POTENTIAL FOR WIND-BORNE SPREAD OF FOOT-AND-MOUTH DISEASE VIRUS IN AUSTRALIA

A report prepared for the Australian Meat Research Corporation

by

M.G. Garner and R.M. Cannon

with assistance from

W.P. Roberts and J.K. Bowyer

Bureau of Resource Sciences

There are minor differences (mainly to do with page breaks) between this electronic version of the document and the original printing. All the coloured figures and some of the black and white figures were produced directly from computer programs to paper in the original printing. Scanned versions of these figures are available separately on the web site www.affa.gov.au.

Note:

This paper was prepared for the purposes of discussion, and should not be cited without the prior permission of the Executive Director of the Bureau of Resource Sciences.

To ensure early publication, this report has received only limited editorial input.

CONTENTS   Page

EXECUTIVE SUMMARY

- Foot-and-mouth disease (FMD) is one of the most contagious of animal diseases. Animals may be infected by inhalation or ingestion. Ruminants are especially sensitive to infection via the respiratory tract.

- Movement of infected animals is the most important method of spread of FMD from one property to another. However, on occasions movement of airborne virus particles by wind has been responsible for infecting properties some distance downwind. Under favourable climatic conditions wind-borne spread can be an important factor in FMD epidemics.

- For wind-borne spread to occur, virus must be able to survive long enough and in sufficiently high concentrations to infect livestock downwind. Our analysis of weather data shows that for much of Australia, conditions, particularly at night, are suitable for survival of FMD virus in aerosols. Long-distance spread of virus particles requires stable atmospheric conditions and low wind speeds. Such conditions are common in Australia. Thus, for much of Australia, for much of the year, weather conditions will not be a limiting factor for wind-borne spread.

- For infection to occur downwind, animals must be exposed to sufficient virus particles. This depends on the amount of virus produced and the volume of air breathed by exposed animals. The risk of wind-borne spread is proportional to the strength of the virus source. As infected pigs excrete 1000–3000 times as much virus as cattle or sheep they pose the greatest threat.

- The risk of spread is proportional to the density of livestock downwind, with large concentrations of animals such as saleyards and feedlots being particularly vulnerable. Cattle are more likely to be infected than are sheep or pigs because of their higher respiratory volume — sheep have one quarter, and pigs one twelfth, the risk of cattle. Hence, the typical pattern of wind-borne spread is from pigs to cattle. Once one animal has become infected, the disease will spread rapidly through the herd by close contact.

- These findings have been confirmed in simulated outbreak studies. Except in close proximity to an infected property, there is minimal risk of wind-borne spread from typical beef, dairy and sheep properties in Australia. Cattle feedlots, because of their size pose a greater risk, especially if slaughtering of infected animals is delayed. However, infected piggeries represent the greatest threat, with spread greater than 10 km likely. Even a small number of infected pigs pose a significant risk of wind-borne spread.

- The weather conditions at the time of the outbreak will determine the survival of airborne virus and how far it spreads. These cannot be predicted in advance and must be analysed at the time to determine premises at risk. Surveillance effort can then be targeted accordingly.

- Several countries have developed tactical models and decision aids to evaluate the risks of wind-borne spread during FMD outbreaks. It is recommended that a tactical FMD wind-borne spread model suitable for use in Australia be developed and made available to disease control authorities.

PREFACE

The Bureau of Resource Sciences was contracted by the Australian Meat Research Corporation to undertake a study to assess the potential for wind-borne spread of foot-and-mouth disease (FMD) in Australia.

The terms of reference for the project were to:

- *Review overseas and Australian literature and computer models on the wind-borne spread of FMD virus and related subjects*

- *Specify the geographic areas and conditions under which wind-borne spread may occur and set out results on surface maps of Australia showing areas conducive to FMD virus survival*

- *Review the distribution of livestock in at-risk areas of Australia*

- *Assess the likelihood of wind-borne spread of FMD in Australia and present as contour diagram of virus concentrations downwind from sources of various sizes*

- *Integrate the findings of the above work by making use of real livestock and weather data to define actual scenarios which model the likely concentrations of FMD virus produced after introduction and potential spread downwind*

- *Make recommendations on any changes to AUSVETPLAN in order to take account of wind-borne spread*

- *Make recommendations concerning the need to develop computer models or other tools for tactical use in an outbreak*

In accordance with instructions by the MRC, the project was undertaken in two stages.

Stage one of this project used long-term weather records to assess regions of Australia for their suitability for survival of FMD virus in aerosols based on relative humidity and temperature criteria. The relative risk of wind-borne spread across Australia was assessed using information on the distributions and densities of livestock and feral pigs.

Stage two of the project examined factors affecting the concentration of virus in plumes, the extent of spread from various sources under different conditions, probabilities that infection would result from various levels of exposure to airborne virus, and the risks under Australian conditions posed by typical livestock enterprises should they become infected.

In order to simplify presentation of the work undertaken in this project, the report contains three parts. Part A provides a concise overview of the key findings and conclusions, Part B is a literature review and Part C contains detailed descriptions of the methodologies used and the results obtained from the various analyses.

POTENTIAL FOR WIND-BORNE SPREAD OF FOOT-AND-MOUTH DISEASE VIRUS IN AUSTRALIA

PART A — KEY FINDINGS AND CONCLUSIONS

The answer, my friend, is blowin' in the wind,
The answer is blowin' in the wind.

Bob Dylan (Robert Zimmerman), 1962

1.   Introduction

Foot-and-mouth disease (FMD) is one of the most contagious of animal diseases. Animals may be infected by inhalation or ingestion. Primary infection is most likely to occur via the respiratory tract. Ruminants are especially sensitive to this route, while pigs are relatively more commonly infected via the oral route. Once introduced into a herd, disease will spread rapidly to animals in close contact.

The most common mechanism of spread of FMD is by the movement of infected animals. Indirect transmission of infection through contaminated products and fomites is also important. Under favourable climatic conditions, wind-borne spread of FMD will occur as well. This involves spread to animals remote from known foci of infection without any history of contact.

Most wind-borne spread over land is thought to be over distances less than 10 km. However, spread over distances of 60 km over land, and some 250 km over sea, are also believed to have occurred.

Wind-borne spread of virus can be an important factor in FMD epidemics. For example, extensive wind-borne spread of FMD is believed to have occurred at the start of the 1967−68 United Kingdom epidemic when more than 300 farms were affected in the first three weeks of the outbreak. A number of studies have demonstrated the role that wind-borne spread has played in disseminating FMD in previously free countries.

Anecdotal evidence suggests that wind-borne spread of FMD is not important in tropical and sub-tropical environments or in the hot dry climates of countries in Africa, Asia and the Middle East, a view supported by the findings from a number of recent overseas study tours. However, the endemic nature of FMD in many of these countries, and a lack of epidemiological studies make it difficult to draw firm conclusions. Wind-borne spread will be less likely in countries with low pig populations.

Caution is required in extrapolating from the experience of other countries and it is important that their FMD status is considered. In endemically-affected countries and countries that use prophylactic vaccination, the presence of varying levels of immunity in the population will modify the patterns of spread that are observed, as compared to fully susceptible populations because:

- the presence of antibodies will reduce the amount of virus produced by infected animals;
- the presence of immune animals will lower the *effective* density of animals exposed;
- a higher dose of virus will be required to infect animals if a degree of immunity is present.

These factors will serve to reduce the probability of wind-borne spread occurring in the first place and reduce the likelihood of it being recognised if it did occur because of a lack of further spread. Although it is true that many of the reports of wind-borne spread have come from Europe, there have also been reports from drier countries such as Malta and Israel.

As Australia is a large land mass with a range of geographic and climatic conditions, it is important to have an understanding of the likely extent and importance of wind-borne spread, should an FMD outbreak occur. This study, by using information on the survival of FMD virus, and on factors that affect its dispersion in the air, has been able to make an assessment of this potential.

The study contains four components. The first component was a review of the literature to ascertain the *importance* of wind-borne spread in the epidemiology of FMD and the conditions under which it may occur (Sections 2–4). The review also considered models of wind-borne spread (Section 5).

The second component of the study used long-term records of relative humidity and temperature to assess the suitability of different regions of Australia for survival of FMD virus in aerosols. By linking this information to livestock distributions and densities, this analysis (Sections 6 and 7) provided a national overview of the *relative potential* for wind-borne spread of FMD to occur in different parts of Australia (Section 8)

The third component of the study looked at the potential *extent* of wind-borne spread that could occur. The Gaussian plume model was used to examine the factors affecting the spread and dispersion of virus plumes. By using a standard source strength, and determining virus concentrations at a set point downwind, comparisons within and between sites can be made of the suitability of conditions for long-distance spread of FMD (Section 9 and 10).

The fourth component looked at aerosol virus production that could be expected from typical Australian livestock enterprises. A within-herd disease spread model was used to estimate virus production under various outbreak scenarios, and real weather data was used to ascertain how far virus would spread in the period until the disease was recognised (Section 11).

In the course of the study, techniques were developed that allowed assessments of areas to be made not only for the suitability of conditions for the survival of FMD virus in aerosols, but also of the extent of potential wind-borne spread that could occur under various outbreak conditions. These techniques can be applied to any site for which standard meteorological data is available. A wind-borne spread model was developed, that used with a within-herd disease spread model, enables a spatial representation of potential spread from an FMD-infected premise to be made.

1.1 Conditions under which wind-borne spread may occur

From overseas experience and studies we can identify the factors important in wind-borne spread. Wind-borne spread of FMD occurs where the virus:

- is produced in large quantities and become airborne,
- remains airborne long enough to reach a potential recipient host, and
- reaches the recipient host in sufficiently large quantities to cause infection.

2

The respiratory tract is the main source of airborne virus from infected animals. Pigs are potent excretors of airborne virus with one infected pig excreting 1,000 to 3,000 times that of a cow. Hence infected piggeries pose the greatest threat of wind-borne spread.

The most important conditions for survival of FMD virus in aerosols are relative humidity greater than 60%, followed by temperature less than 27°C. Other weather factors have only a minor effect on virus survival.

Virus particles from an infected source form a plume that is subject to both horizontal and vertical dispersion. The extent of spread of the plume depends on a number of factors, including the strength of the virus source, wind speed and direction, stability of the atmosphere and the topography of the area. The stability of the atmosphere and the terrain will affect the amount of turbulent mixing and thus the concentration of virus downwind. Topographic features will affect the path of the plume. It is well-recognised that most wind-borne spread of FMD occurs at night when the atmosphere tends to be more stable and wind speeds are low.

The probability of infection depends on exposure dose and susceptibility. While there is little difference in susceptibility, the higher respiratory volume of cattle means that cattle are more likely to be infected than are sheep or pigs — sheep have one quarter, and pigs one twelfth, the chance of becoming infected compared to cattle. Thus, the pattern of wind-borne spread that has been observed most often is from pigs to cattle downwind. The larger the concentration of animals that are exposed to the virus plume, the greater the risk. Larger cattle herds are more likely to be infected than smaller ones because of the greater probability that at least one animal will inhale an infectious dose.

The greatest risk of wind-borne spread occurs where pigs are infected, relative humidity is high, wind speed is low, the atmosphere is stable (particularly at night) and density of cattle downwind is high.

1.2   Relative risk of wind-borne spread occurring in Australia

In Sections 6 and 7 long-term meteorological records were used to assess the suitability of weather conditions (relative humidity and temperature) around Australia for the survival of airborne virus. This analysis has shown that there are large areas of Australia where FMD virus could survive in aerosols for a considerable portion of the year, and where wind-borne spread of FMD *could* occur.

For much of Australia, for at least part of the year, weather conditions would be suitable for survival of airborne FMD virus.

Although there are pronounced seasonal effects on the number of days at risk, it is clear that even when conditions during the day are unsuitable, in many locations, night conditions would favour survival of the virus. Figure 13 is a map, using a quarter degree grid, showing the number of days per year that are conducive to the survival of FMD virus in aerosols for different parts of Australia. From this map, it is apparent that survival of airborne virus is unlikely to be a limiting factor in wind-borne spread for much of Australia.

It is recommended that the possibility of wind-borne spread of FMD and the conditions under which it is likely to occur be highlighted in the AUSVETPLAN National Strategy for FMD.

The areas with the highest potential are in southern and eastern Australia, and correspond to the areas of highest livestock densities and higher-valued livestock in Australia. The study has also shown that year-to-year variations in periods conducive to survival of FMD virus can be large. This variation is less pronounced for coastal and high rainfall sites, but for inland and low rainfall sites it could be a significant feature, that may not be apparent when looking at long-term average weather records.

The results from the initial analysis identifying areas in Australia where FMD virus could survive should not be confused with areas where wind-borne spread would occur.

A comparison of weather conditions is only one component to be considered in assessing the risk of wind-borne spread in Australia. Animal density also plays an important part — animals can be viewed both as a potential source of virus (should they become infected) and as receptors of infection (should they be exposed). In Section 8, livestock and feral pigs numbers were converted into cattle equivalents and mapped on a quarter degree grid cell basis for the whole of Australia. As an example, Figure 19 shows Australia's cattle, sheep and pig numbers in terms of their potential as receptors of wind-borne virus.

By combining these animal density maps with the data on the number of days per year that are conducive to the survival of FMD virus (Figure 13), it was possible to rank areas according to the risk of wind-borne spread of FMD occurring. Three measures of relative risk were used — potential for virus production; receptiveness to airborne infection; and, by combining both of these, potential for transmission. The areas with the highest potential for wind-borne spread are south-eastern Queensland, eastern and central New South Wales, southern and western Victoria, northern Tasmania and south western Western Australia.

It must be emphasised that although analyses like this can demonstrate areas where conditions suitable for wind-borne spread of FMD could be expected to occur in Australia, whether spread occurs or not will be determined by the weather conditions at the time of an outbreak, and not by long-term averages.

Using historical weather records and livestock distribution data is useful for identifying areas at higher risk, however, this type of analysis does not allow any firm conclusions to be made about the probability of wind-borne spread actually occurring from these areas, nor about how far virus might be spread. To examine these issues, it is necessary to consider virus production from different herd types and sizes, how far virus might be carried by the wind and the degree of exposure for susceptible livestock downwind. These issues cannot be addressed on a national scale as they depend on the livestock and weather characteristics of individual sites.

1.3 Extent of wind-borne spread of FMD in Australia

The Gaussian plume model has been used by many investigators to study the spread of wind-borne contaminants. This model provides a method of calculating the concentration of virus downwind from an infected source. The dispersion of virus particles in the plume depends mainly on the atmospheric stability — the greater the stability, the less the dispersion and consequently the higher the concentration of virus. Atmospheric stability is determined by

wind speed and cloud cover, with the greatest stability being at night with low wind speeds. Section 9 discusses the plume model and shows the effect of different parameters on the concentration of virus downwind.

The initial analyses of the weather data used only temperature and relative humidity data, to assess virus survival. In Sections 10 and 11 cloud cover and wind speed information are combined with the virus survival data and, using the Gaussian plume model, the extent of wind-borne spread is assessed.

Section 10 compares the potential extent of spread at different sites around Australia and times of the year based on weather conditions, independent of livestock density. Each comparison used a standard virus source and the same density of livestock downwind. Ten kilometres downwind was used as the reference point for the comparisons because national and international guidelines for FMD recommend that initial restrictions around an outbreak should extend at least 10 km. Spread beyond this point would not be contained by control procedures, if the minimum restrictions were to be used.

The results were not unexpected and mirrored the virus survival patterns already described. The risk of long distance (10 km or beyond) spread is highest for coastal sites and for sites in southern Australia. However, even for sites in northern and inland Australia, there will be periods suitable for long-distance spread. The analysis also confirmed the importance of night time, with long-distance spread much more likely at night because of the more stable atmospheric conditions.

The findings show that it is not weather conditions that will be a limiting factor for the occurrence of wind-borne spread of FMD in much of Australia.

There was a good correlation between these findings, and the relative risk of wind-borne spread determined from the livestock density and virus survival data (Section 8), suggesting that the latter is an adequate measure for comparing sites.

1.4 Potential for wind-borne spread of FMD from Australian livestock enterprises

In Section 11, seven outbreak scenarios involving typical extensive and intensive livestock enterprises, backyard pigs and feral pigs were considered, to illustrate to what extent wind-borne spread may occur under Australian conditions. A composite figure using extracts from Figures 43–50 illustrates the potential for spread from these simulated outbreaks.

The probability of wind-borne spread from typical beef, dairy and sheep enterprises in Australia is very low. Even a typical south-eastern Victorian dairy farm poses a low risk. Cattle feedlots pose some risk and under favourable weather conditions could be a cause of concern. However, piggeries, with their enormous virus production potential, clearly pose the greatest threat for spreading FMD over large areas.

Infected pigs and piggeries pose the greatest threat of wind-borne spread of FMD in Australia.

A 100 sow piggery, even if the disease were recognised promptly, would put large areas of the surrounding countryside at risk, with a high probability of infection occurring beyond 10 km. Even a small number of infected backyard or feral pigs pose a significant threat of spreading

disease to surrounding livestock. Interestingly, the feral pigs presented only a low risk of infecting other feral pigs. This suggests that spread of FMD in feral pig populations will largely depend on close contact between groups.

1.5   Consequences of the findings for FMD preparedness

The study has shown that for much of Australia, the weather is not a constraint to wind-borne spread of FMD. The amount of virus produced on the source property is the major factor that determines whether wind-borne spread would take place or not. For typical cattle and sheep properties, there is a minimal risk of wind-borne spread. However for feedlots, there is a small risk, and for piggeries, a high risk.

Whatever the conditions, the closer to the source, the greater the risk of wind-borne spread. While this study has focussed on spread beyond 10 km, the possibility of wind-borne spread over lesser distances should certainly be considered in assigning surveillance priorities in the event of an outbreak. The risk also increases with the number of animals exposed. With very high livestock densities, even a relatively small source property could pose a significant threat. Such a situation could arise, for example, if a large cattle feedlot were in the path of the plume

The actual extent of spread will depend on the size of the source, type and density of exposed livestock, and weather conditions that prevail at the time of an outbreak. The size of the source will depend on both the type of enterprise and the period of virus emission. These factors cannot be predicted in advance. Simple models to predict potential wind-borne spread of FMD from infected premises have been developed and are used in a number of countries.

It is recommended that a tactical FMD wind-borne spread model suitable for use in Australia be developed and made available for use by the Epidemiological Section in the local disease control centre (LDCC).

For tactical use, a wind-borne spread model should take into account changes in wind direction and speed, atmospheric conditions and topography. Weather parameters would need to be measured at least hourly, with wind direction particularly important.

At its simplest, the model should operate on a microcomputer and be capable of generating printouts on overhead transparencies of contours of virus concentration (isopleths) at various scales. These can be overlaid on paper maps in the LDCC to facilitate identification of properties at risk.

In identifying surveillance priorities, from the point of view of wind-borne spread, one needs to consider:

- the species exposed — cattle are more at risk than sheep or pigs;
- distance from the source of infection ;
- the number of animals exposed — large herds are more at risk than small herds (because of the high stock concentrations, feedlots and saleyards are especially vulnerable);
- potential for further spread should that property become infected.

POTENTIAL FOR WIND-BORNE SPREAD OF FOOT-AND-MOUTH DISEASE VIRUS IN AUSTRALIA

PART B — LITERATURE REVIEW

Ill blows the wind that profits nobody

William Shakespeare (Henry VI, Part 3, Act II, Scene 5)

2.   EVIDENCE FOR WIND-BORNE SPREAD

Although FMD will readily spread between animals in close contact via the respiratory route, wind-borne spread refers to infection of animals remote from known foci, without any history of contact (Donaldson 1983). Distances may be measured in kilometres and movement of virus on the wind is implied. This is the definition that is used in this report.

Hardy and Milne (1938) first postulated that FMD virus may be spread by wind. Subsequently, Hyslop (1965a, 1965b) demonstrated the presence of FMD virus aerosols in coarse-filtered air from loose boxes containing infected cattle and showed that virus is released into the air before clinical signs appear.

There is now considerable evidence from past outbreaks that supports the view that FMD can be spread by the airborne route. Such spread can sometimes be quite extensive. For example, Fogedby et al. (1960) described the appearance of FMD in Scandinavia after it occurred in northern Germany. Hurst (1968) showed that many of the outbreaks in the east and south of England since 1937 could be due to wind-borne spread from Europe. Extensive wind-borne spread of FMD is believed to have occurred at the start of the 1967−68 United Kingdom epidemic when more than 300 farms were affected in the first three weeks of the outbreak (Donaldson 1988). Wind is thought to have been responsible for secondary outbreaks of FMD in Denmark and Sweden during 1966, and in France and the Channel Islands during 1974 (Gloster et al. 1982). Wind is also considered to have introduced FMD to the Isle of Wight from France in 1981 (Donaldson et al. 1982b) and to Denmark from East Germany in 1982 (Stougaard 1982). Analysis also suggested that an FMD outbreak in Israel in March–April 1985 could have been due to wind-borne spread from a preceeding outbreak in Jordan (Donaldson et al. 1988).

3. EXTENT AND IMPORTANCE OF WIND-BORNE SPREAD

Anecdotal evidence suggests that wind-borne spread is not a feature of FMD in tropical and sub-tropical environments. However, a lack of epidemiological studies and the endemic nature of the disease in many tropical and sub-tropical countries mean that wind-borne spread may not be recognised even if it occurs. According to Griffiths (1994), although the most important mechanism of spread in southern African countries such as Botswana and Zimbabwe is probably by respiratory aerosols between animals in close contact, FMD is not believed to be transmitted long distances in the air. Wind-borne aerosols are also not considered important in the spread of FMD between villages in middle eastern countries such as Turkey (Brightling 1994). Experience in Turkey suggests that with separation of more than 50 metres, the probability of aerosol spread between livestock is low (Brightling 1994). Wind was considered a possible mechanism of spread in Malto in 1975 (Sellers et al. 1981) and in Israel in 1985 (Donaldson et al. 1988)

Although FMD in south east Asian countries mainly occurs in the wet season (W.A. Geering, personal communication, August 1994), in countries like India, Venezuela and Zimbabwe where there is a wet and dry season, most FMD spread occurs during the dry season (Sellers et al. 1973) when conditions are least suitable for wind-borne spread. Experimental evidence also shows that although FMD strains from dry countries are more resistant to dessication than are temperate strains, they produce lower yields of airborne virus (Donaldson et al. 1970). In northern hemisphere countries, temperate FMD strains tend to produce higher yields of airborne virus and conditions can favour survival of virus in aerosols for much of the year (Sellers et al. 1973). Hence it is not surprising that most wind-borne spread has been reported from northern Europe.

The extent of wind-borne spread appears to depend on the number of animals affected, the site of the infected farm, the topography of the affected area and the wind speed. Detailed epidemiological studies of a number of outbreaks have demonstrated the potential for wind-borne spread to be a significant factor in some FMD outbreaks (eg. Hugh-Jones and Wright 1970, Sellers and Forman 1973, Sellers et al. 1975, Sellers and Gloster 1980, Daggupaty and Sellers 1990).

The initial pattern of spread in the 1967−68 United Kingdom outbreak strongly suggested wind-borne spread, and extensive investigations were conducted. Wind carriage of virus, together with deposition by rain at night, was considered to have been a major cause of secondary outbreaks, particularly in the first month, with a range of spread up to 30 km (Committee of Inquiry 1969).

In the 1967 Hampshire and 1966 Northumberland epidemics, 70−80% of the spread could be attributed to airborne transmission (Sellers and Forman 1973, Sellers and Gloster 1980). However, in the 1975 Malta epidemic, wind-borne spread was considered responsible for 37% of the outbreaks at most (Sellers et al. 1981). Of the 24 infected farms in the 1951−52 Canadian epidemic, wind-borne spread could have been responsible for infection on six farms and a possible source of infection on another six (Sellers and Daggupaty 1990).

Gloster et al. (1982) suggest that in 90% of outbreaks, wind-borne spread over land occurs over distances of up to 10 km. The remaining 10% includes spread over distances of 60 km or more. For the Hampshire epidemic, wind-borne spread up to 10 km was considered possible

(Sellers and Forman, 1973). In the Northumberland epidemic a longest distance of 20 km was considered likely (Sellers and Gloster, 1980). At a wind speed of 6 knots, this distance would be covered in 2 hours. Wind-borne spread of up to 20 km was considered possible in the Canadian 1951−52 epidemic (Daggupaty and Sellers, 1990). Hugh-Jones and Wright (1970) showed that airborne virus was the most likely cause of infection of farms up to 60 km from a known source in the 1967−68 United Kingdom outbreak.

Conditions over the sea are likely to be more favourable for survival of the virus and maintenance of higher virus concentrations in plumes than occurs over land (see below). Hence it is not surprising that the longest distances of wind-borne spread of FMD have been reported over the sea. Gloster et al. (1982) and Donaldson et al. (1982b) review outbreaks where wind-borne spread over long sea passages is believed to have occurred.

4.   PREREQUISITES FOR WIND-BORNE SPREAD TO OCCUR

Wind-borne spread of FMD can occur only if the virus (1) becomes airborne; (2) remains airborne long enough to reach a potential recipient host; and (3) reaches the recipient host in sufficiently large quantities to cause infection.

Thus wind-borne spread of FMD requires a source of infected particles (virus in aerosol form), atmospheric conditions that are suitable for virus survival, and persistence of particles in the air long enough and in sufficiently high concentrations to infect susceptible livestock downwind. Aerosol particles originate in the infected animal's respiratory tract and are emitted in its breath (Rumney 1986). Virus particles from an infected source form a plume that is subject to dispersion in both the horizontal and vertical planes. High concentrations of virus in the plume are maintained when the wind speed is low and the atmosphere is stable (Donaldson et al. 1982), but strong and variable winds will reduce virus concentrations by spreading particles over large areas (Rumney 1986). Morgan (1993) has reviewed and summarised many of the conditions required for long distance spread of FMD virus in aerosols.

Pigs are potent excretors of airborne virus with one infected pig equivalent to about 3,000 cattle (Donaldson 1987). Cattle are readily infected by airborne virus, and the pattern of spread that has been observed most often is from pigs to cattle downwind.

Thus there are a number of factors to be considered in assessing potential for wind-borne spread:

- Emission of virus
- Survival of virus
- Spread of virus
- Exposure and infection

4.1   Emission of Virus

Aerosol emissions of FMD originates mainly from exhaled breath and lymph from ruptured vesicles. The respiratory tract is the main source of airborne virus from infected animals (Donaldson 1986). For all species, the excretion of airborne virus lasts for 4−5 days (Sellers and Parker 1969, Donaldson et al. 1970, Sellers et al. 1971, Donaldson 1988). The amount of virus excreted varies with the species, numbers of animals infected, stage of the disease, and strain of virus. In sheep, the maximum amount of virus is excreted before lesions appear, but in pigs and cattle this occurs when lesions first appear (Sellers and Parker 1969, Donaldson et al. 1970).

Two periods of aerosol production from exposed animals have been identified (see Donaldson, 1983). The first period, from 30 minutes to 22 hours after exposure, probably corresponds to virus trapped on the bristles, hair, wool and in the lumen of the upper respiratory tract, with virus dislodged by mechanical movement and airflow. The second phase, two to seven days after exposure, follows replication of the virus in the upper respiratory tract.

Different strains of virus vary in their resistance to desiccation as measured by survival at various relative humidities. There appears to be an inverse relationship between the quantity of

virus excreted and its stability in aerosols. The strains that have been reported to result in the highest yields of airborne virus are $C_{noville}$ and several O strains, while lower yields have been recovered from animals infected with $C_{Lebanon}$, A_5 and A_{22} strains (Table 1).

Table 1: Strain differences in amount of airborne FMD virus emitted (from Donaldson et al. 1970). The amounts are in Infectious Dose $(ID)_{50}$ per minute.

{PRIVATE }Strain	Cattle	Sheep	Pigs
0_1	80	60	10,000
0_2	5	2	2,000
A_5	130	0.83	800
A_{22}	10	0.38	280
$C_{Noville}$	30	80	60,000
$C_{Lebanon}$	8	0.5	360

Tissue culture ID_{50} ($TCID_{50}$) is a measure of virus concentration or dose. Serial tenfold dilutions of virus with medium are made and added to test-tube cultures of a susceptible cell strain. Following incubation, the cultures are examined for evidence of cytopathic effects (CPE). The dilution of virus at which half of the cultures are infected is called the $TCID_{50.}$ One infectious unit (IU) is considered to be equivalent to 1.4 ID_{50} assuming a Poisson distribution. Hereafter, to avoid confusion all virus amounts will be reported in IUs.

The species affected markedly influences the potential for wind-borne spread. Pigs excrete considerably more airborne virus than do sheep or cattle. One pig can excrete 1000 to 3000 times as much as a cow or sheep over a 24 hour period (Donaldson 1986).

Reports by Donaldson (1983, 1987) and Garland and Donaldson (1990) suggest that pigs may excrete 280 million IU of airborne virus per animal per day while cattle and sheep excrete a maximum of 180,000 IU per day. The pattern of excretion and the quantities of airborne virus obtained from other ruminants such as deer and goats have been found to be similar to those with sheep and cattle (Donaldson 1983). For all species, excretion of airborne virus occurs for four to five days after the first development of vesicles.

When a number of animals are infected and atmospheric conditions are right, the infective particles form a virus plume. Within the plume there is a range of particle sizes. In aerosols from pigs, 65−71% of the total infectivity was found to be associated with particles greater than 6 microns in diameter, 19−24% with particles between 3 and 6 microns and 10−11% with particles less than 3 microns (Gloster et al. 1981, Donaldson 1988). There is no information on the percentage of virus exhaled that will be absorbed onto surfaces (ground, other animals, buildings, etc) in the immediate vicinity of the infected animals and therefore does not contribute to the plume.

In addition to respiratory aerosols, other possible sources of airborne FMD virus may include the splashing of contaminated milk or faecal slurry, the spray disposal of infected slurry, rain falling onto contaminated ground, and burning of infected carcases (Donaldson 1986). The quantities that these procedures could generate have not been determined. Bulk milk tankers containing contaminated milk are another potential source since, air displaced during filling,

can produce milk aerosols near the air-outlet vent. However, experimental studies using spores and tracers suggest that the quantities of virus likely to be emitted are unlikely to constitute a serious hazard (Donaldson 1986).

4.2   Survival of virus

Survival of virus released into the air is largely related to relative humidity (RH). Studies examining the loss of infectivity under different environmental conditions show that RH is the factor that has the greatest influence on survival of airborne virus (Donaldson 1988).

At a RH greater than 60%, survival of FMD virus is measured in hours or days (Blackall and Gloster 1981). Below this level, virus soon becomes inactivated (Barlow 1972, Donaldson 1972, Gloster et al. 1981, Donaldson 1983, Donaldson 1987). A critical RH range of 55−60% separates good from poor survival (Barlow 1972, Donaldson 1972), with virus rapidly inactivated at a RH below 55% (Donaldson 1988). The relationship between RH and survival of FMD virus is shown in Figure 1. Low RH is believed to have played a role in terminating the 1975 FMD outbreak in Malta (Sellers et al. 1981). Virus strains originating from regions with relatively dry climates retain infectivity longer than do those from more temperate regions (Donaldson 1983).

The fluid from which aerosols originate also influences airborne survival. In general, survival is high in aerosols generated from nasal fluid, milk, faecal slurry, and tissue culture but poor in aerosols from bovine salivary fluid (Donaldson 1988). The respiratory tract is recognised as the main source of airborne virus from infected animals (Donaldson 1986).

Strains vary in their resistance to temperature. At 37°C, virus *in suspensions* will retain infectivity for up to 10 days. At higher temperatures, inactivation is more rapid (Donaldson, 1987). The effect of temperature on survival of FMD virus *in aerosols* has not been adequately investigated. Gloster et al. (1981) suggest that the influence of temperature is secondary to that of RH. High recoveries of infectivity were obtained from microthread studies after 30−60 minute exposure at 27°C, and experience with other viruses in aerosols suggests that the effect of temperature is minor compared with RH (Donaldson and Ferris 1975). Rumney (1986) thought the effect of temperature to be minor since the virus can survive sub-freezing temperatures and exposure to temperatures of 27°C. Gainaru et al. (1986) identified conditions favourable for virus survival in aerosols as a RH greater than 60%, environmental temperatures less than 21°C and little or no wind.

Despite early supposition (eg. Henderson 1969) that strong sunlight is likely to decrease the infectivity of FMD virus, the available evidence suggests that any such effect is extremely small. Donaldson and Ferris (1975) found no effect of strong sunlight on survival of FMD virus in aerosols deposited on microthreads. These authors demonstrated a general photoresistance of FMD virus, though Rumney (1986) notes that this may depend on the nature of the aerosol, since certain substances can sensitise the virus to ultraviolet (UV) radiation.

Figure 1: Relationship of FMD virus survival to relative humidity (from Rumney 1986)
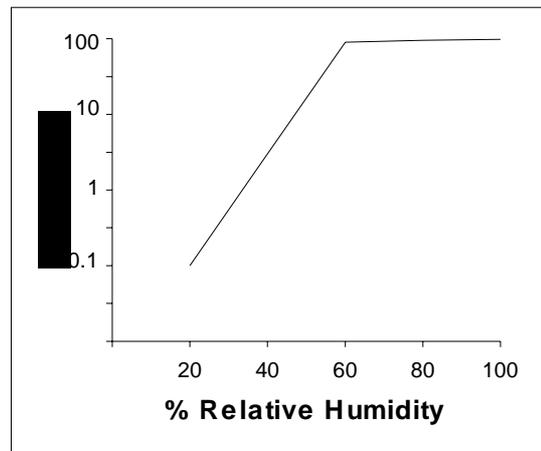
Morgan (1993) points out, RNA viruses, such as FMD virus, are inactivated by ultraviolet. light through changes in their uracil residues (Brown et al. 1963). The RNA core is destroyed but the protein coat is unaffected. Morgan (1993) considers UV light to be *'detrimental to the survival of FMDV and it could be expected that under Australian conditions, survival of FMDV in aerosols might be somewhat less than that recorded in the United Kingdom'*. Recent advice from the Australian Animal Health Laboratory (AAHL) suggests that, at the wavelengths occurring in the atmosphere, UV radiation will have a negligible effect on infectivity of FMD virus (L. Gleeson, personal communication, August 1994). In any case, the most likely time for wind-borne spread is night time, when UV levels are low or zero. UV levels are also likely to be lower at times of favourable daytime spread. Midwinter noon UV levels are much lower than midsummer levels. Cloudy conditions can reduce UV levels to about half that expected in cloudless conditions.

4.3   Spread of Virus

Spread of airborne virus will depend on weather conditions. Under suitable conditions virus emitted into the air will form a plume that may spread over large distances. Wind-borne spread of FMD is simply a special case of the dispersion of material injected into the air as a plume. Many authors have studied such dispersal in general and FMD in particular. This section of the literature review is a brief summary of findings with emphasis on those concerning FMD.

The concentration of virus in the air will depend on the dispersion of the plume and the deposition from the plume.

The plume will be dispersed both horizontally and vertically. This dispersion is mainly affected by atmospheric stability. The atmospheric stability depends not only on atmospheric conditions such as wind speed, cloud cover, and level of sunlight but also on other factors such as ground conditions (temperature, type, roughness) and topography. Wind speed also causes a dilution effect: the higher the wind speed, the lower the concentration because more air passes the source of the virus per second.

In the horizontal plane, along-wind dispersion is not important for a continuously emitting source, but cross-wind dispersion can typically give rise to a plume width of 5 km at a distance

of 10 km downwind (Rumney 1986). Plume widths generally grow at a less than linear rate with distance. Smith (1983) gives a value of $w = x^{0.875}$ where w is the plume width and x is the distance downwind.

To maintain high concentrations of virus near the ground, vertical dispersion must be limited. The amount of vertical dispersion depends on the vertical temperature structure in the lower atmosphere, wind speed, and the surface over which the air is passing (Gloster et al. 1982). Virus will be trapped near the surface by a stable layer of air in which the temperature increases with height (temperature inversion).

The rougher the surface, the greater will be the turbulence. For a given wind speed and stability, a plume is likely to be confined closer to the surface over the sea than it is over the land. Stable air is also more likely to persist over the sea than over the land because the sea surface is less affected by diurnal heating and cooling (that leads to atmospheric instability when the ground is heated by the sun). The cases where FMD is believed to have spread the greatest distances (over 100 km) have all been over the sea (Donaldson 1988).

There is commonly a pronounced diurnal variation. During the day, winds are usually stronger and atmospheric turbulence greater, leading to more rapid dilution. Thus the highest concentrations of FMD virus occur at night when the lower atmosphere is stable, inhibiting vertical dispersion, and when wind speeds are low (Donaldson 1988, Gloster et al. 1981).

Pasquill (1962) and others have studied the effects of atmospheric turbulence on dispersion of material injected into the air. An equation using a Gaussian description of plume spread has been derived for calculating the concentration of virus, at ground level, downwind from a source of given strength (eg Blackall and Gloster, 1981). For a source of height h, and sink at height z, the concentration at point x,y,z is given by:

$$C_{x,y,z} = (Q/2\pi u\sigma_y\sigma_z).\exp(-y^2/2\sigma_y^2).[\exp(-(z-h)^2/2\sigma_z^2)+\exp(-(z+h)^2/2\sigma_z^2)]$$

where:

        C = virus concentration (particles per m^3)
        Q = source strength (number of virus particles released per second)
        u = wind speed, usually measured at 10 metres
        σ_y and σ_z = are the dispersion coefficients in the crosswind and vertical directions respectively, and depend on the distance downwind

If the source and sink heights are both set to ground level the equation simplifies to:

$$C_{x,y} = (Q/\pi u\sigma_y\sigma_z).\exp(-y^2/2\sigma_y^2)$$

The concentration of virus at any point downwind depends on the strength of the source and the effects of dispersion between the source and the point in question. The source strength can be estimated from the number and type of infected animals. Dispersion is controlled by atmospheric turbulence, which can be defined in terms of atmospheric heat flux, which in turn, depends on factors such as solar elevation, cloud cover and state of the ground (Rumney 1986). This Gaussian plume dispersion equation forms the basis of models used to predict short distance (up to 10 km) spread of airborne virus (Blackall and Gloster 1981, Daggupaty and Sellers 1990).

W Grace of the Bureau of Meteorology (personal communication, July 1995) mentions that the Gaussian plume model works well for horizontal dispersion but can be improved for vertical dispersion, often by incorporating two processes, one for upwards dispersion, and one for downwards. The model can also be adjusted for topography, deposition and virus inactivation. He adds that other plume models are being developed but are not yet suitable for tactical use. He comments that the Gaussian model is known to work best and to be reasonably accurate for surface releases in stable conditions for distances of the order of 10 km. This is the typical situation when wind-borne spread of FMD could be expected.

Deposition depends on the size of particles. In still air, a particle of 6 µm would sink at 0.001 m/s but the effective settling speed is increased by atmospheric turbulence, which during daylight conditions may increase the speed of settling to 0.02 m/s (Gloster et al. 1981). However, this is still very small compared with the large scale movements of atmospheric air that spread and dilute the virus plume. Convection currents and natural or artificial ventilation may lift and keep particles in suspension. At a wind speed of 5 m/s, virus may travel 36 km in 2 hours; at 10 m/s virus could travel 100 km in 3 hours. Dilution of the plume due to deposition is considerably less than that due to turbulent dispersion in the atmosphere, and it is considered that there is little transfer of airborne virus to the ground within about 10 km of the source (Rumney 1986).

The reported role of precipitation in wind-borne spread of FMD is variable. Some early reports (Henderson 1969, Wright 1969, Hugh-Jones and Wright 1970) identified precipitation as an important factor in disease spread. However, later reports (Sellers and Forman 1973, Sellers et al. 1973, Sellers and Gloster 1980) found little correlation between precipitation and disease spread. In fact, heavy rainfall may reduce wind-borne spread by washing virus out of the air (Donaldson 1993). The amount washed out will depend on the rainfall rate, the size of the raindrops, the wind speed, duration of the rainfall, and the efficiency with which virus particles are captured (Gloster et al. 1982).

If precipitation is important, virus particles have to be captured by falling raindrops. Animals must inhale contaminated raindrops directly or inhale any particles that are emitted when raindrops hit the ground and break up (Gloster et al. 1981). They also consider that neither of these events is likely or of major importance and suggest that any apparent relationship between precipitation and disease spread may be due to the conditions that normally accompany warm frontal precipitation. These include very high RH (over 90%), the stability of the atmosphere (with attendant low vertical dispersion), and a wind speed capable of carrying airborne virus tens of kilometres.

Topography will affect the path of the virus plume (Blackall and Gloster 1981). The airstream will tend to take the path of least resistance around hills and along watercourses and valleys, rather than rising over obstacles (Donaldson 1988). The effects can be quite large in stable conditions (Gloster et al. 1981). Night-time and stable atmospheric conditions with low wind speeds are favourable for wind-borne spread of FMD virus, and it is under these conditions that the effect of topography is most marked. The nature of the terrain will also have an effect, as rough terrain will increase turbulence leading to greater dilution of virus particles (Donaldson, 1988) as discussed above.

4.4   Exposure and Infection

The amount of virus that can initiate infection in livestock via the respiratory route is considerably less than that required for infection by the oral route. Animals can become infected by inhaling infected particles directly; by disturbing particles deposited on the ground while grazing and subsequently inhaling them; or by inhaling particles released when raindrops containing virus break up and splash when hitting the ground (Gloster et al. 1981). Sheep and cattle are especially susceptible to infection by the respiratory route (Donaldson 1986). Gloster (1979) suggested that the number of particles required to cause infection by inhalation is between one and two orders of magnitude less than the number required by ingestion. Sanson (1994) quotes even greater differences between the oral and respiratory route for cattle.

Under field conditions, cattle are more easily infected by inhalation than by ingestion (McVicar 1977). Pigs require higher doses than cattle to be infected by inhalation (Gloster et al. 1981). Cattle are therefore more susceptible and most likely to be first infected from airborne virus, since on an individual animal basis they sample more air than do sheep or pigs (Sellers 1971). Under experimental conditions, when sheep and cattle were exposed to an environment containing airborne virus, the cattle did become infected before the sheep (Burrows, 1968).

In theory, a single infectious particle could establish infection in a susceptible animal. However, in practice, because of virus inactivation and clearance by the host, a larger dose is required (Sanson 1994). According to Sellers (1971), cattle, sheep and goats require a dose of $7-7000$ IU to become infected by the respiratory route. Sellers and Forman (1973) reported the infective dose for cattle to be 7 IU. Terpestra (1973) found that the minimum dose needed to infect a pig by the respiratory route was 284 IU.

Experimental studies have been undertaken to clarify the doses required to initiate infection. Donaldson et al. (1987a) found that the minimal dose for natural infection of calves was 18 IU). Gibson and Donaldson (1986) reported that a dose of 7 IU could infect sheep after 10-15 minutes exposure by inhalation. Donaldson (1988) reported that the minimal aerosol doses required to initiate infection to be 18 IU for cattle, 7 IU for sheep and 11 IU for pigs.

More recently Sanson (1994), quoting a personal communication from A. Donaldson, suggests the minimum doses to be 9, 7 and 14 IU for cattle, sheep and pigs respectively.

Daggupaty and Sellers (1990), in a modelling study, recognising that virus output could be underestimated by a factor of 5 to 100 times, used one IU as the infectious dose for cattle. To calculate the dose received, these authors multiplied the concentration downwind by the volume of air breathed by one animal during the hours that the wind was blowing (i.e. time of exposure) expressed as a daily amount.

There may be a potential problem in relating a naturally-acquired dose per day to experimentally-determined minimal infective doses, since, for the latter, exposure occurred over a period of minutes. It is not known what effect, if any, the animal's respiratory clearance mechanism will have on the same dose acquired over hours rather than minutes. It is probable that more virus would be required to establish infection over the longer period. The difference is not considered to be large, perhaps a factor of two (Donaldson et al. 1987a).

16

Typical respiratory rates for cattle, sheep and pigs are 100 L/min, 10 L/min and 5 L/min respectively (Gloster et al. 1981). For cattle, this equates to an intake of 6 m^3/hour or 144 m^3/day. Sellers (1971) provides information on daily intake of air by different species — cattle: 86–167m^3; calves: 20–72 m^3; sheep: 7–10 m^3; and pigs: 4–32 m^3.

Large herds will be more at risk than small herds because of the greater air sampling capacity (Hugh-Jones 1972). This was demonstrated in the 1967 Hampshire epidemic where the mean sizes of herds believed to be affected by wind-borne spread was higher than those not affected (Sellers and Forman 1973). In the 1966 Northumberland epidemic, where spread was ascribed to the airborne route, farms with large numbers of animals were affected but small holdings escaped infection (Sellers and Gloster 1980). Outbreaks did not occur on moorland areas where livestock density was low. Thus, it is advisable when attempting to trace possible wind-borne spread to concentrate surveillance on larger herds.

The incubation period for FMD (ie. time from exposure until the appearance of clinical disease) in animals is 2–4 days for cattle, sheep and goats, 3–6 days for pigs, but the incubation period for farms is 4–14 days (Hugh-Jones and Wright 1970, Sellers and Forman 1973). Once one or more animals in a herd have been infected, the quantity of virus in the environment will increase and transmission to other animals will occur via a variety of methods and incubation periods will be shorter.

4.5   Summary

Survival of FMD virus in aerosols will be high if :

- the temperature is less than 27°C;
- the relative humidity is greater than 60%.

The factors that favour wind-borne spread of FMD virus are:

- a stable atmosphere, particularly a temperature inversion;
- low to moderate wind speed;
- absence of heavy rain (which will tend to wash virus out of the air)

The extent of spread depends on a number of factors:

- a concentrated source of virus in aerosol form;
- a high stocking density of cattle downwind;
- species type involved;
- the topography of the area around the outbreak.

Infected piggeries pose the greatest threat of wind-borne spread. At the time of peak excretion a pig can emit about 2.8 x 10^8 IU per day compared to cattle or sheep with 1.8 x10^5 IU per day.

Cattle are more likely to be infected than are sheep or pigs because of their higher respiratory tidal volume. Larger cattle herds are more likely to be infected than smaller ones because of the greater probability that at least one animal will inhale an infectious dose.

5.   MODELS OF WIND-BORNE SPREAD OF FMD

Computer simulation models have been developed to model potential wind-borne spread from infected sources in several countries (Gloster et al. 1981, Donaldson et al. 1982b, Gloster 1983, Donaldson et al. 1987b, Donaldson et al. 1988, Daggupaty and Sellers 1990, Sanson et al. 1991a,b, Moutou and Durand 1994). These models have been used in both predictive and analytical roles. Prediction of wind-borne spread is based on weather records rather than weather forecasts, since the main potential for wind-borne spread occurs before the disease is confirmed. According to Rumney (1986) predictions of wind-borne spread can be made when it is possible to estimate:

- the quantity of virus released into the atmosphere;
- how well the virus survives and how it disperses; and
- the method and dose required to cause infection.

It is possible to make predictions of the spread of FMD over land and sea, but different techniques are used for each situation (Rumney 1986). Long distance spread over the sea is most likely to occur with high output of virus, low dispersion of virus, conditions suitable for survival of virus in aerosols and large numbers of susceptible livestock exposed to virus for many hours (Gloster 1983). For short distance spread over land, models have been developed that require estimates of daily output of virus, meteorological data, and details of the topography of the area surrounding an outbreak (Gloster 1983).

The following sections describe models of wind-borne spread of FMD virus developed in a number of countries.

5.1   United Kingdom

Mathematical models have been developed to provide an objective assessment of the area most at risk from wind-borne spread of FMD in the event of an outbreak. The models have been developed jointly by the Animal Virus Research Institute, Pirbright, and the Meteorological Office, Bracknell. They have been developed by combining data on the parameters influencing dispersion of particles in the atmosphere with data on the aerobiological properties of FMD virus. Donaldson (1988) provides a concise overview of the development and application of these models.

Two separate computer-based models are available — one for short-range and one for long-range prediction. The short range model is used to model the extent of spread over land within a 10 km radius of a known source (Gloster et al. 1981). The long-range model is used for analysing the dispersion of airborne virus across the sea over long distances (Gloster et al. 1982).

Data required to run the models:

- Estimates of the daily output of aerosol virus from infected animals. This is estimated by determining the total number of infected animals at the source of virus release.
- Hourly or three hourly observations of wind speed, wind direction, RH, cloud cover and precipitation in the area of the outbreak

- Latitude (for the short term model)

- Topographical features of the area (short term model)

- Hourly or three-hourly recordings of sea and air temperature to determine atmospheric stability at sea level (long term model).

The start of the period of emission of airborne virus on the premises is determined from the estimated age of vesicular lesions. The period of emission and the total daily output of virus are determined, and plume profiles generated. The plume profiles can be overlaid on animal distribution maps to determine the premises and herds potentially at risk.

The validity of the models has been tested by analysing past outbreaks where there has been strong circumstantial evidence for wind-borne spread. Good agreement between the predicted and actual spread has been found (Gloster et al. 1981). The models were used operationally in March 1981 when a risk of spread from Brittany, France, to the United Kingdom was successfully forecast. The forecasts were for a high risk of spread for the Channel Islands, but low for southern England (Donaldson et al. 1982b). Outbreaks subsequently occurred on Jersey and the Isle of Wight. The short-range model was used to assess the risk of further spread on Jersey and the Isle of Wight, the results indicating the risk in both places was very low. In fact no further spread took place.

Recent experimental work (Donaldson et al. 1987) has focused on clarifying minimal doses required to infect animals.

The short-range model has also been used to analyse an outbreak of FMD in Israel in March-April 1985 (Donaldson et al. 1988a). The results of the analysis suggested that the origin could have been airborne transmission of type 0_1 strain from a preceding outbreak in Jordan.

5.2   Canada

Daggupaty and Sellers (1990) have described a short-range Gaussian plume dispersion model used to analyse potential wind-borne spread in the 1951−52 Saskatchewan outbreak. The model was developed from a computer program designed to assist meteorologists in regional weather centres to respond to accidental release of toxic chemicals into the atmosphere (Daggupaty 1988).

The model requires data on temperature, relative humidity, wind direction and speed, and cloud amount and ceiling. In their study, Daggupaty and Sellers (1990) assumed that infected pigs were excreting aerosol virus at the rate of 3.23 x 10^3 IU/s, and cattle and sheep at 1.98 IU/s. One IU was taken as the infectious dose for cattle.

5.3   New Zealand

Studies in New Zealand have indicated that meteorological conditions favourable to wind-borne spread of FMD virus do occur, and a short distance wind-borne spread prediction model has been developed (Sanson 1994).

Sanson et al. (1991a,b) describe the virus plume simulation model as part of a computerised disease recording and information system (EpiMAN) for use in an exotic disease emergency. The system incorporates a database management system, a geographic information system (GIS), a simulation model of FMD, and expert system elements. The system can overlay simulated plumes of FMD virus on farm distribution maps. The following description has been supplied by Professor Roger Morris and is taken from work undertaken at Massey University on behalf of the NZ Ministry of Agriculture and Fisheries.

The New Zealand approach is based on an on-farm virus production model that calculates the amount of virus released into the atmosphere, during the period from initial infection to diagnosis. The model is a stochastic simulation model that uses Monte Carlo techniques. The model simulates the progression of infection on the farm from the known or estimated date of infection based on aging of the oldest lesions. Emission of virus by infected animals is based on reports in the scientific literature (Sellers and Parker 1969, Donaldson et al. 1970, Donaldson et al. 1982a) and amounts of virus released are estimated on a daily basis.

The model thus recreates the epidemic curve for the farm under consideration and calculates daily output of virus. This daily quantity is divided into hourly periods, according to the frequency of weather recordings. It is assumed that virus emission is a point source from the affected farm. A meteorological model based on the Gaussian plume dispersion equation is then run. This uses weather parameters recorded on farm — wind direction, wind speed, relative humidity and cloud cover — to estimate lateral and vertical dispersion, and calculates concentrations of virus at various points downwind. Virus concentrations at the centre of 50 m x 50 m cells in a 20 km x 20 km grid are calculated and the concentration of virus for each cell is summed for each 24 hour period. It is assumed that if there is insufficient virus to infect an animal over a 24 hour period, then it will be inactivated. A separate grid is created for each day.

The grid concentrations are read into the GIS and converted to categories based on ranges of concentrations of virus important for the various farm species (taken from reported minimum doses required to initiate infection by the respiratory route). A risk rating for each exposed farm is defined based on the numbers of each species present, the concentration of virus that the farm is exposed to, and the proportion of the total farm area that is covered by the plume. For multi-species farms, the risk rating is calculated for each species separately, with the highest risk rating being assigned to the farm. If required, a plume map showing the likely affected farms can be generated.

The model does not take into account underlying topography. Where the terrain is judged to be a strong influence on plume behaviour, manual interpretation by a meteorologist would be undertaken.

5.4   Europe

Moutou and Durand (1994) describe an airborne transmission model developed in France . The model is based on a model, developed by the Atomic Energy Agency (Commissariat a l'Energie Atomique: CEA), used for predicting chemical or nuclear industrial pollution (Doury 1982). The model represents virus emission as a sequence of instantaneous emissions of individual puffs. The sum of the puffs forms the infectious cloud. Virus concentration around the centre of each puff is assumed to follow a three-dimensional Gaussian dispersion. When far enough from the source, and when the wind speed is greater than 1 m/s, the puff model is simplified to the standard Gaussian plume model as described earlier.

The model is considered suitable for use on land over distances of up to 10 km. It has been tested on past outbreaks of FMD in Brittany in 1981, the last episode of FMD in France, where it confirmed that airborne transmission could have been responsible for 10 out of 13 secondary outbreaks. The model has also been used in real time during the recent outbreak of FMD in Italy, in April 1993 (Montou and Durand 1994, Maragon et al. 1994). It was used to assess the risk of further spread following four outbreaks in beef fattening units in the Po valley in northern Italy. Although the risk was found to be confined to small areas around each farm, the proximity of two large pig fattening units to the last outbreak, together with the predicted large potential for spread from these piggeries if they became infected, led to the decision to pre-emptively slaughter-out the piggeries despite there being no evidence of disease in them.

5.5   Australia

Discussions with the Bureau of Meteorology have indicated that directives are in place detailing the Bureau's role in providing support in animal health emergencies, particularly FMD, for all States and the Northern Territory (G. Bedson, Bureau of Meteorology, personal communication, July 1994). Included in the directives are instructions for preparing estimates of dispersion of FMD virus using a program RADSPIN. The following description is taken from the Western Australian directive (Anon, 1990).

The RADSPIN program is used to estimate the accumulated dosages of FMD virus at 1 km intervals, to 10 km, along each of 36 equally spaced radials. It is based on material contained in the publication *Workbook of Atmospheric Dispersion Estimates* (Turner 1967). The program can be used both retrospectively and predictively to estimate radial dosages.

Inputs required are mean values of temperature, dewpoint, wind direction and speed, emission source rate, and atmospheric stability. The program relies on a Gaussian plume model of dispersion and assumes a ground level emission, constant wind speed/direction in the dispersion layer, no deposition, and flat terrain. No dispersion is assumed to occur for RH of 60% or less.

The source emission strength is given in relative units and it is assumed that for FMD virus this will increase as a squared function of the duration of the outbreak, in days. Advice would be sought from veterinary authorities on when emission of virus commenced. The default source emission assumes that initially one unit of FMD virus is emitted per unit time, increasing to 4 units after 24 hours, 9 units after 48 hours, and so on. The program calculates the dose of FMD virus that occurs at each point on a radial grid. When RH is 60% or less no

virus is dispersed from the source, but each receptor point is assumed to maintain the previously accumulated dose.

In its present form the program does not store the meteorological data and it is necessary to re-enter these data to determine dispersion from a second source.

Recently, a Gaussian dispersion model (BolSol) developed by the Bureau of Meteorology for monitoring emissions from a sewerage treatment works has been described (Grace and Schahinger 1994). This is a set of software programs that, for specified meteorological conditions provides contours of odour, superimposed on a general purpose map. The authors suggest other potential applications of the model include predicting concentrations of airborne virus downwind of infected animals, odour levels from cattle feedlots and dispersion of fumes from large scale chemical accidents or fires involving toxic material, and in education and training.

POTENTIAL FOR WIND-BORNE SPREAD OF FOOT-AND-MOUTH DISEASE VIRUS IN AUSTRALIA

PART C — METHODOLOGY AND RESULTS

Who has seen the wind?
Neither you nor I:
But when the trees bow down their heads,
The wind is passing by.

Christina Rossetti (1830–94)

6. ASSESSING THE POTENTIAL FOR SURVIVAL OF AIRBORNE FMD VIRUS UNDER AUSTRALIAN CONDITIONS

If FMD virus emitted in aerosols is inactivated by external conditions then wind-borne spread can be discounted. The first step in assessing the likelihood of wind-borne spread in Australia is to see if the virus, while airborne, would survive the local conditions. Other requirements for wind-borne spread are an adequate number of susceptible livestock downwind (considered in Section 8) and suitable atmospheric conditions for spread to take place (Section 10).

It is worthwhile summarising the findings of Section 4.2. The key factor that has been identified for survival of FMD in aerosols is relative humidity. FMD virus survives well at a relative humidity above 60% but is rapidly inactivated by a relative humidity of less than 55%. Temperature criteria are less clear. Gainaru et al. (1986) suggest environmental temperatures less than 21°C are favourable for virus survival, but Donaldson (1987) states that virus *in suspensions* at 37°C will retain infectivity for up to 10 days although inactivation is more rapid at higher temperatures. Donaldson (1983) found that virus will survive for at least an hour at 27°C, based on studies of aerosol particles exposed on microthreads.

In this study, the criteria used to define suitability for FMD virus survival in aerosols were environmental relative humidity > 60% and temperature < 27°C.

Bureau of Meteorology records were used to identify regions of Australia that meet these criteria. Detailed daily meteorological records from selected weather stations were reviewed. In some cases, these records include three-hourly observations. These stations were chosen to provide long runs of data and to provide a representative cross section of the areas of interest.

To produce surface maps of survival patterns of virus, the detailed daily data were supplemented by average monthly temperature and RH (9.00 am and 3.00 pm) available from the Bureau of Meteorology.

6.1 Weather Data Used

Detailed records were obtained from the Bureau of Meteorology's *Three Hourly Surface Data* collection of weather observations. Logistic considerations made it necessary to select a series of sites from those available. It was essential that the sites chosen were widely spread over Australia. The length of data collection was also important, and was used to choose between nearby sites — some sites have over 40 years of data, but others have less than 10 years. In all,

113 sites were chosen. Figure 2 shows the location of these sites and Table 3 lists site details and weather data that will be used in the next section.

Table 2: Number of sites with weather readings at different times of the day

Time	0 am	3 am	6 am	9 am	Noon	3 pm	6 pm	9 pm
Number of sites	35	62	76	113	66	113	58	62

All the sites took readings at 9 am and 3 pm. In addition, 79 sites had readings at other times of the day, as shown in Table 2. Adjustments were made for daylight saving time where appropriate.

Like any weather records, there are occasional gaps in the data, ranging from a single reading, a few days or even a few months. Only records that had both a temperature and RH readings were included. Depending on the analysis being done, some records might be excluded to ensure that like was being compared with like.

For preparing surface maps, the detailed daily data were supplemented by average monthly data from the Weather Bureau Climate Data CD-ROM. These data provided a wider base for extrapolating the results over all of Australia. Information on long-term average monthly RH (9.00 am and 3.00 pm recordings) and average monthly maximum and minimum temperatures is available for some 721 weather stations across Australia (see Figure 3).
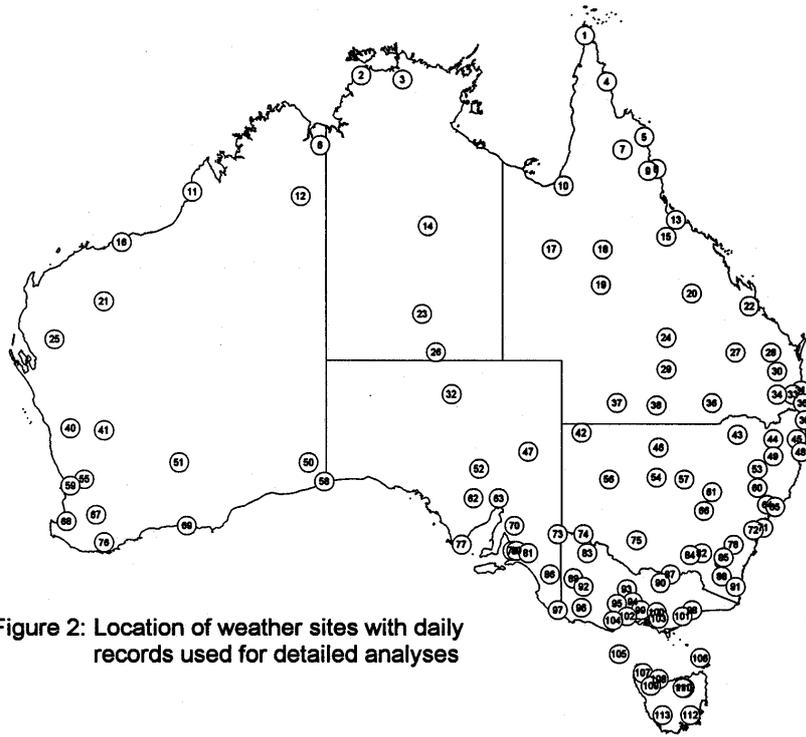
Figure 2: Location of weather sites with daily
records used for detailed analyses
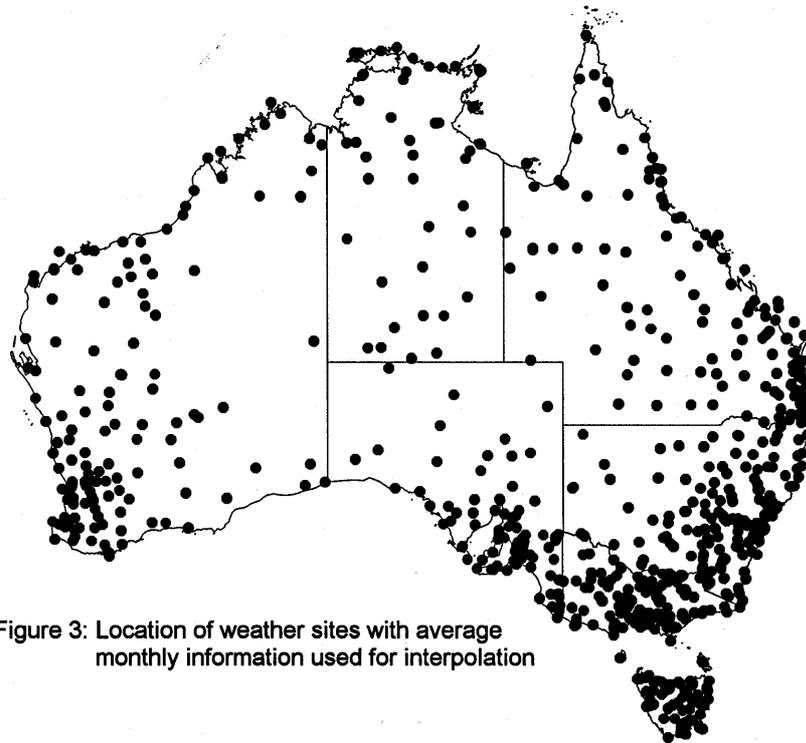
Figure 3: Location of weather sites with average
monthly information used for interpolation

Table 3 **List of weather sites used for the detailed analyses**

Fig 2 No.	BOM Site ID	Location	Lat	Long	Years of data	Readings per year suitable for aerosol FMD survival							
						0:00	3:00	6:00	9:00	12:00	15:00	18:00	21:00
1	027022	Thursday Island	10.6	142.2	28	199	319	330	197	72	72	195	273
2	014161	Darwin	12.5	130.8	34	185	228	254	141	19	18	38	127
3	014198	Jabiru	12.7	132.9	35				90		13		
4	028008	Lockhart River	12.8	143.3	29	303	347	354	213	99	98	217	283
5	031016	Cooktown	15.4	145.2	30				201		132		
6	002038	Kununurra	15.8	128.7	7				17		11		
7	028004	Palmerville	16.0	144.1	29				211		14		
8	031011	Cairns	16.9	145.8	31	341	352	354	238	86	81	206	312
9	031066	Mareeba	17.0	145.4	24				278		84		
10	029041	Normanton	17.7	141.1	17		163	198	59	13	10	14	75
11	003003	Broome	18.0	122.2	10	196	207	210	50	14	16	98	176
12	002012	Halls Creek	18.2	127.7	42	73	100	116	25	8	10	21	
13	032040	Townsville	19.3	146.8	25	311	332	336	175	51	60	184	282
14	015135	Tennant Creek	19.6	134.2	30	41	72	90	44	13	9	14	31
15	034002	Charters Towers	20.1	146.3	32		328	331	211		29		
16	004032	Port Hedland	20.4	118.6	26	225	240	240	34	10	11	67	190
17	029009	Cloncurry	20.7	140.5	34	103	88	121	54	14	10	15	37
18	030045	Richmond	20.7	143.1	13		134	185	67	10	11	21	48
19	037051	Winton	22.4	143.0	51		133	166	66	20	11		61
20	035019	Clermont	22.8	147.6	9		305	326	190		26		
21	007178	Paraburdoo	23.2	117.7	29				38		10		
22	039083	Rockhampton	23.4	150.5	45	345	350	351	243	58	44	138	312
23	015590	Alice Springs	23.8	133.9	54	107	121	150	82	22	15	21	52
24	035069	Tambo	24.9	146.3	45				125		25		
25	006022	Gascoyne Junction	25.0	115.2	30				120		19		
26	015526	Finke	25.6	134.6	9				55		12		
27	035070	Taroom	25.6	149.8	34			338	212		32		
28	039039	Gayndah	25.6	151.6	54		347	352	233	46	39		251
29	044021	Charleville	26.4	146.3	50	152	195	231	116	30	24	41	104
30	040112	Kingaroy	26.5	151.9	32			354	296	137	78		317
31	040223	Brisbane	27.4	153.1	21	328	336	341	229	77	90	239	315
32	017043	Oodnadatta	27.6	135.4	14	52	93	125	84	20	11	12	30
33	040004	Amberley	27.6	152.7	36	340	347	351	258	64	55	167	311
34	041103	Toowoomba	27.6	151.9	28		339	344	290		117		303
35	040197	Mount Tamborine	28.0	153.2	17				285		222		
36	043034	St George	28.0	148.6	24		234	280	148		32		143
37	045017	Thargomindah	28.0	143.8	36			178	94	32	19	38	89
38	044026	Cunnamulla	28.1	145.8	24		172	207	108	42	24	46	105
39	058037	Lismore	28.8	153.3	55				298		95		
40	008093	Morawa	29.2	116.0	52				183		48		
41	007139	Paynes Find	29.3	117.7	16	169	217	239	131		30	80	
42	046037	Tibooburra	29.4	142.0	21			167	114		19	32	87
43	053048	Moree	29.5	149.9	15		278	319	198	54	38	89	178
44	056011	Glen Innes	29.7	151.7	31		353	353	297	129	96	130	
45	058130	Grafton	29.7	152.9	21				302		86		
46	048013	Bourke	30.1	145.9	16		192	224	141	51	27	41	133
47	017099	Arkaroola	30.3	139.3	23				133		28		
48	059040	Coffs Harbour	30.3	153.1	35	354	338	338	248	179	207	298	330
49	056002	Armidale	30.5	151.7	25			347	263	78	82	142	293
50	011004	Forrest	30.8	128.1	51	285	302	304	139	40	32	94	235
51	012038	Kalgoorlie	30.8	121.5	35	170	226	272	163	54	33	64	139
52	016001	Woomera	31.1	136.8	51	122	205	246	153	38	20	36	84
53	055054	Tamworth	31.1	150.9	32		326	334	213	66	50	94	187
54	048027	Cobar	31.5	145.8	34		189	225	153	54	36	64	116
55	010111	Northam	31.6	116.7	53				219		68		
56	046043	Wilcannia	31.6	143.4	32				159	55	32	59	

Fig 2 No.	BOM Site ID	Location	Lat	Long	Years of data	Readings per year suitable for aerosol FMD survival							
						0:00	3:00	6:00	9:00	12:00	15:00	18:00	21:00
57	051039	Nyngan	31.6	147.2	35			303	181		37		167
58	011003	Eucla	31.7	128.9	34			319	203	216	212	302	
59	009021	Belmont	31.9	116.0	17	293	296	309	220	101	84	177	277
60	061069	Scone	32.0	150.9	33		348	344	267		66		
61	065012	Dubbo	32.2	148.6	32		328		244		54		225
62	016032	Nonning	32.5	136.5	33			302	209		36		
63	019066	Port Augusta	32.5	137.8	37		266	280	201	76	47	114	216
64	061242	Cessnock	32.8	151.4	17				272		90		
65	061055	Newcastle	32.9	151.8	48		328	338	309	244	240	280	305
66	065026	Parkes	33.1	148.2	15		303	321	208	115	69		225
67	010647	Wagin	33.3	117.3	25			336	261	42	89		
68	009534	Donnybrook	33.6	115.8	37				227		83		
69	009789	Esperance	33.8	121.9	48	328	333	333	242	145	167	294	325
70	021014	Clare	33.8	138.6	52				234		96		
71	066037	Sydney	33.9	151.2	39	315	328	332	260	153	153	239	291
72	068192	Camden	34.0	150.7	20		326	334	284	144	84	178	276
73	024016	Renmark	34.2	140.8	45		280	314	207	73	44	105	171
74	076031	Mildura	34.2	142.1	12	179	251	293	228	89	48	84	153
75	075031	Hay	34.5	144.8	28			311	216		64		181
76	009581	Mt Barker	34.6	117.7	53				292		177		
77	018070	Port Lincoln	34.7	135.9	34		342	342	297	216	177	226	328
78	070263	Goulburn	34.7	149.7	53				313		138		
79	023034	Adelaide	35.0	138.5	36	278	291	299	212	136	119	178	251
80	023785	Stirling	35.0	138.7	44				273		188		
81	024521	Murray Bridge	35.1	139.3	42			340	262	97	72	236	
82	073128	Gundagai	35.1	148.1	32				294		112		
83	076047	Ouyen	35.1	142.3	37				226		57		
84	072150	Wagga	35.2	147.5	55	286	311	328	244	137	94	162	231
85	070014	Canberra	35.3	149.2	16	328	341	346	280	123	84	174	288
86	025507	Keith	36.1	140.4	32		324	328	239	44	97		252
87	082039	Rutherglen	36.1	146.5	17				244		101		
88	070094	Cooma	36.2	149.1	28				258	126	84		
89	078031	Nhill	36.3	141.6	30	304	319	331	258	131	87	148	220
90	082002	Benalla	36.5	146.0	34			343	253	114	91	154	242
91	069002	Bega	36.7	149.8	23				297		137		
92	079023	Horsham	36.7	142.1	23		337	345	258		94		
93	081003	Bendigo	36.8	144.3	29		330	339	265	138	96	148	233
94	087036	Macedon	37.4	144.6	10				310		186		
95	089002	Ballarat	37.5	143.8	19		346	347	310	234	177	233	321
96	090103	Hamilton	37.7	142.0	14				318		282		
97	026021	Mount Gambier	37.8	140.8	22	342	342	342	295	192	174	258	333
98	084080	Bairnsdale	37.8	147.6	31				276		135		
99	086071	Melbourne	37.8	145.0	13	309	327	332	276	137	116	184	267
100	086094	Powelltown	37.9	145.8	39				326		193		
101	085072	Sale	38.1	147.1	25	346	352	352	319	187	159	264	337
102	087117	Geelong	38.1	144.3	51				322		172		
103	085093	Warragul	38.2	145.9	52				313		152		
104	090147	Colac	38.3	143.6	13				340		160		
105	098001	King Island	39.9	143.9	36		354	355	333	314	293	333	352
106	099005	Flinders Island	40.1	148.0	23	340	340	349	321	273	270	301	345
107	091092	Smithton	40.8	145.1	30			358	340	269	230		
108	091009	Burnie	41.1	145.9	36			292	305	214	229		
109	097014	Waratah	41.4	145.5	55				346		279		
110	091104	Launceston	41.5	147.2	29	346	354	355	305	180	151	227	315
111	091123	Launceston	41.5	147.1	26				305		178		
112	094008	Hobart	42.8	147.5	15	314	324	329	266	152	142	227	292
113	097053	Strathgordon	42.8	146.1	14			347	346	155	241		

7.   SURVIVAL OF FMD VIRUS IN AEROSOLS IN AUSTRALIA

This section looks at weather data around Australia to see the proportion of time that temperature and relative humidity conditions are suitable for the survival of FMD in aerosol. For this study, these criteria are:

- relative humidity greater than 60%
- temperature less than 27°C.

Section 8 will look at livestock density and Sections 9 – 11 will look at weather records in terms of how virus might spread by wind.

7.1   Analysis of the Daily Records

Figure 4 shows the number of days per year that each site met the criteria for survival of FMD virus for each particular three-hourly reading time. Missing points for particular times indicate that data were not available for this time for the site. Figure 2 can be used to relate the sites to Table 3 where the site names and actual values are given.

The maps show that, over almost the entire eastern and southern Australian coast, most of Victoria, and eastern New South Wales, more than 270 days per year meet the criteria for virus survival for the 3 am and 6 am reading. Although the data are less complete, similar conclusions apply to the midnight reading. This indicates that, on average, these areas have at least six hours of conditions conducive to survival of FMD virus in aerosols for at least 270 days of the year.

As would be expected, weather conditions during the day are less conducive to survival of FMD virus than at night. Nevertheless, there are still substantial periods that meet the conditions for survival of FMD virus. For example, even at noon the eastern and southern coastal fringe of New South Wales and Victoria average more than 150 days a year that are conducive to FMD virus survival.

The importance of night compared to day is clearly shown in Figure 5. The number of nights (based on the 9.00 am readings) ranked as suitable for survival of FMD virus clearly outweighs the number of days (based on 3.00 pm readings). Also apparent in this figure is a general trend for the number of suitable periods to increase with increasing latitude. However, a number of sites deviate from this trend, particularly sites on the coast or sites at higher elevations. Sites with a higher average at 9 pm also tended to have a higher average at 3 pm.

The sites with the lower average number of periods conducive to survival of FMD virus are found in inland Northern Territory and South Australia but even these sites have significant periods that meet the criteria, particularly between 9 pm and 6 am. For example, Alice Springs averages over 90 days a year that meet the criteria from midnight to 9 am. Kununurra was the lowest risk site with an average of only 16.5 days a year meeting the criteria at 9 am, and an average of 10 days a year meeting the criteria at 3 pm.

[Figure 5]

7.2   Sensitivity to the End-points of Viability

The criteria used for assessing sites as being suitable for survival of FMD virus in aerosols were based on data reported in the scientific literature. However, there are no definitive studies that provide clear cut-off points for survival of virus, particularly in relation to temperature. To test the sensitivity of the findings to the relative humidity and temperature criteria, the analysis of Figure 4 was repeated with both less stringent and more stringent criteria, as outlined in Table 4.

Table 4:   Sensitivity analysis on criteria used for classifying survival of FMD virus

Criteria	RH (%)	Temperature (^{o}C)
Less stringent	> 55	< 30
Standard	> 60	< 27
More stringent	> 65	< 24

Figure 6 shows the proportion of days in the year that met these criteria for the 3 am, 9 am, 3 pm and 9 pm readings for all sites. Within the graph, the sites have been sorted by the standard criteria (plotted as a line) to show the trends more clearly.

As expected, the less stringent conditions gave a higher number of days and the more stringent gave a lower number of days suitable for survival of FMD virus. Using the new criteria, the change for most sites was about 5 to 10%. That is, choosing the less stringent criteria increased the proportion of suitable days at each site by 5 to 10%, and a change to the more stringent condition decreased the proportion of suitable days at each site by 5 to 10%. Depending on the time some sites showed a much greater change in the proportion of days conducive to survival of virus.

Figures 7 and 8 show the spatial pattern of suitability under the new criteria and should be compared to Figure 4 (standard conditions). These maps show that the changes in the criteria do not result in any large change in the pattern or distribution of number of days conducive to virus survival. For example, if the more stringent criteria are used, most of the eastern and southern coast still has over 270 days meeting the criteria for the midnight, 3 am, and 6 am readings although some of the north eastern sites drop down into the 210 days a year category.

This sensitivity analysis indicates that changes of about 8% in the temperature and humidity criteria do not result in very large changes in the proportion of days meeting the criteria. This shows that there are not a large number of sites just meeting or just missing the criteria and suggests that choice of the exact criteria will not alter the conclusions.

7.3　Seasonal Differences

The 9 am and 3 pm readings have been used to show the differences between the four seasons. The average number of 12 hour periods per year was determined from the readings that satisfied the suitability criteria (RH > 60% , temperature < 27°C) for each site for each month. The analysis was based on all days for which both the morning and afternoon readings were available. The findings are shown in Figure 9.

Predictably, winter had the most periods conducive to survival of FMD virus, and summer the least. Autumn and spring have about the same number of periods conducive for most sites. The pattern is similar for all seasons, showing that a site that has a higher number of days in winter compared to other sites is also likely to have a greater number of days in the other seasons as well. Once again, the trend for the number of periods to increase with increasing latitude is evident. There is no season for any site that does not have at least a few 9 am or 3 pm readings that are conducive to survival of FMD virus.

7.4　Differences Between Years.

The analyses reported to date have been based on long-term averages. To illustrate the year-to-year variation that may occur, the number of day and night periods (based on 3.00 pm and 9.00 am readings) per month suitable for survival of FMD virus are shown for 12 sites over a 16 year period.
The sites were chosen to give a wide geographic distribution.

The mini-bar charts in Figure 10 show the proportion of suitable 3 pm readings (top) and 9 am readings (bottom) by month over 16-year periods. Gaps in the charts indicate gaps in the availability of weather data. Effectively the amount of 'black' on the chart indicates the number of readings that meet the criteria. The charts can be used to assess both within-year and between-year variation at individual sites. The data clearly show the contrast between lower risk sites (e.g. Normanton) and higher risk sites (e.g. Launceston).

Not surprisingly, most sites show some degree of year-to-year variation. However, even in the lower risk sites such as Alice Springs and Normanton there are a number of periods in even the lowest risk years that meet the criteria for survival of FMD virus. At sites like Bega and Launceston it can be seen clearly that conditions are suitable for survival of FMD virus in aerosols for a large proportion of the year.

The more detailed charts in Figure 11 use 3-hourly readings over a 12 month period to demonstrate the variability of conditions suitable for survival of FMD virus in aerosols at selected sites. These charts show not only the number and distribution of days and nights suitable for survival of FMD virus throughout the year, but also the length of the suitable periods, in three-hourly increments. Thus, for a site like Canberra there are a large number of days each year where conditions are suitable 6 to 9 hours and, during winter, 12 hours at a time. For a site like Launceston the length of continuous periods suitable for FMD virus survival in aerosols can be measured in days or weeks. Charts for different years are shown for Alice Springs, Cobar and Mildura to emphasise the differences that can occur between years.

[Figure 9]

32

[Figure 10 page 1]

[Figure 10 page 2]

[Figure 11 page 1]

[Figure 11 page 2]

7.5   Extrapolation to other sites across Australia.

The detailed information from the 113 sites was not in itself sufficient to estimate the proportion of days suitable to survival of FMD virus at any particular point in Australia. Obtaining daily data readings for other sites was both expensive (daily weather records from the 113 sites alone amounted to more than 500 MB of computer data) and unnecessary given the level of precision needed for this broad summary of risk. Use was made of the Weather Bureau Climate Data CD-ROM, which provides long-term average monthly RH (9.00 am and 3.00 pm recordings), and average monthly maximum and minimum temperatures for some 721 weather stations across Australia. The detailed information from the 113 sites was used to find an approximate relationship between:

- average monthly RH and the number days with a RH less than 60%;
- average monthly temperature and the number of days less than 27°C; and
- temperature and RH.

Figure 12:   Relationship between average monthly RH and temperature and number of days per month satisfying criteria for survival of FMD virus.
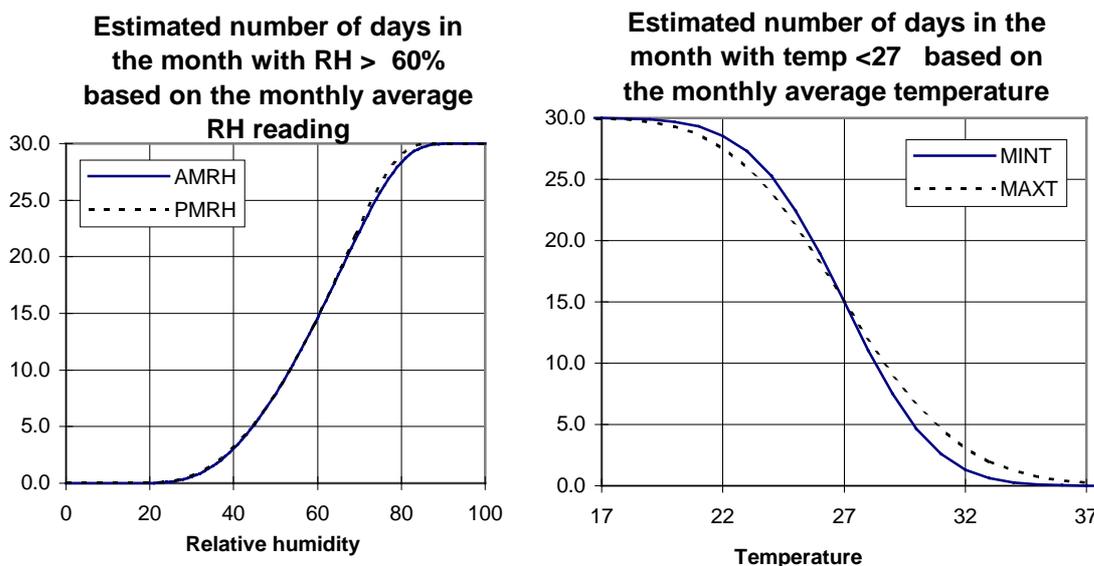
The annual estimate of the number of days suitable for aerosol survival can then be determined by adding the estimates for each monthly set of averages. When this method was applied to the monthly averages for the 113 sites, the overall average error was 0.8%, and the average absolute error was 6.2%. The estimates were within 20 days per year of the actual value and most were within 10 days.

This approximation was applied to the average monthly information for the 721 sites in the Bureau of Meteorology's Climate Data CD-ROM. The standard ArcInfo smoothing functions were then used to extrapolate across the whole of Australia using a quarter-degree grid. Figure 13 is a surface map of Australia showing the number of days a year conducive to survival of FMD virus in aerosols. The map is based on quarter degree grid cells (about 25 x 25 km) and is intended to give a broad guide to the likelihood of survival of virus in aerosol. Within any of the quarter degree grid cells there will, of course, be places with a greater or lesser risk because of localised conditions. It will be the weather conditions at the time of an outbreak that will determine if the virus will survive in an aerosol, and not any long term average.

7.6   Summary

The analyses of weather data shows that much of rural Australia has weather conditions suitable for the survival of FMD virus in aerosols for some part of the year. The difference between night and day is the most striking feature: such conditions are much more prevalent at night.

Not surprisingly there are seasonal and latitude effects. Winter has the most frequent, and summer the least, number of 12 hour periods suitable for survival of FMD virus in aerosol. There is a general trend of increasing suitability as one goes south, although this is modified partly by the distance inland.

While survival of airborne FMD virus is a necessary requirement for wind-borne spread to occur,.livestock density must also be considered when assessing the risk. There must be sufficient animals exposed down-wind to ensure that it is likely that at least one animal becomes infected. The next section looks at Australia's livestock distribution in terms of potential virus production and virus inhalation. Sections 10 and 11 look at weather conditions further to investigate how far the virus might spread downwind from a source.

8.   LIVESTOCK DENSITY

The previous section looked at the likelihood of weather conditions suitable for the survival of FMD virus in aerosol. However it matters little if the virus can survive somewhere if there are no animals present. This section looks at livestock distribution and density, the second component needed to assess the risk of wind-borne spread of FMD over Australia

This study of livestock density is only intended to give a broad picture of risk. Greater detail is not necessary. In the event of an outbreak, it will be the actual number of surrounding animals that will be used in assessing risk and assigning surveillance priorities. The aim of this section is to allow areas to be ranked in terms of their potential for wind-borne spread, using data on the type and number of stock present, and the proportion of the year that conditions are conducive to virus survival.

The first set of maps in this section give livestock distribution and density for cattle, sheep and pigs in animals per hectare. That we are looking at the broad picture is reflected in the distribution maps being based on quarter degree grid cells (about 25 x 25 km).

Given suitable weather conditions, the more animals that there are, the greater the risk of wind-borne spread. In assessing risk, we can look at animals in two ways – as potential virus producers and as potential receptors. Indeed we can combine both production and reception to give some measure of transmission potential.

The second set of maps combine the different number of each species in terms of cattle equivalents per hectare. We have used *cattle* equivalents since cattle are recognised as the species most likely to be infected by wind-borne spread. The *equivalents* in the different maps will depend on whether we are considering the potential production, reception or transmission of virus.

The third set of maps combines animal density information with the virus survival information of Figure 13. These maps are derived by multiplying the proportion of the year suitable for virus survival with the corresponding potential map on a cell-by-cell basis.

8.1   Livestock Distribution and Densities

Livestock numbers were mapped on a quarter degree grid cell basis for the whole of Australia.

Figures 14 and 15 give the distribution of cattle (including feedlots) and sheep. These maps were prepared by the National Resource Information Centre of the Bureau of Resource Sciences from the 1988/89 Australian Bureau of Statistics (Agstats) data and have been published in *Australian Agriculture: The Complete Reference on Rural Industry* (National Farmers' Federation, 1993).

Figure 16 gives the distribution of pigs and feral pigs. Feral pigs need to be considered because of the high virus production capabilities of pigs. The information on the location and size of piggeries was provided by the Pig Research and Development Corporation (PRDC). The distribution and relative density of feral pigs comes from *Pest Animals in Australia. A Survey of Introduced Wild Animals* (Wilson et al. 1992). The information on feral pig is less reliable that

the information on domestic pigs, and the subsequent analyses has been done both with and without feral pigs.

8.2 Livestock density in terms of potential virus production

One measure of risk is the potential virus production. The amount of virus produced by an infected animal will depend on many factors – species, type of virus, age of animal, and stage of disease are just some. The variation due to these factors may well be of use in considering the specifics of an actual outbreak, but in general add little to the broad picture. Species is the most clearly defined factor and we have used typical values of virus emissions reported in the literature (Section 4.4). To combine the different species numbers we have converted each species to cattle equivalents and then added them on a cell-by-cell basis.

Table 5 shows typical virus production by species in IU/day and corresponding cattle equivalents.

Table 5: Virus production capability by species, converted to cattle equivalents

Species	Virus	Production
	IU/day	Cattle equivalents.
Cattle	1.79×10^5	1
Sheep	1.79×10^5	1
Pigs	2.84×10^8	1,585

Potential virus production will be greatly affected by the numbers of pigs in an area, since one pig is equivalent to 1,585 cattle. Thus, the highest risk areas using this measure will be associated with areas of pig production.

Feral pigs are a potentially important source of FMD virus as well as domestic pigs. However, because of concerns about the quality and reliability of the data available on their distribution and densities (M Braysher, personal communication, August 1994), the reported densities of feral pigs could represent one potential source of error. Accordingly, we have undertaken the analysis both with and without feral pigs.

Figure 17 is a surface map for Australia showing potential virus production. This is based on density of livestock species (excluding feral pigs) — converted to cattle equivalents. It is readily apparent that southern and eastern Australia are the highest risk areas using this measure of risk.

Figure 18 shows potential virus production, with feral pigs included. The reported high densities of feral pigs in northern Queensland and central-western New South Wales account for the large increase in potential virus production.

This message has been included to indicate that printing has taken place.

42

8.3 Livestock density in terms of receptiveness to airborne infection

Another measure of risk is to consider the receptiveness to airborne infection, based on the infective doses and air sampling capacity of the different species. This measure is particularly useful for ranking the risk of spread at the start of an outbreak. Risk based on transmission potential (Section 8.4) is a better measure once the outbreak is well-established.

As with virus production, many factors influence whether an animal becomes infected from an aerosol dose – species, breed, sex, age, respiratory volume, time of year, etc. Again for our broad picture, we have treated species as the most clearly defined factor, and used typical values from the literature for susceptibility and respiratory volume as shown in Table 6.

The values for minimum infective doses are taken from Donaldson (1988). It should be noted that Sanson (1994) reports the minimum doses for cattle, sheep and pigs to be 9, 7 and 14 IU respectively. However, as these data originate from a personal communication rather than a published scientific report, the values of Donaldson (1988) were used here.

Table 6: Susceptibility to airborne infection by species, in cattle equivalents.

Species	Minimum Infective dose (IU)	Air sampling capacity (m^3/day)	Cattle equivalents
Cattle	18	144.0	1.00
Sheep	7	14.4	0.25
Pigs	11	7.2	0.08

While virus production is influenced by the distribution of pigs, receptiveness to infection depends largely on the distribution of cattle, since they are the species most likely to be infected, based on minimum infective dose and air sampling capacity. On an individual animal basis cattle are 4 times more susceptible to infection by inhalation than sheep, and 12.5 times more susceptible than pigs.

Figure 19 shows receptiveness of areas to airborne infection based on livestock (not including feral pigs) density. The susceptibility of each grid cell was rated according to the total number of cattle equivalents present. In Figure 20 the *relative* receptiveness of areas has been determined by multiplying the number of cattle equivalents by the number of days per year suitable for survival of virus in aerosols for each cell. The corresponding maps when feral pigs are included in the calculations are shown in Figures 22 and 23.

While the concept of a 'minimum' dose to infect animals is a convenient way to compare species susceptibility, it is important to recognise that the risk of infection in exposed animals is a probability function. Theoretically, a single virus particle could establish infection, although, practically, a larger dose is generally required. There is a limited amount of experimental work on the response of animals to aerosol doses of FMD virus. Section 9.4 discusses this further.

8.4 Livestock density in terms of transmission potential

The third measure of risk gives an indication of transmission potential. It combines the receptiveness to infection with the consequent virus production that could occur if the area became infected by wind-borne spread. The relative transmission potential is calculated for each cell of the map as the product of receptiveness (in cattle equivalents/ha), virus production (in cattle equivalents/ha) and proportion of the year suitable for survival of virus. The map emphasises areas where livestock numbers would lead to both a high virus receptiveness and a high level of virus production.

The findings, excluding feral pigs, are shown in Figure 21. The areas with the greatest potential for wind-borne spread occur in eastern and southern Australia. The corresponding findings, when feral pigs are included in the analysis, are shown in Figure 24.

8.5 Summary

This section has looked at the role of livestock in wind-borne spread: animals can both produce virus and inhale airborne virus. In order to rank areas of Australia for their suitability for wind-borne spread, we have defined three measures of risk based on livestock numbers — potential virus production, potential receptiveness to infection, and, by combining these, potential transmission. When the proportion of the year conducive to airborne virus survival for each area is taken into account, we can assign relative risk rankings.

Not surprisingly, the areas of greatest potential virus production are closely linked with the distribution of pigs. However all three measures gave a similar pattern: the relative risk is highest in southern and eastern Australia.

It needs to be emphasised that this analysis provides a simple overview of the suitability of different parts of Australia for wind-borne spread. It does not give the probability of spread actually occuring nor how extensive the spread might be. We still need to consider if the weather conditions are suitable to carry the virus downwind in sufficient quantity for infection to occur.

Section 9 describes the Gaussian plume model that has been used to predict the wind-borne spread of many substances. Sections 10 and 11 look at the component of Australia's weather that influences wind-borne spread.

9.   ASSESSING THE EXTENT OF WIND-BORNE SPREAD IN AUSTRALIA

The Gaussian plume model provides a method of calculating the concentration of virus downwind from a source of any given size. It is a useful tool that enables the effects of different source strengths, wind speeds and atmospheric conditions to be quantified. The advantages and limitations of the Gaussian approach are discussed in Sections 9.1 and 9.2. Section 9.3 describes the development of a plume model that was used to assess the factors affecting virus concentration in plumes. Section 9.4 examines the relationship between virus concentration and the likelihood that infection will result following exposure to various doses of virus. Section 9.5 shows the effects that different variables have on virus concentrations in plumes, a necessary precursor to understanding how wind-borne spread of virus will occur from infected properties.

9.1   Gaussian Plume Model

Aerosol virus emitted from infected animals will form a plume. As the plume moves downwind, it will be dispersed both vertically and at right angles to the wind direction. As has been described in Section 4.3, the key weather factors influencing this dispersion of FMD virus (as distinct to virus survival) are atmospheric stability and wind speed. Cloud cover is one of the prime determinants of atmospheric stability. Many other factors influence the behaviour of the plume such as the temperature and roughness of the surface over which the air passes, the presence of an inversion layer, and topographic features.

For a continuously emitting source, the Gaussian plume model has been widely used to quantify the effects of atmospheric turbulence on the dispersion of material injected into the atmosphere. The Gaussian model forms the basis for a number of FMD wind-borne spread models (e.g. Gloster et al. 1981, Donaldson et al. 1982, Gloster 1983, Donaldson et al. 1987b, Donaldson et al. 1988, Daggupaty and Sellers 1990, Sanson et al. 1991, Moutou and Durand 1994). This approach is popular because:

- it produces results that agree with experimental data;
- it is relatively straightforward to perform the calculations;
- it is conceptually appealing;
- it is consistent with the random nature of turbulence;
- it is compatible with input weather observations that are readily available;
- results can be obtained quickly to satisfy the demands of emergency decision-making.

9.2   Limitations of the Gaussian model

9.2.1   Distance from source

Daggupaty (1988) suggests that the Gaussian model can be used for ranges up to 50 km from source. However, in practice, most authors of FMD wind-borne spread models suggest that the use is limited to distances of 10 km from source. As the distance downwind increases, the predictions become less reliable. This is because the approach assumes that the properties of

the atmosphere remain essentially uniform in time and space (Bartlett 1973). Once distances exceed about 10 km from source the assumption is less likely to hold.

9.2.2 Topography

The Gaussian model assumes flat terrain. In fact, both topography and terrain affect the virus plume. The rougher the terrain, the greater the amount of turbulent mixing and therefore the greater the vertical and horizontal dispersion that will occur.

Topography will affect the path of a plume. An airstream will take the path of least resistance around hills and along watercourses and valleys (Donaldson 1988). It appears that towns form a barrier to airborne spread, although whether this is due to lack of susceptible animals, pollutants, or dispersal by convection currents is not known (Sellers et al. 1973).

Studies on the Hampshire outbreak (1966/67) in the United Kingdom (Blackall and Gloster 1981) found that the United Kingdom FMD model predictions of airborne spread from an infected source gave good agreement over flat terrain, but were less accurate over hilly terrain. Studies of mountain airflow show that hills and valleys influence the surface wind direction and although the effects are small in conditions of strong convection, they are large in stable conditions. The U.K. model has been modified to take into account gradients (Blackall and Gloster 1981).

In practice, when using a plume model, it may be necessary to modify the model or manually interpret its outputs where topography could have a strong influence on plume behaviour.

9.2.3 Deposition

The simple Gaussian plume model as given in Section 4.3 assumes that there is no loss of infective material. Often this does not matter. For a plume of, say, a toxic chemical, the severity of the response is related directly to the level of exposure and a low exposure level will not be severe, even if it is over a very wide area. Contrast this with a plume of virus particles. The response to a virus dose is either 'becomes infected' or 'remains uninfected'. Since the probability of infection depends on the dose, there is a minute probability that a very small dose can cause infection. Because of the highly infectious nature of FMD, it only takes one animal to become infected for the disease to establish.

Deposition needs to included in the model. Otherwise the mathematics of the model shows that while the concentration decreases with distance, the area covered by the plume increases is such a way that it is inevitable that an animal further downwind will become infected. This is not a realistic conclusion.

9.2.4 Weather data availability

The type of weather data that is readily available determines to a large extent what variables can be included in a model. For example, the Gaussian model can be improved by being expressed in terms of actual angular dispersion readings instead of an empirical horizontal dispersion coefficient. However such observations are not routinely made.

46

As another example, an atmospheric inversion layer can act as a reflecting barrier to the upwards dispersion of virus particles. Such a barrier can be readily incorporated into the plume model (eg Grace and Schahinger 1994). However data on inversions is not routinely available from weather records. In addition, the model without deposition shows that concentration at ground level increases by at most a factor of three over the model with no barrier. This increase will actually be less because the increase in ground level concentration will in turn increase the rate of deposition onto the ground. For these reasons, accounting for an inversion layer has not been included in our model.

9.2.5 Dispersion

Experimental studies show that the basic Gaussian model gives a good representation for the horizontal dispersion, but is not as good for vertical dispersion. Vertical dispersion is skewed and the Gaussian model can be modified with vertical dispersion being represented by two Gaussian processes, one each for upwards and downwards dispersion. This modification is especially relevant to convective conditions.

9.2.6 Summary

The Gaussian plume model works well for surface releases in stable conditions for distances of about 10 km. Various modifications can be made to increase the sophistication of the model, and other types of models have been or are being developed. However, these inevitably require more input data that is not usually available in routinely collected weather recordings.

A tactical plume model in the event of an outbreak could well use more parameters to increase the accuracy of its predictions provided that the appropriate weather observations are made.

The methodology and models used in this report are appropriate for the first order estimates of relative risk associated with wind-borne spread that are required by this report (W Grace, personal communication, July 1995).

9.3   Project model for FMD spread

A computer program of the Gaussian model coupled with a deposition model has been prepared. The program calculates the concentrations of FMD virus in the air downwind from a source of virus based on wind speed, time of day and cloud cover and deposition velocity. The variables used in the model are listed in Table 7 as a convenient point of reference for the subsequent description.

Table 7 List of variables used in FMD wind-borne spread model

x	the **distance downwind** from the source (metres)
y	the **distance at right angle** to the wind direction (metres)
z	the **height** above the ground (metres)
A - F	**atmospheric stability class**
u	the **wind speed** at the height of 10 metres (metres per second)
$\sigma_y(x)$	the **crosswind dispersion coefficient**
$\sigma_z(x)$	the **vertical dispersion coefficient**
α, β	constants used to relate σ_y to the distance downwind.
γ, δ	constants used to relate σ_z to the distance downwind.
$C(x,y)$	the **concentration at ground level** (particles per cubic metre)
$G(x)$	the **ground contact rate** (particles per square metre)
$Q(x)$	the **effective source strength** (particles released per second)
v	the **deposition velocity** (metre per second)
t	the **exposure time** (hours)
a	**air intake** (cubic metres per hour per animal)
d	animal **density** (animals per square metre)
$D(x,y)$	the **dose** inhaled (particles per hour per square metre)
$E(x)$	the total **exposure** over area under consideration (particles per hour)
θ	**infectability**: probability that one virus particle will infect an animal
$P(x)$	**probability** that at least one animal downwind of x is infected

9.3.1 Atmospheric stability

Atmospheric stability is the major factor in determining how the plume spreads. This spread is reflected in the crosswind and vertical dispersion coefficients. The stability of the atmosphere generally depends on wind speed and the amount of sunlight (insolation) during the day or cloud cover at night. There have been many attempts to categorise weather conditions and give a mathematical relationship between the dispersion coefficients and stability.

Pasquill (1961) defined six different categories (A-F) for describing atmospheric stability (Table 8). He related them to five classes of surface wind speeds, three levels of daytime insolation and two levels of night-time cloudiness (Table 9). Since the Bureau of Meteorology reports cloud cover in oktas (1/8ths of the sky covered by cloud) we have interpolated this relationship as shown in Table 10.

Table 8 Atmospheric stability categories, with constants used for dispersion coefficients

Pasquill category	Atmospheric stability	α	β	γ	δ
A	Very unstable	1.38	0.76	0.32	0.95
B	Mod unstable	1.00	0.76	0.68	0.81
C	Slightly unstable	0.71	0.76	0.96	0.67
D	Neutral	0.50	0.76	1.32	0.53
E	Slightly stable	0.33	0.76	1.98	0.39
F	Moderately stable	0.27	0.76	2.28	0.31

Table 9 Atmospheric stability categories (Pasquill 1961)

Surface wind speed (m/sec) at 10 m	Daytime insolation			Night	
	strong	moderate	slight	thinly overcast or >4/8 cloud	<=3/8 cloud
<2	A	A-B	B	—	—
2-3	A-B	B	C	E	F
3-5	B	B-C	C	D	E
5-6	C	C-D	D	D	D
>6	C	D	D	D	D

Table 10 Atmospheric stability categories based on wind speed and cloud cover

speed m/s	Day time: cloud cover (oktas)									Night time: cloud cover (oktas)								
	0	1	2	3	4	5	6	7	8	0	1	2	3	4	5	6	7	8
1	A	A	A	A	B	B	B	B	B	F	F	F	F	E	E	E	E	E
2	A	A	A	A	B	B	B	B	B	F	F	F	F	E	E	E	E	E
3	A	B	B	B	B	B	C	C	C	F	F	E	E	E	E	D	D	D
4	B	B	B	B	C	C	C	C	C	E	E	D	D	D	D	D	D	D
5	C	C	C	C	C	D	D	D	D	D	D	D	D	D	D	D	D	D
6	C	C	C	C	D	D	D	D	D	D	D	D	D	D	D	D	D	D
7+	C	C	C	D	D	D	D	D	D	D	D	D	D	D	D	D	D	D

9.3.2 Dispersion coefficients

The dispersion coefficients σ_y and σ_z used in the Gaussian model equation are functions of downwind distance and atmospheric stability. There are several methods for deriving the form of the coefficients, based on a combination of experimental results and theory. One relationship between the dispersion coefficient and distance downwind that has been used by several authors is a simple power relationship:

$$\sigma_y = \alpha\, x^\beta \quad \text{and } \sigma_z = \gamma\, x^\delta$$

As an example, such a relationship is implicit in the spore dispersal models described in Gregory (1961). This form of the dispersion coefficients was chosen because it simplifies the calculation of deposition (9.3.3). The four coefficients, α, β, γ and δ depend on the stability category and have been calculated (Table 8) to match the widely used formulas recommended by Briggs (1973). Dispersion is discussed in detail by Hanna et al. (1982).

The dispersion coefficients, σ_y and σ_z, determine the concentration of virus downwind. If crosswind dispersion (σ_y) is small, the plume is narrow with high virus concentration. Increasing σ_y will increase the width of the plume but reduce the peak concentration in such a way that the overall risk of infection is unchanged. Increasing σ_z reduces the concentration at ground level, and consequently the risk of infection.

9.3.3 Deposition

The concentration of airborne virus in a plume will be reduced by physical loss through deposition. Virus particles deposited on the ground are not considered likely to cause infection because of the much greater dose required for infection by ingestion. If the vertical dispersion coefficient (σ_z) is large, the virus particles rapidly move above ground level, and so the loss from deposition is small. However, when σ_z is small, more of the particles stay at ground level and the rate of loss through deposition is greater.

A common method of adding deposition to the plume model is to use the *source depletion model* in which the apparent strength of the source is reduced as one goes downwind to allow for the diminishing amount of virus remaining aloft (Hanna et al. 1982). The constant source Q in the Gaussian plume equation is replaced by a function Q(x) so that virus concentration reduces with distance downwind. The derivation of this function for the project model is described below.

For wind-borne spread of FMD virus, both the height of the source and the height of the receiver can be treated as zero. With deposition, the *concentration at ground level* from a source at ground level is:

$$C(x,y) = Q(x) \exp(-\tfrac{1}{2}y^2/\sigma_y^2) / \pi / u / \sigma_y / \sigma_z$$

If there was no deposition, the function Q(x) would simply be a constant equal to the strength of the source, the usual form of the Gaussian plume equation.

The starting point in deriving Q(x) is to calculate the amount of virus in contact with the ground at a distance x from the source. This is given by:

$$G(x) = \int C(x,y)\, dy = \sqrt{(2/\pi)}\, Q(x) / u / \sigma_z$$

The rate that Q(x) changes is proportional to the amount of virus in contact with the ground

$$dQ(x)/dx = -v\, G(x) = -v \sqrt{(2/\pi)}\, Q(x) / u / \sigma_z$$

The constant **v** is called the *deposition velocity* and depends on the size of particles and the roughness of the terrain. For FMD virus it is typically 0.01 metres/sec (Rumney 1986).

For $\sigma_z = \gamma\, x^\delta$, the strength of the apparent source becomes:

$$Q(x) = Q(0) \exp(-\sqrt{(2/\pi)}\, v\, x / u / \sigma_z / (1-\delta))$$

9.3.4 Inactivation

As well as deposition, the plume model needs to include the inactivation of the virus. We have assumed that there will be minimal inactivation while the temperature is below 27°C and the relative humidity is above 60%. Once these bounds are exceeded we have assumed inactivation occurs immediately and completely.

9.3.5 Calm air

The Gaussian plume model is undefined if the wind speed is zero. For the project we have assumed that calm air corresponds to a speed of 0.5 m/s as suggested by Hanna (1982).

9.4 Relating virus concentration to probability of infection in exposed animals

Establishment of infection in a susceptible animal depends on the dose of airborne virus to which it is exposed. The dose is determined by the concentration of virus, the air sampling capacity of the animal, and the period of exposure. The probability of infection following exposure to a low dose is small, but increases as the size of the dose increases.

Although mechanisms such as virus inactivation and clearance by the host means that a 'large' dose is required to establish infection, in theory, a single infectious particle could do so. In this report, *infectability* (θ) has been defined as the probability that one virus particle will infect an animal. The probability (P) that at least one animal in a group will become infected following exposure of the group to a combined dose (D) is given by the formula:

$$P = 1 - (1 - \theta)^D$$

There is a limited amount of experimental work on the response of animals to aerosol doses of FMD virus. For example, at the Animal Virus Research Institute, Pirbright, sheep (Gibson and Donaldson 1986) and calves (Donaldson et al. 1987a) were exposed individually to aerosols of FMD. By calculating the exposure doses the authors were able to estimate minimum infective doses. We have re-analysed the results of these experiments to estimate the small probability that exposure to a single IU would result in infection in individual animals. The estimates obtained for θ were $\theta = 0.06$ for sheep, and $\theta = 0.03$ for calves. Figure 25 shows the dose-response curves that can be drawn from this re-analysis.

Figure 25:   Dose-response curves for exposure to FMD virus in aerosols
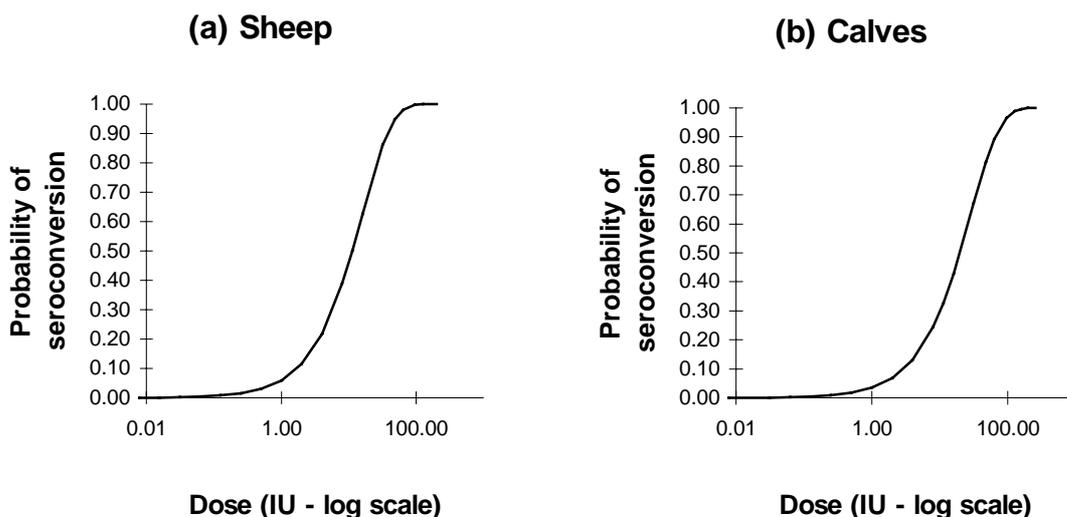
Although these curves are based on limited data, they do give some indication of how the probability of infection increases within increasing exposure to FMD virus. From these curves, a dose of 11 IU per sheep or 20 IU per cow would infect about 50% of the animals exposed.

Group size is also important. For example for cattle, the probability of an individual animal becoming infected after exposure to 1 IU is 3%, but the probability that at least one animal in a group of 100, each exposed to 1 IU, will become infected is 97%. Although the infectability for cattle is less than sheep, for the same concentration of virus, cattle are a greater risk of becoming infected because they sample air at a greater rate than sheep, and so are exposed to a higher dose.

Given the highly contagious nature of FMD, it only takes one animal in a group to become infected for the disease to establish. Once one animal in a group is infected, the disease will rapidly spread to animals in close contact. Thus for the same virus concentration, the size of herd/flock exposed to virus becomes a key issue in determining the likelihood that infection will occur on a farm downwind. From the point of view of initial establishment of disease into a group, it matters little whether one animal receives a dose of 100 IU or 100 animals receive a dose of 1 IU, although the latter has the potential to provide more infected animals initially.

Within a Gaussian plume, the *dose,* $D(x,y)$ at a point (x,y) downwind, is the product of the concentration of virus at that point, $C(x,y)$, the air intake of the species, a, the animal density, d, and the exposure time, t:

$$D(x,y) = a\ d\ t\ C(x,y)$$

In the examples used in Section 9.5 to illustrate the effects of different weather conditions on virus plumes, we have defined *exposure*, $E(x)$, as the total amount of virus inhaled by all animals further downwind than a point, x. Exposure will be related to the amount of virus in contact with the ground, $G(x)$, by:

$$E(x) \quad = \int_{x}^{\infty} a\ d\ t\ G(w)\ dw$$

But $dQ(w)/dw = -v\ G(w)$ and so

$$E(x) \quad = \ a\ d\ t\ Q(x)\ /\ v$$

The probability that at least one animal downwind from x will become infected is:

$$P(x) = 1 - (1 - \theta)^{E(x)}$$

It should be pointed out that for the small probabilities that we are dealing with, the effect of change in parameters such as time, density and size of source is simply multiplicative. For example doubling the cattle density will double the risk. Similarly, halving the number of source animals, will half the risk.

These formulae allow us to determine the probability of infection resulting at various distances downwind. As international guidelines (OIE 1992) recommend that an *infected zone* should extend for a minimum of 10 km around foci of infection and as this distance is used in the AUSVETPLAN strategy for FMD (DPIE, 1990), 10 km downwind becomes a convenient reference point. This enables us to calculate the probability of animals outside of the restricted area becoming infected through wind-borne spread. This is of key importance to disease control authorities because such spread may not be contained by the normal control measures put in place. This distance also represents the generally recommended distance over which the Gaussian plume model is considered appropriate.

9.5   Effects of different variables on the plume

This section discusses how different factors such as source strength, deposition rate and weather conditions affect the concentration of FMD virus in plumes.

Examples provide the easiest way to illustrate the effects that the different parameters have on the concentration of the virus downwind of the source. In the examples presented, unless otherwise stated, *the same values for key variables will be used* with only one condition changed at a time. The following standard values have been used:

- wind velocity is 3 m/s
- deposition velocity is 0.01 m/s
- atmospheric stability category is D
- source of virus is 100 pigs
- animals at risk are cattle breathing at 6 cubic metres/hour
- animal density is 1 per hectare
- period of exposure is one hour

Three types of figures will be used to illustrate virus concentrations in plumes.

Three-dimensional graphs show actual virus concentrations at ground level downwind and crosswind from the source.

The second type of graph shows the contour line for a given dose of virus (isopleth) for different values of the parameter being investigated. The line on the graph represents the virus concentration of one millionth of the source strength. This corresponds, for a source of 100 infected pigs over a 5 hour period, to a cattle dose of FMD virus of about 10 IU.

The third type of graph relates distance from the source to the probability that animals further downwind would become infected following one hour's exposure to the plume.

9.5.1   Effect of atmospheric stability:

Three-dimensional graphs have been used to show the effects of different atmospheric stability conditions on the virus concentration (IUs per cubic metre) at ground level under the standard parameters listed above. Three atmospheric stability categories (B, D, and F) are illustrated in Figure 26.

The viewpoint of the graphs is some 20 km downwind looking towards the source of the plume. The graphs start 2.5 km from the source. Near the source, the concentration of virus is, naturally enough, quite high. The graph for stability category B (moderately unstable) shows much lower virus concentration compared to the stability category D (neutral). This, in turn, is much lower than stability category F (moderately stable) which is still showing a peak concentration of 0.3 IU per cubic metre 20 km downwind of 100 infected pigs.

Figure 26   Effect of atmospheric stability on virus concentration downwind

Stability category B
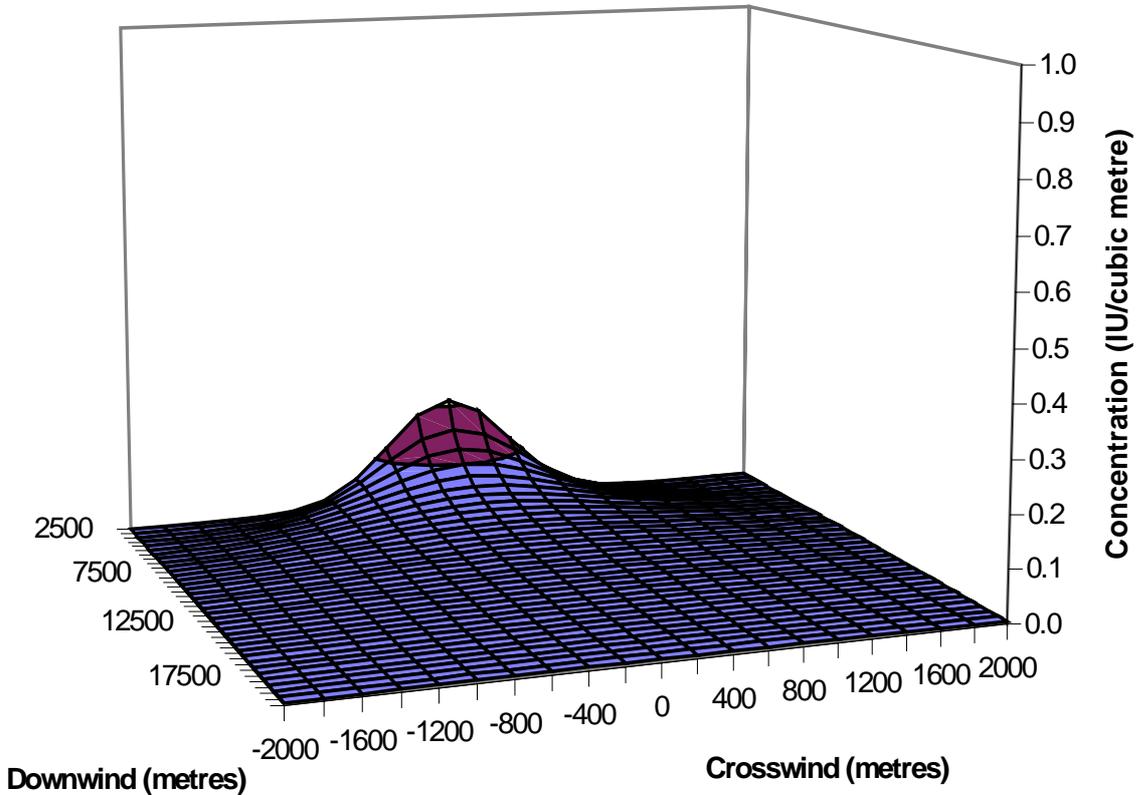
Stability category D
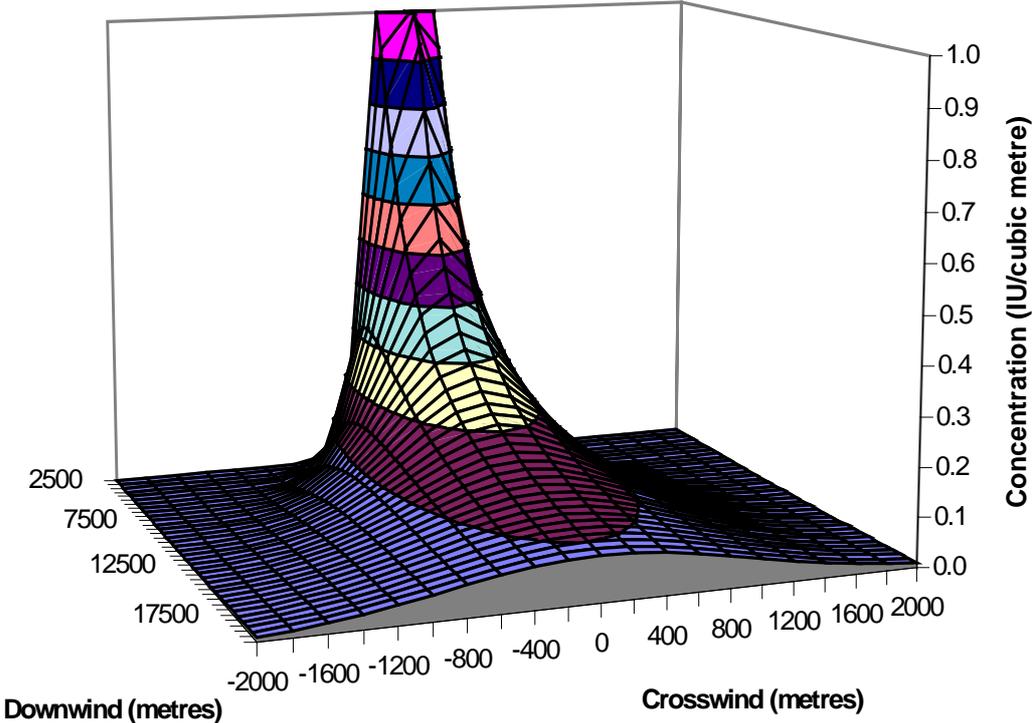
Stability category F
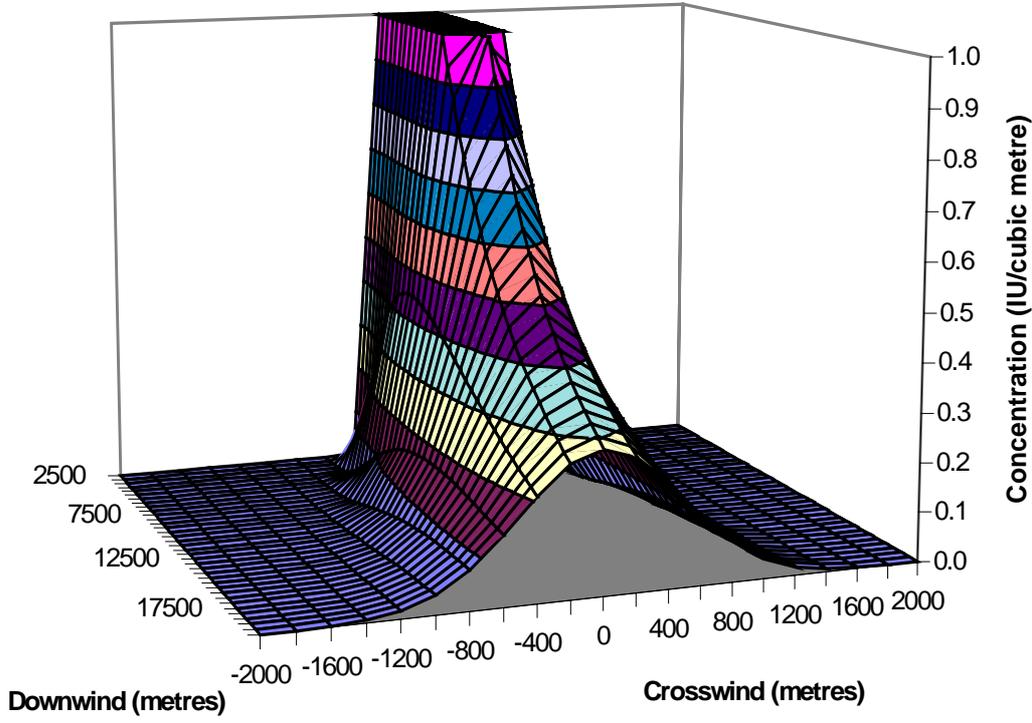
We can also show the concentration information of Figure 26 in the other two formats that we are using to illustrate the effects of the various parameters for the plume model. Figure 27 shows the 10 IU isopleth with different atmospheric stability conditions. The effect on the probability that animals downwind would become infected is shown in Figure 28.

Figure 27   Virus plume isopleths (10 IU) with different atmospheric stabilities

Atmospheric stability categories with deposition
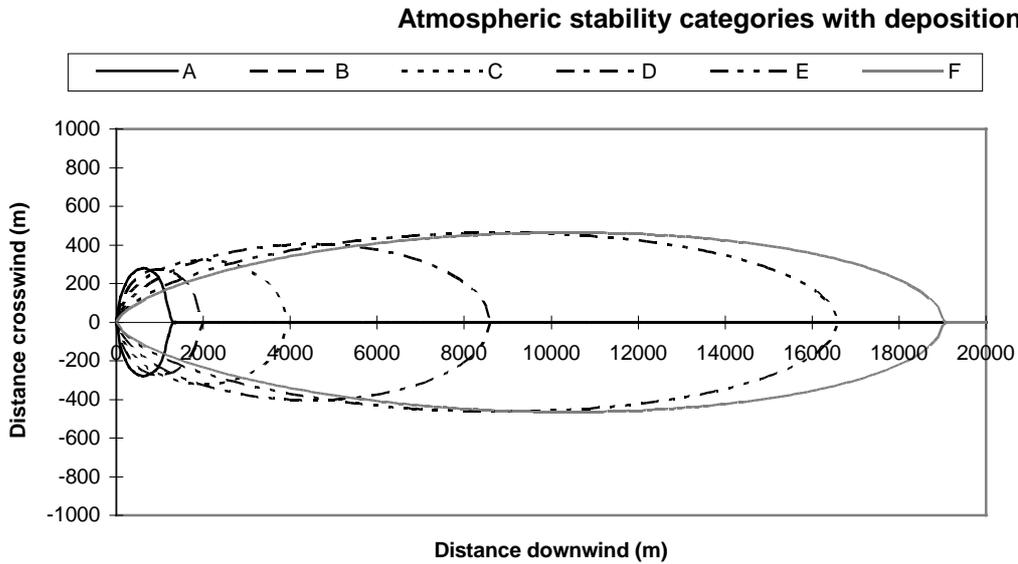
Figure 28   Probability that at least one animal will be infected further downwind after one hour's exposure for different atmospheric stabilities

Atmospheric stability categories with deposition
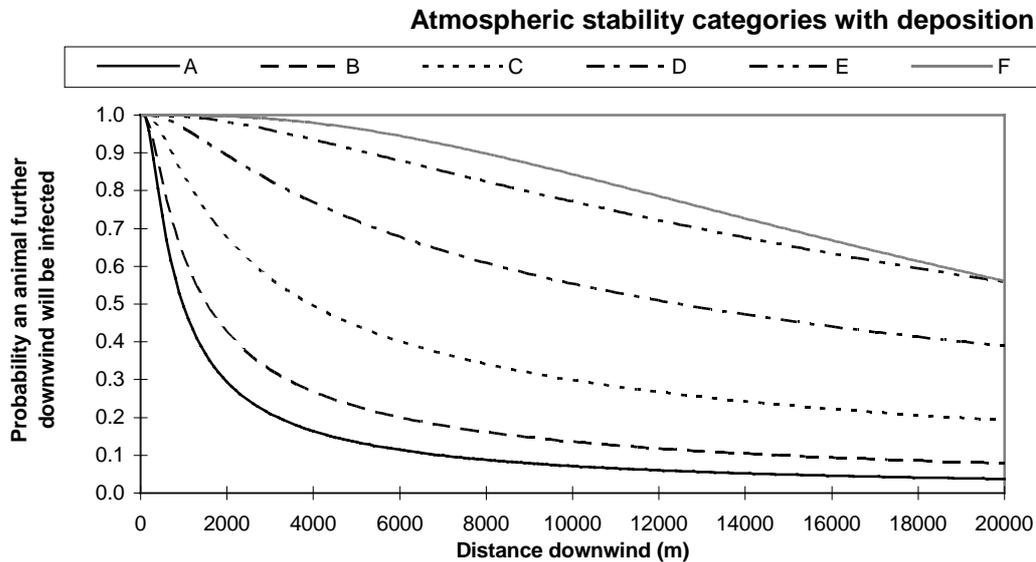
9.5.2 Effect of no deposition

Figure 29 shows isopleths with the different atmospheric stability categories if there is no deposition. Compare this with Figure 27. Ignoring the effects of deposition leads to a higher estimate of the concentration in the plume. There is little difference for the less stable atmospheric conditions, but considerable difference for the plumes in stable atmospheric conditions.

Figure 29   Virus plume isopleths (10 IU) with no deposition for different atmospheric stabilities
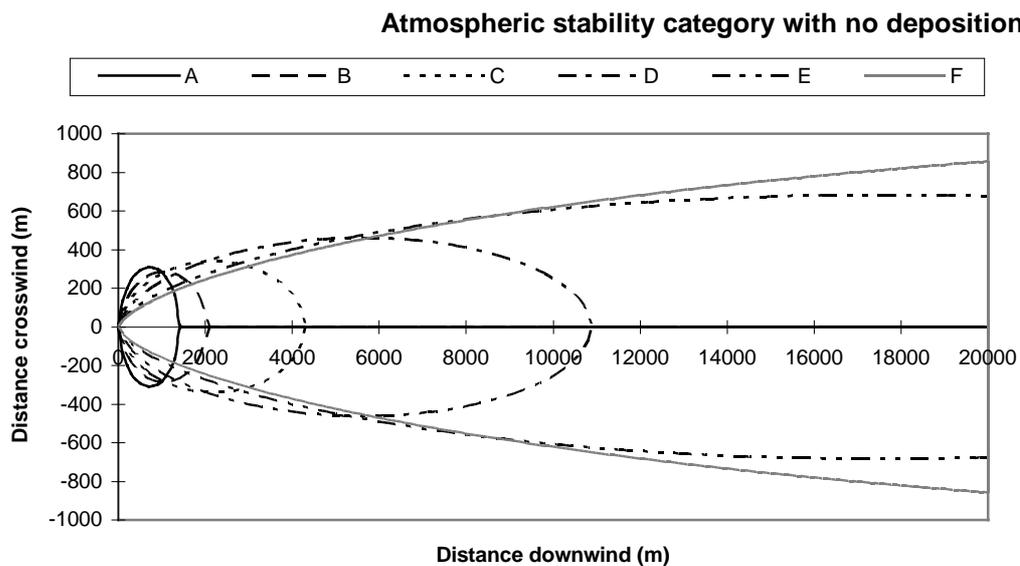
9.5.3 Effect of non-point source

The Gaussian model is based on a point source. Where there are multiple sources, one can combine the plumes from each source to obtain the concentration downwind. Close to the sources, the concentration along a line at right angles to the wind direction will show peaks ('hot spots') corresponding to the individual sources. Further from the sources, the peaks will coalesce, giving a single, broader peak. The distance at which this happens will depend on the separation distance of the sources. Because of this we can treat a property as a *point* source rather than an *area* source. However if the source is spread over a large area, such as a dispersed group of feral pigs, the plume would be diffuse with virus concentration downwind at right angles to wind direction practically uniform.

Figure 30 illustrates this effect by plotting the concentration of virus at ground level from 100 pigs divided into several groups. The second graph shows virus concentration downwind from 5 groups, 200 metres apart along a line at right angles to the wind direction. The maximum concentration across a line at right angles to the wind direction is less than if the 100 pigs were located at the one point but the spread is broader. The third graph, with 11 separate groups of 9 pigs, shows an almost constant concentration for several kilometres downwind.

Having multiple sources might change the maximum concentration downwind, but does not have a great effect on the risk that infection would result. This is because while the maximum concentration is lower, the area over which it spreads is greater. The total amount of virus downwind depends on the strength of the source regardless of how the source is distributed.

Figure 30   Effect of a non-point source on virus concentration downwind

Stability category D, 1 source of 100 pigs
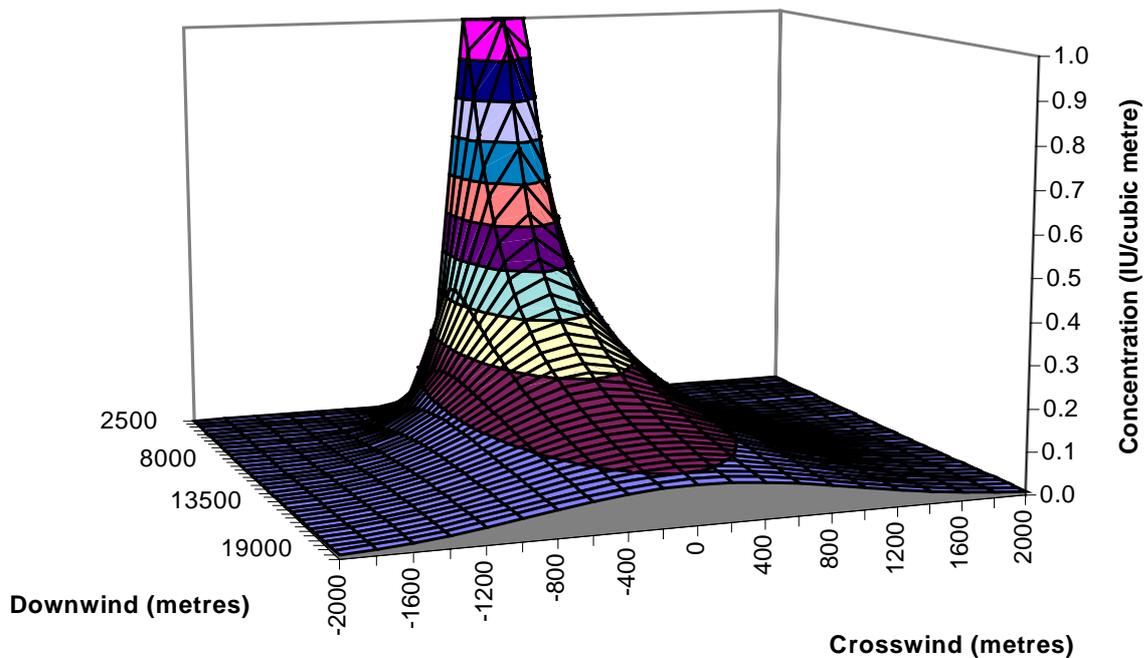
Stability category D, 5 sources of 20 pigs
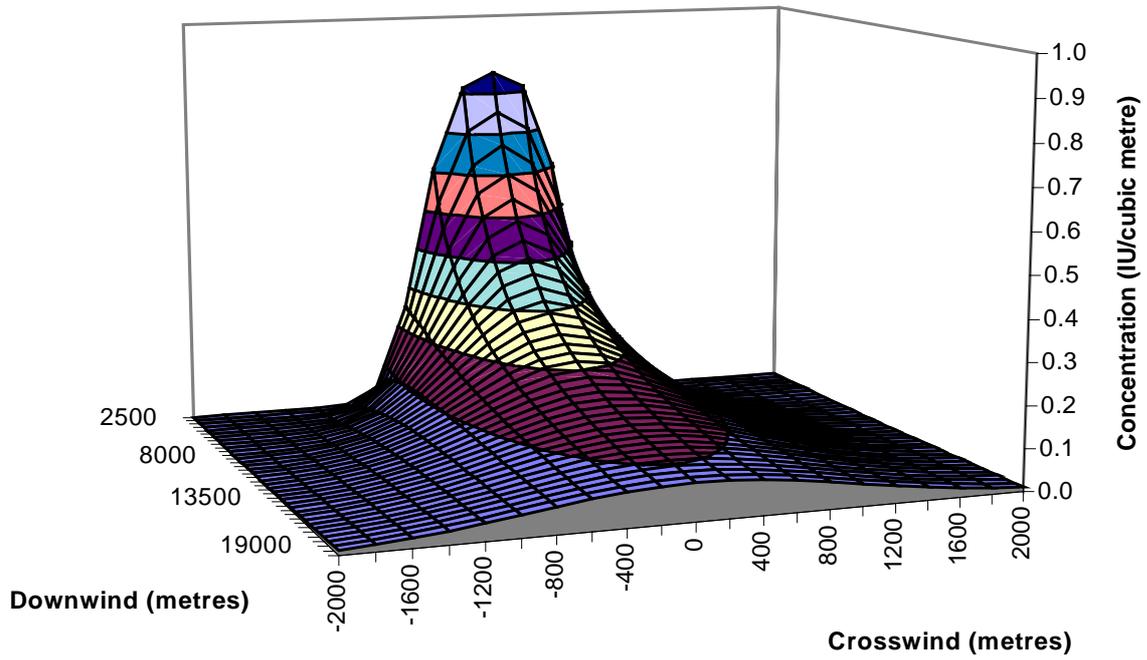
Stability category D, 11 sources of 9 pigs
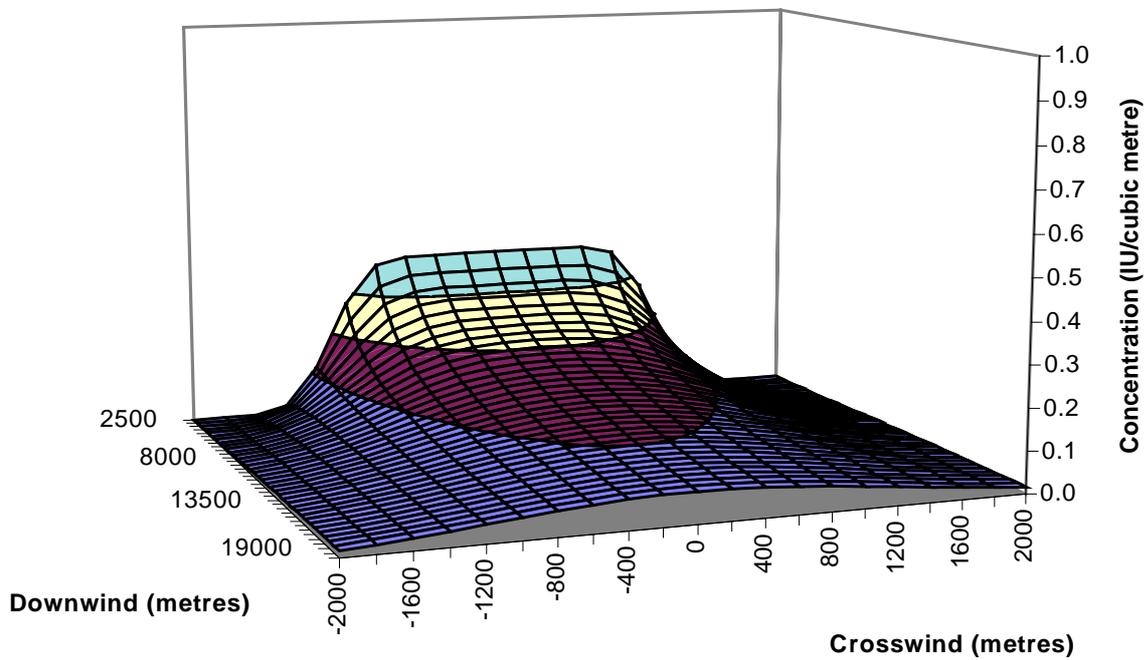
9.5.4 Effect of wind speed

Wind speed is one of the factors that determines atmospheric stability.However, for a given category of atmospheric stability, as wind speed rises the effective strength of the source is diluted because more air passes the source per second. Figure 31 shows this dilution effect of wind speed on the concentration of virus downwind for the stability category D. Figure 32 shows what effect wind speed has on the likelihood that animals downwind would become infected if exposed.

Figure 31   Virus plume isopleths (10 IU) with different wind speeds
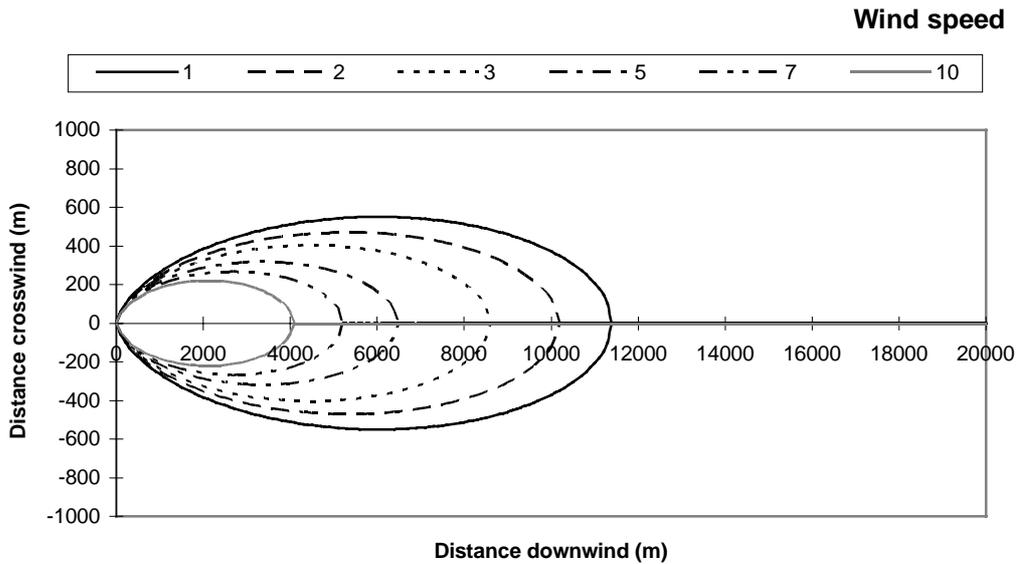
Figure 32   Probability that at least one animal will be infected further downwind after one hour's exposure at different windspeeds
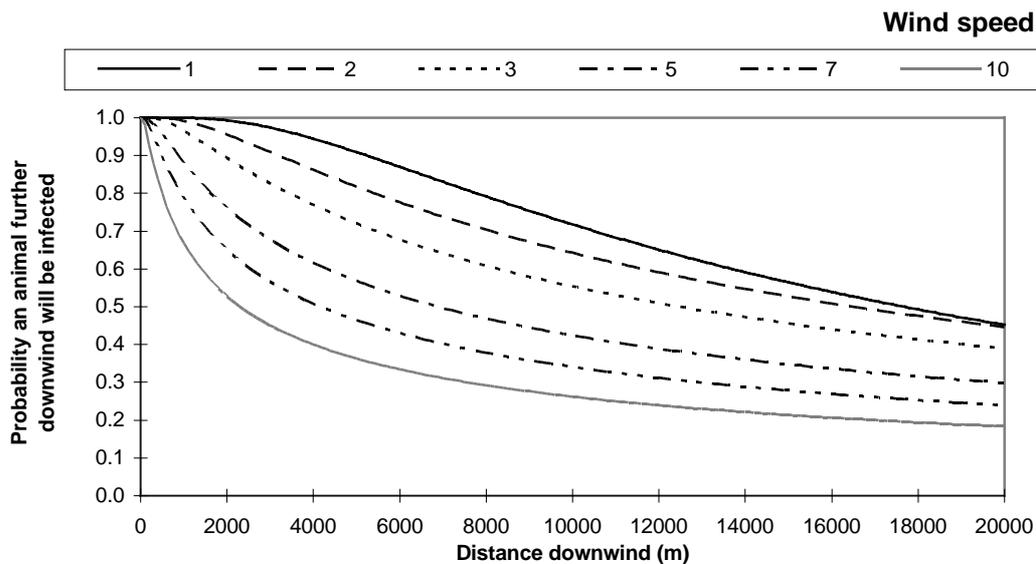
9.5.5 Effect of deposition velocity

The effect of deposition on virus concentration in plumes is clearly seen in Figure 33. The greater the deposition rate Q(x), the more quickly the fall in virus concentration as one moves downwind. Q(x) depends on deposition velocity, the value of which is typically taken as 0.01 m/s (Section 9.3.4). Figure 33 shows isopleths with various deposition velocities from 0 to 0.05 m/s. The effect of these deposition velocities on the probability that animals downwind would become infected is shown in Figure 34.

Figure 33   Virus plume isopleths (10 IU) with different deposition rates
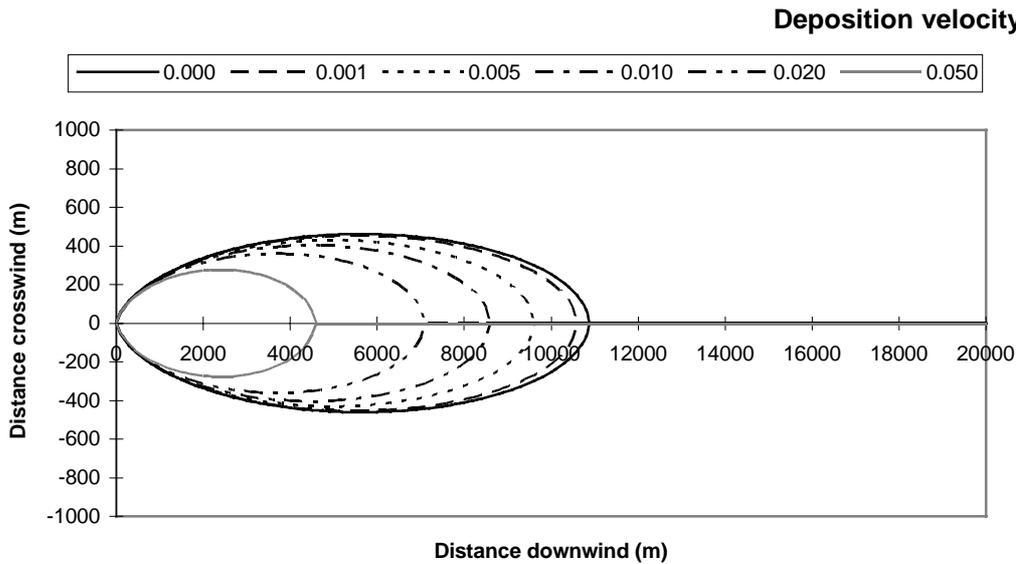
Figure 34   Probability that at least one animal will be infected further downwind
after one   hour's exposure for different deposition velocities
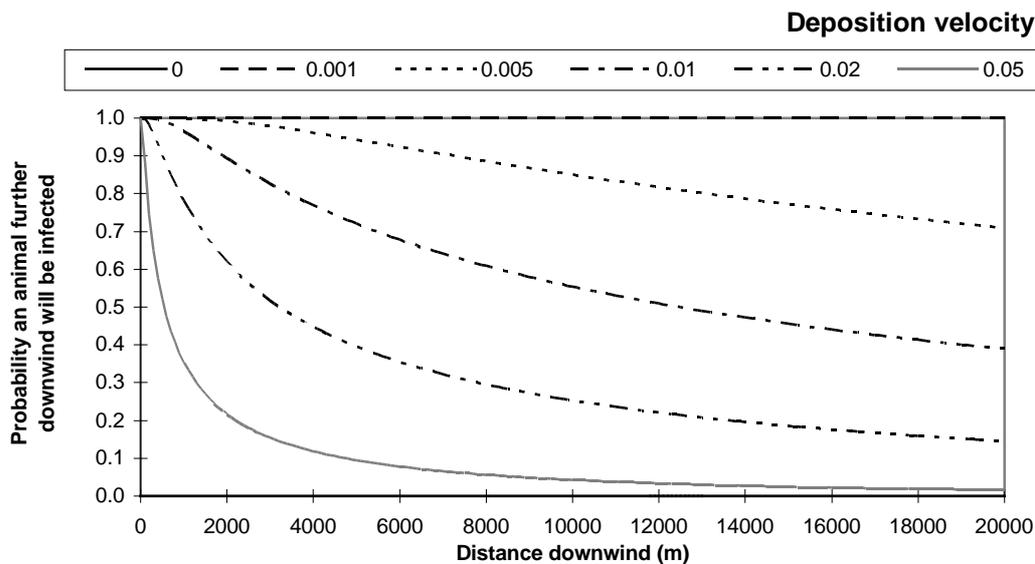
9.5.6 Effect of source strength

From the Gaussian plume equation, the concentration of virus downwind is directly proportional to the source strength. Increasing the source strength increases the concentration of virus at any given point downwind and increases the total area at risk. Figure 35 shows isopleths, under the standard conditions for sources of 5, 10, 25, 50, 100 or 250 infected pigs.

The effect of source strength on the probability that animals downwind would become infected is shown in Figure 36.

Figure 35   **Virus plume isopleths (10 IU) with different source strengths**
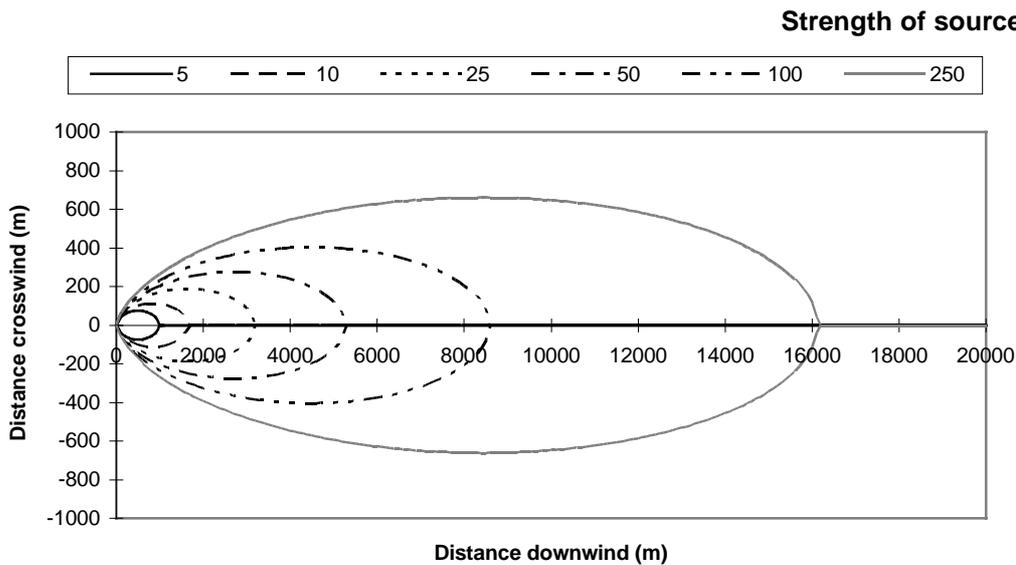
Figure 36   **Probability that at least one animal will be infected further downwind after one hour's exposure for a different number of infected pigs**
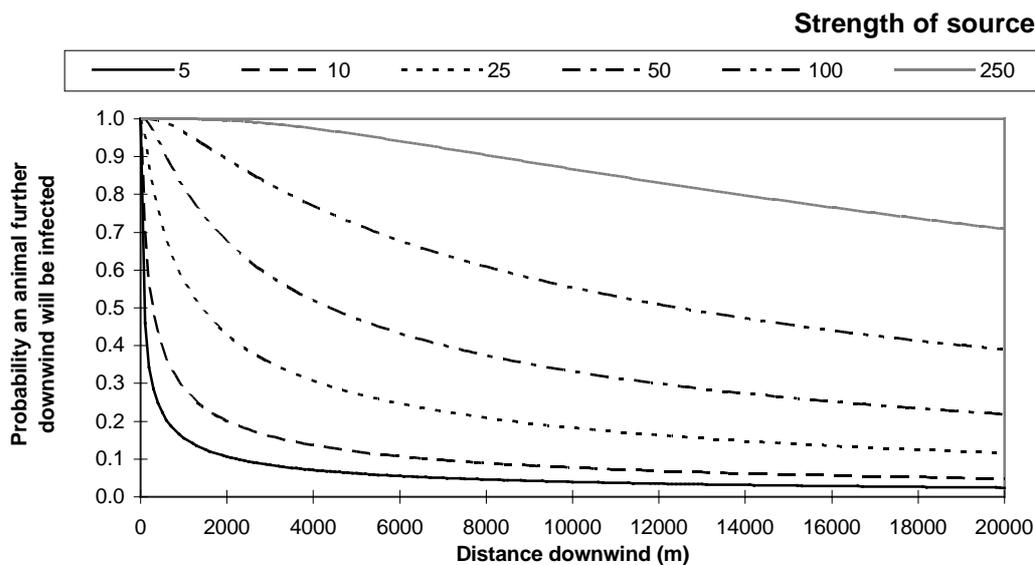
9.5.7 Effect of exposure time

As there is a direct relationship between exposure time and dose that an animal downwind receives, the longer an animal is exposed the greater the probability that it will become infected.

Figure 37 shows how long animals at different points downwind would need to be exposed to the plume to receive a dose of 10 IU. For example, under the standard set of conditions, cattle 10 km downwind would need to be exposed for more than 6 hours to receive a dose of 10 IU.

Figure 37   Isopleths corresponding to a dose of 10 IU for different exposure times

Hours until a dose of 10 IU
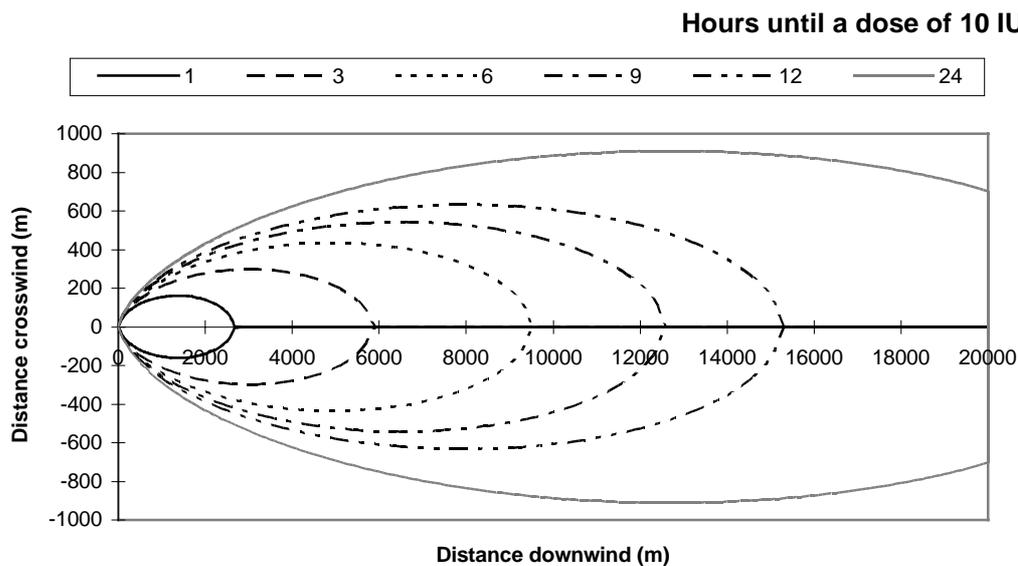
9.5.8 Effect of wind direction

Wind direction, of course, has no direct effect on the concentration of virus downwind. However in the event of an outbreak wind direction is possibly the most important weather parameter to monitor frequently in assessing *where* airborne virus might be expected and hence allocating surveillance effort.

The analysis of Section 11 illustrates the effect of wind direction.

9.5.9 Effect of animal density downwind

The higher the stock density downwind, the greater the probability that infection will result, as shown in Figure 38.

Figure 38 **Probability that at least one animal will be infected further downwind after one hour's exposure with different livestock densities (cattle per ha)**
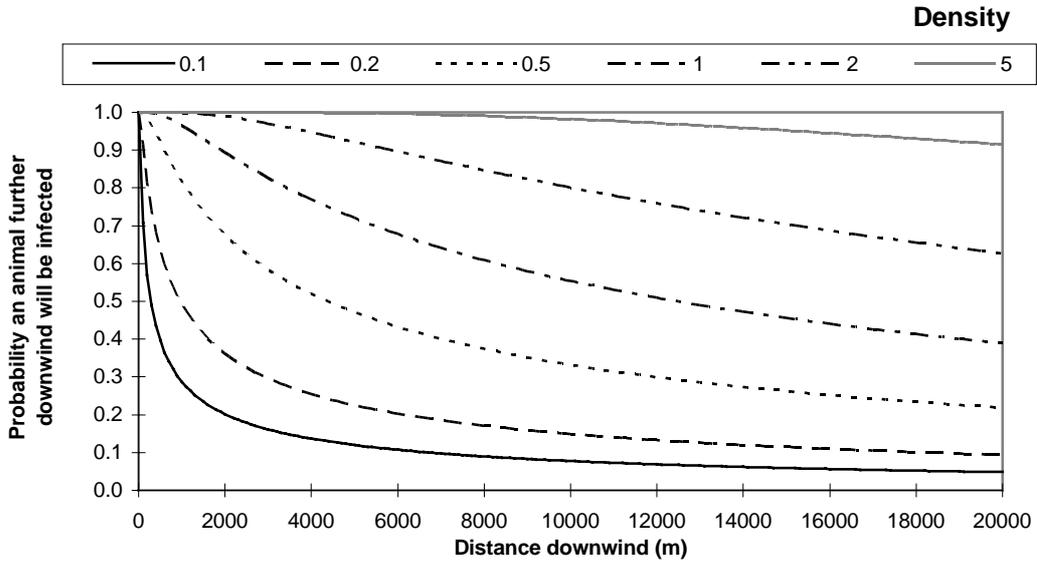
10. COMPARING SITES FOR POTENTIAL EXTENT OF WIND-BORNE SPREAD OF FMD

In Section 7 of this report, individual sites in Australia were compared for their suitability for survival of FMD virus in aerosols, based on temperature and relative humidity criteria. To assess the *extent* of possible spread it is necessary to take into account factors such as atmospheric stability and wind speed.

Locations with similar numbers of days per year conducive to virus survival in aerosols could have quite different potential for spread because of different wind speed and atmospheric conditions. To illustrate differences in the extent of spread that could occur in different parts of Australia due to these parameters, actual weather data from a range of sites have been used. By using a standardised source of virus and a standardised group of animals exposed, it is possible to make comparisons between different areas. This Section compares sites by looking at the concentration of virus to which 100 cattle at a point 10 km downwind would be exposed, from a source of 100 infective pigs. The results are presented as hourly mini-bar charts, each covering a period of 3 months.

We have used the same sites as were used in Section 7 so that comparisons can be made between the number of days suitable for survival of FMD virus in aerosols and the potential extent of FMD spread that could occur from these sites.

10.1 Weather Data

Data from the Bureau of Meteorology's three-hourly series was used to provide realistic weather patterns for the analyses in both Sections 10 and 11.

The weather data was interpolated to one-hourly values to smooth the transitions between time periods. Figure 39 shows the data used to derive the concentration of virus at a point 10 km downwind from the source. The data are presented as mini weather charts. The horizontal scale has one dot corresponding to one hour. The vertical scale on the various weather charts depends on the parameter being graphed — it is the general shape that is important, not the absolute values.

The model looks at what happens to each hour's virus production as it moves the 10 km downwind. If, at any time on this journey, the temperature or relative humidity become unsatisfactory for virus survival (temp $> 27^{\circ}$C, RH $< 60\%$) it is assumed that no virus will reach the destination. Such events are shown by the *hollow parts* of the temperature and relative humidity charts. These events are incorporated as *hollow parts* of the wind speed chart to show those times when viable virus particles would not reach the destination.

Wind direction is needed for deriving plume 'rosette' diagrams in Section 11, but is not needed in this analysis since we are simply looking at the variable point 10 km downwind. The graph plots wind direction twice because wind direction uses a circular scale (0 deg = 360 deg).

The wind speed, time of day and cloud cover determine the atmospheric stability category. For simplicity, we have treated the period from 6 am to 6 pm as daytime regardless of season or

geographic location. Because the atmospheric stability differs considerable between day and night, there is a complication with journeys covering dawn and dusk

This issue has been resolved, pragmatically, by using the starting time for most journeys as the time of day for determining the atmospheric stability. For journeys that start less than two hours before dawn (6 am), night conditions are assumed to apply if the journey finishes within two hours of dawn, and day conditions otherwise. Similarly a journey starting less than two hours before dusk (6 pm) will be a day one if it finishes within two hours of dusk, and a night one otherwise.

Having determined the atmospheric stability, the concentration of virus at the 10 km point can be calculated using the formulas of Section 9.

10.2   Findings

The results of the analysis are shown in Figure 40a–f. The sites were chosen on the basis of availability of weather data with no consideration given to the values of the actual data. Each location is represented by four mini-bar charts each covering a 3 month period, so that a full year is shown for each site. The charts show graphically how much virus 100 cattle, 10 km downwind from a source of 100 infective pigs, would be exposed to on an hourly basis. For this set of conditions, the maximum hourly dose is nearly 1000 IU, corresponding to an average dose of 10 IU per animal. Suggested minimum infectitive dose for cattle range from 9 IU (Sanson 1994) to 18 IU (Donaldson 1988).

The charts show how often, throughout the year, virus would reach 10 km downwind. The higher and wider the peaks, the more virus reaches this point, the more likely that infection would result and the greater the extent of wind-borne spread of FMD from that site. The higher the peaks, the greater the virus concentration, and the wider the peaks, the longer that conditions are suitable for spread.

These charts should be treated as a means of comparing the *relative risk* of long distance spread (10 km and beyond) between sites and between seasons. It is the pattern of the peaks that is the important feature of the charts, rather than the absolute height of the various peaks since we have standardised the outbreak conditions (size of source and exposed livestock). In an actual outbreak, amount of virus inhaled is directly proportional to the strength of the source, and the density and type of livestock downwind.

The analysis can be applied to any site for which standard meteorological data is available. We have used the same sites as were used in Section 7 so that comparisons can be made between the number of days suitable for survival of FMD virus in aerosols and the potential extent of FMD spread that could occur from these sites.

The most noticeable feature of the charts is the pronounced diurnal variation in potential spread shown by spikes in virus concentration at night. Section 7 has already shown the difference in virus survival between night and day but this difference in potential spread is greatly magnified by the central role that day/night has in determining atmospheric stability. Atmospheric stability is the most important factor affecting the dispersal of virus particles. As the atmosphere tends to be more stable at night, it is not surprising that the highest concentrations of virus downwind occur then.

Figure 39   Weather data, interpolated to hourly values, used to derive virus
             concentrations downwind

[Figure 40 a]

[Figure 40　b]

[Figure 40 c]

70

[Figure 40 d]

[Figure 40 e]

72

[Figure 40　f]

Many of the night time spikes are narrow because the temperature and relative humidity conditions are only met for a short time during the night. Other spikes are narrow because the virus does not reach 10 km downwind before day time either changes the atmospheric stability conditions or the conditions become unsuitable for FMD virus survival. Virus will not reach the 10 km point downwind if temperature exceeds 27°C, relative humidity is less than 60% or atmospheric stability and wind speed result in excessive plume dispersal. These periods can be seen in the examples shown in Figure 39.

The spikes of high night time virus concentrations are evident in all the charts. The number of such spikes per year varies from the occasional (Alice Springs), through seasonal (Darwin, Cobar, Mildura) to almost every night (Sale, Launceston). Since the night time peaks are much greater than the day time peaks, wind-borne spread is more likely at night. Nonetheless there are sites and times for which daytime conditions are almost continuously suitable for spread (e.g. Mildura − winter, Launceston − all year). A long exposure to a low dose can be just as risky as a short exposure to a high dose.

Table 11 summarises the data in Figure 40 by tabulating the average hourly intake for each mini-bar chart. Even with a relatively large source of virus (100 infected pigs), the risk of long distance wind-borne spread associated with inland sites such as Alice Springs is low. The risk increases for northern sites such as Darwin and inland sites further south such as Canberra and Tamworth. Sites near the eastern and southern coast of Australia provide the highest risk. The table also shows the seasonal effects that occur at sites such as Amberley, Cobar, Mildura.

Table 11   Summary of virus concentrations shown in Figure 40

Average hourly dose inhaled by 100 cows 10km from 100 infected pigs						
Site	Year	Jan-Mar	Apr-Jun	Jul-Sep	Oct-Dec	Full year
Alice Springs	1947	21.3	12.7	58.4	30.1	30.6
Cobar	1980	20.4	170.7	109.4	27.1	81.9
Darwin	1972	50.1	59.6	147.3	89.2	86.6
Belmont	1975	93.7	114.5	108.0	124.5	110.2
Canberra	1975	82.5	111.0	138.0	123.1	113.6
Amberley	1980	140.9	89.1	90.5	141.0	115.3
Forrest	1970	100.3	149.6	155.8	92.7	124.6
Tamworth	1978	123.7	156.1	179.8	93.7	138.3
Mildura	1978	70.0	185.6	214.7	84.9	138.8
Rockhampton	1978	141.8	164.9	150.6	161.1	154.6
Launceston	1963	145.3	180.7	145.1	150.0	155.3
Sale	1975	134.1	135.6	183.6	180.6	158.5

To complete the analysis of the risk by site one could adjust the dose for the average density of livestock (expressed both as an emitter of virus and a recipient of virus) in the same manner as Section 8. This has not been done for several reasons. The more detailed weather data that is needed is not readily available. The correlation in results (discussed below) between the two methods is good and the overall picture of risk should not change significantly. Finally, a map of risk based on average weather conditions does not need to have high precision. The likelihood of wind-borne spread during an outbreak will depend solely on the actual conditions at the time of the outbreak, and not on the average conditions.

It is useful to compare the methods of Sections 7 and 10 for assessing the risk of wind-borne spread. Section 7 uses the proportion of the year that temperature and relative humidity data is suitable for survival of FMD virus in aerosol. Section 10 incorporates wind speed and cloud cover to determine atmospheric stability and hence the area of spread. This requires considerably more weather data.

For the weather data used in Sections 7 and 10, there is a 97% correlation between the proportion of time suitable for virus survival when calculated simply using the 9 am and 3 pm readings and when calculated on an hourly basis. There is a 82% correlation between this proportion and the hourly dose of virus at 10 km. Figure 41 plots the quarterly values.

Figure 41   **Correlation between proportion of time suitable for virus survival in aerosols and hourly dose 10 km downwind**
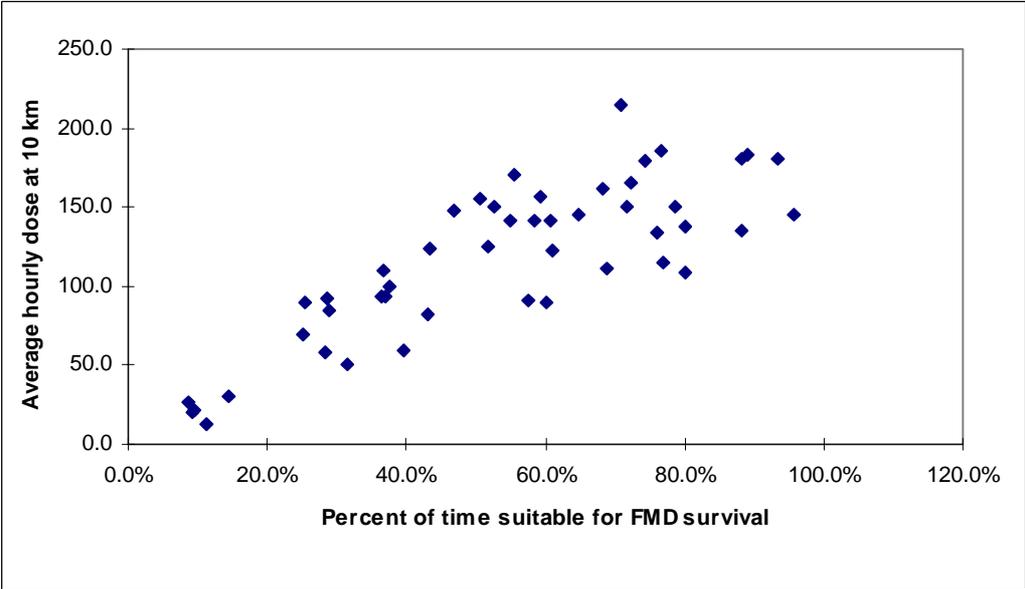
These findings suggest that the proportion of time suitable for survival of FMD virus in aerosols provides a simple and adequate indicator for comparing the risk of long-distance FMD spread between sites associated with weather conditions.

11.   FMD OUTBREAK SCENARIOS

To illustrate the potential for airborne spread of FMD under Australian conditions, several plausible FMD outbreak scenarios involving typical Australian production systems will be described and the extent of wind-borne spread that could occur in each case explored.

In these scenarios, FMD is assumed to be introduced into a property or enterprise. Virus aerosols will be produced until the disease is recognised and appropriate controls imposed. A within-herd disease model was used to determine the amount of virus produced on the infected property. The virus production data, together with actual historical weather data, were used with the Gaussian plume model to determine the extent of wind-borne spread from the infected property to properties downwind that could have taken place. Wind direction is taken into account and the model provides a spatial representation of potential spread.

11.1   FMD Scenarios

The recent report from the EXANDIS-funded study on insuring for consequential losses associated with exotic animal disease outbreaks (Minet Agricultural Insurance Brokers, 1994) describes typical farm sizes and herd structures for a number of livestock enterprises. These have been used as the basis for the scenarios. In each case, disease is assumed to result from the introduction of a single infected animal. FMD subsequently spreads through the herd until, it is recognised and appropriate action (stamping-out) takes place. Seven scenarios were used:

1. Beef cattle: 500 cow breeding/fattening property in the central Queensland coastal area.
2. Beef cattle feedlot: 10,000 head operation in south eastern Queensland.
3. Dairy herd: 130 cow herd in Gippsland, Victoria.
4. Sheep property: 4,000 ewe self-replacing merino ewe flock on the north west slopes of NSW.
5. Pig herd: 100 sow unit producing heavy porkers from WA
6. Backyard pigs: 5 backyard pigs, Tasmania
7. Feral pigs: central western NSW

In each scenario, we are considering virus production from a single property only and have not included any neighbouring properties that may have become infected, e.g. through direct contact, in estimating potential virus spread.

The feral pig scenario is slightly different to the other six scenarios since the population can move about, and this increases the risk of direct contact spread. Nonetheless it is useful to consider the threat that feral pigs pose for long distance wind-borne spread.

11.2   Within-Herd Disease Spread Model

To determine the numbers of stock infected at any point in time after FMD introduction onto the property, a simplified within-herd FMD stochastic simulation model was used. This model is based on a State-Transition herd model for FMD described previously (Hassall and Assoc. 1993). The model enables the daily virus output to be determined in the period up to when disease is recognised and the herd stamped-out. Individual animals are the units in the model, and a daily time frame is used.

The model uses species-specific ranges for duration of latent and infectious periods for FMD. It also includes disease-related mortality. Rates of spread can be chosen to reflect animal to animal spread for the property under consideration. Similarly an appropriate interval for the time from disease introduction until disease recognition needs to be specified to define the period of virus excretion. The parameters used are tabulated below, in Table 12.

Table 12   **Values for key parameters use in FMD outbreak scenarios**

Scenario No.	1	2	3	4	5	6	7
Location	Rockhampton Qld	Toowoomba Qld	Sale Vic	Tamworth NSW	Belmont WA	Launceston Tas	Cobar NSW
Enterprise type	cattle herd	beef feedlot	dairy herd	merino flock	pork production	backyard pigs	feral pigs
Total animals	1684	10000	232	8286	1006	5	5
Management type	semi-extensive	intensive	semi-intensive	extensive	intensive	hobby	feral
Dissemn rate	4	33	9	3	40	NA	NA
Delay (days)	14	10	7	21	7	NA	NA
Mortality (%)	2.5	1	1	8	15	NA	NA
Stock density	0.2	0.3	1	0.5	0.4	0.3	0.1
Weather data	1978	1960	1975	1968	1975	1973	1980

11.2.1 Dissemination rates

Dissemination rate is defined as the expected number of animals coming in contact with each infected animal per day. This will vary with species, time of year and management practices. The rates used have been chosen to reflect animal to animal spread of FMD under the various management systems. Dissemination rates can be estimated from disease incidence data (see Hassall and Assoc. 1993). However it is difficult to predict likely values of the dissemination rate in advance of an outbreak.

11.2.2 Delay — periods of virus emission

The periods from disease introduction until virus emission ceases have been chosen to be realistic. It was assumed that FMD would be identified relatively quickly in intensively managed livestock, but that this would take longer with extensively managed enterprises. While an outbreak of FMD is likely to be recognised relatively quickly in a feedlot, it would take some time to dispose of the stock, hence the period of virus excretion could be quite long. For backyard and feral pigs, the disease was allowed to run its course.

The actual periods of virus emission could be longer or shorter depending on individual circumstances. Feedlots, in particular, might be much longer than the period of 10 days we have used in light of comments by the Australian Lot Feeders Association that it could take weeks to slaughter and dispose of stock on a large feedlot.

11.2.3 Virus production

Aerosol virus output from the infected herd was calculated using excretion rates taken from Donaldson (1983, 1987) and Garland and Donaldson (1990) as listed in Table 4. It is assumed that the whole herd will be slaughtered once the disease is recognised. The results of the within-herd simulations are shown in Figure 42 as epidemic curves, and daily aerosol virus production for each outbreak scenario. Day one is the first day of virus emission.

Figure 42   **Epidemic curves and daily aerosol virus production for outbreak scenarios**

Scenario 1: Beef breeding/fattening

Scenario 2: Beef feedlot

Scenario 3: Dairy herd
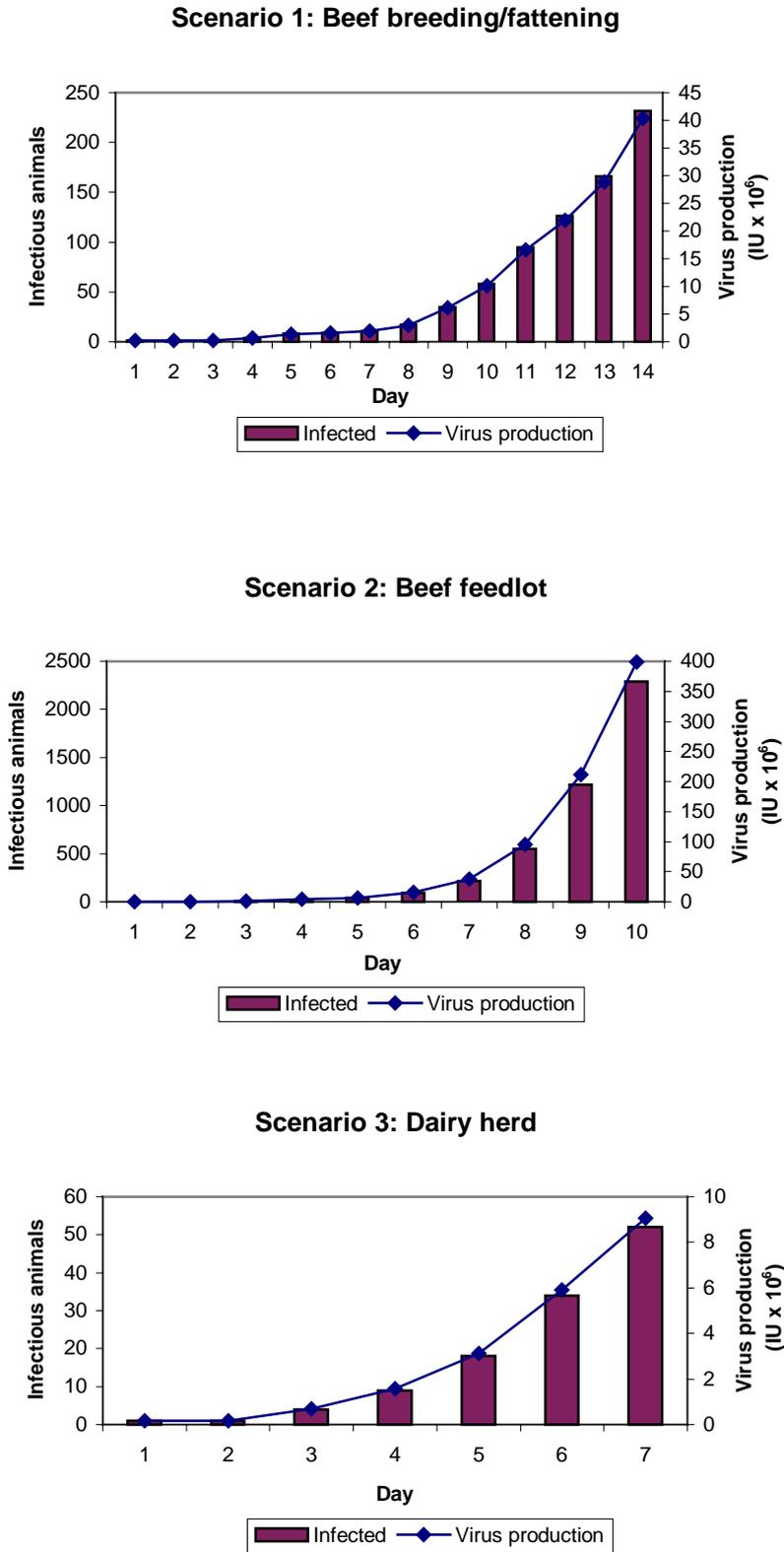
Scenario 4: Merino flock
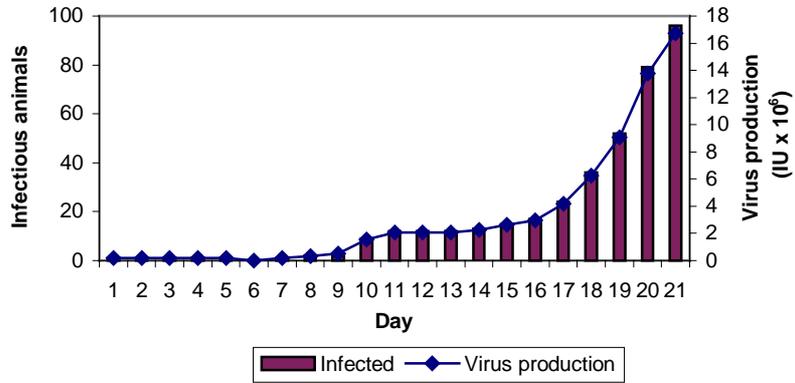
Scenario 5: Intensive piggery
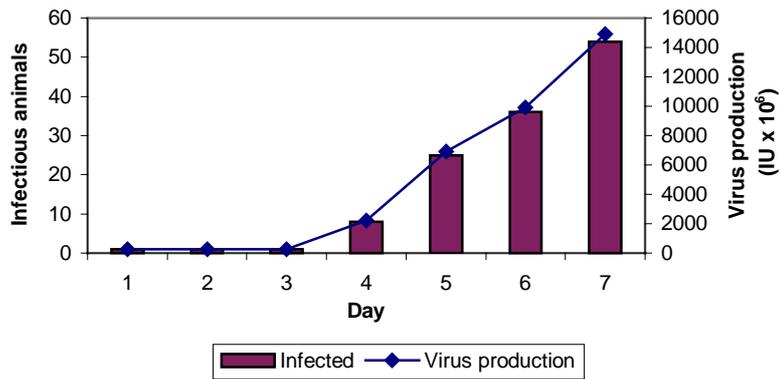
Scenario 6: Feral pig group
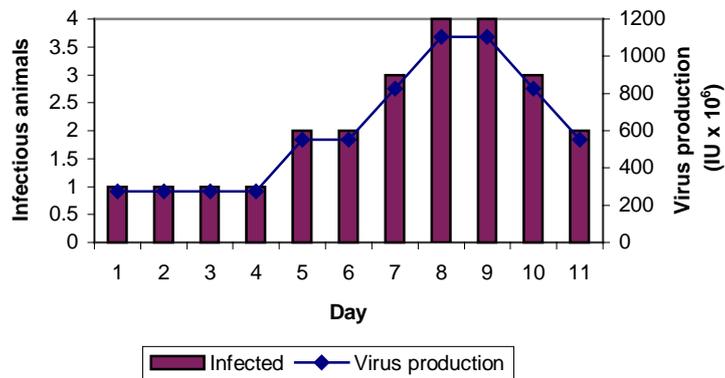
11.2.4 Mortality rates

Mortality from FMD is generally low, although in some circumstances it can be high. According to Geering and Forman (1987) the mortality rate in adult animals is usually less than 5%, although in young animals it can be up to 50%. In piglets in large intensive units in Europe, mortality rates up to 90% have been seen (Donaldson 1993). Brightling (1994) also reports potentially high mortalities in young lambs in Turkey (up to 90%, but rates of 20-40% are more common).

11.2.5 Enterprise size

Sizes typical of the type of enterprise have been used for the size of the initially infected property. We have used a size of 5 for our feral pig group. Saunders and Kay (1993) reports feral pig group sizes to range from 1 to 22 with a mean and standard deviation of 4.2 ± 3.9 .

11.2.6 Stock density

The density of stock at risk was calculated from livestock census data of the shire or local government area containing the outbreak and is expressed in cattle equivalents per hectare. In the case of feral pigs at risk in Scenario 7, published density estimates were used (Pech and Hone, 1990). Representative densities for feral pigs are <5/sq km for dry sclerophyll forests, 2−20/sq km for wet sclerophyll forests and 20−80/sq km for marshes.

11.2.7 Feral pigs

A slightly different approach was required for the feral pigs. Estimating rates of spread among feral populations is more difficult than for 'managed' domestic herds. As most feral species, including feral pigs form social groups, there are two components to be considered — within group spread and between group spread. For a highly contagious disease like FMD, within group spread will be rapid, but between group spread will depend on inter-group contacts and may be quite low depending on various factors such as seasonal conditions, time of year, etc.

We are modeling the start of a possible epidemic, before the disease is rife, and assuming that FMD is introduced into a single group of pigs by a means such as the ingestion of virus contaminated material. The initially infected pig quickly infects the other members of the group. The situation we are considering here is analogous to infection of a small group of 'backyard' pigs and the same approach was used for Scenario 6.

The illustrations using rosette diagrams are not appropriate if we have a number of infected pigs wandering over wide areas. The source of infection is no longer a point source but can be treated as a line source along the limit of the pigs. The comments of Section 9.5.2 apply and the virus concentration will be almost uniform parallel to this line.

11.3   Plume Model

The Gaussian plume model was used to determine the extent of the spread of FMD virus produced by the simulated outbreaks.The Bureau of Meteorology's three-hourly weather data was used to determine both the suitability of conditions for survival of airborne FMD virus and the parameters for the Gaussian plume model as described in Section 10. Instead of the constant artificial virus source (100 pigs) of Section 10, the model uses the varying hourly level of virus production from each enterprise, based on the within-herd disease spread model.

The model uses as realistic values as possible for its various parameters. However, there are two parameters (percent virus becoming airborne and deposition rate) for which there is little information. A conservative approach was adopted and the values used in the model were chosen to overestimate, rather than underestimate, the concentration of FMD virus downwind.

While there is information on virus production from individual animals, there is little information on how much of this virus will remain airborne outside of the immediate vicinty of the animal and so contribute to the plume. The model assumes that 100% of the virus exhaled becomes airborne and this is certain to be an overestimate.

The model assumes a virus deposition velocity of 0.01m/s. As discussed in Section 9.3.4, deposition velocity depends on size of particles and rougness of terrain. In many practical situations the rate of deposition will be much greater because of the type of vegetation and terrain.

The model program looks at the concentration of virus at each point of a 250 m by 250 m grid within 20 km around the source. It tracks the position of the emitted virus particles as they travel downwind until the weather conditions become unfavourable to FMD aerosol virus survival. The amount of virus to which animals in each grid cell would be exposed is accumulated over the period of aerosol virus excretion.

The sites of the outbreaks were selected from those used in Sections 7 and 10 as being appropriate for the enterprise types. Weather sites were chosen as the nearest site to the point of interest that had a full range of weather data. The year was chosen with no consideration given to the actual values of the data The only case where the animal data and the weather data did not coincide was for Toowomba (Scenario 2) — Amberley has much more detailed weather records. To allow for different effects associated with outbreaks occurring at different times of the year, outbreaks were simulated at the start of each month over a 12 month period.

11.4   Results

The results have been depicted in *rosette* diagrams that show, graphically, the amount, direction and extent of FMD virus exposure of areas surrounding the outbreak The circles are drawn at 5 km intervals as a scale. The relative areas covered by the rosettes give a visual way of comparing the relative risks both near to the the outbreak, and at greater distances. The *spikes* in the diagrams result partly from the simulations being hourly coupled with the accuracy of the wind direction data (22.5°) and partly because night time conditions are more suitable for spread than daytime conditions.

Rosette diagrams (Figures 43–48) show the amount and extent of virus spread from the infected properties. These diagrams plot the amount of virus (IU) that would be inhaled per hectare by susceptible stock around the outbreak site. Figure 49 and 50 consider the risk of wind-borne spread of FMD from feral pigs, both to livestock in the surrounding region and to other nearby feral pigs.

As mentioned in Section 11.3, the model is likely to overestimate the concentration of virus downwind. The model's main use is in comparing the relative risks between different situations rather than in giving an absolute risk. The relationship between dose inhaled and probability of infection has been discussed in Sections 8.5 and 9.4. The ratio of the virus doses inhaled at each site provides a convenient estimate of the relative risk between two sites (Table 13).

Under Australian conditions, extensively and semi-extensively managed cattle and sheep (Figures 43, 45 and 47) would appear to pose little threat of long distance wind-borne spread of FMD, even where the disease is not recognised for several weeks. Under suitable conditions, it would appear that feedlots could pose problems. Figure 44 suggests that for most of the year a small feedlot in south eastern Queensland would be capable of spreading disease more than 5 km.

The importance of pigs in wind-borne spread is readily apparent. The infected piggery (Figure 47) poses the greatest threat of wind-borne spread, by far. For all months of the year, virus could spread well beyond 10 km. Even small groups of pigs (Figures 48 and 49) in Scenarios 6 and 7 pose a significant threat of spreading disease to surrounding livestock, mainly because the disease is likely to be unrecognised and run its full course.

Some sites, such as Cobar (Figure 49), show significant seasonal variation, with potential wind-borne spread much higher in the winter months. The effect of weather conditions on virus survival and dispersal (see Figure 39) explain this variation.

The feral pig simulations also show the importance of species at risk and stock density on the risk of wind-borne spread. Figure 49 shows the potential for wind-borne spread to domestic livestock (average stock density is 0.1 cattle equivalents per ha). This is considerably greater than potential wind-borne spread to other feral pigs in the vicinity (Figure 50). On an individual animal basis, pigs have one eighth the susceptibility of cattle to infection via the airborne route, largely because of their lower air intake. A density of 10 pigs per km^2 (0.1 pigs per ha) is equivalent to 0.008 cattle per ha and at these densities potential for wind-borne spread is much reduced.

Throughout this report, we have used 10 km from the source of infection as a key reference point because this represents the recommended extent of the Restricted Area that would be imposed around an outbreak site. To compare the relative risk of wind-borne spread of FMD beyond the 10 km, we calculated the total amount of virus that would be inhaled by all stock in a 10–20 km annular ring around the outbreak for each scenario. It is important to appreciate that these amounts of virus depend on stock density of the surrounding areas, the periods of virus emission as well as the weather conditions. The results are shown in Table 13, following the rosette diagrams. The top half of the table reflects the comments already made about the different scenarios. The relative risk of wind-borne spread from infected pigs is 100 to 200 times that from infected cattle.

Table 13   **Virus inhaled (IU) during the course of the outbreaks by all animals in a 10–20 km annulus around each outbreak site.**

Scenario	1	2	3	4	5	6	7
Enterprise type	Cattle	Feedlot	Dairy	Sheep	Piggery	Backyard	Feral pigs
Total virus emitted (MIU = million IU)	133.0	771.8	20.7	67.6	34769.3	6898.7	6898.7
Weather site used	R'ton	Amberley	Sale	Tamworth	Belmont	Launc'n	Cobar
(a) actual stock density							
Density (cattle equiv / ha)	0.2	0.3	1.0	0.5	0.4	0.3	0.1
Virus inhaled (IU) during							
Jan	8.8	45.1	6.0	20.2	5163.1	1048.1	309.9
Feb	3.3	46.7	6.0	6.2	1247.0	1137.5	16.7
Mar	18.1	39.0	3.6	9.9	339.2	1440.1	185.5
Apr	11.9	54.8	9.5	12.8	1498.3	1532.0	0.0
May	11.3	6.9	3.8	20.4	2553.8	2044.1	496.8
Jun	16.8	9.0	8.9	13.1	3008.4	1161.4	630.9
Jul	10.6	35.5	6.2	20.0	1797.2	1082.7	655.0
Aug	13.3	5.7	11.8	22.7	3008.3	1453.5	164.1
Sep	14.5	17.0	12.7	13.0	1677.3	1763.9	14.5
Oct	13.9	63.4	10.8	11.2	5579.1	1205.0	42.2
Nov	12.5	48.3	8.9	1.5	2828.1	1427.1	42.0
Dec	8.2	53.8	13.3	5.9	4442.8	651.4	43.4
Average	11.9	35.4	8.5	13.1	2761.9	1328.9	216.8
Relative risk	1.0	3.0	0.7	1.1	232.1	111.7	18.2
(b) standardised density							
Density	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Average inhaled (IU)	59.7	118.1	8.5	26.2	6904.7	4429.7	2167.5
Ratio inhaled (IU) to emitted (MIU)	0.45	0.15	0.41	0.39	0.20	0.64	0.31

The bottom half of Table 13 use the data to demonstrate the effect that weather conditions at different sites have on possible spread by considering a standardised density of animals at risk. The table shows the average number of virus particles inhaled if the surrounding density were 1 cow per hectare. With the same source strength and same (standardised) density, the amount of virus inhaled at Launceston (Scenario 6) is double that at Cobar (Scenario 7). Similarly the virus inhaled at Launceston (Scenario 6) is 64% that inhaled at Belmont (Scenario 7) even though the virus emission at Launceston was only 20% of that at Belmont. In both cases, the difference is due entirely to the weather conditions.

Another way to compare the effect of weather at different sites is to look at the ratio of virus particles inhaled (by a standard stock density) to virus particles emitted. This ratio provides a measure of the potential to spread that is independent of the strength of the source, its type, or stock density in the surrounding countryside. For the sites used in these scenarios, there is a considerable range, with Launceston having the most suitable conditions for wind-borne spread and Amberley the least.

While it may be of interest to compare sites, to have an idea of the measure of risk associated with weather, it is important to reiterate that the threat posed by wind-borne spread would depend on the conditions at the time of an outbreak, and not on historical long-term averages.

References

Anon (1990) Department of the Arts, Sport, The Environment, Tourism and Territories - Bureau of Meteorology - Directive for the Provision of Meteorological support in Animal Health Emergencies. 18 pp.

Barlow DF (1972) The aerosol stability of a strain of foot-and-mouth disease virus and the effects on stability of precipitation with ammonium sulphate, methanol or polyethylene glycol. *J Gen Virol* 15: 17–24.

Bartlett JT (1973) Meteorological factors influencing the long-range transmission of micro-organisms. In *Proceedings VIth International Symposium on Aerobiology*, Enschede, The Netherlands. Pp. 385–391.

Blackall RM and Gloster J (1981) Forecasting the airborne spread of foot and mouth disease. *Weather* 36: 162–167.

Briggs GA (1973) Diffusion Estimation for Small Emissions, ATDL Contribution File No 79, Atmospheric Turbulence and Diffusion Laboratory

Brightling T (1994) Report on a visit to Turkey to study foot and mouth disease. Unpublished Report to the Meat Research Corporation. 17 pp.

Brooksby JB (1969) Laboratory investigations on the 1967/8 outbreak of foot-and-mouth disease in Great Britain. *Veterinary Annual* 1969, pp. 1–11.

Brown F, Cartwright B and Stewart DL (1963) The effect of various inactivating agents on the viral and ribonucleic acid infectivities of Foot-and-Mouth disease virus and its attachment to susceptible cells. *J Gen Microbiol.* 31: 179–186.

Burrows R (1968) Excretion of foot-and-mouth disease virus prior to the development of lesions. *Vet Rec* 82: 387–388.

Committee of Inquiry (1969) Report of the Committee of Inquiry on Foot-and-mouth Disease 1968. Part One. Her Majesty's Stationery Office, London. 135 pp.

Daggupaty SM (1988) Response to accidental release of toxic chemicals into the atmosphere using - AQPAC. In *Natural and Man-Made Hazards*, edited by MI El-Sabh and TS Murty. D Reidel Publishing Company. Pp. 599–608.

Daggupaty SM and Sellers RF (1990) Airborne spread of foot-and-mouth disease in Saskatchewan, Canada, 1951–1952. *Can J Vet Res* 54: 465–468.

Donaldson AI (1972) Influence of relative humidity on the aerosol stability of different strains of foot-and-mouth disease virus suspended in saliva, *J Gen Virol* 15: 25–33.

Donaldson AI (1983) Quantitative data on airborne foot-and-mouth disease virus; its production, carriage and deposition. *Phil Trans R Soc Lond B* 302: 529–534.

Donaldson AI (1986) Aerobiology of foot-and-mouth disease (FMD): an outline and recent advances. *Rev Sci Tech Off Int Epiz* 5: 315–321.

Donaldson AI (1987) Foot-and-mouth disease: the principal features. *Irish Vet J* 41: 325–327.

Donaldson AI (1988) Development and use of models for forecasting the spread of foot-and-mouth disease. *J Royal Agric Soc England* 149: 184–194.

Donaldson AI (1993) Epidemiology of foot-and-mouth diesease: the current situation and new perspectives. Unpublished report 23pp.

Donaldson AI and Ferris NP (1975) The survival of foot-and-mouth disease virus in open air conditions. *J Hyg Camb* 74: 409–416.

Donaldson AI, Ferris NP and Gloster J (1982a) Air sampling of pigs infected with foot-and-mouth disease virus: comparison of Litton and cyclone samples. *Res Vet Sci* 33: 384–385.

Donaldson AI, Gloster J and Harvey LDJ (1982b) Use of prediction models to forecast and analyse airborne spread during the foot-and-mouth disease outbreaks in Britanny, Jersey and the Isle of Wight in 1981. *Vet Rec* 110: 53–57.

Donaldson AI, Gibson CF, Oliver R, Hamblin C and Kitching RP (1987a) Infection of cattle by airborne foot-and-mouth disease virus: minimal doses with O1 and SAT2 strains. *Res Vet Sci* 43: 339–346.

Donaldson AI, Herniman KAJ, Parker J and Sellers RF (1970) Further investigations on the airborne excretion of foot-and-mouth disease virus. *J Hyg Camb* 69: 557–564.

Donaldson AI, Lee M and Gibson CF (1987b) Improvement of mathematical models for predicting airborne spread of foot-and-mouth disease. In *Proceedings of the 3rd International Conference on Aerobiology*, August 6–9, 1986, Basel, Switzerland, Birkhauser Verlag, Basel. Pp. 351–355.

Donaldson AI, Lee M and Shimshony A (1988) A possible airborne transmission of foot and mouth disease virus from Jordan to Israel — a simulated computer analysis. *Isr J Vet Med* 44: 92–96.

Doury A (1982) Operational calculation aids for atmospheric dispersion. *Sci Total Environ* 25: 3–17.

DPIE (1990) Australian Veterinary Emergency Plan: National Disease Strategy, Foot-and-Mouth Disease. Vol 2, p1.14.

Fogedby EG, Malmquist WA, Osteen OL and Johnson ML (1960) Air-borne transmission of foot and mouth disease virus. *Nordisk Veterinaermedicin* 12: 490–498.

Gainaru MD, Thomson GR, Bengis RG Esterhuysen JJ, Bruce W and Pini A (1986) Foot-and-mouth disease and the African buffalo (Synerus caffer). II. Virus excretion and transmission during acute infection. *Onderstepoort J Vet Res* 53: 75–85.

Garland AJM and Donaldson AI (1990) Foot-and-mouth diesease. Surveillance 17(4): 6–8.

Geering WA and Forman AJ (1987) Foot-and-mouth disease. In *Animal Health in Australia Vol. 9 Exotic Diseases*. Australian Government Publishing Service, Canberra, Pp. 111–117.

Gibson CF and Donaldson AI (1986) Exposure of sheep to natural aerosols of foot-and-mouth disease virus. *Res Vet Sci* 41: 45–49.

Gloster J (1983) Forecasting the airborne spread of foot-and-mouth disease and Newcastle disease. *Phil Trans R Soc Lond B* 302: 535–541.

Gloster J, Blackall RM, Sellers RF and Donaldson AI (1981) Forecasting the airborne spread of foot-and-mouth disease. *Vet Rec* 108: 370–374.

Gloster J, Sellers RF and Donaldson AI (1982) Long distance transport of foot-and-mouth disease over the sea. *Vet Rec* 110: 47–52.

Grace W and Schahinger R (1994) An operational air quality model for Bolivar Sewerage Treatment Works. *Bulletin of the Australian Meteorological and Oceanographic Society* 7(4): 66–71.

Gregory PH (1961) The Microbiology of the Atmosphere. Leonard Hill, London, pp 45–57.

Griffiths GL (1994) Report of Study Trip. Unpublished report on an EXANDIS-funded study tour to southern Africa. 44 pp.

Hanna SR, Briggs GA and Hosker Jr RP (1982) Handbook on Atmospheric Diffusion. UD Department of Energy, Springfield, 135 pp.

Hardy AC and Milne PS (1938) Aerial drift of insects. *Nature* 141: 602–603.

Hassall and Associates (1993) Impacts of exotic animal diseases on regional economies within Australia. Unpublished report to EXANDIS, pp. 15–16.

Henderson RJ (1969). The outbreak of foot-and-mouth disease in Worcestershire an epidemiological study with special reference to spread of the disease by wind carriage of the virus. *J Hyg Camb* 67: 21–33.

Hugh-Jones ME (1972) Epidemiological studies on the 1967–68 foot and mouth epidemic: attack rates and cattle density. *Res Vet Sci* 13: 411–417.

Hugh-Jones ME and Wright PB (1970) Studies on the 1967–8 foot-and-mouth disease epidemic the relation of weather to the spread of disease. *J Hyg Camb* 68: 253–271.

Hurst GW (1968) Foot and mouth disease, the possibility of continental sources of the virus in England in epidemics of October 1967 and several other years. *Vet Rec* 81: 610–617.

Hyslop N St G (1965a) Secretion of foot and mouth disease virus and antibody in the saliva of infected and immunised cattle. J *Comp Path* 75: 111–117.

Hyslop N St G (1965b) Airborne infection with the virus of foot and mouth disease. *J Comp Path* 75: 119–126.

Maragon S, Facchin E, Moutou F, Massirio I, Vincenzi G and Davies G (1994) The 1993 Italian foot-and-mouth disease epidemic: epidemiological features of the four outbreaks identified in Verona province (Veneto region). *Vet Rec* 135: 53–57.

McVicar (1977) The pathobiology of foot-and-mouth disease in cattle. A review. *Boletin del Cento Panamericano de Fiebre Aftosa* 26: 9–14.

Minet Agricultural Insurance Brokers Pty Ltd (1994) A feasibility study of a commercial insurance scheme covering the consequential loss associated with exotic animal disease outbreaks in Australia. Unpublished report to EXANDIS. 139 pp.

Moutou F and Durand B (1994) Modelling the spread of foot-and-mouth disease virus. *Vet Res* 25: 279–285.

Morgan IR (1993) Spread of foot-and-mouth disease. A review prepared for the Australian Meat Research Corporation. 115 pp.

National Farmers Federation (1993) *Australian Agriculture. The Complete Reference on Rural Industry*. 4th Edition. Morecope Publishing, Camberwell. 437 pp.

OIE (1992) Foot and mouth dosease (FMD). In Office International des Epizooties International Animal Health Code – Mammals, Birds and Bees, 6th edn, OIE, Paris. Pp 57–69.

Pasquill F (1961) The estimation of the dispersal of windborne material. *Meteorological Magazine (London)* 90: 33.

Pech RP and Hone J (1988) A model of the dynamics and control of an outbreak of foot and mouth disease in feral pigs in Australia. *J Applied Ecol* 25: 63–77.

Rumney RP (1986) Meteorological influences on the spread of foot-and-mouth disease. *J Appl Bacteriol Symposium Supplement:* 105S–114S

Sanson RL (1994) The epidemiology of foot-and-mouth disease. Implications for New Zealand. *New Zealand Vet J* 42: 41–53.

Sanson RL, Liberona H and Morris RS (1991a) The use of a geographical information system in the management of a foot-and-mouth disease epidemic. *Prev Vet Med* 11: 309–313.

Sanson RL, Pfeiffer DU and Morris RS (1991b) Geographic information systems: their application in animal disease control. Rev Sci Tech Off Int Epiz 10: 179–195.

Saunders G and Kay B (1993) The ecology and control of feral pigs: implications for exotic disease preparidedness. Unpublished report to WEDPP. 99pp.

Sellers RF (1971) Quantitative aspects of the spread of foot-and-mouth disease. *Vet Bull* 41: 431–439.

Sellers RF, Barlow DF, Donaldson AI, Herniman KAJ and Parker J (1973) Foot-and-mouth disease, a case study of airborne disease. In *Proceedings of the VIth International Symposium on Aerobiology*, Enschede, the Netherlands, pp. 405–411.

Sellers RF and Daggupaty SM (1990) The epidemic of foot-and-mouth disease in Saskatchewan, Canada, 1951–1952. *Can J Vet Res* 54:457–464.

Sellers RF and Forman AJ (1973) The Hampshire epidemic of foot-and-mouth disease, 1967. *J Hyg Camb* 71:15–34.

Sellers RF, Garland AJM, Donaldson AI and Gloster J (1981). The 1975 foot-and-mouth disease epidemic in Malta. IV: Analysis of the epidemic. *Br Vet J* 137: 608–620.

Sellers RF and Gloster J (1980) The Northumberland epidemic of foot-and-mouth disease, 1966. *J Hyg Camb* 85: 129–140.

Sellers RF and Parker J (1969) Airborne excretion of foot-and-mouth disease virus. *J Hyg Camb* 67: 671–677.

Smith FB (1983) Meteorological factors influencing the dispersion of airborne diseases. *Philosophical Transactions of the Royal Society of London* B302: 439–450.

Stougaard E (1982) Information on the eradication of FMD on the islands of Funen and Zeeland in Denmark (interim report). In *Proceedings of the XVIth Conference of the OIE Foot-and-Mouth Disease Commission*, Paris, 14–17 September 1982, pp. 535–545.

Terpstra C (1972) Pathogenesis of foot-and-mouth disease in experimentally infected pigs. *Bull Off Int Epiz* 77: 859–874.

Turner DB (1967) Workbook of Atmospheric Dispersion Estimates. Public Health Service publication 999-AP-26, Robert A Taft Sanitary Engineering Centre, Cincinatta, Ohio.

Wilson G, Dexter N, O'Brien P and Bomford M (1992) Feral pig (Sus scrofa). *In Pest Animals in Australia. A Survey of Introduced Wild Animals.* Bureau of Rural Resources and Kangaroo Press, Kenthurst, pp. 42–46.

Wright PB (1969) Effects of wind and precipitation on the spread of foot-and-mouth disease. *Weather* 24: 204–213.