rel_4 was used to calculate Rel_{5a} (the likelihood that an uninfected carcass from an infected flock will become contaminated during slaughter and processing) and Rel_{5b} (the likelihood that an uninfected carcass from an uninfected flock will become contaminated during slaughter and processing).

For FAdV-4, Rel_{5a} was calculated as ***moderate***, and Rel_{5b} was calculated as ***low***.

Rel_6: The likelihood that the carcass of a bird that was *infected* before slaughter will be removed as a result of inspections before or during processing

Carcasses with visible haemorrhages in skeletal muscle would, most likely, be detected during processing. Similarly, birds with enlarged livers and hydropericardium may be detected and removed. However, birds in the viraemic stages of infection may show no gross lesions that would prompt removal from the processing line. The IRA team considered that the rejection rate of infected carcasses would be greater than the background rejection rate but still very low. The likelihood that a contaminated/infected carcass will be removed during processing inspections was therefore assessed as ***very low***.

Rel_7: The likelihood that the carcass of a bird that was *not infected* before slaughter will be removed as a result of inspections before or during processing

The likelihood that an uncontaminated/uninfected carcass will be removed during processing inspections was considered to be equal to the background rejection rate of 0.75% as described in the Method section (page 59, Part B).

Rel_8: Inactivation of the agent during further processing, storage, handling and transport

Fowl adenovirus 4 can withstand freezing and the alterations in pH that occur in carcasses after processing. Given that the carcasses for importation will be chilled or frozen, the likelihood of inactivation of the virus during further processing, storage, handling and transport was assessed by the IRA team as ***extremely low***.

Conclusions – Release assessment

After inserting the above estimates into the simulation model, and using the outputs from the model as a guide, the IRA team concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a ***moderate*** likelihood that imported chicken meat would be infected or contaminated with FAdV-4.

Exposure assessment

Exposure Group 1: Wild birds

Of the steps identified as determinants in the infection of wild birds by exposure to scraps from an imported contaminated chicken carcass, $WB_{agentsurvival}$ and $WB_{infectivedose}$ are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$WB_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by a wild bird

Little is known about the resistance of FAdV-4 to environmental conditions. The virus can withstand heating within liver suspensions, and it was assumed that, protected within chicken meat scraps, it is likely to survive in the environment under ambient temperatures of 10 ºC to 35 ºC for several days. This likelihood was assessed by the IRA team as ***high***.

$WB_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a wild bird is sufficient to produce infection

HPS has not been reported to occur in birds known to frequent refuse dumps. Infection in a flock of pigeons has been reported, but the susceptibility of other species of birds has not been investigated. While the virus appears to cause disease almost exclusively in chickens, transmission of infection by wild birds has not been ruled out. Taking account of this information, the IRA team considered that there was a ***low*** likelihood that FAdV-4 would infect a wild bird consuming the contaminated meat scraps.

Exposure Group 2: Low biosecurity poultry

Of the steps identified as determinants in the infection of low biosecurity poultry by exposure to material from an imported contaminated chicken carcass, $BP_{agentsurvival}$, $BP_{infectivedose}$, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$BP_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by low biosecurity poultry

The time between feeding of scraps and consumption by low biosecurity poultry is likely to be very short, so environmental degradation of the disease agent will be minimal. The IRA team considered that this, together with the high temperature resistance of FAdV-4, means that the likelihood that the agent will remain viable is ***certain (=1)***.

$BP_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a low biosecurity bird is sufficient to produce infection

Transmission of HPS to chickens by feeding of infected meat has not been documented; however, the oral administration of infected liver homogenates to chickens can result in clinical disease (Abdul-Aziz and Hasan 1995). The titre of virus likely to be present in the muscle of an infected or contaminated carcass is unknown; titres will be higher in internal organs, such as liver, kidney heart and spleen. This risk assessment considers the importation of whole carcasses, including heart, gizzard and liver; therefore the assumption was made that these organs may be fed to low biosecurity poultry. Birds of all ages can be infected with this virus, with the mortality being highest in younger birds. Given that a chicken can consume up to 150g of feed per day, the IRA team considered that it was highly likely that a sufficient dose of virus would be available to initiate infection.

The overall likelihood that low biosecurity poultry would be infected with FAdV-4 as a result of consuming the contaminated chicken meat scraps was assessed by the IRA team as ***high***.

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

The likelihood that FAdV-4 would survive the rendering process was negligible. The IRA team considered that the likelihood the product would be re-contaminated with virus post-processing was negligible. Therefore, the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with FAdV-4 was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

Given that FAdV-4 would be destroyed by rendering as discussed above, and that feed derived from contaminated carcasses would be diluted with feed from non-risk material, the likelihood that the amount of final poultry ration eaten by a bird would contain an oral infectious dose of virus was considered by the IRA team to be ***negligible***.

Exposure Group 3: Medium biosecurity commercial poultry

Of the steps identified as determinants in the infection of medium biosecurity commercial poultry by exposure to material from an imported contaminated chicken carcass, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that poultry feed would be contaminated with FAdV-4 was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that the final poultry ration would contain an oral infectious dose of FAdV-4 was considered to be ***negligible***.

Exposure Group 4: Non-avian species

As discussed above, $NAS_{agentsurvival}$ was considered to be equal to $WB_{agentsurvival}$.

HPS has not been reported in non-avian species. Therefore this exposure group was not considered further in relation to this disease. $NAS_{infectivedose}$ was set to a value of zero.

Conclusions – Exposure assessment

Using the simulation model as a guide, the partial likelihood of exposure for each of the exposure groups was determined, taking the above estimates of the exposure variables into account. A summary of the outcomes determined by the IRA team is set out in Table 74.

Consequence assessment

Establishment and spread was considered in the context of the outbreak scenarios, described in the Method of Risk Assessment.

Table 74. Partial likelihoods of exposure (PLE)

Exposure group	PLE
Wild birds	Extremely low
Low biosecurity poultry	Very low
Medium biosecurity poultry	Negligible
Non-avian species	Zero

Estimating the likelihood of each outbreak scenario

The partial likelihood of establishment and spread (PLES) of HPS for the different exposure groups is described below. Four outbreak scenarios were considered relevant:

1. Disease agent does not establish or is not recognised within the directly exposed population
2. Disease agent establishes within the directly exposed population, is identified and is eliminated by human efforts or by natural means
3. Disease agent establishes in the directly exposed population, spreads within a district/region, including into other exposure groups if applicable, and is eliminated by human action or by natural means
4. Disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia.

Wild birds

Outbreaks of HPS in wild birds have not been reported, although infection has been reported in a single flock of domestic pigeons with an increased but unspecified mortality rate. The IRA team considered that the most likely outcome of infection of a wild bird resulting from scavenging the imported contaminated chicken meat scraps would be a single or a few isolated occurrences of infection in wild birds, with the virus being unable to establish ongoing infection in the population. Infection of wild birds with FAdV-4, with subsequent spread to poultry, has not been reported, so it must be considered an unlikely event. In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 75).

Table 75. Estimated partial likelihood of establishment and spread (PLES) values for FAdV-4 in wild birds

Outbreak scenario 1:	$PLES_{(wb)}1$	**High**
Outbreak scenario 2:	$PLES_{(wb)}2$	**Very low**
Outbreak scenario 3:	$PLES_{(wb)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(wb)}4$	**Very low**

Low biosecurity poultry

FAdV-4 is associated with HPS primarily in chickens (Chandra, Shukla, and Kumar 2000). Although this exposure group includes commercial free-range poultry and ratites, the IRA team considered that the sub-set of this group most likely to be exposed to scraps of imported chicken meat was small flocks of backyard poultry, because these are more likely to be directly fed table scraps. In such flocks, there is less opportunity for the generation of high levels of environmental contamination than might occur with an outbreak of infectious disease in a large commercial flock. The most likely outcome of infection would be a single or a few isolated occurrences of infection.

If the disease were to establish in a low biosecurity backyard flock, the level of expertise in disease recognition is likely to be low. Mechanical transmission of the virus by contaminated persons or fomites, and transmission by movement of birds may facilitate spread of the virus beyond the initially infected backyard flock before it is recognised; however, such spread is likely to be local, and spread to low or medium biosecurity commercial poultry will not necessarily follow. If the infection does spread to low or medium biosecurity commercial poultry, it is more likely to be diagnosed. However, there may be some delay in distinguishing the infection from endemic inclusion body hepatitis. HPS is not a notifiable disease in Australia, and it is likely that no official control action will be taken to eradicate the disease. In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 76).

Table 76. Estimated partial likelihood of establishment and spread (PLES) values for FAdV-4 in low biosecurity poultry

Outbreak scenario 1:	$PLES_{(bp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(bp)}2$	**Low**
Outbreak scenario 3:	$PLES_{(bp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(bp)}4$	**Low**

Medium biosecurity commercial poultry

The IRA team considered that the most feasible route for exposure of medium biosecurity commercial poultry to imported carcass components would be through poultry feed containing inadequately rendered processing waste. The likelihood that FADV-4 would remain viable following rendering of contaminated imported carcasses and parts was assessed as negligible (page 232). Nevertheless, the IRA team estimated the PLES values, based on their assessment of the likely outcomes in the improbable event that exposure of medium biosecurity commercial poultry did occur via this route.

Because of its highly infectious nature, spread within an exposed flock will be rapid. However, difficulty in differentiation between HPS and the endemic inclusion body hepatitis may lead to delays in recognising an outbreak of exotic disease, especially because the combination of husbandry factors that contribute to the severity of the disease in other countries does not occur commonly in Australian commercial flocks. This, combined with the fact that the disease is not notifiable, nor subject to the Emergency Animal Disease Response Agreement, led the IRA

team to conclude that early recognition and eradication of HPS is unlikely. In view of these factors, outbreak scenario 4 (disease becomes endemic) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 77).

Table 77. Estimated partial likelihood of establishment and spread (PLES) values for FAdV-4 in medium biosecurity commercial poultry

Outbreak scenario 1:	$PLES_{(cp)}1$	**Very low**
Outbreak scenario 2:	$PLES_{(cp)}2$	**Low**
Outbreak scenario 3:	$PLES_{(cp)}3$	**Low**
Outbreak scenario 4:	$PLES_{(cp)}4$	**Moderate**

Non-Avian Species

As discussed above, this exposure group was not considered further in relation to this disease.

Conclusion – Likelihood assessments

The estimates for the likelihood of release, the partial likelihoods of exposure for each of the exposure groups, and the partial likelihood of establishment and spread for each of the outbreaks scenarios were combined with the expected volume of trade using the simulation model. This allowed the calculation of partial annual likelihood of entry, exposure, establishment and spread for each of the outbreak scenarios. The results of this calculation are shown in Table 78.

Table 78. Partial annual likelihood of entry, exposure, establishment and spread (PALEEES) for the outbreak scenarios

Exposure group	Outbreak scenario	PALEEES
Wild birds	Scenario 1	High
	Scenario 2	High
Low biosecurity poultry	Scenario 1	High
	Scenario 2	High
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
Total all exposure groups	Scenario 3	High
Total all exposure groups	Scenario 4	High

Estimating the impacts associated with each outbreak scenario

For each outbreak scenario the direct and indirect impacts of HPS were estimated at the national, State or Territory, regional and local levels as described in the Methods section (page 90-95, Part B).

The likelihood of FAdV-4 affecting non-avian species (exposure group 4) was considered to be remote. Therefore, the impacts of HPS occurring in this exposure group were not considered further.

Outbreak Scenario 1

By definition, outbreak scenario 1 means that the disease agent does not establish in the initially exposed population, or does not cause sufficient disease to lead to investigation and recognition of the infection. Therefore the impacts will be ***unlikely to be discernible*** for all exposure groups and all criteria.

Outbreak Scenario 2

The impacts of this outbreak scenario (disease agent establishes within the directly exposed population, and is identified and eliminated) will differ among exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

HPS has not been reported to occur in birds known to frequent refuse dumps. Since wild birds do not play a significant part in production, direct economic loss from death of wild birds, in the unlikely event that it were to occur, is not measurable. Other impacts from the death of wild birds will be considered under direct criterion 2 and indirect criterion 4. The direct impacts of disease on wild birds were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

An outbreak of HPS contained within the low biosecurity poultry population will result in losses to individual owners. Impacts of such losses on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Loss of birds and production may occur, especially in meat chicken flocks. Impacts in medium biosecurity commercial poultry flocks were assessed as by the IRA team ***unlikely to be discernible*** at all levels.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

Impacts of an outbreak of disease in any exposure group on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

Because the disease is not likely to be recognised in wild birds, and is not notifiable in Australia, the impacts of an outbreak of HPS in wild birds on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels. The impacts of a recognised outbreak of HPS in low biosecurity poultry, were assessed as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level with regard to destruction of affected flocks and increased surveillance and monitoring of the poultry population, at least until the disease was diagnosed and other more serious diseases were ruled out.

While no official action will be taken to eradicate the disease, individual growers will need to take some control action. It was considered likely that the affected flock would be destroyed and surveillance and monitoring of the surrounding poultry population increased. The impacts of a recognised outbreak of HPS in a medium biosecurity commercial poultry flock were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

The impacts of an outbreak of HPS in wild birds on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak of HPS in low biosecurity poultry were assessed as ***unlikely to be discernible*** at all levels. Production losses may occur in commercial poultry flocks. A local outbreak of disease may result in affected flocks being withheld from slaughter for human consumption. However, it is expected that the impacts in Australia will not be as great as in countries where multifactorial husbandry failures lead to severe disease. Impacts of an outbreak of HPS in medium biosecurity commercial poultry on domestic trade and industry were assessed as ***unlikely to be discernible*** at all levels.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

HPS is not an OIE-listed disease; it is therefore difficult to predict the impact of a localised outbreak on international trade. Impacts for all exposure groups were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of an outbreak of HPS in all exposure groups were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impacts of an outbreak of HPS in wild birds were assessed as ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak of HPS in low biosecurity poultry were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Temporary restrictions imposed on movement of birds, eggs, poultry products and people until the diagnosis of HPS may lead to community disruption. The impacts of a disease outbreak in the medium biosecurity commercial poultry on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Outbreak Scenario 3

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a district/region, and is eliminated) will be the same for all exposure groups, no matter which exposure group has been directly exposed to FAdV-4 in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

HPS is likely to cause significant mortality in a naïve flock, and large numbers of birds may be involved on commercial properties. The direct costs of dead birds will be larger for commercial enterprises than for low biosecurity or aviary birds, but with prompt diagnosis and eradication, the costs will be limited to the local area. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory, and district/region levels, and ***minor*** at the local level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

Impacts were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

Spread of HPS to a local population of poultry or caged birds would lead to isolation of affected farms, and most likely eradication, surveillance and monitoring programs. The disease is not subject to the Emergency Animal Disease Response Agreement between Commonwealth and State governments and industry, however, as an exotic disease with high mortality, attempts would almost certainly be made to eradicate the disease. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, and ***minor*** at the local level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Impacts of movement restrictions associated with control and eradication programs on domestic trade and local industry were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

HPS is not an OIE-listed disease; it is therefore unlikely to cause serious on-going impacts on international trade. Impacts at all levels were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impact of an outbreak of HPS was assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Temporary restrictions may be imposed on movement of birds, eggs, poultry products and people until the diagnosis is confirmed. However, these restrictions were considered by the IRA team to be of short duration, and the impacts were assessed as ***unlikely to be discernible*** at all levels.

Outbreak Scenario 4

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a State/Territory, and becomes endemic in Australia) will be the same for all exposure groups, no matter which exposure group has been directly exposed to FAdV-4 in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

If the disease spreads more widely through medium biosecurity commercial poultry and low biosecurity poultry, there will be significant losses of birds and production. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, and ***minor*** at the local level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

Impacts were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

The IRA team considered that if HPS spreads to a more general population of poultry, efforts will be made, at least at the individual company level, to eradicate the disease, and internal monitoring and surveillance programs will be implemented. The impact at the national, State/Territory and district/region levels, were assessed by the IRA team as ***unlikely to be discernible***. There may be ***minor*** impacts at the local level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

If HPS spreads to a more general population of poultry, impacts on domestic trade and industry at the national level were assessed by the IRA team as ***unlikely to be discernible***. There may be ***minor*** impacts at the local level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

HPS is not an OIE-listed disease and the IRA team therefore considered that the impact of an outbreak on international trade would be ***unlikely to be discernible***.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The IRA team considered that a generalised outbreak of HPS would have ***no discernible impacts*** on this criterion at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impacts of a general outbreak of HPS on communities were assessed by the IRA team as ***unlikely to be discernible*** at the national, State/Territory or district/region levels, but ***minor*** at the local level.

Conclusions – impact assessment

The above estimates for each of the impact criteria were entered into the simulation model, and the overall impact of each outbreak scenario was estimated. Results are shown in Table 79.

Table 79. Impacts of each outbreak scenario

Exposure group	Outbreak scenario	Impact
Wild birds	Scenario 1	Negligible
	Scenario 2	Negligible
Low biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
	Total Scenario 3	Negligible
	Total Scenario 4	Negligible

Partial annual risk estimate for each outbreak scenario

The partial annual likelihood of entry, exposure, establishment and spread were combined with the impacts of the relevant outbreak scenarios to provide an estimate of the partial annual risk associated with each outbreak scenario. The results of this process are shown in Table 80.

Table 80. Partial annual risk (PAR) of each outbreak scenario

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Medium biosecurity poultry	Scenario 1	Negligible	Negligible	Negligible
	Scenario 2	Negligible	Negligible	Negligible
Non-avian species	Scenario 1	Zero	Zero	Zero
	Scenario 2	Zero	Zero	Zero
	Total Scenario 3	High	Negligible	Negligible
	Total Scenario 4	High	Negligible	Negligible

Unrestricted risk estimate

The overall risk associated with the import of whole chicken carcasses was assessed as ***negligible*** for FAdV-4. As the unrestricted risk estimate meets Australia's ALOP, no risk management was considered necessary.

Direct impact on human life or health

HPS is not known to affect humans and is not considered to be a threat to public health.

Group 2 Avian Adenovirus

Technical Information

Background

Avian adenovirus splenomegaly (AAS) is a viral disease of chickens related to Haemorrhagic Enteritis of turkeys and Marble Spleen Disease of pheasants. Although haemorrhagic enteritis and marble spleen disease have been reported in Australia (Tham and Thies 1988), AAS of chickens is an exotic disease. It is not an OIE-listed disease.

Agent taxonomy

Avian adenovirus splenomegaly is caused by a Group 2 avian adenovirus. AAS of chickens, Marble Spleen Disease Virus (MSDV) of pheasants and Haemorrhagic Enteritis Virus (HEV) of turkeys are indistinguishable in agar gel immunodiffusion tests but can be differentiated by restriction endonuclease fingerprinting and monoclonal antibody affinity (Zhang and Nagaraja 1989).

Agent characteristics

AAS virus is closely related to turkey HEV (Pierson and Fitzgerald 2003), and in the absence of published information on the resistance of AAS virus to physical and chemical agents, the IRA team assumed that data for turkey HEV will be applicable. Infectious gut contents from turkeys with HEV, maintained in a moist condition at 4 ºC, remain infectious for at least six months (Domermuth and Gross 1971). Infectivity of HEV can be destroyed by heating at 70 ºC for one hour, drying at 25 ºC for one week, treatment with 0.0086% sodium hypochlorite, 1.0% sodium lauryl sulphate, 0.4% chlorocide, 0.4% phenocide or 1% lysol (Pierson and Fitzgerald 2003). Infectivity is not destroyed by heating to 65 ºC for one hour; exposure to pH 3 at 25 ºC for 30 minutes; treatment with 50% chloroform or ether, or storage for four weeks at 37 ºC, six months at 4 ºC or four years at –40 ºC (Pierson and Fitzgerald 2003).

Epidemiology

Turkeys, pheasants and chickens are the natural hosts of Group 2 avian adenoviruses; however, group 2 adenovirus-associated disease has also been reported in guinea fowl and psittacines (Massi et al. 1995; Gomez-Villamandos et al. 1995). Antibodies to HEV/MSDV were not detected in the serum of 618 wild birds from 42 species in the southern United States, indicating that infection of wild birds is probably not commonplace (Domermuth et al. 1977).

While AAS has not been demonstrated to be present in Australia, HEV is found wherever turkeys are commercially reared, and MSDV has been diagnosed in North America, Europe, Korea and Australia (Tham and Thies 1988; Pierson and Fitzgerald 2003).

Unlike Group I adenoviruses, there is no evidence for vertical transmission of Group 2 adenoviruses in birds. Horizontal transmission appears to be via faeces, or fomites contaminated with the faeces of infected birds. No animal or insect vectors have been implicated in the transmission of HEV (Pierson and Fitzgerald 2003).

AAS affects young or market-age meat chickens and mature birds (Pierson and Domermuth 1997). Experimentally-infected five- to eight-week-old chickens developed splenomegaly within 5–7 days of oral inoculation with AASV (Domermuth et al. 1978b). HEV in turkeys and MSDV in pheasants have an incubation period of 3–6 days after experimental inoculation, depending on the route of infection (Pierson and Fitzgerald 2003).

Because of immunological cross reactivity of the group 2 avian adenoviruses, specific prevalence data for AAS are difficult to obtain. However, a serosurvey of 174 broiler-breeder farms in nine states of the United States showed that 46% of flocks tested positive for HEV/MSDV precipitating antibody (Domermuth et al. 1979). Interestingly, in that study, seroconversion was detected only in flocks over 19 weeks of age, despite the fact that the disease has been diagnosed in market-age meat chickens (Domermuth et al. 1978b). Within-flock seroprevalence appears to increase with the age of the flock to 50% (Domermuth et al. 1978b). Splenomegaly in market-age chickens resulted in 4% carcass condemnation at processing, while a total flock mortality rate of 9% was reported in chickens aged from 22 weeks old (Domermuth et al. 1978b; Domermuth, van der Heide, and Faddoul 1982).

Clinical Signs

Ante-mortem clinical signs were not described in market-age meat chickens condemned for splenomegaly at postmortem inspection (Domermuth et al. 1978b). In unusual cases, peracute or acute death of chickens due to respiratory compromise associated with pulmonary congestion and oedema may occur following infection with AAS (Pierson and Fitzgerald 2003). Ante-mortem clinical signs were not seen in mature chickens from a flock with increased mortality associated with AAS (Domermuth, van der Heide, and Faddoul 1982).

Pathogenesis

The pathogenesis of AAS infection in chickens is poorly understood. Group 2 avian adenoviruses appear to have an affinity for lymphoid tissue, including spleen, thymus and intestinal lymphoid tissue (Veit, Domermuth, and Gross 1981), and are lymphocytopathic (Pierson and Fitzgerald 2003). Immunosuppression occurs in birds affected with HEV and MSDV.

Pathology

In experimental infections of seven-week-old meat chickens, splenomegaly and splenic mottling were the main post-mortem findings (Veit, Domermuth, and Gross 1981). Mean spleen weights in experimentally-infected eight-week-old chickens varied from 4.85 to 6.4 grams (depending on time since inoculation) compared with a mean weight of 1.95 grams for spleens from control birds (Domermuth et al. 1978b).

In mature birds, splenomegaly, oedema and congestion of the lungs, and in some cases enteritis, hydropericardium and hepatomegaly were described (Domermuth, van der Heide, and Faddoul 1982).

Histologically, there was reticuloendothelial hyperplasia and intranuclear inclusions in the spleen, lymphoid degeneration within the lungs, and enlarged lymph nodules in the duodenum and ileum of inoculated birds (Veit, Domermuth, and Gross 1981).

Immunology

Due to the serologic cross-reactivity, avirulent HEV and MSDV can be used in the production of vaccines for ring-necked pheasants and turkeys (Pierson and Domermuth 1997; Fadly, Cowen, and Nazerian 1988; Domermuth et al. 1978a).

There is no vaccine currently available against AAS, nor does it seem likely that one will be developed, with some authors stating that it does not seem to be necessary (Pierson and Fitzgerald 2003).

Diagnosis

Diagnosis is made by isolation and identification of the causal organism from the spleen of AASV-infected chickens (Pierson and Fitzgerald 2003). Viral antigens can be detected in fixed tissues using immunofluorescence or immunoperoxidase tests, and in fresh or frozen tissue using agar gel precipitation, antigen-capture ELISA, restriction endonuclease fingerprinting and PCR (Pierson and Fitzgerald 2003). Serological tests are unlikely to be useful in market-age birds, as seroconversion is most common in older flocks (Domermuth et al. 1978b).

Transmission in chicken meat

Infection with AAS virus occurs in young and market-age meat chickens, which do not show clinical signs of disease. Although virus is most concentrated in the spleen, it is possible that asymptomatic chickens could be slaughtered during the viraemic phase, resulting in contamination of the entire carcass. Despite condemnation rates of 4% due to splenomegaly, it is likely that most infected birds would remain undetected during slaughter and processing. The virus survives refrigeration and freezing, therefore transmission of AAS could occur via chicken meat.

Quarantine significance

AAS is not an OIE-listed disease. It is not notifiable in any State or Territory of Australia, and is not subject to official controls. AAS is not included in the Emergency Animal Disease Response Agreement. Disease associated with AAS infection is not considered a major problem, since mortality is unusual and adverse effects are limited to some carcass condemnation at slaughter for enlarged spleens (McFerran and Smyth 2000). Therefore AAS is considered to be of relatively minor concern, and unlikely to have adverse effects beyond the local level.

Risk Assessment

Release Assessment

Rel_1: Selection of source flock (between flock prevalence)

The seroprevalence of AAS in some areas approaches 50%. For an unrestricted risk estimate, it was assumed that the prevalence is at the highest sustainable level in an endemically infected country or zone (see Method for Risk Assessment). The likelihood that a source flock will be infected with AAS was assessed by the IRA team as ***moderate***.

Rel_2: Infection detected through flock surveillance and the flock withdrawn from slaughter

AAS may be present in a flock with few clinical signs, although on rare occasions flock mortality of mature chickens may be increased. The likelihood that an infected flock will be detected through routine flock surveillance, and the entire flock withheld from slaughter, was assessed by the IRA team as ***extremely low***.

Rel_3: Selection of an infected chicken from an infected flock (within-flock prevalence)

Within-flock seroprevalence of 50% has been reported in mature flocks; however, seroconversion may be delayed to 19 weeks of age. If it is assumed that the infection rate is similar in market-age birds, the likelihood that a selected individual chicken will be infected was assessed by the IRA team as ***moderate***.

Rel_4: Background cross-contamination rate

This likelihood represents the background cross-contamination rate during slaughter and processing and applies to cross-contamination with material, infectious or otherwise, from other carcasses, including those tissues or materials in which the disease agent tends to localise.

AAS is a systemic infection affecting lymphoid tissue, including intestinal lymphoid tissue, and affected birds excrete virus in the faeces. The IRA team considered that the likelihood of a carcass being contaminated with potentially contaminated material from other birds, especially from the digestive tract, was ***high***.

Rel_5: Likelihood that an uninfected carcass will be contaminated with the disease agent during slaughter and processing

As discussed in the Method for Risk Assessment (page 58, Part B), Rel_4 was used to calculate Rel_{5a} (the likelihood that an uninfected carcass from an infected flock will become contaminated during slaughter and processing) and Rel_{5b} (the likelihood that an uninfected carcass from an uninfected flock will become contaminated during slaughter and processing).

For AAS, Rel_{5a} was calculated as ***moderate***, and Rel_{5b} was calculated as ***low***.

Rel_6: The likelihood that the carcass of a bird that was *infected* before slaughter will be removed as a result of inspections before or during processing

Condemnation rates of 4% due to splenomegaly have been reported. The IRA team considered that the rejection rate of infected carcasses would be greater than the background rejection rate but still very low. The likelihood that a contaminated/infected carcass will be removed during processing inspections was therefore assessed as ***very low.***

Rel_7: The likelihood that the carcass of a bird that was *not infected* before slaughter will be removed as a result of inspections before or during processing

The likelihood that an uncontaminated/uninfected carcass will be removed during processing inspections was considered to be equal to the background rejection rate of 0.75% as described in the Method section (page 59, Part B).

Rel_8: Inactivation of the agent during further processing, storage, handling and transport

Group 2 avian adenoviruses are resistant to alterations in pH and temperature. Given that the carcasses for importation will be chilled or frozen, and that the virus can persist for weeks or months at low temperatures, the likelihood of inactivation of the virus during further processing, storage, handling and transport was assessed by the IRA team as ***extremely low***.

Conclusions – Release assessment

After inserting the above estimates into the simulation model, and using the outputs from the model as a guide, the IRA team concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a ***moderate*** likelihood that imported chicken meat would be infected or contaminated with AAS virus.

Exposure assessment

Exposure Group 1: Wild birds

Of the steps identified as determinants in the infection of wild birds by exposure to scraps from an imported contaminated chicken carcass, $WB_{agentsurvival}$ and $WB_{infectivedose}$ are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$WB_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by a wild bird

Group 2 adenovirus, protected within chicken meat scraps, is likely to survive in the environment under ambient temperatures of 10 °C to 35 ºC for at least several days, giving ample time for wild birds to locate and scavenge the material. This likelihood was assessed by the IRA team as ***high***.

$WB_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a wild bird is sufficient to produce infection

AAS is a disease of domestic chickens, although related viruses can cause disease in turkeys, pheasants, guinea fowl and psittacines. Antibodies to Group 2 avian adenoviruses were not detected in a limited survey of wild birds in the United States, suggesting that infection of wild birds is not common.

Taking account of this information, the IRA team considered that there was a ***very low*** likelihood that AAS virus would infect a wild bird consuming the contaminated meat scraps.

Exposure Group 2: Low biosecurity poultry

Of the steps identified as determinants in the infection of low biosecurity poultry by exposure to material from an imported contaminated chicken carcass, $BP_{agentsurvival}$, $BP_{infectivedose}$, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$BP_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by low biosecurity poultry

The time between feeding of scraps and consumption by low biosecurity poultry is likely to be very short, so environmental degradation of the disease agent will be minimal. The likelihood that the agent will remain viable was considered by the IRA team to be ***certain (=1)***.

$BP_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a low biosecurity bird is sufficient to produce infection

Transmission of AAS is probably by the faecal-oral route (Pierson and Fitzgerald 2003), and chickens fed a spleen homogenate from infected birds developed clinical disease. The pathogenesis of infection, tissue distribution (other than lymphoid tissues) and oral infectious dose, are not documented. Given the lack of firm scientific data, the IRA team assessed that it is ***moderately*** likely that low biosecurity poultry would be infected with the virus as a result of consuming the contaminated imported chicken meat.

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

The likelihood that the virus would survive the rendering process was negligible. The IRA team considered that the likelihood the product would be re-contaminated with virus post-processing was negligible. Therefore the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with AAS virus was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

Given that the virus would be destroyed by rendering as discussed above, and that feed derived from contaminated carcasses would be diluted with feed from non-risk material, the likelihood that the amount of final poultry ration eaten by a bird would contain an oral infectious dose of virus was considered by the IRA team to be ***negligible***.

Exposure Group 3: Medium biosecurity commercial poultry

Of the steps identified as determinants in the infection of medium biosecurity commercial poultry by exposure to material from an imported contaminated chicken carcass, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with AAS virus was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As discussed above (low biosecurity poultry), the likelihood of the amount of final poultry ration consumed containing an oral infectious dose of AAS virus was considered to be ***negligible***.

Exposure Group 4: Non-avian species

As discussed above, $NAS_{agentsurvival}$ was considered to be equal to $WB_{agentsurvival}$.

There is no evidence that this virus infects non-avian species. Therefore this exposure group was not considered further in relation to this disease. $NAS_{infectivedose}$ was set to a value of zero.

Conclusions – Exposure assessment

Using the simulation model as a guide, the partial likelihood of exposure for each of the exposure groups was determined, taking the above estimates of the exposure variables into account. A summary of the outcomes determined by the IRA team is set out in Table 81.

Table 81. Partial likelihoods of exposure (PLE)

Exposure group	PLE
Wild birds	Extremely low
Low biosecurity poultry	Very low
Medium biosecurity poultry	Negligible
Non-avian species	Zero

Consequence assessment

Establishment and spread was considered in the context of the outbreak scenarios, described in the Method for Risk Assessment.

Estimating the likelihood of each outbreak scenario

The partial likelihood of establishment and spread (PLES) of AAS for the different exposure groups is described below. Four outbreak scenarios were considered relevant:

1. Disease agent does not establish or is not recognised within the directly exposed population
2. Disease agent establishes within the directly exposed population, is identified and is eliminated by human efforts or by natural means

3. Disease agent establishes in the directly exposed population, spreads within a district/region, including into other exposure groups if applicable, and is eliminated by human action or by natural means

4. Disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia.

Wild birds

Evidence suggests that wild birds are not susceptible to infection with AAS virus (Domermuth et al. 1977). The IRA team considered that the most likely outcome of infection of a wild bird resulting from scavenging chicken meat scraps would be a single or a few isolated occurrences of infection in wild birds, with the virus being unable to establish ongoing infection in the population. Infection of wild birds with AAS, with subsequent spread to poultry, has not been reported. In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 82).

Table 82. Estimated partial likelihood of establishment and spread (PLES) values for AASV in wild birds

Outbreak scenario 1:	$PLES_{(wb)}1$	**High**
Outbreak scenario 2:	$PLES_{(wb)}2$	**Very low**
Outbreak scenario 3:	$PLES_{(wb)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(wb)}4$	**Very low**

Low biosecurity poultry

Turkeys, pheasants and chickens are the natural hosts of group 2 avian adenoviruses. AAS virus affects young and market-age meat chickens and mature birds, causing increases in mortality. Although this exposure group includes commercial free-range poultry and ratites, the IRA team considered that the sub-set of this group most likely to be exposed to scraps of imported chicken meat was small flocks of backyard poultry, because these are more likely to be directly fed table scraps. In such flocks, there is less opportunity for the generation of high levels of environmental contamination than might occur with an outbreak of infectious disease in a large commercial flock. The most likely outcome of infection would be a single or a few isolated occurrences of infection.

If the disease were to establish in the flock, the level of expertise in disease recognition is likely to be low. Mechanical transmission of the virus by contaminated persons or fomites, and transmission by movement of birds may facilitate spread of the virus beyond the initially infected flock before it is recognised and eradication measures are implemented. In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. However, if infection did spread to involve commercial poultry, the absence of clinical signs, lack of official control program and the resistant nature of the virus could contribute to the infection becoming widespread. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 83).

Table 83. Estimated partial likelihood of establishment and spread (PLES) values for AASV in low biosecurity poultry

Outbreak scenario 1:	$PLES_{(bp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(bp)}2$	**Low**
Outbreak scenario 3:	$PLES_{(bp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(bp)}4$	**Low**

Medium biosecurity commercial poultry

The IRA team considered that the most feasible route for exposure of medium biosecurity commercial poultry to imported carcass components would be through poultry feed containing inadequately rendered processing waste. The likelihood that AAS virus would remain viable following rendering of contaminated imported carcasses and parts was assessed as negligible (page 250). Nevertheless, the IRA team estimated the PLES values, based on their assessment of the likely outcomes in the improbable event that exposure of medium biosecurity commercial poultry did occur via this route.

Young and market-age meat chickens infected with AAS do not show clinical signs of disease, and it is likely that most infected birds would remain undetected even during slaughter and processing. High population densities in commercial flocks would facilitate higher levels of environmental contamination, and lead to an increased probability of spread of the virus within flocks. Despite higher levels of management expertise, formal flock health monitoring and biosecurity in commercial flocks, spread from the affected flock could well occur. In view of these factors, outbreak scenario 4 (disease agent establishes, spreads to other exposure groups and becomes endemic) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 84).

Table 84. Estimated partial likelihood of establishment and spread (PLES) values for AASV in medium biosecurity commercial poultry

Outbreak scenario 1:	$PLES_{(cp)}1$	**Very low**
Outbreak scenario 2:	$PLES_{(cp)}2$	**Low**
Outbreak scenario 3:	$PLES_{(cp)}3$	**Low**
Outbreak scenario 4:	$PLES_{(cp)}4$	**Moderate**

Non-avian species

As discussed above, this exposure group was not considered further in relation to this disease.

Conclusion – Likelihood assessments

The estimates for the likelihood of release, the partial likelihoods of exposure for each of the exposure groups, and the partial likelihood of establishment and spread for each of the outbreaks scenarios were combined with the expected volume of trade using the simulation model. This allowed the calculation of partial annual likelihood of entry, exposure,

establishment and spread for each of the outbreak scenarios. The results of this calculation are shown in Table 85.

Table 85. Partial annual likelihood of entry, exposure, establishment and spread (PALEEES) for the outbreak scenarios

Exposure group	Outbreak scenario	PALEEES
Wild birds	Scenario 1	High
	Scenario 2	High
Low biosecurity poultry	Scenario 1	High
	Scenario 2	High
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
Total all exposure groups	Scenario 3	High
Total all exposure groups	Scenario 4	High

Summary: Impact assessment

Group II adenoviruses have only rarely been isolated from non-poultry species, and then only from aviary-kept birds. The IRA team considered that any direct or indirect environmental effects of infection with AAS virus were ***unlikely to be discernible***.

Due to the relatively minor level of disease reported in chickens, the IRA team considered that impacts arising from infection of low biosecurity poultry with AAS would be ***unlikely to be discernible*** and that any impacts arising from infection of medium biosecurity commercial poultry would be limited to ***minor*** effects on direct and indirect criteria 1 at the local level.

Because AAS is neither OIE-listed, nor notifiable, it is unlikely there will be adverse effects on international or domestic trade arising from infection with this virus, and no indirect effects on communities are expected.

Therefore the IRA team considered that the overall impact of infection with AAS virus would be ***negligible***.

Conclusions – impact assessment

The above estimates for each of the impact criteria were entered into the simulation model, and the overall impact of each outbreak scenario was estimated. Results are shown in Table 86.

Table 86. Impacts of each outbreak scenario

Exposure group	Outbreak scenario	Impact
Wild birds	Scenario 1	Negligible
	Scenario 2	Negligible
Low biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
	Total Scenario 3	Negligible
	Total Scenario 4	Negligible

Partial annual risk estimate for each outbreak scenario

The partial annual likelihood of entry, exposure, establishment and spread were combined with the impacts of the relevant outbreak scenarios to provide an estimate of the partial annual risk associated with each outbreak scenario. The outcomes of this process are shown in Table 87.

Table 87. Partial annual risk (PAR) of each outbreak scenario

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Medium biosecurity poultry	Scenario 1	Negligible	Negligible	Negligible
	Scenario 2	Negligible	Negligible	Negligible
Non-avian species	Scenario 1	Zero	Zero	Zero
	Scenario 2	Zero	Zero	Zero
	Total Scenario 3	High	Negligible	Negligible
	Total Scenario 4	High	Negligible	Negligible

Unrestricted risk estimate

The overall risk associated with the import of whole chicken carcasses was assessed as ***negligible*** for this virus. As this unrestricted risk estimate meets Australia's ALOP, risk management was not considered necessary.

Direct impact on human life or health

AAS virus is not known to affect humans and is not considered to be a threat to public health.

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Technical Information

Background

Mycoplasma iowae is primarily a pathogen of turkeys, causing embryo mortality and reduced hatchability; however, infections of chickens can occur. While clinical disease in adult turkeys is rare, *M. iowae* has been isolated from 17-day-old turkey poults with clinical signs of leg weakness (Trampel and Goll 1994). It has not been reported in domestic ducks. The organism has not been isolated from avian species in Australia (Al-Ankari and Bradbury 1996), and is not an OIE-listed agent.

Agent taxonomy

M. iowae is a species of the genus *Mycoplasma*. Mycoplasmas are micro-organisms of the Class Mollicutes. *M. iowae* is coccobacillary in form, pleomorphic with a three-layered cell membrane and, like all *Mycoplasma*, lacks a cell wall. There are many different strains of *M. iowae*, and marked within-species antigenic variation (Rhoades 1984).

Agent characteristics

Little is known about the susceptibility of *M. iowae* to disinfectants, and its survival in the environment. Mycoplasmas are presumed to be susceptible to thorough cleaning and disinfection. *M. gallisepticum* can be inactivated by phenol, formalin, B-propiolactone and merthiolate. The organism remains viable in chicken faeces for one to three days at 20 ºC, on cloth for three days at 20 ºC, and in egg yolk for 18 weeks at 37 ºC, or six weeks at 20 ºC. *M. gallisepticum* was stable in powdered skim milk, phosphate buffered saline, tryptose broth and distilled water for 24 hours at 4 ºC and 22 ºC (Ley and Yoder 1997).

M. iowae has been shown to survive on cotton, rubber, straw, feathers and human hair for at least six days, which was the duration of the experiment (Christensen et al. 1994). *M. iowae* appears to be slightly hardier to environmental conditions than *M. gallisepticum* and *M. synoviae.* Some strains of *M. iowae* are able to survive in culture media at pH 5.5 and in the presence of bile salts (Barber and Fabricant 1971).

Epidemiology

Turkeys and chickens are the natural hosts of *M. iowae*, although it is found most frequently in turkeys (Bradbury et al. 1990). The organism has also been isolated from geese (Bradbury and Kleven 2003), Amazon parrots, and from wild birds including starling, cormorants, heron, wood pigeons and an eider duck in a zoo (Al-Ankari and Bradbury 1996). Naturally-occurring disease appears to be restricted to turkeys, with occasional reports of disease in chicken flocks. *M. iowae* was one of several organisms isolated from parrots with upper respiratory disease, but no causal association was made between *M. iowae* and clinical disease (Bozeman, Kleven, and Davis 1984).

M. iowae occurs in North America, Europe, India and Asia, and is presumed to exist worldwide, with the exception of Australia and New Zealand.

Transmission in turkeys is by both vertical and horizontal routes (Bradbury and Kleven 2003). The rate of vertical transmission appears to decline with age, and varies with individuals in a flock. Some birds lay no or few infected eggs, while others lay many infected eggs (Bradbury and Kleven 2003). Horizontal transmission probably occurs in the hatchery, as the organism is found in the respiratory tract, the alimentary tract, and the meconium (Bradbury et al. 1990; Bradbury, Ideris, and Oo 1988). Unlike most avian mycoplasmas, *M. iowae* exhibits a predilection for the digestive tract (Mirsalimi, Rosendal, and Julian 1989). Day-old turkey poults orally inoculated with *M. iowae* developed intestinal infection, and became persistent faecal shedders of the organism, indicating that at least some strains are able to survive passage through the acid pH of the proventriculus and gizzard and colonise the wall of the intestinal tract (Shah-Majid and Rosendal 1987). The organism may persist in the cloaca until maturity, and can be found in the oviduct and semen of mature turkeys, facilitating venereal spread (Al-Ankari and Bradbury 1996). *M. iowae* has been isolated from naturally-infected chicken embryos, and from the respiratory tract, oviduct and hock joint of chickens (Yoder and Hofstad 1962; Al-Ankari and Bradbury 1996). It is assumed that horizontal spread among chicks could occur in the hatchery, as with poults.

M. iowae causes late embryonic death in turkey eggs, with reduced hatchability being the main sign of infection in turkey flocks. Signs of clinical disease in turkey poults or adult birds are uncommon, and little is known about the incubation period following horizontal transmission. In a turkey flock with naturally-occurring disease, onset of clinical signs of leg problems in poults was at nine days of age (Trampel and Goll 1994). There appears to be little or no information on the incubation period in naturally-infected chickens. Experimentally infected one-day-old poults showed depression in the second week of life, and leg abnormalities by the third week (Bradbury, Ideris, and Oo 1988), while inoculated chicks showed minimal clinical signs up to six weeks of age, when the experiment concluded (Bradbury and McCarthy 1984).

The prevalence of *M. iowae* infection in turkey and chicken flocks is unknown. Documentation of prevalence is complicated by the poor serological response to infection, even in persistently infected birds, and the difficulty in isolating the organism, especially from live adult birds (Bradbury et al. 1990). Of pooled serum samples taken from 122 commercial market turkey flocks in the United States, 18% had antibodies against *M. iowae* (Cummins and Reynolds 1990). However, both false positive and false negative results are possible in serological testing for mycoplasma infection (Bradbury 2001). In a study on mycoplasma species isolations from six avian species, the incidence of *M. iowae* in 696 chickens and chicken embryos was less than 2% (Bencina, Dorrer, and Tadina 1987). *M. iowae* was isolated from over 8% of chickens on one poorly managed commercial poultry farm, and was absent from three other farms which were considered to be well-managed (Bencina et al. 1987).

Clinical Signs

Clinical signs of disease are rarely observed during natural infections in chicken and turkey flocks (Al-Ankari and Bradbury 1996; Trampel and Goll 1994; Bradbury et al. 1990). The most common indication of infection is a reduction in hatchability of 2–5% of eggs in turkey flocks (Bradbury and Kleven 2003). However, a report exists of an outbreak of *M. iowae* infection in a large turkey flock in Iowa, in which poults developed leg abnormalities such as hock swelling, lameness, valgus deformities, splay legs and curling of the toes, with an onset of disease at nine days of age (Trampel and Goll 1994). In a report on naturally-occurring clinical disease associated with *M. iowae* in chickens, the organism was isolated from the swollen hock joints

of three meat chicken-breeder chicks in a flock with persistent lameness problems (Bradbury et al. 1990).

Clinical signs following experimental infection in turkey poults varied with the age of the bird and route of inoculation and with the strain of *M. iowae* used. Turkeys infected *in ovo* failed to hatch or were stunted and died within three weeks. Those infected at one day of age were stunted, and developed poor feathering and leg abnormalities such as ruptured tendons, swollen hocks and splayed legs (Bradbury, Ideris, and Oo 1988).

Following experimental infection at one day of age, some meat chicken breeder chicks showed depression, reluctance to stand, inability to flex the digits, poor feathering and poor growth rates (Bradbury and Kelly 1991). Clinical signs were less marked in light hybrid chicks inoculated with *M. iowae,* with most chicks remaining clinically normal (Bradbury and McCarthy 1984).

Pathogenesis

The disease characteristics of *M. iowae* have largely been determined by experimental infection rather than by observation of natural field cases. The pathology of natural and experimental infection with *M. iowae* is described below, but little information is available on the pathogenesis of disease in hatched birds. Embryos infected *in ovo* show stunting, congestion, hepatitis, oedema and splenomegaly (Bradbury and Kleven 2003).

Pathology

Turkeys infected with *M. iowae* in a natural outbreak had enlarged hock joints, valgus deformities, curling of the toes, and increased fluid in the hock joint space (Trampel and Goll 1994). Experimentally-infected turkeys showed chondrodystrophy, rotated tibias, splayed legs, deviated toes, excess fluid in the hock joints and rupture of the digital flexor tendons (Bradbury and Ideris 1982; Bradbury, Ideris, and Oo 1988).

Chicks experimentally infected at one day of age had leg abnormalities including tendon sheath exudation, adhesions and sometimes rupture of the digital flexor tendons in the hock area (Bradbury and Kelly 1991). There was mild thickening of the walls of the air sacs in some inoculated chicks (Bradbury and McCarthy 1984). Detailed post-mortem findings on naturally-infected chickens are not available.

Immunology

Little information is available on immunity to *M. iowae*, and while antibody responses have been observed, the serological response to infection is weak. There is no reliable serological test available for widespread use (Bradbury and Kleven 2003). *M. iowae* infection frequently fails to stimulate an immune response in mature turkeys, with the ELISA being more sensitive than the rapid serum agglutination test at detecting an immune response (Shah-Majid and Rosendal 1992). Organisms can persistently colonise organ systems, such as the reproductive and alimentary tracts (Fiorentin, Zhang, and Panangala 2000). Intravaginal inoculation of turkey hens with *M. iowae*-contaminated semen failed to stimulate an antibody response (Shah-Majid and Rosendal 1992). Similarly, experimental infection of one-day old chicks failed to stimulate an antibody response in most chicks, with the results of serological testing depending to some extent on the strain of *M. iowae* inoculated (Bradbury and McCarthy 1984). Long-term

infection with mycoplasmas is thought to be possible, because the organisms evade the host immune system by antigen switching (Fiorentin, Zhang, and Panangala 2000).

Diagnosis

Diagnosis of infection is by isolation of *M. iowae* from tissues of infected birds or dead embryos. However, the organism has specific growth requirements, and cannot always be isolated from persistently infected live birds (Bradbury et al. 1990). Serological detection of infection is unreliable, both because of a poorly detectable immune response in exposed birds, and because of the antigenic variation between the many strains of *M. iowae* (Rhoades 1984). PCR has been used for direct detection of *M. iowae* DNA (Zhao and Yamamoto 1993) and may become a more useful diagnostic procedure for field infections in the future.

Transmission in chicken meat

No information is available on the prevalence of *M. iowae* infection in meat chickens. However, clinical disease has only rarely been reported in meat chickens infected by *M. iowae*. The organism is capable of persistence in the alimentary and reproductive tracts and has been isolated from the hock joints of infected birds. Therefore, there is potential for carcass contamination to occur with *M. iowae*.

Quarantine significance

M. iowae is not an OIE-listed disease agent.

M. iowae is not notifiable in any State or Territory of Australia, and is not subject to official controls within Australia. *M. iowae* is not included in the Emergency Animal Disease Response Agreement. Therefore it is considered to be of relatively minor concern, and is unlikely to have consequences for the poultry industry that are discernible beyond the district/region level.

Risk Assessment

Release assessment

Rel_1: Selection of source flock (between-flock prevalence)

For an unrestricted risk estimate, it was assumed that the prevalence is at the highest sustainable level in an endemically infected country or zone (see Method for Risk Assessment). Prevalence of *M. iowae* infection in chicken flocks is unknown. However, since *M. iowae* is primarily a disease of turkeys, the IRA team considered it to be unlikely that chicken flocks would be infected. The likelihood that a source flock will be infected with *M. iowae* at the time of slaughter was estimated to be ***very low***.

Rel_2: Infection detected through flock surveillance and the flock withdrawn from slaughter

Clinical signs of *M. iowae* infection may be inapparent in chickens, although rarely, flocks may have an increased occurrence of lameness. The likelihood that an infected flock will be detected through routine flock surveillance, and the flock withheld from slaughter, was assessed by the IRA team as ***extremely low***.

Rel_3: Selection of an infected chicken from an infected flock (within-flock prevalence)

The within-flock prevalence of *M. iowae* in slaughter-age chickens is not known. In one instance of documented *M. iowae* infection on a poorly managed farm, within-flock prevalence, based on isolation or the organism, was 8%. If an infected flock was sent to slaughter, the IRA team considered that the likelihood that a selected individual chicken will be infected was ***low***.

Rel_4: Background cross-contamination rate

This likelihood represents the background cross-contamination rate during slaughter and processing and applies to cross-contamination with material, infectious or otherwise, from other carcasses, including those tissues or materials in which the disease agent tends to localise. *M. iowae* persists in the intestinal and reproductive tracts and has been isolated from the hock joints of infected birds. The IRA team considered that the likelihood of a carcass being contaminated with potentially contaminated material from other birds was ***very low***.

Rel_5: Likelihood that an uninfected carcass will be contaminated with the disease agent during slaughter and processing

As discussed in the Method for Risk Assessment (page 58, Part B), Rel_4 was used to calculate Rel_{5a} (the likelihood that an uninfected carcass from an infected flock will become contaminated during slaughter and processing) and Rel_{5b} (the likelihood that an uninfected carcass from an uninfected flock will become contaminated during slaughter and processing).

For *M. iowae*, Rel_{5a} was calculated as ***very low***, and Rel_{5b} was calculated as ***extremely low***.

Rel_6: The likelihood that the carcass of a bird that was *infected* before slaughter will be removed as a result of inspections before or during processing

No obvious post-mortem lesions are likely to be present during slaughter and processing. The IRA team considered that the rejection rate of infected carcasses would be equal to the background rejection rate of 0.75%.

Rel_7: The likelihood that the carcass of a bird that was *not infected* before slaughter will be removed as a result of inspections before or during processing

The likelihood that an uncontaminated/uninfected carcass will be removed during processing inspections was considered to be equal to the background rejection rate of 0.75% as described in the Method section (page 59, Part B).

Rel_8: Inactivation of the agent during further processing, storage, handling and transport

Little is known about the stability of *M. iowae* at carcass pH. Some strains of *M. iowae* have been documented to survive at pH 5.5 (carcass pH). The likelihood of inactivation of the bacteria during further processing, storage, handling and transport was assessed by the IRA team as ***moderate***.

Conclusions – Release assessment

After inserting the above estimates into the simulation model, and using the outputs from the model as a guide, the IRA team concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a ***very low*** likelihood that imported chicken meat would be infected or contaminated with *M. iowae*.

Exposure assessment

Exposure Group 1: Wild birds

Of the steps identified as determinants in the infection of wild birds by exposure to scraps from an imported contaminated chicken carcass, $WB_{agentsurvival}$ and $WB_{infectivedose}$ are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$WB_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by a wild bird

M. iowae, protected within chicken meat scraps, is likely to survive in the environment under ambient temperatures of 10 ºC to 35 ºC for at least 24 hours, giving ample time for wild birds to locate and scavenge the material. This likelihood was assessed by the IRA team as ***moderate***.

$WB_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a wild bird is sufficient to produce infection

M. iowae has been isolated from numerous species of wild birds, although no reports of clinical disease were found in association with the organism. While clinical disease may not occur in wild birds, they may act as mechanical vectors, shedding the bacteria through their faeces. Contamination of chicken carcasses with *M. iowae* is most likely to result from faecal contamination during processing, although some organisms may also persist in joints; therefore, the dose of bacteria present on the carcass is likely to be low.

The IRA team considered that there was an ***extremely low*** likelihood that *M. iowae* would infect a wild bird consuming the contaminated meat scraps.

Exposure Group 2: Low biosecurity poultry

Of the steps identified as determinants in the infection of low biosecurity poultry by exposure to material from an imported contaminated chicken carcass, $BP_{agentsurvival}$, $BP_{infectivedose}$, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$BP_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by low biosecurity poultry

The time between feeding of scraps and consumption by low biosecurity poultry is likely to be very short, so environmental degradation of the disease agent will be minimal. The likelihood that the agent will remain viable was assessed by the IRA team as ***certain (=1)***.

$BP_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a low biosecurity bird is sufficient to produce infection

Horizontal transmission of *M. iowae* can occur via the oral route (Shah-Majid and Rosendal 1987), and birds of all ages can be affected, although clinical signs may be inapparent in older birds (Al-Ankari and Bradbury 1996). The likelihood that chicken meat scraps would contain a sufficient dose of the agent to produce infection was assessed by the IRA team as ***very low***.

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

The likelihood that the agent would survive the rendering process was negligible. For *M. iowae* the IRA team considered that the likelihood that the product will be re-contaminated post-processing was negligible. Therefore, the likelihood that poultry feed would be contaminated with *M. iowae* was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

Given that *M. iowae* would be destroyed by rendering, and that even rendered waste contaminated with viable mycoplasma would be diluted with feed from non-risk material, the likelihood that the amount of final poultry ration eaten by a bird would contain an oral infectious dose was considered by the IRA team to be ***negligible***.

Exposure Group 3: Medium biosecurity commercial poultry

Of the steps identified as determinants in the infection of medium biosecurity commercial poultry by exposure to material from an imported contaminated chicken carcass, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with *M. iowae* was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that the final poultry ration consumed would contain an oral infectious dose of *M. iowae* was considered to be ***negligible***.

Exposure Group 4: Non-avian species

As discussed above, $NAS_{agentsurvival}$ was considered to be equal to $WB_{agentsurvival}$.

M. iowae has not been isolated from non-avian species. Therefore this exposure group was not considered further in relation to this disease. $NAS_{infectivedose}$ was set to a value of zero.

Conclusions – Exposure assessment

Using the simulation model as a guide, the partial likelihood of exposure for each of the exposure groups was determined, taking the above estimates of the exposure variables into account. A summary of the outcomes determined by the IRA team is set out in Table 88.

Table 88. Partial likelihoods of exposure (PLE)

Exposure group	PLE
Wild birds	Extremely low
Low biosecurity poultry	Extremely low
Medium biosecurity poultry	Negligible
Non-avian species	Zero

Consequence assessment

Establishment and spread was considered in the context of the outbreak scenarios, described in the Method for Risk Assessment.

Estimating the likelihood of each outbreak scenario

The partial likelihood of establishment and spread (PLES) of *M. iowae* for the different exposure groups is described below. Four outbreak scenarios were considered relevant:

1. Disease agent does not establish or is not recognised within the directly exposed population
2. Disease agent establishes within the directly exposed population, is identified and is eliminated by human efforts or by natural means
3. Disease agent establishes in the directly exposed population, spreads within a district/region, including into other exposure groups if applicable, and is eliminated by human action or by natural means
4. Disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia.

Wild birds

Outbreaks of disease associated with *M. iowae* have not been reported in wild birds, although the organisms have been isolated from several species. The IRA team considered that the most likely outcome of infection of a wild bird resulting from scavenging imported contaminated chicken meat scraps would be a single or a few isolated occurrences of infection in wild birds, with the organism being unable to establish ongoing infection in the population. Infection of wild birds with *M. iowae*, with subsequent spread to poultry, has not been reported and is considered to be of extremely low likelihood. In view of these factors, outbreak scenario 1

(disease does not establish or is not recognised) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 89).

Table 89. Estimated partial likelihood of exposure (PLES) values for *M. iowae* in wild birds

Outbreak scenario 1:	PLES(wb)1	**High**
Outbreak scenario 2:	PLES(wb)2	**Extremely low**
Outbreak scenario 3:	PLES(wb)3	**Extremely low**
Outbreak scenario 4:	PLES(wb)4	**Extremely low**

Low biosecurity poultry

Although this exposure group includes commercial free-range poultry and ratites, the IRA team considered that the sub-set of this group most likely to be exposed to scraps of imported chicken meat was small flocks of backyard poultry, because these are more likely to be directly fed table scraps. Turkeys and chickens are the natural hosts of *M. iowae*, although it is found most frequently in turkeys. Infected flocks may show no recognisable clinical disease. Moreover, turkeys represent a small proportion of the backyard poultry kept by households. In such flocks, there is less opportunity for the generation of high levels of environmental contamination than might occur with an outbreak of infectious disease in a large commercial flock. The most likely outcome of infection would be a single or a few isolated occurrences of infection.

If the disease was to establish in the flock, the level of expertise in disease recognition is likely to be low. Transmission of the organism by movement of birds or mechanical means may facilitate spread of the organism beyond the initially infected flock before it is recognised, and control measures are implemented. In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered to be moderately likely. *M. iowae* infection rarely causes clinical signs in chickens, and it is unlikely to be detected in low or medium biosecurity commercial poultry before becoming reasonably widespread. If detected, the infection will not be subject to Government-sponsored control programs. The IRA team assessed that it was extremely unlikely the infection would spread to involve high biosecurity breeder flocks but it was likely to become endemic in low and medium biosecurity poultry. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 90).

Table 90. Estimated partial likelihood of exposure (PLES) values for *M. iowae* in low biosecurity poultry

Outbreak scenario 1:	$PLES_{(bp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(bp}$ 2	**Very low**
Outbreak scenario 3:	$PLES_{(bp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(bp)}4$	**Moderate**

Medium biosecurity commercial poultry

The IRA team considered that the most feasible route for exposure of medium biosecurity commercial poultry to imported carcass components would be through poultry feed containing inadequately rendered processing waste. The likelihood that *M. iowae* would remain viable following rendering of contaminated imported carcasses and parts was assessed as negligible (page 267). Nevertheless, the IRA team estimated the PLES values, based on their assessment of the likely outcomes in the improbable event that exposure of medium biosecurity commercial poultry did occur via this route.

M. iowae in turkeys is transmitted by both vertical and horizontal routes, with spread of the organism probably occurring mainly within hatcheries. Clinical signs of disease due to *M. iowae* infections are rarely observed in chicken and turkey flocks, and the absence of clinical signs may delay the recognition of infection. This may result in the disease agent becoming widespread. Once diagnosed, it is likely that efforts would be made by industry to eradicate the disease, at least from turkeys. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 91).

Table 91. Estimated partial likelihood of exposure (PLES) values for *M. iowae* in medium biosecurity commercial poultry

Outbreak scenario 1:	$PLES_{(cp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(cp)}2$	**Very low**
Outbreak scenario 3:	$PLES_{(cp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(cp)}4$	**Moderate**

Non-Avian Species

As discussed above, this exposure group was not considered further in relation to this disease.

Conclusion – Likelihood assessments

The estimates for the likelihood of release, the partial likelihoods of exposure for each of the exposure groups, and the partial likelihood of establishment and spread for each of the outbreaks scenarios were combined with the expected volume of trade using the simulation model. This allowed the calculation of partial annual likelihood of entry, exposure, establishment and spread for each of the outbreak scenarios. The results of this calculation are shown in Table 92.

Estimating the impacts associated with each outbreak scenario

For each outbreak scenario the direct and indirect impacts of *M. iowae* infection were estimated at the national, State or Territory, district/region and local levels, as described in the Methods section (page 90-95, Part B).

M. iowae infection has not been reported in non-avian species (exposure group 4). Therefore, the impacts of infection occurring in this exposure group were not considered further.

Table 92. Partial annual likelihood of entry, exposure, establishment and spread (PALEES) for the outbreak scenarios

Exposure group	Outbreak scenario	PALEEES
Wild birds	Scenario 1	Low
	Scenario 2	Extremely low
Low biosecurity poultry	Scenario 1	High
	Scenario 2	High
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
Total all exposure groups	Scenario 3	High
Total all exposure groups	Scenario 4	High

Outbreak Scenario 1

By definition, outbreak scenario 1 means that the disease agent does not establish in the initially exposed population, or does not cause sufficient disease to lead to investigation and recognition of the infection. Therefore the impacts will be ***unlikely to be discernible*** for all exposure groups and all criteria.

Outbreak Scenario 2

The impacts of this outbreak scenario (disease agent establishes within the directly exposed population, and is identified and eliminated) will differ between exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

Since wild birds do not play a significant part in production, direct economic loss from death of these birds, were it to occur, is not measurable. Other impacts from the death of wild birds will be considered under direct criterion 2 and indirect criterion 4. The direct impacts of disease on wild birds were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Infection with *M. iowae* in low biosecurity poultry is unlikely to be recognisable, but even if clinical signs are recognised, the impacts on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

The most common indication of *M. iowae* infection is a reduction in hatchability of 2–5% of eggs in turkey flocks. Leg abnormalities in poults, such as hock swelling, lameness, valgus deformities, splay legs and curling of the toes, have also been reported. Impacts of an outbreak, contained within the exposed group of medium biosecurity commercial poultry, were assessed

by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels but ***minor*** at the local level due to the loss of production on the affected farm.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

An outbreak of disease in wild birds, if it occurs, is likely to be transient, with few detectable impacts on the population. The direct impacts on the environment were assessed as ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak of disease in low biosecurity poultry were assessed as ***unlikely to be discernible*** at all levels. The impacts of an outbreak of disease in medium biosecurity commercial poultry on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

M. iowae is not notifiable in any State or Territory of Australia, and is not subject to official controls. The impacts of an outbreak of *M. iowae* in wild birds were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Control and eradication programs may be instigated if *M. iowae* spreads to commercial flocks. Inactivated vaccines are available overseas and may be introduced to control disease, particularly in turkey flocks. The impacts of an outbreak in low biosecurity poultry were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels but ***minor*** at the local level, especially if the outbreak involved turkey flocks. Similarly, the impacts of an outbreak in medium biosecurity commercial poultry were assessed as ***unlikely to be discernible*** at national, State/Territory and district/region levels but ***minor*** at the local level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

The impacts of an outbreak of *M. iowae* in all exposure groups were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

M. iowae is not an OIE-listed disease, and the impacts of outbreaks on international trade were assessed by the IRA team as ***unlikely to be discernible*** at all levels for all exposure groups.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of an outbreak of *M. iowae* infection on the environment were assessed by the IRA team as ***unlikely to be discernible*** at all levels for all exposure groups.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impacts of a disease outbreak in all exposure groups were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Outbreak Scenario 3

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a district/region, and is eliminated) will be the same for all exposure groups, no matter which exposure group has been directly exposed to *M. iowae* in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

M. iowae infection may lead to production losses in affected flocks, particularly if the outbreak spreads to involve turkey flocks. The IRA team considered that the impacts at national, State/Territory and district/region levels were ***unlikely to be discernible***. However, the IRA team considered that animal production losses would be ***minor*** at the local level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

The impacts of an outbreak of disease were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

It is possible that control and eradication programs may be instigated if *M. iowae* spreads to commercial turkey flocks. Inactivated vaccines are available overseas and may be introduced to control disease in turkey flocks. The IRA team considered that the impacts were ***unlikely to be discernible*** at the national, State/Territory and district/region levels, and ***minor*** at the local level, especially in areas where the turkey industry is concentrated.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

The impact on turkey breeding operations would be significant because of depressed hatchability and reduced growing performance. Sales of poults from infected breeder flocks would impact on breeders' existing customers, who may have difficulty sourcing poults from elsewhere. Impacts on domestic trade and local industry were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory or district/region levels, and ***minor*** at the local level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

M. iowae is not recognised by the OIE as a disease of international trade significance. Impacts on local, district/region, State/Territory and national economies were assessed by the IRA team as ***unlikely to be discernible***.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of a local or general outbreak of *M. iowae* infection were assessed by the IRA team as ***unlikely to be discernible***.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impact of a localised outbreak of *M. iowae* on communities was assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Outbreak Scenario 4

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a State or Territory, and becomes endemic in Australia) will be the same for all exposure groups, no matter which exposure group has been directly exposed to *M. iowae* in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

Impacts of a more general spread of infection were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels. Impacts at the district/region level were assessed as ***minor***, particularly if the outbreak involved turkey flocks.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

The impacts of an outbreak of *M. iowae* infection were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

Spread of *M. iowae* infection to a general population of poultry or caged birds would lead to an increase in surveillance and monitoring, particularly of turkey flocks. It is possible that control and eradication programs may be instigated if *M. iowae* spreads to commercial turkey flocks.

Inactivated vaccines are available overseas and may be introduced to control disease in turkey flocks. A feature of the turkey industry is its concentration within local areas, which would assist in limiting impacts to the local level. The impacts on the national, State/Territory and regional economies were assessed by the IRA team as ***unlikely to be discernible***. Impacts were assessed as ***minor*** at the local level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Impacts on domestic trade and local industry were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory or district/region levels, and ***minor*** at the local level, for reasons explained previously, under outbreak scenario 3.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

M. iowae is not an OIE-listed disease, and the impacts of local or generalised outbreaks on international trade were assessed by the IRA team as ***unlikely to be discernible***.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of a local or general outbreak of *M. iowae* infection were assessed by the IRA team as ***unlikely to be discernible***.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impact of a general outbreak of *M. iowae* on communities was assessed by the IRA team as ***unlikely to be discernible*** at any level.

Conclusions – impact assessment

The above estimates for each of the impact criteria were entered into the simulation model, and the overall impact of each outbreak scenario was estimated. Results are shown Table 93.

Partial annual risk estimate for each outbreak scenario

The partial annual likelihood of entry, exposure, establishment and spread were combined with the impacts of the relevant outbreak scenarios to provide an estimate of the partial annual risk associated with each outbreak scenario. The outcomes of this process are shown in Table 94.

Unrestricted risk estimate

The overall risk associated with the import of whole chicken carcasses for *M. iowae* was assessed as ***very low***. As this unrestricted risk estimate meets Australia's ALOP, no risk management was considered necessary.

Direct impact on human life or health

M. iowae is not known to affect humans and is not considered to be a threat to public health.

Table 93. Impacts of each outbreak scenario

Exposure group	Outbreak scenario	Impact
Wild birds	Scenario 1	Negligible
	Scenario 2	Negligible
Low biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
	Total Scenario 3	Negligible
	Total Scenario 4	Very low

Table 94. Partial annual risk of each outbreak scenario

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	Low	Negligible	Negligible
	Scenario 2	E. Low	Negligible	Negligible
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Medium biosecurity poultry	Scenario 1	Negligible	Negligible	Negligible
	Scenario 2	Negligible	Negligible	Negligible
Non-avian species	Scenario 1	Zero	Zero	Zero
	Scenario 2	Zero	Zero	Zero
	Total Scenario 3	High	Negligible	Negligible
	Total Scenario 4	High	Very low	Very low

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Technical Information

Background

Mycoplasma synoviae causes respiratory disease and infectious synovitis in chickens and turkeys. Flocks infected with *M. synoviae* may have higher condemnation rates from airsacculitis and arthritis lesions, decreased body weight and increased feed conversions, and decreased egg production and hatchability rates, leading to economic losses (Ewing et al. 1998; Marois, Dufour-Gesbert, and Kempf 2000). There is substantial variation in pathogenicity and tissue tropism among isolates of *M. synoviae*, with many strains causing subclinical disease, and some resulting in significant disease problems (Kang, Gazdzinski, and Kleven 2002; Stipkovits and Kempf 1996; Senties-Cue, Shivaprasad, and Chin 2005). Some strains of *M. synoviae* occur in Australia (Gilchrist and Cottew 1974; Morrow et al. 1990).

M. synoviae infection was included as an OIE-listed disease for the first time in 2005, on the basis that 'several countries have eradication schemes' and 'the disease has significant morbidity' (World Organisation for Animal Health (OIE) 2005b).

Agent taxonomy

M. synoviae is a species of the genus *Mycoplasma*. Mycoplasmas are micro-organisms of the Class Mollicutes. *M. synoviae* is pleomorphic in form, with a three-layered cell membrane and an extracellular surface layer, and like all *Mycoplasma*, lacks a cell wall. There are many different strains of *M. synoviae*, with little antigenic heterogeneity being demonstrated among strains (Kleven 2003).

Agent characteristics

Resistance of *M. synoviae* to disinfectants has not been determined but is assumed to be similar to *M. gallisepticum* and other mycoplasmas (Kleven 2003). Mycoplasmas are presumed to be susceptible to thorough cleaning and disinfection. *Mycoplasma gallisepticum* can be inactivated by phenol, formalin, B-propiolactone and merthiolate. The organism remains viable in chicken faeces for one to three days at 20 ºC, on cloth for three days at 20 ºC, and in egg yolk for 18 weeks at 37 ºC, or six weeks at 20 ºC. *Mycoplasma gallisepticum* was stable in powdered skim mild, phosphate buffered saline, tryptose broth and distilled water for 24 hours at 4 ºC and 22 ºC (Ley and Yoder 1997).

M. synoviae is unstable at pH 6.8 or lower, and sensitive to temperatures above 39 ºC (Kleven 2003). The organism remains viable in egg yolk material for at least two years at –20 ºC, and for longer at lower temperatures, although at freezing temperatures, the titre is reduced (Kleven 2003).

M. synoviae, or its DNA, has been detected in drinking water, droppings, feed, feathers and dust in the environment of infected hens (Marois, Dufour-Gesbert, and Kempf 2000). Outside the host, *M. synoviae* is less resilient than *M. gallisepticum* and *M. iowae*, surviving for two to three days on feathers and several hours on cotton, straw and timber. The organism has been recovered from a human nose 12 hours after exposure (Christensen et al. 1994).

Pathogenicity of *M. synoviae* strains appears to be multifactorial, involving attachment and colonisation and unidentified factors associated with systemic invasion and lesion production (Lockaby et al. 1999). The only method currently available for assessing virulence of specific strains is by inoculation into chickens (S.H. Kleven, Department of Avian Medicine, University of Georgia, pers. comm. March 2002).

Epidemiology

Chickens, turkeys and guinea fowl are the natural hosts of *M. synoviae*, while ducks, geese, pigeons, Japanese quail, red-legged partridge, sparrows and pheasants have been found to be naturally-infected without showing clinical signs of disease (Kleven 2003; Bradbury, Yavari, and Dare 2001; Yamada and Matsuo 1983; Kleven and Fletcher 1983). Rabbits, rats, guinea pigs, mice, pigs and lambs did not show clinical signs or gross post-mortem lesions consistent with infectious synovitis following experimental inoculation with *M. synoviae* (Olson et al. 1956).

M. synoviae is present in poultry-producing countries worldwide, including Australia. It is likely that variations in strain and pathogenicity occur between countries, but to date there has been no evidence presented that exotic strains of *M. synoviae* are more virulent than Australian strains.

Transmission of *M. synoviae* is both vertical and horizontal, with spread occurring via direct and indirect contact. Transovarial transmission is greatest within the first four to six weeks after infection and, while transmission may cease after this time, infected flocks may continue to shed organisms (Kleven 2003). Vertical transmission is an important means of spread, with outbreaks in commercial flocks often being traced to infected breeder birds (Morrow et al. 1990; Droual et al. 1992; Ewing et al. 1996). Transovarial transmission also occurs in naturally-infected ducks and geese (Bencina, Tadina, and Dorrer 1988a; Bencina, Tadina, and Dorrer 1988b). Lateral transmission probably occurs via aerosol, with *M. synoviae* being demonstrated in the respiratory tract of control chickens within one to four weeks of commingling with infected birds (Ewing et al. 1998; Kleven 2003). Spread within a flock may be rapid or slow. Up to 100% of birds may become infected, although clinical signs may occur in few or no birds (Kleven 2003).

Signs of infection may be present in chickens as early as the first week of life, but acute infection is more generally seen when chickens are four to sixteen weeks old. Following acute infection, chronic infection may persist for the life of the flock. Turkeys may be affected with airsacculitis as early as one-day-old, but acute infection is more common when turkeys are 10–24 weeks old. Infection of the upper respiratory tract may be permanent (Kleven 2003).

The incubation period varies with the route of infection and the titre and pathogenicity of the organism. The incubation may be less than one week in birds infected by egg transmission, and 11–21 days following contact exposure. Intra-tracheal inoculation may result in infection within four days, followed by spread to in-contact birds (Kleven 2003). The incubation period following experimental inoculation by other routes varies from 2–21 days (Stipkovits and Kempf 1996).

The prevalence of *M. synoviae* infection is difficult to estimate. In a 1984 study, seroprevalence rate of *M. synoviae* in commercial pullet and layer flocks in southern California was estimated to be 91% and in central California was 32% (Mohammed et al. 1986). While eradication has been attempted by many commercial breeder and meat-chicken establishments, the standard

testing procedures for determining flock freedom have recently been called into question. In a study evaluating diagnostic tests for *M. synoviae* infection, the standard serum plate agglutination (SPA) and hemagglutination-inhibition (HI) tests were negative in some flocks from which *M. synoviae* was both cultured and detected by PCR (Ewing et al. 1996). Thus, the prevalence of infection in commercial flocks may be underestimated in some circumstances.

Clinical signs

Clinical signs of infectious synovitis include depression, poor growth rates, paleness of the comb, swelling of the hock joints and surrounding tissues, diarrhoea and ruffled feathers. While the hock joints and foot pads are most commonly affected, in some birds most joints can be affected; others a generalised infection can occur without apparent swelling of the joints (Kleven 2003). Morbidity generally approaches 15%, while mortality varies from 1–10% (Stipkovits and Kempf 1996).

Respiratory infection may be subclinical; however, respiratory signs, lameness, reduced growth rates, decreased egg production and mortality can occur (Stipkovits and Kempf 1996). Clinical disease is exacerbated by cold environmental temperatures and by simultaneous infection or vaccination with Newcastle disease, IBD or infectious bronchitis viruses (Kleven 2003). Air sac infection is the most often observed as a cause of condemnation in meat chickens (Kleven 2003), although other lesions leading to condemnation may include sternal (keel) bursitis and hock lesions (Senties-Cue, Shivaprasad, and Chin 2005). In one study, birds experimentally infected with *M. synoviae* via aerosol had condemnation rates of 13–39% at the processing plant (Vardaman, Reece, and Deaton 1973).

Egg production can be reduced by 5–10%, with a reduction of hatchability of 5–7% and approximately 5% mortality in the offspring flock (Stipkovits and Kempf 1996).

Pathogenesis

Factors determining pathogenesis include the pathogenicity and tissue tropism of the infective strain of *M. synoviae*, the number of infecting organisms, and the route of infection. Infection acquired through the egg can result in respiratory or synovial disease, while experimental inoculation via the footpad induces synovitis, and aerosol penetration causes airsacculitis. Infection can become systemic, leading to a combination of airsacculitis and infectious synovitis.

In the respiratory tract, *M. synoviae* colonises the tracheal epithelial surface, leading to loss of cilia and erosion of epithelial cells. Spread of the organism to the joints is probably via the bloodstream (Lockaby et al. 1998).

M. synoviae infection causes anaemia via erythrocyte damage and, when localised in tissue, attracts inflammatory cells leading to progressive accumulation of exudates around tendons and bursae and in joints (Stipkovits and Kempf 1996).

Pathology

Birds with infectious synovitis have swelling, often bilateral, of the footpads or hock joints and surrounding tissues. Joint fluid may vary from clear to purulent, with oedema, granulation tissue production and fibrosis occurring in affected areas. The sternal bursa may be affected in some birds (Morrow et al. 1990).

Airsacculitis may be present in the respiratory form of the disease, and may contribute to increased carcass condemnation rates (Kleven 2003). Infection with some strains may result in fibrinous pericarditis, and enlarged heart, liver, spleen and kidneys (Lockaby and Hoerr 1999).

Immunology

The immune response induced by *M. synoviae* is not strong, and is influenced by environmental conditions, the presence of respiratory viruses and the strain of the organism (Stipkovits and Kempf 1996).

There may be a long lag phase between infection with *M. synoviae* and development of detectable serum antibodies. The progeny of infected parents become seropositive by 8–12 weeks of age (Stipkovits and Kempf 1996). In an experimental trial, in which five-week-old control birds were intermingled with birds experimentally inoculated with *M. synoviae*, infection was detected by culture and PCR three or more weeks prior to seroconversion. Serum plate agglutination (SPA) tests were able to detect some infected groups of birds, while Haemagglutination Inhibition (HI) tests remained negative for more than six weeks (Ewing et al. 1998). In naturally-occurring outbreaks, SPA was not adequate as a sole screening test for *M. synoviae* infection, and HI was inadequate for confirmation of flock infection status. Both tests failed to detect early infections following repopulation of disinfected farms. ELISAs were found to be more reliable than HI in determining flock status, but PCR and culture were most sensitive and specific (Ewing et al. 1996). In one report, the ELISA produced no false positive results, but failed to identify some positive samples (i.e. the test produced false negative results) (May and Branton 1997). PCR is now commonly used for confirmation of *M. synoviae* infection in chickens and turkeys in the United States (Hong et al. 2004),

High concentrations of *M. synoviae*-specific antibodies may be found in synovial exudates of birds with infectious synovitis (Morrow et al. 1990). Antibodies are also detectable in egg yolk, in bile and in the bursa of Fabricius. In some cases, bile antibody assays may be positive while the serum assays are negative (Stipkovits and Kempf 1996).

Cell-mediated immunity may be an important contributor to the inflammatory response seen locally, at infected joints, tendon sheaths and bursae (Morrow et al. 1990).

Vaccines against *M. synoviae* are still being evaluated. In one large clinical trial conducted in Australia, serological response was slow to establish after vaccination with an attenuated live vaccine, taking 16–20 weeks to become universally positive. The vaccine did not provide protection against colonisation with a field strain of *M. synoviae*, but was reported to assist in improving performance of broilers (Markham, Scott, and Whithear 1998).

Diagnosis

Diagnosis is best made by isolation and identification of the causative organism in culture. The upper respiratory tract is the most reliable source of organisms in chronically affected birds, while lesions are a source of organism in acute infection. Colonies may be identified using fluorescent antibody techniques (Kleven 2003).

PCR is a highly sensitive method of detecting *M. synoviae* DNA in tissues, culture or environmental samples (Hong et al. 2004; Kleven 2003; Marois, Dufour-Gesbert, and Kempf 2000; Ewing et al. 1996).

Serological tests are often used to determine flock status, but false negative and false positive tests can occur (see section on Immunology).

Transmission in chicken meat

The potential for disease transmission in chicken meat is unknown. Lesions of the respiratory system, particularly the air sacs, and of synovial structures have the potential to contaminate the chicken carcass with viable organisms. Similarly, the presence of organisms in the intestinal tract could lead to carcass contamination and cross-contamination.

M. synoviae is not stable at pH 6.8 or lower. Breast meat de-boned immediately after chilling has a higher pH (6.2) than meat removed from the bones after four hours of chilling (pH: 5.5). However, in general, chilled chicken meat attains a pH of 5.5–5.9 in the first 24 hours, depending on processing methods (Lyon, Hamm, and Thomson 1985). It is therefore expected that *M. synoviae* organisms present in or on the carcass will be inactivated during packaging and storage.

Quarantine significance

Mycoplasma synoviae was added to the OIE list of notifiable disease agents in 2005 (World Organisation for Animal Health (OIE) 2005a).

M. synoviae infection is not currently notifiable in any State or Territory in Australia, and is not subject to official controls. M. *synoviae* is not included in the Emergency Animal Disease Response Agreement. Some strains of *M. synoviae* are endemic in Australian poultry.

As no evidence was found that exotic strains of *M. synoviae* are more virulent than endemic Australian strains, and because it is unlikely that the organism will survive in chicken carcasses, no further risk assessment was considered necessary for *M. synoviae*.

Direct impact on human life or health

M. synoviae is not known to affect humans and is not considered to be a threat to public health.

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Technical Information

Background

O. rhinotracheale is a respiratory pathogen of avian species, and has been implicated in both primary and secondary infections in chickens and turkeys. In meat chickens, infection results in mild respiratory signs, decreased growth rate and slight increases in mortality (van Veen, van Empel, and Fabri 2000). Increases in carcass condemnation rates at processing have also been reported (van Veen et al. 2000). Infection is more common in meat chicken breeders, especially during the period of peak egg production and results in mild respiratory signs, reduction in egg production and size, and poor egg shell quality (Chin, van Empel, and Hafez 2003). Infection with *O. rhinotracheale* has not been documented in avian species in Australia. It is of no known public health significance, and is not OIE-listed.

Agent taxonomy

O. rhinotracheale is a Gram-negative, highly pleomorphic, non-motile, non-sporulating bacterium belonging to the rRNA superfamily V (van Empel and Hafez 1999). Eighteen serotypes have been reported within the species (van den Bosch 2001), of which serotype A is the most prevalent, especially in chickens; 95% of chicken strains and 58% of turkey strains are serotype A (van Empel and Hafez 1999). The majority of strains belong to serotypes A, B, D and E. There is no evidence of host specificity. Serotypes A and C from chickens, and serotypes B, D, and E from turkeys have similar virulence for both turkeys and chickens (van den Bosch 2001). Differences in the pathogenicity of strains have been reported (van Veen, van Empel, and Fabri 2000).

Agent characteristics

There is little information on the susceptibility of *O. rhinotracheale* to physical or chemical action. However, the organism is reported to be highly sensitive to chemical disinfectants containing organic acids such as formic and glyoxyl acids, or aldehydes (van Empel and Hafez 1999).

O. rhinotracheale in poultry litter survived for one day at 37 °C, six days at 22 °C, 40 days at 4 °C and at least 150 days at –12 °C (Lopes et al. 2002). When sprayed on egg shells, *O. rhinotracheale* survived less than 24 hours at 37 °C, and less than three days at room temperature. However, at 4 °C the bacterium remained viable for 11 days (Varga, Fodor, and Makrai 2001).

Epidemiology

O. rhinotracheale infection in commercial poultry has been reported in the United States, Germany, South Africa, Netherlands, France, Belgium, Hungary, Japan, Canada, the United Kingdom (Turan and Ak 2002), Brazil (Canal et al. 2003), and Pakistan (Naeem, Malik, and Ullah 2003), but not Australia. Natural infections have been reported in chickens and turkeys, and the organism has been isolated from wild birds (gulls, rooks, partridge, chukar, pheasant, pigeon, quail, duck, ostrich, geese and Guinea fowl) (van Empel and Hafez 1999; van den

Bosch 2001). It has been postulated that infection in the poultry population may have resulted from the relatively recent transmission of *O. rhinotracheale* to domestic poultry from wild birds (Amonsin et al. 1997). Experimental studies have shown that all strains tested seem able to infect turkeys and meat chickens with comparable severity (van den Bosch 2001).

O. rhinotracheale spreads both vertically and horizontally (van Veen, Vrijenhoek, and van Empel 2004). Vertical and horizontal transmission have been demonstrated in turkeys within the hatchery (van Veen, Vrijenhoek, and van Empel 2004). Egg-transmission (either trans-ovarially or by cloacal contamination) is supported by the isolation of *O. rhinotracheale* from the oviducts and ovaries of experimentally infected 56-week-old turkey breeder hens (Back et al. 1998). Horizontal transmission is by direct or indirect contact through aerosols or drinking water (Chin, van Empel, and Hafez 2003).

Reports of the seroprevalence of *O. rhinotracheale* suggest that infection is common in domestic poultry flocks. Antibodies to *O. rhinotracheale* have been reported in 13% to 64% of meat chicken flocks, 79% to 100% of meat chicken breeder flocks, 90% to 100% of commercial layer flocks and 96.6% of meat turkey flocks in various countries (Heeder et al. 2001; Ryll et al. 1997; van Empel and Hafez 1999; Canal et al. 2003).

Clinical signs

The severity of clinical signs, duration of disease, and mortality in chickens and turkeys infected with *O. rhinotracheale* are extremely variable, and may be aggravated by other factors such as infections with viruses or bacteria, and climatic or environmental conditions. Infection commonly occurs in young chickens to slaughter age (van Veen et al. 2005), but is more common in meat chicken breeders of 24 to 52 weeks of age, especially during the period of peak egg production (van Empel and Hafez 1999). Inapparent infections may also occur (van Empel et al. 1999; Sprenger et al. 2000).

Young chickens generally show mild respiratory signs including sneezing, nasal discharge and airsacculitis lasting five to eight days, reduced weight gain, and a slight increase in mortality. Increased rates of condemnation at processing have been reported (van Veen et al. 2000). In a European survey, more than one third of the respiratory lesions in meat chickens at slaughter age were associated with *O. rhinotracheale* infection (van Veen et al. 2005). In breeders, decreased feed intake, mild respiratory signs (lasting over 21 days) and a slight increase in mortality are observed. Egg production may decrease, concomitant with poor egg shell quality and a decrease in egg size. Mortality rates generally range from 2% to 11% (van Empel and Hafez 1999).

Infection in young turkeys is generally mild. Affected birds may show nasal discharge, facial oedema and swelling of the infraorbital sinuses. More severe clinical signs and lesions are reported in birds over 14 weeks of age, and in breeders. Depression, gasping, dyspnoea and a slight decrease in egg production often precede a sudden increase in mortality which may reach 50% in older birds (van Empel and Hafez 1999).

Pathogenesis

In natural infections of turkeys and chickens, *O. rhinotracheale* is primarily a pathogen of the respiratory tract and is isolated most frequently from lungs and airsacs of affected birds (de Rosa et al. 1996). Isolation of *O. rhinotracheale* from trachea, sinus, pericardium, peritoneum and bone marrow has also been reported (de Rosa et al. 1996; van Veen et al. 2000).

Degeneration of the muscles has been described, and antigen demonstrated in tendon by immunohistochemical methods, indicating that systemic infection may occur (van Veen et al. 2000).

Disease can be induced in chickens and turkeys by intrathoracic, intratracheal, intra-airsac and intravenous inoculation of *O. rhinotracheale*, with or without priming by other pathogens such as Newcastle disease virus (Back et al. 1998; van Empel et al. 1999; Sprenger et al. 1998). Intravenous infection results in joint infections, meningitis and osteitis (Goovaerts, Vrijenhoek, and van Empel 1998). In turkeys infected by routes other than aerosol, *O. rhinotracheale* has been isolated from heart, liver, brain, oviduct and spleen, as well as from respiratory tissues (Back et al. 1998; Ryll et al. 1996). The incubation period varies with the route of infection, and may be from 24 hours (intratracheal infection) to more than seven days (aerosol infection) (Sprenger et al. 1998).

Pathology

Lesions in uncomplicated natural infections are generally confined to the respiratory tract where pneumonia, airsacculitis and fibrinopurulent pleuritis have been described (van Veen, van Empel, and Fabri 2000). More severe lesions are generally observed in older birds, especially breeders. Histologically, congestion, oedema, infiltration of inflammatory cells (macrophages and heterophils), and necrosis are observed in lungs, pleura and air sacs.

Immunology

ELISAs have been developed to detect antibodies to *O. rhinotracheale*, but serotype specificity may be a problem (Chin and Charlton 1998; van Empel et al. 1997). A serum plate agglutination (SPA) test has also been developed, but is limited in the serotypes it can detect. It is reported to be less sensitive than ELISA (Heeder et al. 2001). A commercial ELISA to serotypes A to M (IDEXX FlockChek *O. rhinotracheale* test kit) is available and will detect antibodies in both chicken and turkey serum. Antibody titres peak one to four weeks after natural infection but decline rapidly. Therefore, serum samples for flock screening should be taken at different ages within the flock.

An inactivated, water in oil vaccine (serotype A) is commercially available for use in meat chicken breeders to protect against shedding of the organism, and to obtain protection of the progeny (Intervet 2003).

Diagnosis

The clinical signs and lesions of ornithobacteriosis are not pathognomonic, and confirmation of the diagnosis relies on isolation and identification of *O. rhinotracheale*. Samples should be collected from the trachea, lungs and airsacs, infraorbital sinuses and nasal cavity early in the course of the disease (van Empel and Hafez 1999). Optimal growth of *O. rhinotracheale* occurs on 5% sheep blood agar in micro-aerophilic conditions and cultures should be observed for more than 48 hours (Chin, van Empel, and Hafez 2003). Growth of *O. rhinotracheale* may be masked by overgrowth of other bacteria. A PCR test has been developed and is useful for identification of the organism and in epidemiological studies. Immunofluorescence assay (IFA) and immunohistochemical tests were more sensitive than bacteriology and serology in detecting *O. rhinotracheale* in infected meat chicken flocks in Europe (van Veen et al. 2005).

Transmission in chicken meat

Carcasses may be condemned at slaughter due to purulent airsacculitis, ascites and polyserositis, and condemnation rates of 5% to 10% are considered 'usual' but rates of up to 50% have been reported (van Veen et al. 2000). Although *O. rhinotracheale* is primarily a pathogen of the respiratory tract and tissues of the respiratory tract are generally removed from carcasses during processing, systemic infection may occur. Therefore, carcasses could be infected with *O. rhinotracheale* or become contaminated during processing.

Quarantine significance

O. rhinotracheale is not an OIE-listed disease agent.

O. rhinotracheale is not notifiable in any State or Territory of Australia, and is not subject to official controls within Australia. *O. rhinotracheale* is not included in the Emergency Animal Disease Response Agreement. Therefore, it is considered to be of relatively minor concern, and is unlikely to have consequences for the poultry industry that are discernible beyond the district/region level.

Risk Assessment

Release Assessment

Rel_1: Selection of source flock (between flock prevalence)

Reports of the seroprevalence of *O. rhinotracheale* suggest that infection is common in domestic poultry flocks. In meat chicken flocks the reported seroprevalence is up to 64% (Canal et al. 2003). For an unrestricted risk estimate, it was assumed that the prevalence is at the highest sustainable level in an endemically infected country or zone (see Method for Risk Assessment). The likelihood that a source flock will be infected with *O. rhinotracheale* was therefore assessed by the IRA team as ***moderate***.

Rel_2: Infection detected through flock surveillance and the flock withdrawn from slaughter

The mild respiratory signs of *O. rhinotracheale* infection may not be obvious to the producer and are not sufficiently specific to be diagnostic. In addition, inapparent infections are common. The likelihood that an infected flock will be detected through routine flock surveillance, and the flock withheld from slaughter was assessed by the IRA team as ***extremely low***.

Rel_3: Selection of an infected chicken from an infected flock (within flock prevalence)

Infection with *O. rhinotracheale* occurs commonly in young meat chickens. The organism spreads horizontally primarily by aerosol and it would be expected to spread rapidly once introduced to a meat chicken flock. The IRA team considered that if an affected flock were sent to slaughter, the likelihood that a selected individual chicken will be infected was ***moderate***.

Rel_4: Background cross-contamination rate

This likelihood represents the background cross-contamination rate during slaughter and processing and applies to cross-contamination with material, infectious or otherwise, from other carcasses, including those tissues or materials in which the disease agent tends to localise. *O. rhinotracheale* localises predominantly in the respiratory tract, which is removed during processing. The IRA team assessed that the likelihood of a carcass being contaminated with potentially contaminated material from other birds was ***low***.

Rel_5: Likelihood that an uninfected carcass will be contaminated with the disease agent during slaughter and processing

As discussed in the Method for Risk Assessment (page 58, Part B), Rel_4 was used to calculate Rel_{5a} (the likelihood that an uninfected carcass from an infected flock will become contaminated during slaughter and processing) and Rel_{5b} (the likelihood that an uninfected carcass from an uninfected flock will become contaminated during slaughter and processing).

For *O. rhinotracheale*, Rel_{5a} was calculated as ***low***, and Rel_{5b} was calculated as ***very low***.

Rel_6: The likelihood that the carcass of a bird that was *infected* before slaughter will be removed as a result of inspections before or during processing

Carcasses showing obvious pneumonia, airsacculitis, pleuritis, ascites and polyserositis would, most likely, be detected during processing. However, birds in the acute stages of infection may show no gross lesions. The IRA team considered that the rejection rate of infected carcasses would be greater than the background rejection rate but still very low. The likelihood that a contaminated/infected carcass will be removed during processing inspections was therefore assessed as ***very low.***

Rel_7: The likelihood that the carcass of a bird that was *not infected* before slaughter will be removed as a result of inspections before or during processing

The likelihood that an uncontaminated/uninfected carcass will be removed during processing inspections was considered to be equal to the background rejection rate of 0.75% as described in the Method section (page 59, Part B).

Rel_8: Inactivation of the agent during further processing, storage, handling and transport

O. rhinotracheale in poultry litter has been shown to survive 40 days at 4°C and at least 150 days at –12°C (Lopes et al. 2002). Given that the carcasses for importation will be chilled or frozen, the likelihood of inactivation of *O. rhinotracheale* during further processing, storage, handling and transport was assessed by the IRA team as ***very low***.

Conclusions – Release assessment

After inserting the above estimates into the simulation model, and using the outputs from the model as a guide, the IRA team concluded that, in the absence of risk management and without

considerations regarding the exporting country, there was a ***low*** likelihood that imported chicken meat would be infected or contaminated with *O. rhinotracheale*.

Exposure assessment

Exposure Group 1: Wild birds

Of the steps identified as determinants in the infection of wild birds by exposure to scraps from an imported contaminated chicken carcass, $WB_{agentsurvival}$ and $WB_{infectivedose}$ are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$WB_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by a wild bird

There is little information on the effects of temperature on *O. rhinotracheale*, however the IRA team considered that there is a ***low*** likelihood that *O. rhinotracheale* would survive in the environment under ambient temperatures of 10 ºC to 35 ºC.

$WB_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a wild bird is sufficient to produce infection

O. rhinotracheale has been isolated from several species of wild birds including gulls. *O. rhinotracheale* is primarily a pathogen of the respiratory tract and infection is most likely to be by aerosol. However, organisms may be sequestered in the heart, liver and gizzard, and if these organs were available to wild birds, the risk of infection associated with their consumption would be higher than for other carcass scraps. This risk assessment considers the importation of whole carcasses, including heart, gizzard and liver, therefore $WB_{infectivedose}$ was estimated on the assumption that these organs may be discarded in refuse.

Because horizontal transmission of *O. rhinotracheale* is primarily via aerosol, the IRA team considered that there was a ***very low*** likelihood that the pathogen would infect a wild bird consuming the contaminated meat scraps.

Exposure Group 2: Low biosecurity poultry

Of the steps identified as determinants in the infection of low biosecurity poultry by exposure to material from an imported contaminated chicken carcass, $BP_{agentsurvival}$, $BP_{infectivedose}$, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$BP_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by low biosecurity poultry

The time between feeding of scraps and consumption by low biosecurity poultry is likely to be very short, so environmental degradation of the disease agent will be minimal. The IRA team considered that the likelihood that the agent will remain viable is ***certain (=1)***.

$BP_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a low biosecurity bird is sufficient to produce infection

O. rhinotracheale is primarily a pathogen of the respiratory tract and natural transmission is by aerosol. However, organisms may be sequestered in the heart, liver and gizzard, and if these organs were available to low biosecurity poultry, the risk of infection associated with their consumption would be higher than for other carcass scraps. This risk assessment considers the importation of whole carcasses, including heart, gizzard and liver, therefore $BP_{infectivedose}$ was estimated on the assumption that these organs may be fed to low biosecurity poultry.

Because horizontal transmission of *O. rhinotracheale* is primarily via aerosol, the IRA team considered that there was a ***very low*** likelihood that the pathogen would infect low biosecurity poultry consuming imported contaminated chicken meat scraps.

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

The likelihood that the agent would survive the rendering process was negligible. For *O. rhinotracheale* the IRA team considered that the likelihood that the product will be re-contaminated post processing was negligible. Therefore, the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with *O. rhinotracheale* was estimated by the IRA team to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

Given that *O. rhinotracheale* would be destroyed by rendering, and that even rendered waste contaminated with viable organisms would be diluted with feed from non-risk material, the IRA team considered that the likelihood that the final poultry ration eaten by a bird would contain an oral infectious dose of organisms was considered to be ***negligible***.

Exposure Group 3: Medium biosecurity commercial poultry

Of the steps identified as determinants in the infection of medium biosecurity commercial poultry by exposure to material from an imported contaminated chicken carcass, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that poultry feed derived from the imported contaminated carcass, would be contaminated with *O. rhinotracheale* was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that the final poultry ration would contain an oral infectious dose of *O. rhinotracheale* was considered to be ***negligible***.

Exposure Group 4: Non-avian species

As discussed above, $NAS_{agentsurvival}$ was considered to be equal to $WB_{agentsurvival}$.

There are no reports of the infection of non-avian species with *O. rhinotracheale,* and it was considered extremely unlikely that disease would establish and spread in non-avian species gaining access to discarded imported contaminated chicken meat scraps. Therefore this exposure group was not considered further in relation to this disease. $NAS_{infectivedose}$ was set to a value of zero.

Conclusions – Exposure assessment

Using the simulation model as a guide, the partial likelihood of exposure for each of the exposure groups was determined, taking the above estimates of the exposure variables into account. A summary of the outcomes determined by the IRA team is set out in Table 95.

Table 95. Partial likelihoods of exposure

Exposure group	PLE
Wild birds	E. Low
Low biosecurity poultry	E. Low
Medium biosecurity poultry	Negligible
Non-avian species	Zero

Consequence assessment

Establishment and spread was considered in the context of the outbreak scenarios, described in the Method of Risk Assessment.

Estimating the likelihood of each outbreak scenario

The partial likelihood of establishment and spread (PLES) of *O. rhinotracheale* for the different exposure groups is described below. Four outbreak scenarios were considered relevant:

1. Disease agent does not establish or is not recognised within the directly exposed population
2. Disease agent establishes within the directly exposed population, is identified and is eliminated by human efforts or by natural means
3. Disease agent establishes in the directly exposed population, spreads within a district/region, including into other exposure groups if applicable, and is eliminated by human action or by natural means

4. Disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia.

Wild birds

O. rhinotracheale has been isolated from a number of species including gulls, although no clinical data is available to show if these birds showed signs of disease, or were able to transmit the organism to other species of birds. The IRA team considered that the most likely outcome of infection of a wild bird resulting from scavenging imported contaminated chicken meat scraps would be a single or a few isolated occurrences of infection in wild birds, with the organism being unable to establish ongoing infection in the population. Infection of wild birds with *O. rhinotracheale*, with subsequent spread to poultry, has not been reported although it has been postulated that infection in the poultry population may have resulted from the relatively recent transmission of *O. rhinotracheale* to domestic poultry from wild birds. If infection were to occur in wild birds, it is unlikely to be recognised or naturally eliminated. If infection occurs in low or medium biosecurity commercial poultry, overseas experience suggests that it is likely to become widespread. In the absence of official control programs, the disease agent is likely to become endemic rather than be eradicated. In view of these factors, outbreak scenarios 1 (disease does not establish or is not recognised) and 4 (disease agent becomes endemic) were considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 96).

Table 96. Estimated partial likelihood of exposure (PLES) values for *O. rhinotracheale* in wild birds

Outbreak scenario 1:	$PLES_{(wb)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(wb)}2$	**Very low**
Outbreak scenario 3:	$PLES_{(wb)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(wb)}4$	**Moderate**

Low biosecurity poultry

O. rhinotracheale is a respiratory pathogen of avian species, causing primary and secondary infections in chickens and turkeys. Although this exposure group includes commercial free-range poultry and ratites, the IRA team considered that the sub-set of this group most likely to be exposed to scraps of imported chicken meat was small flocks of backyard poultry, because these are more likely to be directly fed table scraps. In such flocks, there is less opportunity for the generation of high levels of environmental contamination than might occur with an outbreak of infectious disease in a large commercial flock. The most likely outcome of infection would be a single or a few isolated occurrences of infection.

If the disease were to establish in the flock, the level of expertise in disease recognition is likely to be low, and transmission by movement of poultry or wild birds may facilitate spread of the organism beyond the initially infected flock before it is recognised, and eradication measures could be implemented. If infection occurs in low or medium biosecurity commercial poultry, overseas experience suggests that it is likely to become widespread. In the absence of official control programs, the disease agent is likely to become endemic rather than be eradicated. In

view of these factors, outbreak scenarios 1 (disease does not establish or is not recognised) and 4 (disease agent becomes endemic) were considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 97).

Table 97. Estimated partial likelihood of exposure (PLES) values for *O. rhinotracheale* in low biosecurity poultry

Outbreak scenario 1:	$PLES_{(bp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(bp)}2$	**Very low**
Outbreak scenario 3:	$PLES_{(bp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(bp)}4$	**Moderate**

Medium biosecurity commercial poultry

The IRA team considered that the most feasible route for exposure of medium biosecurity commercial poultry to imported carcass components would be through poultry feed containing inadequately rendered processing waste. The likelihood that pathogens would remain viable following rendering of contaminated imported carcasses and parts was assessed as negligible (page 293). Nevertheless, assessment of PLES was based on the assumption that medium biosecurity commercial poultry had been exposed to poultry feed contaminated with the disease agent.

O. rhinotracheale is primarily a pathogen of the respiratory tract of chickens and turkeys, causing mild respiratory disease in uncomplicated infections. Transmission is both vertical and horizontal (via aerosol), and infection is likely to spread rapidly once introduced into a flock. The clinical signs and lesions of ornithobacteriosis are not pathognomonic, and it is likely that the disease will escape recognition until it has spread widely. In the absence of official control programs, the disease agent is likely to become endemic rather than be eradicated. *O. rhinotracheale* has become endemic in the intensive poultry industry in many overseas countries. In view of these factors, outbreak scenarios 1 and 4 are considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 98).

Table 98. Estimated partial likelihood of exposure (PLES) values for *O. rhinotracheale* in medium biosecurity commercial poultry

Outbreak scenario 1:	$PLES_{(cp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(cp)}2$	**Very low**
Outbreak scenario 3:	$PLES_{(cp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(cp)}4$	**Moderate**

Non-Avian Species

As discussed above, this exposure group was not considered further in relation to this disease.

Conclusion – Likelihood assessments

The estimates for the likelihood of release, the partial likelihoods of exposure for each of the exposure groups, and the partial likelihood of establishment and spread for each of the outbreaks scenarios were combined with the expected volume of trade using the simulation model. This allowed the calculation of partial annual likelihood of entry, exposure, establishment and spread for each of the outbreak scenarios. The results of this calculation are shown in Table 99.

Table 99. Partial annual likelihood of entry, exposure, establishment and spread (PALEES) for the outbreak scenarios

Exposure group	Outbreak scenario	PALEEES
Wild birds	Scenario 1	High
	Scenario 2	High
Low biosecurity poultry	Scenario 1	High
	Scenario 2	High
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
Total all exposure groups	Scenario 3	High
Total all exposure groups	Scenario 4	High

Estimating the impacts associated with each outbreak scenario

For each outbreak scenario, the direct and indirect impacts of *O. rhinotracheale* infection were estimated at the national, State or Territory, district/region and local levels, as described in the Methods section (page 90-95, Part B).

The likelihood of *O. rhinotracheale* affecting non-avian species (exposure group 4) was considered to be remote. Therefore, the impacts of *O. rhinotracheale* occurring in this exposure group were not considered further.

Outbreak Scenario 1

By definition, outbreak scenario 1 means that the disease agent does not establish in the initially exposed population, or does not cause sufficient disease to lead to investigation and recognition of the infection. Therefore the IRA team considered that the impacts will be ***unlikely to be discernible*** for all exposure groups and all criteria.

Outbreak Scenario 2

The impacts of this outbreak scenario (disease agent establishes within the directly exposed population, and is identified and eliminated) will differ between exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

Because wild birds do not play a significant part in production, direct economic loss from death of these birds is not measurable. Other impacts from the death of wild birds will be considered under criterion 2: the environment. The direct impacts of disease on wild birds were assessed by the IRA team as ***unlikely to be discernible.***

O. rhinotracheale infection in young chickens and turkeys generally results in mild respiratory disease and reduction in weight gain. In older birds, there may be a decrease in egg production, and increased mortality. An outbreak contained within a local population of low biosecurity poultry will result in losses to individual owners only. The impacts of such losses on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

If *O. rhinotracheale* were to infect medium biosecurity commercial poultry, losses due to decreased egg production, increased carcass condemnation, reduced weight gain and increased mortality rates may result in ***minor*** impacts at the local level. The IRA team considered that the impacts were ***unlikely to be discernible*** at national, State/Territory and district/region levels.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

O. rhinotracheale has been isolated from wild birds including gulls, but there are no reports of clinical disease in these species. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at all levels for all exposure groups.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

O. rhinotracheale is not notifiable in any State or Territory of Australia, and is not subject to official controls. The impacts of an outbreak of *O. rhinotracheale* in wild birds on this criterion were assessed as ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak in low biosecurity poultry were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

While it is unlikely that official action will be taken to eradicate the disease if *O. rhinotracheale* were diagnosed in medium biosecurity commercial poultry flocks, individual growers will need to take some control action to minimise losses. Such action may include the use of antimicrobials or vaccination. Impacts of such programs were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels but ***minor*** at the local level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

The impacts of an outbreak of *O. rhinotracheale* in wild birds on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak of

O. rhinotracheale in low biosecurity poultry were assessed as ***unlikely to be discernible*** at all levels.

If an outbreak were to occur in medium biosecurity commercial poultry, especially turkeys, production losses may result in ***minor*** impacts at the local level. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

O. rhinotracheale is not recognised by the OIE as a disease of international trade significance. Impacts for all exposure groups were assessed as ***unlikely to be discernible*** at all levels.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of an outbreak of *O. rhinotracheale* in all exposure groups were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impacts of an outbreak of *O. rhinotracheale* in all exposure groups were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Outbreak Scenario 3

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a district/region, and is eliminated) will be the same for all exposure groups, no matter which exposure group has been directly exposed to *O. rhinotracheale* in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

Losses due to decreased egg production, reduced weight gain, increased carcass condemnation and increased mortality rates were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels but ***minor*** at the local level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

There have been no reports of *O. rhinotracheale* outbreaks in the wild. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

It is possible that control and eradication programs may be instigated if *O. rhinotracheale* were to spread to commercial flocks. Vaccines are available overseas and may be introduced to control disease in chicken and turkey breeders, and possibly also in turkey poults. The IRA team considered that the impacts were ***unlikely to be discernible*** at national, State/Territory and district/region levels, and ***minor*** at the local level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Reduced weight gains may lead to longer grow-out periods, impacting on the returns to contract growers. Impacts on domestic trade and industry were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, and ***minor*** at the local level.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

O. rhinotracheale is not recognised by the OIE as a disease of international trade significance. Impacts on the local, district/region, State/Territory and national economies were assessed by the IRA team as ***unlikely to be discernible***.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impact of an outbreak of *O. rhinotracheale* on this criterion was assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Temporary restrictions may be imposed on movement of birds, eggs, poultry products and people until the diagnosis is confirmed. However, the IRA team considered that the impacts were ***unlikely to be discernible*** at all levels.

Outbreak Scenario 4

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a State/Territory, and becomes endemic in Australia) will be the same for all exposure groups, no matter which exposure group has been directly exposed to *O. rhinotracheale* in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

If the disease spreads more widely through medium biosecurity commercial poultry and low biosecurity poultry, losses of birds and production may be substantial. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, and ***minor*** at the district/region level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

There have been no reports of *O. rhinotracheale* outbreaks in the wild. Impacts on the environment were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

Vaccines may be introduced to control disease, resulting in added costs to poultry producers. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, and ***minor*** at the district/region level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

If *O. rhinotracheale* spreads to a more general population of poultry, the impacts at State/Territory and national levels were assessed as ***unlikely to be discernible***. The IRA team considered that there would be ***minor*** impacts at the district/region level on account of the capacity of the causative organism to reduce productivity in the chicken and turkey industries.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

Impacts of a more general outbreak were assessed as ***unlikely to be discernible*** at all levels.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of a generalised outbreak of *O. rhinotracheale* were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Temporary restrictions may be imposed on movement of birds, eggs, and poultry products until the diagnosis is confirmed. This may lead to community disruption. The impacts were assessed

by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level.

Conclusions – impact assessment

The above estimates for each of the impact criteria were entered into the simulation model, and the overall impact of each outbreak scenario was estimated. Results are shown in Table 100.

Table 100. Impacts of each outbreak scenario

Exposure group	Outbreak scenario	Impact
Wild birds	Scenario 1	Negligible
	Scenario 2	Negligible
Low biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
	Total Scenario 3	Negligible
	Total Scenario 4	Very low

Partial annual risk estimate for each outbreak scenario

The partial annual likelihood of entry, exposure, establishment and spread were combined with the impacts of the relevant outbreak scenarios to provide an estimate of the partial annual risk associated with each outbreak scenario. The outcomes of this process are shown in Table 101.

Unrestricted risk estimate

The overall risk associated with the import of whole chicken carcasses was assessed as ***very low*** for *O. rhinotracheale*. As this unrestricted risk estimate meets Australia's ALOP, no risk management was considered necessary.

Direct impact on human life or health

O. rhinotracheale is not known to affect humans and is not considered to be a threat to public health.

Table 101. Partial annual risk (PAR) of each outbreak scenario

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Medium biosecurity poultry	Scenario 1	Negligible	Negligible	Negligible
	Scenario 2	Negligible	Negligible	Negligible
Non-avian species	Scenario 1	Zero	Zero	Zero
	Scenario 2	Zero	Zero	Zero
	Total Scenario 3	High	Negligible	Negligible
	Total Scenario 4	High	Very low	Very Low

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Technical Information

Background

Avian reoviruses are the cause of infectious viral arthritis/tenosynovitis of poultry. Although reoviruses have also been associated with a number of other poultry disease conditions including malabsorption syndrome, runting/stunting syndrome, diarrhoea, respiratory disease and sudden death, in many cases a causal association is not proven. This risk assessment considers only arthritis/tenosynovitis due to exotic strains of reovirus.

While reovirus infections are widespread, 85–90% of avian reoviruses are considered to be non-pathogenic (Jones 2000). Production losses due to viral arthritis/tenosynovitis overseas have prompted the development of vaccines to prevent infections in commercial poultry. Vaccination is not practised in Australia, because Australian reovirus strains appear to be of low virulence, and are rarely found as the sole causative organism in arthritis/tenosynovitis of chickens (Hussain, Spradbrow, and Mackenzie 1981; Meanger et al. 1997). Reovirus infection is not an OIE-listed disease.

Agent taxonomy

Avian reoviruses are non-enveloped double-stranded RNA viruses, belonging to the genus *Orthoreovirus*, family *Reoviridae*. Eleven serotypes and many subtypes of avian reoviruses exist, only some of which are pathogenic for poultry (Saifuddin et al. 1989). Most reovirus isolations in the United States and most American vaccine strains belong to the standard S1133 serotype (van der Heide 1996). Australian strains appear to have evolved separately from United States strains, with variation in the nucleotide sequences between United States and Australian strains. The serological relationship between United States and Australian strains is unknown (Liu, Giambrone, and Nielsen 1997). In Europe and Israel, variant serotypes of reovirus exist, which are different from United States strains and are not neutralised by the S1133 antiserum (van der Heide 2000).

Agent Characteristics

Avian reoviruses are stable at pH 3 to 9, and at normal ambient temperatures. Reoviruses are stable for up to two months at room temperature (Robertson and Wilcox 1986), more than three years at 4 ºC, and over four years at –20 ºC (Rosenberger 2003). Reoviruses are heat resistant, and are able to withstand 60 ºC for eight to ten hours (Rosenberger 2003). They are relatively resistant to disinfectants such as 2% formaldehyde at 4 ºC and 2% phenol at room temperature, but sensitive to 100% ethyl alcohol and to chlorine disinfectants (Robertson and Wilcox 1986).

Avian reoviruses have been reported to persist for at least 10 days on the surface of egg shells when organic material is present, at least 10 days on feathers, wood shavings, chicken feed, metal, glass and rubber, and for at least 10 weeks in water (Savage and Jones 2003; Jones 2000).

Epidemiology

Avian reoviruses have been associated with various disease syndromes in chickens, including in Australia, although disease produced in Australian chickens appears less severe than in overseas flocks. Meanger et al. (1997) stated,

'avian reoviruses have frequently been isolated from lesions of tenosynovitis in meat-type chickens but attempts to experimentally reproduce the lesions by infection of chickens with the avian reovirus isolates have, in general, been unsuccessful. The role of avian reoviruses in the production of tenosynovitis in Australia therefore remains uncertain'.

Disease occurs predominantly in meat chickens, although layer breeds can be affected (Schwartz, Gentry, and Rothenbacher 1976; Robertson and Wilcox 1986). Reoviruses have also been isolated from turkeys with tenosynovitis, from Muscovy ducks with high morbidity and mortality, African Grey parrots with necrotic lesions in the liver, normal mallard ducks, pigeons with diarrhoea and American woodcocks with generalised infection and emaciation (McNulty 1993; Jones 2000). In many cases, the role of the reovirus in the disease condition has not been determined, although in some the isolated reovirus has been capable of causing tenosynovitis when experimentally inoculated into chicks (Jones 2000). It appears that avian reoviruses can be transmitted between avian species, but it is not clear whether wild birds serve as reservoirs of infection for poultry (Jones 2000). Chickens and turkeys are the only recognised natural hosts for reovirus-induced arthritis (Rosenberger 2003).

Avian reoviruses have worldwide distribution, with the importance of infections depending, at least partly, on the presence and prevalence of pathogenic strains (Jones 2000). Australian strains appear to be of low virulence in comparison with reovirus strains causing arthritis/tenosynovitis in North America and Europe (Meanger et al. 1997). No markers have been found for pathogenicity or tissue tropism, and experimental infection of SPF chicks with reovirus is required to determine these characteristics of a particular isolate (Jones 2000).

While horizontal faecal-oral transmission is the predominant means of virus dissemination, vertical transmission of reovirus does occur. In addition to faecal-oral transmission, infection can occur via the respiratory tract or through broken skin of the feet (Jones 2000). The rate of experimental transovarial transmission varied from less than 2% in one experimental study (Menendez, Calnek, and Cowen 1975a), to 33% in another (Deshmukh and Pomeroy 1969), but is probably low in nature, resulting in a small infected nucleus of chicks from which virus is transmitted to other birds (Al-Muffarej, Savage, and Jones 1996; Jones 2000).

Chickens are susceptible to reovirus arthritis/tenosynovitis infection from one day old, and develop resistance to clinical disease with age. The older the birds at the time of infection, the shorter was the duration of viral persistence in the joints (Jones and Georgiou 1984b), and the shorter the duration of faecal shedding of virus (Jones and Georgiou 1984a). Chickens infected earlier in life may develop more severe joint lesions than those infected when older (Jones 2000). Chickens inoculated with reovirus at five months of age by nasal, tracheal and oesophageal routes showed no clinical signs, but virus was detected in the tendons, oviduct and intestinal tract of some birds 14–15 days later (Menendez, Calnek, and Cowen 1975b).

In experimental studies, chickens inoculated with high doses of virus developed clinical signs within three days (Gouvea and Schnitzer 1982). While infection most probably occurs before or soon after hatching in natural infections, severe clinical lesions may not occur until five to seven weeks later (Kibenge and Wilcox 1983). In an experimental study using chickens inoculated by the footpad, horizontal transmission of reovirus to uninoculated chicks occurred

within a week, but signs of tenosynovitis did not develop until six to seven weeks later (Jones and Onunkwo 1978).

Reovirus has rarely been isolated as the sole causative organism of arthritis/tenosynovitis in Australian chicken flocks, with most documented cases having multiple infections involving *Staphylococcus aureus* and/or other organisms in synovitis lesions (Mackenzie and Bains 1976; Mackenzie and Bains 1977; Hussain, Spradbrow, and Mackenzie 1981; Kibenge et al. 1982). Attempts to reproduce viral arthritis/tenosynovitis by experimental inoculation of Australian strains were complicated by the low virulence of the strains, with transient synovitis being induced only after injection of very high doses of concentrated virus into the footpad (Meanger et al. 1997; G. Wilcox, Murdoch University, pers. comm. December 2002). This is in contrast to experimental studies using overseas strains, with which arthritis/tenosynovitis lesions are readily induced, using various routes of inoculation.

In a study on prevalence of reoviruses in commercial chickens in Australia, all 30 adult meat-breeder chicken flocks examined showed serological evidence of previous exposure to reovirus, in the absence of vaccination. Reoviruses were isolated from tissues and rectal contents of chickens showing runting/stunting syndrome and tenosynovitis, and from the rectal contents of six flocks of normal three-week-old chickens. Within-flock prevalence varied from 50–100%. The results indicated that infection with reoviruses is widespread, and isolation of virus does not necessarily imply any aetiological relationship with disease (Robertson, Wilcox, and Kibenge 1984).

Avian reovirus infection is widespread in overseas flocks; however, the prevalence of pathogenic strains varies from region to region (Jones 2000). Viral arthritis/tenosynovitis is an important cause of production losses in Europe and North America, and as a result, vaccination of meat-chickens and breeders is commonly practised (Meanger et al. 1997). The prevalence of pathogenic strains is unknown.

Clinical Signs

Several clinical syndromes have been associated with reovirus infection in poultry. Of these, only the viral arthritis/tenosynovitis syndrome has a proven causal association with reovirus infection (Jones 2000).

Clinical signs, after oral inoculation at one-day-old, included depression at three to six days after inoculation, and lameness and reluctance to move 6–8 weeks later. Less than half the infected birds showed clinical signs (Jones and Georgiou 1984b). Leg swellings occurred above and below the hock joint at four to seven weeks. In natural infections, lameness and stunting occur between three and eight weeks, and enlargement of the hock region may be observed at slaughter (Rosenberger 2003).

Morbidity is usually below 10% (McNulty 1993), but may be as high as 100%, while mortality is less than 6% (Rosenberger 2003). Commercial white leghorn layers with clinical tenosynovitis had production losses of 15–20% and mortality of 5% (Schwartz, Gentry, and Rothenbacher 1976).

Reoviruses have been implicated in the malabsorption syndrome, also known as runting/stunting syndrome, although they may occur in combination with other organisms and may, therefore, not be the sole cause of the clinical signs. Affected birds show reduction in growth rates of 5–20%, elevated feed conversion ratios, lameness, diarrhoea and feathering defects (Robertson and Wilcox 1986).

Other disease syndromes, including sudden deaths with hydropericardium, hepatitis, cloacal pasting, and respiratory disease have been reported, but the association between these conditions and reovirus is not certain (Jones 1976; Robertson and Wilcox 1986).

Pathogenesis

Following experimental oral inoculation of day-old SPF chickens, virus was isolated from the blood within 30 hours, and was widely distributed within 24–48 hours to other organs, including bursa of Fabricius, liver, spleen, pancreas, heart, kidney and hock joints (McNulty 1993; Jones, Islam, and Kelly 1989). Inoculation of adult (five-month-old) chickens resulted in virus dissemination in virtually all organ systems, including respiratory, digestive, reproductive and musculoskeletal systems. Virus was detectable at four days post-inoculation and persisted in the tendons, oviduct and intestinal tract for at least 15 days (Menendez, Calnek, and Cowen 1975b). Virus appears to persist for long periods in these tissues after replication in the digestive tract has ceased, and has been isolated from tendon sheaths and hock joints for more than six months after experimental infection (Olson and Kerr 1967). Virus can be isolated from cloacal swabs for 14–16 weeks from both chickens inoculated with reovirus and in-contact control (not inoculated) chickens (Robertson and Wilcox 1986).

Pathology

Lesions of viral arthritis/tenosynovitis include swellings of the digital flexor and extensor tendons and sheaths, and swelling around the hock (tibiotarsal) joints. Thickening and adhesions of the tendons and surrounding tissues, excess clear or turbid fluid in the joints, erosions in the articular cartilage of affected joints, and occasionally rupture of the gastrocnemius and other tendons may be seen on post-mortem examination (Jones 2000).

Other gross pathological changes described include feather abnormalities, pericarditis, hepatomegaly and splenomegaly (McNulty 1993). Microscopic lesions of hepatic necrosis, pericarditis, bursal atrophy, myocarditis and thymitis have been described after experimental infection with highly pathogenic reovirus (McNulty 1993).

Immunology

Circulating antibodies can be detected in the sera of infected birds using AGID, indirect immunofluorescence, ELISA and virus neutralisation. Only the latter differentiates between antigenically different strains of reovirus (Jones 2000).

Maternally derived antibodies may protect chicks against infection with reovirus, if present at a sufficiently high titre (Takase, Fujikawa, and Yamada 1996). Vaccination of breeder birds is the most commonly used means of preventing reovirus infection in chicks overseas, although vaccination of day-old chicks with a live vaccine has also been used to provide early active immunity (Jones 2000). Breeder birds are generally vaccinated with a live vaccine early in life, followed by two or more inactivated vaccines prior to point of lay.

Vaccination of breeder birds does not necessarily prevent infection of progeny chicks. In an experimental study, one-day-old progeny chicks of vaccinated parents were challenged with a virulent reovirus. Although vaccination reduced the incidence and severity of tenosynovitis lesions, challenge virus was isolated from the hock joints in nine out of 12 of the progeny chicks (Jones and Nwajei 1985). Furthermore, vaccination of one-day-old SPF chickens with an attenuated live virus resulted in systemic infection, with vaccine virus localising in the joints

(Gouvea and Schnitzer 1982). Vaccination of birds against one antigenic type of reovirus will not necessarily confer protection against heterologous antigenic types (Meanger et al. 1997).

Vaccination against avian reovirus is not currently practised in Australia. There are no avian reovirus vaccines registered for use in Australia, nor are any requests for vaccine registration pending (Leigh Nind, Australian Pesticides and Veterinary Medicines Authority, pers. comm. March 2002).

Diagnosis

Clinical cases of viral arthritis/tenosynovitis must be differentiated from *Mycoplasma synoviae* infection, bacterial arthritis, and lameness resulting from anatomical abnormalities (Rosenberger, Olson, and van der Heide 1998).

Because of the high prevalence of subclinical and clinical reovirus infections in poultry, serology is not generally useful for diagnosis, which is best achieved using virus isolation (McNulty 1993). However, serology may be used to monitor the status of SPF flocks, or antibody levels in vaccinated breeder flocks, and commercial ELISA tests are now available for this purpose. Virus isolation can be attempted from various organs, with the most reliable sites being the subtarsal sesamoid bone and associated tendons, and the hock articular cartilage (Jones and Georgiou 1984b). Virus can be isolated in embryos or cell cultures, with confirmation by detection of reovirus-specific antigens (Rosenberger, Olson, and van der Heide 1998). Reovirus, isolated from the hock joints and tendon sheaths, is diagnostic for viral arthritis/tenosynovitis; however, reovirus isolations from other organs or intestinal contents may not be significant as they may represent isolation of non-pathogenic strains (McNulty 1993). Virus can also be demonstrated in tissues with the use of PCR, direct immunofluorescence staining and other techniques, but these tools are not likely to be as useful as virus isolation in the diagnosis of disease in the field (Jones 2000).

Transmission in chicken meat

Viral arthritis/tenosynovitis is predominantly a disease of meat chickens, with infection occurring early in life, and disease manifesting itself at around slaughter-age. The virus persists in synovial structures, predominantly around the hock region, but it can also be isolated from the femoral head, in the absence of grossly detectable lesions of the hip joint (Jones and Georgiou 1984b). The titre of reovirus in the affected synovial structures of chickens is as high as $10^{4.5}$ CID_{50}/g of tissue two weeks after inoculation of day-old chicks, with the titre declining over a number of weeks (Jones and Georgiou 1984b). Vaccination of parent birds may not prevent infection and localisation of field virus in synovial structures of meat chickens. Vaccination of young birds with live vaccines may result in vaccine virus persisting in joints of the carcass. Reovirus could be reisolated from the legs of experimentally infected chickens 285 days post-inoculation and from the spleen for 235 days (Olson and Kerr 1967).

Reovirus replicates in the intestinal tract, where it may persist for weeks to months (Robertson and Wilcox 1986), and the virus is also distributed to liver and heart.

This suggests that viable reovirus could be present in chicken carcasses, with or without faecal contamination during processing.

Quarantine significance

Reovirus is not an OIE-listed disease agent.

Reovirus is not notifiable in any State or Territory of Australia, and is not subject to official controls within Australia. Reovirus is not included in the Emergency Animal Disease Response Agreement, has no impacts on human health or the environment, and no national socio-economic effects.

Some strains of avian reovirus are endemic in Australian poultry. The low quarantine significance afforded to this organism, together with the difficulty in determining that exotic strains are more virulent than endemic Australian strains, and the fact that 85–90% of reovirus strains are considered to be non-pathogenic, making it difficult to accurately target risk management efforts at particular pathogenic types, resulted in the IRA team concluding that no further risk assessment is necessary for this disease agent.

Direct impact on human life or health

Avian reovirus is not known to affect humans and is not considered to be a threat to public health.

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Technical Information

Background

Avian Metapneumovirus (AMPV) is a significant respiratory pathogen in both turkey and chicken flocks, causing serious economic losses in birds of any age. In turkeys, initial infection of the upper respiratory tract with AMPV is frequently complicated by secondary infections with *E. coli*, infectious bronchitis virus, or *Mycoplasma* spp. The disease caused by AMPV in turkeys is known as turkey rhinotracheitis or turkey coryza. AMPV also infects chickens and is associated with swollen head syndrome in meat chickens and breeders (Cook 2000).

AMPV has not been isolated from avian species in Australia. AMPV was included as an OIE-listed disease agent in 2005, on the basis that 'there has been proven international spread and the disease has significant morbidity and mortality' (World Organisation for Animal Health (OIE) 2005a).

Agent taxonomy

AMPV is the type species for the genus *Metapneumovirus* (subfamily *Pneumovirinae* of family *Paramyxoviridae*). AMPV strains may have significant antigenic diversity, shown by reactions with monoclonal antibody panels. Based on virus neutralisation and sequence analysis, at least four antigenically different subgroups have been described (Toquin et al. 2000), tentatively designated type A, B, C (also known as the Colorado strain) and D (or non-A, non-B strain). AMPV isolates from different geographical locations may show marked variation in serological tests (Toquin et al. 2000) and are also reported to show different species tropism (Cook, Kinloch, and Ellis 1993).

Agent characteristics

AMPV is an enveloped RNA virus and is destroyed rapidly outside the host. The virus remained infective for three days in inoculated turkey litter kept at room temperature, and up to a month when kept at 8 ºC (Velayudhan et al. 2003). The resistance of AMPV to physical and chemical action (Townsend et al. 2000) is shown below. The level of inactivation achieved depends on the concentration of the inactivating agent, the length of time of exposure, the titre of the virus and the nature of the suspending medium (Table 102). A cell culture-grown preparation of AMPV fluid was not inactivated by freezing at –20 °C, and there was no loss of infectivity after multiple cycles of freezing and thawing (Townsend et al. 2000).

Epidemiology

AMPV occurs in most countries of the world, although reports are frequently based only on serological evidence. Published reports indicate that Canada (Heckert and Meyers 1993) and Australia (Bell and Alexander 1990) are free of AMPV. However, antibodies to AMPV have reportedly been detected in commercial turkey flocks from Canada (Njenga, Lwamba, and Seal 2003).

Table 102. Resistance of agent to physical and chemical action

Temperature:	Inactivated by heat: six hours at 50 °C; 72 hours at 37 °C; <4 week at 20 °C; <26 weeks at 4 °C
pH:	Inactivated by extremes of pH (<3 and >10)
Chemicals:	Inactivated by chloroform, ethyl alcohol
Disinfectants:	Inactivated by sodium hypochlorite, quaternary ammonium compounds, iodophors, phenol derivatives, chlorhexidine
Survival:	Sensitive to desiccation

(Townsend et al. 2000)

AMPV is highly infectious, and spreads rapidly once it is introduced into susceptible flocks. Where birds are housed in close proximity, AMPV spreads rapidly, but spread is slower over greater distances. Given the nature of the infection, transmission is likely to be airborne, although faecal shedding may occur. Transmission by contaminated water, movement of infected birds, and fomites (personnel, vehicles, egg trays) is also possible, although at present only contact spread has been confirmed (Gough 2003). In some countries where AMPV infection has appeared as a new disease, it has spread rapidly (Gough 2003). In the United Kingdom, the disease was widespread within nine weeks of the first reported outbreak in turkeys (Alexander 1993). However, spread of AMPV in North America has been slower than that described in the United Kingdom (Bennett et al. 2004).

AMPV can be isolated for only a short time in the infected bird, although detection of viral RNA in cloacal and pharyngeal swabs of experimentally-infected chickens can be achieved for several weeks (Hess et al. 2004; Hess et al. 2004). Field evidence suggests that transmission of AMPV through the egg (either transovarially or by egg contamination) is unlikely to occur (Cook 2000). However, simultaneous AMPV infections in neonatal turkey flocks were recently reported in three separate states of the United States. A common parent breeder source flock was determined for some of the affected flocks, raising the possibility that the infections were egg-transmitted (Shin et al. 2002). AMPV has been transmitted experimentally by contact, or by intranasal, ocular or oculonasal inoculation, and there are reports of transmission via the oral route (Shin et al. 2001; Nagaraja et al. 2002).

AMPV replicates in guinea fowl, pheasants and ducks (Gough et al. 1988) and it has been suggested that ducks may act as non-clinical carriers of AMPV (Shin et al. 2002; Shin et al. 2001). Muscovy ducks are susceptible to experimental infection (Toquin et al. 1999), but geese and pigeons are apparently refractory (Gough et al. 1988). AMPV antibodies have been reported in ostrich (Cadman et al. 1994) and gulls (Heffels-Redmann et al. 1998), and AMPV RNA has been demonstrated in wild geese, sparrows, swallows, starlings and Mallard ducks, suggesting that wild birds may be involved in the circulation of AMPV (Shin et al. 2000; Shin et al. 2002; Bennett et al. 2004). However, there have been no confirmed reports of spread of AMPV by wild birds, and wild birds testing positive for AMPV RNA have shown no clinical signs of disease (Bennett et al. 2004). There are no reports of natural or experimental infection of non-avian species with AMPV.

Reports of the seroprevalence of AMPV in poultry suggest that infection is common and widespread. In the United Kingdom, 20 of 99 meat chicken flocks were seropositive at the time of slaughter (Pattison et al. 1989), and in Japan, analysis of 4,111 serological samples taken from 137 farms in 36 prefectures over the period from 1985 to 1995 showed that prevalence in chickens increased from 2.2% in 1988 to 54.2% in 1995 (Tanaka et al. 1996).

Clinical signs

Infection in chickens is not always associated with clinical signs (Cook 2000; Gough 2003). Swollen head syndrome has been described in three- to five-week-old meat chickens (Gough et al. 1994) and in older meat chicken breeders, but not in commercial egg laying flocks to any extent (Naylor and Jones 1993). In chickens, swollen head syndrome is characterised by respiratory disease, apathy, swelling of infraorbital sinuses and facial swelling. These signs may be followed by cerebral disorientation, torticollis (twisted neck) and opisthotonus (arching of the neck). The incubation period in experimentally infected three-week-old chicks was three to six days (Majó et al. 1995). The morbidity is generally low (around 10%), and mortality also low (1–2%). Mortality of up to 20% has been reported in flocks with concurrent bacterial infection (Morley and Thomson 1984). Although reduced egg production and changes in egg shell quality were described in laying chickens infected with AMPV intravenously, egg production and quality were unaffected in hens infected by the oculonasal route (Cook 2000).

Infection in turkeys may occur from a very young age. There is a rapid onset of clinical signs, and sneezing, tracheal rales, nasal and ocular discharge, conjunctivitis and swollen infraorbital sinuses, submandibular oedema, coughing and head shaking have been described in infected birds. The incubation period in experimentally infected four-week-old poults was two to three days (Jirjis et al. 2002). In uncomplicated infection, affected birds generally recover in 10 to 14 days. Respiratory infection is generally less severe in laying turkeys but reductions in egg production of up to 70% have been reported. AMPV may cause significant reductions in egg quality, and an increased incidence of thin-shelled eggs has been reported. Affected birds recover in approximately three weeks. Morbidity is generally very high (up to 100%), while mortality is variable (0.4 to 50%). Mortality is generally highest in young poults (Gough 2003). In some adult flocks, seroconversion has been reported in the absence of clinical signs.

Infection with AMPV predisposes birds to secondary infection, which may produce more serious disease and higher mortality (Cook 2000).

Pathogenesis

Replication of AMPV in growing turkeys appears to be limited to the respiratory tract, and is generally of short duration (up to 10 days post-inoculation). A recent report studied the tissue distribution of a virulent isolate of AMPV in turkeys. Viral RNA could be detected for 14 days post-inoculation from nasal turbinates, and for nine days from trachea. Viral RNA was not detected in lungs, liver or spleen (Velayudhan et al. 2005). AMPV has been isolated from the vagina and magnum of turkeys, and viral antigen was detected in uterine epithelium and the oviduct. However, replication of AMPV in oviduct could not be demonstrated (Jones et al. 1988).

AMPV has been demonstrated in respiratory tract tissues, including nasal tissue, sinus, trachea and lung of inoculated chickens, but only for a short time after infection (Catelli et al. 1998). Virus was rarely found after six days post inoculation, and was not recovered from non-respiratory tissues at any time (Catelli et al. 1998). In another study, intestinal samples were

positive for the presence of viral RNA by PCR for up to nine days post-inoculation (Shin et al. 2000). AMPV antigen was demonstrated in the reproductive tract of adult hens after intravenous inoculation in one study (Cook et al. 2000), but not in another (Kherna and Jones 1999). Using explants of oviduct tissue, the oviduct was found to be susceptible to infection, but virus replication could not be demonstrated *in vivo* (Kherna and Jones 1999).

Pathology

In chickens, the only significant lesions are those associated with swollen head syndrome, although histological lesions may be detected in the trachea and lungs. When infection is complicated by co-infection with bacteria and other viruses, pneumonia and airsacculitis may be present (Gough et al. 1994). In naturally-occurring infections in turkeys, complicated by secondary infections, post-mortem findings include airsacculitis, pericarditis, pneumonia and perihepatitis (Gough 2003).

Immunology

Live attenuated and inactivated vaccines are available to control AMPV infections in turkeys and chickens. Live vaccines are used to control infections in growing turkeys and meat chickens, and to prime future layers and breeders before the injection of inactivated vaccine prior to onset of lay. The duration of immunity following vaccination is at least 14 to 22 weeks. Vaccination appeared to reduce or prevent virus excretion in vaccinated, challenged laying chickens (Hess et al. 2004). Maternal immunity does not appear to protect against infection (Catelli et al. 1998), or prevent successful immunisation. Good cross protection has been shown between the A and B subgroup strains (Cook et al. 1995).

Diagnosis

In the chicken, diagnosis based only on clinical signs is unreliable. Similar respiratory signs are observed in other diseases of chickens such as Newcastle disease, infectious bronchitis, avian influenza and infections with *Mycoplasma* spp. Virus isolation and identification, the detection of AMPV using PCR and the demonstration of specific antibodies in acute and convalescent serum samples will confirm the diagnosis. In both chickens and turkeys, serology is the most common method of diagnosis (Cook 2000).

AMPV in infective mucus, nasal secretions or sinus scrapings may be isolated in chicken or turkey embryo tracheal organ culture, or in embryonated eggs inoculated via the yolk sac. Due to the short duration of virus shedding, isolation should be attempted at the first sign of clinical disease. Embryo mortality will occur after four or five passages, but virus is generally of low titre. Cell culture is not recommended for primary isolation of AMPV but after isolation, AMPV may be grown in chick embryo fibroblasts, chick embryo liver cells or Vero cells (Gough, Alexander, and Wyeth 1998). The pathogenicity of AMPV isolates is difficult to assess in the laboratory. Differentiation of vaccine strains and virulent field virus of AMPV is also difficult (Gough, Alexander, and Wyeth 1998). Viral RNA can be detected in infected birds for some time after isolation of virus is no longer possible.

Several commercial ELISA kits for AMPV are available but differences in sensitivity and specificity between tests have been reported (Gough, Alexander, and Wyeth 1998). ELISAs are very useful tests for disease surveillance, but in situations where all known AMPV types are to be detected with high sensitivity, more than one type of ELISA should be used (Cook 2000).

Transmission in chicken meat

There is no data on the survival of AMPV in chicken or poultry carcasses. Viral replication occurs principally in tissues of the upper respiratory tract, which are generally removed from chicken carcasses at slaughter. However, contamination of the carcass could occur during processing, and some remnants of respiratory tissue may remain in the carcass after evisceration.

Quarantine significance

AMPV was added to the OIE list of notifiable disease agents in 2005 (World Organisation for Animal Health (OIE) 2005b). However, AMPV is not subject to official controls within Australia and is notifiable only in South Australia. AMPV is not included in the Emergency Animal Disease Response Agreement. Experience from the United Kingdom suggests that spread of infection may be rapid once introduced (Alexander 1993), and consequences within an individual flock may be serious if infection was introduced to a fully susceptible population (Cook 2000).

Risk Assessment

Release Assessment

Rel_1: Selection of source flock (between flock prevalence)

There are few reports on the prevalence of AMPV infection in meat chicken flocks. One study reported seroprevalence of around 20% (Pattison et al. 1989). For an unrestricted risk estimate, it was assumed that the prevalence is at the highest sustainable level in an endemically infected country or zone (see Method of Risk Assessment). The likelihood that a source flock will be infected with AMPV at slaughter age was assessed by the IRA team as ***low***.

Rel_2: Infection detected through flock surveillance and the flock withdrawn from slaughter

AMPV infection in chickens may not be associated with clinical signs. The IRA team considered that the likelihood that an infected flock will be detected through routine flock surveillance and the flock withheld from slaughter was ***extremely low***.

Rel_3: Selection of an infected chicken from an infected flock (within flock prevalence)

AMPV is highly infectious, and spreads rapidly once it is introduced into susceptible flocks, especially where birds are housed in close proximity. Therefore, in an outbreak, many birds within a shed are likely to be infected. If an affected flock were sent to slaughter, the IRA team considered that the likelihood that a selected individual chicken will be infected was ***high***.

Rel_4: Background cross-contamination rate

This likelihood represents the background cross-contamination rate during slaughter and processing and applies to cross-contamination with material, infectious or otherwise, from other carcasses, including those tissues or materials in which the disease agent tends to localise.

Replication of AMPV appears to be limited to the upper respiratory tract, and is generally of short duration. In addition, the head of the bird is removed during processing. The IRA team considered that the likelihood of a carcass being contaminated with potentially contaminated material from other birds was ***very low***.

Rel_5: Likelihood that an uninfected carcass will be contaminated with the disease agent during slaughter and processing

As discussed in the Method for Risk Assessment (page 58, Part B), Rel_4 was used to calculate Rel_{5a} (the likelihood that an uninfected carcass from an infected flock will become contaminated during slaughter and processing) and Rel_{5b} (the likelihood that an uninfected carcass from an uninfected flock will become contaminated during slaughter and processing).

For AMPV, Rel_{5a} was calculated as ***very low***, and Rel_{5b} was calculated as ***very low***.

Rel_6: The likelihood that the carcass of a bird that was *infected* before slaughter will be removed as a result of inspections before or during processing

Lesions of AMPV infection appear to be restricted to tissues of the upper respiratory tract, and these are generally removed from carcasses at slaughter. The IRA team considered that the rejection rate of infected carcasses would be greater than the background rejection rate but still very low. The likelihood that a contaminated/infected carcass will be removed during processing inspections was assessed by the IRA team as ***very low***.

Rel_7: The likelihood that the carcass of a bird that was *not infected* before slaughter will be removed as a result of inspections before or during processing

The likelihood that an uncontaminated/uninfected carcass will be removed during processing inspections was considered to be equal to the background rejection rate of 0.75% as described in the Method section (page 59, Part B).

Rel_8: Inactivation of the agent during further processing, storage, handling and transport

Given that the carcasses for importation will be chilled or frozen, and that the virus can persist for weeks at low temperatures, the IRA team considered that the likelihood of inactivation of the virus during further processing, storage, handling and transport was ***extremely low***.

Conclusions – Release assessment

After inserting the above estimates into the simulation model, and using the outputs from the model as a guide, the IRA team concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a ***low*** likelihood that imported chicken meat would be infected or contaminated with AMPV.

Exposure assessment

Exposure Group 1: Wild birds

Of the steps identified as determinants in the infection of wild birds by exposure to scraps from an imported contaminated chicken carcass, $WB_{agentsurvival}$ and $WB_{infectivedose}$ are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$WB_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by a wild bird

There are no data on the survival of AMPV in carcasses; however the virus can remain infective in inoculated turkey litter at room temperature for three days. This likelihood was assessed by the IRA team as ***moderate.***

$WB_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a wild bird is sufficient to produce infection

Serological evidence of AMPV infection has been demonstrated in gulls, and AMPV RNA has been demonstrated in a number of species of wild birds. However, virus has not been isolated from these species. The low, transient viraemia and restricted tissue distribution of virus in the live bird suggest that AMPV is unlikely to be present in high quantities in the majority of chicken meat scraps. The IRA team considered that there was an ***extremely low*** likelihood that AMPV would infect a wild bird consuming the contaminated meat scraps.

Exposure Group 2: Low biosecurity poultry

Of the steps identified as determinants in the infection of low biosecurity poultry by exposure to material from an imported contaminated chicken carcass, $BP_{agentsurvival}$, $BP_{infectivedose}$, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$BP_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by low biosecurity poultry

The time between feeding of scraps and consumption by low biosecurity poultry is likely to be very short, so environmental degradation of the disease agent will be minimal. The likelihood that the agent will remain viable was assessed by the IRA team as ***certain (=1)***.

$BP_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a low biosecurity bird is sufficient to produce infection

Transmission of infection via the oral route has been demonstrated, but the likelihood that a carcass would be contaminated with AMPV was considered low, given that the organism has respiratory tissue tropism, and the majority of the respiratory tract is removed during processing. When these factors were combined the IRA team considered that there was an ***extremely low*** likelihood that AMPV would infect a low biosecurity bird that consumed the contaminated imported chicken meat scraps.

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

The likelihood that AMPV would survive the rendering process was negligible. The IRA team considered that the likelihood the product would be re-contaminated with AMPV post-processing was negligible. Therefore the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with AMPV was estimated by the IRA team to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

Given that AMPV would be destroyed by rendering as discussed above, and that feed derived from contaminated carcasses would be diluted with feed from non-risk material, the IRA team considere that the likelihood that the amount of final poultry ration eaten by a bird would contain an oral infectious dose of virus was ***negligible***.

Exposure Group 3: Medium biosecurity commercial poultry

Of the steps identified as determinants in the infection of medium biosecurity commercial poultry by exposure to material from an imported contaminated chicken carcass, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that poultry feed would be contaminated with AMPV was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that the final poultry ration would contain an oral infectious dose of AMPV was considered by the IRA team to be ***negligible***.

Exposure Group 4: Non-avian species

As discussed above, $NAS_{agentsurvival}$ was considered to be equal to $WB_{agentsurvival}$.

There are no reports of natural or experimental infection of non-avian species with AMPV. Therefore this exposure group was not considered further in relation to this disease. $NAS_{infectivedose}$ was set to a value of zero.

Conclusions – Exposure assessment

Using the simulation model as a guide, the partial likelihood of exposure for each of the exposure groups was determined, taking the above estimates of the exposure variables into account. A summary of the outcomes determined by the IRA team is set out in Table 103.

Table 103. Partial likelihoods of exposure (PLE)

Exposure group	PLE
Wild birds	Negligible
Low biosecurity poultry	Extremely low
Medium biosecurity poultry	Negligible
Non-avian species	Zero

Consequence assessment

Establishment and spread was considered in the context of the outbreak scenarios, described in the Method of Risk Assessment.

Estimating the likelihood of each outbreak scenario

The partial likelihood of establishment and spread (PLES) of AMPV for the different exposure groups is described below. Four outbreak scenarios were considered relevant:

1. Disease agent does not establish or is not recognised within the directly exposed population

2. Disease agent establishes within the directly exposed population, is identified and is eliminated by human efforts or by natural means

3. Disease agent establishes in the directly exposed population, spreads within a district/region, including into other exposure groups if applicable, and is eliminated by human action or by natural means

4. Disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia.

Wild birds

AMPV infection has been demonstrated in a number of avian species, and antibodies have been demonstrated in gulls. However, outbreaks of disease associated with AMPV have not been reported in wild birds, and birds with positive tests for AMPV RNA have shown no clinical signs of infection (Bennett et al. 2004). The IRA team considered that the most likely outcome of infection of a wild bird resulting from scavenging chicken meat scraps would be a single or a few isolated occurrences of infection in wild birds. It has been postulated that wild birds may be involved in the circulation of AMPV (Shin et al. 2002). However, there have been no reports of spread of AMPV by wild birds, and their role in transmission of this virus remains unclear (Bennett et al. 2004). In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 104).

Table 104. Estimated partial likelihood of establishment and spread (PLES) values for AMPV in wild birds

Outbreak scenario 1:	$PLES_{(wb)}1$	**High**
Outbreak scenario 2:	$PLES_{(wb)}2$	**Extremely low**
Outbreak scenario 3:	$PLES_{(wb)}3$	**Extremely low**
Outbreak scenario 4:	$PLES_{(wb)}4$	**Extremely low**

Low biosecurity poultry

Although this exposure group includes commercial free-range poultry and ratites, the IRA team considered that the sub-set of this group most likely to be exposed to scraps of imported chicken meat was small flocks of backyard poultry, because these are more likely to be directly fed table scraps. Moreover, turkeys represent a small proportion of the backyard poultry kept by households. In such flocks, there is less opportunity for the generation of high levels of environmental contamination, such as might occur with an outbreak of infectious disease in a large commercial flock. The most likely outcome of infection would be a single or a few isolated occurrences of infection.

If the disease were to establish in the flock, the level of expertise in disease recognition is likely to be low. Movement of infected birds and fomites (personnel, vehicles, egg trays) may facilitate spread of the virus beyond the initially infected flock before it is recognised and eradication measures are implemented. Once established, overseas experience suggests that infection might rapidly become widespread, at least in turkey flocks. AMPV infection is not notifiable in most states of Australia, nor subject to official control measures. In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. However, if spread of infection did occur, it is likely that it would become widespread. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 105).

Table 105. Estimated partial likelihood of establishment and spread (PLES) values for AMPV in low biosecurity poultry

Outbreak scenario 1:	$PLES_{(bp)}1$	**High**
Outbreak scenario 2:	$PLES_{(bp)}2$	**Low**
Outbreak scenario 3:	$PLES_{(bp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(bp)}4$	**Low**

Medium biosecurity commercial poultry

The IRA team considered that the most feasible route for exposure of medium biosecurity commercial poultry to imported carcass components would be through poultry feed containing inadequately rendered processing waste. The likelihood that pathogens would remain viable following rendering of contaminated imported carcasses and parts was assessed as negligible (page 322, Part C). Nevertheless, assessment of PLES was based on the assumption that

medium biosecurity commercial poultry had been exposed to poultry feed contaminated with the disease agent.

AMPV is highly infectious, and spread in and between susceptible flocks is rapid, especially where birds are housed in close proximity. Although higher levels of management expertise and formal flock health monitoring should ensure that an outbreak would be recognised sooner than in backyard poultry, diagnosis based only on clinical signs is unreliable. Overseas experience suggests that, once established, transmission between commercial flocks could be rapid and widespread. The IRA team considered early recognition and eradication of AMPV infection to be unlikely. In view of these factors, outbreak scenarios 1 and 4 are considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 106).

Table 106. Estimated partial likelihood of establishment and spread (PLES) values for AMPV in medium biosecurity commercial poultry

Outbreak scenario 1:	$PLES_{(cp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(cp)}2$	**Very low**
Outbreak scenario 3:	$PLES_{(cp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(cp)}4$	**Moderate**

Non-Avian Species

As discussed above, this exposure group was not considered further in relation to this disease.

Conclusion – Likelihood assessments

The estimates for the likelihood of release, the partial likelihoods of exposure for each of the exposure groups, and the partial likelihood of establishment and spread for each of the outbreaks scenarios were combined with the expected volume of trade using the simulation model. This allowed the calculation of partial annual likelihood of entry, exposure, establishment and spread for each of the outbreak scenarios. The results of this calculation are shown in Table 107.

Estimating the impacts associated with each outbreak scenario

For each outbreak scenario the direct and indirect impacts of AMPV infection were estimated at the national, State or Territory, district/region and local levels, as described in the Methods section (page 90-95, Part B).

The likelihood of AMPV affecting non-avian species (exposure group 4) was considered to be remote. Therefore, the impacts of AMPV occurring in this exposure group were not considered further.

Outbreak Scenario 1

By definition, outbreak scenario 1 means that the disease agent does not establish in the initially exposed population, or does not cause sufficient disease to lead to investigation and recognition of the infection. Therefore the impacts will be ***unlikely to be discernible*** for all exposure groups and all criteria.

Table 107. Partial annual likelihood of entry, exposure, establishment and spread (PALEEES) for the outbreak scenarios

Exposure group	Outbreak scenario	PALEEES
Wild birds	Scenario 1	High
	Scenario 2	Very low
Low biosecurity poultry	Scenario 1	High
	Scenario 2	High
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
Total all exposure groups	Scenario 3	High
Total all exposure groups	Scenario 4	High

Outbreak Scenario 2

The impacts of this outbreak scenario (disease agent establishes within the directly exposed population, and is identified and eliminated) will differ between exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

Disease in wild birds associated with AMPV infection has not been reported. Since wild birds do not play a significant part in production, direct economic loss from death of wild birds, were it to occur, is not measurable. Other impacts from the death of wild birds will be considered under direct criterion 2 and indirect criterion 4. The direct impacts of disease on wild birds were assessed as ***unlikely to be discernible***.

AMPV causes respiratory signs and decreased egg production in infected turkeys. An outbreak contained within a local population of low biosecurity poultry will result in losses to individual owners only. The impacts of such losses on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Coughing, sneezing, rales, sinusitis, conjunctivitis, depression and dyspnoea have been described in AMPV-infected turkeys. If an outbreak were to occur in medium biosecurity commercial poultry, especially turkeys, egg production losses, decreased fertility and hatchability may result in ***minor*** impacts at the local level. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

There are no reports of AMPV outbreaks in the wild. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at all levels for all exposure groups.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

AMPV is notifiable only in South Australia, and is not subject to official controls. The impacts of an outbreak of AMPV in wild birds on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

While no official action will be taken to eradicate the disease if AMPV were diagnosed in poultry flocks, individual growers will need to take some control action, such as eradication or vaccination. The impacts of an outbreak in low biosecurity poultry were assessed by the IRA team as ***unlikely to be discernible*** at any level. Impacts of such programs in medium biosecurity commercial poultry were assessed as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level when poultry industry action is taken to control the disease.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

The impacts of an outbreak of AMPV in wild birds, low biosecurity poultry or medium biosecurity commercial poultry on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

AMPV is recognised by the OIE as a disease of international trade significance because ‘there has been proven international spread and the disease has significant morbidity and mortality’. However, recognising that Australia’s export markets for poultry, especially turkey products, are small, the IRA team considered that the impacts for all exposure groups were ***unlikely to be discernible*** at all levels.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of an outbreak of AMPV in all exposure groups were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impacts of an outbreak of AMPV in wild birds were assessed by the IRA team as ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak in low biosecurity poultry were assessed as ***unlikely to be discernible*** at all levels.

If commercial turkey flocks were infected, temporary restrictions may be imposed on movement of birds, eggs, poultry products and people until the diagnosis was confirmed. This may lead to community disruption. The impacts of a disease outbreak in the medium biosecurity commercial poultry on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level.

Outbreak Scenario 3

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a district/region, and is eliminated) will be the same for all exposure groups, no matter which exposure group has been directly exposed to AMPV in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

Production losses in turkeys outlined above were assessed as ***unlikely to be discernible*** at national and State/Territory levels. Impacts at the district/region level were assessed by the IRA team as ***minor***.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

There have been no reports of AMPV outbreaks in the wild. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

It is possible that control and eradication programs may be instigated if AMPV was to spread to commercial turkey flocks. Inactivated vaccines are available overseas and may be introduced to control disease. The IRA team considered that the impacts were ***unlikely to be discernible*** at national State/Territory and district/region levels, and ***minor*** at the local level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

The impacts of an outbreak of AMPV in wild birds, low biosecurity poultry or medium biosecurity commercial poultry on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

AMPV is recognised by the OIE as a disease of international trade significance because 'there has been proven international spread and the disease has significant morbidity and mortality'. However, recognising that Australia's export markets for poultry, especially turkey products, are small, the IRA team conmsidered that the impacts for all exposure groups were ***unlikely to be discernible*** at all levels.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impact of an outbreak of AMPV was assessed as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Temporary restrictions may be imposed on movement of birds, eggs, poultry products and people until the diagnosis is confirmed. This may lead to community disruption. The impacts were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but may be ***minor*** at the local level.

Outbreak Scenario 4

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a State/Territory, and becomes endemic in Australia) will be the same for all exposure groups, no matter which exposure group has been directly exposed to AMPV in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

If the disease spreads more widely through medium biosecurity commercial poultry and low biosecurity poultry, losses of birds and production may be significant, especially if complicated by other infections. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, but ***minor*** at the district/region level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

There have been no reports of AMPV outbreaks in the wild. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

Control and eradication programs may be instigated if AMPV was to spread to commercial turkey flocks. Inactivated vaccines are available overseas and may be introduced to control disease. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, and ***minor*** at the district/region level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

The impacts of an outbreak of AMPV in wild birds, low biosecurity poultry or medium biosecurity commercial poultry on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

AMPV is recognised by the OIE as a disease of international trade significance because 'there has been proven international spread and the disease has significant morbidity and mortality'. However, recognising that Australia's export markets for poultry, especially turkey products, are small, impacts for all exposure groups were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of a generalised outbreak of AMPV on the environment were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Temporary restrictions may be imposed on movement of birds, eggs, poultry products and people until the diagnosis is confirmed. This may lead to community disruption. The impact of a general outbreak of AMPV on communities was assessed as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level.

Conclusions – impact assessment

The above estimates for each of the impact criteria were entered into the simulation model, and the overall impact of each outbreak scenario was estimated. Results are shown in Table 108.

Table 108. Impacts of each outbreak scenario

Exposure group	Outbreak scenario	Impact
Wild birds	Scenario 1	Negligible
	Scenario 2	Negligible
Low biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
	Total Scenario 3	Very low
	Total Scenario 4	Very low

Partial annual risk estimate for each outbreak scenario

The partial annual likelihood of entry, exposure, establishment and spread were combined with the impacts of the relevant outbreak scenarios to provide an estimate of the partial annual risk associated with each outbreak scenario. The outcomes of this process are shown in Table 109.

Table 109. Partial annual risk (PAR) of each outbreak scenario

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	High	Negligible	Negligible
	Scenario 2	Very low	Negligible	Negligible
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Medium biosecurity poultry	Scenario 1	Negligible	Negligible	Negligible
	Scenario 2	Negligible	Negligible	Negligible
Non-avian species	Scenario 1	Zero	Zero	Zero
	Scenario 2	Zero	Zero	Zero
	Total Scenario 3	High	Very low	Very low
	Total Scenario 4	High	Very low	Very low

Unrestricted risk estimate

The overall risk associated with the import of whole chicken carcasses was assessed as ***very low*** for AMPV. As this unrestricted risk estimate meets Australia's ALOP, no risk management was considered necessary.

Direct impact on human life or health

Avian metapneumovirus is not known to affect humans and is not considered to be a threat to public health.

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Technical Information

Background

Avian paramyxoviruses, other than APMV-1 (Newcastle disease virus), have received little attention due to their relatively low pathogenicity in domestic poultry and lower economic impact. Except where infection is complicated by the presence of other pathogens, avian paramyxovirus-2 (APMV-2) infection in poultry has been associated with inapparent or mild respiratory disease. Although infection has been reported in chickens, turkeys, and caged passerine and psittacine birds, the primary natural host appears to be small passerine birds (Alexander 1993). APMV-2 is also known as Yucaipa virus as it was first isolated in Yucaipa, California in 1956 (Bankowski, Corstvet, and Clark 1960).

APMV-2 has not been isolated from avian species in Australia. APMV-2 infection is not an OIE-listed disease.

Agent taxonomy

APMV-2 is a member of the genus *Avulavirus* of the family *Paramyxoviridae* (Buchen-Osmond 2002). There is considerable antigenic and structural diversity among APMV-2 isolates, and isolates have been classed into four groups based on their reactions with a panel of monoclonal antibodies (Ozdemir et al. 1990). APMV-2 is serologically distinct from Newcastle disease virus.

Agent characteristics

APMV-2 is an enveloped RNA virus and is therefore destroyed rapidly outside the host species. In general, paramyxoviruses are sensitive to thermal inactivation, lipid solvents and chlorine-based disinfectants. In the absence of any specific reports on the inactivation of APMV-2, it is assumed that APMV-2 has a similar spectrum of sensitivity to APMV-1.

Epidemiology

APMV-2 appears to have a worldwide distribution in various hosts. In poultry, APMV-2 has been isolated from chickens and/or turkeys in countries of North and Central America, Asia, the Middle East and Eastern and Western Europe (Alexander 2000). In countries where monitoring and surveillance is carried out, APMV-2 has been isolated from wild passerine birds, and from captive caged passerine and psittacine birds. APMV-2 has not been reported in poultry in Australia or New Zealand.

Reports of the prevalence of APMV-2 in poultry are few. In Spain, 14.7% (50 of 341 birds sampled) of layer hens on 43.7% (21 of 44 farms) of farms surveyed, and 39% (48 of 123 birds sampled) of meat chickens from 80% (4 of 5 farms) of farms surveyed were shown to be serologically positive for APMV-2 antibodies (Maldonado et al. 1994). However, these results may not represent the true prevalence since information on the study design is lacking. (Alexander 2003). Serological surveys of poultry in the United States and elsewhere indicate that this virus is widespread, and more common in turkeys than chickens (Alexander 2003).

There are few reports of studies of the transmission of APMV-2. However, since infection of poultry leads to shedding of APMV-2 from the respiratory and intestinal tracts, it is assumed that the methods of spread would be similar to APMV-1. In field infections APMV-2 spreads only slowly through the flock, and flock-to-flock transmission, even between flocks in close proximity, does not always occur (Alexander 1993).

It has been postulated that wild passerine birds are responsible for spread to other species, by contact or invasion of poultry houses (Ozdemir et al. 1990). APMV-2 has also been isolated from cattle egrets and mallard ducks during an epizootic of respiratory disease in turkeys in Israel (Lipkind et al. 1982) and it was suggested that these birds could be responsible for the spread of APMV-2.

Clinical signs

Where infection is uncomplicated by other infections, APMV-2 infection generally results in mild respiratory or inapparent disease (Bradshaw and Jensen 1979). Dyspnoea, ocular and nasal discharge, conjunctivitis and sinusitis have been reported in infected turkeys and chickens (Alexander 1993; Weisman, Malkinson, and Yuval 1999). Reduction in egg production and hatchability in turkeys and chickens may also occur.

The disease caused by APMV-2 is reported to be more severe in turkeys than in chickens, but may be significant in both species if complicated by other infections or environmental conditions. Severe respiratory disease, sinusitis, increased mortality and low egg production may result. Egg production losses with decreased hatchability and low poult yield have been reported but no effect on fertility has been reported (Alexander 2003). Mortality may be 5% to 50% (Lipkind et al. 1982).

High morbidity and mortality were reported in mixed infections with APMV-2 and APMV-3 (Weisman, Malkinson, and Yuval 1999).

Pathogenesis

There are no reports of studies on the pathogenesis of APMV-2.

Pathology

APMV-2 infection in poultry generally causes inapparent or mild respiratory disease. No specific lesions due to APMV-2 infection have been reported.

Immunology

In field infections of turkeys, seroconversion takes up to five weeks, and antibody titres remain low. In some cases, seroconversion occurs only in a proportion of the affected flock (Le Gros 1986).

Diagnosis

The samples taken and methods used to isolate APMV-2 are the same as those used for isolation of APMV-1. In addition, inoculation of six- to seven-day-old embryonated eggs via the yolk sac may be used.

Transmission in chicken meat

There is no data on the presence and survival of APMV-2 in carcasses. However, if it is assumed that the methods of spread of APMV-2 are similar to APMV-1, APMV-2 could contaminate the carcass via the airsacs, lungs and from the intestinal tract during evisceration of infected birds.

Quarantine significance

APMV-2 is not an OIE-listed disease agent.

APMV-2 infection is not notifiable in any State or Territory of Australia, and is not subject to official controls. APMV-2 is not included in the Emergency Animal Disease Response Agreement. Therefore, it is considered to be of relatively minor concern, and is unlikely to have significant consequences for the poultry industry that are discernible beyond the district/region level. There are no reports of outbreaks in the wild so there is little cause for concern for wild bird populations.

Risk Assessment

Release Assessment

Rel_1: Selection of source flock (between flock prevalence)

The primary natural host of APMV-2 appears to be small passerine birds. APMV-2 infection is reportedly widespread overseas in domestic turkeys, and to a lesser extent in chickens, but reliable prevalence data for meat chickens flocks are unavailable. The likelihood that a source flock will be infected with APMV-2 was assessed by the IRA team as ***low***.

Rel_2: Infection detected through flock surveillance and the flock withdrawn from slaughter

APMV-2 infection in chickens generally results in mild or inapparent respiratory disease. In an outbreak of APMV-2, clinical signs may not be apparent to the producer. The likelihood that a diseased flock will be detected through routine flock surveillance and the flock withheld from slaughter was assessed by the IRA team as ***extremely low***.

Rel_3: Selection of an infected chicken from an infected flock (within flock prevalence)

In field infections, APMV-2 appears to spread only slowly through the flock, and flock-to-flock transmission, even between flocks in close proximity, does not always occur. Therefore, the likelihood that a selected individual chicken will be infected was assessed by the IRA team as ***low***.

Rel_4: Background cross-contamination rate

This likelihood represents the background cross-contamination rate during slaughter and processing and applies to cross-contamination with material, infectious or otherwise, from other carcasses, including those tissues or materials in which the disease agent tends to localise.

Since it is assumed that the methods of spread of APMV-2 are similar to APMV-1, carcasses could be infected with the virus or become contaminated during processing. The IRA team considered that the likelihood of a carcass being contaminated with potentially contaminated material from other birds, especially from the digestive tract or the bursa, is ***moderate***.

Rel_5: Likelihood that an uninfected carcass will be contaminated with the disease agent during slaughter and processing

As discussed in the Method for Risk Assessment (page 58, Part B), Rel_4 was used to calculate Rel_{5a} (the likelihood that an uninfected carcass from an infected flock will become contaminated during slaughter and processing) and Rel_{5b} (the likelihood that an uninfected carcass from an uninfected flock will become contaminated during slaughter and processing).

For APMV-2, Rel_{5a} was calculated as ***low***, and Rel_{5b} was calculated as ***very low***.

Rel_6: The likelihood that the carcass of a bird that was *infected* before slaughter will be removed as a result of inspections before or during processing

APMV-2 infection in poultry generally causes inapparent or mild respiratory disease and no specific lesions due to APMV-2 infection have been reported. No obvious post-mortem lesions are likely to be present during slaughter and processing. The IRA team considered that the rejection rate of infected carcasses would be equal to the background rejection rate of 0.75%, as described in the Method section.

Rel_7: The likelihood that the carcass of a bird that was *not infected* before slaughter will be removed as a result of inspections before or during processing

The likelihood that an uncontaminated/uninfected carcass will be removed during processing inspections was considered to be equal to the background rejection rate of 0.75% as described in the Method section (page 59, Part B).

Rel_8: Inactivation of the agent during further processing, storage, handling and transport

In the absence of any specific reports on the inactivation of APMV-2, it was assumed that APMV-2 has a similar spectrum of sensitivity to APMV-1. Therefore, the likelihood of inactivation of the virus during further processing, storage, handling and transport was assessed by the IRA team as ***extremely low***.

Conclusions – Release assessment

After inserting the above estimates into the simulation model, and using the outputs from the model as a guide, the IRA team concluded that, in the absence of risk management and without considerations regarding the exporting country, there was a ***low*** likelihood that imported chicken meat would be infected or contaminated with APMV-2.

Exposure assessment

Exposure Group 1: Wild birds

Of the steps identified as determinants in the infection of wild birds by exposure to scraps from an imported contaminated chicken carcass, $WB_{agentsurvival}$ and $WB_{infectivedose}$ are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$WB_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by a wild bird

In the absence of any specific reports on the inactivation of APMV-2, it was assumed that APMV-2 has a similar spectrum of sensitivity to APMV-1. Therefore, APMV-2, protected within the chicken meat scraps, is likely to survive in the environment under ambient temperatures of 10 ºC to 35 ºC for several days, giving ample time for wild birds to locate and scavenge the material. This likelihood was assessed by the IRA team as ***high***.

$WB_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a wild bird is sufficient to produce infection

APMV-2 has not been reported in the species of birds known to frequent refuse dumps, but infection of passerine birds, such as sparrows and magpies, would be possible from backyard disposals of chicken meat. The IRA team considered that there was a ***low*** likelihood that APMV-2 would infect a wild bird consuming the contaminated meat scraps.

Exposure Group 2: Low biosecurity poultry

Of the steps identified as determinants in the infection of low biosecurity poultry by exposure to material from an imported contaminated chicken carcass, $BP_{agentsurvival}$, $BP_{infectivedose}$, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$BP_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by low biosecurity poultry

The time between feeding of scraps and consumption by low biosecurity poultry is likely to be very short, so environmental degradation of the disease agent will be minimal. The likelihood that the agent will remain viable was assessed by the IRA team as ***certain (=1)***.

$BP_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a low biosecurity bird is sufficient to produce infection

The natural hosts of APMV-2 are passerine birds. Infection of poultry does not produce systemic infection, and therefore contamination of the carcass would be limited to internal and external surfaces. The likely titre in muscle of infected or contaminated birds is assumed to be less than with NDV (APMV-1) infection. Transmission of APMV-2 to chickens by feeding of infected meat has not been documented. The IRA team considered that the likelihood that low biosecurity poultry would be infected with APMV-2 as a result of consuming the imported contaminated chicken meat scraps was ***low***.

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

The likelihood that APMV-2 would survive the rendering process was negligible. The IRA team considered that the likelihood the product would be re-contaminated with APMV-2 post-processing was negligible. Therefore the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with APMV-2 was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

Given that APMV-2 would be destroyed by rendering as discussed above, and that feed derived from contaminated carcasses would be diluted with feed from non-risk material, the likelihood that the amount of final poultry ration eaten by a bird would contain an oral infectious dose of virus was considered by the IRA team to be ***negligible***.

Exposure Group 3: Medium biosecurity commercial poultry

Of the steps identified as determinants in the infection of medium biosecurity commercial poultry by exposure to material from an imported contaminated chicken carcass, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that poultry feed would be contaminated with APMV-2 was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that the final poultry ration would contain an oral infectious dose of APMV-2 was considered to be ***negligible***.

Exposure Group 4: Non-avian species

As discussed above, $NAS_{agentsurvival}$ was considered to be equal to $WB_{agentsurvival}$.

APMV-2 infection has not been reported in non-avian species. Therefore this exposure group was not considered further in relation to this disease. $NAS_{infectivedose}$ was set to zero.

Conclusions – Exposure assessment

Using the simulation model as a guide, the partial likelihood of exposure for each of the exposure groups was determined, taking the above estimates of the exposure variables into account. A summary of the outcomes determined by the IRA team is set out in Table 110.

Table 110. Partial likelihoods of exposure (PLE)

Exposure group	PLE
Wild birds	E. Low
Low biosecurity poultry	E. Low
Medium biosecurity poultry	Negligible
Non-avian species	Zero

Consequence assessment

Establishment and spread was considered in the context of the outbreak scenarios, described in the Method of Risk Assessment.

Estimating the likelihood of each outbreak scenario

The partial likelihood of establishment and spread (PLES) of APMV-2 for the different exposure groups is described below. Four outbreak scenarios were considered relevant:

1. Disease agent does not establish or is not recognised within the directly exposed population
2. Disease agent establishes within the directly exposed population, is identified and is eliminated by human efforts or by natural means
3. Disease agent establishes in the directly exposed population, spreads within a district/region, including into other exposure groups if applicable, and is eliminated by human action or by natural means
4. Disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia.

Wild birds

Small passerine birds are assumed to be the primary natural hosts of APMV-2. APMV-2 infection has not been reported in birds known to scavenge at rubbish dumps, however, infection of passerine birds would be possible from backyard disposals of chicken meat. The IRA team considered that the most likely outcome of infection of a wild bird resulting from scavenging chicken meat scraps would be a single or a few isolated occurrences of infection in wild birds, with the virus being unable to establish ongoing infection in the population. Spread of APMV-2 from wild birds to poultry has been suggested as a means of dissemination of this virus overseas (Lipkind et al. 1982). If infection were to spread from wild birds to other exposure groups, diagnosis may be delayed by the mild or inapparent clinical disease that accompanies infection with APMV-2. Infection may be diagnosed if production deficits were being investigated in medium biosecurity commercial poultry; however, it is unlikely that diagnosis would be followed by government-backed control programs. In view of these factors, outbreak scenarios 1 (disease does not establish or is not recognised) and 4 (disease agent becomes endemic) were considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 111).

Table 111. Estimated partial likelihood of establishment and spread (PLES) values for APMV-2 in wild birds

Outbreak scenario 1:	$PLES_{(wb)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(wb)}2$	**Very low**
Outbreak scenario 3:	$PLES_{(wb)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(wb)}4$	**Moderate**

Low biosecurity poultry

Although this exposure group includes commercial free-range poultry and ratites, the IRA team considered that the sub-set of this group most likely to be exposed to scraps of imported chicken meat was small flocks of backyard poultry, because these are more likely to be directly fed table scraps. Of domestic poultry species, turkeys are the usual natural hosts of APMV-2. Infection generally results in mild respiratory or inapparent disease, unless complicated by other infections. In addition, turkeys represent a small proportion of the backyard poultry kept by households. In such flocks, there is less opportunity for the generation of high levels of environmental contamination, such as might occur with an outbreak of infectious disease in a large commercial flock. The most likely outcome of infection would be a single or a few isolated occurrences of infection.

If the disease were to establish in the flock, the level of expertise in disease recognition is likely to be low. Mechanical transmission of the virus by contaminated persons or fomites, and transmission by movement of birds may facilitate spread of the virus beyond the initially infected flock before it is recognised, and before control measures are implemented. Wild passerine birds may also spread the virus to other species, by contact or invasion of poultry houses. If infection did spread to commercial poultry, the mild clinical nature of the disease may delay diagnosis, and the lack of official control measures may lead to the disease agent becoming widespread. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 112).

Table 112. Estimated partial likelihood of establishment and spread (PLES) values for APMV-2 in low biosecurity poultry

Outbreak scenario 1:	$PLES_{(bp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(bp)}2$	**Very low**
Outbreak scenario 3:	$PLES_{(bp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(bp)}4$	**Moderate**

Medium biosecurity commercial poultry

The IRA team considered that the most feasible route for exposure of medium biosecurity commercial poultry to imported carcass components would be through poultry feed containing inadequately rendered processing waste. The likelihood that pathogens would remain viable following rendering of contaminated imported carcasses and parts was assessed as negligible (page 342). Nevertheless, assessment of PLES was based on the assumption that medium

biosecurity commercial poultry had been exposed to poultry feed contaminated with the disease agent.

Although APMV-2 infection has been reported in chickens and turkeys, passerines are considered the usual natural hosts of APMV-2. Of the poultry species, turkeys are more commonly infected. The virus is shed from the respiratory and intestinal tracts of infected birds, and it is assumed that the methods of spread would be similar to APMV-1. APMV-2 infection is reported to spread slowly in affected flocks, and flock-to-flock transmission, even between flocks in close proximity, does not always occur. Clinical signs may be mild or inapparent, and therefore the infection may initially go undiagnosed. Although infection appears to spread slowly between flocks, lack of diagnosis and control measures may lead to the disease being spread to other flocks during normal commercial activities. APMV-2 infection is unlikely to be subject to official control measures, and so may eventually become widespread. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 113).

Non-Avian Species

As discussed above, this exposure group was not considered further in relation to this disease.

Table 113. Estimated partial likelihood of establishment and spread (PLES) values for APMV-2 in medium biosecurity commercial poultry

Outbreak scenario 1:	$PLES_{(cp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(cp)}2$	**Very low**
Outbreak scenario 3:	$PLES_{(cp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(cp)}4$	**Moderate**

Conclusion – Likelihood assessments

The estimates for the likelihood of release, the partial likelihoods of exposure for each of the exposure groups, and the partial likelihood of establishment and spread for each of the outbreaks scenarios were combined with the expected volume of trade using the simulation model. This allowed the calculation of partial annual likelihood of entry, exposure, establishment and spread for each of the outbreak scenarios. The results of this calculation are shown in Table 114.

Estimating the impacts associated with each outbreak scenario

For each outbreak scenario the direct and indirect impacts of APMV-2 infection were estimated at the national, State or Territory, district/region and local levels, as described in the Methods section (page 90-95, Part B).

The likelihood of APMV-2 affecting non-avian species (exposure group 4) was considered to be remote. Therefore, the impacts of APMV-2 infection occurring in this exposure group were not considered further.

Table 114. Partial annual likelihood of entry, exposure, establishment and spread (PALEEES) for the outbreak scenarios

Exposure group	Outbreak scenario	PALEEES
Wild birds	Scenario 1	High
	Scenario 2	High
Low biosecurity poultry	Scenario 1	High
	Scenario 2	High
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
Total all exposure groups	Scenario 3	High
Total all exposure groups	Scenario 4	High

Outbreak Scenario 1

By definition, outbreak scenario 1 means that the disease agent does not establish in the initially exposed population, or does not cause sufficient disease to lead to investigation and recognition of the infection. Therefore the impacts will be ***unlikely to be discernible*** for all exposure groups and all criteria.

Outbreak Scenario 2

The impacts of this outbreak scenario (disease agent establishes within the directly exposed population, and is identified and eliminated) will differ between exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

The primary natural host of APMV-2 appear to be small passerine birds, which are unlikely to be found scavenging chicken meat at dump sites. Wild birds do not play a significant part in production, so direct economic loss from death of wild birds, if it occurs, is not measurable. Other impacts from the death of wild birds will be considered under direct criterion 2 and indirect criterion 4. The direct impacts of disease on wild birds were assessed by the IRA team as ***unlikely to be discernible*** at all levels for this criterion.

APMV-2 infection in chickens generally results in mild respiratory or inapparent disease. The disease is reported to be more severe in turkeys than in chickens, and if complicated by other infections or by environmental conditions may result in severe respiratory disease, sinusitis, increased mortality and low egg production in both species. However, an outbreak contained within a local population of low biosecurity poultry will result in losses to individual owners only. The impacts of such losses on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

If an outbreak were to occur in medium biosecurity commercial poultry, especially turkeys, egg production losses, decreased hatchability and low poult yield may result in ***minor*** impacts at the local level. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

There are no reports of APMV-2 outbreaks in the wild. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at all levels for all exposure groups.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

APMV-2 is not notifiable in any State or Territory of Australia, and is not subject to official controls. The impacts of an outbreak of APMV-2 in wild birds on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels. The impacts of an outbreak in low biosecurity poultry, even if involving a free-range flock, were assessed as ***unlikely to be discernible*** at all levels.

While no official action will be taken to eradicate the disease if APMV-2 were diagnosed in medium biosecurity commercial poultry flocks, individual growers will need to take some control action. Impacts of such programs were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, and ***minor*** at the local level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

The impacts of an outbreak of APMV-2 in wild birds on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak of APMV-2 in low biosecurity poultry were assessed as ***unlikely to be discernible*** at all levels.

Because APMV-2 infection results in mild or inapparent disease in most cases, impacts of an outbreak in medium biosecurity commercial poultry were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

APMV-2 has relatively low pathogenicity in domestic poultry. It is not recognised by the OIE as a disease of international trade significance. Impacts for all exposure groups were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of an outbreak of APMV-2 on the environment in all exposure groups were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impacts of an outbreak of APMV-2 in wild birds on communities were assessed by the IRA team as ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak of APMV-2 in low biosecurity poultry were assessed as ***unlikely to be discernible*** at all levels.

If commercial flocks were infected, temporary restrictions may be imposed on movement of birds, eggs, poultry products and people until the diagnosis was confirmed. This may lead to community disruption. The impacts of a disease outbreak in medium biosecurity commercial poultry on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level.

Outbreak Scenario 3

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a district/region, and is eliminated) will be the same for all exposure groups, no matter which exposure group has been directly exposed to APMV-2 in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

Production losses outlined above were assessed as ***unlikely to be discernible*** at national, State/Territory and district/region levels. Impacts at the local level were assessed by the IRA team as ***minor***.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

There have been no reports of APMV-2 outbreaks in the wild. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

It is possible that control and eradication programs may be instigated if APMV-2 were to spread to commercial flocks. Inactivated vaccines are available overseas and may be introduced to control disease, particularly in turkey flocks. Impacts were assessed by the IRA team as

unlikely to be discernible at national and State/Territory levels, and ***minor*** at the district/region level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Because APMV-2 infection results in mild or inapparent disease in most cases, impacts were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

APMV-2 is not recognised by the OIE as a disease of international trade significance. Impacts on the local, district/region, State/Territory and national economies were assessed by the IRA team as ***unlikely to be discernible***.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impact of an outbreak of APMV-2 on the environment was assessed as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Temporary restrictions may be imposed on movement of birds, eggs, poultry products and people until the diagnosis is confirmed. This may lead to community disruption. The impacts were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level.

Outbreak Scenario 4

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a State/Territory, and becomes endemic in Australia) will be the same for all exposure groups, no matter which exposure group has been directly exposed to APMV-2 in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

If the disease spreads more widely through medium biosecurity commercial poultry and low biosecurity poultry, losses of birds and production may be considerable, especially if complicated by other infections or by environmental conditions. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, but ***minor*** at the district/region level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

There have been no reports of APMV-2 outbreaks in the wild. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

Inactivated vaccines may be introduced to control disease, particularly in turkey flocks. Therefore, producers will incur additional costs. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory level, and ***minor*** at the district/region level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Because APMV-2 infection results in mild or inapparent disease in most cases, impacts were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

Impacts of a more general outbreak were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of a generalised outbreak of APMV-2 on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impacts of a general outbreak of APMV-2 on communities were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level.

Conclusions – impact assessment

The above estimates for each of the impact criteria were entered into the simulation model, and the overall impact of each outbreak scenario was estimated. Results are shown in Table 115.

Table 115. Impacts of each outbreak scenario

Exposure group	Outbreak scenario	Impact
Wild birds	Scenario 1	Negligible
	Scenario 2	Negligible
Low biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
	Total Scenario 3	Very low
	Total Scenario 4	Very low

Partial annual risk estimate for each outbreak scenario

The partial annual likelihood of entry, exposure, establishment and spread were combined with the impacts of the relevant outbreak scenarios to provide an estimate of the partial annual risk associated with each outbreak scenario. The outcomes of this process are shown in Table 116.

Table 116. Partial annual risk of each outbreak scenario

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Medium biosecurity poultry	Scenario 1	Negligible	Negligible	Negligible
	Scenario 2	Negligible	Negligible	Negligible
Non-avian species	Scenario 1	Zero	Zero	Zero
	Scenario 2	Zero	Zero	Zero
	Total Scenario 3	High	Very low	Very Low
	Total Scenario 4	High	Very low	Very Low

Unrestricted risk estimate

The overall risk associated with the import of whole chicken carcasses was assessed as ***very low*** for APMV-2. As the unrestricted risk estimate meets Australia's ALOP, no risk management was considered necessary.

Direct impact on human life or health

APMV-2 is not known to affect humans and is not considered to be a threat to public health.

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Technical Information

Background

Avian paramyxovirus-3 (APMV-3) causes respiratory signs and decreased egg production in infected turkey flocks. Although turkeys are considered to be the primary natural host of APMV-3, experimental studies have shown that chickens are susceptible to infection (Alexander 2003) and there is one report of isolation of APMV-3 from a flock of chickens with respiratory disease (Shihmanter et al. 2000). APMV-3 has also been isolated from captive psittacine and passerine birds held in quarantine (Alexander 2003).

APMV-3 has not been reported in avian species in Australia. APMV-3 infection is not an OIE-listed disease.

Agent taxonomy

APMV-3 is a member of the genus *Avulavirus* of the family *Paramyxoviridae* (Buchen-Osmond 2002). Isolates show considerable diversity and two antigenically distinguishable groups are recognised. The first has been isolated only from turkeys, and the second from captive, caged psittacine and passerine birds, and recently from a chicken flock in Israel (Shihmanter et al. 2000; Shihmanter et al. 1998b).

Agent characteristics

APMV-3 is an enveloped RNA virus and is therefore destroyed rapidly outside the host species. In general, paramyxoviruses are sensitive to thermal inactivation, lipid solvents and chlorine-based disinfectants. In the absence of any specific reports on the inactivation of APMV-3, it is assumed that APMV-3 has a similar spectrum of sensitivity to APMV-1 (Newcastle disease virus).

Epidemiology

Turkeys are considered to be the primary natural host of APMV-3 and most reports of natural APMV-3 infections in domestic poultry are from turkeys in Western Europe, North America and Israel (Alexander 2000). There is one report of isolation of APMV-3 from the cloacal swab of a chicken showing clinical signs of respiratory disease in Israel (Shihmanter et al. 2000). Antigenically different strains of APMV-3 have also been isolated from captive birds, principally imported exotic caged passerine and psittacine birds in Europe and Asia, but there are no reports of isolation of APMV-3 from wild birds (Alexander 2000). APMV-3 has not been reported in avian species in Australia or New Zealand.

Reports of the prevalence of APMV-3 in chickens are few and are complicated by cross reactivity with APMV-1 antisera in serological tests. In Spain, 14.7% (50 of 341 birds sampled) of layer hens on 36.4% (16 of 44 farms) of farms surveyed, and 4.9% (6 of 123 birds sampled) of meat chickens on 20% (1 of 5 of farms) were shown to be serologically positive for APMV-3 antibodies (Maldonado et al. 1994). However, it is likely that most of these results corresponded to vaccine-induced APMV-1 antibodies.

Similarly, there are few reports of studies of the transmission of APMV-3. Since mild respiratory disease is the earliest sign of infection, the primary site of infection is likely to be the respiratory tract. Virus has been isolated from tracheal and cloacal swabs of domestic poultry, so transmission probably occurs through aerosols and ingestion of material contaminated with faeces. In field infections it is reported that APMV-3 spreads only slowly through the flock, and flock-to-flock transmission, even between flocks in close proximity, does not always occur (Alexander, Pattison, and Macpherson 1983; Alexander 1993). In the absence of wild bird hosts for APMV-3 viruses, it seems likely that introduction of this virus to different countries has been through importation of poultry or via humans (Alexander 2003).

There are no reports of transovarial transmission of APMV-3 (Alexander 1993).

Clinical signs

Respiratory signs occur early in the course of the disease. Coughing, sneezing, rales, sinusitis, conjunctivitis, depression and dyspnoea have been described in APMV-3 infected turkeys. A reduction in egg production may occur, and fertility and hatchability may be affected. More serious losses and respiratory disease have been reported where infection has occurred around or at point of lay (Alexander 2000). Inapparent infections have also been reported in turkeys (Alexander, Pattison, and Macpherson 1983).

Lethargy, respiratory distress and deaths were reported in chickens experimentally infected as day-olds with a strain of APMV-3 isolated from psittacine birds (Alexander and Collins 1982). Stunting also occurred and the authors postulated that considerable production and economic losses could occur in chickens infected with APMV-3 at an early age. Older chickens showed no clinical signs. Sneezing, rales, sinusitis and difficult breathing were reported in chickens naturally-infected with a psittacine strain of APMV-3 (Shihmanter et al. 2000). Chickens with secondary infections or infections with both APMV-2 and APMV-3 show more severe respiratory signs (Weisman, Malkinson, and Yuval 1999).

Cachexia and diarrhoea have been reported in passerine birds infected with APMV-3; and weakness, anorexia, vomiting and sneezing in infected psittacine birds (Shihmanter et al. 1998a). Infection has been associated with encephalitis and high mortality in caged psittacine birds.

Pathology

APMV-3 infection in poultry generally causes inapparent or mild respiratory disease. No specific lesions due to APMV-3 infection have been reported.

Immunology

Inactivated, oil emulsion vaccine is used to prevent serious production losses associated with infection in laying turkeys in the United States and Europe (Alexander 2003). Vaccine is usually administered twice, four weeks apart before the birds begin to lay.

APMV-3 viruses may show sufficiently high levels of cross reactivity with conventional APMV-1 (ND virus) antisera to cause problems with interpretation of serological tests. Apparent antibodies to APMV-3 in chickens have been attributed to high antibody levels to ND virus as a result of vaccination (Box, Holmes, and Webb 1988). Prior infection of chickens with

APMV-3 conferred some protection against challenge with a virulent strain of ND virus (Alexander, Chettle, and Parsons 1979).

Diagnosis

Virus isolation is often difficult since the virus is shed for only a short time. The virus may be isolated from swabs or samples of trachea, lung, sinus, cloaca and pharynx inoculated into embryonated chicken eggs via the allantoic cavity. Inoculation of six- to seven-day-old embryonated eggs via the yolk sac may sometimes yield better results. APMV-3 may also be cultured in chicken embryo kidney cells, monkey kidney or bovine kidney (Awang and Russell 1990).

A number of serological tests are available to assist with diagnosis of APMV-3. At present, the haemagglutination inhibition (HI) test is used most widely (Alexander 2000). APMV-3 may show some antigenic cross-reactions in HI tests with APMV-1 (Alexander 2003), but these can be resolved by the use of suitable antigen and antiserum controls.

Transmission in chicken meat

There is no data on the survival or presence of APMV-3 in carcasses. However, if it is assumed that the methods of spread of APMV-3 are similar to APMV-1, carcasses could be infected with the virus or become contaminated during processing.

Quarantine significance

APMV-3 is not an OIE-listed disease agent.

APMV-3 infection is not notifiable in any State or Territory of Australia, and is not subject to official controls. APMV-3 is not included in the Emergency Animal Disease Response Agreement. Therefore, it is considered to be of relatively minor concern, and is unlikely to have consequences that are discernible beyond the district/region level. Even uncomplicated APMV-3 infection in turkeys, especially laying birds, is considered to be of sufficient economic significance to warrant the use of expensive inactivated vaccines for its control overseas (Alexander 2003). Control and eradication programs may be warranted in countries with large commercial turkey populations (Awang and Russell 1990).

Risk Assessment

Release Assessment

Rel_1: Selection of source flock (between flock prevalence)

Although experimental studies have shown that chickens are susceptible to infection, there is only one published report of isolation of APMV-3 from a flock of chickens with respiratory disease (Shihmanter et al. 2000). Outbreaks of APMV-3 in domestic chickens are therefore considered very rare events. For an unrestricted risk estimate, it was assumed that the prevalence is at the highest sustainable level in an endemically infected country or zone (see Method for Risk Assessment). The likelihood that a source flock will be infected with APMV-3 was assessed by the IRA team as ***extremely low***.

Rel_2: Infection detected through flock surveillance and the flock withdrawn from slaughter

APMV-3 infection in poultry generally causes inapparent or mild respiratory disease. In an outbreak of APMV-3, clinical signs may not be apparent to the producer. The likelihood that a diseased flock will be detected through routine flock surveillance and the flock withheld from slaughter was assessed by the IRA team as ***extremely low***.

Rel_3: Selection of an infected chicken from an infected flock (within flock prevalence)

In field infections, APMV-3 appears to spread only slowly through the flock, and flock-to-flock transmission, even between flocks in close proximity does not always occur. Therefore, the likelihood that a selected individual chicken will be infected was assessed by the IRA team as ***low***.

Rel_4: Background cross-contamination rate

This likelihood represents the background cross-contamination rate during slaughter and processing and applies to cross-contamination with material, infectious or otherwise, from other carcasses, including those tissues or materials in which the disease agent tends to localise.

Since it was assumed that the methods of spread of APMV-3 are similar to APMV-1, it is possible that carcasses could be infected with the virus or become contaminated during processing. The IRA team considered that the likelihood of a carcass being contaminated with potentially contaminated material from other birds, especially from the digestive tract, is ***moderate***.

Rel_5: Likelihood that an uninfected carcass will be contaminated with the disease agent during slaughter and processing

As discussed in the Method for Risk Assessment (page 58, Part B), Rel_4 was used to calculate Rel_{5a} (the likelihood that an uninfected carcass from an infected flock will become contaminated during slaughter and processing) and Rel_{5b} (the likelihood that an uninfected carcass from an uninfected flock will become contaminated during slaughter and processing).

For APMV-3, Rel_{5a} was calculated as ***low***, and Rel_{5b} was calculated as ***extremely low***.

Rel_6: The likelihood that the carcass of a bird that was *infected* before slaughter will be removed as a result of inspections before or during processing

APMV-3 infection in poultry generally causes inapparent or mild respiratory disease, and no specific lesions due to APMV-3 infection have been reported. No obvious post-mortem lesions are likely to be present during slaughter and processing. The IRA team considered that the rejection rate of infected carcasses would be equal to the background rejection rate of 0.75%, as described in the Method section.

Rel_7: The likelihood that the carcass of a bird that was *not infected* before slaughter will be removed as a result of inspections before or during processing

The likelihood that an uncontaminated/uninfected carcass will be removed during processing inspections was considered to be equal to the background rejection rate of 0.75% as described in the Method section (page 59, Part B).

Rel_8: Inactivation of the agent during further processing, storage, handling and transport

In the absence of any specific reports on the inactivation of APMV-3, it was assumed that APMV-3 has a similar spectrum of sensitivity to APMV-1. Therefore, the likelihood of inactivation of the virus during further processing, storage, handling and transport was assessed by the IRA team as ***extremely low***.

Conclusions – Release assessment

After inserting the above estimates into the simulation model, and using the outputs from the model as a guide, the IRA team concluded that, in the absence of risk management and without considerations regarding the exporting country, there was an ***extremely low*** likelihood that imported chicken meat would be infected or contaminated with APMV-3.

Exposure assessment

Exposure Group 1: Wild birds

Of the steps identified as determinants in the infection of wild birds by exposure to scraps from an imported contaminated chicken carcass, $WB_{agentsurvival}$ and $WB_{infectivedose}$ are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$WB_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by a wild bird

In the absence of any specific reports on the inactivation of APMV-3, it was assumed that APMV-3 has a similar spectrum of sensitivity to APMV-1. Therefore, APMV-3, protected within chicken meat scraps, is likely to survive in the environment under ambient temperatures of 10 ºC to 35 ºC for several days, giving ample time for wild birds to locate and scavenge the material. The IRA team considered that this likelihood was ***high*.**

$WB_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a wild bird is sufficient to produce infection

APMV-3 has been isolated from turkeys and from psittacine and small passerine birds. Infection has not been reported in species of birds known to frequent refuse dumps. While passerine and psittacine birds could gain access to chicken meat scraps in backyards, most such susceptible species do not consume meat. Furthermore, APMV-3 is likely to be present in low concentrations only on the surface of contaminated/infected carcasses. The IRA team considered that there was a ***negligible*** likelihood that APMV-3 would infect a wild bird consuming the contaminated meat scraps.

Exposure Group 2: Low biosecurity poultry

Of the steps identified as determinants in the infection of low biosecurity poultry by exposure to material from an imported contaminated chicken carcass, $BP_{agentsurvival}$, $BP_{infectivedose}$, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

$BP_{agentsurvival}$: The likelihood that the disease agent remains viable after exposure to the environment over the period before consumption by low biosecurity poultry

The time between feeding of scraps and consumption by low biosecurity poultry is likely to be very short, so environmental degradation of the disease agent will be minimal. The likelihood that the agent will remain viable was assessed by the IRA team as very close to ***certain (approx =1)***.

$BP_{infectivedose}$: The likelihood that the amount of the contaminated chicken waste eaten by a low biosecurity bird is sufficient to produce infection

In the absence of specific data on APMV-3, the oral infectious dose of APMV-3 in poultry is assumed to be similar to APMV-1. However, the presence of virus in meat is expected to be limited to surface contamination only. Transmission of APMV-3 to chickens by feeding of infected meat has not been documented. The likelihood that a sufficient dose of virus would be present to initiate infection was considered by the IRA team to be ***low***.

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

The likelihood that APMV-3 would survive the rendering process was negligible. The IRA team considered that the likelihood the product would be re-contaminated with APMV-3 post-processing was negligible. Therefore, the likelihood that poultry feed derived from the imported contaminated carcass would be contaminated with APMV-3 was estimated by the IRA team to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

Given that APMV-3 would be destroyed by rendering as discussed above, and that feed derived from contaminated carcasses would be diluted with feed from non-risk material, the likelihood that the amount of final poultry ration eaten by a bird would contain an oral infectious dose of virus was considered by the IRA team to be ***negligible***.

Exposure Group 3: Medium biosecurity commercial poultry

Of the steps identified as determinants in the infection of medium biosecurity commercial poultry by exposure to material from an imported contaminated chicken carcass, FEEDCONTAMINATED and INFECTDOSEINFEED are pathogen dependent. All other determinants are pathogen independent and are discussed in detail in the Method section (pages 67-86, Part B).

FEEDCONTAMINATED: The likelihood that poultry feed, produced from the rendered contaminated imported carcass, will be contaminated with the disease agent

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that poultry feed would be contaminated with APMV-3 was estimated to be ***negligible***.

INFECTDOSEINFEED: The likelihood that the amount of the contaminated commercial poultry feed eaten by a bird is sufficient to produce infection

As discussed above (Exposure Group 2: Low biosecurity poultry), the likelihood that the final poultry ration would contain an oral infectious dose of APMV-3 was considered to be ***negligible***.

Exposure Group 4: Non-avian species

As discussed above, $NAS_{agentsurvival}$ was considered to be equal to $WB_{agentsurvival}$.

APMV-3 infection has not been reported in non-avian species. Therefore this exposure group was not considered further in relation to this disease. $NAS_{infectivedose}$ was set to a value of zero.

Conclusions – Exposure assessment

Using the simulation model as a guide, the partial likelihood of exposure for each of the exposure groups was determined, taking the above estimates of the exposure variables into account. A summary of the outcomes determined by the IRA team is set out in Table 117.

Table 117. Partial likelihoods of exposure (PLE)

Exposure group	PLE
Wild birds	Negligible
Low biosecurity poultry	Very low
Medium biosecurity poultry	Negligible
Non-avian species	Zero

Consequence assessment

Establishment and spread was considered in the context of the outbreak scenarios, described in the Method of Risk Assessment.

Estimating the likelihood of each outbreak scenario

The partial likelihood of establishment and spread (PLES) of APMV-3 for the different exposure groups is described below. Four outbreak scenarios were considered relevant:

1. Disease agent does not establish or is not recognised within the directly exposed population
2. Disease agent establishes within the directly exposed population, is identified and is eliminated by human efforts or by natural means

3. Disease agent establishes in the directly exposed population, spreads within a district/region, including into other exposure groups if applicable, and is eliminated by human action or by natural means
4. Disease agent establishes in the directly exposed population, spreads within a State/Territory, including to other exposure groups if applicable, and becomes endemic in Australia.

Wild birds

There is no evidence of APMV-3 infection in species likely to consume meat scraps. APMV-3 has been isolated from captive birds, principally imported exotic caged passerine and psittacine birds, but there are no reports of isolation of APMV-3 from wild birds. The IRA team considered that the most likely outcome of infection of a wild bird resulting from scavenging chicken meat scraps would be a single or a few isolated occurrences of infection in wild birds, with the virus being unable to establish ongoing infection in the population. Infection of wild birds with APMV-3, with subsequent spread to poultry, has not been reported, but cannot be excluded. If spread of the disease agent were to occur, it may not be detected until it had become widespread, because of the mild nature of the clinical signs in most chicken flocks. In view of these factors, outbreak scenario 1 (disease does not establish or is not recognised) was considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 118).

Table 118. Estimated partial likelihood of establishment and spread (PLES) values for APMV-3 in wild birds

Outbreak scenario 1:	$PLES_{(wb)}1$	**High**
Outbreak scenario 2:	$PLES_{(wb)}2$	**Very low**
Outbreak scenario 3:	$PLES_{(wb)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(wb)}4$	**Very low**

Low biosecurity poultry

Although this exposure group includes commercial free-range poultry and ratites, the IRA team considered that the sub-set of this group most likely to be exposed to scraps of imported chicken meat was small flocks of backyard poultry, because these are more likely to be directly fed table scraps. Turkeys are considered to be the primary natural host of APMV-3 but turkeys represent a small proportion of the backyard poultry kept by households. In such flocks, there is less opportunity for the generation of high levels of environmental contamination than might occur with an outbreak of infectious disease in a large commercial flock. The most likely outcome of infection would be a single or a few isolated occurrences of infection.

If the disease were to establish in the flock, the level of expertise in disease recognition is likely to be low. Mechanical transmission of the virus by contaminated persons or fomites, and transmission by movement of birds may facilitate spread of the virus beyond the initially infected flock before it is recognised, and control measures are implemented. Spread of APMV-3 occurs only slowly, and flock-to-flock transmission does not always occur. Therefore, infection may be limited to this exposure group, or it may be eliminated by natural means. If

the infection were to spread to involve commercial poultry, lack of official control measures may eventually result in the disease becoming widespread. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 119).

Table 119. Estimated partial likelihood of establishment and spread (PLES) values for APMV-3 in low biosecurity poultry

Outbreak scenario 1:	$PLES_{(bp)}1$	**High**
Outbreak scenario 2:	$PLES_{(bp)}2$	**Low**
Outbreak scenario 3:	$PLES_{(bp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(bp)}4$	**Very low**

Medium biosecurity commercial poultry

The IRA team considered that the most feasible route for exposure of medium biosecurity commercial poultry to imported carcass components would be through poultry feed containing inadequately rendered processing waste. The likelihood that pathogens would remain viable following rendering of contaminated imported carcasses and parts was assessed as negligible (page 361). Nevertheless, assessment of PLES was based on the assumption that medium biosecurity commercial poultry had been exposed to poultry feed contaminated with the disease agent.

Respiratory signs, reduction in egg production, fertility and hatchability have been described in APMV-3 infected turkeys. Inapparent infections may also occur. Transmission probably occurs through aerosols and ingestion of material contaminated with faeces. APMV-3 is reported to spread only slowly through infected flocks, and flock-to-flock transmission, even between flocks in close proximity does not always occur. In the event that infection did spread beyond the initially infected flock, however, the mild clinical signs and lack of official control measures may result in the infection becoming widespread. In view of these factors, outbreak scenarios 1 and 2 are considered the most likely. The IRA team assigned the following likelihoods to the four outbreak scenarios (Table 120).

Table 120. Estimated partial likelihood of establishment and spread (PLES) values for APMV-3 in medium biosecurity commercial poultry

Outbreak scenario 1:	$PLES_{(cp)}1$	**Moderate**
Outbreak scenario 2:	$PLES_{(cp)}2$	**Moderate**
Outbreak scenario 3:	$PLES_{(cp)}3$	**Very low**
Outbreak scenario 4:	$PLES_{(cp)}4$	**Very low**

Non-Avian Species

As discussed above, this exposure group was not considered further in relation to this disease.

Conclusion – Likelihood assessments

The estimates for the likelihood of release, the partial likelihoods of exposure for each of the exposure groups, and the partial likelihood of establishment and spread for each of the outbreaks scenarios were combined with the expected volume of trade using the simulation model. This allowed the calculation of partial annual likelihood of entry, exposure, establishment and spread for each of the outbreak scenarios. The results of this calculation are shown in Table 121.

Table 121. Partial annual likelihood of entry, exposure, establishment and spread (PALEES) for the outbreak scenarios

Exposure group	Outbreak scenario	PALEEES
Wild birds	Scenario 1	Extremely low
	Scenario 2	Negligible
Low biosecurity poultry	Scenario 1	High
	Scenario 2	High
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
Total all exposure groups	Scenario 3	Moderate
Total all exposure groups	Scenario 4	Moderate

Estimating the impacts associated with each outbreak scenario

For each outbreak scenario the direct and indirect impacts of APMV-3 infection were estimated at the national, State or Territory, district/region and local levels, as described in the Methods section (page 90-95, Part B).

The likelihood of APMV-3 affecting non-avian species (exposure group 4) was considered to be remote. Therefore, the impacts of APMV-3 occurring in this exposure group were not considered further.

Outbreak Scenario 1

By definition, outbreak scenario 1 means that the disease agent does not establish in the initially exposed population, or does not cause sufficient disease to lead to investigation and recognition of the infection. Therefore the impacts will be ***unlikely to be discernible*** for all exposure groups and all criteria.

Outbreak Scenario 2

The impacts of this outbreak scenario (disease agent establishes within the directly exposed population, and is identified and eliminated) will differ between exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

Since wild birds do not play a significant part in production, direct economic loss from death of wild birds, were it to occur, is not measurable. Other impacts from the death of wild birds will be considered under direct criterion 2 and indirect criterion 4. The direct impacts of disease on wild birds were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

APMV-3 causes respiratory signs and decreased egg production in infected turkeys. An outbreak contained within a local population of low biosecurity poultry would result in losses to individual owners only. The impacts of such losses on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Coughing, sneezing, rales, sinusitis, conjunctivitis, depression and dyspnoea have been described in APMV-3 infected turkeys. If an outbreak were to occur in medium biosecurity commercial poultry, especially turkeys, egg production losses, decreased fertility and hatchability may result in ***minor*** impacts at the local level. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

There are no reports of APMV-3 outbreaks in the wild. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at all levels for all exposure groups.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

APMV-3 is not notifiable in any State or Territory of Australia, and is not subject to official controls within Australia. The impacts of an outbreak of APMV-3 in wild birds on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak in low biosecurity poultry were assessed as ***unlikely to be discernible*** at all levels.

While no official action would be taken to eradicate the disease if APMV-3 were diagnosed in medium biosecurity commercial poultry flocks, individual growers would need to take some control action. Impacts of such programs were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

The impacts of an outbreak of APMV-3 in wild birds on this criterion were assessed as ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak of APMV-3 in low biosecurity poultry were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Because APMV-3 infection results in mild or inapparent disease in most cases, impacts of an outbreak in medium biosecurity commercial poultry were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

APMV-3 has relatively low pathogenicity in domestic poultry. It is not recognised by the OIE as a disease of international trade significance. Impacts for all exposure groups were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of an outbreak of APMV-3 in all exposure groups were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impacts of an outbreak of APMV-3 in wild birds were assessed by the IRA team as ***unlikely to be discernible*** at all levels. Similarly, the impacts of an outbreak of APMV-3 in low biosecurity poultry were assessed as ***unlikely to be discernible*** at all levels.

If commercial turkey flocks were infected, temporary restrictions may be imposed on movement of birds, eggs, poultry products and people until the diagnosis was confirmed. This may lead to community disruption. The impacts of a disease outbreak in the medium biosecurity commercial poultry on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level.

Outbreak Scenario 3

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a district/region, and is eliminated) will be the same for all exposure groups, no matter which exposure group has been directly exposed to APMV-3 in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

Production losses in turkeys outlined above were assessed as ***unlikely to be discernible*** at national, State/Territory and district/region levels. Impacts at the local level were assessed by the IRA team as ***minor***.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

There have been no reports of APMV-3 outbreaks in the wild. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

It is possible that control and eradication programs may be instigated were APMV-3 to spread to commercial turkey flocks. Inactivated vaccines are available overseas and may be introduced to control disease. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, and ***minor*** at the district/region level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Because APMV-3 infection results in mild or inapparent disease in most cases, impacts of an outbreak were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

APMV-3 is not recognised by the OIE as a disease of international trade significance. Impacts at all levels were assessed by the IRA team as ***unlikely to be discernible***.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impact of an outbreak of APMV-3 was assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

Temporary restrictions may be imposed on movement of birds, eggs, poultry products and people until the diagnosis is confirmed. This may lead to community disruption. The impacts were assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level.

Outbreak Scenario 4

The impacts of this outbreak scenario (disease agent establishes in the directly exposed population, spreads to other exposure groups within a State/Territory, and becomes endemic in Australia) will be the same for all exposure groups, no matter which exposure group has been directly exposed to APMV-3 in imported chicken meat, since by definition the scenario extends to all susceptible exposure groups.

Direct impacts of a disease agent on host species and the environment

1. The life or health (including production impacts) of production, domestic or feral animals

If the disease spreads more widely through medium biosecurity commercial poultry and low biosecurity poultry, losses of birds and production may be considerable, especially if complicated by other infections. The impacts were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, but ***minor*** at the district/region level.

2. The environment, including life and health of native animals and direct impacts on the non-living environment

There have been no reports of APMV-3 outbreaks in the wild. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

Indirect impacts

1. New eradication, control, surveillance/monitoring and compensation strategies/programs

Inactivated vaccines may be introduced to control disease in turkey flocks. Impacts were assessed by the IRA team as ***unlikely to be discernible*** at national and State/Territory levels, and ***minor*** at the district/region level.

2. Domestic trade or industry impacts, including changes in consumer demand and impacts on other industries supplying inputs to or using outputs from directly affected industries

Because APMV-3 infection results in mild or inapparent disease in most cases, impacts of an outbreak were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

3. International trade impacts, including loss of markets, meeting new technical requirements to enter/maintain markets, changes in international consumer demand

Impacts of a more general outbreak were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

4. Impacts on the environment, including biodiversity, endangered species and the integrity of ecosystems

The impacts of a generalised outbreak of APMV-3 on this criterion were assessed by the IRA team as ***unlikely to be discernible*** at all levels.

5. Impact on communities, including reduced tourism, reduced regional and economic viability, loss of social amenity and side effects of control measures

The impact of a general outbreak of APMV-3 on communities was assessed by the IRA team as ***unlikely to be discernible*** at national, State/Territory and district/region levels, but ***minor*** at the local level.

Conclusions – impact assessment

The above estimates for each of the impact criteria were entered into the simulation model, and the overall impact of each outbreak scenario was estimated. Results are shown in Table 122.

Table 122. Impacts of each outbreak scenario.

Exposure group	Outbreak scenario	Impact
Wild birds	Scenario 1	Negligible
	Scenario 2	Negligible
Low biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Medium biosecurity poultry	Scenario 1	Negligible
	Scenario 2	Negligible
Non-avian species	Scenario 1	Zero
	Scenario 2	Zero
	Total Scenario 3	Very low
	Total Scenario 4	Very low

Partial annual risk estimate for each outbreak scenario

The partial annual likelihood of entry, exposure, establishment and spread were combined with the impacts of the relevant outbreak scenarios to provide an estimate of the partial annual risk associated with each outbreak scenario. The outcomes of this process are shown in Table 123.

Table 123. Partial annual risk of each outbreak scenario

Exposure group	Outbreak scenario	PALEEES	Impact	PAR
Wild birds	Scenario 1	E. Low	Negligible	Negligible
	Scenario 2	Negligible	Negligible	Negligible
Low biosecurity poultry	Scenario 1	High	Negligible	Negligible
	Scenario 2	High	Negligible	Negligible
Medium biosecurity poultry	Scenario 1	Negligible	Negligible	Negligible
	Scenario 2	Negligible	Negligible	Negligible
Non-avian species	Scenario 1	Zero	Zero	Zero
	Scenario 2	Zero	Zero	Zero
	Total Scenario 3	Moderate	Very low	Very Low
	Total Scenario 4	Moderate	Very low	Very Low

Unrestricted risk estimate

The overall risk associated with the import of whole chicken carcasses was assessed as ***very low*** for APMV-3. As this unrestricted risk estimate meets Australia's ALOP, no risk management was considered necessary.

Direct impact on human life or health

APMV-3 is not known to affect humans and is not considered to be a threat to public health.

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Arboviruses[3]

The term ‘arbovirus’ is an abbreviation of ‘arthropod-borne virus’ (Guy and Malkinson 2003). Arboviruses are transmitted between susceptible vertebrate hosts by blood-feeding arthropods (such as mosquitoes and ticks). Arboviruses belong to several different virus families, including the *Togaviridae*, *Flaviviridae*, *Bunyaviridae*, *Arenaviridae*, *Rhabdoviridae* and *Reoviridae*, but share the common characteristic of being transmitted by arthropod vectors (Guy and Malkinson 2003). The term 'arbovirus' itself has no taxonomic significance (Centers for Disease Control 2005a).

More than 100 arboviruses have been isolated from avian species or vectors that feed on them. However, only five arboviruses from two virus families have been identified as causes of disease in domestic poultry and farm-reared game birds (Guy and Malkinson 2003). The two families of arboviruses identified as causes of disease in poultry and game birds are the *Togaviridae*, which includes Eastern equine encephalomyelitis (EEE) virus, Western equine encephalomyelitis (WEE) virus and Highlands J (HJ) virus, and the *Flaviviridae*, which includes West Nile virus (WNV) and Israel turkey meningoencephalitis (IT) virus (Guy and Malkinson 2003). Japanese Encephalitis (JE) virus is a *Flavivirus*, which causes disease in pigs, humans and horses, and circulates in birds without causing clinical disease.

Chickens are competent hosts for only some of these arboviruses. There is no evidence that fresh chicken meat is involved in the transmission or maintenance of arbovirus infections.

[3] The entire arbovirus chapter is new, inserted in response to comments from stakeholders.

Togaviruses

Technical Information

Background

Eastern equine encephalomyelitis (EEE), Western equine encephalomyelitis (WEE) and Highlands J (HJ) viruses are arthropod-borne, circulating between birds and mosquito vectors in the Americas and Caribbean. Birds act as amplifying hosts for the viruses, which are then transmitted to susceptible hosts by mosquito vectors. While most birds show no clinical signs of infection with these viruses, some species including emus and cranes are susceptible to clinical disease. In addition to disease and mortality in birds, EEE and WEE viruses cause serious disease in horses, donkeys, mules, and in humans. HJ virus has been associated with encephalitis in horses but has not been recognised to cause disease in humans (Griffin 2007). Humans and horses act as accidental, 'dead-end' hosts for EEE, WEE and HJ viruses.

EEE and WEE are OIE-listed diseases.

Agent taxonomy

EEE, WEE and HJ viruses are serologically distinct members of the *Alphavirus* genus of the *Togaviridae* family. Alphaviruses are enveloped RNA viruses which share common antigenic sites, as revealed by haemagglutination inhibition and complement fixation tests with polyclonal sera. Seven broad antigenic groups have been identified within the alphavirus serogroup (Griffin 2007).

Agent characteristics

Togaviruses are enveloped, and are not considered to be stable outside the infective host. They are generally susceptible to inactivation by most disinfectants with 10 minutes of exposure at 20 °C to 25 °C (Prince and Prince 2001). These viruses are also sensitive to drying, heat and acidic conditions (pH 3), and are inactivated by lipid solvents such as ether and chloroform (Calisher and Walton 1996).

Epidemiology

Many species of birds are susceptible to infection with EEE, WEE and HJ viruses, and provide the reservoir for the virus in nature. The prevalence of these viruses in certain areas of the United States may be controlled by the regional distribution of the mosquitoes that transmit the viruses (Stamm and Kissling 1957).

Alphaviruses have rarely been implicated as causes of clinical disease in chickens. However, chickens are known to be susceptible to infection with these viruses and are commonly used as sentinel animals in epidemiological surveillance studies (Guy, Barnes, and Smith 1994; Guy and Malkinson 2003).

EEE virus

EEE is endemic in North America, South America and the Caribbean. South American strains are less virulent than North American strains in experimental animals (Griffin 2007).

For EEE virus, the major insect vector is the ornithophilic mosquito *Culiseta melanura*. EEE occurs in natural cycles involving birds and *Culiseta melanura* in swampy areas of the Americas during warm months. This species of mosquito feeds exclusively on passerine birds, which are amplifying hosts. Transmission between birds and other vertebrates usually involves species of mosquitoes with more varied feeding habits, such as *Aedes* and *Coquillettidia* species. Transmission in mosquito saliva can occur within four days of feeding on an infected bird (Johnston and Peters 1996).

Avian outbreaks of EEE have been reported mainly in pheasants, turkeys, ducks, chukars, pigeons (Guy and Malkinson 2003), emus and cranes (Hansen and Docherty 1999). Chickens are also susceptible to experimental infection but disease signs are not observed in birds older than 3 weeks (Guy and Malkinson 2003). Experimentally inoculated two-week-old chickens developed clinical signs of depression and inappetence, with a mortality rate of 80%. Two of five in-contact sentinel birds also developed clinical signs and died, while the remaining three sentinels survived and did not develop antibodies to EEE.

In pheasants and ducks, EEE can spread through direct contact. Feather-picking and cannibalism were found to be responsible for the rapid dissemination of the virus among susceptible pheasants, but virus transmission did not occur in pheasants through contact with aerosols of faecal material from infected birds (Ritchie 1995). In experimentally-infected pheasants, virus could be recovered from the feather quills for up to six days after infection, and persisted in this site for 24 to 48 hours longer than in the blood. Experimentally inoculated turkeys shed virus in their semen, and semen is a potential vehicle for transmission of EEE virus in this species (Guy et al. 1995). Although virus is present in the faeces and feathers of some infected birds, these sources of virus transmission are considered of little significance when compared to the likelihood of virus transmission by infected mosquitoes (Ritchie 1995).

Many birds infected with EEE virus develop viraemia within 24–72 hours after infection, but remain asymptomatic (Ritchie 1995). The incubation period in pheasants inoculated with the EEE virus by the intramuscular route averages 3.8 days. The incubation period in immunologically naive birds exposed to experimentally-infected pheasants is 4.5 days (Ritchie 1995). The viraemic period in birds generally persists for up to five days (Ritchie 1995). However, English sparrows have been shown to develop high-level viraemia that persists for up to seven days (Stamm and Kissling 1957).

EEE virus is most frequently recovered from nestling birds in areas where the virus is endemic, probably because they lack immunity, and their sparse feathering and inability to move make them easy targets for feeding mosquitoes (Ritchie 1995; Griffin 2007). Recovered birds develop high levels of antibody, which limits the duration of viraemia (Ritchie 1995).

Non-avian species, such as humans and horses, may develop disease as a result of infection with EEE virus, but do not develop sufficient viraemia to transmit the virus to arthropod vectors. EEE viraemia in horses lasts one to three days, and may be asymptomatic or associated with fever; viraemia in humans is generally undetectable.

WEE virus

WEE is endemic in the western plains and valleys of the United States and Canada and in South America (Griffin 2007). In North America, WEE virus is maintained in a cycle involving mainly passerine birds and the mosquito *Culex tarsalis.* Other major mosquito vector species include *Aedes melanimon, Ae. dorsalis* and *Ae. campestris*. In South America, antibodies to WEE virus are common in small mammals as well as birds.

Wild ducks can be experimentally inoculated with WEE virus via the oral route through drinking water (Burton et al. 1961). The elapsed time between initial ingestion of the virus by the vector and acquisition of competence for transmission to a new vertebrate host (the extrinsic incubation period) is approximately one week (Johnston and Peters 1996). For WEE, under experimental conditions, incubation periods in mosquitoes of 10 days have been observed before transmission to chickens (Hammon, Reeves, and Gray 1943).

Falls in egg production associated with WEE virus infection were identified in three turkey breeder flocks in California during the summer of 1993 and again in one flock the following year (Cooper and Medina 1999). Preliminary studies indicate that WEE virus infection results in mortality amongst nestling house finches and mourning doves (Reisen et al. 2003).

A low-titre viraemia in virus-inoculated 19-week-old hens persisted for only one day, and viraemia was undetectable in 38-week-old inoculated hens (Reisen et al. 1994). Reports of the duration of viraemia in slaughter-age chickens were not found.

Early studies demonstrated that WEE virus may persist in avian hosts for as long as 10 months, and that the virus may recirculate in the bloodstream subsequent to the period of active viraemia (Reeves et al. 1958). More recent studies indicate that a low percentage of birds experimentally infected with WEE virus developed chronic infections in the spleen or lung that could be detected by RT-PCR (Reisen et al. 2001). However, they were unable to demonstrate a recurrence of viraemia even with immunosuppressive treatment. In another study, 3.5% of birds had detectable RNA in one or more tissues six to eight weeks after inoculation with WEE virus (Reisen et al. 2003). These studies suggest that avian hosts with chronic latent infections may potentially serve as long-term reservoirs and sources of virus infection for arthropod vectors.

Highlands J virus

HJ virus is endemic in North America along the eastern coast of the United States (Griffin 2007). It is part of the WEE antigenic complex of alphaviruses. It is maintained in a cycle similar to EEE virus, with *Culiseta melanura* the main arthropod vector and migrating birds the primary reservoir.

Experimental inoculation of HJ virus into two-week-old chickens induced clinical signs of abdominal distension in only one of 30 birds (Guy, Barnes, and Smith 1994), and HJ infection has not been reported as a cause of clinical disease in naturally-occurring infections in chickens.

Outbreaks associated with mortality and reduced egg production have been reported in turkeys and chukars and HJ virus is an occasional cause of encephalitis in horses (Guy and Malkinson 2003; Griffin 2007). Experimentally-inoculated turkeys shed virus in their semen, and semen is a potential vehicle for transmission of HJ virus in this species (Guy et al. 1995).

Pathogenesis

Alphavirus replication in the susceptible host occurs near the portal of entry or in the regional lymph nodes, before release into the circulation results in a primary viraemia. The virus is disseminated through the body and then replicates in other tissues, leading to a secondary viraemia that is sufficient in many avian hosts to infect feeding insects. Viraemia may last from several days to a week.

In some avian species, such as the pheasant, the central nervous system is a primary target for EEE replication, whereas in others, such as chickens and turkeys, the virus is viscerotropic and associated with multifocal necrosis in the heart, liver, pancreas and lymphoid tissue (Griffin 2007). In cranes and some other avian species, the liver is the main affected organ, while in emus the virus causes severe enteritis (Ritchie 1995). In humans and horses, viraemia and fever occur soon after infection, although virus is generally undetectable in human blood. Neutralizing antibody fails to clear virus from the central nervous system, in which neuronal destruction occurs via direct cytopathic effects, inflammatory damage and vasculitis (Johnston and Peters 1996). The pathogenesis of WEE infection is similar to that of EEE.

Pathology

On post-mortem examination, two-week-old chickens experimentally-inoculated with EEE virus were dehydrated and had pale, enlarged livers and focal pale areas on the heart. Microscopic lesions were observed in the heart, liver, bursa, spleen, thymus and kidneys of chickens that died within six days of inoculation. Affected chickens that survived beyond six days had pericardial effusion and ascites (Guy, Barnes, and Smith 1994).

EEE infection results in few gross post-mortem findings in pheasants, with histological changes confined to the central nervous system (Guy and Malkinson 2003). Pale focal areas in the heart and splenic enlargement have been described in partridges (Guy and Malkinson 2003). Pathological changes in affected turkey flocks were minimal (Ficken et al. 1993). Reported post-mortem findings in emus infected with WEE virus include accumulation of fluid around the heart, enlarged liver and kidneys and meningitis (Ritchie 1995).

Clinical signs

Chickens experimentally-infected with EEE virus showed signs of depression, inappetence and death. Clinical signs were only observed in one of 30 two-week-old chickens inoculated with HJ virus (Guy, Barnes, and Smith 1994). Experimental inoculation of WEE virus into one-day-old chickens resulted in lethal infection, but clinical signs were not described (Hardy et al. 1997). No clinical signs were reported in adult chickens inoculated with WEE virus (Reisen et al. 1994).

One flock of turkeys was reported to have increased mortality and signs of depression and diarrhoea in association with seroconversion to EEE virus (Ficken et al. 1993). EEE virus infections have been associated with reproductive problems and decreased egg production in chukars and turkeys. Captive whooping cranes, emus, partridges, and pheasants and other affected species may show signs of lethargy, diarrhoea and ataxia before succumbing to EEE infection (Dein J.F. et al. 1986; Ritchie 1995; Guy and Malkinson 2003).

Drops in egg production of 8.7–10.1% have been reported in turkeys in association with WEE infection (Cooper and Medina 1999). Depression and neurological signs have been described in birds affected by WEE infection (Ritchie 1995).

Reduced egg production and mortalities have been reported in turkeys and recumbency and death have been reported in chukars with HJ virus infection.

Immunology

Passerine and gallinaceous birds that survive EEE will have detectable antibodies as early as five days to two weeks after infection. WEE antibodies in chickens were detected 6–10 days after experimental inoculation (Reisen et al. 1994).

The duration of immunity following natural infection in birds is not known, and seronegativity does not necessarily indicate lack of previous exposure to the virus (Kuno 2001).

Polyvalent vaccines are available for protection of horses against EEE and WEE.

Diagnosis

These viruses can be diagnosed by isolation and identification of the causative agent, detection of viral antigens using ELISA or immunohistochemistry, detection of viral RNA using RT-PCR procedures, or by serological methods including virus neutralization, haemagglutination-inhibition, ELISA and complement fixation (Guy and Malkinson 2003). When using viral isolation, the OIE recommends precautions be taken against human infection in the laboratory.

Transmission in chicken meat

Transmission of these viruses in nature is between mosquitoes and some species of birds which act as amplifying hosts. Oral transmission of EEE virus has been reported in viraemic pheasants, and under experimental conditions, ducks and pheasants can be orally infected with WEE and EEE viruses (Burton et al. 1961; Holden 1955). Episodes of clinical disease have not been confirmed in naturally-infected chickens, although young chickens are susceptible to experimental infection.

Despite the evidence that oral transmission of these viruses is possible in some species of birds, there is no evidence that transmission of these viruses has ever been associated with human or animal consumption of meat of infected chickens. Despite decades of trade in fresh chicken meat from countries endemically infected with EEE, WEE and HJ virus, these viruses have remained within established geographical limits in the Americas. The Australian Government Department of Health and Ageing (DoHA) sought advice from the National Arbovirus and Malaria Advisory Committee (NAMAC) on the risk of transmission of arboviruses to mhumans through imported chicken meat. The advice received from NAMAC was that the risk of importation and establishment of these viruses via imported uncooked chicken carcasses is negligible.

Quarantine Significance

EEE and WEE are OIE-listed diseases. EEE and WEE are also notifiable in all States of Australia. Equine encephalitides are not covered in the AUSVETPLAN.

Two hundred confirmed human cases of EEE and 639 human cases of WEE were reported in the United States between 1964 and 2001 (Centers for Disease Control 2003a). The total case costs ranged from $US 21,000 for transiently infected individuals to $US3 million for severely infected individuals. Insecticide applications can cost as much as $US1.4 million depending on the size of area treated (Centers for Disease Control 2003a).

After considering the available evidence suggesting that there is a negligible likelihood of introduction of these viruses in chicken meat, the IRA team concluded that no further risk assessment for alphaviruses was necessary.

Flaviviruses

Technical Information

Background

Flaviviridae, a large family of RNA viruses, currently consists of three genera – *Flavivirus*, *Pestivirus* and *Hepacivirus* – and two groups of unassigned viruses (Lindenbach, Thiel, and Rice 2007). The genus *Flavivirus* includes more than 70 viruses, including two that have been documented to cause disease in farmed poultry (Israel turkey meningoencephalitis virus and West Nile virus) (Guy and Malkinson 2003).

Most flaviviruses are arboviruses, transmitted by arthropod vectors, such as mosquitoes and ticks, and most replicate alternately in susceptible vertebrate and arthropod hosts (Mackenzie, Barrett, and Deubel 2002). However, some viruses can be transmitted directly from vertebrate host to vertebrate host (Gubler, Kuno, and Markoff 2007).

Agent characteristics

Flaviviruses are enveloped and therefore readily inactivated by organic solvents and detergents. They are inactivated by 3–8% formaldehyde, 2% glutaraldehyde, 2–3% hydrogen peroxide; 500 to 5000ppm chlorine, alcohol, 1% iodine and phenol iodophors, by ultraviolet light and gamma irradiation, and by heating to 56 ºC for 30 minutes in biological fluids, although they are stable when frozen. They are most stable between pH 8.4–8.8 and are generally sensitive to acid pH (Monath and Heinz 1996).

West Nile virus (WNV) has been recovered from samples air-dried on filter paper stored at room temperature for up to 60 days (Guzman et al. 2005).

Israel Turkey Meningoencephalitis Virus

This virus is a member of the Ntaya virus group of Flaviviruses (Fauquet et al. 2005). The virus has been isolated from turkeys in Israel and South Africa, and is transmitted by biting midges (*Culicoides* species) and some mosquitos (Komarov and Kalmar 1960; Barnard et al. 1980; Gubler, Kuno, and Markoff 2007).

IT has been reported only in turkeys (Guy and Malkinson 2003), although quail are susceptible to experimental infection. Some day-old chicks inoculated intramuscularly and intracerebrally died within one to two weeks of inoculation with IT virus. However, three-week-old chicks and pullets inoculated intracerebrally showed no clinical effects. The authors of that study concluded that chickens, ducks, geese and pigeons are refractory to infection with IT virus (Komarov and Kalmar 1960).

Because IT virus is a disease of turkeys only, with a limited geographic distribution, and chickens appear to be refractory to infection, this virus is not considered further in this IRA report.

Japanese Encephalitis Virus

Agent taxonomy

JE virus belongs to the family *Flaviviridae* and genus *Flavivirus*. The Japanese encephalitis serological group also includes West Nile virus (WNV), Murray Valley encephalitis (MVE) virus and St Louis encephalitis (SLE) virus (Gubler, Kuno, and Markoff 2007). Murray Valley encephalitis, the related Alfuy virus and Kunjin virus, a subtype of WNV, are enzootic in Australia (Mackenzie, Barrett, and Deubel 2002).

Epidemiology

JE virus is endemic in southern and eastern Asia, with its geographic range extending from India and Sri Lanka to Japan and the Philippines, and from maritime Siberia in the north, to the Indonesian archipelago and New Guinea in the south. The distribution of JE virus is significantly linked to irrigated rice production and pig-rearing (Endy and Nisalak 2002). In 1995, JE virus was first detected in the Torres Strait islands, and in 1998 a single human isolation was made in mainland Queensland (Endy and Nisalak 2002). Simultaneously, pigs in the Cape York area of northern Queensland were shown to have seroconverted to JE virus. Seroconversions of sentinel pigs have been regularly documented in the Torres Strait islands, but no further locally acquired human cases have been documented on the Australian mainland (Liu et al. 2006).

Birds and pigs serve as effective amplifier hosts for JE virus (Gubler, Kuno, and Markoff 2007). Ardeid birds (herons and egrets) are believed to be the natural wildlife hosts for JE virus; however, many species of birds are susceptible to infection while showing no clinical signs of disease. Susceptible avian species, other than herons and egrets, include chickens, house finches, blackbirds (Hammon, Reeves, and Sather 1951), ducks (Dhanda et al. 1977), pigeons (Chunikhin and Takahashi 1971), Japanese tree sparrows (Hasegawa, Takehara, and Takahashi 1975), Indian Starling, crow, pied mynah, kingfisher (Sarkar, Mal, and Malik 1995) and other species. Australian bird species susceptible to experimental infection include Rufous or Nankeen night herons (*Nycticorax caledonicus*), Pacific herons (*Ardea pacifica*), and egrets (*Egretta* species) (Boyle, Dickerman, and Marshall 1983). Other potential Australian hosts include bitterns, ducks and cormorants, which have been implicated in JE virus transmission in other countries (Mackenzie et al. 2002). Chickens in endemic areas have a high JE antibody seroprevalence (Endy and Nisalak 2002; Ting et al. 2004).

Other vertebrate hosts include pigs, cattle, buffalo, sheep, goats, bats, horses and humans (Endy and Nisalak 2002; Mackenzie et al. 2002). Pigs (domestic and feral) serve as important amplifying hosts in endemic areas, although they are not necessary for the basic mosquito-vertebrate transmission cycle to occur (Rosen 1986). Bats develop a prolonged viraemia after infection with JE virus and may serve as potential amplifying hosts, while cattle may be involved in natural transmission cycles in some countries, such as Indonesia (Mackenzie et al. 2002). The potential role of marsupials in JE virus transmission is still unclear. Most host species, other than pigs, horses and humans, do not exhibit disease in response to infection with JE virus. JE infection can cause reproductive losses in pigs, and both horses and humans are susceptible to fatal encephalitis (Rosen 1986; Ellis, Daniels, and Banks 2000).

Insect vectors for JE include *Culex* spp., *Mansonia* spp., *Aedes* spp., some *Anopheles* spp. and *Ochlerotatus* spp (Endy and Nisalak 2002; Mackenzie et al. 2002). Mosquitoes capable of transmitting JE are present in Australia (Endy and Nisalak 2002). Behavioural characteristics, such as feeding behaviour and host preference, determine the efficiency of transmission by each species of mosquito vector.

Infection of insect vectors is dependent on the ingestion of a blood meal from a viraemic host. The level of virus in the blood must be sufficient to establish midgut infection in the vector. Once ingested, flaviviruses replicate in the insect gut, with subsequent spread to the salivary tissues, from which they are inoculated into the host during feeding (Monath and Heinz 1996; Tyler K.L. and Fields 1996). Mosquitoes held for 9–12 days after ingesting a blood-meal from a viraemic avian host were capable of transmitting virus to uninfected birds (Soman et al. 1977; Dhanda et al. 1977).

Young birds (nestlings) are more susceptible to infection than older birds. Transmission of infection via eggs has not been reported. Young pigs are susceptible to infection after waning of maternal antibody (in endemic areas). Pigs are important amplifying hosts because of the high turnover rate of the population and continuous replenishment of a susceptible young population (Endy and Nisalak 2002).

Birds do not show signs of disease following infection with JE virus, however, viraemia has been detected 2–6 days after feeding by infected mosquitoes (Soman et al. 1977). Experimentally infected ardeid birds have a viraemic period of two to four days, with occasional birds being viraemic for at least eight days post-inoculation (Soman et al. 1977). Pigs develop a viraemia of two to four days duration, with viral levels capable of infecting mosquito vectors (Endy and Nisalak 2002). The viraemia in other species is, as a rule, insufficient to infect insect vectors.

JE virus has been detected in the kidney and liver of a pigeon 39 days after experimental inoculation; however secondary viraemia was not detected in any pigeon during a 15 week observation period (Chunikhin and Takahashi 1971). Persistence or recurrence of viraemia, sufficient to infect vectors, has not been reported in birds following the end of the primary viraemia.

Humans, horses and pigs are the only vertebrate species in which JE virus is known to cause disease under natural conditions (Rosen 1986). In humans in endemic areas, the ratio of inapparent to apparent infections is 200:1 to 300:1, with age, acquired immunity and virus strain influencing the ratio (Monath and Heinz 1996). During epidemics, higher attack rates are seen, and approximately 35,000 cases and 10,000 deaths are recognised annually in Asia, with the disease being greatly underreported. Children and the elderly are at increased risk of infection with clinical disease; the case fatality rate varies from 5–40% (Monath and Heinz 1996). In horses, inapparent infections are more common than cases of recognised clinical disease. However, case fatality rates of 5–15% in endemic areas, and 30–40% in epidemics, have been reported (Ellis, Daniels, and Banks 2000). In endemic areas, horses are frequently vaccinated against JE. In pigs, reproductive failures due to foetal encephalitis, abortions, still births in sows and hypospermia in boars may occur (Mackenzie et al. 1998).

Pathogenesis

Following inoculation of virus into a susceptible host, primary replication occurs at the site of entry (dermal or subcutaneous tissues), followed by spread to regional lymph nodes then via the

lymphatic system to the systemic circulation (Tyler K.L. and Fields 1996). After a primary viraemia, further viral replication may occur in extraneural tissues, from which virus may be re-released into the circulation. The viraemia is terminated by the appearance of humoral antibodies, usually within a week after infection (Monath and Heinz 1996). In some species, viral invasion of the central nervous system occurs, followed by the onset of neurological signs.

Day-old chicks inoculated intracerebrally or intravenously with JE virus showed no clinical signs, but the virus had a wide tissue distribution, with higher concentrations in the blood than in the brain. When 45-day-old chickens were inoculated intravenously and killed 48 hours later the virus was detected only in the blood and was not detectable in the other organs tested, which included skeletal muscle, heart, liver and bone marrow (Miyamoto and Nakamura 1969).

Pathology

Pathological changes including splenomegaly have been described in chicken embryos inoculated with JE virus. However, chickens inoculated at one, three and 45 days of age remained clinically normal and no post-mortem changes were described (Miyamoto and Nakamura 1969).

Transplacental infections in pigs leads to foetal encephalitis, abortion and stillbirths (Gubler, Kuno, and Markoff 2007). A nonsuppurative encephalitis with neuronal degeneration may be seen in pigs up to six months of age (Joo and Chu 1999).

In affected horses, there is microscopic evidence of diffuse nonsuppurative encephalomyelitis (Ellis, Daniels, and Banks 2000).

Immunology

Birds are not vaccinated against JE virus. In one study, the available inactivated JE vaccine (JE-VAX) was evaluated for its potential to protect birds against WNV. The vaccine appeared to be safe to use in birds, but did not induce antibody response in all birds, and nor did it induce a neutralising antibody response to WNV in previously seronegative birds (Clippinger et al. 2001).

Vaccination of humans, particularly children, is practiced in some Asian countries, including Japan, Korea and parts of China. However, the cost of vaccination in some developing countries, and the prevalence of adverse effects, preclude widespread vaccination of both the local population and travellers. Vaccination of horses is practiced in many parts of Asia, and vaccination of racehorses is compulsory in some areas, such as Japan, Singapore, Malaysia, Macau and Hong Kong. Vaccination of breeder pigs is recommended in endemic areas (Joo and Chu 1999). However, the high turnover rate of young pigs raised for meat production makes widespread vaccination uneconomical.

Diagnosis

There is a high level of antibody cross-reactivity between flaviviruses, making serological diagnosis problematic in areas where more than one flavivirus circulate in susceptible populations (Endy and Nisalak 2002; Gubler, Kuno, and Markoff 2007). JE virus can be isolated by intracerebral inoculation of susceptible mice or cell cultures with homogenates prepared from tissue specimens, heparinized whole blood, blood clots or mosquitos. Viral isolates can be identified as flaviviruses by ELISA or haemagglutination inhibition and can be confirmed as JE virus using serum neutralization or PCR tests.

Transmission in chicken meat

The oral transmission of JE virus has not been reported. Aerosol infection has been used experimentally in mice, hamsters, guinea pigs, rats and squirrel monkeys (Larson, Dominik, and Slone 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.

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 chicken meat, either orally to another (carnivorous) host or to a mosquito vector is unknown. However, when 45-day-old chickens were inoculated intravenously with JE virus, the virus could be detected at a low level only in the blood, but not in skeletal muscle, bone marrow, heart or liver 48 hours later. The authors of the study concluded that infection of chickens at this age or older, if it occurs, may be 'extremely limited' in both tissue distribution and intensity of viraemia (Miyamoto and Nakamura 1969).

Virus titres as low as $10^{1.2}$ suckling mouse intracerebral LD_{50} in 0.03 ml of horse blood have been used to infect the mosquito *C. tritaeniorhynchus* (Gould, et al. 1964) but it is not known whether titres of this magnitude would remain in 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 (Biosecurity Australia 2004).

DoHA sought advice from NAMAC on the risk of transmission to humans of arboviruses through imported chicken meat. The advice received from NAMAC was that the risk of importation and establishment of these viruses via imported uncooked chicken carcasses is negligible.

Quarantine Significance

JE is included in the EAD Response Agreement of Australia as a Category 1 disease. Category 1 diseases are emergency animal diseases that predominantly seriously affect human health and/or the environment (depletion of native fauna) but may only have minimal direct consequences to the livestock industries (Animal Health Australia 2006). JE is covered by AUSVETPLAN. Surveillance for JE virus is carried out in northern areas of Australia and results of surveillance are reported annually (Liu et al. 2006). The disease is nationally notifiable in Australia.

About 50,000 human cases and 10,000 deaths are recognised annually throughout Asia, but the disease is greatly under-reported (Gubler, Kuno, and Markoff 2007). The incidence in some countries is declining because of widespread vaccination. In other areas, an increase in JE transmission has been associated with agricultural development and irrigation for rice

cultivation, with a resultant increase in mosquito vectors, and with pig breeding (Gubler, Kuno, and Markoff 2007).

JE virus has been detected several times in sentinel pigs and in mosquito traps on the Australian mainland, although only one locally-acquired human case has been reported in mainland Australia.

Chickens are not recorded as playing a significant part in the natural transmission of JE in endemic areas. However, pigs are recognised as amplifier hosts, and proximity of pigs is a risk factor for human infection in endemic areas (Endy and Nisalak 2002; Liu et al. 2006).

The unrestricted annual risk estimate for introduction of JE virus in pig meat has been previously determined to be extremely low or negligible (Biosecurity Australia 2004). DoHA 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 (Biosecurity Australia 2004).

Risk management for JE virus was not required for pig meat imports, and therefore, the IRA team concluded that no further risk assessment was necessary for JE virus in chicken meat.

West Nile Virus

West Nile virus (WNV) is a member of the JE virus antigenic complex of arthropod-borne flaviviruses. WNV, like other members of the JE complex, typically circulates in nature between *Culex* mosquitoes and avian reservoir hosts in sylvatic transmission cycles. Humans and horses are considered as incidental (dead-end) hosts (Steele et al. 2000). A percentage of infected humans and horses develop clinical signs of encephalitis, which may be fatal. Birds are the primary amplifying hosts (Monath and Heinz 1996).

WNV is an OIE-listed disease.

Agent taxonomy

WNV is a *Flavivirus* belonging to the family *Flaviviridae*. Two lineages of WNV are described (I and II), based on genetic sequencing (Scherrett et al. 2001).

Epidemiology

Lineage I strains of WNV have been isolated from Africa, India, Europe, the Middle East and North America. All viruses isolated in the past decade during WNV outbreaks of human or avian disease belong to Lineage I (Scherrett et al. 2001). WNV spread to the United States in 1999, and subsequently to Canada, causing fatal neurological disease in humans, horses and a variety of native and exotic bird species (Steele et al. 2000). WNV has since spread to central and South America and the Caribbean (Komar and Clark 2006; Bosch et al. 2007). Kunjin virus, a subtype of Lineage I WNV, is present in Australia (Hayes 2001). The Kunjin virus occasionally causes mild encephalitis in humans with full recovery occurring after a few weeks. In an experimental study, house sparrows inoculated with Kunjin virus developed low viraemia and suffered no mortality, with authors concluding that the strain appeared to be ‘non-virulent’ (Langevin et al. 2005).

Lineage II strains are maintained in epizootic foci in West, Central and East Africa and Madagascar (Scherrett et al. 2001), and have not been associated with human encephalitis cases (McLean et al. 2002).

Wild birds are the principal hosts of WNV. The virus has been isolated from a number of wetland and terrestrial avian species in diverse areas. Migratory birds are also suspected as instrumental in the introduction of the virus to temperate areas during spring migration (Rappole, Derrickson, and Hubalek 2000).

In the 1999 United States epizootic involving birds in zoological gardens, 27 birds that died were of 14 different species that represented the following eight diverse orders: *Passeriformes*, *Ciconiiformes*, *Pelicaniformes*, *Charadriiformes*, *Anseriformes*, *Galliformes*, *Falciniformes*, and *Strigiformes* (Steele et al. 2000). Deaths in wild birds at first appeared to be restricted to the order *Passeriformes*, and corvids accounted for over 80% of the deaths. However, deaths have since been reported in wild and captive raptors and owls (Wunschmann et al. 2005; Wunschmann et al. 2004; Gancz et al. 2004b; Gancz et al. 2004a). WNV has been isolated from parrots in Madagascar (Fontenille et al. 1989) and deaths have been reported in many species of birds in North America due to WNV since 2000 (Komar 2003).

Although turkeys and chickens generally do not show clinical signs of infection with WNV, infected domestic geese may present with neurological signs and high morbidity and mortality (Malkinson and Banet 2002). Neurological signs and death have been reported in ducks and geese on a mixed species commercial waterfowl farm in the United States (Meece et al. 2006). Transmission of WNV from asymptomatic turkeys to farm workers has also been recorded (Glaser et al. 2003).

WNV has also been demonstrated in mammals and reptiles, although they are less important than birds in maintaining transmission cycles of the virus in ecosystems. WNV or antigen has been demonstrated in bats, chipmunks, raccoons, skunks, squirrels, domestic rabbits (Centers for Disease Control 2003b), canids, including a dog and a wolf (Lichtensteiger et al. 2003), and alligators (Klenk et al. 2004; Jacobson et al. 2005).

Cats experimentally infected with WNV by mosquito bite developed detectable viraemia and displayed lethargy and a fluctuant febrile response for several days. Cats infected by ingestion of WNV-inoculated mice also developed viraemia, but displayed no clinical signs of infection. The peak viraemia developed by infected cats was considered sufficient to infect some mosquito hosts at a low level of efficiency. Experimentally-infected dogs developed no clinical signs, and the low-level viraemia they developed was considered insufficient to infect mosquitos (Austgen et al. 2004).

The natural life cycle of WNV involves the transmission of virus from mosquitoes to wild birds. High viraemia of several days' duration in wild birds leads to infection of susceptible mosquitoes to complete the life cycle (McLean et al. 2002). The virus has been isolated from numerous mosquito species, predominantly of the genus *Culex*, but also from *Aedes*, *Ochlerotatus, Anopheles*, *Mimomyia* and *Mansonia* species (Monath and Heinz 1996; Turell et al. 2005). The *Culex* spp. and *Aedes* spp. are considered to be important vectors (Turell et al. 2005). WNV has been isolated from hibernating *Culex* mosquitoes in the winter, and may be vertically transmitted between generations of mosquitoes (McLean et al. 2002). Australian *Culex spp* are competent vectors for Kunjin and Murray Valley Encephalitis viruses and, therefore, can be expected to be able to transmit WNV. Virus isolations have occasionally been reported from other hematophagous arthropods such as bird-feeding ticks (Monath and Heinz 1996).

There is experimental and circumstantial evidence of bird-to-bird transfer in American crows, ring-bill gulls, black-billed magpies, blue jays (Komar et al. 2003), and geese (Banet-Noach, Simanov, and Malkinson 2003; Austin et al. 2004) with other species, including raptors, being confirmed as susceptible to orally-acquired WNV infection (Komar et al. 2003). WNV is excreted in faeces and is present in oral secretions of infected birds for up to 9–10 days (Komar et al. 2003).

In birds experimentally exposed to WNV by infectious mosquito bite, death occurred from 3–13 days post-inoculation (Komar et al. 2003). In humans and horses, the incubation period is thought to be between three and ninedays. WNV has an incubation period of 10–14 days in the mosquito, after which it can infect birds, animals or humans (Centers for Disease Control 2003b).

Clinical signs

Chickens and turkeys show few or no clinical signs of infection with WNV. However, young chicks are known to succumb to WNV infection (Komar 2003). Domestic geese can develop

neurological disease from WNV infection (Guy and Malkinson 2003), as can some waterfowl (Meece et al. 2006; Austin et al. 2004).

Infected psittacines show neurological signs, including incoordination and seizures (Clubb 2002). Clinical signs in zoo birds of a variety of species included neurological signs, anorexia, depression and weight loss (D'Agostino and Isaza 2004). Owls and raptors may show evidence of chronic disease, including emaciation, neurological signs, and impaired vision over a period of one to four weeks (Wunschmann et al. 2005).

Pathogenesis

Early experimental studies in chickens indicate that the outcome of exposure was affected by the age of the chicken (McLean et al. 2002). Chicks aged between one and eleven days were infected by exposure to infected mosquitoes, but three-week-old chicks were more resistant to infection (Taylor et al. 1956; McLean et al. 2002). Seven-week-old SPF chickens inoculated with WNV developed no clinical signs, but virus was re-isolated from the blood for up to eight days post-inoculation, and from myocardium, spleen, kidney, lung and intestine for up to 10 days (Senne et al. 2000). Although the authors of that study concluded that chickens during peak viraemia could infect competent mosquitos, the authors of a later study disputed this assertion, at least for the New York strain of WNV and the predominant mosquito vector in the United States, *Culex pipiens* (Langevin et al. 2001). They proposed that chickens are a suitable sentinel species for WNV, because they are susceptible to infection, produce detectable antibodies, do not show clinical disease, and are unlikely to infect either mosquitos or their flock mates through direct contact (Langevin et al. 2001).

Experimental studies show the viraemic period varying from 2–10 days in turkeys (Swayne, Beck, and Zaki 2000). High and long-term (four to seven days) viraemia, sufficient to infect vector mosquitoes, has been observed in some birds (Komar et al. 2003). Of the 25 bird species examined, passerine species produced viremia greater in magnitude and duration, and psittacines and gallinaceous birds (pheasants and quail) the lowest titres and shortest duration of viremia. The virus may persist in the internal organs for at least 14 days in some avian species (Komar et al. 2003), and has been documented in one study to persist for 20–100 days in the organs of inoculated ducks and pigeons (Sharp et al. 1993). In addition to passerines, juvenile alligators have been shown to have a maximum duration of viremia of over two weeks, suggesting that they may play a role in WNV transmission in the wild (Klenk et al. 2004).

In susceptible bird species experimentally infected by exposure to infectious mosquito bite, the viraemic period persisted for around seven days, although a second viraemia was observed in at least one bird at day 11, just before its death (Komar et al. 2003). Signs of illness, including lethargy, ruffled feathers and neurological signs were followed in most cases by death within 24 hours. Virus was detectable in oral secretions and faeces of some bird species, including passerine birds (sparrows and crows) after several days of viraemia, with shedding persisting for 9–10 days. Some birds, including crows, sparrows and an owl, were susceptible to orally acquired infection in the absence of mosquitos. In some cases, the oral infection was acquired by feeding infected mosquitos, water, or carcasses of infected birds or mice; in others, the mechanism of transmission was unknown but was thought to be related to contact with infected secretions (Komar et al. 2003). WNV shows tropism for the central and peripheral nervous systems, the myocardium, cells of the mononuclear phagocyte system, multiple epithelial cell types, fibrous connective tissues and oocytes (Steele et al. 2000). Virus can be isolated from multiple organs for at least 13 days beyond the period of viraemia (Komar et al. 2003).

In experimentally infected mice, virus was found in lymphoid organs, followed by the heart and kidney, with viraemia peaking two to three days after inoculation. While virus was found in brain as early as 28 hours after infection, it was consistently present in brain tissue by day 4 (Kramer and Bernard 2001).

Horses developed low-level viraemias one to six days after exposure to mosquitoes experimentally infected with WNV. One experimentally infected horse, which became febrile and showed neurological signs eight days after infection, developed encephalomyelitis and high titres of virus in the brain and spinal cord. However, the horses did not develop viraemias of sufficient magnitude to re-infect susceptible mosquitoes, and the authors concluded that horses are unlikely to serve as important amplifying hosts for WNV in nature (Bunning et al. 2002).

Pathology

In chickens, there may be no gross evidence of infection, although in some cases white flecks may be visible on the heart surfaces and myocardium (Senne et al. 2000). Histopathological evidence of myocarditis, nephritis, pneumonitis and encephalitis has been reported following experimental infection of chickens (Senne et al. 2000).

In some birds, brain haemorrhage, enlarged spleen and kidneys and intestinal inflammation or haemorrhage are evident (Steele et al. 2000). External haemorrhage from the cloaca or mouth is sometimes observed in corvids dying of WNV infection (Komar et al. 2003), and emaciation is seen in infected raptors (Wunschmann et al. 2005; Wunschmann et al. 2004).

Immunology

Birds develop humoral antibody after exposure to WNV (Komar et al. 2001) however, it is not known how long birds remain seropositive, and seronegativity does not necessarily indicate lack of previous exposure to the virus (Kuno 2001). Seroprevalence rates in wild-caught rock pigeons (*Columba livia*) in the United States varied seasonally between 11% and 50% (Allison et al. 2004). Antibody to WNV can be detected as early as five to seven days after experimental inoculation in chickens (Senne et al. 2000) and turkeys (Swayne, Beck, and Zaki 2000).

Commercial WNV vaccines are available for use in horses in the United States (American Association of Equine Practitioners 2005). Other vaccines have been experimentally evaluated for use in birds (Turell et al. 2003). The inactivated equine vaccine has been used to vaccinate zoo and other birds, with limited success. Some bird species fail to produce detectable antibody after vaccination with the inactivated equine WNV vaccine (Nusbaum et al. 2003).

Diagnosis

Diagnosis of WNV infection based on laboratory testing is complicated by the antigenic cross-reactivity of members of the JE serocomplex of viruses. Diagnosis can be confirmed by virus isolation, detection of virus RNA or from serological evidence of recent infection. Antemortem diagnosis of infection can be difficult in live birds, and a combination of tests may be required (D'Agostino and Isaza 2004).

Virus isolation: Virus isolation from serum, cerebrospinal fluid (CSF) or tissue specimens can be amplified in cell culture or in suckling mice, and identified by virus neutralization assays using antisera, by ELISA or by detection of WNV-specific sequences (Komar 2000). However,

virus isolation requires special facilities that are not widely available (D'Agostino and Isaza 2004).

Detection of virus RNA: Viral RNA or antigen can be detected using reverse transcriptase PCR, immunohistochemistry or antigen capture assay on post-mortem tissue. However, the tests lack sensitivity in some species (Gancz et al. 2004b; Wunschmann et al. 2005).

Serology: The IgM and IgG ELISA are first line testing for human and animal serum and cerebrospinal fluid specimens. In addition plaque reduction neutralisation tests (PRNTs) will identify specific flavivirus antibody. In birds, indirect ELISA testing for diverse orders of avian species has been evaluated recently. IgG isolated from the sera of four species representing four orders, namely *Passeriformes*, *Columbiformes*, *Galliformes* and *Anseriformes* showed reactivity in 23 species from 12 avian orders. It did not detect positive sera from the orders *Ciconiiformes*, *Gruiformes* and *Charadriiformes* (Ebel et al. 2002).

The epitope blocking ELISA assays also provide a rapid, reliable, and inexpensive diagnostic and surveillance technique to monitor WNV activity in multiple avian species (Blitvich et al. 2003).

Serologic diagnosis of WNV in birds can be achieved by PRNT. However, PRNTs for type specific diagnosis are laborious, expensive, require live virus and for these reasons are not ideal for large scale testing (Blitvich et al. 2003).

Transmission in chicken meat

Transmission of WNV in nature is between mosquitoes and some species of birds which act as amplifying hosts.

Oral transmission of WNV has been reported in birds and other animals consuming whole infected carcasses. WNV persists for some days in the heart, brain and some other organs of infected animals and these organs may serve as sources of infectious virus if affected animals are eaten by predators. Oral and contact transmission of WNV in chickens are rare (Langevin et al. 2001), but direct transmission between turkeys has been proposed as a means of spread of WNV on a commercial turkey farm (Glaser et al. 2003). Chickens are not an efficient amplifier host for WNV, and there is no evidence that transmission of this virus has ever been associated with human or animal consumption of the meat of commercially slaughtered chickens.

Oral transmission of WNV has been reported in birds and mammals consuming all or part of the carcasses of infected animals. However, there is no evidence that fresh chicken meat is involved in the transmission or maintenance of WNV infection. In countries with endemic WNV, the epidemiological spread of the virus is more consistent with spread by vectors and not by spread via distribution of chicken meat. Chickens are unlikely to infect either mosquitos or their flock mates through direct contact because they are not believed to develop sufficiently high viraemia (Langevin et al. 2001). Transmission of WNV to humans via handling or eating infected meat has not been recorded, suggesting that the ‘risk of WNV infection though these behaviours is exceedingly low, or possibly overlooked’ (Komar 2003). No precautions are taken to prevent infection of humans working in chicken processing plants, or to prevent contact by humans or animals with chicken meat, in countries with endemic WNV.

DoHA sought advice from NAMAC on the risk of transmission of arboviruses through imported chicken meat. The advice received from NAMAC was that the risk of importation and

establishment of these viruses via imported uncooked chicken carcasses is negligible. For these reasons, the IRA team considered that no further risk assessment was necessary for WNV.

Quarantine Significance

WNV is currently not included in the EAD Response Agreement of Australia, and is not covered by the AUSVETPLAN. It is nationally notifiable in Australia. WNV is an OIE-listed disease agent. Kunjin virus, which is a subtype of WNV, is present in Australia.

The establishment and spread of WNV has had a significant impact in the United States since 1999. The virus spread from the east coast to the west coast in four years, with major epidemics of neurological disease occurring in 2002 and 2003 in humans and horses. The movement of the virus westward was compatible with bird migratory pathways. As of October 2005, 16,706 human cases of WNV illness were recorded in the United States, with over 7,000 cases of neurological disease and 666 deaths (Gubler, Kuno, and Markoff 2007). Although most WNV infections in humans are asymptomatic, overt disease occurs in approximately 1% of infections (Nash et al. 2001).

WNV has also affected many bird species, with 284 species being reported to the Centers for Disease Control (CDC) avian mortality data base between 1999 and January 2005 (Centers for Disease Control 2005b). Equine cases of WNV have been reported throughout the United States and more recently in South America. According to the,CDC approximately 40% of clinical equine cases result in the death of the horse (Centers for Disease Control 2003c).

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Risk Management

The method adopted by Biosecurity Australia for performing import risk analysis conforms to that recommended by the OIE, and is described in earlier sections of this report. The method for risk management described here is consistent with that described by the OIE, and is applied in turn to each of the disease agents identified following the risk assessment step.

Because of the generic nature of this risk analysis, the IRA team has based its evaluation of the effectiveness of risk management measures on their estimates of the most likely situation in an infected country. Where exporting countries can provide specific data on their own disease status, Biosecurity Australia will reconsider the risk assessment based on that data.

In the risk assessment chapters, the IRA team assessed the unrestricted risk estimate for each disease agent, to ascertain whether it exceeded Australia's ALOP. In cases where the unrestricted risk was found to be 'very low' or 'negligible' it was concluded that no risk management measures were required for that disease agent. In situations where the unrestricted risk estimation has confirmed that the biosecurity risk associated with importation is in excess of Australia's ALOP, consideration is given to measures that could be used to reduce the biosecurity risks associated with the importation of the commodity to acceptable levels (i.e. very low to negligible).

For the following disease agents, the unrestricted risk estimate was higher than Australia's ALOP, and risk management measures were deemed necessary (Table 124).

Table 124. Disease agents requiring risk management

Disease Agent	Unrestricted annual risk
Highly pathogenic notifiable avian influenza virus	High
Low pathogenicity notifiable avian influenza viruses	Moderate
Newcastle disease virus	Moderate
Very virulent infectious bursal disease virus	Moderate
Exotic antigenic variant strains of infectious bursal disease virus	Low
Salmonella Pullorum and *S.* Gallinarum	Low
Salmonella Enteritidis and multi-drug resistant *S.* Typhimurium	Moderate

In this chapter, having identified and evaluated the disease agents that would require risk management before importation could be permitted, the least trade restrictive risk management measures that could be applied, which would achieve Australia's ALOP, were evaluated. These options were selected from a range of measures considered practicable by the IRA team, and form the basis for the recommendations for the importation of chicken meat. However, alternative risk management measures that are demonstrated, to the satisfaction of Australian Government authorities, to provide equivalent quarantine protection will also be considered. Those seeking to propose alternative risk management measures should provide a submission

for consideration, and should include supporting scientific data that explain the extent to which the alternative measures would achieve Australia's ALOP.

The method for risk management was described in detail in Part B of this IRA report (page 101). 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 poultry 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.

Risk management measures can either:

- reduce the likelihood of the disease agent's entry in imported commodities by imposing conditions on one or more steps in the release scenario (i.e. pre-import measures), or
- reduce the likelihood that susceptible host species in Australia would be exposed to the pathogenic agent in a contaminated imported commodity or in products or waste derived from that commodity, by imposing conditions on one or more steps in the exposure scenario (i.e. post-import measures).

Pre-import measures

Risk management options considered by the IRA team included:

OPTION 1: Allow import from countries or zones free of the pathogen of concern. As discussed above, this option reduces the risk to negligible and is considered to achieve Australia's ALOP.

OPTION 2: Allow import of product which has been processed off-shore to ensure destruction of the pathogen of concern. As discussed above, this option reduces the risk to negligible and is considered to achieve Australia's ALOP.

OPTION 3: Allow import of product subject to the product being processed post-arrival, at an establishment that is operating under a compliance agreement for this purpose with AQIS, to ensure destruction of the pathogen of concern. All packaging associated with the imported product is to be treated as quarantinable waste, and to minimise risks associated with transport of the unprocessed product, the processing establishment should ideally be located in the port of entry. As discussed above, this option reduces the risk to negligible and is considered to achieve Australia's ALOP.

OPTION 4: Allow importation of bone-in cuts only. The effect of this option on the various steps in the release and exposure pathways was discussed on pages 105–109 of Part B.

OPTION 5: Allow importation of boneless cuts only. The effect of this option on the various steps in the release and exposure pathways was discussed on pages 105–109 of Part B.

Post-import measures

Australia has a long history of implementing measures to reduce the likelihood of susceptible host exposure, for example farmer awareness, and biosecurity practices. 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 access of wild birds to waste products and to some classes of poultry. The programs that are in 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 HPNAI and Newcastle disease, are serious both in terms of reduced production efficiency through increased costs and loss of access to export markets, and impact on native fauna.

Australia is committed to exotic disease preparedness and will continue to investigate and develop emergency programs for the rapid identification of exotic disease outbreaks, for limiting the impact of exotic disease incursions, and for stamping out of exotic diseases.

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 to the country's veterinary services having the capacity to determine and maintain disease freedom.

Another example was the requirement that the product be treated by cooking or other means that have been demonstrated, to the satisfaction of Australian Government authorities, to effectively destroy the infective agent. Such processing, if undertaken off-shore, and in combination with measures to ensure protection from post-processing contamination, would reduce the risk to negligible. Similarly, processing could be carried out on-shore at an establishment that has entered into a compliance agreement for this purpose with AQIS. The compliance agreement would cover such things as the siting of the processing establishment at the port of entry, and disposal of packaging, waste water and trimmings.

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. The end result of the risk management measure was evaluated by modifying the parameters of the risk simulation model 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.

Biosecurity Australia recognises that some exporting countries may wish to make a claim for access based on equivalent risk management measures, such as flock accreditation schemes or the concept of compartmentalisation, recently introduced by the OIE. These would need to be assessed on a case-by-case basis. A rigorous assessment of any application for approval of compartmentalisation or flock accreditation schemes will be undertaken to ensure that effective biosecurity measures are implemented and maintained throughout the complete chain from farm to slaughter to export. A detailed submission will need to be provided by the veterinary authority of the exporting country and Australia will conduct an on-ground assessment of the proposed compartment or flock accreditation scheme. In order for Australian Government 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.

Auditing and monitoring of application of requirements

Where generic conditions for the importation of animals or animal products are developed as a result of a generic risk analysis, it will generally be appropriate to specify as part of the conditions that permits will only be issued for importations from countries that have been specifically approved by Australia. Australia's 'Guidelines for the approval of countries to export animals (including fish) and their products to Australia' have been published (ABPM 1999/41) and are included at Appendix 5. This document sets out the procedures by which Australian authorities would approve exporting countries for the purposes of export of commodities (including chicken meat) to Australia. These guidelines were produced in 1999, prior to the creation of Biosecurity Australia as a separate entity from AQIS. At that time, all of the approval procedures would have been carried out by AQIS. Since the creation of Biosecurity Australia, administrative arrangements have been established whereby BiosecurityAustralia would perform the systems review of the country's veterinary services as described in points a) to g) below, while AQIS would undertake the on-plant inspections as described in points h) to l).

As chicken meat has not previously been exported to Australia, all potential exporting countries, including those that routinely export animals and animal products to Australia, would be subject to a review to ensure that countries would meet Australia's requirements. This would include an on-ground assessment. For those countries that do not routinely export animals and animal products to Australia an assessment by Biosecurity Australia of their veterinary services would also be undertaken. Specific quarantine requirements would be developed, applicable to the exporting country, based on the generic quarantine requirements and the exporting country's avian health status.

The review process involves assessment of the country in relation to the following criteria:
a) An effective veterinary/fish health service
b) Animal health status of the country of origin/export
c) Quarantine measures
d) Animal health controls
e) Performance in reporting disease
f) Access to laboratories that can conduct recognised diagnostic tests to an international standard of competence; and
g) Appropriate arrangements for certification/documentation.

In addition to this "systems review" of potential exporting countries, the Guidelines contain a requirement for assessment of particular export processing facilities. As stated above, these assessments would normally be undertaken by AQIS. Assessment of such facilities involves demonstration that the plant has systems in place for:
h) Suitable separation of raw and processed product
i) Reliable compliance with minimum processing requirements for the product
j) Auditable records of information required by AQIS, for example on the source of raw materials and ingredients, processing records and test results
k) Controls to prevent post-processing contamination; and
l) Standards of hygienic construction and operation that provide equivalent public health safeguards to those provided by relevant Australian standards.

If approval is given for a country to export chicken meat to Australia, AQIS will monitor the performance of the exporting country in relation to certification and compliance with import conditions. Detection of non-compliance would trigger a follow-up audit by appropriate Australian authorities.

Where an exporting country makes an application for consideration of zoning or compartmentalisation arrangements, Biosecurity Australia would audit these arrangements prior to approval. Principles of zoning and compartmentalisation, including relevant OIE code chapters, are included at Appendix 6.

Heat treatments for management of risk associated with avian influenza and Newcastle disease viruses

After consideration of the comments received from stakeholders regarding the proposed risk management measures for notifiable avian influenza (NAI) and Newcastle disease (ND) viruses in the draft IRA report, the IRA team has reconsidered the available heat inactivation data for these two viruses. The major concerns raised by stakeholders related to research conducted on heat inactivation of NAI viruses and Newcastle disease virus (NDV) in chicken products with some stakeholders commenting that the time/temperature combinations proposed in the draft IRA report (NAI viruses 70 °C for one minute and ND virus 70 °C for 12 minutes) were too stringent and not supported by scientific data (Alexander and Manvell 2004; Thomas and Swayne 2007a; Thomas and Swayne 2007b).

In a published experimental report, Thomas and Swayne have estimated a D value[1] of approximately 0.5 seconds at 70 °C for AI virus (Thomas and Swayne 2007a). Using this D value, a heat treatment at 70 °C for 3 seconds would be sufficient to achieve a 6 log reduction in NAI virus titre. This compares with an OIE recommendation that chicken meat be heated at 70 °C for 3.5 seconds, in order to inactivate NAI. The World Health Organization (WHO) also recommends that chicken meat be cooked to a core temperature of 70 °C (World Health Organisation 2005).

Thomas, King and Swayne investigated the thermal inactivation of avian influenza and Newcastle disease viruses, and concluded that under conditons of natural infection, 70 °C for 5 seconds was sufficent to inactivate both viruses, indicating similar D values for both viruses. There was one instance where a small amount of NDV remained after heating to 73.9 °C. The authors considered that this result was due to the starting titre being much higher than was likely in natural infections, and discounted this result in reaching their conclusion (Thomas, King and Swayne 2008). In contrast, the work of Alexander and Manvell (2004) estimated a D value of 82 seconds at 70 °C for NDV. OIE does not provide recommendations for heat treatments for inactivation of NDV.

The IRA team is of the view that the large differences in D values for ND virus would not be accounted for by large differences in heat resistance of different strains of the virus, as may be suggested by these data. After consultation with an expert on the heat treatments of foods, the IRA team recognised that there are a number of differences in the methods used by Thomas and Swayne and by Alexander and Manvell that could explain the difference in D values reported. The most significant of these differences relate to:

> a) Size and nature of the test sample: Thomas and Swayne treated 0.05g homogenised samples of raw, skinless meat in thin-walled polypropylene PCR tubes that were centrifuged before heat treatment. Alexander and Manvell used a medium consisting of homogenised muscle skin and fat in normal saline. Samples (1 ml) of the homogenate were placed in glass 'bijou' bottles for treatment.

[1] ***D-value*** *refers to* ***decimal reduction time*** *- The time required at a certain temperature to kill 90% of the organisms being studied.*

b) The nature of the heat source used to apply the heat treatments: Thomas and Swayne heated their samples in a tube-holding heating block of a thermocycler with a heated lid that provided precise control of the temperature and the time, and the small sample size minimised problems associated with heat transfer through the product. To ensure even heating Alexander and Manvell treated their samples by immersion and agitation in a water bath. Care was taken to ensure that the individual bottles were immersed to a level well above the level of the added suspension. This method is unlikely to allow as precise control over the heating process as use of the thermocycler, but the IRA team considered that it represented a process that is closer to commercial practice than the procedures used by Thomas and Swayne.

c) The temperatures at which heat treatments were carried out: Thomas and Swayne treated samples for various times at temperatures ranging from 57 °C to 61 °C. They subsequently used results obtained at these temperatures to calculate D values that were then extrapolated to 70 °C. Alexander and Manvell treated samples for various times at temperatures ranging from 60 °C to 80 °C. This approach allowed the direct calculation of D values at the temperature of interest (i.e. 70 °C). Advice provided to the IRA team was that directly calculated values would be expected to be more reliable than those obtained by extrapolation, especially when extrapolating more than two Z values[2] above the level of the experimental data.

Some stakeholders stated that the approach taken by Thomas and Swayne would provide a more accurate estimate of the heat resistance of the viruses concerned. It was suggested that artefacts of the heating process, such as air pockets in the test sample, which could inhibit heat transfer, would be less likely to interfere with the outcome. The approach taken by Alexander and Manvell more closely reflected a commercial process, in which the treated product could contain skin and fat and potentially air pockets. The level of fat in meat products has been shown to affect the D values for microorganisms in heated foods (Juneja, Eblen, and Marks 2001), and this could in part explain the observed differences in heat resistance.

d) The method of calculation of the D values: When experimentally assessing the heat resistance of microorganisms, the inactivation curve is often biphasic. In an unpublished report on NDV, Thomas and Swayne stated that ‘some of the curves had a slightly biphasic shape’, but ‘a linear model provided a fair-to-good fit for all curves’. This linear model was used to determine D and Z values. Alexander and Manvell took a more conservative approach, stating ‘where curves were biphasic, the D value was calculated from the gradient of the second (shallowest) phase’.

The IRA team considered that both experimental methods were valid. The methods of Thomas and Swayne provided a useful assessment of the relative heat resistance of different microorganisms under similar conditions. On the other hand, the IRA team considered that the work of Alexander and Manvell on NDV may reflect the likely commercial situation. After considering the information contained in the quoted literature, and the OIE recommendations, in the case of NAI viruses, the IRA team has adopted the following recommendations for heat inactivation of AI and ND viruses in commercial chicken meat products.

[2] ***Z-value*** *of an organism is the temperature, in degrees Fahrenheit, that is required for the thermal destruction curve to move one log cycle.*

Taking into account the available scientific data and the difficulty in accurately determining an instant core temperature in commercial situations a process equivalent to one minute at 70 °C was considered appropriate for a 6 log reduction in HPNAI and LPNAI. Biosecurity Australia considers that further information on thermal inactivation of avian influenza viruses in chicken meat under commercial type situations would be useful. Accordingly Biosecurity Australia is commissioning the Australian Animal Health Laboratory (AAHL) to undertake a project to assess the effectiveness of this time and temperature under conditions to reflect those used commercially. The recommendation for the inactivation of NAI viruses will be reviewed when the report of this study has been received.

A process equivalent to 8.2 minutes at 70 °C was considered appropriate for a 6 log reduction of NDV in chicken products, based on the approach of Alexander and Manvell (2004) in setting a D value for NDV.

Highly pathogenic avian influenza virus

In relation to the risks associated with highly pathogenic notifiable avian influenza (HPNAI) and low pathogenicity notifiable avian influenza (LPNAI), the OIE Animal Health Code 2008 (World Organisation for Animal Health (OIE) 2007b) provides that, when importing fresh meat of poultry, veterinary administrations should require for notifiable avian influenza (NAI) free countries, zones or compartments,

‘the presentation of an international veterinary certificate attesting that the entire consignment of fresh meat comes from poultry:

‘1. which have been kept in an NAI free country, zone or compartment since they were hatched or for at least the past 21 days;

2. which have been slaughtered in an approved abattoir in an NAI free country, zone or compartment and have been subjected to ante-mortem and post-mortem inspections for NAI in accordance with Chapter 6.2 and have been found free of any signs suggestive of NAI.’

For meat products, the Code requires that the commodity is derived from fresh meat which meets the requirements outlined above, or the commodity has been processed to ensure destruction of the NAI virus, and that precautions were taken to avoid contact of the commodity with any source of NAI virus. The provisions of the Code, so far as are relevant to this IRA, are reproduced in Appendix 7.

After taking into account the available information, the IRA team considered that heating to a minimum core temperature of 70 °C for at least one minute would be sufficient to ensure the inactivation of AI virus in chicken meat under commercial conditions. Because the unrestricted risk associated with the introduction of HPNAI virus was assessed as high, processing of chicken meat to ensure destruction of the virus must be undertaken off-shore (i.e. before importation into Australia).

Therefore the IRA team considered that, in order to achieve Australia’s ALOP, the following risk management measures are required for HPNAI viruses:

a. Certification of country or zone freedom from HPNAI virus, subject to Australian Government authority satisfaction.[3]

OR

b. Processing of chicken meat to ensure destruction of AI virus, before importation of the commodity. The product must be heated to a minimum core temperature of 70 °C for at least 1 minute or equivalent time/temperature.

Low pathogenicity notifiable avian influenza virus

In relation to the risks associated with low pathogenicity notifiable avian influenza (LPNAI), the OIE Animal Health Code 2008 (World Organisation for Animal Health (OIE) 2007b) provides that, when importing fresh meat of poultry, veterinary administrations should require for notifiable avian influenza (NAI) free countries, zones or compartments,

'the presentation of an international veterinary certificate attesting that the entire consignment of fresh meat comes from poultry:

'1. which have been kept in an NAI free country, zone or compartment since they were hatched or for at least the past 21 days;

2. which have been slaughtered in an approved abattoir in an NAI free country, zone or compartment and have been subjected to ante-mortem and post-mortem inspections for NAI in accordance with Chapter 6.2 and have been found free of any signs suggestive of NAI.'

For meat products, the Code requires that the commodity is derived from fresh meat which meets the requirements outlined above, or the commodity has been processed to ensure destruction of the NAI virus, and that precautions were taken to avoid contact of the commodity with any source of NAI virus. The provisions of the Code, so far as are relevant to this IRA, are reproduced in Appendix 7.

After taking into account the available information, the IRA team considered that heating to a minimum core temperature of 70 °C for at least 1 minute would be sufficient to ensure the inactivation of AI virus in chicken meat under commercial conditions.

Therefore the IRA team considered that, in order to achieve Australia's ALOP, the following risk management measures are required for LPNAI viruses:

a. Certification of country or zone freedom from LPNAI virus, subject to Australian Government authority satisfaction[3].

OR

b. Processing of chicken meat, off-shore or on-shore under quarantine control, to ensure destruction of AI virus. The product must be heated to a minimum core temperature of 70 °C for at least one minute, or equivalent time/temperature.

[3] In order to satisfy Australian Government authorities that a country or zone is free of NAI, it is expected that the use of live avian influenza virus vaccine in poultry would not be permitted in that country or zone.

Newcastle disease virus

In relation to risks associated with Newcastle disease virus (NDV), the OIE Terrestrial Animal Health Code 2008 (World Organisation for Animal Health (OIE) 2007c) provides that, when importing fresh meat of poultry from ND-free countries, zones or compartments Veterinary Authorities should require

'the presentation of an international veterinary certificate attesting that the entire consignment of meat comes from poultry:

1. which have been kept in an ND free country, zone or compartment since they were hatched or for at least the past 21 days;

2. which have been slaughtered in an approved abattoir in an ND free country, zone or compartment and have been subjected to ante-mortem and post-mortem inspections in accordance with Chapter 6.2 and have been found free of any signs suggestive of ND.'

The provisions of the Code, so far as are relevant to this IRA, are reproduced in Appendix 7.

As discussed in the introduction to this chapter, the IRA team considered that country or zone freedom to the satisfaction of Australian Government authorities would achieve Australia's ALOP. Similarly, the IRA team considered that the risk would be negligible where the product has been processed to ensure, to the satisfaction of Australian Government authorities, the destruction of NDV. Based on the experimental data of Alexander and Manvell (2004), the IRA team considered that a heating process equivalent to 8.2 minutes at 70 °C would inactivate Newcastle disease virus:

Where the exporting country is considered free of ND but allows vaccination with live vaccines, any live vaccines used must have been produced from lentogenic strains of NDV, as recommended by the OIE.

Following risk mitigation assessment, it was determined that, in order to achieve Australia's ALOP, the following risk management measures are required for Newcastle disease virus:

a. Certification of country or zone freedom from NDV, subject to Australian Government authority satisfaction.

AND

Certification that any live vaccines used on chickens from which the meat was derived had an ICPI of <0.7.

OR

b. Processing of chicken meat, off-shore or on-shore under quarantine control, to ensure destruction of ND virus. The product must be heated to a minimum core temperature of 70 °C for at least 8.2 minutes, or equivalent time/temperature.

Very virulent infectious bursal disease virus

The OIE Code does not provide guidelines relating to the import of chicken meat from countries where very virulent infectious bursal disease virus (vvIBDV) is present. Therefore,

risk mitigation measures for this disease agent were assessed in accordance with the procedure described in the Method for Risk Assessment.

As discussed in the introduction to this chapter, the IRA team considered that country or zone freedom to the satisfaction of Australian Government authorities would achieve Australia's ALOP. Similarly, the IRA team considered that the risk would be negligible where the product has been processed to ensure, to the satisfaction of Australian Government authorities, the destruction of infectious bursal disease virus. Unpublished work conducted in 1997 at the Quality Control Unit, Central Veterinary Laboratory, Alderstone, United Kingdom, showed that a mix of bursal homogenate (23%), skin and fat (4%), muscle tissue (23%) and peptone broth (50%) contained no viable IBDV only after cooking at 80 °C for at least 120 minutes (Quality Control Unit 1997). Option 1 (country or zone freedom), Option 2 (off-shore processing) and Option 3 (on-shore processing) were considered to be appropriate risk management measures.

Other options were evaluated using the risk assessment model. An outline of risk mitigation measures considered, and their effects on estimated annual risk, is presented in Table 125 below.

Table 125. Assessment of risk management options for vvIBDV

Risk management option(s)	Step in the release/ exposure pathway	Original likelihood	Managed likelihood	Restricted risk	Achieves ALOP
OPTION 4[1]	$Expo_1$	0.5±10%	0.1±10%	Moderate	No
Importing bone-in cuts only	$Expo_3$	0.29±10%	0.3±10%		
	$Expo_5$	0.4±10%	0.3±10%		
OPTION 5[1]	$Expo_1$	0.5±10%	Zero	Moderate	No
Import boneless cuts only	$Expo_3$	0.29±10%	0.0±10%		
	$Expo_5$	0.4±10%	0.1±10%		

[1] For risk management options 4 and 5, the values of $Expo_1$, $Expo_3$, $Expo_5$ are as described in the Method for Risk Management (pages 106–107 of Part B).

Following risk mitigation assessment, it was determined that, in order to achieve Australia's ALOP, the following risk management measures are required for vvIBDV:

a. Certification of country or zone freedom from vvIBDV, subject to Australian Government authority satisfaction.

OR

b. Processing of chicken meat, off-shore or on-shore under quarantine control, to ensure destruction of very virulent IBDV. The product must be heated to a minimum core temperature of 80° C for at least 125 minutes, or equivalent time/temperature.

Requirements for thermal processing to ensure destruction of vvIBDV in chicken meat have been published previously and are included at Appendix 8.

Exotic antigenic variant strains of infectious bursal disease virus

For the purposes of this risk assessment, exotic antigenic variant strains of infectious bursal disease viruses (IBDV) have been defined as variant strains that are antigenically and genetically different from those that exist in Australia, and include United States variant strains.

The OIE Code does not provide guidelines relating to the import of chicken meat from countries where exotic antigenic variant strains of IBDV are present. Therefore, risk mitigation measures for these disease agents were assessed in accordance with the procedure described in the Method for Risk Assessment.

As discussed in the introduction to this chapter, the IRA team considered that country or zone freedom, to the satisfaction of Australian Government authorities, would achieve Australia's ALOP. Similarly, the IRA team considered that the risk would be negligible where the product has been processed to ensure, to the satisfaction of Australian Government authorities, the destruction of infectious bursal disease virus. Therefore, Option 1 (country or zone freedom), Option 2 (off-shore processing) and Option 3 (on-shore processing) were considered to be appropriate risk management measures.

Other options were evaluated using the risk assessment model. An outline of risk mitigation measures considered, and their effects on estimated annual risk, is presented in Table 126 below.

Table 126. Assessment of risk management options for exotic antigenic variant strains of IBDV

Risk management option(s)	Step in the release/ exposure pathway	Original likelihood	Managed likelihood	Restricted risk	Achieves ALOP
OPTION 4[1]	$Expo_1$	0.5±10%	0.1±10%	Low	No
Importing bone-in cuts only	$Expo_3$	0.29±10%	0.3±10%		
	$Expo_5$	0.4±10%	0.3±10%		
OPTION 5[1]	$Expo_1$	0.5±10%	Zero	Low	No
Import boneless cuts only	$Expo_3$	0.29±10%	0.0±10%		
	$Expo_5$	0.4±10%	0.1±10%		

[1] For risk management options 4 and 5, the values of $Expo_1$, $Expo_3$, $Expo_5$ are as described in the Method for Risk Management (pages 106–107 of Part B).

Following risk mitigation assessment, it was determined that, in order to achieve Australia's ALOP, the following risk management measures are required for exotic antigenic variant strains of IBDV:

a. Certification of country or zone freedom from exotic antigenic variant IBDV, subject to Australian Government authority satisfaction.

OR

b. Processing of chicken meat, off-shore or on-shore under quarantine control, to ensure destruction of the IBD virus. The product must be heated to a minimum core temperature of 80° C for at least 125 minutes, or equivalent time/temperature.

Requirements for thermal processing to ensure destruction of IBD virus in chicken meat have been published previously and are included Part D at Appendix 8.

Salmonella Pullorum and *Salmonella* Gallinarum

The OIE Code does not provide guidelines relating to the import of chicken meat from countries where pullorum disease and/or fowl typhoid are present.

As discussed in the introduction to this chapter, the IRA team considered that country or zone freedom to the satisfaction of Australian Government authorities would achieve Australia's ALOP. Similarly, the IRA team considered that the risk would be negligible where the product has been processed to ensure, to the satisfaction of Australian Government authorities, the destruction of *Salmonellae*. Extrapolating from the available literature, and for the purposes of this risk analysis, it is considered by the IRA team that heating of chicken meat to a core temperature of 70 °C for a minimum of 2.5 minutes will result in a 6 log reduction in *Salmonella* species of concern (Murphy et al. 2002; Murphy et al. 2004; Murphy et al. 2004; Schnepf and Barbeau 1989; Veeramuthu et al. 1998).

Option 1 (country or zone freedom), Option 2 (off-shore processing) and Option 3 (on-shore processing) were considered to be appropriate risk management measures.

Other options were evaluated using the risk assessment model. An outline of risk mitigation measures considered, and their effects on estimated annual risk, is presented in Table 127 below.

Table 127. Assessment of risk management options for *Salmonella* Pullorum and *Salmonella* Gallinarum

Risk management option(s)	Step in the release/ exposure pathway	Original likelihood	Managed likelihood	Restricted risk	Achieves ALOP
OPTION 4[1]	$Expo_1$	0.5±10%	0.1±10%	Low	No
Importing bone-in cuts only	$Expo_3$	0.29±10%	0.3±10%		
	$Expo_5$	0.4±10%	0.3±10%		
OPTION 5[1]	$Expo_1$	0.5±10%	Zero	Low	No
Import boneless cuts only	$Expo_3$	0.29±10%	0.0±10%		
	$Expo_5$	0.4±10%	0.1±10%		

[1] For risk management options 4 and 5, the values of $Expo_1$, $Expo_3$, $Expo_5$ are as described in the Method for Risk Management (pages 106–107 of Part B).

Following risk mitigation assessment, it was determined that, in order to achieve Australia's ALOP, the following risk management measures are required for *S.* Pullorum and *S.* Gallinarum:

a. Certification of country or zone freedom from S. Pullorum and S. Gallinarum, subject to Australian Government authority satisfaction.

OR

a. Processing of chicken meat, off-shore or on-shore under quarantine control, to ensure destruction of *Salmonella*. The product must be heated to a minimum core temperature of 70°C for at least 2.5 minutes, or equivalent time/temperature.

Salmonella Enteritidis/Typhimurium

The OIE Code does not provide guidelines relating to the import of chicken meat from countries where *S.* Enteritidis or multi-drug resistant *S.* Typhimurium is present.

As discussed in the introduction to this chapter, the IRA team considered that country or zone freedom to the satisfaction of Australian Government authorities would achieve Australia's ALOP. Similarly, the IRA team considered that the risk would be negligible where the product has been processed to ensure, to the satisfaction of Australian Government authorities, the destruction of *Salmonellae*. Extrapolating from the available literature, and for the purposes of this risk analysis, it is considered by the IRA team that heating of chicken meat to a core temperature of 70 °C for a minimum of 2.5 minutes will result in a 6 log reduction in

Salmonella species of concern (Murphy et al. 2002; Murphy et al. 2004; Murphy et al. 2004; Schnepf and Barbeau 1989; Veeramuthu et al. 1998).

Option 1 (country or zone freedom), Option 2 (off-shore processing) and Option 3 (on-shore processing) were considered to be appropriate risk management measures.

Other options were evaluated using the risk assessment model. An outline of risk mitigation measures considered, and their effects on estimated annual risk, is presented in Table 128 below.

Table 128. Assessment of risk management options for *S*. Enteritidis and multi-drug resistant *S*. Typhimurium

Risk management option(s)	Step in the release/ exposure pathway	Original likelihood	Managed likelihood	Restricted risk	Achieves ALOP
OPTION 4[1]	$Expo_1$	0.5±10%	0.1±10%	Moderate	No
Importing bone-in cuts only	$Expo_3$	0.29±10%	0.3±10%		
	$Expo_5$	0.4±10%	0.3±10%		
OPTION 5[1]	$Expo_1$	0.5±10%	Zero	Moderate	No
Import boneless cuts only	$Expo_3$	0.29±10%	0.0±10%		
	$Expo_5$	0.4±10%	0.1±10%		

[1] For risk management options 4 and 5, the values of $Expo_1$, $Expo_3$, $Expo_5$ are as described in the Method for Risk Management (pages 106–107 of Part B).

Following risk mitigation assessment, it was determined that, in order to achieve Australia's ALOP, the following risk management measures are required for *S*. Enteritidis and multi-drug resistant *S*. Typhimurium:

a. Certification of country or zone freedom from *S.* Enteritidis and multi-drug resistant *S.* Typhimurium, subject to Australian Government authority satisfaction.

OR

b. Processing of chicken meat, off-shore or on-shore under quarantine control, to ensure destruction of *Salmonella*. The product must be heated to a minimum core temperature of 70°C for at least 2.5 minutes, or equivalent time/temperature.

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Quarantine Requirements

These quarantine requirements were prepared by Biosecurity Australia based on the results of the evaluation of risk management options considered in this risk analysis report. Where an exporting country can demonstrate to the satisfaction of Australian Government authorities that alternative risk management options provide an equivalent level of quarantine protection, those alternative risk management options will be acceptable.

IMPORTERS SHOULD NOTE: Imported chicken meat must comply with the Imported Food Control Act 1992 and the Australia New Zealand Food Standards Code (FSC) in its entirety. Under the Imported Food Control Act 1992, AQIS may inspect, or inspect and conduct an analysis of imported chicken meat to determine its compliance with the FSC. These food safety requirements are separate from, and additional to, Australian quarantine requirements. Information on the Food Standards Code may be obtained from Food Standards Australia New Zealand (FSANZ).

These quarantine requirements apply to the importation of chicken meat and are issued under the authority of Quarantine Proclamation 1998.

1. Documentation

1.1 A Permit to Import chicken meat into Australia (the Permit includes an Approval Advice for the source establishment) must be obtained 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.

1.3 The application will be assessed on the above criteria as well as any other criteria deemed relevant by the Director.

2. Requirements

2.1 The chickens 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 current Australian Standard for Construction of Premises and Hygienic Production of Poultry Meat for Human Consumption and the Australian Standard for the Hygienic Production and Transportation of Meat and Meat Products for Human Consumption, or any subsequent amended standards, or any standards agreed by AQIS to be equivalent. AQIS may take into account existing approvals granted by the relevant overseas veterinary authorities.

2.2 While preparing product for Australia, establishments must conduct slaughter, preparation and storage of the meat in accordance with quality assurance principles such as the Hazard Analysis and Critical Control Point (HACCP) approach. Where establishments process product that is not suitable for export to Australia, a quality assurance program must be in place to ensure that poultry destined for export to Australia is kept separate from poultry/meat not destined for Australia, and is handled in such a way as to ensure that there is no cross-contamination. This includes ensuring that product destined for Australia is produced following a complete clean and sanitisation of the entire processing plant, and before product not destined for export to Australia.

2.3 Each consignment of chicken meat must be accompanied by official certification in accordance with these requirements.

2.4 Quarantine clearance of each consignment will remain subject to satisfactory examination of accompanying certification and documentation.

2.5 The product and consignment details must correspond with certification and Permit to Import.

3. Certification

3.1 Each consignment must be accompanied by a Government Veterinary Certificate in accordance with the Office International des Epizooties (OIE) International Terrestrial Animal Health Code 'Model international veterinary certificate for meat of domestic animals' signed by an Official Government 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 Government Veterinarian of the source country must certify in English, under

IV. Attestation of wholesomeness, that:

3.2.1 The chickens from which the meat was derived have been continuously resident in the source country since hatching and were slaughtered on ..... (dates).

3.2.2 The chickens from which the meat was derived passed ante- and post-mortem veterinary inspection under official veterinary supervision, and the meat is considered to be fit for human consumption.

3.3. All of the following risk management measures apply:

a. Highly pathogenic avian influenza virus

EITHER

(i) The chickens from which the meat was derived have been kept since hatching in a country or zone which is recognised by Australian Government authorities as free of highly pathogenic notifiable avian influenza (HPNAI).

OR

(ii) The chicken meat has been processed to ensure the destruction of the AI virus and has been heated to a minimum core temperature of 70 °C for at least one minute (or time/temperature equivalent).

b. Low pathogenicity notifiable avian influenza virus

EITHER

(i) The chickens from which the meat was derived have been kept since hatching in a country or zone which is recognised by Australian Government authorities as free of low pathogenicity notifiable avian influenza (LPNAI).

OR

(ii) The chicken meat has been processed to ensure the destruction of the AI virus and has been heated to a minimum core temperature of 70 °C for at least one minute (or time/temperature equivalent).

OR

(iii) Neither paragraph b.(i) nor b.(ii) is applicable to this consignment.

Note: In this case, the meat must be processed in Australia to ensure destruction of AI virus - see Section 4.

c. Newcastle disease virus

EITHER

(i) The chickens from which the meat was derived have been kept since hatching in a country or zone which is recognised by Australian Government authorities as free from Newcastle disease.

AND

Any live vaccines used on chickens from which the meat was derived were produced from lentogenic strains of Newcastle disease virus.

OR

(ii) The chicken meat has been processed to ensure the destruction of the Newcastle disease virus and has been heated to a minimum core temperature of 70 °C for at least 8.2 minutes (or time/temperature equivalent).

OR

(iii) Neither paragraph c.(i) nor c.(ii) is applicable to this consignment.

Note: In this case, the meat must be processed in Australia to ensure destruction of ND virus- see Section 4.

d. Very virulent infectious bursal disease virus (vvIBD)

EITHER

(i) The chickens from which the meat was derived have been kept since hatching in a country or zone which is recognised by Australian Government authorities as free from very virulent infectious bursal disease (vvIBD)

OR

(ii) The chicken meat has been processed to ensure the destruction of the vvIBD virus to the satisfaction of Australian Government authorities. The product must be heated to a minimum core temperature of 80 °C for at least 125 minutes (or time/temperature equivalent).

OR

(iii) Neither paragraph d.(i) nor d.(ii) is applicable to this consignment.

Note: In this case, the meat must be processed in Australia to ensure destruction of vvIBD virus - see Section 4.

e. Exotic antigenic variant strains of IBD virus[7]

EITHER

(i) The chickens from which the meat was derived have been kept since hatching in a country or zone which is recognised by Australian Government authorities as free from exotic antigenic variant infectious bursal disease (var IBD).

OR

(ii) The chicken meat has been processed to ensure the destruction of the var IBD virus to the satisfaction of Australian Government authorities. The product must be heated to a minimum core temperature of 80 °C for at least 125 minutes (or time/temperature equivalent).

OR

(iii) Neither paragraph e.(i) nor e.(ii) is applicable to this consignment.

Note: In this case, the meat must be processed in Australia to ensure destruction of var IBD virus - see Section 4.

f. *Salmonella* Gallinarum (fowl typhoid) & *Salmonella* Pullorum (pullorum disease)

EITHER

(i) The chickens from which the meat was derived have been kept since hatching in a country or zone which is recognised by Australian Government authorities as free from fowl typhoid and pullorum disease.

OR

[7] For the purposes of this document, exotic antigenic variant strains are defined as variant strains that are antigenically and genetically different from those that exist in Australia, and include United States variant strains.

(ii) The chicken meat has been processed to ensure the destruction of S. Pullorum and S. Gallinarum and has been held at a minimum core temperature of 70 °C for at least 2.5 minutes (or time/temperature equivalent).

OR

(iii) Neither paragraph f.(i) nor f.(ii) is applicable to this consignment.

Note: In this case, the meat must be processed in Australia to ensure destruction of Salmonella organisms - see Section 4.

g. *Salmonella* Enteritidis and multi-drug resistant *S*. Typhimurium

EITHER

(i) The chickens from which the meat was derived have been kept since hatching in a country or zone which is recognised by Australian Government authorities as free from S. Enteritidis and multi-drug resistant S. Typhimurium.

OR

(ii) The chicken meat has been processed to ensure the destruction of S. Enteritidis and multi-drug resistant S. Typhimurium and has been held at a minimum core temperature of 70 °C for at least 2.5 minutes (or time/temperature equivalent).

OR

(iii) Neither paragraph g.(i), nor g.(ii) is applicable to this consignment.

Note: In this case, the meat must be processed in Australia to ensure destruction of Salmonella organisms - see Section 4.

3.4 The establishment where the chickens 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;

3.5 Officials of the Veterinary Authority of the source country were present in plants at all times when chickens were being slaughtered for export to Australia.

3.6 A quality assurance program is in place to ensure that poultry/meat destined for export to Australia is kept separate from poultry/meat not destined for Australia, and is handled in such a way as to ensure that there is no cross-contamination.

3.7 Product destined for Australia was processed and produced following a complete clean and sanitisation of the entire processing plant, and before product not destined for export to Australia.

3.8 The meat has been prepared for export and packed on ..... (dates) in bags, wrappers or packing containers which were clean and new, and in a manner which prevented contamination.

3.9 The identification number of the slaughtering establishment and/or the establishment where the meat was prepared is readily visible on the package or wrapping containing the meat,

in such a way that the numbers cannot readily be removed without damaging the package or wrapping.

3.10 The meat was not exposed to contamination prior to export.

3.11 The meat is being transported to Australia in a clean container sealed with an Official Government seal; the container contains only meat eligible for entry into Australia.

4. Post-Entry Control and Processing Requirements

Where chicken meat is identified as requiring processing in Australia, as specified in 3.3, the following conditions apply.

4.1 The chicken meat and its derivatives must be securely transported from the port of entry to the approved storage establishment(s) thence to the processing establishment(s) and finally, with respect to inadequately processed surplus wastes and by-products, to the place(s) of disposal of quarantinable waste. The transport of imported chicken meat will require appropriate security arrangements to prevent spillage (e.g. refrigerated container) and be transported by the most appropriate route as determined by AQIS.

The meat must be processed in accordance with an approved AQIS arrangement. The approved AQIS arrangement also covers such things as disposal of packaging, wastewater 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.

Further Steps in the Import Risk Analysis Process

The administrative process adopted requires that the following steps be undertaken:

- consideration of appeals, if any
- if there are no appeals or the appeals are rejected, the recommended policy will be submitted to the Director of Animal and Plant Quarantine for a policy determination
- if an appeal is allowed the IRA Appeal Panel may advise the Chief Executive of Biosecurity Australia on how to overcome the identified deficiencies. When this process is completed the recommended policy will be submitted to the Director of Animal and Plant Quarantine for a policy determination
- Notification of the proponent/applicant, registered stakeholders, and the WTO of the policy determination.

Stakeholders will be advised of any significant variation to the process.