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A Risk Assessment and Simulation Modelling Framework for Exotic Disease Prioritisation in the Australian Pig Industry: Part 2

Final Report
APL Project 2010/1012.361

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Introduction

This report describes completion of tasks to satisfy project 2010/1012.361 'A risk assessment and simulation modelling framework for exotic disease prioritisation in the Australian pig industry'. Part 2 of this project is focused on development of methods for assessing the disease status of feral pig populations.

Project Objectives and Outcomes

1. Conduct an Aerial Survey of Feral Pigs

Two aerial surveys of feral pigs in the Fitzroy River basin centred on Fitzroy Crossing, Western Australia were conducted in August 2011 (with additional funding and in-kind support from the Department of Agriculture and Food, Western Australia). See milestone reports 1 and 3 for details.

2. Using a Disease Spread Model, Simulate Likely Disease Spread Scenarios Based on the Estimated Population at-Risk and Population Demographics

The spread of classical swine fever in the Fitzroy River basin feral pig population was successfully simulated. The estimated population at-risk and population demographics were initially based on expert opinion and published literature. See milestone report 2 for details. In addition, the results of this research has been published (and is freely available online at http://www.veterinaryresearch.org/content/43/1/3/abstract) in the peer-reviewed journal *Veterinary Research*. This journal is ranked number 1 out of 140 journals in the field of Veterinary Science, with a current impact factor of 3.6.

3. *Calculate the Minimum Number of Feral Pigs that Would Need to Be Sampled to Detect Disease Presence or to Demonstrate Freedom from Exotic Diseases that Threaten the Australian Pig Industry, such as Classical Swine Fever, by Post-Hoc Sampling of the Simulated Outbreaks*

Details of methods to calculate the sample size to detect an exotic disease incursion – or to demonstrate freedom – have previously been reported in milestone report 3. In summary, early in an outbreak many more herds are required to undergo surveillance than later (when the outbreak has spread across the landscape). Additionally, random surveillance across a region is less efficient than spatially targeted surveillance around the index case (e.g. radial surveillance). See Appendix 1 for details.

4. *Investigate the Influence of the Spatial Distribution of Feral Pigs, Family Groupings, and Age and Gender Distributions on the Methodology Developed*

See milestone report 3 for details. In summary, the aerial survey revealed a much smaller (lower density and distribution) feral pig population in the study area than in the previously simulated feral pig population. This resulted in simulated epidemics that generally died out several weeks or months after an incursion occurred (compared with previous epidemics that lasted several years). This suggests that the Fitzroy River basin population would only sometimes sustain an incursion of a transboundary disease such as classical swine fever, and that an incursion may never be detected due to a rapid fadeout of the disease (i.e. before passive surveillance could detect the outbreak). This is not true of all populations across northern Australia where population densities and distribution are much higher (for example, Cape York Peninsula). This is also unlikely to be true in the Kimberley region of Western Australia in all years because sometimes higher densities of feral pigs will occur (due to a good season, less flood related mortality, less culling and so on). However, results suggest that the assumption that these disease incursion would continue to spread in the absence of control efforts might not be valid. Our results suggest that a 'wait-and-see' (whilst conducting intensive surveillance) policy could be feasible where surveys demonstrate pig densities are low and biosecurity is sufficient to prevent disease spread from infected areas (e.g. public adherence to movement restrictions).

5. *Develop a Decision Making Tool for APL Use Following the Discovery of an Outbreak of Disease in Feral Pigs*

Details are provided in Appendix 2.

Project Outputs

1. Feral pig population distributions simulated and reported
   (Milestone report 3)

2. Disease Spread Model created, applied and results reported
   (Milestone report 2; *Veterinary Research* 2012; 43:3)

3. Sample size calculations to detect disease and demonstrate freedom
   (Appendix 1)

4. Sample size calculations to detect disease and demonstrate freedom
   (Appendix 1)

5. Decision support flow chart for APL use during a classical swine fever epidemic that involves feral pigs
   (Appendix 2)

Conclusion

This work has been completed.

Useful findings from the research are that according to simulation modelling, classical swine fever will not always establish for any length of time in feral pigs in some low density areas of northern Australia. The feral pig distributions in the Kimberley appear to be on the cusp. Some years disease may establish and spread for some time, but in other years, disease may not establish. For example, in the year we conducted aerial surveys the abundance and population structure was not sufficient to sustain infection for longer than 2-3 months. This was likely caused by a "big wet" season, which reduced the feral pig population substantially.

Our simulation modelling reveals that targeted, spatially explicit surveillance around an index case is the most efficient sampling strategy. Depending on the length of time an epidemic had been progressing, the densities of herds and the prevalence of disease, as few as two herds or as many as 230 herds were required to be surveyed before an epidemic was detected.

The research team thanks APL and pig producers for providing the funding to successfully complete this research.

Appendix 1 - Effective Surveillance Techniques Following a Classical Swine Fever Incursion in the Kimberley Region, Western Australia

Note: This represents Chapter 8 of a thesis to be submitted 31 March 2012 by Ms Edwina Leslie B.An.Vet.Biosci (Hons) for award of a PhD degree, Faculty of Veterinary Science, The University of Sydney

Introduction

Surveillance and control strategies can significantly influence the outcome of an infectious disease incursion (Klinkenberg *et al.*, 2005). The ability to detect infection and to prove disease freedom influences decision making. Factors such as domestic and international trade, economic and social loss and public health need to be considered (Boklund *et al.*, 2008; Domenech *et al.*, 2006; Thulke *et al.*, 2009). Surveillance methods can be assessed and improved through the use of epidemiological models. Such models, which include important disease spread parameters, can be used to map potential outbreaks and evaluate the effectiveness of surveillance and control strategies (Willeberg *et al.*, 2011). Over the last several decades models have been developed to simulate outbreaks of important transboundary diseases such as foot-and-mouth disease (FMD) and classical swine fever (CSF) to assist in preparedness planning for policy formulation, decision making and economic impact assessments (Harvey *et al.*, 2007). Garner *et al.* (2007) suggested that models are most useful prior to an outbreak to allow for contingency and resource planning, risk assessment and appropriate training for application during the event of a disease incursion.

Transboundary animal diseases (TAD's) such as CSF have been reported to the OIE from over 60 countries during the last 15 years (Donahue *et al.*, 2011). The highly contagious nature of the CSF virus and ability to spread rapidly amongst both domestic swine and wild pig species (*Sus scrofa*) has substantial economic and social impacts (Donahue *et al.*, 2011; Meuwissen *et al.*, 1999). Wild pig species refers to both feral pigs and wild boar in reference to the chapter. TAD's are diseases that are of economic, trade and/or food security importance for a large number of countries and can rapidly spread through susceptible populations reaching epidemic levels, irrespective of country borders (Otte *et al.*, 2004). The close vicinity of eastern Indonesia to northern Australia poses a risk for the reintroduction of CSF into Australia. Since its eradication in 1961, Australia has maintained its CSF free status through the establishment of biosecurity policies implemented by organisations such as the Australian Quarantine and Inspection Service (AQIS) and its Northern Australia Quarantine Strategy (NAQS) (AQIS, 2005). Animal Health Australia (AHA) developed the Australian Veterinary Emergency Plan for classical swine fever (Animal Health Australia, 2009) following eradication to provide contingency plans in the event of an outbreak and methods for use to provide proof of freedom. The reintroduction of CSF into Australia would have devastating impacts on the pig industry with the immediate loss of export markets taking effect, until eradication was completed (Animal Health Australia, 2009).

A variety of different diagnostic tests are available for CSF. The use of virus isolation in cell culture is classified as the 'gold standard' diagnostic tool for CSFV (Moennig, 2000). Alternative virus detection methods including enzyme-linked immunosorbent assays (ELISA's) (Greiser-Wilke *et al.*, 2007; Koppel *et al.*, 2007) and polymerase chain reaction (PCR) assay's (Greiser-Wilke *et al.*, 2007) have been developed.

Serosurveillance has been used as a diagnostic tool for monitoring both wild boar and domestic pig populations (Elbers *et al.*, 2000; Suradhat *et al.*, 2007). However, for early detection of CSF in

disease-free populations, Crauwels *et al*. (1999) found that the probability of detecting an epidemic in its early stages was low.

The closest islands of Indonesia to the Northern Territory and Western Australia are those in Nusa Tenggara Timur province. This chapter is an important component to the thesis because it demonstrates what would be the most appropriate surveillance and control strategies to adopt in the event of a CSF incursion in Australia. CSF in wild pig herds has been a recognised source of infection for many domestic herd outbreaks (Fritzemeier *et al.*, 2000; Lipowski, 2003). For example, in Germany during the 1990's Fritzemier *et al*. (2000) determined that 59% of index cases for CSF were a result of infected wild boar populations (Boklund *et al.*, 2008).

The wild pig (*Sus scrofa*) is an invasive species in Australia that impacts agricultural land and the environment while also competing with native and domestic animals (Cowled *et al.*, 2008a; Spencer *et al.*, 2005). Their role as a disease reservoir for infections such as CSF, brucellosis, swine vesicular disease virus and porcine reproductive and respiratory syndrome (PRRS) has been recognised in the scientific literature (Montagnaro *et al.*, 2010; Wyckoff *et al.*, 2009). In Australia, the wild pig population is predominantly distributed throughout the eastern and northern regions of Australia, with the densest population being located in north Queensland (West, 2008). Studies have suggested that wild pig herd structure can be divided into two categories, female mobs and solitary boars (Spencer *et al.*, 2005). This can influence disease transmission between herds. The high level of connectivity in densely populated areas can influence disease spread in the event of an outbreak and the persistence of CSFV (Cowled *et al*., 2012; Siembieda *et al*., 2011).

The objectives of this study were to analyse simulated CSF outbreaks in The Kimberley region to:

- Determine the effectiveness and efficiency of different surveillance strategies to detect and delineate infection, including random surveillance and two more practical methods of surveillance.

Methods

Model Description

A model developed by Cowled *et al*. (2012) was used to simulate wild pig inter herd CSF spread in time and space following an incursion in The Kimberley region, Western Australia. Within The Kimberley region the Fitzroy River area was selected as a representative population for disease introduction (Cowled *et al.*, 2012). The location of the study area can be viewed in Figure 8.1. The model was coded in MapBasic® and output data obtained from MapInfo® Professional Version 10.5 (Release Build 15, Pitney Bowes Software, Inc.).

The population dataset used in the model was derived from wild pig biology and ecology literature. The total number of herds simulated in the model was 5304. This number was generated using several data sources. Questionnaire surveys conducted by Cowled *et al*. (2009) and Woolnough *et al*. (2004) obtained information on pig distribution and densities. Results demonstrated that pigs were found across approximately 26,000km^2 of The Kimberley region. A 'core' habitat of 16,701km^2 was then identified and divided into polygons, classified as having low, medium or high pig densities. Published literature was then used to extract density and population parameters to use in the model allowing an estimate for the number of pigs herds present in the region (refer to Cowled *et al*. (2012) for details).

The model considered factors including pig density and movements, herd, age, social structure and the population distribution and habitat connectivity. Population parameters set in the model can be viewed in Table 8.1 where both herd types, solitary boars and female groups were considered. Each herd in the model was allocated an annual home range. Where applicable, probability distributions were placed around input parameters to account for uncertainty and variability (Table 8.1).

The model was able to simulate CSF outbreaks according to different scenarios. The spread of CSF could be investigated without the use of surveillance or control strategies by altering input parameters (refer to Figure 8.2). In addition, the model was also coded for surveillance techniques including radial and leap frog sampling (defined in section 8.2.5.2 and 8.2.5.3). The population was aggregated into cells where a 10km x 10km grid based system was applied to the study area under the assumption that three helicopters were available for surveillance. The model factored in a lag period of three days following disease detection in order to account for the allocation of resources and organisation of control measures. Finally, control options were coded into the model where culling or vaccination could be implementation in the event of an outbreak. If surveillance and control were used in a simulation, surveillance was activated first, and once completed, control was then initiated. Assumptions included four helicopters for control and the expected number of herds to be surveyed per team per day was 40.

Important baseline outputs to consider for this chapter included the number of susceptible, latent, infected, immune and dead pigs. It was assumed that once a pig was classified as immune it could no longer infect other pigs. The model also provided epidemic length as an output to allow comparisons between different simulation scenarios. When surveillance was simulated, reference could be made to the number of surveillance days and the number of cells being sampled. The inclusion of control in a simulation allowed reference to the total area covered (km^2) from beginning of surveillance to completion of control to enable comparison of surveillance effectiveness. For further model details refer to Cowled *et al.* (2012).

Model Limitation

Cowled *et al.* (2012) investigated available literature to determine input values and validate assumptions. Several limitations of this model need to be highlighted. The model assumed that infection was not spread through human movements of infected wild pigs or fomites, only simulating spread directly as a result from wild pig populations. As a result, we can expect these values to be lower estimates for the potential spread of CSF. As mentioned by Cowled *et al*. (2012), human population density is low in The Kimberley (0.1 person/km^2; ABS, 2011). This supported their assumption for limited CSF transmission via human movements.

It has been recognised in domestic pigs that the process of CSF infection can be acute, subacute or chronic (Dahle & Liess, 1992; Floegel-Niesmann *et al*., 2003). The presence of chronic CSF infection in wild pigs has yet to be documented (Artois *et al*., 2002; Cowled *et al*., 2012). Consequently, the model assumed only the presence of acute infection in wild pigs. The course of CSF infection is influenced by various factors including age, breed and environment (Floegel-Niesmann *et al*., 2003). Literature has suggested the presence of both high and moderate strains of CSF in Indonesia which can both result in acute forms of CSF infection (Frias-Lepoureau & Greiser-Wilke, 2002; Paton & Greiser-Wilke, 2003; Paton *et al*., 2000) aligning with this assumption.

Previous models investigating wild pigs have identified water sources as primary environmental consideration for pig population distribution. Choquenot *et al.* (1996) suggested that the distribution of feral pigs in eastern and northern Australia is largely dictated by the vicinity of watercourses and flood plains. The Kimberly region is a hot, dry area with the Fitzroy River catchment being identified

as an area with high feral pig abundance (Woolnough *et al.*, 2005). This model assumed pigs to be located within 2km of a water source where daily requirements for water access were also assumed. Previous models in the literature have demonstrated limitations regarding this parameter were a model developed by Milne *et al.* (2008) assumed pigs would only require water every 4 to 8 days in northern Australia.

Model Use

Using this model, two different approaches were taken to investigate disease surveillance. Approach 1 aimed to determine the number of herds required for sampling to detect disease following a CSF outbreak. Approach 2 aimed to compare three different surveillance techniques, simple random sampling (SRS), radial and leap sampling and their effectiveness in detecting and delineating CSF infection. For both approaches time points were compared for disease detection on day 42 (6 weeks), 168 (6 months) and 365 (1 year) (Figure 8.3). These time points were selected to demonstrate the extent of a CSF outbreak following delays in disease detection. Hone and Pech (1990) using a model to investigate FMD estimated minimum time to detection of an FMD outbreak with a sample size of 200 to be 35 days. Cowled *et al.* (2012) utilised similar estimates with a minimum of 42 days for disease detection. For clarity, when a reference is made to infected pigs, this should be taken to mean latent, infectious and immune pig herds. It was assumed that detection of CSF index herds was via passive surveillance.

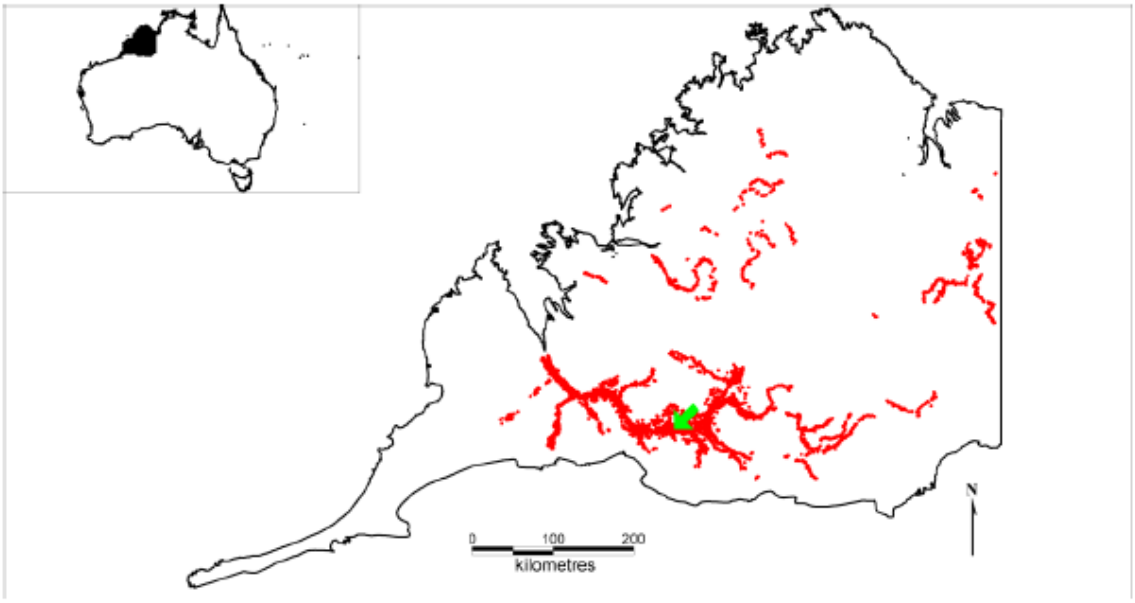

Figure 8.1: Wild pig herd distribution in The Kimberley region, Western Australia. Map obtained from Cowled et al (2011). The inset identifies the location of The Kimberley region in Western Australia. Red dots represent pig herds simulated within known Wild pig distributions. The green arrow identifies the introduction site for classical swine fever for all simulations.

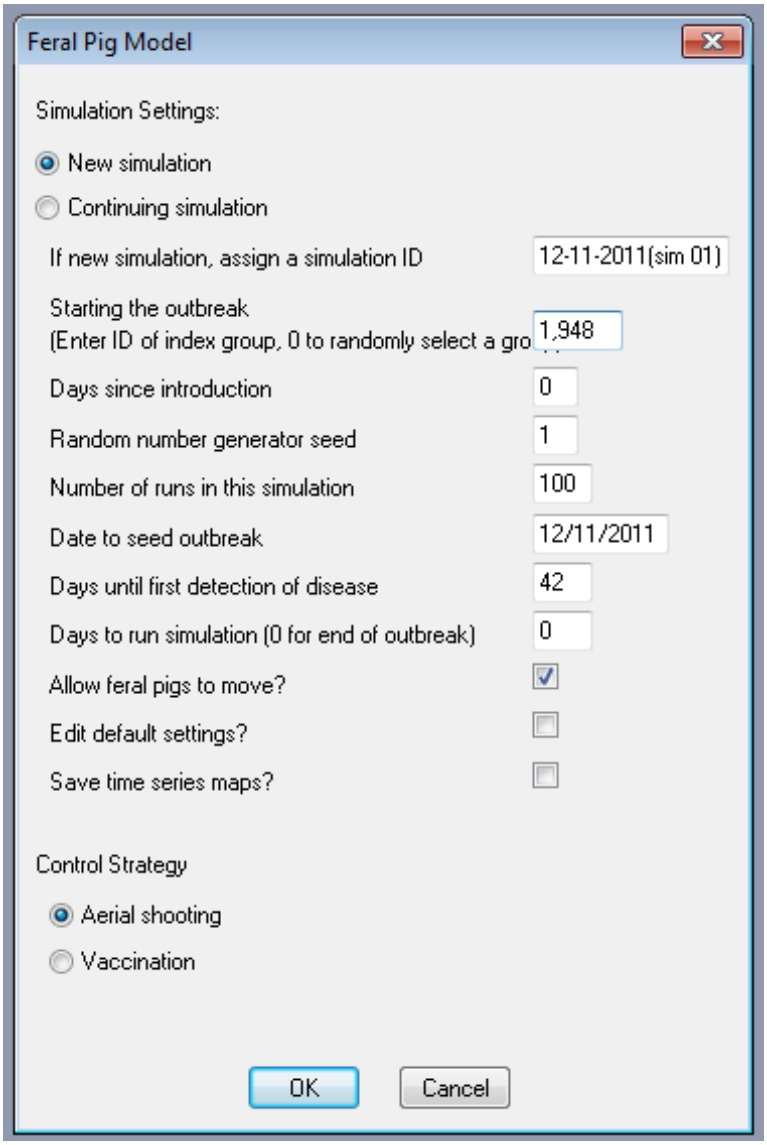

Figure 8.2: Start up screen for wild pig model to simulate a CSF outbreak in The Kimberley region, Western Australia. Input parameters can be changed according to the scenario being simulated.

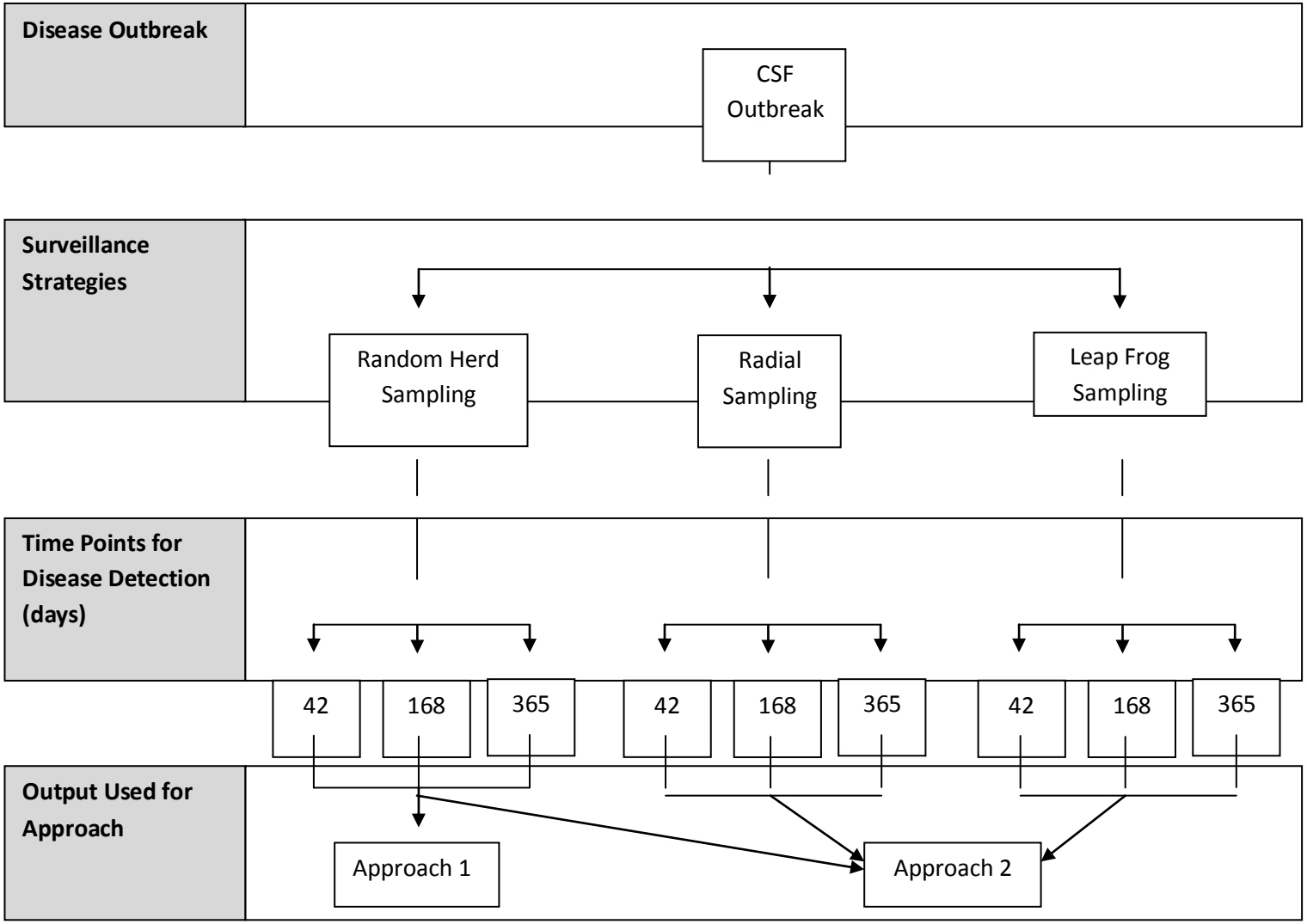

Figure 8.3: Disease outbreak scenarios simulated by a between herd model investigating classical swine fever transmission and spread developed by Cowled et al (2011) using the pig population in The Kimberley region, Western Australia.

Table 8.1: Ecological Parameters taken from Cowled et al (2012) for between herd model.

Parameter	Estimate	High	Low	Probability Distribution
Herd size[1]	7	45	5	Beta Pert
Pig density (km²)	1 - 3	-	-	-
Male home range (km²)	12	31.2	3.7	Triangular
Female home range (km²)	7	19.4	2.5	Triangular
Male daily home range (km²)	1.5	9.99	0.2	Triangular
Female daily home range(km²)	0.9	3.6	0.06	Triangular
Male daily linear movements (km²)	1	2	0.1	Triangular
Female daily linear movements (km²)	0.7	1.8	0.1	Triangular

[1]12% herds were assumed solitary male boars and the remaining distributed into female groups.

Approach 1: Detecting Disease Following CSF Outbreak Using Estimated Population Data

Approach 1 modelled an outbreak with a single entry point in a high pig density area with no control measures implemented. One hundred simulations were run with disease detection occurring at 42, 168 and 365 days post incursion to determine the extent of CSF spread. A total of 5304 herds were present in the model, representing all herds across The Kimberley under the assumption of 1-3 pigs/km^2 (refer to Cowled et al (2012) for more details). Comparisons were then made to determine the success of a random surveillance strategy to detect infection at these time points. The model was simulated at the herd level under the assumption of approximately 7 pigs per herd (Cowled *et al.*, 2012). At each time point for each simulation, a SRS of 290 herds were selected according to a sample size calculation to detect disease with 95% confidence level assuming 1% herd prevalence and a finite population (Equation 8.1; Thrusfield, 2007. Herds were selected using a random number generator (PopTools Version 3.2.5, Hood, 2011). The same random herd sample was used for all simulations and time points to allow comparisons to be made across different time points.

$n = [1-(\alpha)^{1/D}] [N – (D-1)/2]$……..Equation 8.1

Where: n = required sample size

α = 1- confidence level (0.05)

D = estimated minimum number of diseased animals (5304*0.01)

N = population size 5304.

Approach 2: Comparisons between Surveillance Techniques - Simple Random Sampling, Radial and Leap Frog Sampling

Approach 2 enabled the comparison of surveillance techniques including SRS, radial and leap frog sampling that could be implemented in the event of a CSF incursion. Simulations were run until the end of an outbreak and comparisons made for each surveillance technique at different time points to determine their effectiveness in detecting and delineating disease (Figure 8.3). Culling was used as the control measure for each surveillance technique, implemented following surveillance completion.

Simple Random Sampling (SRS)

Baseline information was initially generated with simple random sampling used as the surveillance technique. Simulations were run until the end of an outbreak with surveillance initiated following disease detection at time points 42, 168 and 365 days. One hundred simulations were run with 15 simulations randomly selected for comparisons between each time point. Fifteen simulations were selected based on the proportion of simulations where disease was delineated with the presence of a buffer.

In a pilot assessment, 1 out of 5 simulations was not delineated. As a result, using a binomial confidence interval with 95% confidence and 10% precision, a sample of 15 was deemed adequate. The spread of CSF from these simulations was mapped taking into consideration latent, infectious, immune and dead pigs. A total of 290 randomly selected herds (calculated based on 1% prevalence and 95% confidence level; Equation 8.1) were then identified on each map. It was then determined

whether disease was detected and delineated during the outbreak based on locations of sampled and diseased herds. Disease detection in the population was assumed if a single herd that was infected was sampled using the surveillance strategy. Disease delineation was assessed assuming a buffer zone of 10 km x 10km (as recommended by (Animal Health Australia, 2009). Delineation was assumed to have occurred if the buffering of infected herds identified during surveillance was sufficient to include all other infected herds within the study population. Delineation was further assessed with the removal of the buffer zone to determine whether delineation was successful.

Radial Surveillance

Radial sampling is a surveillance technique that uses an index herd as the starting point. In the model, this sampling technique was initiated by a randomly selected index cell. Then all cells immediately surrounding the index cell that contained wild pig herds underwent surveillance progressively according to resource constraints. Within each selected surveillance cell, sufficient herds were sampled to detect disease with 95% confidence, assuming 1% prevalence (Equation 8.1; Thrusfield, 2007). If an infected cell was found then the sampling area was increased to adjacent cells until no infected cells were detected. Surveillance stopped when all scheduled cells had been sampled. Sampling occurred day by day under realistic resource constraints. For comparison with leap frog surveillance and simple random sampling, 100 simulations were run till the end of an outbreak with disease detected at time points 42, 168 and 365 days (Figure 8.3). The simulations were run until surveillance was completed.

Leap Frog Surveillance

Leap frog sampling followed the same initial stages as radial sampling, where an index cell was randomly selected. The difference is when there is the detection of an infected cell, surveillance occurs in every second cell away from the index cell. Using the concept of the nearest neighbour, surveillance begins at the level k = 2 and continues at k = 4, k = 6 and so on until all cells are negative (Beyer *et al.*, 1999; Dubé *et al.*, 2009; Raine *et al.*, 2009). Using Figure 8.4 as an example, the index cell (yellow) is first identified as infected with sampling first occurring in the red cells, following this pattern. The motivation for developing this surveillance method was the increased efficiency for widespread CSF outbreaks. For comparison with radial surveillance and SRS, 100 simulations were run till the end of an outbreak with disease detected at time points 42, 168 and 365 days (Figure 8.3). The simulations were run until surveillance was completed.

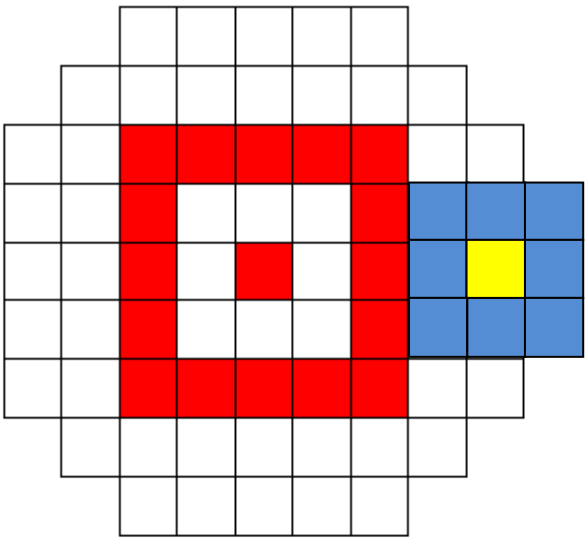

Figure 8.4 (above): Leap frog sampling approach used as a surveillance technique to identify infected wild pig herds. The index cell (yellow) was identified as infected with classical swine fever. Surveillance is then initiated with sampling at the level of k = 2 continuing away from the index cell.

Statistical Analysis

General descriptive analysis was conducted on each scenario using Genstat 11th Edition (PC/Windows XP, 2006, VSN International Ltd., Hemel Hempsted, UK).

Approach 1

The infection status of randomly selected herds (n = 290) were determined in each simulation and for each time point (42, 168 and 365 days). A detected herd was an infected herd identified in the surveillance sample. A pre-determined list of herds was used to consecutively identify in a binary process (1 = yes or 0 = no) whether a herd was infected. This process aimed to identify the number of herds it took to sample before the first case of infection was detected. This was then repeated for each time point.

Approach 2

To assess the effectiveness of the different surveillance techniques, a chi-squared test was performed to determine significant differences between herd type (herd types: susceptible, latent, infectious, immune or dead) at different time points (42, 168 and 365 days) during outbreak duration. A z-test using a Bonferroni adjustment was used to compare which of the herd types and time points were significantly different due to comparisons of multiple outcomes. Data were analysed for normality to fulfil the assumptions of a z-test. Significance was indicated by *P*-values < 0.05. Using descriptive statistics to determine the similarities between simulations at each time point, a sample of 15 simulations for each time point was selected for comparison. For each simulation, the extent of the outbreak was mapped using MapInfo Professional Version 11 displaying latent, infectious, immune and dead herds. Following this, 290 randomly selected herds were then identified on the map. Using visual observations and recordings of the number of infected pigs (latent, infectious and immune) detected through SRS, it was determined whether at least 1 infected herd was detected with SRS and whether disease was delineated with the presence and absence of a buffer zone.

Results

Approach 1

The model output obtained for approach 1 demonstrated that in the early stages following an incursion (< 6 weeks post incursion) a minimum of 28 herds needed to be sampled to detect disease (Table 8.2). In the first few weeks, only the presence of latent and infectious herds were detected. Immune pigs were not detected till later in the outbreak (> 6 months). The number of infected herds peaked at day 290 post incursion (Figure 8.5).

Table 8.2: Model estimates following a typical incursion of classical swine fever in the Kimberley region, Western Australia with random herd selection to detect disease (L = latent, I = infectious, Im = Immune; standard deviation displayed in table).

Day post incursion	No. of herds sampled to detect disease	No. of days sampling	Latent, infectious, immune,	No. Infected Pigs (L, I and Im)	No. deaths (no. herds)
42	28	4.8	L, I	75 ± 13.3	5
168	10	1.9	L, I, Im	259 ± 39.6	30
365	2	0.3	L, I, Im	525 ± 94.9	82

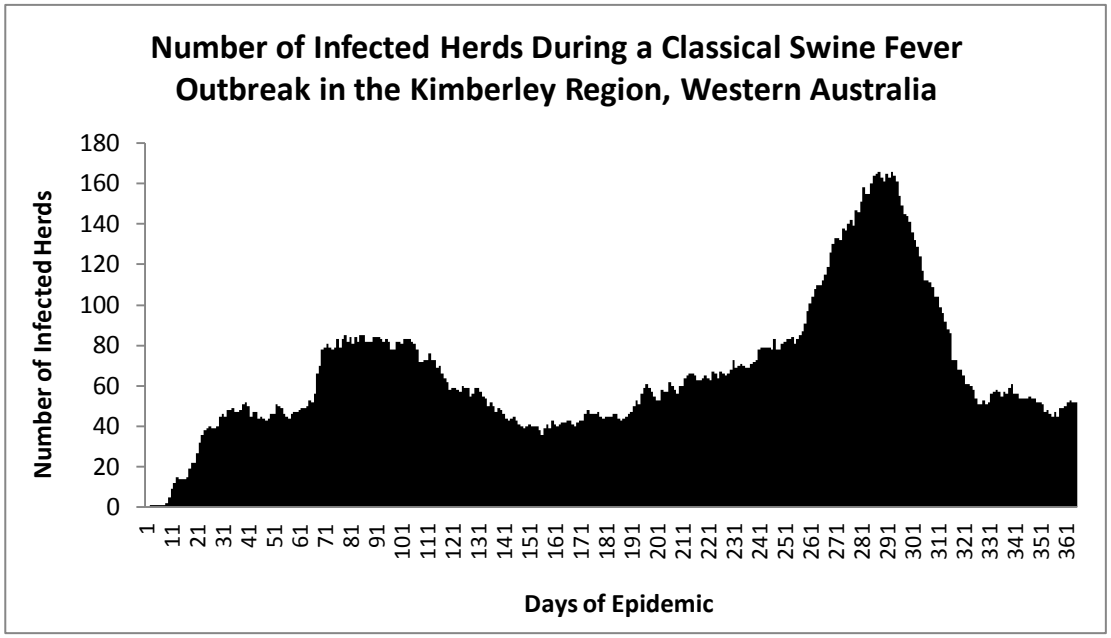

Figure 8.5: An epidemic curve for one Classical Swine Fever outbreak simulation in wild pigs in The Kimberley region, Western Australia (n = 290).

Approach 2

Simple Random Sampling of Herds (SRS)

Statistical analysis of the 290 randomly selected herds demonstrated that the number of infected, susceptible, immune and dead herds significantly differed between time points for disease detection at 42, 168 and 365 days (Table 8.3). The number of latent and infectious herds at these time points following the outbreak did not vary significantly (Table 8.3). Demonstrated from Table 8.4 it can be seen that in 86.7% of simulations (13/15), disease was detected and delineated through the use of SRS with a buffer zone around selected herds. When a buffer zone was not applied, infection was detected, however not delineated.

Table 8.3: Model estimates following classical swine fever introduction identifying extent of spread at time points 42, 168 and 365 post incursion across the Kimberley region, Western Australia, 2011 (standard deviation displayed in table).

Model Duration (days)	Time Point (days)	Susceptible Herds	Latent Herds	Infectious Herds	Immune Herds	Dead from Disease	Average Total Infected[1]
365	42	5209 ± 13.8[a]	16 ± 6.2 [a]	51 ± 9.0[a]	8 ± 2.4[a]	20 ± 4.5[a]	75 ± 13.3[a]
	(min, max)	5168-5239	3-41	29-73	2-14	8-34	
	168	4891 ± 57.4[ab]	15 ± 6.5[a]	45 ± 16.6[a]	198 ± 28[b]	154 ± 21.3[ab]	259 ± 39.6[ab]

[a,b,c] Within a column, values without a common superscript letter differ significantly ($P < 0.05$); [1]Total infected refers to the number of latent, infectious and immune herds

Table 8.4: Model estimates comparing different model simulation runs following a classical swine fever introduction identifying extent of spread at time points 42, 168 and 365 post incursion using random selection of 290 herds across the Kimberley region, Western Australia, 2011 (Y = yes; N = No).

Simulation	Day	Disease Detected	Disease Delineated (with buffer)	Infected Herds Outside Buffer (no. latent, infectious, immune)	Disease Delineated (no buffer)	No. Detected Infected Herds (no. latent, infectious, immune)	Susceptible	Latent	Infectious	Immune	Dead
75	42	Y	Y	-	N	0,4,0	5202	9	51	14	25
	168	Y	Y	-	N	1,1,9	4941	18	36	179	130
	365	Y	Y	-	N	0,1,22	4608	5	22	370	299
3	42	Y	Y	-	N	1,2,0	5218	14	48	9	15
	168	Y	Y	-	N	1,2,5	4872	18	69	172	173
	365	Y	Y	-	N	2,4,21	4297	18	96	443	450
35	42	Y	Y	-	N	0,2,1	5211	7	55	8	23
	168	Y	Y	-	N	1,0,5	4882	21	36	211	154
	365	Y	N	1,4,0	N	0,0,15	4498	1	19	408	378
28	42	Y	Y	-	N	1,2,0	5211	16	53	6	18
	168	Y	Y	-	N	1,1,7	4946	11	15	187	145
	365	Y	Y	-	N	4,12,18	4708	0	31	288	277
49	42	Y	Y	-	N	0,2,0	5217	20	45	7	15

	168	Y	Y	-	N	0,2,7	4904	10	26	205	159
	365	Y	Y	-	N	2,5,21	4284	43	67	476	443
17	42	Y	Y	-	N	0, 1,1	5208	14	63	8	11
	168	Y	Y	-	N	0,0, 5	5010	2	11	171	110
	365	Y	Y	-	N	1,3,0	4513	15	69	381	326
79	42	Y	Y	-	N	0,2,1	5196	17	57	8	26
	168	Y	Y	-	N	0,2,7	4965	16	38	153	132
	365	Y	N	1,1,8	N	1,0,20	4518	7	9	430	340
46	42	Y	Y	-	N	0,2,0	5227	10	47	9	11
	168	Y	Y	-	N	3,2,11	4876	14	46	212	156
	365	Y	Y	-	N	1,4,26	4193	22	80	541	468
81	42	Y	Y	-	N	0,0,1	5209	18	44	9	24
	168	Y	Y	-	N	0,1,9	4872	14	34	227	157
	365	Y	Y	-	N	3,6,24	4286	25	95	486	412
25	42	Y	Y	-	N	1,1,1	5210	17	55	4	18
	168	Y	Y	-	N	2,2,5	4966	15	30	161	132
	365	Y	Y	-	N	0,0,22	4520	14	54	389	327
12	42	Y	Y	-	N	1,1,0	5217	15	45	8	19

	168	Y	Y	-	N	0,3,8	4878	13	41	221	151
	365	Y	Y	-	N	1,3,30	4230	17	52	550	455
67	42	Y	Y	-	N	2,1,1	5230	14	41	7	12
	168	Y	Y	-	N	0,3,5	4914	12	37	198	143
	365	Y	Y	-	N	0,2,23	4395	14	56	448	391
93	42	Y	Y	-	N	0,2,0	5199	16	55	13	21
	168	Y	Y	-	N	1,2,4	4892	10	42	204	256
	365	Y	Y	-	N	0,6,24	4309	9	43	532	414
22	42	Y	Y	-	N	0,1,0	5229	9	43	9	14
	168	Y	Y	-	N	3,0,10	4882	12	39	206	165
	365	Y	Y	-	N	2,3,0	4430	25	80	383	386
78	42	Y	Y	-	N	0,2,0	5216	16	44	5	23
	168	Y	Y	-	N	2,4,9	4793	23	72	212	204
	365	Y	Y	-	N	1,4,25	4179	16	63	523	523

Surveillance Strategy Comparison with Disease Detection at Day 42

Surveillance length did not vary significantly between radial and leap frog sampling when infection was detected at day 42, 168 and 365 (P = 0.208 and P = 0.175, respectively; Table 8.5). A greater number of cells were sampled when adopting a leap frog approach. At day 42 of disease detection, the epidemic lasted on average 31 days longer when using leap frog sampling in comparison to radial sampling. This was not significantly different (P = 0.927; Table 8.5). In terms of the number of total infected herds, leap frog sampling resulted in a higher number of infected herds during the course of an outbreak in comparison to radial sampling, however this was not significant (P = 0.715; Table 8.5). With the application of a buffer, all infection was delineated when SRS was used.

Surveillance Strategy Comparison with Disease Detection at Day 168

A greater total area was covered following surveillance and control using a radial sampling approach (Table 8.5). The number of cells sampled was equivalent across both sampling techniques. At day 168, radial sampling resulted in a maximum epidemic length of 489 days, 56 days longer in comparison to leap frog sampling. There was one isolated case (simulation 28) that lasted until day 1,371 post incursion. However the remaining simulations were ≤ 489 days. The maximum epidemic length for leap frog sampling was 433 days. The length of an epidemic and the number of infected herds did not significantly vary between sampling type (P = 0.222 and P = 0.831 respectively; Table 8.5). With the application of a buffer, all infection was delineated when SRS was used with infection detected at day 168.

Surveillance Strategy Comparison with Disease Detection at Day 365

Surveillance, using a radial approach, saw an average of 106 cells sampled with disease detected at 365 days (Table 8.5). This was one cell greater when comparing with leap frog sampling. At day 365 post incursion, radial sampling resulted in an average epidemic length of 500 days with a maximum of 663 days reached (Table 8.5). Leap Frog sampling resulted in an average epidemic length of 498 days, with a maximum of 792 days. These did not significantly differ (P = 0.999; Table 8.5). The total number of infected herds did not differ significantly between sampling approaches (P = 0.963; Table 8.5). Although a greater total area was covered using a radial sampling approach, this was found to be similar between sampling types when disease was detected at 365 days (P = 0.991; Table 8.5). Overall if we compare the total area covered following disease detection at different time points, the area covered was significantly less if infection was detected at day 42 in comparison to day 365 (P < 0.001; Table 8.5). When SRS was conducted, delineation of infection was not successful even with the application of a buffer zone.

Table 8.5: Model estimates following classical swine fever detection at day 42, 168 and 365 with radial and leap frog sampling for surveillance and control of disease across the Kimberley region, Western Australia, 2011 (standard deviation displayed in table).

Disease Detection (day)	Sampling Type	Average Epidemic length (days)	Average Dead from Disease (no. herds)	Average Total Area covered (km^2)	Average Total Infected (no. herds)	Average Culled (no. herds)	Surveillance Length (days)	No. cells sampled
42	Radial	159 ± 31.6^{ax}	74 ± 18.3^{ax}	377 ± 103.5^{ax}	183 ± 40.8^{ax}	411 ± 30.7^{ax}	8 ± 0.6^{ax}	38 ± 3.6^{ax}
	Leap Frog	166 ± 29.1^{as}	80 ± 20.6^{as}	414 ± 112.8^{as}	199 ± 44.5^{as}	419 ± 30.3^{as}	9 ± 0.7^{as}	39 ± 3.6^{as}
168	Radial	321 ± 115.2^{axy}	220 ± 60.0^{axy}	1,522 ± 894.1^{ay}	538 ± 144.1^{axy}	609 ± 31.5^{ax}	13 ± 0.9^{ax}	68 ± 5.6^{ax}
	Leap Frog	306 ± 39.3^{as}	218 ± 39.5^{ast}	1,463 ± 397.9^{at}	534 ± 92.8^{ast}	609 ± 31.3^{as}	14 ± 1.4^{as}	68 ± 5.7^{as}

^{a,b} Within a column comparing sampling type for each time point, values without a common superscript letter differ significantly ($P < 0.05$)
^{x,y,z} Within a column comparing disease detection day for radial sampling, values without a common superscript letter differ significantly ($P < 0.05$); ^{s,t,u} Within a column comparing disease detection day for leap frog sampling, values without a common superscript letter differ significantly ($P < 0.05$).

Discussion/Conclusion

Disease Detection

The earlier disease detection can occur, the more effective control strategies can be in containing an outbreak (Klinkenberg *et al.*, 2005). Early detection can also help to minimise costs by reducing the amount of resources required for control (Nusser *et al.*, 2008). At day 42 it was found that 28 herds were required for sampling to detect disease using a random sampling approach. Although this is a higher sample size in comparison to 130 pigs at 168 days and 24 pigs at 365 days, by detecting and containing an outbreak it the early phases, the impacts can be reduced. This was demonstrated in the model whereby when there was a delay in disease detection, an increase in the number of infected herds was seen. On day 42 of detection, there was the presence of only 75 infected herds. By day 365, 525 herds were infected, identifying a much greater spread of disease. This is further supported by (Shirley & Rushton, 2005a) who determined that during a FMD outbreak, if disease had been detected two days earlier, the eventual epidemic would have been half its size. It needs to be noted that there are limitations to simulation modelling due to variability and uncertainty of input parameters. Their results need to be considered along with other information sources for use in decision making (Clifford *et al.*, 2011). Rather than focusing on specific output values, the trends in the results should be the focus (Clifford *et al.*, 2011).

Pig density is an ecological factor that can determine the potential for disease to be maintained and transmitted within a population (Doran & Laffan, 2005). Authors such as Pech and Hone (1988) have also discussed this effect in relation to threshold densities. They suggested that for disease to persist in a semi-arid Australian environment a threshold of 2.3-14km^2 was required, in association with FMD. For CSF to be eradicated from a population, transmission needs to be reduced to a level where the virus can no longer maintain itself (Weesendorp *et al.*, 2010).

The model utilised for this chapter used estimated pig herd data based on several data sources (refer to section 8.2.1). This identifies a need for further research into the current pig population based in The Kimberley region as this is such as important parameter regarding CSF transmission. Similarly, Cowled *et al.* (2012) identified the spatial structure and behaviour of wild populations as an important factor to be considered for wildlife disease management. The limitations of surveillance and control, in that they are only able to reach a proportion of a given population, indicates that more efficient strategies need to be developed to maximise the use of resources (Thulke *et al.*, 2009). Investigations into herd structure and the interactions of wild pig populations have been conducted (Cowled *et al.*, 2008a; Spencer *et al.*, 2005). It has been confirmed that sows will accept multiple matings and for lone boars to travel distances of up to 2km daily and with home ranges of up to 10km^{2,} facilitating potential disease spread (Cowled *et al.*, 2012; Spencer *et al.*, 2005).

Surveillance Strategies

The use of a SRS surveillance approach was found to be effective across days 42, 168 and 365 post incursion for detection of infection. When a 10km x 10km buffer zone was in place, delineation of infection was successful following disease detection at days 42 and 168. By day 365 of an epidemic, a buffer zone did not delineate all infection. Moreover, in the absence of a buffer zone, delineation of infection was not successful in any simulation. Although a SRS approach was effective in detecting disease, it was suboptimal in terms of resource allocation and time. Wildlife disease surveillance is more complex than in livestock diseases. Due to limited knowledge on species abundance, distribution and susceptible populations at risk, thereby necessitating the consideration of alternative sample designs as effective means of tracking disease spread (Thulke *et al.*, 2009). Similar to that of Nusser *et al.* (2008), a SRS approach was used to develop a baseline in which alternative surveillance

strategies were compared against. This further supported that SRS was not a practical strategy for effective allocation of resources in terms of disease detection in wildlife populations.

The use of radial and leap frog sampling allowed a more targeted approach to be utilised in the event of an outbreak. By adapting sampling techniques based on a specific epidemic event, this can assist in improving effectiveness (Thulke *et al.*, 2009). On day 42 post incursion, the most effective surveillance strategy to contain an outbreak was radial sampling. During the early stages of the modelled outbreak, disease dispersal was limited. By implementing a surveillance area around the index cell, this allowed delineation within an average of 159 days. Disease detection on day 168 demonstrated that further spread of disease had occurred through the population. In this situation, the most appropriate sampling technique was leap frog sampling. This allowed for a reduction in total area covered and a lower average for total herds infected. Although surveillance duration was slightly longer, by an average of only 1 day, this was not significant (Table 8.5). Following a year of disease spread before detection of infection, when comparing radial and leap frog sampling, the total area covered during surveillance and control was greater for radial sampling. Surveillance length was slightly greater for leap frog sampling, however, not significantly different (Table 8.5). The total number of herds infected and dead were similar ($P = 0.963$ and $P = 0.965$, respectively). For this time point, the use of leap frog sampling would be more appropriate. Although similar figures were obtained from the model for both radial and leap frog approaches, the ability for leap frog surveillance to detect the extent of an outbreak more rapidly due to the process of sampling, this can be seen as a more suitable approach when an outbreak has not been detected for a lengthy period and there has been a greater level of spread through a population.

We can conclude from this analysis that, due to the complexity of wildlife population dynamics and herd behaviour, a targeted approach to surveillance needs to be conducted for the effective use of resources and time. The use of SRS can be seen as suboptimal, although disease was detected. The detection and containment of an outbreak needs to be as early and rapid as possible. Using a more situation-based surveillance approach and accounting for disease distribution and the time period over which an epidemic had occurred was the best way to approach the selection of a control strategy. Radial and leap frog surveillance have demonstrated their ability to improve the effectiveness of disease detection at various stages of a disease outbreak. These sampling strategies have been able to model potential outcomes following a CSF incursion and their ability to minimise impacts. These results can be used to assist in the allocation of resources, decision making and improving the efficiency of intervention strategies.

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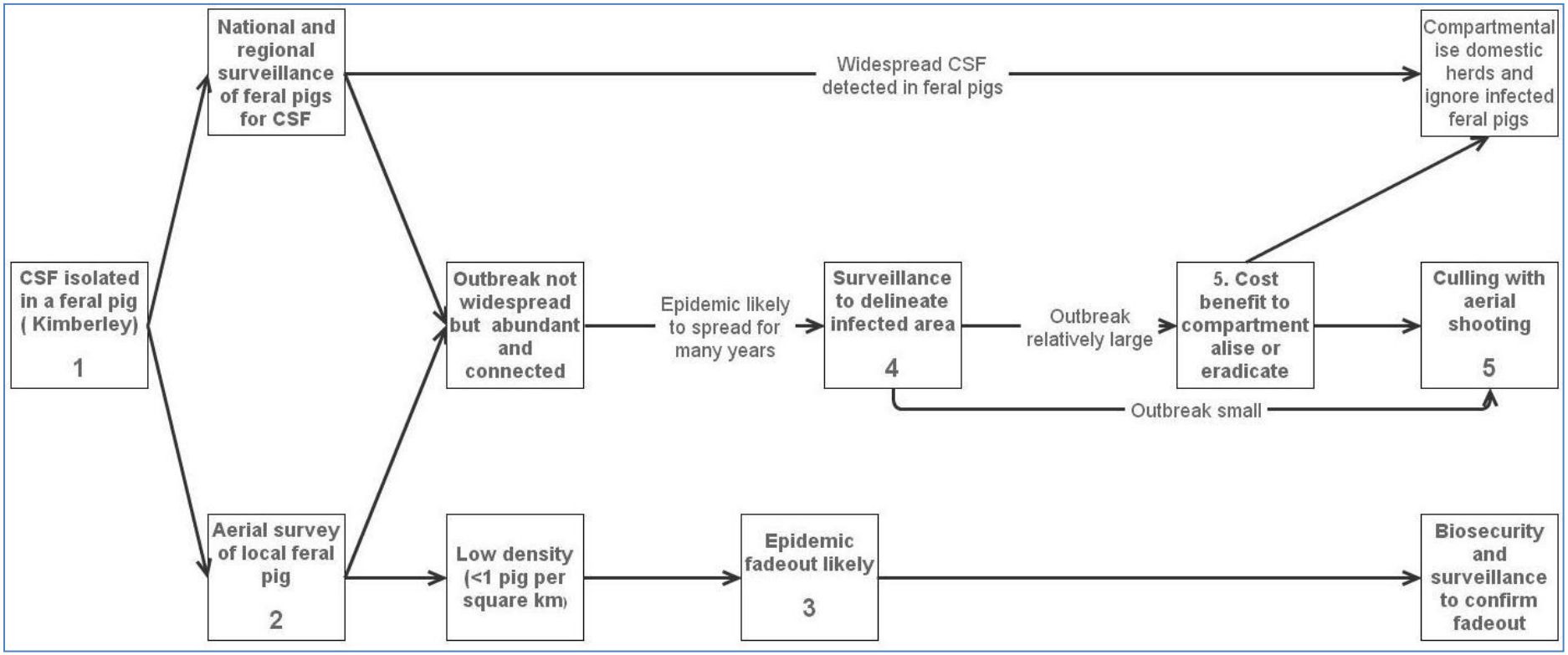
Appendix 2 - Decision Support Flow Chart for APL Use During a CSF Epidemic in Feral Pigs



Explanatory Notes (Numbers on Flow Diagram Correspond to the Text below)

Background: APL has previously signed a deed under the emergency animal disease response agreement to fund 50% of the costs of responding to an outbreak of a Classical Swine Fever (CSF). This response may reach 1% of GVP (approximately $8 million) but can only exceed this with further agreement. An initial response will be shaped by an emergency animal disease response plan (EADRP) (written by the state department of agriculture where the outbreak has occurred in consultation with other parties such as industry). This document will be written in haste in real time and will direct the response to the epidemic.

APL has an opportunity to influence the EADRP at several levels, but this will occur formally through membership of the consultative committee on emergency animal diseases[i] (CCEAD) and the national management group (NMG). These groups have technical and management oversight of emergency animal disease responses respectively. Practically, APL's membership of the CCEAD and NMG will allow it to contribute to decisions on the EADRP. However, APL will require a good understanding of the technical issues in order to make excellent decisions that may bind members to significant future cost recovery levies. This document is designed to provide a simple technical tool to assist this decision making in response to a potential CSF outbreak in feral pigs.

1) Assuming there is a single passive surveillance detection of an infected feral pig herd

2) Aerial surveys using slow, low flying helicopters are a well documented means of counting and observing distributions of wild animals. Peter Fleming and John Tracey at NSW DPI are very experienced in this method (Fleming and Tracey 2008)[ii] (Fleming and Tracey, 2008).

3) Epidemic fadeout occurred almost invariably during simulation modelling of CSF in feral pigs where densities were less than 1 pig km^{-2} (Cowled et al 2012)[iii]. That is, simulated epidemics of CSF were not sustained longer than a few weeks when introduced to low density populations.

4) An effective surveillance technique was progressively expanding the infected area using a 10x10 km grid template. 4-5 pigs from each herd within a grid cell can be sampled. Assume approximately 60-80% of pig herds could be sampled, but surveillance is discontinued when disease is detected. This allows effective delineation of the epidemic area. See Cowled et al. (2012).

5) Saunders and Bryant (1988)[iv] demonstrated that 80% of feral pigs in a local area can be shot from a helicopter. Cowled et al. (2012) used simulation modelling to demonstrate that various combinations of culling proportion and culling zone width will lead to disease fadeout (e.g. 60% of pigs culled over a 20 km zone around an epidemic).

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[i] http://www.daff.gov.au/animal-plant-health/animal/committees/ccead

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