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Simulating the spatial dynamics of foot and mouth disease outbreaks in feral pigs and livestock in Queensland, Australia, using a susceptible-infected-recovered cellular automata model

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Abstract

We describe an approach to modelling the spatio-temporal spread of foot and mouth disease through feral animal and unfenced livestock populations. We used a susceptible-infected-recovered model, implemented in a cellular automata framework, to assess the spread of FMD across two regions of Queensland, Australia. Following a sensitivity analysis on the infectious states, scenario analyses were conducted using feral pigs only as the susceptible population, and then with the addition of livestock, and initiated in the wet season and in the dry season. The results indicate that, depending on the season the outbreak is initiated, and without the implementation of control measures, an outbreak of Foot and Mouth Disease around Winton could continue unchecked, while an outbreak around Cape York may die out naturally. The approach explicitly incorporates the spatial relationships between the populations through which the disease spreads and provides a framework by which the spread of disease outbreaks can be explored through varying the model parameters. It highlights the emergence and importance of spatio-temporal patterns, something that previous modelling of FMD in feral animal and unfenced livestock populations has lacked.

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Keywords: Foot and mouth disease; Cellular automata; Susceptible-infected-recovered models; Spatial modelling; Feral animals

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1. Introduction

Foot and mouth disease is considered the most economically devastating livestock disease worldwide (Berinstein et al., 2000; Meyer and Knudsen, 2001). It is an extremely contagious virus that affects cloven-hoofed animals and can cause severe economic losses (Alexandersen et al., 2001; Cannon and Garner, 1999; Ferguson et al., 2001; Samuel and Knowles, 2001; Sorensen et al., 2000).

Australia is presently free of FMD, and so there is concern regarding the impact that an outbreak may have. Estimates of the cost to the Australian economy range from \$AU 2–13 billion depending on the severity of an outbreak (Gee, 2003), although little is known about the potential impact of the disease on feral and livestock populations (Pech and Hone, 1988).

Australia has many feral animal and unfenced livestock species that could act as hosts of FMD including pigs, goats, buffalo, camels, cattle, sheep and deer. These are of great concern because there is far less control over their interactions than there is with fenced livestock. Of these species, feral pigs are of most concern for five reasons. First, pigs are highly susceptible to the FMD Virus (FMDV, the etiologic agent of FMD) and act as amplifiers because they excrete large amounts of the virus (Durand and Mahul, 2000; Pech and Mcllroy, 1990; Wilson et al., 1992). Second, northern Australia has a dense population of feral pigs (Caley, 1993). Third, feral pigs are abundant, widely distributed and are often in close contact with livestock (Eggleston and Korn, 1992; Wilson et al., 1992). Estimates of the Australian feral pig population range from 13.5 to 23.5 million (Bunn, 2003). Fourth, pigs are scavengers, and often live near settled areas where the disease is more likely to enter the country (Eggleston and Korn, 1992; Wilson et al., 1992). In addition, as the virus is present in all excretions, secretions and carcasses of infected animals (Callis and McKercher, 1981) and can persist outside the host for up to a month (Ferguson et al., 2001), feral pigs could easily contract the disease if it were present in livestock. Fifth, feral pigs can be difficult to control (Pech and Mcllroy, 1990).

The aim of this research is to model the spatial dynamics of a foot and mouth disease (FMD) outbreak in feral pig and livestock populations to examine how such an epidemic would spread through time and space. We use a susceptible-infected-recovered approach, implemented within a cellular automata model, to assess the potential spread of FMD through two areas in Queensland, Australia. There are several variables in the model, and we explore its sensitivity to a subset of these. Other variables need to be assessed in future research.

2. Materials and methods

FMD and feral pigs have many characteristics that need to be considered before any modelling is conducted. One also must use an appropriate modelling approach that can represent these characteristics. We now describe these, following which we describe the model implementation.

2.1. How FMD spreads

FMDV is highly transmissible. It is rapidly replicated in hosts, and only very small quantities of the virus are required to initiate infection. There are numerous ways by which it can infect animals (Alexandersen et al., 2001), including via aerosol transmission (exhaled particles) from infected animals or by direct contact with infected animal products (Cannon and Garner, 1999; Eggleston and Korn, 1992; Samuel and Knowles, 2001). Infected animals are the main source of transmission (Samuel and Knowles, 2001). For example, after the primary infection of a pig herd, the 2001 FMD outbreak in the UK spread rapidly over long distances via the transport of livestock and by contact at markets (Ferguson et al., 2001). The virus can also be spread via contaminated materials transported by humans, birds or wild animals (Cannon and Garner, 1999; Eggleston and Korn, 1992; Samuel and Knowles, 2001). FMDV may also persist in the tissues of dead animals for up to several months if refrigerated (Meyer and Knudsen, 2001).

Long distance transport of FMDV by air currents is possible (Donaldson, 1983), and there is evidence to suggest that a major outbreak in the UK in 1981 was initiated by windborne particles carried across the English Channel from France (Alexandersen et al., 2001; Donaldson, 1983; Sorensen et al., 2000). However, long distance windborne spread has not commonly been reported because very specific conditions such as stable air currents are required, and they all must occur simultaneously (Bunn et al., 1998; Cannon and Garner, 1999; Mikkelsen et al., 2003).

2.2. Feral pigs and FMD

Latency, the time from exposure until a herd is capable of infecting others, lasts for approximately 7–8 days in pigs (Garner, 1992; Garner and Lack, 1995; Pech and McIlroy, 1990). Pig herds have been reported to be infective for between 10 and 17 days (Garner and Lack, 1995). Immunity depends on herd size, contact and number of susceptible animals, and in a pig herd may last between 3 and 6 months (Garner and Lack, 1995; Pech and Hone, 1988).

Associated with these disease states, there are ten ecological factors that determine whether a disease will be maintained and transmitted by wild animals (see Ausvetplan, 2000). These are the population density and distribution, the reproductive status and seasonality, interactions between wildlife and livestock, animal response to disturbance, animal movement and distances travelled, habitat requirements, social organisation, age structure and population, home range, and barriers to dispersal. These need to be incorporated into modelling approaches, either implicitly or explicitly.

Given that FMDV is easily transmitted by numerous mechanisms, it is also likely that all animals in a herd will rapidly contract the disease once one member is infected (Alexandersen et al., 2001; Sorensen et al., 2000). When a herd becomes infected there are usually other susceptible herds nearby. Thus, the most important factors determining the propagation and maintenance of FMD in susceptible herds are their geographical density, connectivity and size (Durand and Mahul, 2000).

2.3. Modelling the spread of FMD

It is vital to have some understanding of how a disease as serious as FMD would spread through time and space (Garner, 1992). Epidemiological models are useful in this regard, allowing an assessment of the impact an outbreak may have under a variety of conditions (e.g. Cannon and Garner, 1999; Garner and Lack, 1995; Suttmoller et al., 2000). Such models are recognised as fundamental to developing management strategies for disease control (Durand and Mahul, 2000; Ferguson et al., 2001; Schoenbaum and Disney, 2003; Sørensen et al., 2001).

While there are models that analyse the spatial spread of FMD between farms (e.g. Gerbier et al., 2002; Keeling et al., 2001; Morris et al., 2001), most simulation models for FMD outbreaks amongst feral animals and unfenced livestock do not examine the spatial spread of such an outbreak (Dexter, 2003; Pech and Hone, 1988). Susceptible-infected-recovered models of disease are usually based on differential equations (Ahmed and Agiza, 1998; Sirakoulis et al., 2000) and neglect the spatial dynamics of dissemination. By not incorporating geographic relationships, these models are inadequate for detailed response planning in the event of outbreaks.

An alternative modelling approach to differential equations are artificial life models such as cellular automata (Fuentes and Kuperman, 1999; Openshaw and Openshaw, 1997; Sirakoulis et al., 2000). Cellular automata explicitly incorporate geographic relationships (Mackey and Laffan, 2002). They are models of physical systems in which space and time are treated as discrete units and interactions occur between local neighbours (Balzter et al., 1998; Benyoussef et al., 1999; Fuentes and Kuperman, 1999; Sirakoulis et al., 2000). Cellular automata are based on the assumption that a set of simple rules which operate on a local neighbourhood, but that apply globally, underlie complex systems. The repetitive application of these rules within the local neighbourhood allows an analysis of complex behaviour, generating emergent, unpredictable patterns from local deterministic interactions (Openshaw and Openshaw, 1997).

A cellular automata model is implemented using a lattice of cells, where each cell interacts with other cells within a local neighbourhood. These interactions are controlled by a set of rules concerning the neighbourhood and cell states at earlier time steps (Ahmed and Agiza, 1998; Ahmed and Elgazzar, 2001; Fuentes and Kuperman, 1999; Sirakoulis et al., 2000). Cells are normally unable to directly communicate at a global scale (Duryea et al., 1999).

Cellular automata have several advantages for epidemiological modelling. They can deal with complex initial conditions and geographical boundaries (Filipe and Gibson, 1998; Sirakoulis et al., 2000), are relatively simple to construct and understand, and are computationally efficient (Clarke et al., 1997; Fuentes and Kuperman, 1999; Openshaw and Openshaw, 1997). Cellular automata are more general than differential equations (Ahmed and Elgazzar, 2001) and do not depend on generalised probability distributions derived from observations or hypothetical data.

Cellular automata have been applied to a wide range of pathogen and disease spread problems (Ahmed and Agiza, 1998; Benyoussef et al., 1999; Duryea et al., 1999; Johansen, 1996; Rousseau et al., 1997; Vlad et al., 1996). Susceptible-infected-recovered models are often built into cellular automata to examine the spatial and temporal propagation of

epidemics (Benyoussef et al., 1999; Filipe and Gibson, 1998; Johansen, 1996; Kleczkowski and Grenfell, 1999; Rousseau et al., 1997; Sirakoulis et al., 2000), and form the framework of our model.

2.4. The data set

As we have noted, the most important factors for modelling the spread of FMD are the geographical density, connectivity and size of susceptible species distributions (Section 2.2, Durand and Mahul, 2000). All of these can be represented by spatial data sets of the susceptible population.

A feral pig density and distribution map was obtained from the Australian Bureau of Rural Sciences. Densities used are low, medium and high, and were based on questionnaires and aerial surveys conducted between 1986 and 1988 (Wilson et al., 1992). While these are old surveys, the relative abundances and spatial patterns are expected to be the same as the present. High density locations had regular sightings, day or night, and abundant fresh droppings and signs. Medium density locations had sightings mainly at night, and moderate signs, many of which were old. Low density had very few sightings or signs of pigs.

Sheep and cattle density and distributions were constructed using sheep and cattle figures for statistical local areas (SLAs) from the Australian Bureau of Statistics' 1988–1989 AgStats database (ABS, 1989). Sheep and cattle were assumed to be evenly distributed across the SLA units.

Two feral pig density data sets were generated, one each for the dry and wet seasons. The feral pig density estimates are spatially and temporally coarse (0.25° cells), and so more detail was inferred using knowledge of seasonal behaviour and habitat information (Table 1) (Caley, 1993; Hone, 1990a,b). Habitat areas were derived from the National Vegetation Information System Major Vegetation Groups data set (ERIN, 2001), and allowed us to represent such factors as the seasonally high density of feral pigs along river courses and on floodplains (Caley, 1997; Pech and Hone, 1988; Wilson et al., 1992). The newly assigned feral pig densities for each cell were then divided by 10, which is a higher estimate for the average family group size (1–10 animals) (Ausvetplan, 2000), to estimate the number of herds per cell for both the wet and dry seasons. These two data sets were used to simulate the spread of FMD amongst feral pig populations only.

Table 1

Density values (pigs km^{-2}) assigned to the feral pig distribution map based on the relative density and distribution map and habitat utilisation between seasons (wet and dry)

Habitat class	High density		Medium density		Low density	
	Wet	Dry	Wet	Dry	Wet	Dry
Mixed woodland and cereal	10.9	10.9	6.1	6.1	2.2	3.5
Eucalyptus woodland	1.5	0	1.2	0	0.9	0
Other woodland	2.8	2.8	2.2	2.2	1.6	0.8
Other habitat types	3.2	12.3	2.6	10.9	2.0	9.5

Data from Caley (1993); Hone (1990a,b).

It is difficult to maintain the distinction between susceptible species when using a single layer state transition model to simulate the propagation of disease, as separate dissemination rates cannot be applied for each species. An average composite herd must therefore be used (Durand and Mahul, 2000). For this study, data for all species included in the model (sheep, cattle and feral pigs) were converted into feral pig and pig equivalent herds per cell. The number of sheep and cattle herds in each cell was converted to pig equivalent herds based on the minimum dosage required to infect each species. Different species are susceptible to FMD to varying degrees (Durand and Mahul, 2000). The minimum dosage required to infect sheep, cattle and pigs are 7, 9 and 14 infectious units (viral particles), respectively (Bunn et al., 1998). This is largely based on inhalation rates (Cannon and Garner, 1999; Sørensen et al., 2001) and the amount of virus exhaled varies considerably between species (Sørensen et al., 2001). One sheep flock was considered equivalent to 0.5 pig herds, and one cattle herd equivalent to 0.64 pig herds. The pig herd equivalent data sets were added to the seasonal feral pig herd data sets to examine the potential spread of FMD amongst both feral pigs and livestock.

2.5. Analyses

A series of models were run to gain an understanding of the general pattern of FMD spread through space and time. Following a set of sensitivity analyses to determine a subset of the model parameters, we used a series of models where we included or excluded livestock, initiated the model in the wet or the dry season, and used two different initiation locations.

Two locations in Queensland were selected as sources of infection for FMD, one in Cape York Peninsula (“the Cape”) and one outside Winton (Fig. 1). Both sites were selected because of the high density of feral pigs in the area (at least at certain times of year) as an FMD outbreak is more likely to occur at such locations (Durand and Mahul, 2000). One cell in each location was “infected” as the primary source of infection to initiate each model scenario.

Each model was run for 360 days based on estimates that an outbreak would be detected by this time (Eggleston and Korn, 1992). Of the ecological factors affecting disease transmission by wild animals (Section 2.2), we explicitly incorporated the population density and distribution, seasonality, habitat requirements, and home ranges. We did not model barriers to dispersal, reproductive status and age structure. The remainder were implicit in the population data sets and model structure. We also did not model mortality and control measures such as culling, although the model is sufficiently flexible for such measures to be incorporated.

2.5.1. Model implementation

We used a purpose-built cellular automata model, implemented using the map algebra system in ArcInfo GRID version 8.2. No interactions took place beyond the eight nearest neighbours of a three by three cell square. Diagonally adjacent cells in the lattice were assigned a reduced probability of interacting to account for their reduced contiguity and greater distance from the centre cell’s centroid. This value was 71% of non-diagonal neighbours, which is proportional to the Euclidean distance of the cell centroids from the central cell.

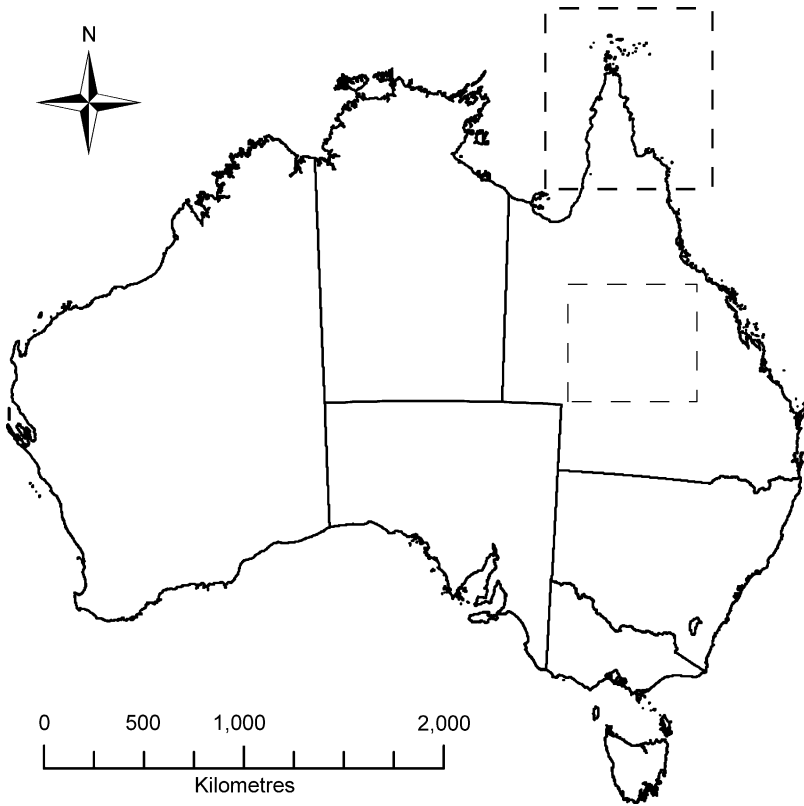


Fig. 1. Regions for which the models were run are bounded by the dashed lines. Cape York is in the north, Winton is in the south.

Bastos et al. (2000) found that relationships for buffalo contracting FMD were strongly related to home ranges. Similarly, Benyoussef et al. (1999) used a cell size that approximated the average territory size of a fox family when modelling the spread of rabies. A similar approach was adopted for this study where a cell size of 29 km (5.385×5.385 km) was chosen to represent the average feral pig home range size for both boars and sows in tropical Australia (Caley, 1993). Feral pig home ranges overlap (Pech and McIlroy, 1990), and using an average size implicitly accounts for this in the modelled interactions because the larger ranges will overlap with adjacent smaller ranges.

In the model the herds in a cell could pass, sequentially, through four model states: from susceptible to latent, to infectious, to immune and then to susceptible again. These transitions partly determined the dissemination rate between herds (Garner and Lack, 1995).

As in Garner's (1992) model, the first transition (secondary infection) depended on contact rates between susceptible and infected herds in the previous time step. Feral pigs are very mobile so herds within cells were assumed to come into close enough contact to

transmit the virus every 1–6 days (Pech and Hone, 1988; Pech and McIlroy, 1990). We therefore assume complete contact between all pigs in a cell after 6 days, and complete infection of the cell after that. Only after that period was an infected cell capable of infecting its neighbours. One of the main assumptions of this approach is that homogenous mixing of herds took place within cells but not between cells.

The probability of interactions between cells depends on the density of susceptible herds. Cells with more than a maximum threshold of pig equivalent herds were assigned a 100% probability (1.0) of becoming infected if they had a neighbour that was infected, assuming that contact between these herds will always occur. Cells with less than a minimum threshold of pig equivalent herds had a 0% probability of becoming infected, as population densities would be too low for contact to occur. The remaining cells were linearly scaled into the interval [0,1] by subtracting the minimum threshold and dividing by the threshold range (maximum threshold – minimum threshold). Non-linear and discontinuous functional forms could be used where they are supported by known responses. To incorporate chance into the model, interactions between a susceptible cell and an infectious neighbour occurred when a random value from a pseudo-random number generator (PRNG) was below a cell's probability threshold.

The randomness of sequences generated using PRNGs is crucial to the model results. However, the quality of PRNG algorithms can vary greatly, and many have serial correlations and other issues of non-randomness (Van Neil and Laffan, 2003). To reduce these effects we used the Mersenne Twister mt19937 algorithm (Matsumoto and Nishimura, 1998). This algorithm has a large period length before the sequence of random values repeats ($2^{19937}-1$) and good spectral properties where correlation structures within the random number sequence are very small.

Once a cell was infectious the second, third, and fourth transitions in the model depended on the length of the latent, infectious and immune periods assigned in the model parameterisation.

We do not consider long distance wind borne transmission in these analyses. They were assessed separately, but the results were the same as those presented below.

2.6. Model sensitivity

There is a range of estimates for the parameters used in the model. For example, the minimum and maximum probability threshold values cannot be based upon information from other studies. The estimated threshold density below which FMD would not persist in feral pig populations (made by Pech and Hone, 1988) was confined to specific habitat types, while there is a range of habitats across the study area. Importantly, the carrying capacity of different habitats determines the threshold density for FMD, and the smaller the difference between the two, the longer the outbreak will be (Eggleston and Korn, 1992).

To assess the impact of varying the model parameters, and to select a standard model for the scenario analyses, we conducted a series of sensitivity analyses on a subset of the parameters. We focussed first on the duration of the latent and infectious periods, and then on the minimum and maximum probability threshold values. Each sensitivity analysis assessed four parameterisations for five separate model runs over 365 days.

Based on figures in [Garner and Lack \(1995\)](#), the latent and infectious parameters assessed were combinations of, respectively, 7 and 14, 13 and 14, 7 and 17 and 13 and 17, each with the immune period set to 90 days. The maximum probability thresholds assessed were 30, 40, 50 and 60 herds per cell, and the minimum threshold densities were 5, 10, 15 and 20 herds per cell.

2.7. Scenario analyses

Once the sensitivity analyses were completed, we conducted a series of scenario analyses to examine where and when livestock would be most at risk in the case of an FMD outbreak.

The population within the lattice was varied to include only feral pigs or feral pigs plus livestock. The impact that seasonal variation could have upon the propagation of FMD was examined by initiating outbreaks during both the wet and dry season, and running simulations for 360 days (180 days for each season) using the wet and dry season population density data sets in the appropriate seasons.

3. Results

3.1. Sensitivity

The peaks of the modelled outbreaks reduced with the higher latency period, and became more extensive with the higher infectious period ([Fig. 2](#)). This is to be expected, as these parameters regulate the opportunity for transmission in the model. A short latency period and long infectious period will increase the probability of an interaction occurring

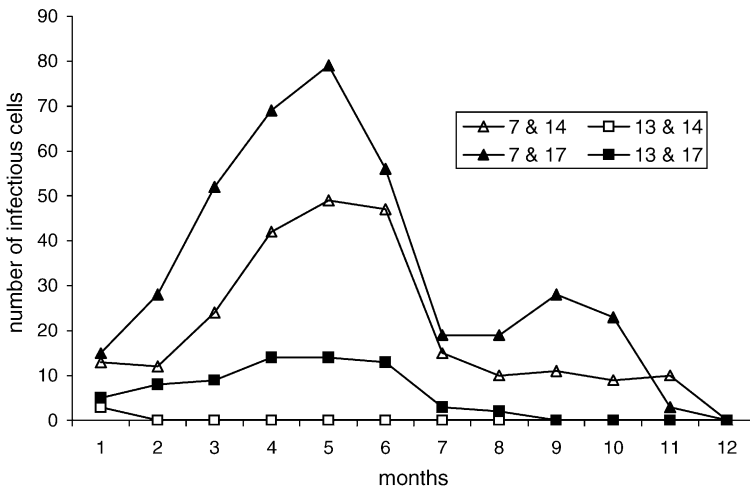


Fig. 2. The impact of varying the latency and infectious periods on the number of infectious cell per month for an outbreak amongst feral pigs in the Cape during the wet season.

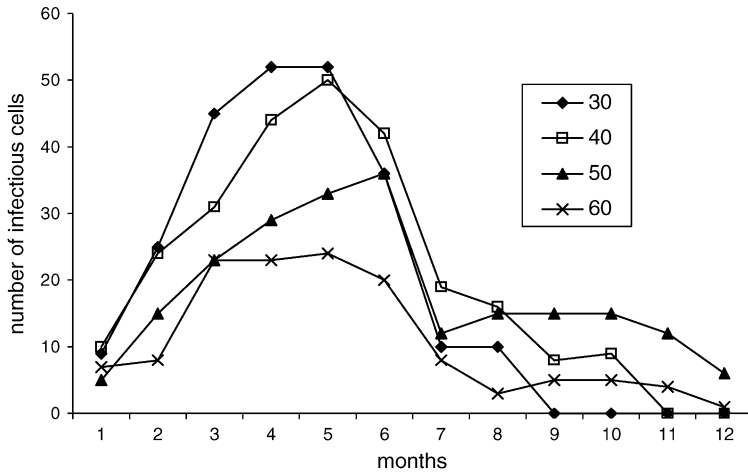


Fig. 3. The impact of varying the maximum probability threshold (herds per cell) on the number of infectious cells per month for an outbreak amongst feral pigs in the Cape during the wet season.

and so resulted in the highest number of infected cells in the model. For analysis purposes, a standard model was chosen to have a latency period of 7 days and an infectious period of 14 days.

Varying the maximum probability threshold did not influence the overall pattern of spread through time, with the peaks occurring at approximately the same time (Fig. 3). However, it did affect the magnitude of spread, which increased as the maximum probability threshold was lowered. The higher rates of spread far exceed those that have been observed or modelled previously, and so a standard model was chosen with a maximum probability threshold of 50 pig equivalent herds per cell.

Varying the minimum probability threshold had a greater impact upon the number of cells affected by FMD than did varying the maximum probability threshold (Fig. 4). The results indicate that a simulated outbreak would be more likely to die out within the first month with higher minimum probability thresholds. This is because raising the minimum threshold above zero excludes parts of the susceptible population from possible contact. Based on these findings, the minimum probability threshold was set to zero herds per cell to include all susceptible members of the population in the scenario analyses.

3.2. Outbreaks in feral pigs only

There was little variation between the five model runs for each scenario with feral pigs only, so common patterns could be distinguished (Figs. 5 and 6). The results indicate that an outbreak around Winton would probably be more severe than one in Cape York Peninsula, as it would spread rapidly and extensively, especially during the dry season, and not die out naturally. An outbreak in Cape York Peninsula during the wet season would peak after approximately 6 months and then begin to wane during the dry season. In contrast, an outbreak in Cape York Peninsula during the dry season would not spread

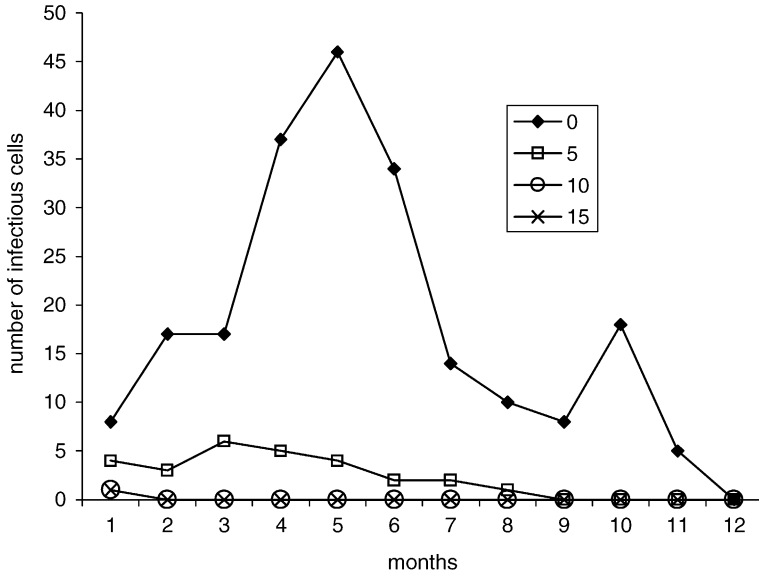


Fig. 4. The impact of varying the minimum probability threshold (herds per cell) on the number of infectious cells per month for an outbreak amongst feral pigs in the Cape during the wet season.

extensively and would die out within 6 months because of the low density of susceptible animals.

We suggest that an outbreak during the wet season in feral pigs only would be of greatest concern around Winton. This is based on the risk posed to livestock using the pattern of

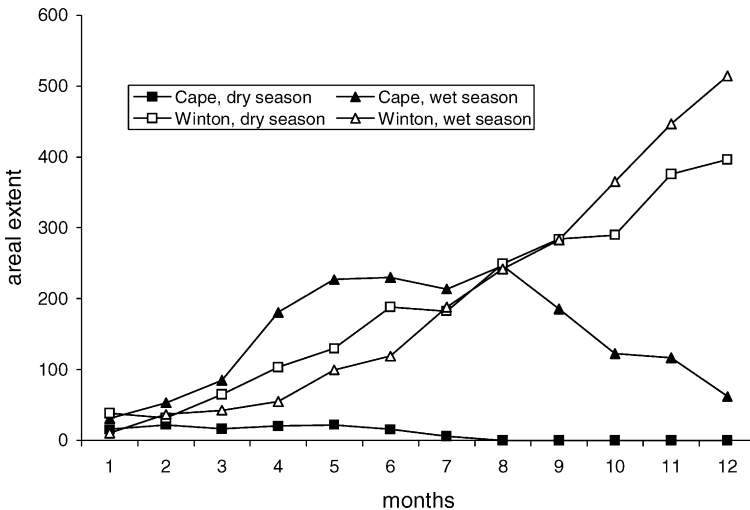


Fig. 5. The estimated areal extent (km) of infectious cells through time for the scenarios including feral pigs only.

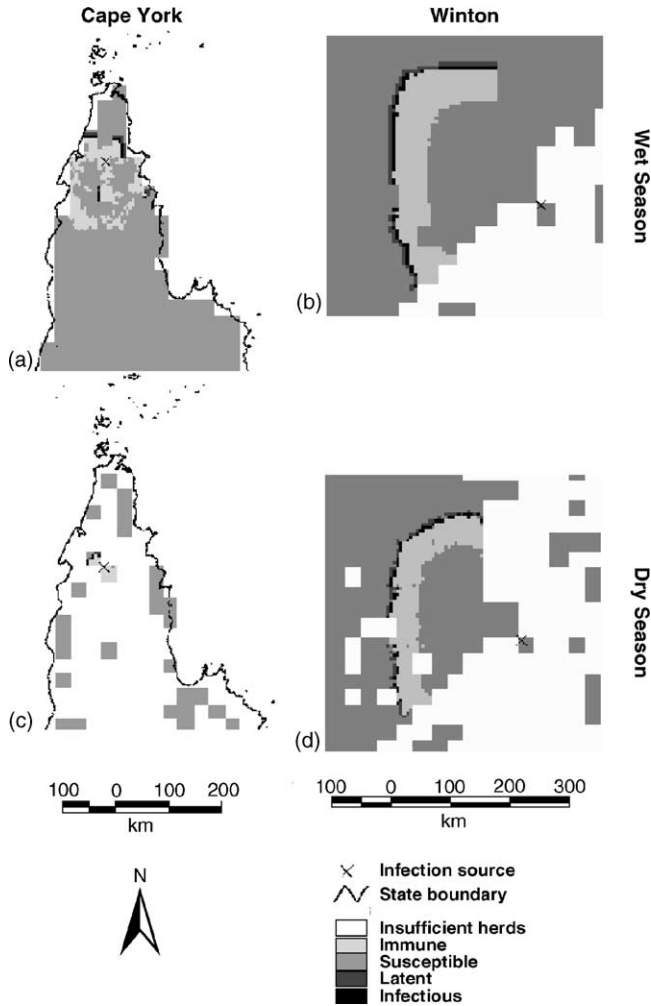


Fig. 6. The spatial distribution of FMD infections with the maximum number of cells infected during the simulation for scenarios involving feral pigs only, initiated in the wet and dry seasons: (a) 12 months; (b) 11 months; (c) 3 months; (d) 12 months. The areas are shown in Fig. 1.

spread through time and space as well as the greater number of livestock herds near Winton that could potentially be affected.

3.3. Outbreaks in livestock and feral pigs

The results indicate that FMD outbreaks including sheep, cattle and feral pigs are more likely to become established, and not die out naturally, than those that include feral pigs only. This is with the exception of an outbreak initiated in the dry season in Cape York Peninsula, where livestock densities are very low. An outbreak occurring around Winton at

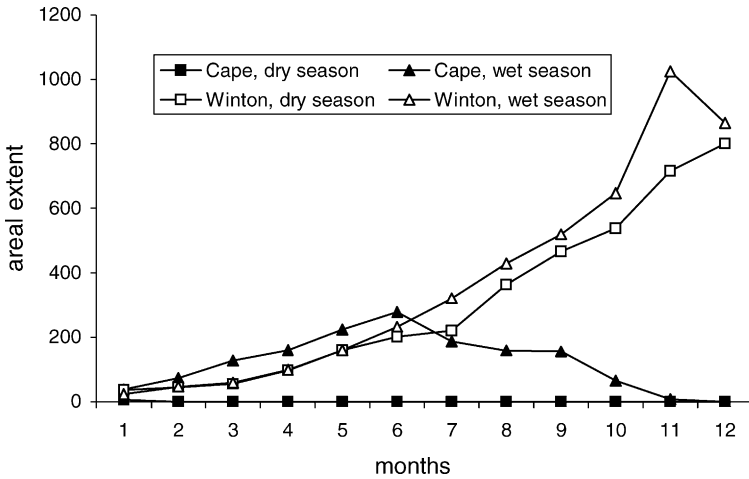


Fig. 7. The estimated areal extent (km) of infectious cells through time for the scenarios including sheep, cattle and feral pigs.

any time of the year would spread rapidly and extensively, whereas an outbreak in the north of Queensland during the wet season would spread more slowly and begin to die out naturally after 6 months (Figs. 7 and 8). The spiral patterns in Winton (Fig. 8) are the result of the infection front re-infecting a population that has become susceptible again after the immunity period has ended.

The estimated rate of infection between herds at the peak of each outbreak also gives an indication of how quickly the disease could travel between susceptible populations in Australia. The rate of infection around Winton in the wet season is approximately 40 herds per week, and 26 herds per week in the dry season. Conversely, the Cape is 18 herds per week in the dry season, and only 1 herd per week in the wet season. An epidemic around Winton would therefore be of greater concern than one in Cape York Peninsula.

4. Discussion

We now discuss the results of the sensitivity and scenario analyses and consider alternate parameterisations that would extend the utility of the model for understanding FMD transmission in feral animal and unfenced livestock populations.

4.1. Sensitivity analyses

The length of the latency period is important for the spread of FMD in the model. Latent cells may form a barrier around infectious cells and prevent transmission of the disease front to susceptible cells. Therefore, longer latency periods increase the likelihood that the propagation of FMD will be moderated, something also found by *Pech and McIlroy (1990)*. Conversely, longer infectious periods make it less likely for the simulated spread to be moderated.

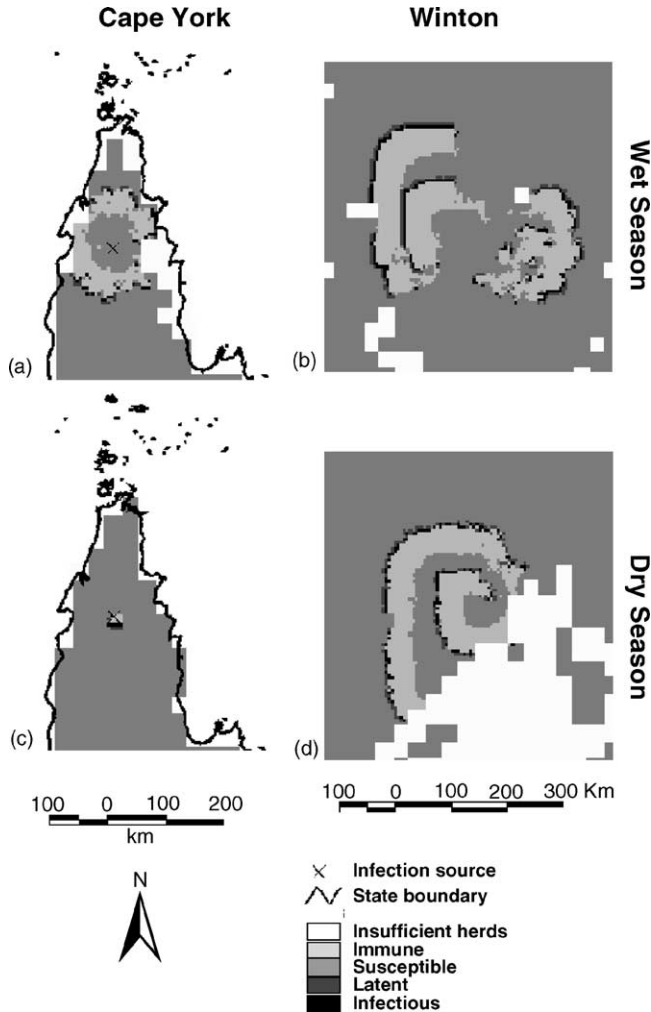


Fig. 8. The spatial distribution of FMD infections with the maximum number of cells infected during the simulation for scenarios involving feral pigs and livestock: (a) 11 months; (b) 11 months; (c) 1 month; (d) 12 months. The areas are shown in Fig. 1.

The model was very sensitive to variations in the probability threshold layer. Raising the maximum threshold slowed the rate of spread as it decreased the probability of cells interacting. Raising the minimum threshold slowed the rate of spread because it prevented cells with low densities from interacting with infected cells. A minimum threshold of 5, 10 or 15 pig herds per cell excludes respectively, 41.2, 91.6 and 99.9% of the susceptible population in the wet season.

It is important in disease modelling to estimate the threshold density of susceptible animals below which the disease would die out and above which it would become well

established and/or persist indefinitely (Caley, 1993; Pech and McIlroy, 1990). Sensitivity analysis on the probability threshold layer of the model did not actually provide information on FMD threshold densities, it merely established how sensitive the model was. As Benyoussef et al. (1999) state, establishing a threshold density using an epidemiological model is dependent upon its other parameters. Thus, the high degree of uncertainty in the model associated with the density and distribution of feral pigs, the interactions between feral pigs and livestock as well as the potential behaviour of FMD in Australia make it impossible to establish true threshold densities for FMD.

Despite the probability threshold layer not providing information on FMD threshold densities, it could be used to incorporate the impact of control measures. The dissemination rate of FMD slows once quarantine and control measures are implemented (Garner and Lack, 1995). Therefore, by using different probability layers with progressively higher thresholds, the dissemination rate could be slowed and the impact of control measures could be simulated. Varying the probability threshold layer could also be used to calibrate the model against other FMD exercises.

4.2. Scenario analyses

The modelled rate of spread appears to be an overestimate compared with previous results. However, this is not necessarily a disadvantage because FMD outbreaks can be catastrophic, and so it is better to overestimate the rate of spread than to underestimate it. Consequently, the simulated spatial pattern of spread is a very useful indication of how FMD would propagate throughout Queensland without control measures, especially amongst feral pig herds.

The model results can largely be explained in terms of population densities, the season an outbreak commenced and the inclusion of other animal species. If susceptible population densities are low then FMD may die out naturally before it becomes established. These results are to be expected given the structure of the model, but are consistent with Durand and Mahul (2000) who suggested that the geographical density of susceptible herds is the most important factor in controlling the spread of FMD.

The higher density of susceptible herds around Winton is clearly the reason why the FMD simulations in the model are more likely to become established, not die out naturally, and spread rapidly when initiated in that part of the state. An outbreak near Winton could also spread in multiple directions and possibly re-infect recovered herds if they have not been culled, vaccinated or removed.

The more extensive spread of outbreaks in the Cape initiated during the wet season in comparison with the dry season is explained by the more continuous distribution and the increased density of feral pigs in many habitats at that time of year (Caley, 1993). The discontinuous distribution (Pech and McIlroy, 1990) and lower average feral pig densities in northern Australia in the dry season (Caley, 1993) incorporated into the model explain why FMD died out quickly in Cape York Peninsula during the simulations commenced in the dry season. These findings are comparable to the spread of the disease amongst wildlife populations in southern Africa, where FMD is often confined to localised geographical areas in the dry season (Sutmoller et al., 2000). However, while outbreaks in Cape York Peninsula are comparatively smaller than in Winton, they may not be detected in feral pig

populations for many months and would be difficult to control in the rugged terrain (Eggleston and Korn, 1992; Pech and McIlroy, 1990).

4.3. Alternate parameterizations

There are many parameters in this model, and we have experimented with only a subset of them. We now consider alternate parameterisations to pursue in future work, all of which can be implemented within the cellular automata framework.

The first consideration is that we have initiated the model at only two locations in the study area, based on population densities (Durand and Mahul, 2000). An FMD outbreak could potentially be initiated anywhere there are susceptible species, albeit with varying degrees of probability. It would be useful to conduct a formal risk analysis to identify areas of probable introduction of FMD infected material and subsequent initiation of an outbreak.

Feral animal species which were not included in the model but that are susceptible to FMD, for example goats, deer and buffalo, may also play a role in the spread of FMD in Australia. Including these species in the model as it stands would obviously increase the rate of spread. Wild cattle and buffalo populations in northern Australia could be especially important as they may interact with domestic stock and other feral animals that are susceptible to FMD. However, the lack of knowledge regarding such interactions in Australia limits modelling exercises of this type (Pech and Hone, 1988).

However, there is also some disagreement concerning the interactions between feral pigs and other species and therefore the potential for the disease to spread between these populations. The assumption that cells with feral pigs or feral pigs plus livestock were as likely to interact with other cells with livestock, and thereby spread FMD, was fundamental to the functioning of the model. This assumption is reasonable when susceptible species share the same habitat (Bastos et al., 2000; Pech and McIlroy, 1990), and FMD in Australia would rarely be restricted by the supply of susceptible animals as feral pig populations tend to be contiguous and will come into contact with livestock at water sources. However, where species do not share habitats, but do co-occur within a model cell, interaction between feral pigs and livestock leading to inter-species transmission may be rare (Pech and McIlroy, 1990). For example, FMD has rarely been known to spread from cattle or buffalo to pigs in tropical countries (Animal Health Australia, 2001). Part of the reason for this is that feral pigs would become lame when infected with FMD, thereby limiting contact with other feral pigs and so would effectively be infectious for only a very limited period (M.G. Garner personal communication 27/09/02). However, one cannot discount the possibility of contact between a healthy, susceptible animal and a lame, infected animal, or alternately between a healthy animal and the faeces of an infected animal.

The estimates for the minimum dosage required to infect different species vary considerably (Bunn et al., 1998; Cannon and Garner, 1999) and could alter the spread of a simulated outbreak. For example, if a different estimate (22 infectious units) for the minimum dosage required to infect cattle (Cannon and Garner, 1999) was used instead of nine infectious units, the probability of cells containing cattle herds interacting with cells containing other herds would have been lowered. This would slow the rate of spread

amongst susceptible populations. One could also consider the volume exhaled by different species, although this may be of less concern because only small volumes are needed for transmission.

The model was also limited in how it treated the infectious states because, although sheep and cattle were incorporated, the model was based on how FMD affects pigs and assumed a complete random mixing between all population units. With the considerations discussed above, this can explain the higher than expected rate of spread within the model. Future research needs to incorporate separate layers for each species to allow modelling of the different exhalation rates between species, interspecies interactions (where known) and the differing spatial patterns of dissemination. These could also incorporate sensitivity to particular strains of FMD. For example, the epidemic in North Africa and the Middle East between 1987 and 1994 was caused by FMDV type O, to which sheep and goats are more susceptible than other species (Samuel et al., 1999). In the case of our study areas, an outbreak caused by FMD type O near Winton would probably spread more rapidly to the south due to the greater numbers of sheep in that area.

Furthermore, one must consider varying the latent, infectious and immune periods. For example, the infectious period is determined by the herd size and type, and a cattle herd can be infectious for between 9 and 66 days (Garner, 1992). Rather than assuming a constant rate over time, these parameters could be varied stochastically on a cell-by-cell basis. One could also treat the population densities and average herd size variables in a stochastic manner to allow for some of their associated uncertainty.

Finally, if the impact of transported contaminated animals or products were taken into account then the distances over which FMD could spread would be even greater than modelled here. The large-scale movement of livestock has been crucial during FMD outbreaks in Africa and South America and would probably be of major importance in Australia (Garner and Lack, 1995; Samuel et al., 1999), and as indicated by Exercise Minotaur (AFFA, 2002). One should also consider the transport of feral pig carcasses by hunters. This could be incorporated by adding extra sources of infection at probable locations at appropriate model iterations.

5. Conclusions

This study, as it examined the propagation of FMD from a spatio-temporal perspective, provides new general representations of how FMD may behave under Australian conditions. Little is known about the role that feral pigs would play in the instance of an FMD outbreak in Australia (Pech and Hone, 1988) and, despite the potential sources of error discussed above, our model provides valuable information to inform contingency plans. In the case of an outbreak, the model could be used to predict the progress of an epidemic and is flexible enough to incorporate management actions, such as culling, as they are conducted.

The model also provides a framework to explore the interactions between the different factors that control the spread of FMD, and further exploration of the model parameters will provide valuable insights into the spread FMD in feral and unfenced animal populations in Australia and similar environments.

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