An integrated modelling approach to assess the risk of wind-borne spread of foot-and-mouth disease virus from infected premises

M.G. Garner^{a,*}, G.D. Hess^b and X. Yang^b

^a Epidemiology and Modelling Section, Office of the Chief Veterinary Officer, Department of Agriculture, Fisheries and Forestry, PO Box 858 Canberra, ACT 2601, Australia

Email: graeme.garner@daff.gov.au

^b Bureau of Meteorology Research Centre, GPO Box 1289K Melbourne, VIC 3001, Australia

Email: {d.hess, x.yang}@bom.gov.au

Foot-and-mouth disease (FMD) is a highly contagious disease of livestock that has serious consequences on livestock production and trade. In Australia, preparedness and planning includes the development of decision-support tools that would assist priority setting and resource management in the event of an incursion. In this paper we describe an integrated modelling approach using geographic information system (GIS) technology to assess the risk of wind-borne spread of FMD virus. The approach involves linking an intra-farm virus production model, a wind transport and dispersal model, and an exposure-risk model to identify and rank farms at risk of wind-borne infection of FMD. This will assist authorities by enabling resources for activities like surveillance and vaccination to be allocated on the basis of risk.

Keywords: foot-and-mouth disease (FMD), wind spread, virus, aerosol, disease, model, risk, exposure assessment, decision support

1. Introduction

Foot-and-mouth disease (FMD) is a highly contagious disease of livestock that would have serious consequences were it to occur in a country like Australia [1]. This is not only due to the productivity losses associated with the disease itself, but also due to the restrictions to trade that result [2]. A recent study by the Australian Productivity Commission concluded that an FMD outbreak would result in immediate closure of many of Australia's major export markets. The cumulative loss in export and domestic market revenues would be around \$5.7 billion for a single point outbreak, rising to around \$13 billion for an outbreak lasting 12 months [3].

Recent outbreaks of FMD in countries like the United Kingdom [4] and the Netherlands in 2001 [5] have highlighted the resource requirements needed to manage an outbreak of a disease like FMD and have emphasised the importance of having well-thought-out contingency plans to ensure effective and efficient allocation of these resources. In recognition of the significant consequences, Australia invests considerable resources in FMD prevention and planning. Part of this preparedness includes the development of decision-support tools that would assist priority setting and resource management in the event of an incursion.

In this report we describe an integrated modelling approach using geographic information system (GIS) technology to assess the risk of wind-borne spread of FMD virus. The approach builds on an earlier work [6] and is designed to enhance outbreak management by identifying and prioritising farms at risk of wind-borne infection of FMD. This will assist authorities by enabling resources for activities like surveillance and vaccination to be directed and allocated on the basis of risk.

2. Description of the approach

FMD may be spread by a variety of routes, with movement of infected animals considered the most important [7, 8]. Under the right conditions FMD virus carried by wind may spread infection over long distances [6]. Although this situation may be an uncommon event requiring the right combination of virus strain, environment and suitable weather conditions [9–11], it is of particular concern as it cannot be controlled by the normal measures that are put in place during an outbreak. Recognising this, several countries have developed models that simulate wind-borne spread of FMD virus over short and long distances (e.g., [6, 9, 11–14]). However, managing this threat requires more than just predicting where virus may have spread to.

From an operational perspective, to assess the risk of wind-borne spread from a recently identified infected premise (IP), three issues need to be considered:

- The amount of airborne virus produced on the IP over the period from when it was infected to when it was reported.
- The area(s) that may have been exposed to airborne virus direction, distance and virus concentrations need to be identified.

^{*} Corresponding author.

• The probabilities that farms exposed to airborne virus may have become infected need to be assessed.

The first requires that the pattern of spread on the IP be recreated and the amount of airborne virus produced over the period be estimated. The second requires an assessment of the weather conditions that would affect the survival, dilution and dispersal of the virus in the air, to identify areas and farms that have been potentially exposed to infection. The third component requires an evaluation of the probabilities that farms could have become infected based on the amount of virus to which they have been exposed. We have developed an integrated modelling approach that addresses these components. It includes an intra-farm virus production model, a wind transport and dispersal model, and an exposure-risk model. The application runs within a GIS framework (MapInfo Corporation, Troy, NY, USA) that permits ready visualisation of virus plumes and analysis of premises at risk. In this paper we will briefly review the epidemiology of FMD and describe the methods used in the integrated model.

2.1. Natural history of FMD infection

Animals exposed to FMD incubate the virus for a period before they show clinical signs of disease, although the disease may not always be apparent clinically, especially in sheep [7, 8]. Animals excrete virus and are capable of spreading infection at or shortly before the appearance of clinical signs [8]. Animals infected with FMD virus may die, although mortality rates are usually low, except in very young animals [7], or go on to recover and are then immune to reinfection. Over time, the immunity wanes and animals can become susceptible again. A proportion of recovered cattle and sheep, but not pigs, may continue to excrete virus after they have recovered, referred to as 'carrier' animals [15]. Although carriers have been implicated in field outbreaks, the risk that these animals pose for spreading infection is still unclear [8, 15, 16].

2.2. Intra-farm virus production model

To assess the risk posed by wind-borne spread from an IP, one needs to estimate daily virus production. During a real epidemic, the actual starting point for this exercise is likely to be when a high-risk premise (one with large numbers and high concentrations of animals, like a piggery or feedlot) is found to be infected. As soon as the IP has been found, it is assumed that authorities will undertake appropriate action to eliminate the risk. Under Australian contingency plans for FMD [17], this involves quarantine of IPs and compulsory slaughter of all infected and exposed susceptible animals. Thus, it is the period from infection to removal that is of concern. By estimating how and when the IP became infected, it is possible to use an epidemiological model to recreate the spread of disease and estimate the number of animals infected and the

amount of virus excreted on each day from when the infection was introduced until it was found.

In modelling terms, the period from initial infection to when an animal begins excreting virus is the *latent period*. The period during which infected animals excrete virus and are thus capable of spreading the infection is called the *infectious period*. The period over which recovered animals are immune to reinfection is the *immune period*.

Intra-herd spread of FMD is modelled using a deterministic state-transition simulation model developed from a Markov chain. It is based on the approach described in an earlier model of rinderpest [18]. Briefly, in a state-transition model, animals in the study population (the herd or flock) can be considered to be in one of several mutually exclusive 'states'. There are five basic disease-related states:

- 1. Susceptible able to be infected
- 2. Latent infected but not yet infectious to other animals
- 3. Infectious infected with the disease and capable of spreading infection to other animals
- 4. Immune after recovery from the disease
- 5. Dead as a result of the disease

During any time period, depending on various factors, an animal may remain in that state or move to another state (a 'transition'). Figure 1 shows, diagrammatically, the states and transitions in the basic model. Further states can be added to take into account additional factors like vaccination where this may be used.

The probability of a susceptible animal becoming infected is a function of the number of infected animals in the herd and the effective contact rate (ECR). ECR is defined as the expected number of animals with which one virus-excreting animal will make sufficiently close contact that disease transmission could occur within a given period [18]. This parameter is difficult to estimate directly, but



Figure 1. States (boxes) and transitions (arrows) in intra-herd FMD model.

can be derived from incidence data) and values can be adjusted so that the results conform with a known disease situation [18].

Default values used for key model parameters are given in table 1. The model takes into account the decrease in the latent period as the number of infected animals in the herd, and hence total virus to which susceptible animals are exposed to, increases. Clinical signs of the disease will become apparent 1–2 days after virus excretion has commenced and may persist after the virus ceases. The model also allows for daily virus production from infected animals to vary with time during the virus excretion period, consistent with experimental observations [9].

To recreate an on-farm outbreak, it is necessary to provide information about how and when the infection was introduced. The user specifies:

- Species (cattle, sheep or pigs)
- The size of the flock or herd,
- The estimated time of infection (how many days ago)
- The number on initially infected animals and whether these were incubating or infectious at the time
- The number of animals or proportion of the herd that is affected at the time of inspection
- Effective contact rate between animals on the farm

Where an IP has more than one species on it, virus production is modelled separately for each species group. Virus production is measured in tissue culture infectious dose₅₀ units (TCID50). One TCID₅₀ is the amount of virus that will infect 50% of exposed tissue cultures and is assumed to be directly proportional to the number of infectious virus particles present in a sample [19]. The time and source of infection would be estimated from age of lesions [20, 21] and farm history. If the source is believed to be introduction of infected animals, they could have been incubating or infectious at the time of introduction. Depending on when they were infected, it is necessary to estimate how many days these source animals would have left in the latent or infectious phase of the disease. If the source is believed to be a contaminated product or equipment, then the initially exposed animal(s) begin as incubating rather than infectious, and the latent period is

randomly set. The number of animals affected at the time of inspection is based on animals showing clinical evidence of disease. The model generates a daily breakdown of the status of animals in the herd and total aerosol virus production, with ECR being adjusted until the simulation reproduces the observed number of clinical cases at the time of inspection. The model provides an average or expected daily virus production regime that fits the input parameters. This output, together with the timing of the infection and point location of the IP (latitude and longitude), is used as the input for the wind dispersal modelling.

2.3. Wind transport and dispersion model

The transport and dispersion of FMD virus is modelled by the Australian Bureau of Meteorology (BOM), using a model called HYSPLIT [22, 23]. This is a general model designed to use gridded wind data from numerical weather prediction models or three-dimensional numerical analyses as input. It can be run in either a puff mode or particle mode, or in a combination of the two. In the puff mode, a single puff is released with either a Gaussian or top-hat probability distribution, and when the puff has grown to exceed the size of the meteorological grid, it is split into smaller puffs. In the particle mode, a fixed number of particles is released.

For FMD applications, HYSPLIT is used in the particle mode. The meteorological fields are obtained from the Australian Bureau of Meteorology's operational model, the Limited Area Prediction System (LAPS) [24], whose domain covers all of Australia (from latitude -55°S to 4.875°N and longitude 95.0°E to 169.875°E); the horizontal resolution is 0.125° (approximately 12.5 km) and there are 29 vertical levels with 11 levels within the lowest 1,500 m. The meteorological fields at each horizontal grid point are interpolated at 3-hourly intervals to a terrainfollowing coordinate system from the pressure-sigma surfaces of LAPS. The vertical motion field is included in the meteorological data. The trajectories of the particles are computed using a modified Euler technique. The integration time step is variable (between 1 min and 1 h) during the simulation. The time step is chosen based on the vertical grid spacing, the vertical velocity variance and the vertical Lagrangian time scale (assumed to be 100 s), and

Table 1 Default parameters used in intra-farm FMD model.

	Cattle	Sheep	Pigs	Source
Latent period (days)	8	7	5	8, 34
Infectious period (days)	7	7	7	7, 8, 16, 21
Period over which clinical signs are apparent (days)	11	6	10	20, 21
Mortality (%)	5%	5%	15%	7, 8
Maximum daily airborne virus production (TCID ₅₀)	10 ^{4.3}	10 ^{4.3}	$10^{6.1}$	9, 10, 34
Period of airborne virus excretion (days)	5	4	6	9, 10, 21, 34

is constrained so that the advection distance per time-step is less than the grid spacing.

In the present simulations (which used three processors), 8,640 Lagrangian particles per day were released at 1m height. These particles were transported with the mean wind plus a random component of motion to account for atmospheric turbulence. Thus, the cluster of particles expands in time and space. The stratification of the atmosphere is calculated from the meteorological data and this information allows the vertical and horizontal mixing coefficients, dependent on thermal stability, to be calculated [22]. The turbulent velocity variance profiles are specified through similarity theory relationships [25, 26].

Particle dispersion is calculated following the approach of Fay et al. [27]. The turbulent velocity components are dependent on the auto-correlation coefficient, which is a function of the time step, the horizontal and vertical Lagrangian time scales, and a computer-generated random component. Particle concentrations are calculated as the sum of the virus mass within a grid cell. The cell is defined at the centre of the node and extends halfway to the adjacent nodes. Full reflection is assumed for particles that intersect the ground or the model top (assumed to be 10,000 m for these simulations).

The concentration grid spacing used for the simulations was 0.01° (approximately 1 km) and the grid span was 1.5° (approximately 150 km). The 24-h concentrations are computed at 1-m height. A dry deposition velocity (V_{dry}) of 0.01 m s⁻¹ is assumed in the simulations. Wet deposition includes in-cloud scavenging and below-cloud scavenging. The wet deposition velocity V_{wet} (used to calculate the in-cloud scavenging) is given by $S\mathcal{P}$ where S is the averaging scavenging ratio 3.2×10^5 by volume and \mathcal{P} is the precipitation rate. The below-cloud removal constant (s⁻¹) is given by 5×10^{-5} (1.0 – F_{b}), where F_{b} is the fraction of the polluted layer that is above the cloud bottom.

A virus decay constant is used to simulate the effect of biological ageing. We have adopted the value of the exponential decay constant λ (s⁻¹) suggested by Sørensen et al. [9], $\lambda = 6.4 \times 10^{-4} r$, where r is the virus decay rate for bovine fluids of 0.5 (h⁻¹).

The viability of the virus is dependent on both temperature and humidity [6, 9]. We use a linear decrease in virus concentrations to account for the temperature effect. We multiply the concentrations at temperatures 24° C and below by 1, but apply a linear decrease to the concentrations for temperatures above 24° C, so that at 30° C and above we multiply the concentrations by 0. For humidity, we use an exponential decrease in concentrations. For relative humidity of 60% or higher, we multiply the concentrations decrease exponentially as the relative humidity falls below 60%, so that at 1% relative humidity (the lowest value allowed) we multiply the concentration by 0.0000376. (At a relative humidity of 20% we multiply the concentration by 0.001.)

The outputs of this modelling are spatial plots of virus concentration at 1-m height in log10 TCID₅₀/m³ (showing a minimum concentration down to 10^{-6}), in ArcView shapefile format (ESRI, Redlands, CA, USA). These plots are available daily for the duration of the period of concern and as a cumulative exposure for the whole exposure period.

2.4. Exposure-risk model

Establishment of infection in a susceptible animal depends on the dose of airborne virus to which it is exposed. This exposure dose depends on the concentration of virus in the air, the air sampling capacity of the animal and the period of exposure [28]. When looking at the risk of infection from airborne FMD virus, a commonly used concept is that of minimum infectious dose, e.g., [29], which can be defined as the minimum amount of virus to cause infection (or disease).

Sutmoller and Vose [30] have discussed the issue of minimum infectious dose and recognise that the concept of a minimum threshold, below which an animal will not become infected if exposed, is not particularly useful for quantifying biological risks. The term infective dose should always be qualified with some probability level that indicates the proportion of animals that might succumb, as strictly speaking, even a single virus particle is sufficient to infect an animal although with a minute probability [6].

Cannon and Garner [6] used a binomial distribution to describe the probability of infection. The probability of infection to a low dose of virus is small, but increases with the size of the dose. The probability (P_i) that an animal will be infected when exposed to a given virus dose *d* in TCID₅₀ is given by

$$P_i = 1 - (1 - \theta)^d \tag{1}$$

where θ is the probability that one TCID₅₀ will infect an animal.

There is a limited amount of data on response to different aerosol doses and that which is available usually involves experiments on only small numbers of animals. Using maximum likelihood estimation, the probabilities that exposure to one infectious unit (IU) of virus would result in infection was estimated for calves and sheep [6], based on data from earlier studies [31, 32]. French et al.

Table 2 Default values used in exposure-risk model.

	Cattle	Sheep	Pigs	Source
Air sampling volume (m ³ /24 h) Probability that one virus particle will infect an animal	160 0.031	12 0.045	50 0.003	9, 10 6, 10, 19

[19] used a stochastic approach to analyse the same data and median estimates of θ were 0.031 for cattle and 0.045 for sheep. There is less information available for pigs although it is well recognised that pigs are relatively resistant to airborne FMD virus [8, 10, 29]. The minimum dose of airborne virus to infect a pig is estimated to be more than 800 TCID₅₀ [10]. If we conservatively assume that a dose of 800 TCID₅₀ has a 95% probability of infecting a pig, then θ for pigs can be estimated at about 0.003.

The probability that a group of animals (herd) becomes infected depends not only on the virus dose to which animals are exposed but also on the group size – the larger the group, the greater the probability that at least one



Figure 2. Study region. The cross (near the town of Tara) shows the location of the infected pig farm used in the outbreak scenario.

Table 3 Infected farm profile and outbreak scenario details.

Parameter	Value
Farm ID	3518
Farm type	Intensive piggery
Area (km ²)	1.01
Farm size (N)	11,000
Estimated contact rate (animals/day) ^a	40
Time to detection (days)	8

^a Number of pigs coming into contact with each infected pig sufficiently close that disease transmission could occur.

animal in the group will become infected. The probability (P_h) that a herd is infected is given by:

$$P_h = 1 - (1 - P_i)^n \tag{2}$$

where n = herd size.

Using equations (1) and (2), the risks that farms exposed to virus plumes can become infected can be calculated. To take into account that there may be more than one species present and to allow for exposure on multiple days, we used the following formula.

$$P_h = 1 - \prod_{i=1}^{j} \left[\left(1 - \theta_i\right)^{n_i} \left(\sum_{k=1}^{m} d_k\right) \right]$$
(3)

where i = species (sheep, cattle, pigs); k = day; d = exposure dose; n = number of animals of that species on the farm.

An application has been written in MapBasic (MapInfo Corporation) that overlays the virus concentration plots from the HYSPLIT model onto a digital map of the outbreak region showing farm boundaries. This application identifies those farms under the virus plume and calculates the probability of infection for each of these farms based on the type and number of livestock present. Table 2 shows the default values for calculating exposure doses and probabilities of infection. Based on these probabilities, a relative risk ranking (high, medium, low and very low) is applied. These rankings correspond to calculated probabilities of infection of greater than 50, 10-50, 1-10 and less than 1%, respectively. The results are presented in tabular and map formats.

3. Validation

Model validation is defined as a process of assessing the accuracy of model output and ensuring the usefulness and relevance of the model [33]. This implies that the assumptions underlying the model are correct and that the model representation of the study system is reasonable for the intended purpose. A more comprehensive view of validity considers 'data validity', or the correctness of the data used to construct and parameterise the model;

'conceptual validity', or the correctness of the mathematical and epidemiological logic upon which the model is built; and 'operational validity', or the ability of the model, as implemented, to produce results of sufficient accuracy [34]. Verification is a separate process to establish that the logic upon which the model is based has been correctly written down as code. [34].

Data validity is usually not considered to be part of model validation but is still important because it can be a factor affecting the ability to validate a model [34]. To build a model, adequate data is required to understand the problem and develop mathematical and logical relationships. In this study, we have used real data wherever possible – farm distribution and demographics, and weather data are based on actual records. For key model parameters like virus excretion rates, susceptibilities to infection, and virus decay rates, we have largely relied on published values from field or experimental studies. However, this data is not perfect, e.g. the animal virus dose-response data for estimating risk of infection is quite limited and incomplete.

Conceptual model validity is defined as determining that the theories and assumptions underlying the conceptual model is correct and that the model representation of the study system is reasonable for the intended purpose [34]. Here we are concerned with an integrated modelling system that is designed to identify and rank farms at risk of wind-borne infection of FMD to assist with disease management. A lack of experience with FMD under Australian conditions, and limited data from overseas outbreaks, mean that validation is a difficult issue. However, the modelling methods used are well described in the literature. The disease model is based on a relatively simple, well-established approach to simulating disease spread that is considered suitable for studying livestock diseases, including FMD (e.g., [18, 35-39]). One of the most critical components is the model's ability to deter-



Figure 3. Simulated epidemic curve and estimated virus production by day from infected pig farm.



Figure 4. Virus plume diagrams by day, with virus concentrations for July outbreak. Source farm location is shown as a black cross.

mine where to and in what quantities the virus is transported. Specific validation studies of the HYSPLIT wind-dispersal model for trajectories [the first Aerosol

Characterization Experiment (ACE)] and concentrations [the Across North America Tracer Experiment (ANA-TEX)], deposition (simulation of the Chernobyl accident),

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Figure 5. Exposed farms by risk ranking for July outbreak.

and volcanic ash (Rabaul eruption) have been reported [40]. Additional validation of concentrations is given in a report of the European Tracer Experiment (ETEX) [41].

From an operational perspective, the key output of the model is to identify and rank farms that may have been exposed to wind-borne FMD virus so that they can be dealt with in a logical order based on risk of infection. The riskexposure model is based on accepted risk assessment principles [19, 30] and the approach produces outcomes that are consistent with field observations. That is, cattle herds are more susceptible to airborne infection than sheep flocks and large herds are at greater risk than small herds (e.g., [12, 42-44]), with pigs being relatively resistant to infection [10]. The distances downwind for which farms are at risk are also within the range of field observations [6, 9, 12, 45].



Figure 6. Cumulative virus exposure over the period of virus excretion, by season.

Table 4 Number of exposed farms by risk category, by season.

Season	Category				
	High	Medium	Low	Very Low	
Summer	4	7	39	85	
Autumn	6	7	16	46	
Winter	5	5	19	110	
Spring	6	10	26	83	

Model verification was undertaken by asking an independent epidemiologist to re-read each line of code and evaluate its correctness. In addition, programming tools like structured programming were used. This is a code writing and formatting technique that assists with minimising the occurrence of syntax errors that do not prevent the program from compiling.

4. Application

The integrated modelling approach was evaluated by studying a hypothetical infection, with the same outbreak strain as that in the UK in 2001 [4], on an Australian farm using real weather data. The study region comprised an area of approximately 178,600 km² in southern Queensland. We used a spatial farm dataset (12,872 land parcels) provided by the Queensland Department of Primary Industries (figure 2). This temperate medium-broad acre region of Australia contains 4,196 beef cattle farms, 238 dairy farms, 204 sheep farms, 1,145 beef/sheep farms, 167 pig farms, 2,967 small holders (defined as having less than 50 animals) and 70 beef-cattle feedlots.

A previous study has shown that, even under optimal meteorological conditions, the risk of wind-borne spread from infected cattle and sheep with the UK strain is low even 1,000 infected sheep or cattle would only pose a threat to animal less than 1 km downwind [46]. Accordingly, in this study, we restricted our potential infected source herds to piggeries. A pig farm was randomly selected from the study population to be the IP. A plausible outbreak scenario, in terms of rate of spread within the farm and time to detection of the infection on the farm, was used. It is assumed that infection begins on the IP with introduction of three infected pigs from an outside source. The disease spreads in the piggery from these source animals and is not confirmed until 8 days after infection. The farm animals are slaughtered on day 9. The outbreak is assumed to have occurred in July (Winter). Further information on the source farm and outbreak scenario are provided in table 3.

To assess seasonal effects, we repeated the scenario at three other times of the year – January, April and October – corresponding to the Southern Hemisphere Summer, Autumn and Spring seasons.

Meteorological data. The weather data for study was the latest data available from the Australia Bureau of Meteorology at the time of the analysis. It consisted of gridded data of winds, temperature, humidity, and rainfall at approximately 12.5-km resolution at 3-hourly intervals for October 2003, January 2004, April 2004 and July 2004.

5. Results

Figure 3 shows simulated epidemic curve and virus production by day on the IP over the period from when infection is introduced until the case is reported.

Figure 4 shows plume diagrams with virus concentration isopleths, by day, for the period up until FMD is reported, for the July scenario (i.e., Winter). It is apparent that the areas exposed to wind-borne virus varied over the period. This is a function of both the spread of infection on the IP and daily meteorological conditions. The estimated risk of infection for exposed farms is shown in figure 5. In all, 139 farms containing susceptible livestock were exposed to wind-borne virus with 10 of these (7.2%) rated as a medium or high risk. The higher-risk farms tend to be closer to the source IP; however, there are some exceptions. For example, the two land parcels rated as medium risk approximately 40 km northeast of the IP in figure 5 comprise a very large intensive pig enterprise with 140,000 pigs. The two high-risk land parcels 27 km to the northwest of the IP are part of a larger mixed beef-sheep enterprise with other land parcels close to the IP.

5.1. Effect of season

Figure 6 shows the same scenario repeated in different seasons. The numbers of farms by risk category are shown in table 4. July (Winter) represented the season with the most farms at risk of infection from wind-borne FMD followed by January (Summer) and October (Spring). In terms of the number of farms at greatest risk, i.e. ranked as high and medium risk, Spring had the highest number, followed by April (Autumn). With all other things being equal, the differences in the size of the exposed areas and numbers of farms at risk are functions of the seasonal weather conditions. The meteorological factors affecting the dispersion are mainly the wind speed and the height of the boundary layer, i.e., the height of the turbulent mixing layer. Figure 7 shows plots of wind speed and direction for each of the seasons. In general, the highest wind speeds were found in January (Summer) and July (Winter), and the lowest wind speeds were in the transitional seasons of April and October. The height of the boundary layer, shown in figure 8, was generally lowest in July (Winter) and January (Summer), and highest in the transitional seasons. The low values of boundary-layer height in January (Summer) reflect wet soil conditions and cloudiness during the wet season (during times of drought the

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Figure 7. Wind speed and direction at 10-m height at the closest model grid point to the IP, by season.

boundary-layer height during January would be expected to be higher.) The product of wind speed and boundary-layer height is a measure of the turbulent mixing. Note that figures 6-8 show "snapshots" of the model results for different seasons to indicate the variation over the course of the year. They are not seasonal average values.



Figure 8. Boundary-layer heights at the closest model grid point to the IP, by season.

6. Discussion

A previous study has identified that weather conditions will not be a limiting factor for the potential spread of FMD for much of Australia [6]. The risk will depend on strain of virus, strength of the source and density of livestock exposed downwind. We describe an integrated modelling approach that can be used to identify areas exposed to wind-borne FMD virus and provide a rational basis for identifying and ranking farms at risk. It is based on specialised inputs from animal health and meteorological agencies that are linked through a GIS interface.

For a user like a disease manager to have confidence in a model, it is important that its limitations are recognised. Although FMD has been studied widely, there are several areas that are not well understood. For example, the animal virus dose-response data for estimating risk of infection is based on limited studies, usually with only a small number of animals and involving short-term exposure to airborne virus so it cannot be assumed that these findings can be extrapolated to 24-h averages [10]. The data for pigs, in particular, can be considered very conservative, as recent experimental studies have shown that pigs exposed to concentrations of virus up to 2,500 TCID₅₀/m³ over a 24h period were not infected although 1 out of the 10 pigs exposed did develop a borderline antibody response [29].

The approach also depends on the ability to recreate the pattern of virus excretion on infected farms. This depends on spread within the farm, and the ECR value used will affect the shape of the virus production curve. ECR is recognised as one of the most difficult parameters to estimate [18]. It can be expected to vary depending on factors like species, season, animal density, management systems, etc. The virus production model has been set up such that ECR is modified until the simulated output matches the field situation based on history and numbers of clinically affected animals present at the time of inspection. The accuracy of this approach will depend on obtaining a good history to estimate how and when FMD was introduced onto the farm, and on the ability to recognise affected animals. Thus, it will be less appropriate for use with sheep, where clinical signs in infected animals may be less apparent or absent. However, previous studies [6, 46] have shown that, even under optimal meteorological conditions, the risk of wind-borne spread from infected cattle and sheep farms is low. The highest risk of wind-borne spread of virus will come from large aggregations of intensively managed animals where disease is likely to spread rapidly. Under Australian conditions, these are most likely to be pigs (especially large piggeries) and, to a lesser extent, intensive cattle production (beef feedlots and large dairy farms).

Importantly, the approach takes into account that

• Virus production from infected animals varies with stage of the disease. For example, in cattle and pigs maximal virus production occurs in the early clinical stages of the disease, when vesicles first appear [10, 47].

- Spread of virus depends on the weather conditions at the time so that the number and distribution of farms exposed varies over time.
- Probability of infection depends on the type and number of animals exposed on farms as well as concentration of virus in the air.

Although we present a method for quantifying the risk, because of the many uncertainties in the process, the calculated probabilities of infection for exposed farms should be considered relative, not absolute. Uncertainties arise, because in the absence of data about when animals on an infected farm actually become infected and start shedding virus, it is necessary to estimate daily virus production retrospectively.

The virus production model generates a generalised epidemic curve that is consistent with the observed situation but may not necessarily reflect the true pattern of spread on-farm. In addition, dose-response data for estimating risk of infection is based on limited studies, as discussed above. It is for these reasons we have chosen to present the findings in terms of risk scores. The sensitivity of the model results to parameter choices and dependences will be examined in a future paper.

The approach provides a rational basis for assessing the risk that wind-borne spread of infection from infected premises may have occurred and thus a basis for allocating, possibly scarce, resources to surveillance and control activities. In actual outbreaks it is also important to recognise that wind-borne spread is just one of the potential pathways of spread that will need to be taken into account when setting surveillance and control priorities.

Although Winter is traditionally assumed to be the worst season for pollutant dispersion (because of the lower boundary-layer heights), in the period simulated in this study this was not the case due to the relatively high wind speeds encountered at this time.

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