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Project Description	African swine fever (ASF) represents a significant threat to the Australian pork sector and the economy in general. Estimates of the economic damages from a large multi-state outbreak of ASF in Australia exceed \$A2 billion. ASF outbreaks are widespread and increasing in number in Asia and Europe. Although ASF is not present in Australia, detections of ASF viral fragments in undeclared pork products intercepted at the Australian border and the recent spread of the disease to neighbouring Papua New Guinea demonstrate the significance of the threat.						
	The AADIS model (Bradhurst et al., 2015), simulates the spread and control of contagious emergency animal diseases such as foot-and-mouth disease. The ability to evaluate different outbreak scenarios in time and space, and trial various control measures, assists the development of animal health policy.						
	This project expanded the AADIS modelling framework to simulate the potential spread and control of ASF in Queensland domestic and feral pig populations. Of particular interest was the epidemiological interface between domestic and feral pigs and the potential role of ASF-infectious feral pig carcasses in transmission. The upgraded model will provide a useful decision support tool to assist with preparedness and planning for ASF outbreaks.						

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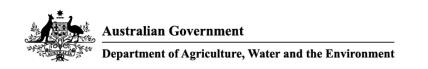


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1.1	Incorporation of contributions and revisions from the project team	R. Bradhurst	3 September 2021
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1.3	Submission for SRP review	R. Bradhurst	6 September 2021
1.4	Revisions stemming from SRP external review and DAWE internal review	R. Bradhurst	3 November 2021

## **Glossary of Acronyms**

Acronym	Definition
AADIS	Australian Animal Disease Spread (model)
ABARES	Australian Bureau of Agricultural and Resource Economics and Sciences
ABM	Agent-based model
APIQ	Australian Pork Industry Quality (Assurance Program)
ARP	At-risk premises (located inside RAs)
ASF	African swine fever
ASFV	African swine fever virus
AUSVETPLAN	Australian Veterinary Emergency Plan
BQ	Biosecurity Queensland, Department of Agriculture and Fisheries
CA	Control area
CEBRA	Centre of Excellence for Biosecurity Risk Analysis, University of Melbourne
DAWE	Department of Agriculture, Water and the Environment
DCP	Dangerous contact premises
DCPF	Dangerous contact processing facility
DES	Deserts and xeric shrublands (wildlife region)
EBM	Equation-based model
FMD	Foot-and-mouth disease
FVAS	Faculty of Veterinary and Agricultural Sciences, University of Melbourne
IA	Infected area
IP	(declared) infected premises
MED	Mediterranean forests, woodlands and shrubs (wildlife region)
MLC	Medium to large commercial (herd)
MON	Montane grasslands and shrublands (wildlife region)





NE	North-east (mega region)
ODE	Ordinary differential equation
OIE	World Organisation for Animal Health
PK	Pig keeper (herd)
PL	Pastoral (mega region)
POR	Premises of relevance (located inside CAs)
QDAF	Queensland Department of Agriculture and Fisheries
RA	Restricted area
RP	Resolved premises
SC	Small commercial (herd)
SE	South-east (mega region)
SEIRD	Susceptible Exposed Infectious Recovered Deceased (model)
SGT	Specialist gene transfer (herd)
SH	Smallholder (herd)
SP	Suspect premises (clinical disease has been reported)
SW	South-west (mega region)
TEF	Temperate broadleaf and mixed forests (wildlife region)
TES	Temperate grasslands, savannas and shrublands (wildlife region)
TP	Trace premises (identified by tracing activities)
TRF	Tropical and subtropical moist broadleaf forests (wildlife region)
VLC	Very large commercial (herd)



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# Modelling the spread and control of African swine fever in domestic and feral pigs

Technical report for CEBRA project 20121501 prepared for the Department of Agriculture, Water and the Environment

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#### 1 EXECUTIVE SUMMARY

African swine fever (ASF) is a contagious and deadly disease of domestic and wild pigs (*Sus scrofa*). An outbreak of a highly virulent strain in Georgia in 2007 has spread to much of Europe and Asia. Recent outbreaks have occurred in the neighbouring countries of Indonesia, Papua New Guinea, and Timor-Leste (Penrith, 2020; Barnes et al., 2020). Although ASF has never been reported in Australia, detection of viral DNA in undeclared pork and pork products seized at the Australian border confirm that it is a significant threat to the Australian pig industry. It has been estimated that a large multi-state outbreak of ASF could impact the Australian economy by up to A\$2 billion (ACIL Allen, 2019).

The risk to livestock from emergency animal disease is often compounded by complex ecological and epidemiological interplay between susceptible livestock, susceptible wild/feral animals, and the environment (Huyvaert et al., 2018). If ASF were to enter the Australian feral pig population it is uncertain whether it would establish and pose an ongoing threat to domestic pigs (similar to experiences with wild boar in parts of Europe (Depner et al, 2017; Mačiulskis et al., 2020)), or whether culling a proportion of the feral pig population might lead to disease fadeout (as per Cowled and colleagues (2012) study on classical swine fever). The likelihood of transmission from ASF-deceased wild pig carcasses (Probst et al., 2017, 2019; Lange & Thulke, 2016), is also unclear in an Australian context, with some authors suggesting that cooler conditions enhance transmission due to prolonged virus viability in carcasses (Schulz et al., 2019).

Epidemiological models can provide insights into the spread and control of emergency animal disease and assist in the formation of animal health policy and preparedness plans. They may be particularly useful when diseases are rare or absent and field data is lacking. Over the past eight

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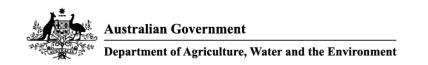
years the Australian Department of Agriculture, Water and the Environment has invested in the Australian Animal Disease Spread model (AADIS) (Bradhurst et al., 2013; 2015; 2016; 2019). The AADIS modelling framework can be used to instantiate national-scale epidemiological models of notifiable livestock disease such as foot-and-mouth disease (FMD). AADIS captures livestock disease epidemiology, regional variability in transmission (for example, due to environmental differences and seasonal livestock production and marketing patterns), and multi-jurisdictional approaches to control. AADIS is a sophisticated decision support tool that can be used to look at the risk of disease introduction, establishment and spread; control approaches in terms of effectiveness and costs; resource management; and post-outbreak management issues. The AADIS framework has also been expanded to model incursions, spread and management of agricultural and environmental pests (Bradhurst et al., 2021a).

This report describes the development of a new AADIS-ASF model that simulates the spread and control of ASF in domestic pigs, in feral pigs, and between domestic and feral pigs. The AADIS-ASF model is the primary outcome of CEBRA project 20121501 which ran from July 2020 to August 2021. The project built upon Biosecurity Innovation Program Project 192027 which ran from February 2020 to January 2021 and focussed on the modelling the spread and control of ASF in domestic pigs.

AADIS-ASF can simulate the introduction of ASF into feral and/or domestic pig populations at configurable points in time and space. The model simulates ASF transmission through live pig movements, fomites, and human movements, as well as spillover transmission between domestic and feral pigs. Control strategies for ASF in the domestic pig population are based on the Australian Emergency Veterinary Plan (AUSVETPLAN) Response Strategy for ASF v5.1 (Animal Health Australia, 2021). This includes movement controls, surveillance, tracing, infected premises operations (destruction, disposal, and decontamination) and post-outbreak surveillance to support the regaining of ASF-free status. Candidate control strategies can be compared in terms of outbreak size and duration, resource requirements, and cost. The AADIS-ASF model allows experimentation with transmission and control of ASF in feral pigs, including passive and active surveillance, and control via population reduction.

#### AADIS-ASF may help in evaluating:

- how ASF may spread in the domestic pig population
- the influence of on-farm biosecurity on ASF spread
- the potential for ASF to spillover between domestic and feral pigs
- how ASF may spread in the feral pig population including the influence of population density, infectious carcasses, and variable contact rates between groups
- regional and seasonal influences on ASF outbreaks in both domestic and feral pigs
- the potential for ASF to establish and become endemic in feral pigs
- resource management and costings





candidate control measures in domestic and feral pigs.

The report provides a literature review on ASF, feral pigs in Australia, and ASF decision support tools. Case studies on the spread and control of ASF in domestic and feral pigs demonstrate the functionality of the new model. Queensland was selected as the test case study area due to the wide distribution and high numbers of feral pigs and the availability of local expertise and data from Biosecurity Queensland, Department of Agriculture and Fisheries, Australian Pork Limited and SunPork Group Pty Ltd. The model was parameterised from the literature review and expert opinion that incorporated local knowledge of Australian production systems and environmental conditions. Note that the model is only parameterised for Queensland and will be scaled up to a national model through Biosecurity Innovation Program project 182021.

A series of simulation studies were carried out and preliminary findings suggest ASF is likely to be controlled in domestic pigs within 6 months of disease introduction (based on the configured assumptions of the scenarios). Indirect transmission of ASF (such as fomites, trucks, and people movements) was an important aspect of outbreaks and on-farm biosecurity played a critical role in reducing ASF spread. The simulations suggest feral pigs have the potential to amplify the size and duration of an outbreak, but their influence will depend on the region, the time of year, the density of the local feral pig population, and the extent of on-farm biosecurity measures. Spillover between domestic and feral pigs was far more likely to involve non-commercial farms (smallholders and pig keepers) than commercial farms. ASF outbreaks are likely to be larger and longer in cooler months and cooler regions due to increased viability of ASFV in the environment, especially in feral pig carcasses. The results of the simulations were coherent, reliable and consistent with international observations on ASF outbreaks and local expectations.

A finding of the project was that there is limited Australian-specific data on contact rates between groups of feral pigs, contact rates and likelihood of disease transmission between feral pigs and domestic pigs, and regional and seasonal influences on the transmission role that ASF-infected feral pig carcasses may play in an outbreak. Previous feral pig movement studies in Australia have collected ecological data on population or individual home ranges, seasonal patterns, and habitat preferences from the behavioural or genetic study of feral pigs. These studies have generally been designed to inform strategies for pest management (Cowled et al. 2008, Mitchell et al. 2009, Lopez et al. 2014., Wilson et al. 2021, in preparation) and have limited capacity to provide supply data to determine contact rates. The proximity of feral pigs to domestic piggeries presents a strong potential for disease transmission (Pearson et al. 2014), and a more directed field studies are recommended to collect data on interactions between feral pigs and their cohorts, and domestic pigs.

The AADIS-ASF model will provide the Animal Health Policy Branch with a useful decision support tool that will enable better preparedness planning for a potential incursion of ASF in Australia. The model will help identify knowledge and data gaps, support preparedness and training exercises, and inform strategic decision making.





#### 2 LITERATURE REVIEW

#### 2.1 African swine fever

#### 2.1.1 Overview

African swine fever (ASF) is a contagious haemorrhagic viral disease of domestic and wild/feral pigs (*Sus scrofa*), with case fatality rates for high-virulence strains approaching 100% (Costard et al., 2013; EFSA, 2014a). Whilst virulence of ASF can vary from acute to subacute and chronic, the global pandemic is driven by transmission of genotype II strains of the Georgia 2007 type, which are high virulence strains with rare mutations to lower virulence (Pikalo et al., 2019). The causative agent of ASF (ASFV) is a large enveloped DNA virus of the genus *Asfivirus* within the *Asfarviridae* virus family (Penrith et al., 2013). ASF was initially reported in 1909 in Africa, where it remained endemic in warthogs, domestic pigs, and ticks. The focus of this project is the high-virulence genotype II strain that emerged in 2007 in the Republic of Georgia and has spread across Europe, Africa and Asia resulting in the deaths of millions of pigs (Gallardo et al., 2015; EFSA, 2019; Gaudreault et al. 2020). The clinical signs of the Georgian strain include high fever, ataxia, loss of appetite, abortion, and depression (Cho et al., 2021), and usually appear 5-7 days after infection (Blome et al., 2013; Walczak et al., 2020). There is typically 1-2 days of pre-symptomatic infectiousness (Penrith & Vosloo, 2009; Beltrán-Alcrudo et al., 2017), and 90-100% of pigs will succumb to the disease within 6-13 days (Pietschmann et al., 2015).

#### 2.1.2 Transmission

#### 2.1.2.1 Direct spread

ASF can spread when an infectious animal comes into direct contact with a susceptible animal. This includes respiratory transmission, which can occur between animals sharing a paddock, yard, pen, or truck (Gallardo et al., 2015; Guinat et al., 2016a; Guinat et al., 2016b; Beltrán-Alcrudo et al., 2017).

#### 2.1.2.2 Indirect spread

Indirect spread is the transmission of infection from infectious pigs to susceptible pigs via indirect contact. Indirect contacts can arise through a variety of mechanisms including environmental contamination, fomites, biological vectors, mechanical vectors, contaminated transport vehicles, swill feeding, etc. In the context of the AADIS modelling framework, indirect spread includes all mechanisms for indirect contact with the exceptions of insect biological vectors (Section 2.1.2.3) and airborne plumes (Section 2.1.2.4) which are modelled separately.

Infectious animals excrete and secrete ASFV into the immediate environment where it can become a resilient source of secondary infections (Sánchez-Vizcaíno et al., 2012; Mazur-Panasiuk et al., 2019; EFSA 2018; EFSA, 2020). ASFV is very stable in blood (Plowright & Parker, 1967), faeces and urine (Davies et al., 2017) and soil (Kovalenko et al., 1965). It can, for example, remain infectious in manure for over 100 days (Blome et al., 2020) and for 1 to 3 weeks in the soil surrounding an infected carcass (Carlson et al. 2020). Wild pigs are known to interact with carcasses and especially the soil underneath carcasses (Probst et al., 2017). Given the stability of ASFV, infectious carcasses and their immediate environment thus pose a transmission risk to susceptible wild pigs (Oļševskis





et al., 2016; Lange & Thulke, 2016; Probst et al., 2019; Chenais et al., 2019; O'Neill et al., 2020). The period that an ASF-deceased feral pig carcass remains infectious will depend on environmental conditions affecting decomposition and virus viability such as heat, humidity, and precipitation (Probst et al., 2020) and the level of activity by scavengers and insects (Probst et al., 2019). It is possible that ASF could be spread mechanically through scavengers such as raptors, wild dogs, and foxes, however, they may in fact reduce the overall likelihood of indirect spread by metabolizing infectious carcasses (Probst et al., 2019). Mechanical transmission of ASFV is also possible through the ingestion of stable flies (Mellor et al., 1987; Olesen et al., 2018) but the level of risk this presents is yet to be clarified (Balmos et al., 2021).

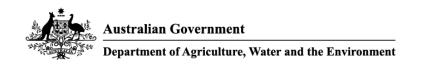
Indirect transmission of ASFV in domestic pigs can arise from movements of contaminated animal products, by-products, and fomites such as equipment, shoes, and vehicles (Penrith and Vosloo, 2009). Potential transmission pathways include veterinarians and stock feed delivery vehicles. ASFV remains viable in pork and pork products for lengthy periods (Farez & Morley, 1997; Costard et al., 2013; Olesen et al., 2018; Mazur-Panasiuk et al., 2019; Petrini et al. 2019). A substantial number of recent outbreaks of ASF in Europe and Asia have been attributed to indirect spread via contaminated fomites, environment, or ingestion of contaminated swill feed (Gogin et al., 2013; Oļševskis et al., 2016; EFSA, 2018a; Mazur-Panasiuk et al., 2019; EFSA, 2020).

#### 2.1.2.3 Vector-borne spread

Some argasid (soft) ticks are natural reservoirs of ASFV and some members of the Ornithodoros genus have been confirmed as competent vectors of ASF (Costard et al., 2013; Pereira de Oliveira et al., 2019). There are three species of Ornithodoros soft ticks in Australia - the seabird soft tick O. capensis, the possum soft tick O. macmillani, and the kangaroo soft tick O. gurneyi (Barker et al., 2014). O. capensis feeds on seabirds (primarily terns, gulls, and penguins), and given the opportunity, humans, and domestic fowl. O. macmillani feeds on possums and birds and is typically found in tree hollows and nests of Australian cockatoos. Ornithodoros gurneyi feeds on macropods (primarily the red kangaroo and the common wallaroo), and given the opportunity, humans, dogs, cattle, and horses), but is found in the arid regions of Australia, generally away from feral pig distributions (Dehhaghi et al., 2019). None of these ticks have been confirmed to feed on pigs and have not been associated with pig diseases. Although the ornate kangaroo tick (Amblyomma triquttatum) is found on pigs, there is no evidence that ixodid (hard) ticks such as this are involved in transmission of ASFV (de Carvalho Ferreira et al 2014; Spickler, 2018). As it is unclear whether Australian ticks could act as a reservoir of ASFV and contribute to spread, vectorborne transmission was not considered in this study. The subject is, however, under study by the Australian Centre for Disease Preparedness (previously the Australian Animal Health Laboratory) and it would be possible to consider this pathway in a future modelling project.

#### 2.1.2.4 Airborne spread

Whilst ASFV can be conveyed from infectious pigs to susceptible pigs via short-range (within-farm) aerosol transmission (Wilkinson et al., 1977; Wilkinson & Donaldson 1977; de Carvalho Ferreira et al., 2013b; Olesen et al., 2017), there is no evidence to date that longer range airborne spread between farms occurs (Guinat et al., 2016a; Guinat et al., 2016b; Olesen et al., 2017; Animal Health Australia, 2020).





## 2.2 The Australian feral pig population

#### 2.2.1 Distribution

Feral pigs are widely distributed across Australia, occurring across a reported 38 – 45% of Australia (Strahan, 1983; Choquenot et al., 1996; West, 2008) (Figure 1). It is thought that feral pigs are expanding in distribution (Cowled et al. 2009; Lewis et al., 2017) due to escapes from domestic production, slow natural dispersal (Caley, 1993) and illegal translocations for hunting resources (Spencer and Hampton, 2005). Feral pig populations can expand and contract in response to local environmental conditions, for example, recent rainfall can trigger rapid rates of increase through breeding (Giles, 1980).

In general, feral pigs are constrained by food and thermoregulation over much of Australia. For example, when it is warm, they are found in vegetated areas, especially riparian vegetation, but distribution may be more driven by food availability in cooler areas or seasons (Dexter, 1998). Feral pigs also utilise pasture and crops for food (Dexter, 1998). Thus, in drier or warmer areas or times, feral pigs frequent swamps, floodplains, and large freshwater rivers with riparian vegetation where they reach their highest abundance after adequate rainfall. However, feral pigs are also found in many other habitats such as and subalpine areas, woodlands, and rainforests and even in some peri-urban areas.

#### 2.2.2 Abundance

Estimates of the number of feral pigs in Australia have been refined over the decades (Tisdell, 1982; Hone 1990; Wilson et al. 1992) and most recently by Hone (2019). Hone (2019) estimated nationally there are 3.2 million pigs (95% CI: 2.4-4 million) at a density of 1.03 pigs per km², although densities of up to 20 pigs per km² have been recorded (Dexter 1990). This estimate of total feral pigs in Australia is much lower than the previous estimate used by industry (13.5 million (95% CI 3.5-23.5 million) but is more accurate given the large number of studies (142) used to obtain the estimate.

#### 2.2.3 Ecology of relevance

Some key aspects of feral pig ecology relevant to ASF modelling include movements of feral pigs (including after persecution), contact distances between groups of feral or wild boar, and the social structure of feral pigs.

Feral pigs are largely sedentary, demonstrating small dispersal distances in general and no tendency to disperse from their home ranges (Caley, 1997). For example, over a multiyear study, boar recapture distances were 3.2 km and sow 1.8 km indicating that these were local movements of feral pigs within their home ranges (Caley, 1997). When feral pig movements and home ranges have been measured in the past during and after intensive persecution (trapping, monitoring and aerial shooting) they have shown no tendency to change movement patterns or to disperse, with collared pigs remaining in their home ranges (Saunders and Bryant 1988; Dexter 1996). Despite this, rare long-distance movements of feral pigs do occur likely over many months (Saunders and Bryant 1988; Caley, 1997). In addition, aerial surveys of pigs indicate that some pigs can hide





following inefficient aerial shooting, but not during well conducted aerial shooting (Choquenot et al., 1995).

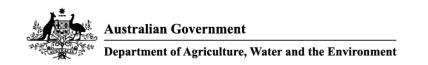
Feral pigs are largely structured across the landscape in female dominated groups of varying sizes, with some solitary males. However, feral pigs are highly sociable and home ranges can overlap, enabling contact and disease transmission between groups of feral pigs. Home ranges vary depending on available resources with larger ranges in areas of poor resources. Some of the larger yearly home ranges have been found to be up to 43 km² for males (Giles, 1980) and 24 km² for sows (Caley 1997), but most observations of home ranges have been smaller than this (Choquenot et al., 1996). However, the home ranges relevant for modelling an infectious disease are those associated with much smaller time intervals such as those that occur in daily home ranges, as these more closely reflect the incubation period of infectious diseases. Cowled et al. (2012) used a daily home range of 1 km² for modelling of CSF, based on research by Caley (1993). Practically, the distance within which most feral pig groups contact one another are most relevant. Several authors have found that for wild boar and feral pigs, most intergroup contacts occur over distances of less than 2 km (Pepin et al., 2016; Podgorski et al., 2018) and modelled home ranges of 4 km² (Scherer et al., 2020).

## 2.2.4 Invasiveness of feral pigs and damage

Feral pig populations can suffer high mortality rates (for example 90-100%) when local food resources are depleted by drought or other adverse seasonal conditions (Giles, 1980; Saunders, 1988). However, in good seasons, for example, following plentiful rain, the instantaneous rate of increase due to reproduction can be very high (Giles, 1980; Caley, 1993) allowing rapid growth of feral pig populations. In addition, feral pigs have an omnivorous diet making them adaptable to a wide variety of habitats. These features enable feral pigs to persist and then expand rapidly in an area when conditions are favourable.

Feral pigs in Australia cause a variety of agricultural damage resulting from disease transmission (for example, *Brucella suis* (Ridoutt et al., 2014)), predation of lambs (Plant et al., 1978; Pavlov and Hone 1982; Choquenot et al., 1997) and consumption of crops and pasture (Gentle et al., 2015). They also cause environmental damage such as predation of wildlife like turtles (Whytlaw et al., 2013), habitat disturbance through rooting and wallowing (Hone, 2002) and competition with native species for food (Energy, 2017). Feral pigs have been estimated to cause more than A\$152 million damage per year in Australia (in 2020 terms) (McLeod, 2004).

Wild boar or feral pigs have been integral in the epidemiology of transboundary diseases of pigs overseas including ASF, pseudorabies and classical swine fever (Artois et al., 2002; Corn et al., 2004, Blome et al. 2020). Previous Australian studies (Pearson, 2012; Pearson et al., 2014; Pearson et al., 2016) and overseas studies (Wyckoff et al., 2009; Wu et al., 2012; Kukielka et al. 2013; Jori et al. 2017; Hayama et al. 2020) have demonstrated the potential for feral pigs to be close to commercial piggeries and have also estimated contact rates. Wild pigs are known reservoirs internationally for major swine diseases such as classical swine fever and African swine fever. It is possible for feral pigs to have direct contact with domestic pigs that have access to non-biosecure outdoor areas, or indirect contact via environmental contamination. Therefore, a key risk to Australian agriculture from feral pigs is their potential involvement in epidemics of transboundary





diseases such as ASF, if these diseases were to enter Australia. Feral pigs may transmit disease to domestic pigs and complicate disease eradication and proof of freedom surveillance.

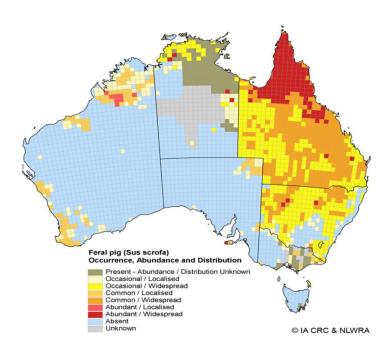


Figure 1. Estimated feral pig distribution and abundance (West, 2008)

## 2.3 Decision support tools for African swine fever

Hayes and colleagues (2021) present a systematic literature review of mechanistic models of ASF. Of the 24 publications reviewed, 16 describe modelling studies of ASF in domestic pigs, 7 describe modelling studies of ASF in wild boar, and only one looks at transmission crossover between domestic pigs and wild boar. This suggests there is a lack of decision support tools that can investigate the epidemiological interface between domestic and wild pigs. There are relatively few distinct ASF models, with just two models accounting for over half of the reviewed modelling studies. Models can be broadly classified as population-based, individual-based, or a hybrid blend of the population-based and individual-based approaches.

An example of a population-based ASF model is Barongo and colleagues' (2016) compartmental mathematical SEICD (susceptible – exposed – infectious – carrier – deceased) model. Control measures are simulated through simple dampening of the transmission rate. A limitation of population-based models such as this is the inability to consider spatial heterogeneities in transmission and control.

An individual-based modelling approach was used by Costard and colleagues (2015) to investigate the transmission risk of farmers quickly selling animals in response to clinical signs of ASF, and by Nigsch and colleagues (2013) to investigate transmission during the high-risk silent spread phase of an outbreak. Whilst individual-based models can represent spatial heterogeneities they are dependent on the quality of the underlying data and tend not to scale well for large populations.





A hybrid model utilises a population-based approach to represent the spread of disease within a herd and an individual-based approach to represent the spread of disease between herds (Bradhurst et al., 2013). An example of a hybrid model is DADS-DTU\_ASF (Halasa et al., 2016a) which represents within-herd spread with a SLSCR (susceptible – latent – subclinical – clinical – removed) state-transition sub-model that operates at both an animal and a herd level. Spread between herds is represented by contact-based direct and indirect spread pathways, and a spatial kernel-based local spread pathway. The within-herd infectious prevalence influences the probability of between-herd transmission. Herd-based hybrid models capture spatial heterogeneities and scale well with population size compared to animal-based individual level models (Bradhurst et al., 2016).

The reviewed literature suggests that the transmission characteristics of an ASF outbreak will depend on a range of country-specific factors such as domestic pig movements, on-farm biosecurity measures, feral pig distribution and abundance, regional and seasonal influences on ASFV viability in feral pig carcasses and in the environment, domestic pig control measures, and feral pig control measures. The development of an ASF decision support tool tailored to Australian conditions will enable better preparedness planning for a potential incursion of ASF in Australia and help identify knowledge and data gaps.





#### 3 CONCEPTUAL MODEL

## 3.1 Representation of the domestic pig population

#### 3.1.1 Epidemiological unit of interest

The AADIS epidemiological unit of interest for domestic pigs is the 'herd', defined as a group of comingling pigs under the same production system. A herd has static attributes such as the type of production system, biosecurity rating, geolocation and jurisdiction, and dynamic attributes such as infection and disease status. As AADIS models at a national scale, in the interests of computational efficiency, a herd is represented spatially as a point with latitude and longitude coordinates (i.e., land area is not explicitly modelled).

A central assumption in AADIS is that the domestic pig population in a study area can be categorised by 'herd type', such that key differences in production system characteristics and buying and selling patterns, can be satisfactorily captured. The stratification of the domestic pig population by herd type was driven by the granularity of available data on pig movements. AADIS allows a user to define custom herd types appropriate to the disease being modelled and the study area.

An AADIS 'farm' is a collection of one or more co-located herds under the same management system. This organisational structure allows AADIS to represent the increased probability of disease transmission between herds that are co-resident on the same farm, due to the higher potential for direct contact and indirect contact via shared equipment and personnel.

It would be possible to use the AADIS farm construct to represent a domestic pig farm and the AADIS herd construct to represent individual sheds on a farm. This would capture the multiscale nature of disease transmission on a pig farm whereby the spread of infection within a shed is typically higher than the spread of infection between sheds (Schultz et al., 2019). Unfortunately, this approach would require detailed shed-level data on pig farms which would be difficult to obtain for a large-scale multi-jurisdictional model. It was decided to represent domestic pig farms as herds with production system characteristics categorised by herd type.

## 3.1.2 Herd types and herd dataset

The herd types defined for the AADIS-ASF Queensland model (AADIS-ASF-QLD) are provided in Table 1 along with their occurrence in the Queensland herd dataset.

**Table 1.** Herd types used in the AADIS-ASF-QLD model

Herd type	Description	Number of herds	Average herd size (pigs)	Distribution of herd sizes (min median max)
Very large commercial	1000+ sows kept indoors. APIQ <sup>1</sup> accredited. Routine biosecurity practices and likely to have a secure (pigproof) perimeter fence. Multiple movements per week	19	24,985	10,110 16,000

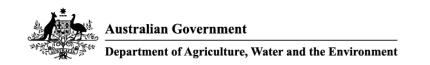


(VLC)	to export and domestic processing plants. Vehicle washdown and decontamination between movements likely			105,000
Medium to large commercial (MLC)	151-1000 sows kept indoors. Usually APIQ accredited. Moderate biosecurity practices and possibility of secure perimeter fencing. Weekly pig movements to domestic and export processing plants using owned vehicles or contractor livestock transport. Possibility of vehicle washdown after movements but decontamination unlikely.	50	4408	1800 4000 10,000
Small commercial (SC)	51-150 sows housed indoors or outdoors. Generally, not APIQ-accredited. Low biosecurity practices with no secure perimeter fencing. Regular pig movements to domestic processing plants using owned vehicles. Generally, no vehicle washdown / decontamination.	50	280	2 203 1114
Specialist gene transfer (SGT)	Specialist producers of boar semen. APIQ accredited. Routine biosecurity practices and likely to have a secure (pig-proof) perimeter fence. Vehicle washdown and decontamination between movements likely.	2	730	260 730 1200
Smallholder (SH)	50 or less sows kept primarily for non-commercial or micro-scale commercial purposes. Not APIQ-accredited. Low biosecurity practices and low awareness of biosecurity. No secure perimeter fencing. Occasional low-biosecurity movements to domestic abattoirs that are recorded under NLIS.	309	50	1 23 400
Pig keeper (PK)	50 or less sows kept outdoors for non-commercial purposes. Not APIQ-accredited. Low biosecurity practices and low awareness of biosecurity. No secure perimeter fencing. Infrequent low-biosecurity movements. The key distinction between smallholders and pig keepers is that movements of live animals off smallholders are recorded under NLIS whereas movements off pig keepers are not.	3587	7	1 2 500
Qld totals		4191	192	

<sup>&</sup>lt;sup>1</sup>APIQ – Australian Pork Industry Quality Assurance Program (www.apiq.com.au)

## 3.1.3 On-farm biosecurity

Prior to this project, the biosecurity characteristics of an AADIS herd were solely described by 'biosecurity weights' defined per herd type. Biosecurity weights provide a means of dampening the probability of disease transmission into a herd via the local and indirect spread pathways, reflecting likely biosecurity measures in place based on the herd type (Bradhurst et al., 2015). It



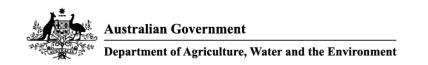


was agreed that assigning biosecurity ratings based on herd type was not granular enough for the pig industry in Australia. For example, from a disease transmission point of view, it is important to be able to distinguish the biosecurity characteristics of a free-range small commercial farm from a small commercial farm where the pigs are permanently housed indoors.

A new AADIS feature was developed that allows biosecurity characteristics to be defined on a perherd basis. A *risk category* attribute was added to the *Herd* database table that allows each herd to be scored in terms of risk factors for introduction of ASF. This includes production system, adopted biosecurity measures, quality assurance provisions, and access to outdoor areas (and thus risk of direct or indirect contacts with feral pigs or environmental contamination). The risk categories and mappings to biosecurity weights for the AADIS-ASF-QLD model are provided in Table 2. The risk category values for the Queensland pig herd dataset and the biosecurity weight mappings were derived through consultation with the Queensland Department of Agriculture and Fisheries (QDAF) and industry. An explanation of how biosecurity weights are used in the local, indirect, and feral pig spread pathways can be found in Section 3.3.

**Table 2.** Risk categories and biosecurity weights used in the AADIS-ASF-QLD model and the occurrences per herd type [VLC=very large commercial, MLC=medium to large commercial, SC=small commercial, BS=specialist gene transfer, SH=smallholder, PK=pig keeper]

Risk	Typical characteristics	Biosecurity	Number of herds per risk category per herd type					
category		weight	VLC	MLC	sc	SGT	SH	PK
1	Poor biosecurity practices and low awareness of biosecurity. No secure perimeter fencing. Pigs may be kept outdoors.	1.0	0	0	164	0	309	3587
2	Low biosecurity practices with no secure perimeter fencing. Pigs may be housed indoors or outdoors. Movements using owned vehicle and generally no vehicle washdown or decontamination	0.8	0	1	11	0	0	0
3	Moderate biosecurity practices and possibility of secure perimeter fencing. Movement using owned vehicles or contractor livestock transport. Possibility of vehicle washdown after movements but decontamination unlikely	0.4	2	39	39	1	0	0
4	Routine biosecurity practices and likely to have a secure (pig-proof) perimeter fence. Vehicle washdown and decontamination between movements likely.	0.15	17	10	10	1	0	0





## 3.2 Representation of the domestic pig study area

For the purposes of capturing regional and seasonal heterogeneity in domestic pig production and marketing systems, AADIS partitions Australia into 12 regions (Table 3 and Figure 2). To further simplify the curation of the pig movement data required to drive the model, the regions are aggregated into four mega-regions (Figure 3). This allows, for example, a large-scale piggery in the north-east of Australia to have direct and indirect movement patterns that are quite distinct from a large-scale piggery in south-west Australia. The Queensland study area spans five regions (Far North, Lower North, Arid Zone, Tropical NE Coast, Central Qld NW NSW) and two mega-regions (Pastoral and North-East).

Table 3. Regions and mega-regions used by the AADIS-ASF model

Region ID	Region name	Mega-region ID	Mega-region name
1	Far north	1	Pastoral (PL)
2	Lower north	1	Pastoral
3	Arid zone	1	Pastoral
4	Barkley Tableland	1	Pastoral
5	Tropical North-East Coast	2	North-east (NE)
6	Central QLD North-West NSW	2	North-east
7	New England	2	North-east
8	Temperate South-East Coast	3	South-east (SE)
9	Temperate slopes & plains	3	South-east
10	Mediterranean	3	South-east
11	Tasmania	3	South-east
12	South-West WA	4	South-west (SW)



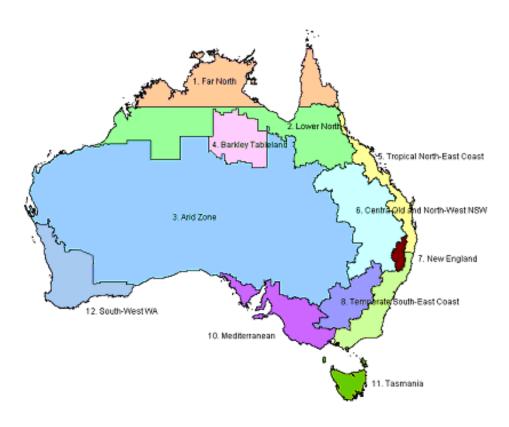


Figure 2. Regions used by the AADIS-ASF model

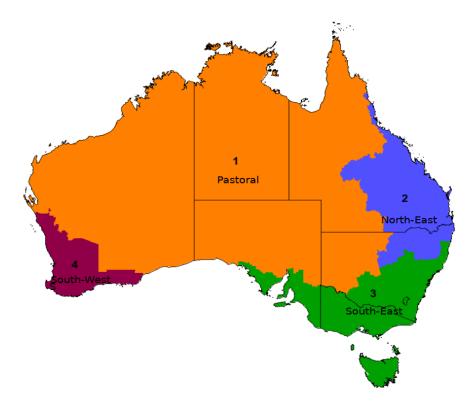


Figure 3. Mega-regions used by the AADIS-ASF model





## 3.3 Transmission of ASF within a domestic pig farm

AADIS considers a herd to be homogeneous and 'well-mixed' from a disease transmission point of view, i.e., all members of a herd are biologically equivalent and equally likely to contract a disease (Keeling and Rohani, 2008). A consequence of the decision to represent domestic pig farms as AADIS herds (Section 3.1.1) is that the spread of infection within a farm is modelled according to typical housing arrangements per herd type.

AADIS employs a deterministic SEIRDC (susceptible, exposed, infectious, recovered, deceased, clinical) compartmental equation-based model (EBM) to represent within-herd spread of the disease under study (Figure 4). The SEIRDC EBM can be thought of as comprising an SEIRD infection model (where exposed animals become infectious and then either recover or die), and a parallel SEC disease model (where exposed animals go on to either develop clinical disease or are asymptomatic). This approach is simple mathematically and is agnostic as to whether the latent period is less than the incubation period (i.e., there may be presymptomatic infectious animals), or whether the latent period is greater than or equal to the incubation period. The Exposed (E) and Infectious (I) compartments influence the progression of infection in a population. It is assumed that carcasses would be removed promptly from a domestic pig farm and the Deceased compartment (D) does not play an ongoing role in transmission (i.e.,  $\beta_D = 0$ ). The Clinical (C) and Deceased (D) compartments inform passive detection and surveillance of infection. The EBM assumes that there will be no surviving long-term carriers of ASFV that pose an ongoing risk of transmission (Stahl et al., 2019).

Each infected herd has a system of ordinary differential equations (ODEs) customised for the herd type, herd size and ASFV strain. AADIS simplifies herd size by assuming that inflows (births and transfers in) are equivalent to outflows (non-ASF related deaths and transfers out). When a susceptible herd becomes infected the ODE system is solved numerically to yield the SEIRDC compartmental counts over time. The EBM generates curves predicting the infected, infectious, and clinical prevalence of the infected herd (Figure 5). This approach is computationally efficient as the solution remains in place up until an external asynchronous event such as destruction acts upon the EBM.

In this project, AADIS-ASF was parameterised for the Georgia 2007/1 (genotype II) strain of ASF. This is a more virulent strain that is considered highly relevant to Australia given its circulation in Asia (Borca et al., 2020; Blome et al., 2020) and the frequent movements of goods and people between Australia and Asia. Details of the preliminary EBM parameterisation for the Georgia 2007/1 strain are provided in Appendix A.



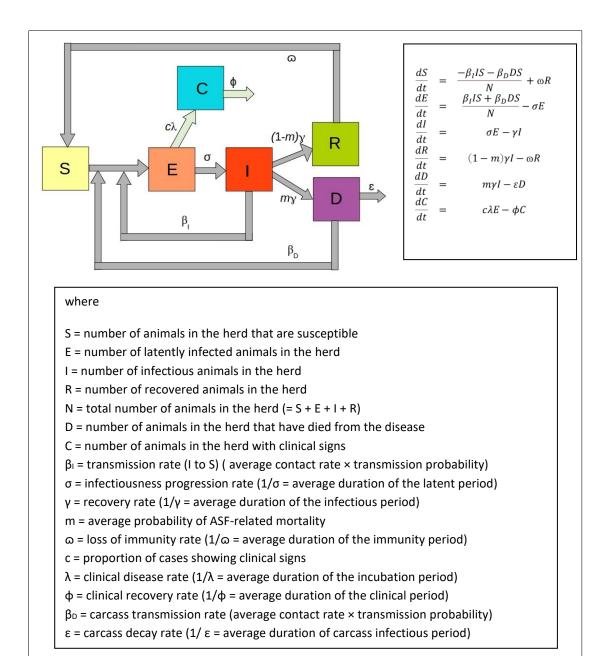


Figure 4. Within-herd SEIRDC model employed by AADIS-ASF

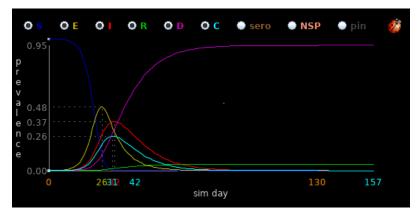
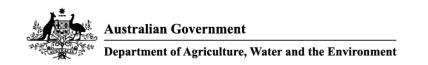


Figure 5. Example prevalence curves generated by the AADIS-ASF within-herd EBM





## 3.4 Transmission of ASF between domestic pig farms

The AADIS modelling platform employs a stochastic and spatially-explicit agent-based model (ABM) to represent the spread of disease between herds (Bradhurst 2015; Bradhurst et al., 2015; 2016). The levels of infected and infectious prevalence predicted by a herd's EBM inform the likelihood that disease will spread between herds. AADIS provides two techniques for spreading disease between herds:

- <u>Data-driven spread pathways</u>: local spread, direct spread between farms, direct spread via saleyards, indirect spread, and airborne spread. These spread pathways capture detailed spatiotemporal heterogeneity but require detailed parameterisation and are dependent on the availability and quality of the underlying data.
- Analytical spread pathways: jump and diffusion. These pathways represent short-range
  local dispersal and ad-hoc longer-range jumps of infection. They are coarser than the datadriven pathways but much simpler to parameterise and can be useful when there is
  inadequate data available to drive explicit spread pathways.

Each spread pathway has a stochastic algorithm that determines on any given simulation day whether disease transfers from infectious herds to susceptible herds (Bradhurst, 2015). AADIS-ASF makes use of the data-driven spread pathways.

## 3.4.1 Local Spread

Local spread is a catch-all pathway for very short-range transmission of disease from an infected herd to neighbouring susceptible herds when the exact spread mechanism may not be known (Sanson, 1994). Local spread might arise from a variety of transmission mechanisms such as:

- direct contacts via unregistered animal movements, the straying of stock, or animals mingling at fences
- indirect contacts via vehicles, people, surface runoff, insects/rodents/birds, or sharing of equipment between neighbours
- short-range aerosol spread

The risk of local spread of ASF between domestic pig farms is not well understood in Australia. Local spread may be less important for ASF as there is no expectation of airborne transmission between farms (Guinat et al., 2016a; Guinat et al., 2016b; Olesen et al., 2017; Animal Health Australia, 2020). Further, large-scale pig production systems that are predominantly indoors with strict biosecurity measures in place will be less conducive to local spread than free-ranging production systems that are common with cattle and sheep. Simulation modelling can be useful in the face of uncertainty as it provides a means for gauging the importance of specific spread pathways to the overall outbreak. Local spread has been explicitly represented in European ASF modelling studies (Halasa et al., 2016a; Mur et al., 2017; Halasa et al., 2018; Andraud et al., 2019) and CSF modelling studies (Boklund et al., 2009; Yadav et al., 2013). In each study, local spread was implemented as a spatial risk kernel operating inside a fixed radius (1 to 2 km) of each infected property.



Local spread in AADIS-ASF is implemented as a spatial kernel that aggregates indirect spread mechanisms (only) inside a circular area enclosing each infected herd. A default local spread radius of 3 km was chosen to reflect the generally lower farm densities in Australia than Europe. The indirect spread pathway does not operate inside the local spread area to avoid double counting of transmissions. All susceptible herds inside a local spread area are deemed at-risk on each simulation day. The probability of transmission is influenced by the distance between an infected herd and a susceptible herd; infectivity of the infected herd; susceptibility of the at-risk herd; biosecurity measures in place at the at-risk premises; and seasonal variations in the ability of the virus to remain viable in the environment (Equation 1).

$$p_i = P_b \ p(t) \ W_i \ W_s \ W_b \ W_x \ w(d) \ W_n$$
 (Equation 1)

where

 $p_i$  = probability that the local contact results in an infection

 $P_b$  = baseline probability that a local contact between farms results in infection

p(t) = normalised infectious prevalence of the source herd at time t

 $W_i$  = infectivity weight of the source herd

 $W_s$  = susceptibility weight of the destination herd

 $W_b$  = biosecurity weight of the destination herd (depends on herd type)

 $W_x$  = seasonal weight (depends on mega-region)

w(d) = distance weight

 $W_n$  = detection weight (reflecting that local spread may organically dampen once an outbreak has been declared due to an increased awareness of risk, decreased movements of people and vehicles, etc.)

The distance weight w(d) can be configured to decay linearly (Equation 2) or exponentially (Equation 3).

$$w(d) = 1 - (d/R)$$
 (linear decay) (Equation 2)

$$w(d) = e^{(C * d/R)}$$
 (exponential decay) (Equation 3)

where

d = distance from the source herd to the destination herd (km)

R = diffusion radius (user configurable, default 3 km)

C = decay constant (user configurable, default -3.4539)

Local spread can also occur between herds that are co-resident on the same holding. In this case the baseline probability of transmission  $P_b$  is increased to reflect the higher potential for indirect contacts between herds managed on the same holding.

Tildesley and colleagues (2012), found that a non-linear relationship between herd size and infectivity/susceptibility better described data from the 2001 UK FMD outbreak than a linear relationship. EuFMDiS provides user-configurable power law parameters  $P_i$  and  $P_s$  that specify the level of influence that herd size has on infectivity and susceptibility. Infectivity weights depend on herd size and are scaled across the herd population (Equation 4). The infectivity powers  $P_i$  allow





tuning of the effect of herd size on infectivity ( $0 \le P_i \le 1$ , where a value of 0 specifies no effect and a value of 1 specifies a linear relationship).

$$W_i = n^{p_i} / population median(n^{p_i})$$
 (Equation 4)

where

 $W_i$  = infectivity weight

n = herd size

 $P_i$  = infectivity power (default = 0.3)

Susceptibility weights also depend on herd size and are scaled across the herd population (Equation 5). The susceptibility powers  $P_s$  allow tuning of the effect of herd size on susceptibility (0  $\leq P_s \leq 1$ , where a value of 0 specifies no effect and a value of 1 specifies a linear relationship).

$$W_s = n^{Ps} / population median(n^{Ps})$$
 (Equation 5)

where

 $W_s$  = susceptibility weight

n = herd size

 $P_s$  = susceptibility power (default = 0.3)

When a susceptible herd becomes infected, an EBM is created and solved with initial conditions based on the estimated number of exposed animals in the destination herd and the size of the destination herd.

The AADIS local spread pathway and parameters are described in Bradhurst 2015 and Bradhurst et al., 2015. The parameterisation of the local spread pathway for AADIS-ASF is provided in Appendix B.

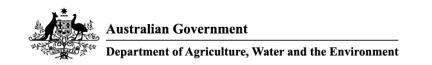
#### 3.4.2 Indirect Spread

The frequency, distance and destination premises of indirect contacts are determined stochastically, taking into account production system and regional and seasonal patterns. Whilst it is possible to implement separate spread pathways for specific types of indirect contacts, the lack of relevant data warrants a simpler approach. AADIS-ASF provides a single aggregative category of indirect contacts with a specified average (baseline) probability of transmission. The user can parameterise this to represent different risk profiles. If a herd is exposed to an indirect contact, the probability of transmission depends on the infectious prevalence of the source herd, the relative infectiousness of the source herd (based on herd size), environmental conditions that influence virus viability, biosecurity practices in place in at-risk premises, and relative susceptibility of the exposed herd (based on herd size) (Equation 6).

$$p_i = P_b p(t) W_i W_s W_b W_x$$
 (Equation 6)

where

 $p_i$  = probability that a specific indirect contact results in infection  $P_b$  = baseline probability that an indirect contact results in infection





p(t) = normalised infectious prevalence of the infectious herd at time t

 $W_i$  = infectivity weight of the source herd (per local spread)

 $W_s$  = susceptibility weight of the exposed herd (per local spread)

 $W_b$  = biosecurity weight of the exposed herd

 $W_x$  = seasonal weight

The AADIS indirect spread pathway and parameters are described in Bradhurst 2015 and Bradhurst et al., 2015. The parameterisation of the indirect spread pathway for AADIS-ASF is provided in Appendix B.

## 3.4.3 Direct Spread

Prior to this project, the AADIS direct spread pathway was purely stochastic. The timing of direct movements and the destination and size of consignment were driven by probability-contact matrices and distance distributions, stratified by herd type, mega-region, and season (Bradhurst et al., 2015). It was determined that a stochastic approach was not appropriate for the Australian pig industry where commercial animal movements are typically more directed and predictable, for example routine transfers between sites of a vertically integrated operation and periodic consignments for specific domestic pork markets.

The direct spread pathway was augmented with the option of replaying historical movements of pigs (as recorded in Australia's National Livestock Information System (NLIS). This provides much more realistic estimations of the direct transmission of infection between farms and between farms and saleyards. Transmission depends on the prevalence of infection in the source herd and the consignment size. When a susceptible herd becomes infected an EBM is created and solved with initial conditions based on the proportion of infectious and exposed animals in the consignment, and the size of the destination herd.

Movements from infected farms to abattoirs are logged but no further spread occurs, i.e., they are considered 'dead-ends' with respect to disease transmission, although they are important locations from which ASF might be first reported. Further movement data would be required to include abattoirs as sources of infection for onward spread of ASFV.

Saleyards have the potential to greatly amplify an outbreak prior to the disease being recognised and controls implemented (Gibbens et al., 2001). The transmission of disease is facilitated by the stresses of transit and handling, large numbers of susceptible animals, and the mixing and partitioning of stock into consignments. Further, outgoing consignments can potentially carry infection to multiple widely dispersed locations. At a saleyard, animals from different sources may be mixed and sorted such that a single infected consignment entering a saleyard may contribute to multiple infected consignments leaving the saleyard. The destination of each infected consignment leaving the saleyard (another farm or an abattoir) is determined via historical NLIS movement data. Infection is transmitted from infected consignments to destination herds with a force relative to the viral load in the consignment. Note that the likelihood of ASF transmission via saleyards will be relatively low in Australia given the very minor role that saleyards play in the pig industry (Hassall and Associates, 2007; East et al., 2014).





## 3.4.4 Vector-borne spread

It is unclear whether soft ticks in Australia could act as a reservoir of ASFV and contribute to spread (Section 2.1.2.3) and tick vector-borne spread pathway was not included in the AADIS-ASF model. However, if competent tick vectors of ASF are identified in Australia, then the model could be revised during subsequent research and development activities.

## 3.4.5 Feral pig spread

If ASF was to enter the feral pig population it would be possible for infection to spillover into domestic pig farms via direct or indirect transmission. This pathway is described separately in Section 3.10. Conversely, if ASF was to enter the domestic pig population it would be possible. for infection to spillover into the feral pig population via direct or indirect transmission. This pathway is described separately in Section 3.19.

#### 3.4.6 Airborne Spread

Airborne spread is not a recognised feature of ASF transmission and as such the airborne spread pathway is disabled for AADIS-ASF. It can easily be enabled in the future if required. Details of the implementation can be found in Bradhurst et al., 2015.

#### 3.5 Surveillance, detection, and control of ASF in domestic pigs

Australia's response strategy to an outbreak of ASF is outlined in the AUSVETPLAN Response Strategy for ASF (Animal Health Australia, 2020). The default ASF response is to control and eradicate ASF in the shortest time possible to regain ASF-free status, whilst minimising socioeconomic impacts. Response activities would be consistent with World Organisation for Animal Health (OIE) guidelines and include implementation of declared areas; movement controls in declared areas; tracing and surveillance to determine the source and extent of infection; valuation, destruction, and disposal of pigs on infected premises and potentially high-risk pigs; decontamination of infected premises; animal welfare management; and potentially zoning and/or compartmentalisation (Animal Health Australia, 2020).

The AADIS-ASF simulated control measures are consistent with the approaches described in the AUSVETPLAN Response Strategy for ASF v5.1 (Animal Health Australia, 2020). The key simulated control measures are biosecurity and movement controls, surveillance, tracing, and infected premises operations (valuation, destruction, disposal, and decontamination). Control measures are configured and resourced per jurisdiction. Selected preliminary parameterisation of AADIS-ASF control measures is provided in Appendix C.

#### 3.5.1 Detection of the index case

The control and eradication phase of an outbreak commences after the declaration of the index case i.e., the first declared infected premises (IP). The day of first detection is either determined stochastically (using pre-configured probabilities of reporting by herd type, and clinical prevalence), or occurs on a fixed day at a specific or randomly selected farm.





#### 3.5.2 Movement Controls

Declared areas are established around each IP to control the movement of pigs, pig products, and other material. The declared areas are defined and enforced per-jurisdiction, and may be designated areas (local administrative area, entire jurisdiction), or radius-based per IP. There are three declared areas: restricted areas (RAs) that enclose IPs, DCPs and as many SPs, TPs and DCPFs as practicable; control areas (CAs) that enclose RAs; and infected areas (IAs) that are defined when ASF is found in feral pigs. RAs have a higher level of control than CAs. AADIS-ASF models the imposition of declared areas in a staged manner. Larger declared areas are enforced at the start of an outbreak. As the control program progresses, the dimensions of the declared areas are amended according to jurisdictional preferences. Jurisdictional declared areas are clipped to fall within the jurisdiction boundaries of the subject IP. When IPs are clustered a meta-IA (if applicable), meta-RA and meta-CA are formed from the union of the constituent RAs and CAs.

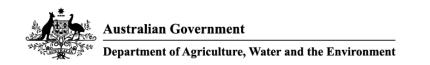
#### 3.5.3 Tracing

Tracing is the identification of movements onto and off IPs, DCPs and DCPFs to ascertain where infection may have come from or gone to. Tracing includes animals, products, equipment, vehicles, and people. Traced premises may be true cases (and thus infected), or false (not infected). AADIS-ASF can readily identify true traces by following infection chains during a simulation, allowing for variable tracing effectiveness by herd type and pathway (direct contact versus indirect contact), and tracing duration. False forward traces are obtained by applying the direct and indirect spread pathways to a premises of interest within the forward tracing window. False backward traces are obtained by reversing the direct and indirect spread pathways over the backwards tracing window (i.e., modelling movements onto the premises of interest). This approach results in a set of plausible false traces to premises (of a suitable type and location) that could well have been sources or destinations of movements of concern. Each false trace triggers a surveillance visit that utilises resources but does not progress the control program. The inclusion of false traces adds realism to AADIS-ASF simulations.

#### 3.5.4 Surveillance

Surveillance is the process by which new infections are identified and declared. During an ASF outbreak, surveillance is used to detect new outbreaks, define the extent and source of infection, and demonstrate freedom in uninfected areas. In turn this will provide data to inform risk analyses and selection of appropriate control measures.

Premises that require visits by surveillance teams are identified through tracing, active inspection of premises within declared areas, reporting of suspect premise and epidemiological analyses. Diagnostic samples are taken and tested when needed. AADIS-ASF maintains a resource-constrained dynamic queue of premises awaiting a surveillance visit. Surveillance visits are prioritised according to a configurable scheme that considers premises classification, declared area and herd type. If multiple premises have the same priority, then arbitration is based on how long a premises has been waiting for a visit. The visit duration (based on herd type), visit frequency (based on priority), and overall surveillance period are configurable.





AADIS-ASF allows for the reporting of suspect cases on an ad hoc basis by pig owners/inspectors, or others. AADIS-ASF commences suspect case reporting the day after the first IP has been declared and allows for both true positive and false positive reports. False positive reports identify herds that are exhibiting consistent clinical signs but are not actually infected with ASF. True positive reports are generated stochastically based on an infected herd's clinical prevalence, the probability of reporting and the expected time to report. The latter two parameters are defined per herd-type in the AADIS-ASF configuration data. The number of false positive reports generated is proportional to an n-day (default n=3), moving average number of true positive reports. The modelling of both true and false reports facilitates more realistic modelling of surveillance as resources are consumed regardless of whether a surveillance visit yields a positive assessment or not. AADIS-ASF also models routine active surveillance of at-risk premises (ARPs) within RAs. All farms within a designated distance of IPs are subject to a configurable inspection schedule (number and frequency of visits).

#### 3.5.5 IP Operations

IP Operations are the valuation, destruction ('stamping out') and disposal of animals, and decontamination of premises. Stamping out of IPs is the default policy for controlling an outbreak of ASF as it is considered the fastest way to reduce viral excretions, limit environmental contamination and dampen spread. AADIS-ASF also provides the option of ring culling farms within a configurable distance of each IP, and pre-emptive culling of farms that are deemed high risk because of a traced direct contact with an IP.

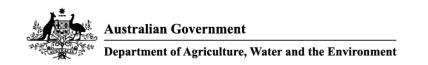
All IP operations are prioritised based on the reason for destruction (stamping out takes precedence over ring culling and preemptive culling), herd type, herd size, proximity to an IP, and (in the case of ring culling and preemptive culling) distance to the nearest IP. The times required for a farm to undergo destruction, disposal and decontamination are defined by herd type in the AADIS-ASF configuration data.

#### 3.5.6 Vaccination

A vaccine for ASF is not currently commercially available (Arias et al., 2017; Sánchez-Cordón et al., 2018; Yoo et al., 2020; Borca et al., 2020), and as such the AADIS-ASF vaccination component is disabled. However, when a vaccine becomes available it will be relatively straightforward to enable and configure vaccination in the AADIS-ASF model.

#### 3.5.7 Post-outbreak surveillance

Disease models often stop simulating once an outbreak has been controlled i.e., all infected herds have been found and the control program has concluded. However, from a disease manager's perspective, additional surveillance must be undertaken to support the regaining of disease-free status. It can be challenging for a disease manager to decide when the final IP of an outbreak has been declared and processed, and post-outbreak surveillance should commence. AADIS represents this with a user-defined rolling countdown timer (e.g., 30 days) that starts whenever a new IP is declared and processed. If the countdown timer expires then the outbreak is assumed over, and post-outbreak surveillance activities commence.





Post-outbreak surveillance is conducted in terms of 'clusters' that represent discrete areas around previously declared infection (IPs and IAs). Post-outbreak surveillance is carried out independently in each cluster to provide statistical support for proof-of-freedom. A user-defined sampling regime determines the number of herds to test within a cluster, and the number of animals to test within a selected herd, to achieve statistical confidence that residual infection would be detected. For example, a 95:5 sampling regime implies that sufficient herds are randomly tested in a cluster to achieve 95% confidence that a residual infected prevalence of at least 5% would be detected (Cannon and Roe, 1982; Cannon, 2001).

Testing regimes are defined in terms of test pairs [screening, confirmatory] that depend on herd type. Tests may be a clinical, serological, or virological, and are defined in terms of sensitivity, specificity, cost, throughput, and pooling rate (Bradhurst et al., 2021). The latter allows for the incorporation of pooled tests such as salivary ropes in domestic pigs. AADIS-ASF reports the number of true/false positives and true/false negatives, and the duration and cost of the post-outbreak surveillance program.

#### 3.5.8 Resourcing

The resources required to manage an emergency animal disease outbreak include personnel (e.g., veterinarians, animal health officers, control centre staff), equipment (e.g., vehicles), facilities (e.g., laboratories) and consumables (e.g., vaccine (when available and used), disinfectant, laboratory diagnostic reagents). Some aspects of disease control and eradication are resource-intensive, and the lack of resources can severely hamper the response to an outbreak (Roche et al., 2014).

AADIS-ASF models the resources required for key operational activities: surveillance, destruction, disposal, decontamination, and vaccination (when vaccines are available and used). An AADIS-ASF 'resource' is abstract in that it can represent whatever is required to complete a specific task. For example, the resource required to conduct a surveillance visit might be a veterinarian, an assistant, sampling equipment, personal protective equipment, decontamination equipment, and a vehicle. As jurisdictions are responsible for emergency animal disease management, resources are allocated per jurisdiction, and organised into 'pools' (i.e., each jurisdiction has five resource pools, one for each key operational activity).

When a field operation is scheduled, a resource is requested from the relevant pool of the jurisdiction. If a resource is available, then it is 'borrowed' from the pool and the field operation commences. If a resource is not available, then the field operation is queued until such time as a resource becomes available. Once a field operation has completed, the resource is 'returned' to the pool.

It is anticipated that resource levels ramp up over time, so initially the pools are small and increase in a linear manner up to a maximum size. The starting point, duration of the ramp-up and maximum pool size are defined in the AADIS-ASF configuration data, by resource type and by jurisdiction. AADIS-ASF tracks the availability and allocation of resources to provide immediate feedback as to whether/where the control program is resource constrained.





Resource pools can be configured to be 'unlimited' in which case requested resources are always immediately granted. In this mode the resourcing profile of an outbreak is a model output, rather than a constraint on the efficacy of the control program.

#### 3.5.9 Outbreak costs

AADIS-ASF keeps track of control costs (control centres, field operations, compensation, vaccine (when available and used), loss of trade), post-outbreak management costs (control centres, field operations, compensation), and loss of trade (estimated simply from the number of days from the declaration of the index case through to the end of the mandatory OIE waiting period).

## 3.6 Representation of the feral pig population

Options for modelling a feral pig population include:

- An individual-based approach whereby the presence and movements of matriarchal family groups (sounders) and solitary boars are represented explicitly in time and space. This approach requires detailed ecological and environmental knowledge and data and is usually suited to smaller scale studies (Cowled et al., 2012; Leslie et al., 2014; Ward et al., 2015; Toger et al., 2018; Croft et al., 2020).
- A raster approach whereby an environment is represented as a lattice in which cells have individual densities/counts/probabilities of feral pigs. This approach greatly simplifies the underlying ecological mechanisms but scales well computationally for larger-scale modelling of habitat suitability and species distribution (Cowled et al., 2009; Froese et al., 2017; Lewis et al., 2017; Pittiglio et al., 2018; Gentle et al., 2019). Examples of animal disease models that have represented feral populations with raster data include Doran & Laffan, 2005; Milne et al., 2008; Ward et al., 2009; Lange et al., 2012.

As AADIS is a national-scale model that focuses on epidemiological processes and transmission risk (rather than ecological processes), it was decided to represent the feral pig population with a raster approach. Representing sounders and solitary boars as individual agents on a national scale would have resulted in a prohibitively high additional number of epidemiological units in the model.

The AADIS modelling framework employs an individual-based modelling approach for livestock diseases (Bradhurst et al., 2013, 2015, 2020b) and a geographic automaton modelling approach for agricultural and environmental pests (Torrens & Benenson, 2005; Laffan et al., 2007; Bradhurst et al., 2020a). The AADIS-ASF model fuses these two approaches into a single model where agents can be point-based herds of domestic pigs or cell-based groups of feral pigs.

#### 3.6.1 Distribution and abundance

An AADIS wildlife study area is represented by a grid delineated by lines of latitude and longitude. Each cell in the grid has environmental attributes such as elevation, average weekly temperature, annual rainfall, human population density, vegetation index, land use category, average weekly wind speed, etc. Each environmental attribute corresponds to a 'layer' of ascii raster data. Layers





can be purely spatial (such as elevation) or spatiotemporal (e.g., average weekly temperature) (Bradhurst et al., 2020a).

The grid extent and cell dimensions are user configurable and facilitate regional studies (inside a localised grid) up to large-scale studies (inside a national grid). The choice of cell size largely depends on the pest/pathogen being modelled, the extent of the study area, and the granularity of the relevant environmental data. A large cell size will not capture within-cell spatial heterogeneities in vegetation, land use, elevation, temperature, etc. A small cell size captures spatial heterogeneities (data granularity permitting) but comes with a computational overhead for large grids. It is advisable to restrict the total number of grid cells to under 1,048,576 so that the raster data input CSV file (which is indexed row-major order on cell ID), can be opened by a standard desktop spreadsheet program.

A cell size of 2 km x 2 km is employed in the AADIS-ASF wildlife raster to reflect the observation that sounders may interact with other sounders within 2 km but are unlikely to interact with other sounders 4-6 km away (Pepin et al., 2016; Podgórski et al., 2018). Any AADIS grid cell can have a feral pig count attribute that varies over time. The periodicity of the population counts is configurable and for AADIS-ASF is set to monthly over a 12-month period. This means that each cell has 12 'time slices' reflecting monthly changes in population count and the 12<sup>th</sup> time slice wraps back to the 1<sup>st</sup> time slice. The time slices are visualised as 'time-normalised' meaning the counts are normalised relative to the maximum count for that cell over time (Figure 6).

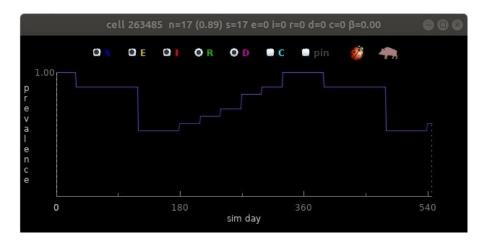


Figure 6. Population time slices for a cell over an 18-month timeframe

#### 3.6.2 Regional and seasonal heterogeneity

For the purposes of capturing regional and seasonal heterogeneity in the feral pig population, AADIS partitions Australia into wildlife regions. The definition of the wildlife regions is flexible and is currently based on the Terrestrial Ecoregions described by the Department of Sustainability, Environment, Water, Population and Communities (2021) (Table 4 and Figure 7). This allows a range of feral pig ecology and disease transmission parameters to be defined per region and per season (Appendices D, E and F).

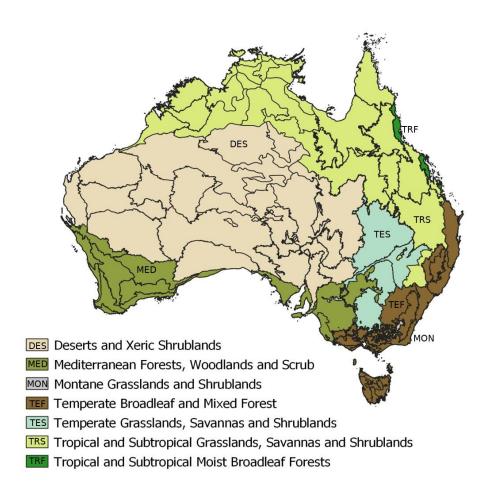
**Table 4.** Wildlife regions used by the AADIS-ASF model (adapted from Department of Sustainability, Environment, Water, Population and Communities, 2021)





Region	Name	Description
DES	Deserts and xeric shrublands	Annual rainfall varies greatly and generally is exceeded by evaporation. Temperature extremes are typical with searing daytime heat and cold nights due to limited insulation from humidity and cloud cover.
MED	Mediterranean forests, woodlands and shrubs	Hot and dry summers, while winters tend to be cool and moist.
MON	Montane grasslands and shrublands	High elevation (montane and alpine) grasslands and shrublands in south-eastern Australia including the Australian Alps and parts of Tasmania.
TEF	Temperate broadleaf and mixed forests	Moderate climate and high rainfall that give rise to unique eucalyptus forests and open woodlands.
TES	Temperate grasslands, savannas and shrublands	Cooler and wider annual temperatures than tropical grasslands. Much of this region has been converted to sheep rearing and wheat cropping, and only small fragments of the original eucalypt vegetation remains.
TRS	Tropical and subtropical grassland, savannas and shrublands	Tropical areas with rainfall levels that do not support extensive tree cover. Examples are the Kimberley, Top End, and Cape York savannas.
TRF	Tropical and subtropical moist broadleaf forests	Low variability in annual temperature and high levels of rainfall.  Dominated by semi-evergreen and evergreen deciduous tree species.  Australia has a small and scattered areas of this type of forest in  Queensland and Norfolk Island. These forests are of particular interest for the high degree of endemism of their plant (many with ancient lineages) and animal species.





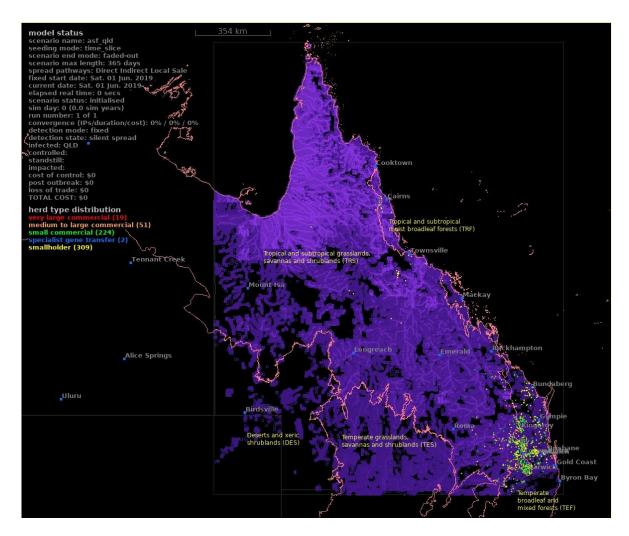
**Figure 7.** Wildlife regions used by the AADIS-ASF model (Department of Sustainability, Environment, Water, Population and Communities, 2021)

## 3.6.3 Baseline Queensland feral pig population dataset

The most recent high quality and nationally consistent data on feral pig distribution and abundance is described by West (2008). It is largely a compilation of previous state-based survey data, as well as surveys of institutional knowledge where data was absent (Woolnough, West et al. 2004). Although the data is presented in a uniform 0.5x0.5 decimal degree national grid (equating to approximately 50x50 km cells), the original scale of the underlying source data varied from a 5x5 km to 125x125 km grid cells which presents difficulties in the uniform use of the data. For example, resampling occurrence data from 125x125 km cells down to 5x5 km cells will lead to overestimation of the contiguity of the feral pig population. This in turn will lead to an overestimation of the potential role of feral pigs in the transmission of disease.

The distribution and abundance data for Queensland feral pig population (Figure 8) was estimated using the West (2008) occurrence data taking into account regional studies on regional feral pig densities (Choquenot et al., 1996; Heise-Pavlov et al., 2003; Cowled et al., 2009), publicly available permanent water and vegetation data, and the wildlife regions defined in Section 3.6.2. If future studies on feral pig ecology in Australia produce better estimates of distribution and abundance, then it will be relatively easy to update the AADIS-ASF baseline feral pig raster data layer.





**Figure 8.** Screenshot of the AADIS-ASF-QLD model illustrating the baseline feral pig distribution and abundance data layer, wildlife regions, and commercial pig farm locations

## 3.6.4 Monthly feral pig population estimates

The baseline feral pig population layer was transformed into monthly layers by taking into account relative changes in the abundance of the feral pig population driven by regional and seasonal influences on mortality and births (e.g., rainfall, land use). The took the form of per-cell multipliers (Table 5) that were largely informed by instantaneous rates of increase observed by Giles (1980), Saunders (1993), Caley (1993), Dexter (1998) and Gentle et al. (2019). The resulting 12 data layers were used to populate the time slices described in Section 3.6.1. Note that the process of deriving the 12 monthly data layers of feral pig counts from the baseline layer is done offline when populating the Postgres relational database. When the model starts up, the 12 layers are read from the Postgres relational database into the in-memory relational database (Bradhurst, 2015).

Table 5. Multipliers used to convert the baseline feral pig population into monthly counts

Region	Area	Dec	Jan	Feb	Ma	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov
		Sı	umm	nmer Autumn			Winter			Spring			
MED	South	0.8	1.0	1.0	1.0	1.0	0.9	0.8	0.7	0.6	0.6	0.6	0.7
TEF	South	0.8	1.0	1.0	1.0	1.0	0.9	0.8	0.7	0.6	0.6	0.6	0.7



DES	Centre	0.8	1.0	1.0	1.0	0.9	0.8	0.7	0.6	0.6	0.7	0.7	0.7
MON	Centre	0.8	1.0	1.0	1.0	0.9	0.8	0.7	0.6	0.6	0.7	0.7	0.7
TES	Centre	0.8	1.0	1.0	1.0	0.9	0.8	0.7	0.6	0.6	0.7	0.7	0.7
		Wet											
				Wet					D	ry			Wet
TRS	North	0.6	0.7	<b>Wet</b> 0.8	0.9	1.0	1.1	1.1	D 1.0	<b>1</b> .0	1.0	0.6	<b>Wet</b> 0.6

## 3.6.5 Statistical summary of the feral pig dataset

The number of cells in each wildlife region that are deemed to have a feral pig population are summarised in Table 6. The modelled area of Queensland (i.e., the sum of the terrestrial cells) is 1.845 million km<sup>2</sup> and this aligns well with the reported figure of 1.853 million km<sup>2</sup>. The modelled occurrence of feral pigs in Queensland is 779,480 km<sup>2</sup> or 42% of the land area.

**Table 6.** Statistical summary of modelled feral pig distribution by wildlife region

Wildlife	Part of	Number	Area (km²)	Populated	Populated	Populated
region	Qld	of cells		cells	cells (%)	area (km²)
TRS	North	290,283	1,161,132	158,390	55%	633,560
TRF	North	8,220	32,880	4,289	52%	17,156
DES	Central	86,503	346,012	4,252	5%	17,008
TES	South	54,443	217,772	23,118	42%	92,472
TRF	South	8,220	32,880	4,289	52%	17,156
Total	All	461,258	1,845,032	194,870	42%	779,480

Note that Figure 8 and Table 6 are presenting the modelled occurrence of feral pigs in Queensland, not habitat suitability, which will be considerable broader.

Figure 9 illustrates the modelled feral pig population in Queensland ranges from a minimum of 1.3 million from October to November, up to a maximum of 2.3 million in May. The number of feral pigs is dominated by the heavily populated TRS wildlife region in the tropical north of the state (Figure 8). This is consistent with West et al., 2008 and Gentle et al., 2019.



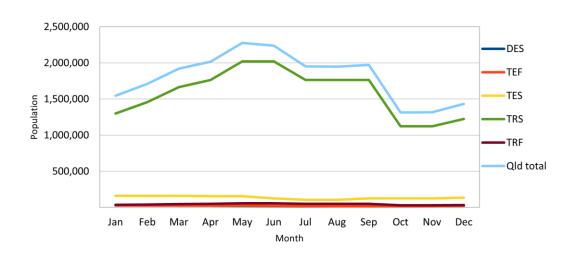
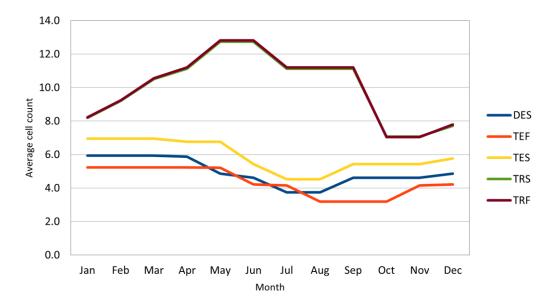


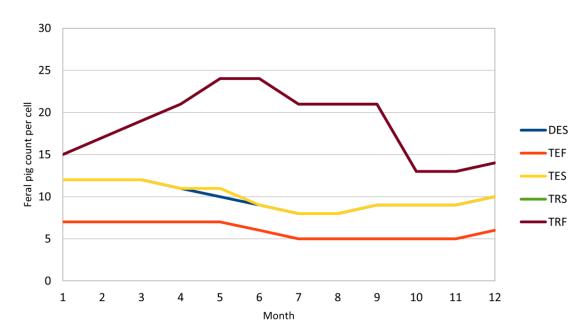
Figure 9. Modelled feral pig population in Queensland by wildlife region by month

Figure 10 illustrates the average number of feral pigs per cell, per month, by wildlife region (populated cells only). Figure 11 illustrates the 95<sup>th</sup> percentile number of feral pigs per cell per month, by wildlife region (populated cells only). The modelled population density ranges from 1.3 pigs km² in southern Qld up to 6.25 pigs km² in northern Qld are consistent with Twigg et al. (2005), Gentle & Pople (2013), Hone et al. (2019) and Gentle et al. (2019). Within-cell populations (representing sounders) range in size from 5 to 25 which is consistent with Choquenot et al. (1996), Twigg et al. (2005) and Cowled et al. (2012).



**Figure 10.** Average number of feral pigs per populated 4 km<sup>2</sup> cell per month, by wildlife region.





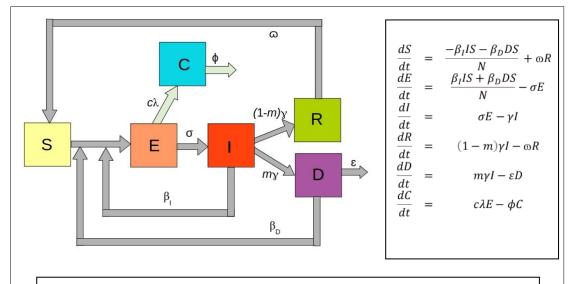
**Figure 11.** 95<sup>th</sup> percentile number of feral pigs per populated 4 km<sup>2</sup> cell per month, by wildlife region.

# 3.7 Transmission of ASF within a group of feral pigs

AADIS considers the feral pigs in a cell to be homogeneous and 'well-mixed' from a disease transmission point of view (Keeling and Rohani, 2008), i.e., all members of a cell are biologically equivalent and equally likely to contract ASF. In other words, the population of a cell is deemed to be a familial sounder. The same ODE system that models the within-herd spread of ASF is used to model the within-cell spread of ASF in feral pigs (Figure 12). A key difference to the domestic pig model is that infectious carcasses are allowed to pose a transmission risk to susceptible feral pigs. The D compartment represents not only the number of infectious carcasses in the cell but also the level of residual infectiousness of the environment near the carcass (Probst et al., 2017; Probst et al., 2019; Chenais et al., 2019; Probst et al., 2020). Carcasses remain infectious in the environment for a configurable period  $(1/\epsilon)$ , during which time they have the potential to infect susceptible feral pigs at a transmission rate of βd. The approach is adapted from an Ebola model described by Weitz and Dushoff (2015).  $\beta$ d and (1/ $\epsilon$ ) are configurable by region and by season as environmental conditions (heat, humidity, and precipitation) will influence carcass decomposition and virus viability (Probst et al., 2020). The EBM assumes that there will be no 'healthy' long-term carriers of ASFV that pose an ongoing risk of transmission (Stahl et al., 2019). Although recent research has demonstrated a carrier state (Eblé et al., 2019), this was only for historical and moderately virulent European isolates that are not associated with the current pandemic.

The parameterisation of the within-cell EBM is provided in Appendix D.





#### where

S = number of animals in the cell that are susceptible

E = number of latently infected animals in the cell

I = number of infectious animals in the cell

R = number of recovered animals in the cell

N = total number of animals in the cell (= S + E + I + R)

D = number of animals in the cell that have died from the disease

C = number of animals in the cell with clinical signs

 $\beta_I$  = transmission rate (I to S) ( average contact rate × transmission probability)

 $\sigma$  = infectiousness progression rate (1/ $\sigma$  = average duration of the latent period)

 $\gamma$  = recovery rate (1/ $\gamma$  = average duration of the infectious period)

m = average probability of ASF-related mortality

 $\omega$  = loss of immunity rate (1/ $\omega$  = average duration of the immunity period)

c = proportion of cases showing clinical signs

 $\lambda$  = clinical disease rate (1/ $\lambda$  = average duration of the incubation period)

 $\phi$  = clinical recovery rate (1/ $\phi$  = average duration of the clinical period)

 $\beta_D$  = carcass transmission rate (average contact rate × transmission probability)

 $\varepsilon$  = carcass decay rate (1/ $\varepsilon$  = average duration of carcass infectious period)

Figure 12. Within-cell SEIRDC model employed by AADIS-ASF





Figure 13. Example prevalence curves generated by the AADIS-ASF within-cell EBM

# 3.8 Transmission of ASF between groups of feral pigs

# 3.8.1 Short-range diffusive spread

Although feral pigs are quite sedentary and in general stay within their home range (Saunders and Kay, 1996; Caley 1997; Saunders and McLeod, 1999; Truvé & Lemel, 2003; Mitchell et al., 2009; Podgórski et al., 2018), home ranges can overlap resulting in the possibility of direct and indirect contact between animals in adjoining groups. This could occur, for example, from shared food/water resources, roaming solitary males, female sounders occupying overlapping home ranges, or a home range expanding due to diminished resources. In the context of a geographic automata this equates to the risk that infectious animals in one cell (referred to as the 'infectious cell'), might occasionally have direct or indirect contact with susceptible animals in neighbouring cells (referred to as 'susceptible cells'). If a contact occurs with a susceptible cell it is referred to as an 'exposed cell'. The set of neighbouring cells can be defined radially (i.e., all cells whose centroid lies within a configured radius  $R_r$  (km) of the centroid of the infectious cell), or as a Moore neighbourhood (of range  $R_m$ ).

The short-range diffusive spread of ASF between groups of feral pigs is modelled with a contact rate approach where the probability of transmission from an infectious cell to specific susceptible cells is dictated by a daily likelihood of a direct/indirect contact, in conjunction with a probability that the contact was effective. The number of daily contacts that a subject cell has with neighbouring cells is determined by sampling a Poisson distribution. This can, for example, be informed by studies on the number of direct/indirect contacts a matriarchal group might be expected to have with a nearby matriarchal group (Pepin et al., 2016; Podgórski et al., 2018). Whenever a contact is generated for an infectious cell, a candidate exposed cell is chosen from the set of neighbouring cells. The choice can be random or influenced by characteristics of the neighbouring cells (most suitable habitat or most populated). The probability that the contact is effective (i.e., results in the transmission of infection), is given by Equation 7.

$$P_d = P_b d_i(t) p(t) d_s(t) W_x$$
 (Equation 7)

where





 $p_d$  = probability of transmission between an infectious cell and a neighbouring exposed cell  $P_b$  = average probability that a contact is effective (defined per region)  $d_i(t)$  = normalised population density of the infectious cell on day t

p(t) = normalised infectious prevalence (including carcasses) of the infectious cell on day t

 $d_s(t)$  = normalised population density of the exposed cell on day t

 $W_x$  = seasonal weight (reflecting how varying environmental conditions influence virus viability, defined per month, per region)

The parameterisation of the between-cell diffusive spread pathway is provided in Appendix E.

# 3.8.2 Longer-range sporadic spread

It is possible for feral pigs to have longer range direct or indirect contact with other feral pigs through sporadic infrequent anthropogenic events such as hunters inadvertently transferring fomites or even purposely relocating animals (Spencer and Hampton, 2005; Chenais et al., 2017; Chenais et al., 2019). It is also possible for feral pigs (usually boars) to occasionally roam longer distances (Andrzejewski & Jezierski, 1978; Caley, 1997; Saunders & Bryant, 1988; Truvé & Lemel, 2003). In the context of a geographic automata spread model, this equates to the risk of an infectious cell having effective direct or indirect contacts with distant susceptible cells.

Longer range transmission of ASF between groups of feral pigs is modelled with a contact rate approach where the probability of transmission from an infectious cell to distant susceptible cells is dictated by a daily likelihood of a 'jump' contact, in conjunction with a probability that the contact was effective. The number of daily jump contacts that a subject cell has with non-adjoining cells is determined by sampling a Poisson distribution. Whenever a jump contact is generated for an infectious cell, a catchment area of candidate exposed cells is chosen by sampling distance and bearing distributions. An exposed cell is chosen from the catchment area either randomly or influenced by characteristics of the candidate cells (most suitable habitat or most populated). The frequency and distance of jumps might be informed by observations of unexpected satellite outbreaks during an actual outbreak (Schulz et al., 2019). The probability that the contact is effective is given by Equation 8.

$$P_i = P_b d_i(t) p(t) d_s(t) W_x$$
 (Equation 8)

where

 $p_i$  = probability of transmission between an infectious cell and a non-adjoining exposed cell

 $P_b$  = average probability that a contact is effective (defined per region)

 $d_i(t)$  = normalised population density of the infectious cell on day t

p(t) = normalised infectious prevalence (including carcasses) of the infectious cell on day t

 $d_s(t)$  = normalised population density of the exposed cell on day t

 $W_x$  = seasonal weight (reflecting how varying environmental conditions influence virus viability, defined per month, per region)

The parameters for the between-cell jump spread pathway are provided in Appendix E. Note, however, that this pathway was disabled for the simulations undertaken for this project as there was no data to support parameterisation.





# 3.9 Transmission of ASF from domestic pigs to feral pigs

Infectious domestic pigs can potentially transmit contagious diseases to feral pigs through direct contacts (e.g., interacting at shared water sources or boundary fences) and indirect contacts (e.g., infectious material such as effluent or other farm waste conveyed into feral pig habitat) (Guinat et al., 2016b). The likelihood of transmission will depend on:

- the prevalence of ASF on the pig farm
- the population density of nearby feral pigs
- the opportunity for direct contact between feral and domestic pigs (e.g., indoor production systems vs free-range production systems)
- the opportunity for infectious material to be conveyed outside a farm by natural means, e.g., via insects or wild birds/rodents interacting with effluent ponds
- the opportunity for infectious material to be conveyed outside a farm by humans (influenced by the biosecurity practices in place on the pig farm)
- seasonal influences on virus viability in the environment

The potential transmission of ASF from domestic pigs to feral pigs is modelled with a spatial kernel approach where all susceptible cells, whose centroid lies within a specified range R<sub>r</sub> (km) of an infectious herd are deemed at-risk on any given day.

The daily probability of ASF transmission from an infectious source herd to a particular at-risk susceptible cell is given by Equation 9.

$$P_t = P_{df} p(t) d_s(t) W_b W_i w(d) W_x$$
 (Equation 9)

where

 $p_t$  = daily probability of transmission between an infectious herd and an at-risk cell  $P_{df}$  = average daily probability of transmission between an infectious herd and an at-risk cell (per region)

p(t) = normalised infectious prevalence of the infectious herd on day t

 $d_s(t)$  = normalised population density of the at-risk cell on day t

 $W_b$  = biosecurity weight of the herd (depends on herd type)

 $W_i$  = infectivity weight of the herd (depends on herd type and herd size)

w(d) = distance weight

 $W_x$  = seasonal weight (environmental influence on virus viability, per month, per region)

The distance weight w(d) decays over the spatial kernel radius  $R_r$  to reflect how the likelihood of transmission decreases as the distance increases between an infectious herd and a susceptible cell. The model provides a choice of tailorable decay profiles (linear, exponential, Gaussian or Hayama) that are illustrated in Figure 14. Hayama (2020) aggregates all risk factors into the kernel to generate probabilities of transmission in space. AADIS simply uses the w(d) kernel to shape the decay of the probability of transmission in space.



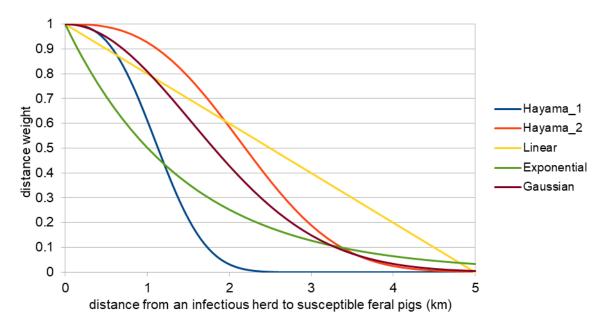


Figure 14. Examples of distance weight profiles over a 5 km (spatial kernel radial) distance

A susceptible cell can be at risk of infection from multiple infectious herds concurrently (due to overlapping spatial kernel risk areas). The parameterisation of the domestic pig to feral pig spread pathway is provided in Appendix F.

# 3.10 Transmission of ASF from feral pigs to domestic pigs

Infectious feral pigs can potentially transmit ASF to domestic pigs through direct contacts (e.g., interacting at shared water sources or boundary fences) and indirect contacts (e.g., infectious material conveyed from feral pig habitat into a pig farm) (Guinat et al., 2016b). The likelihood of transmission will depend on:

- the prevalence of ASF (including carcasses) of the feral pigs
- the population density of feral pigs near at-risk herds
- the opportunity for direct contact between feral and domestic pigs (e.g., indoor production systems vs free-range production systems)
- the opportunity for infectious material to be conveyed into a farm by natural means (e.g., via insects or wild birds/rodents)
- the opportunity for infectious material to be conveyed into a farm by humans (influenced by the biosecurity practices in place on the pig farm)
- seasonal influences on virus viability in the environment

The potential transmission of ASF from feral pigs to domestic pigs is modelled with a spatial kernel approach where all susceptible herds within a specified range  $R_{\rm fd}$  of the centroids of infectious cells are deemed at risk on any given day. The daily probability of ASF transmission from an infectious cell to an at-risk susceptible herd is given by Equation 10.

$$P_t = P_{df} p(t) d_s(t) W_b W_i w(d) W_x$$
 (Equation 10)





where

 $p_t$  = daily probability of transmission between an infectious herd and a nearby at-risk cell  $P_{fd}$  = average daily probability of transmission between an infectious herd and a nearby at-risk cell (per region)

p(t) = normalised infectious prevalence (including carcasses) of the infectious cell on day t

 $d_s(t)$  = normalised population density of the infectious cell on day t

 $W_b$  = biosecurity weight of the at-risk herd (depends on herd type)

 $W_i$  = susceptibility weight of the at-risk herd (depends on herd type and herd size)

w(d) = distance weight

 $W_x$  = seasonal weight (environmental influence on virus viability, per month, per region)

The distance weight w(d) decays over the spatial kernel radius reflecting how the probability of transmission decreases as the distance increases between an infectious cell and a susceptible herd. The model provides a choice of tailorable decay profiles (linear, exponential, Gaussian or Hayama) that are illustrated in Figure 14. The parameterisation of the feral pig to domestic pig spread pathway is provided in Appendix F.

# 3.11 Surveillance, detection, and control of ASF in feral pigs

#### 3.11.1Passive surveillance

The feral pig passive surveillance component is preliminary and will be revisited during a follow-on project. Passive surveillance is a background process that constantly scans the set of cells that have infected live animals or carcasses to assess the likelihood that ASF is detected by an 'observer'. Observers are people that may encounter feral pigs or carcasses during their regular activities. Examples include hunters, landowners, national park rangers, state forest workers, fly-in fly-out mine workers, hikers, etc. The intensity (or degree) of observation is approximated by a raster data layer that can consider such things as human population density, land use, hunting activity, accessibility, etc. In the AADIS-ASF-QLD model this is currently proxied by the human population density data layer and will be refined during future work. The accuracy of the observer is characterised by sensitivity (likelihood of a true positive) and specificity (likelihood of a true negative). The likelihood of a passive surveillance detection depends on the feral pig population density in the cell, the ASF prevalence within the cell's feral pig population and the intensity, sensitivity, and specificity of the observer. The daily probability of a true positive detection on day t is given by Equation 11.

$$p_{tp}(t) = 1 - e^{[-d(t) p(t) \ln Se]}$$
 (Equation 11)

where

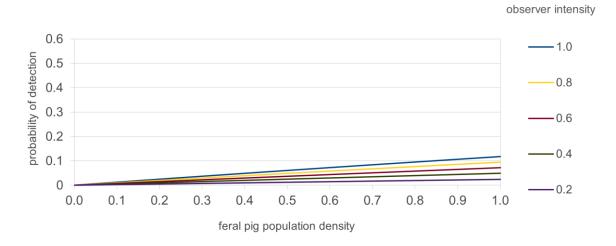
d(t) = feral pig population density on day t (space normalised)

p(t) = ASF infected prevalence (including carcasses) on day t

 $I_n$  = intensity of observers in the cell



S<sub>e</sub> = sensitivity of the observer (defined separately for managed cells vs unmanaged cells. A managed cell is any cell that is undergoing, or has undergone, active surveillance or a control action)



**Figure 15.** Probability of passive surveillance detection over a range of observer intensities for a cell with average infectious prevalence and observer sensitivity = 0.25

A true/false positive detection triggers the formation of a treatment area (Section 3.11.2), a control action inside the treatment area and active surveillance around the treatment area. The first passive detection may be stochastic (per Equation 11), fixed on a specified day, or fixed on a specified day and in a specified cell. The parameterisation of the feral pig passive surveillance component is provided in Appendix G.

## **3.11.2Control**

A feral pig 'treatment area' is formed when an ASF detection is made in a feral pig population or (optionally) when a detection is made in the domestic pig population (i.e., an IP is declared). A treatment area is an annulus with inner radius T1 and outer radius T2 (km) relative to the location of the detection (i.e., either the IP location or the centroid of the cell in which the detection was made).



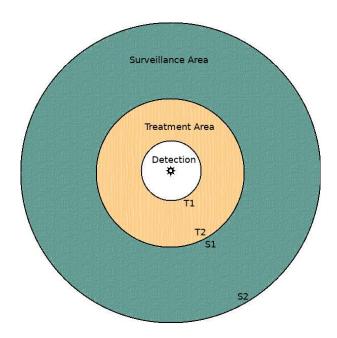


Figure 16. Feral pig treatment and surveillance areas

Control actions are conducted in each cell whose centroid lies inside a control area and are configurable in terms of duration, cost, periodicity, and effectiveness. The only control action currently implemented in AADIS-ASF is the removal of a proportion of the feral pig population ('thinning') to reduce the risk for ongoing transmission both within the feral pig population and into the domestic pig population (O'Neill et al., 2020). This includes destroying live pigs and the removal of carcasses and is represented by evenly reducing the SEIRD compartmental counts. The effectiveness of control (i.e., the resultant population knockdown) is configurable, but the actual method of thinning is not specified (e.g., ground baiting, ground shooting, aerial baiting, helicopter shooting, etc.). Control actions are costed and dynamically constrained by available resources (Section 3.11.5). The completion of control actions inside a control area triggers post-control surveillance. The parameterisation of the feral pig control component is provided in Appendix G.

There is a concern that actively reducing the feral pig population during an ASF outbreak may in fact accelerate transmission as hunting and pest control measures may cause infected animals to disperse (EFSA, 2014b; Animal Health Australia, 2020). However, this may be debatable in an Australian context. The only local studies to examine the issue (Saunders and Bryant 1988; Dexter, 1996) demonstrated no dispersal of collared feral pigs despite significant persecution (capture and collaring, monitoring and aerial shooting most of the population). In fact, only one pig was known to disperse from both studies, and this was detected sometime after the control program and therefore may not have been associated with control.

#### 3.11.3Active surveillance

A feral pig 'surveillance area' is formed when an ASF detection is made in a feral pig population or (optionally) when a detection is made in the domestic pig population (i.e., an IP is declared). A surveillance area is an annulus with inner radius S1 and outer radius S2 (km) relative to the location of the detection (i.e., either the IP location or the centroid of the cell in which the detection was made) (Figure 16).



Surveillance is carried out in each cell whose centroid lies inside a feral pig surveillance area. The user configures the duration, cost, and periodicity of surveillance.

The likelihood of a detection depends on the feral pig population density in the cell, the ASF prevalence within the cell's feral pig population and the intensity, sensitivity, and specificity of the observer. The probability of a true positive detection on day t is given by Equation 12.

$$p_{tp}(t) = 1 - e^{[-d(t) p(t) \ln Se]}$$
 (Equation 12)

where

p(t) = ASF infected prevalence (including carcasses) on day t

 $I_n$  = intensity of observers in the cell

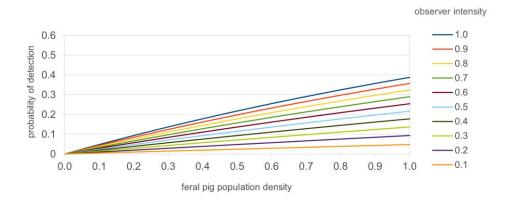
S<sub>e</sub> = sensitivity of the observer (defined separately for managed cells vs unmanaged cells.

The probability of a false positive detection is given by Equation 13.

$$p_{fp} = 1 - S_p (Equation 13)$$

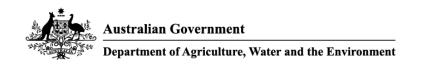
where

S<sub>p</sub> = specificity of the observer



**Figure 17.** Probability of active surveillance detection over a range of observer intensities for a cell with average infectious prevalence and observer sensitivity = 0.98

A true/false positive detection triggers the formation of a treatment area around the new detection and further surveillance outside the new treatment area to delimit the extent of infection. Surveillance actions are costed and dynamically constrained by available resources (Section 3.11.5). The parameterisation of the feral pig active surveillance component is provided in Appendix G. Note that as part of future work, active surveillance may be split into two distinct processes: searching for carcasses and surveillance via sampling of live feral pigs.





#### 3.11.4Post-control surveillance

Post-control surveillance in support of proof of freedom is triggered inside a feral pig treatment area (Figure 16) once all control actions have completed. The user configures the duration, cost, and periodicity of post-control surveillance.

The likelihood of a detection depends on the feral pig population density in the cell, the ASF prevalence within the cell's feral pig population and the intensity, sensitivity, and specificity of the observer. The probability of a true positive detection on day t is given by Equation 14.

$$p_{tp}(t) = 1 - e^{[-d(t) p(t) \ln Se]}$$
 (Equation 14)

where

p(t) = ASF infected prevalence (including carcasses) on day t

 $I_n$  = intensity of observers in the cell

S<sub>e</sub> = sensitivity of the observer

The probability of a false positive detection is given by Equation 15.

$$p_{fp} = 1 - S_p (Equation 15)$$

where

S<sub>p</sub> = specificity of the observer

A true/false positive detection triggers re-treatment plus active delimiting surveillance around the treatment area. Post-control surveillance actions are costed and dynamically constrained by available resources (Section 3.11.5). The parameterisation of the feral pig post-control surveillance component is provided in Appendix G.

## 3.11.5Resourcing

All feral pig surveillance and control actions are configured in terms of the required resources. An AADIS 'resource' is a collection of whatever is required to complete the action. For example, the resources required to conduct a ground baiting control action might be personnel, traps, bait, and a vehicle.

Resources are maintained in dynamic pools. A surveillance or control action can only occur if the required resource can be 'borrowed' from the pool, otherwise the action is queued until resources become available. Once an action has completed the resource is 'returned' to the pool. The capacity of resource pools can be configured to ramp up over time. Resourcing can operate in two modes:

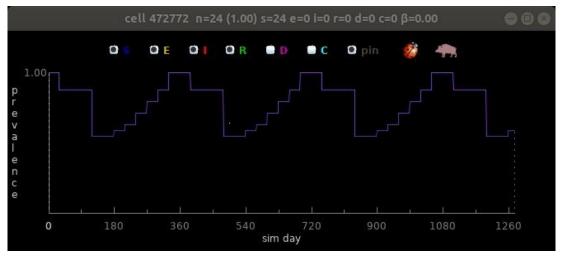
- <u>limited</u>: control actions that can't be resourced are queued and control is constrained
- <u>unlimited</u>: resources are always granted upon request and resourcing levels become a model output rather than an input

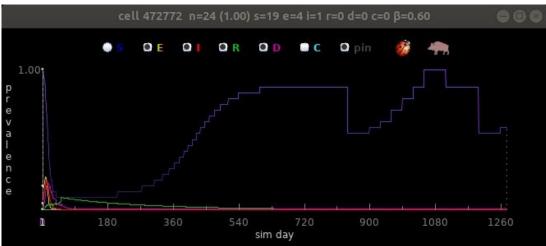


# 3.12 Feral pig population dynamics

Any cell that has feral pigs present has a population model that predicts the within-cell abundance over time (Figure 6). If the feral pigs become infected with ASF then the cell also acquires an infection model (Figure 12). A potential conflict arises between the two models as they both seek to influence the cell population size – the population model via the monthly time slices and the infection model via ASF mortality. The conflict is resolved by allowing the population model to determine the population size when the cell is not infected and allowing the infection model to determine the population size while the cell is impacted by ASF.

Once a cell is free of ASF (in both living and deceased animals) the population model regains control and allows the population to recover over time. The recovery profile is specified with a logistic growth equation with configurable recovery period, lag, and gradient. At the end of the period the population is once again determined by the population model time slices. An example of a population recovery is provided in Figure 18. The parameterisation is provided in Appendix D.





**Figure 18.** A cell the day before infection (top) and the day after infection (bottom) illustrating the population being knocked down and then recovering over time





## 4 IMPLEMENTATION HIGHLIGHTS

AADIS is an agent-based epidemiological modelling framework where agents have embedded equation-based models (representing the spread of a pathogen within the agent) and interact in a highly concurrent spatially explicit environment (Bradhurst, 2015). AADIS facilitates the modelling of contagious livestock disease (Bradhurst et al., 2015), insect vector-borne livestock disease (Kompas et al., 2018; Al-Riyami, 2021), transboundary livestock disease (Bradhurst et al., 2021b), plant and environmental pests (Bradhurst et al., 2021a), and human disease (Geard et al., 2021).

Over the course of this project, the framework was expanded to accommodate the modelling of contagious disease in a wild population and the epidemiological interface between livestock and a wild population. From the (generalised) AADIS framework perspective, the term 'wildlife' is used to refer to any wild population and could refer to feral pigs, insect vectors, tramp ants, etc. From the specific AADIS-ASF-QLD perspective the wild population is specifically feral pigs.

# 4.1 Agent-based model

The agent-based model subsystem was expanded with:

- new wildlife (cell-based) agents that can have both a population model and an infection model
- new wildlife jump and diffusion spread pathways, each operating concurrently on dedicated Java threads
- new wildlife passive surveillance, active surveillance, control, and post-control surveillance components, all operating concurrently on dedicated Java threads
- new dynamic resource pools for the wildlife surveillance and control components
- a new 'directed' option for the direct spread pathway that allows direct contacts between farms and saleyards to be either stochastically derived (via the legacy algorithm) or driven by the replay of historical NLIS movements

## 4.2 Equation-based model

The within-agent SEIR infection model (ODE system) was expanded with:

- a new D (deceased) compartment (representing the number of infectious carcasses as driven by the configured case fatality rate)
- a new relationship between the D and S (susceptible) compartments (representing the transmission risk to susceptible feral pigs from infectious carcasses and their surrounding contaminated environment)
- a new D compartment decay rate (representing the predation and decomposition of infectious carcasses over time until they (and their immediate environment) no longer pose a transmission risk.





# 4.3 Population model

Prior to this project, the only cell-based population model available in the AADIS framework was logistic growth (with optional temperature-dependent growth rate) (Bradhurst 2021a). A new AADIS population model was created that allows population counts to vary stepwise per cell over a configurable time interval (i.e., time slices). The new population model also provides configurable population recovery (based on 5-parameter logistic growth) after knockdown or control.

# 4.4 Graphical user interface

The AADIS Graphical user interface subsystem was updated with

- new visualisations layers for wildlife habitat suitability, wildlife regions, wildlife presence, wildlife outbreaks, wildlife infection networks, and wildlife surveillance and control
- new dialogs for configuring wildlife presence, within-cell transmission, between-cell transmission via diffusion and jumps, transmission to and from domestic animals, surveillance, and control
- new monitors for visualising wildlife outbreaks, wildlife surveillance and control, and the resourcing profile for wildlife surveillance and control
- expansion of the cell popup to display habitat, region, population, infection attributes
- expansion of the cell population popup to display infection status

A selection of screenshots is provided in Appendix H.

#### 4.5 Database

The AADIS relational database subsystem was expanded with four new tables:

- directed\_movements historical NLIS movements to inform the risk of transmission via direct contacts
- management\_group allowing linkages of herds based on shared management/owenership
- wildlife\_population population counts per cell (time slices)
- wildlife\_region key wildlife ecology and epidemiology parameters per wildlife region and in some cases per season

# 4.6 Configuration

The AADIS Configuration subsystem was expanded to allow the configuration of all new model parameters via the disease and scenario configuration files.

## 4.7 Reporting

The AADIS reporting subsystem was updated with a new wildlife summary report that writes a variety of model outcomes for wildlife disease transmission, surveillance, and control.





# 5 SENSITIVITY ANALYSIS

Sensitivity analysis is a technique of systematically varying model parameters to gauge their relative influence on scenario outcomes. It is particularly useful for assessing the influence of parameters that are naturally variable, subject to chance, or uncertain due to inadequate data (Taylor, 2003). It is important that modellers and the users of models know how the quality of specific data influences model outcomes. This helps funnel effort into improving the quality of key data, in lieu of data that does not materially impact the model outputs (Green and Medley, 2002; Taylor, 2003). The identification of parameters that strongly influence scenario outcomes is also useful from an epidemiological perspective, and can then inform planning and preparedness activities, such as a cost benefit analysis of proactive surveillance.

# 5.1 The influence of regionality and seasonality on feral pig outbreaks

#### **5.1.1** Method

The spread and control of ASF in domestic pigs was disabled. Diffusive spread of ASF in feral pigs was enabled and jump spread was disabled. ASF was seeded in each of the wildlife regions in cells with an average-sized feral pig population and a large feral pig population (relative to the region and the season). The model was allowed to run for a maximum of 720 days or until ASF had faded out. Each scenario was repeated 100 times for both summer and winter.

#### 5.1.2 Results

Descriptive statistics are provided in Table 7 for each of the Queensland wildlife regions. The outbreak duration, outbreak size, spread distance, and spread rate outcomes pertain only to those outbreaks that had secondary spread (beyond the seed cell). Boxplots are provided in Figure 19 for the outbreak duration size in winter and summer for the high-density seed cells. The boxplots depict the mean (x), median (line), interquartile range, and outliers.

Table 7. The influence of regionality and seasonality on diffusive ASF spread in feral pigs

Region 1 (DES) - Deserts & xeric shrublands					
	summer	winter	summer	winter	
seed cell density (pigs/km²)	1.8	1.3	3.3	2.0	
seed cell relative density	average	average	high	high	
outbreaks faded-out	100%	100%	100%	100%	
outbreaks with secondary spread	21%	41%	42%	41%	
duration mean[range] (days)	45[36-62]	49[41-68]	67[47-155]	57[42-80]	
size mean[range] (km²)	9[4-16]	9[4-16]	13[4-20]	11[4-28]	
spread distance mean[range] (km)	2[2-3]	3[2-6]	3[2-8]	3[2-6]	
spread rate mean[range] (km/year)	19[12-28]	22[14-34]	19[10-42]	20[11-45]	
simulation time per outbreak (secs)	13.4	15.5	16.7	16.4	

Region 4 (TEF) - Temperate broadleaf & mixed forest)						
summer winter summer winter						
seed cell density (pigs/km²)	1.8	1.3	3.5	2.5		
seed cell relative density	average	average	high	high		
outbreaks faded-out	100%	100%	100%	100%		
outbreaks with secondary spread	18%	37%	36%	50%		



duration mean[range] (days)	48[44-56]	152[151-153]	80[54-157]	169[154-191]
size mean[range] (km²)	9[4-12]	9[4-16]	11[4-20]	12[4-24]
spread distance mean[range] (km)	2[2-3]	3[2-5]	3[2-7]	3[2-7]
spread rate mean[range] (km/year)	19[12-24]	6[5-11]	14[9-22]	7[4-15]
simulation time per outbreak (secs)	15.3	38.4	19.1	39.6

Region 5 (TES) - Temperate grasslands, savannas & shrublands					
	summer	winter	summer	winter	
seed cell density (pigs/km²)	1.8	1.3	3.3	2.0	
seed cell relative density	average	average	high	high	
outbreaks faded-out	100%	100%	100%	100%	
outbreaks with secondary spread	24%	35%	30%	49%	
duration mean[range] (days)	44[36-58]	152[151-153]	66[47-169]	158[153-184]	
size mean[range] (km²)	9[4-12]	2[1-4]	10[4-28]	11[4-24]	
spread distance mean[range] (km)	3[2-3]	3[2-3]	3[2-4]	3[2-6]	
spread rate mean[range] (km/year)	20[13-29]	6[5-7]	15[8-25]	7[4-13]	
simulation time per outbreak (secs)	13.3	37.6	15.6	39.3	

Region 6 (TRS) - Tropical & subtropical grasslands, savannas & shrublands					
	summer	winter	summer	winter	
seed cell density (pigs/km²)	2.3	3.0	3.8	5.3	
seed cell relative density	average	average	high	high	
outbreaks faded-out	100%	100%	100%	100%	
outbreaks with secondary spread	27%	43%	39%	60%	
duration mean[range] (days)	49[37-92]	86[49-154]	60[39-176]	105[52-179]	
size mean[range] (km²)	11[4-24]	17[4-64]	13[4-56]	25[4-84]	
spread distance mean[range] (km)	3[2-6]	4[2-11]	4[2-12]	5[2-12]	
spread rate mean[range] (km/year)	23[14-34]	18[10-33]	23[12-42]	18[9-32]	
simulation time per outbreak (secs)	12.6	18.5	14.7	23.2	

Region 7 (TRF) - Tropical & subtropical moist broadleaf forests					
	summer	winter	summer	winter	
seed cell density (pigs/km²)	2.3	3.0	3.8	5.3	
seed cell relative density	average	average	high	high	
outbreaks faded-out	100%	100%	100%	100%	
outbreaks with secondary spread	32%	34%	54%	56%	
duration mean[range] (days)	43[33-72]	48[38-60]	86[43-184]	105[54-227]	
size mean[range] (km²)	9[4-12]	9[4-24]	19[4-76]	25[4-80]	
spread distance mean[range] (km)	3[2-5]	3[2-5]	4[2-15]	5[2-10]	
spread rate mean[range] (km/year)	24[13-43]	21[11-29]	19[10-36]	17[7-33]	
simulation time per outbreak (secs)	13.7	15.0	19.1	23.1	



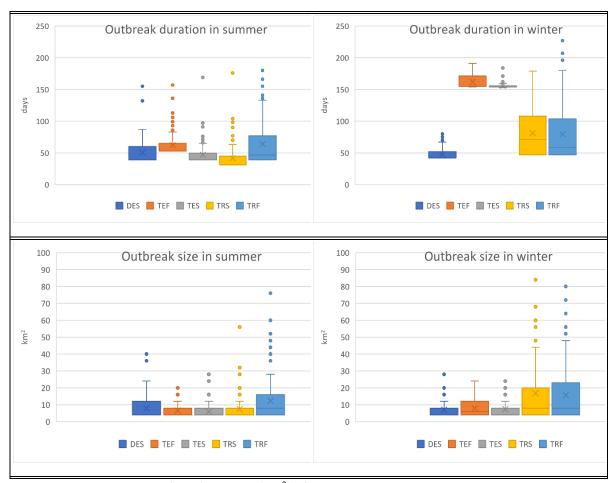
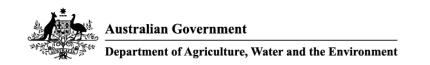


Figure 19. Duration (days) and size (km²) of simulated outbreaks per season, per region

## 5.1.3 Discussion

The simulated outbreaks in the northern tropical regions (TRS and TRF) were larger than those in the southern temperate (TEF and TES) regions and the desert and xeric shrubland region (DES). This is consistent with the high numbers and density of feral pigs in tropical north Qld and the density-dependent nature of diffusive spread. Winter outbreaks in the tropical grasslands, savannas and shrublands region (TRS) were much larger, longer, and more likely to involve secondary spread than summer outbreaks. The distinction between summer and winter outbreaks was less pronounced in the tropical moist broadleaf forests region (TRF) than the TRS region. This is reasonable as tropical forests provide a more consistent habitat for feral pigs than tropical grasslands and savanna (which experience greater seasonal variations). Winter outbreaks in the southern temperate regions (TEF and TES) were longer, and more likely to involve secondary spread than summer outbreaks. This is consistent with the cooler temperatures of winter leading to slower decay carcass decay and increased residual infectiousness in the environment (Schulz et al., 2019). The size and duration of outbreaks in the DES region appeared to be driven more by feral pig population density than season. This could be an artifact of the narrow distribution and low numbers of feral pigs in the desert and xeric shrublands of central Queensland (Figure 8, Table 6, Figure 9).

Spread rates were generally higher in the high feral pig density temperate southern regions (TEF and TES) than the lower density southern temperate (TEF and TES) and central desert (DES)





regions. This is consistent with observations of ASF in wild boar in Europe (EFSA, 2020). Spread rates were higher in summer in the temperate southern regions (TEF and TES) and northern tropical regions (TRS and TRF). The spread rates in the central desert/xeric region (DES) were marginally higher in winter than summer. The overall median spread rate was 19 km/year. This is consistent with overseas estimates of the non-anthropogenic spread rate of ASF in wild boar of 8-24 km/year (Śmietanka et al., 2016; EFSA, 2018a; Wozniakowski et al., 2021; Podgórski & Śmietanka, 2018; Taylor et al., 2020).

# 5.2 The influence of population density on feral pig outbreaks

#### 5.2.1 Method

A high-density feral pig population layer was loaded into the model with abundance estimates four-times that of the baseline population layer (Section 3.6.3). This raised the number of feral pigs in Queensland from approximately 2 million to approximately 9 million. The spread and control of ASF in domestic pigs was disabled. Diffusive spread of ASF in feral pigs was enabled and jump spread was disabled. ASF was seeded in each of the wildlife regions in cells with an average-sized feral pig population and a large feral pig population (both relative to the high-density population layer). This resulted in 'very high' and 'extreme' feral pig densities relative to the 'average' and 'high' densities of the baseline population layer. The model was allowed to run for a maximum of 720 days or until ASF had faded out in the feral population.

#### 5.2.2 Results

Descriptive statistics are provided in Table 8 for each of the Queensland wildlife regions. The outbreak duration, outbreak size, spread distance, and spread rate outcomes pertain only to those outbreaks that had secondary spread (beyond the seed cell). Boxplots are provided in Figure 20 for the outbreak duration size in winter and summer for the high-density seed cells. Boxplots are provided in Figures 21-23 for the duration and size of outbreaks for selected regions by feral pig density. The boxplots depict the mean (x), median (line), interquartile range, and outliers.

**Table 8.** The influence of high-range population density on diffusive ASF spread in feral pigs

Region 1 (DES) - Deserts & xeric shrublands					
	summer	winter	summer	winter	
seed cell density (pigs/km²)	7.5	4.5	14.3	8.8	
seed cell relative density	very high	very high	extreme	extreme	
outbreaks faded-out	100%	100%	100%	100%	
outbreaks with secondary spread	46%	55%	65%	81%	
duration mean[range] (days)	117[59-289]	108[62-200]	150[68-371]	132[69-224]	
size mean[range] (km²)	23[4-160]	22[4-72]	31[4-144]	27[4-100]	
spread distance mean[range] (km)	5[2-13]	5[2-167]	5[2-16]	5[2-12]	
spread rate mean[range] (km/year)	14[6-28]	17[8-35]	12[5-20]	13[6-26]	
simulation time per outbreak (secs)	27.1	27.9	33.5	36.4	

Region 4 (TEF) - Temperate broadleaf & mixed forest					
summer winter summer winter					
seed cell density (pigs/km²)	7.8	5.5	14.8	8.4	



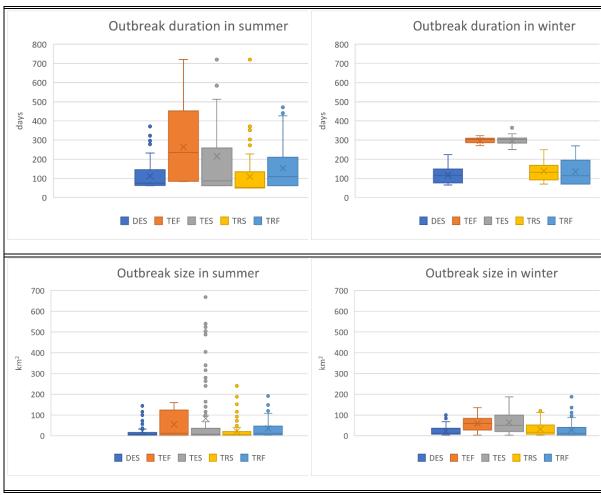
seed cell relative density	very high	very high	extreme	extreme
outbreaks faded-out	100%	100%	99%	100%
outbreaks with secondary spread	45%	55%	70%	92%
duration mean[range] (days)	138[70-396]	228[212-274]	369[86-720]	300[271-323]
size mean[range] (km²)	15[4-88]	12[4-44]	77[4-160]	59[4-136]
spread distance mean[range] (km)	4[2-19]	3[2-9]	8[2-13]	8[2-18]
spread rate mean[range] (km/year)	10[4-17]	5[3-13]	8[3-16]	10[3-21]
simulation time per outbreak (secs)	30.8	60.9	69.4	79.4

Region 5 (TES) - Temperate grasslands, savannas & shrublands					
	summer	winter	summer	winter	
seed cell density (pigs/km²)	7.5	4.5	14.0	8.5	
seed cell relative density	very high	very high	extreme	extreme	
outbreaks faded-out	100%	100%	89%	100%	
outbreaks with secondary spread	40%	65%	61%	92%	
duration mean[range] (days)	140[60-432]	243[204-275]	342[73-720]	302[251-364]	
size mean[range] (km²)	20[4-128]	23[4-96]	134[4-676]	69[4-188]	
spread distance mean[range] (km)	4[2-20]	5[2-12]	10[2-31]	8[2-15]	
spread rate mean[range] (km/year)	12[4-20]	7[3-16]	11[5-24]	9[2-17]	
simulation time per outbreak (secs)	27.7	61.7	59.1	78.1	

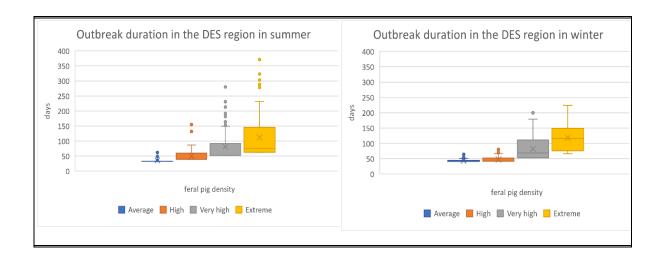
Region 6 (TRS) - Tropical & subtropical grasslands, savannas & shrublands					
	summer	winter	summer	winter	
seed cell density (pigs/km²)	9.5	13.3	15.8	22.5	
seed cell relative density	very high	very high	extreme	extreme	
outbreaks faded-out	100%	100%	99%	100%	
outbreaks with secondary spread	58%	69%	50%	80%	
duration mean[range] (days)	117[52-373]	142[69-261]	170[56-720]	157[79-249]	
size mean[range] (km²)	22[4-144]	36[4-152]	40[4-240]	40[4-120]	
spread distance mean[range] (km)	5[2-19]	7[2-17]	7[2-22]	7[2-14]	
spread rate mean[range] (km/year)	15[8-29]	16[7-36]	15[7-26]	15[6-29]	
simulation time per outbreak (secs)	27.6	35.7	34.0	40.5	

Region 7 (TRF) - Tropical & subtropical moist broadleaf forests					
	summer	winter	summer	winter	
seed cell density (pigs/km²)	9.5	13.5	15.8	22.5	
seed cell relative density	very high	very high	extreme	extreme	
outbreaks faded-out	100%	100%	100%	100%	
outbreaks with secondary spread	60%	46%	75%	66%	
duration mean[range] (days)	96[54-354]	114[62-266]	187[69-471]	172[77-270]	
size mean[range] (km²)	15[4-68]	21[4-128]	46[4-204]	41[4-188]	
spread distance mean[range] (km)	4[2-14]	4[2-15]	7[2-18]	7[2-18]	
spread rate mean[range] (km/year)	14[7-20]	14[6-32]	13[6-24]	14[6-25]	
simulation time per outbreak (secs)	25.5	28.5	43.9	40.4	

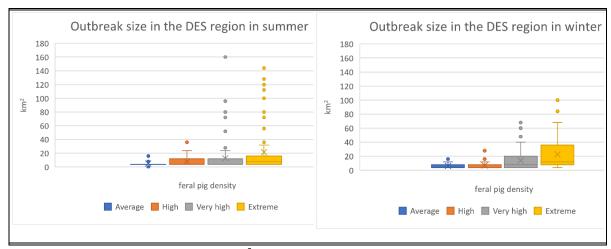




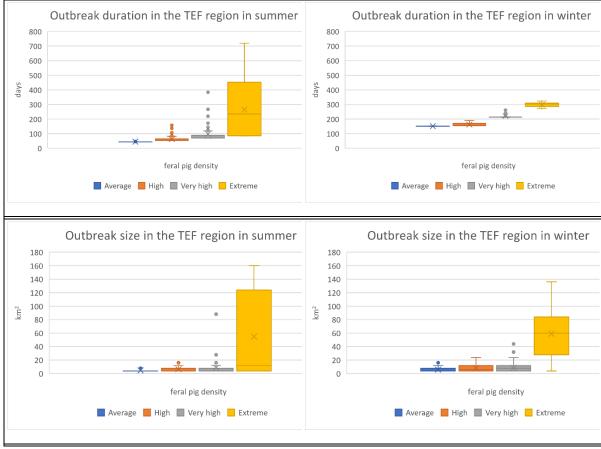
**Figure 20.** Duration (days) and size (km²) of simulated outbreaks per season per region for the high-density feral pig population layer







**Figure 21.** Duration (days) and size (km²) of simulated outbreaks in the DES region for varying feral pig densities



**Figure 22.** Duration (days) and size (km²) of simulated outbreaks in the TEF region for varying feral pig densities



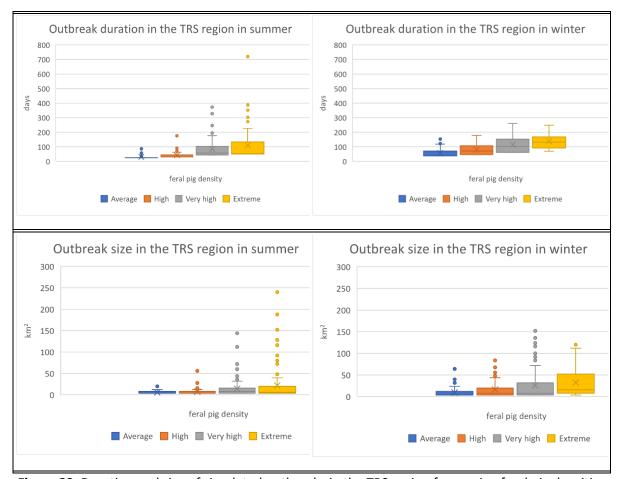


Figure 23. Duration and size of simulated outbreaks in the TRS region for varying feral pig densities

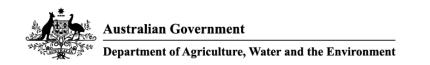
#### 5.2.3 Discussion

The seasonal outbreak patterns observed for the high-density feral pig population layer were broadly similar to those observed for the baseline feral pig population layer (Section 5.1.1). Winter outbreaks in the northern tropical regions (TRS and TRF) and the southern temperate regions (TEF and TES) were generally longer, larger, and more likely to involve secondary spread than summer outbreaks (Table 8). As before, the distinction between summer and winter outbreaks was less pronounced in the central desert and xeric shrubland region (DES) and the tropical & subtropical moist broadleaf forests region (TRF). Summer outbreaks tended to exhibit more variability than winter outbreaks.

For all regions, as feral pig density increases, so too does the likelihood of secondary spread, larger outbreaks, and longer outbreaks. For brevity, only the DES, TES and TRF regions are presented in Figures 20-23.

Average spread rates ranged 5-16 km/year with a median of rate of 14 km/year. Although lower than the rates observed for the baseline feral pig population, it is still consistent with overseas estimates of the non-anthropogenic spread rate of ASF in wild boar of 8-24 km/year.

As the feral pig density increased, so too did outbreak size and duration. These results indicate density-dependent transmission of ASF between sounders (Podgórski et al., 2020). The model may





thus be useful in assessing how targeted reductions in feral pig density may influence the risk of transmission in the domestic pig population (EFSA, 2018b).

# 5.3 The influence of feral pig density, contact rates, and spillover transmission on domestic outbreaks

#### 5.3.1 Method

ASF was introduced into a small commercial farm (ID=59, number of pigs=1200) in June near the town of Dalby, Queensland in region TRS. The feral pig population density and the probability of transmission between domestic and feral pigs was varied to gauge the impact on the size and duration of domestic outbreaks. Detection of ASF in the domestic pig population was fixed at day 28, at which point the default ASF control measures were applied (Appendix C). The feral pig diffusion pathway was enabled, and the jump pathway was disabled. Feral pig surveillance and control was disabled. The scenarios were run until there were no more IPs or infected cells, up until a maximum scenario length of 365 days. Each scenario variant was run 100 times.

Note that this scenario was crafted to always have secondary spread from the seed herd (and thus consistent outbreaks) by timing the introduction of ASF into the herd the day before a known scheduled direct movement of animals out of the herd. This was done to allow differential patterns arising from the systematic variation of feral pig densities, contact rates, and spillover transmission probabilities to be clearer.

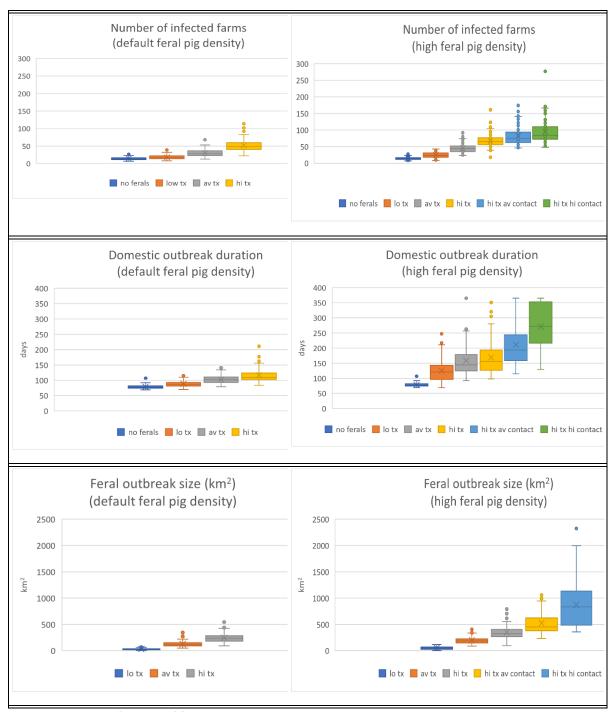
#### 5.3.2 Results

**Table 9.** The influence of feral pig population density, contact rates, and spillover transmission on the size and duration of outbreaks in domestic and feral pigs

Feral pig density	Spillover transmission probabilities	Inter- sounder contact rates	Infected farms mean [range]	Domestic outbreak length (days) mean[range]	Feral outbreak size (km²) mean[range]	Feral outbreak length (days) mean[range]	Feral fade out (%)
zero	0	default <sup>6</sup>	15[7-30]	79[69-107]	0	0	100
default <sup>1</sup>	default <sup>3</sup>	default <sup>6</sup>	18[8-39]	89[70-116]	29[0-80]	155[122-183]	100
default <sup>1</sup>	medium <sup>4</sup>	default <sup>6</sup>	30[12-68]	102[79-148]	125[48-344]	172[149-210]	100
default <sup>1</sup>	high⁵	default <sup>6</sup>	52[22-114]	116[84-211]	243[92-596]	176[145-189]	100
high <sup>2</sup>	default <sup>3</sup>	default <sup>6</sup>	25[8-43]	125[69-247]	50[0-116]	265[126-336]	100
high <sup>2</sup>	medium <sup>4</sup>	default <sup>6</sup>	46[23-92]	158[92-365]	196[88-404]	293[268-365]	98
high <sup>2</sup>	high <sup>5</sup>	default <sup>6</sup>	68[18-161]	169[98-351]	351[96-796]	301[272-365]	95
high <sup>2</sup>	high <sup>5</sup>	medium <sup>7</sup>	83[46-174]	211[115-365]	525[232-1056]	310[270-365]	87
high <sup>2</sup>	high <sup>5</sup>	high <sup>8</sup>	96[48-277]	271[129-365]	873[356-2324]	335[287-365]	47



<sup>8</sup>high inter-sounder contact rates (Appendix E x 3)



**Figure 24.** The influence of feral pig population density and spillover transmission probabilities on the size and duration of outbreaks in domestic and feral pigs

<sup>&</sup>lt;sup>1</sup>default feral pig population (approx. 2M total in Qld)

<sup>&</sup>lt;sup>2</sup>high feral pig population (approx. 9M total in Qld)

<sup>&</sup>lt;sup>3</sup>default spillover transmission probabilities (0.025, 0.05)

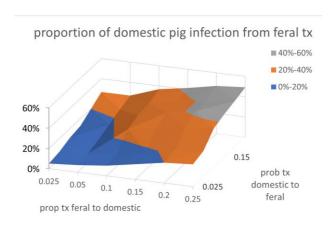
<sup>&</sup>lt;sup>4</sup>medium-high spillover transmission probabilities (0.05, 0.1)

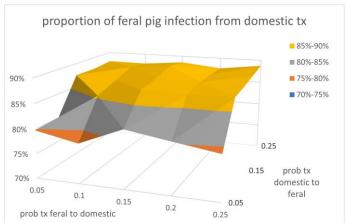
<sup>&</sup>lt;sup>5</sup>high spillover transmission probabilities (0.075, 0.15)

<sup>&</sup>lt;sup>6</sup>default inter-sounder contact rates (Appendix E)

<sup>&</sup>lt;sup>7</sup>medium-high inter-sounder contact rates (Appendix E x 2)







**Figure 25.** The influence of spillover transmission probabilities on the sources of infection in domestic and feral outbreaks

#### 5.3.3 Discussion

There is a clear signal in this scenario that the density of feral pigs, the level of direct/indirect contact between feral pigs, and the level of direct/indirect contact between domestic and feral pigs (proxied by spillover transmission probabilities), strongly influenced the size and duration of ASF outbreaks in domestic pigs. In the baseline case of no feral pig involvement, the domestic outbreaks involved on average 15 farms and lasted 79 days. Outbreaks worsened significantly as feral pig density, spillover transmission probability, and feral pig contact rates increased, ramping up to an average of 96 infected farms over a duration of 271 days. Perhaps more importantly, domestic outbreaks became more variable (i.e., unpredictable) with increases in feral pig density, contact between feral pig groups, and contact between domestic and feral pigs. The influence of feral pigs would probably increase even further in a region such as TEF which has longer periods of carcass infectiousness and/or the inclusion of feral pig anthropogenic transmission jumps.

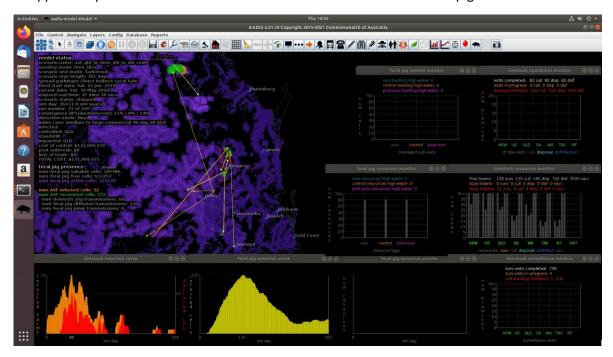
The size and duration of feral outbreaks of ASF were also highly sensitive to feral pig density, the level of direct/indirect contact between domestic and feral pigs, and the contact rate between sounders. The default values for feral pig density, spillover transmission probabilities, and intersounder contact rates lead to outbreaks in feral pigs spanning 29 km², lasting 155 days and 100% fade-out. Outbreaks worsened significantly as feral pig density, spillover transmission probability, and feral pig contact rates increased, ramping up to an average of 873 km², an average duration of 355 days, and only 47% fade-out.

Figure 26 is a screenshot of AADIS-ASF-QLD configured with high feral pig densities, high spillover transmission probabilities, and high inter-sounder contact rates. The bottom left-hand corner of the screen depicts four discrete domestic outbreaks that have arisen through recurring spillover events. The bottom centre of the screen shows that the feral pig outbreak is still well established at the end of the simulation on day 365 and is in fact ramping up again.

This scenario is of course very specific to the seed herd, the study area region, and the time of year. Ideally, the scenario would be repeated for a range of seed herd types, regions, and start



dates, however this was not possible due to time constraints. Nonetheless, the scenario demonstrates that feral pig density, spillover transmission probabilities, and intersounder contact rates are key model 'dials' and will influence emergent outbreak size, duration, and infection networks. This reinforces the critical requirement for the model to be informed by reliable localised data on feral pig distribution and abundance, inter-sounder contact rates, and the level of opportunity for direct and indirect contact between feral and domestic pigs.



**Figure 26.** Screenshot of AADIS-ASF-QLD configured with high feral pig densities, high spillover transmission probabilities, and high inter-sounder contact rates.





# 6 VERIFICATION AND VALIDATION

Verification is the process of ensuring that a model has been implemented correctly (Dent and Blackie, 1979; Sargent, 2013). It targets the mechanics of the software development process and helps assess whether 'the product has been built right'. Validation, on the other hand, looks at the bigger operational picture and helps assess whether 'the right product has been built' (Siviy et al., 2007). It is very challenging to validate epidemiological models when the subject disease has never been detected in the subject country. The AADIS framework has been through extensive verification (Bradhurst, 2015) and validation (Bradhurst, 2015; Bradhurst et al., 2015; Bradhurst et al., 2016) in the context of FMD. Whilst these activities provide confidence in the underlying software processes and broad epidemiological principles of the model, every new disease that is modelled requires a separate validation activity to provide confidence in its fitness for purpose. In the absence of field data on ASF outbreaks in Australia, options for validating ADIS-ASF include:

- adapting the model to a country that has experienced an ASF outbreak (such as Germany or South Korea) and comparing the modelled outcomes with field data
- a comparative validation where several different ASF models are adapted to a common study area and the simulated outbreak results compared (for example, Rawdon et al., (2018) and Roche et al., (2015)).

Adapting a data-driven model to a new study area is a complex process, particularly when both detailed livestock and wildlife data are required. The above validation options would be separate projects themselves and it was not feasible to include them in this project which was focussed on model design and development. Instead, some exploratory simulations and parameter sensitivity analyses were undertaken with the results assessed for congruency with literature on international outbreaks and modelling studies, and expectations of local experts. These activities included:

- isolating the silent spread phase of an ASF outbreak to gauge the influence of production system characteristics and biosecurity on transmission mechanisms (direct movements, indirect movements, and feral pigs) (Section 6.1 and 6.2)
- case studies that include transmission and control of ASF in the domestic and feral pig populations to assess the overall emergent behaviour of the model (Section 7)
- sensitivity analysis on the influence of regionality, seasonality, population density, contact rates, and spillover events on ASF outbreaks (Section 5).

# 6.1 Silent spread in domestic pig-only outbreaks

## 6.1.1 Method

ASF was randomly seeded into herds of each herd type and allowed to spread unchecked for 28 days. Outbreaks were not seeded in SGT herds as there are only two herds of this type in the dataset and this would not provide an adequate sample space. The time of introduction was fixed to be 1<sup>st</sup> June (i.e., winter). 500 iterations were run for each herd type. Feral pig transmission was disabled.



## 6.1.2 Results

**Table 10.** 28-day silent spread outbreaks in domestic pigs for different seed herd types

seed herd type					
outbreak outcome	VLC	MLC	SC	SH	PK
number of infected farms (mean)	6.3	4.3	2.8	1.4	1.2
number of infected farms (min, median, max)	1,6,19	1,3,18	1,2,17	1,1,14	1,1,8
outbreaks with no secondary spread (%)	1.6	10.8	39.4	74.0	89.8
local spread (%)	2.5	4.6	14.9	48.6	52.5
direct spread (%)	30.6	37.4	37.5	21.2	16.3
saleyard spread (%)	0.0	0.1	0.0	0.9	2.5
indirect spread (%)	66.9	58.0	47.6	29.3	28.8
transmissions into VLC herds (%)	26.1	20.6	11.0	4.1	3.8
transmissions into MLC herds (%)	41.7	37.0	35.6	9.0	6.3
transmissions into SC herds (%)	26.9	34.3	31.6	19.4	8.8
transmissions into SH herds (%)	2.0	3.7	10.9	20.7	18.8
transmissions into PK herds (%)	2.7	3.8	10.4	46.8	62.5
simulation time per outbreak (secs)	1.5	1.5	2.4	1.6	1.4

#### 6.1.3 Discussion

Outbreaks that began in commercial farms (VLC, MLC, SC) mainly involved other commercial farms and transmission was mainly due to indirect and direct spread. This is expected as commercial farms generally interact with other commercial farms and there are limited opportunities for direct or indirect contacts with non-commercial farms (smallholders and pig keepers). Outbreaks that began in non-commercial farms mainly involved other non-commercial farms and transmission was mainly due to local spread and indirect spread. This is expected as non-commercial farms generally have fewer biosecurity measures in place and less pig movements than commercial farms.

Outbreaks that began in commercial farms were larger than outbreaks that began in non-commercial farms where there was a much higher probability of fade out with no secondary spread. This is expected as non-commercial farms generally have smaller herd sizes and less frequent and more irregular opportunities for direct and indirect contacts than commercial farms.

These results are consistent with overseas experience (Bellini et al., 2016; Oļševskis et al., 2016; Chenais et al. 2019; Boklund et al., 2020) and local expectations.





# 6.2 Silent spread in outbreaks that involve domestic and feral pigs

## 6.2.1 Method

ASF was randomly seeded into herds of each herd type in winter and allowed to spread unchecked for 28 days. 500 iterations were run for each herd type. Feral pig transmission was enabled.

# 6.2.2 Results

Table 11. 28-day silent spread outbreaks in domestic & feral pigs for different seed herd types

VLC	MLC	SC	SH	PK
6.6	4.2	3.4	1.8	2.0
1,6,22	1,3,19	1,2,16	1,2,11	1,1,14
1.2	12.6	26.6	48.8	56.8
59.0	61.2	66.8	83.0	61.2
2.2	2.2	2.7	2.2	3.8
1,2,12	1,2,12	1,2,11	1,2,8	1,3,17
2.3	4.7	9.1	2.7	13.1
30.4	36.1	23.1	0.5	2.5
0.1	0.1	0.0	0.5	1.0
64.7	52.9	39.0	27.0	16.9
2.6	6.2	28.8	69.2	66.6
26.4	18.2	8.9	0.0	0.8
38.8	37.5	26.9	0.7	2.5
28.5	32.5	26.5	16.6	9.2
2.3	4.7	13.9	43.9	41.1
3.7	6.3	23.6	38.5	46.3
1.4	6.1	4.4	0.0	0.3
4.2	11.2	4.4	0.0	1.2
47.9	21.4	27.9	15.4	11.8
2.8	18.4	18.8	42.7	43.8
43.7	39.8	43.8	41.9	42.7
4.5	5.1	2.4	2.4	2.4
	6.6  1,6,22  1.2  59.0  2.2  1,2,12  2.3  30.4  0.1  64.7  2.6  26.4  38.8  28.5  2.3  3.7  1.4  4.2  47.9  2.8  43.7	6.6       4.2         1,6,22       1,3,19         1.2       12.6         59.0       61.2         2.2       2.2         1,2,12       1,2,12         2.3       4.7         30.4       36.1         0.1       0.1         64.7       52.9         2.6       6.2         26.4       18.2         38.8       37.5         28.5       32.5         2.3       4.7         3.7       6.3         1.4       6.1         4.2       11.2         47.9       21.4         2.8       18.4         43.7       39.8	6.6       4.2       3.4         1,6,22       1,3,19       1,2,16         1.2       12.6       26.6         59.0       61.2       66.8         2.2       2.2       2.7         1,2,12       1,2,12       1,2,11         2.3       4.7       9.1         30.4       36.1       23.1         0.1       0.1       0.0         64.7       52.9       39.0         2.6       6.2       28.8         26.4       18.2       8.9         38.8       37.5       26.9         28.5       32.5       26.5         2.3       4.7       13.9         3.7       6.3       23.6         1.4       6.1       4.4         4.2       11.2       4.4         47.9       21.4       27.9         2.8       18.4       18.8         43.7       39.8       43.8	6.6       4.2       3.4       1.8         1,6,22       1,3,19       1,2,16       1,2,11         1.2       12.6       26.6       48.8         59.0       61.2       66.8       83.0         2.2       2.2       2.7       2.2         1,2,12       1,2,12       1,2,11       1,2,8         2.3       4.7       9.1       2.7         30.4       36.1       23.1       0.5         0.1       0.1       0.0       0.5         64.7       52.9       39.0       27.0         2.6       6.2       28.8       69.2         26.4       18.2       8.9       0.0         38.8       37.5       26.9       0.7         28.5       32.5       26.5       16.6         2.3       4.7       13.9       43.9         3.7       6.3       23.6       38.5         1.4       6.1       4.4       0.0         47.9       21.4       27.9       15.4         2.8       18.4       18.8       42.7         43.7       39.8       43.8       41.9





## 6.2.3 Discussion

Spillover from domestic pigs into feral pigs was likely to occur regardless of the herd type where the outbreak began. When outbreaks began in non-commercial farms and spilled over to feral pigs, feral pig transmission was the dominant spread pathway. When outbreaks began in commercial farms and spilled over to feral pigs, the feral pig spread pathway was not a significant contributor to the overall outbreak.

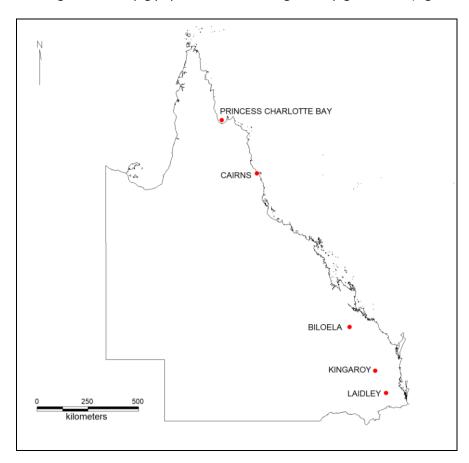
When spillover occurred from feral pigs into domestic pigs the affected farms were mainly non-commercial and small commercial. This is expected behaviour as non-commercial and small commercial farms generally have less biosecurity measures in place and there is more likelihood of direct and indirect contacts with feral pigs than medium, large, and very large commercial farms.

These results are consistent with overseas experience (Bellini et al., 2016; Oļševskis et al., 2016; Chenais et al. 2019; Boklund et al., 2020) and local expectations.



# 7 CASE STUDIES

The case studies presented here are based on incursion scenarios provided by QDAF and adapted in consultation with industry and QDAF. They were selected to represent most likely or highly important potential routes of introduction of ASF into Queensland. ASF was explicitly seeded into areas with either high domestic pig populations and/or high feral pig densities (Figure 27).



**Figure 27.** Map of scenario locations: DS1 - Cairns, DS2 - Laidley, DS3 - Kingaroy, FS1 - Princess Charlotte Bay, FS2 - Biloela

#### 7.1 Outbreak scenario DS1

Illegal contaminated foodstuffs are brought into Australia by an airline passenger and fed to backyard pigs on a peri-urban acreage near Cairns, Queensland. An outbreak of ASF (Georgia 2007/II strain) begins in June and is detected and reported to the authorities 42 days later.

#### **7.1.1** Method

Five smallholder herds near Cairns were selected to represent the primary case (Table 12). ASF was introduced into each herd separately in June and allowed to spread silently for 42 days at which point the default ASF control program (Appendix C) was initiated. The feral pig diffusive spread pathway was enabled and the jump pathway disabled. Control in feral pigs was disabled. 100 outbreaks were simulated for each of the five seed herds and all 500 runs were pooled into a single result. The process was repeated for a 60-day silent spread and for a November start date (i.e., there were four scenario variations: Jun 42d, Jun 60d, Nov 42d, Nov 60d).



Table 12. Seed herds - Scenario DS1

Herd ID	Herd type	Size	Longitude	Latitude	Region
1977	Smallholder	2	146.005	-17.4866	TRF
2177	Smallholder	2	145.555	-16.887	TRF
2539	Smallholder	4	145.280	-17.383	TRF
3378	Smallholder	30	145.555	-17.315	TRF
4175	Smallholder	18	145.572	-17.4699	TRF

# 7.1.2 Results

# 7.1.2.1 Infection in domestic pigs

There was a moderate likelihood (22-52%) that infection would die out before being reported. This was higher for November outbreaks compared to June outbreaks and for 60-day silent spread compared to 42 days.

Table 13. Probability of disease not being detected - Scenario DS1

Scenario variation	Number of runs with no detection	%
Jun 42d	108	21.6%
Jun 60d	179	35.8%
Nov 42d	173	34.6%
Nov 60d	260	52.0%

Relatively few domestic pig herds were infected in this scenario, and this was to be expected given the low density of farms in the study area. When outbreaks did occur, they tended to be larger and last longer in June compared to November and for a 60 -day silent spread phase compared to 42 days. Only in one run was infection still present at the end of the 365-day simulation period (from the June – 60-day silent spread series).

Table 14. Number of IPs for Scenario DS1

Scenario variation	mean	median	min	max
Jun 42d	7.8	4	1	42
Nov 42d	6.4	4	1	31
Jun 60d	11.0	4	1	73
Nov 60d	8.2	5	1	44



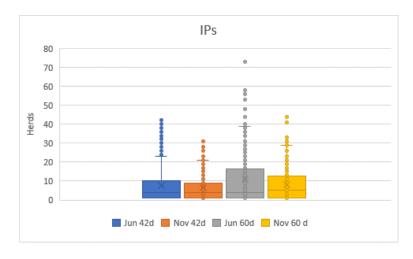


Figure 28. Number of IPs - Scenario DS1

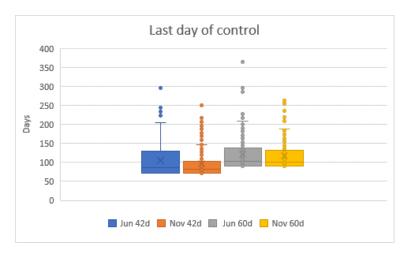


Figure 29. Last day of control - Scenario DS1

# 7.1.2.2 Infection in feral pigs

Nov 60d

There was a high likelihood (76-82%) that infection would spread from domestic to feral pigs in this scenario.

 Scenario variation
 Runs with spread to feral pigs
 %

 Jun 42d
 411
 82.2%

 Jun 60d
 396
 79.2%

 Nov 42d
 394
 78.8%

379

75.8%

Table 15. Spread to feral pigs – Scenario DS1

In only one run was infection still active at the end of the 365-day simulation period (June, 60-day silent spread series). When infection spread to feral pigs, it tended to spread further and persist for longer in June compared to November.



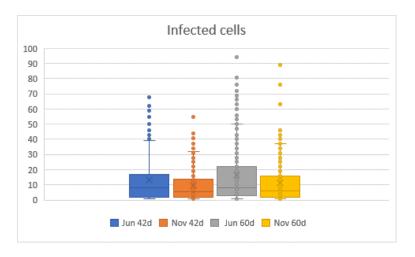


Figure 30. Number of cells with infected feral pigs - Scenario DS1

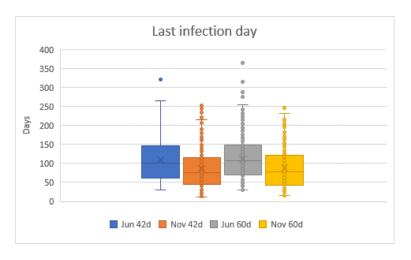


Figure 31. Last day of infection in feral pigs - Scenario DS1

#### 7.1.2.3 Source of infection

When spread of infection occurred in domestic pigs, feral pigs were a major source of infection, accounting for 90% of all infections. Movement of live pigs either directly or via saleyards was a minor contributor to spread between herds.

*Table 16.* Source of infection for domestic pig herds – Scenario DS1

Scenario variation	local	direct	saleyard	indirect	feral pig
Jun 42d	4.35%	0.19%	0.06%	2.96%	92.44%
Nov 42d	3.71%	0.36%	0.09%	2.13%	93.71%
Jun 60d	3.65%	0.22%	0.15%	4.28%	91.71%
Nov 60d	3.59%	0.40%	0.44%	5.24%	90.32%
Average	3.82%	0.29%	0.19%	3.65%	92.04%

Where ASF spread to the feral population, the source of infection for sounders (infected cells) is shown in Table 16. Feral pigs were more likely to be infected from other feral pigs in June, but contact with domestic pigs is relatively more important in November.



**Table 17.** Source of infection for feral pig sounders (cells)

Scenario variation	Feral-to-feral	Farm-to-feral	
Jun 42d	52.81%	47.19%	
Nov 42d	41.38%	58.62%	
Jun 60d	51.37%	48.63%	
Nov 60d	41.28%	58.72%	

# 7.1.2.4 Effect of applying control to feral pigs

Simulations were run in which pre-emptive feral pig control involving surveillance and population reduction was applied around IPs. This approach was evaluated using the June 42d outbreak simulations. For this scenario, there were relatively few IPs and including feral pig control had only a minor effect on these numbers. However, it did reduce the duration of the outbreak

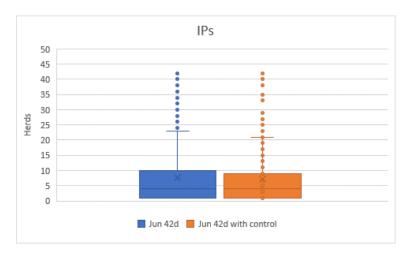


Figure 32. Number of IPs when feral pig control is adopted - Scenario DS1

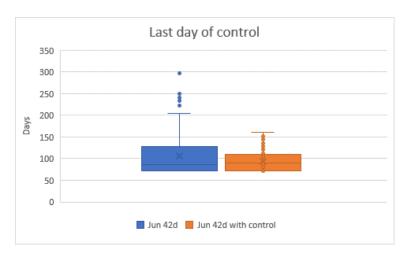


Figure 33. Last day of control when feral pig control is adopted - Scenario DS1

Not surprisingly, applying control measures to feral pigs reduced infection in the feral pig population, both the number of infected cells and the duration of infection. This strategy will only



be effective after the first IP is detected and some sounders would have been infected before this time.

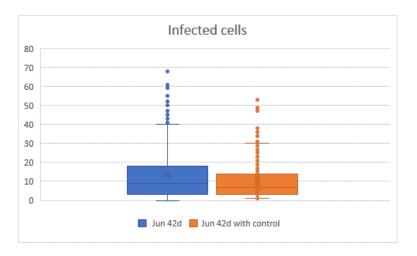


Figure 34. Number of cells with infected feral pigs when feral pig control is used - Scenario DS1

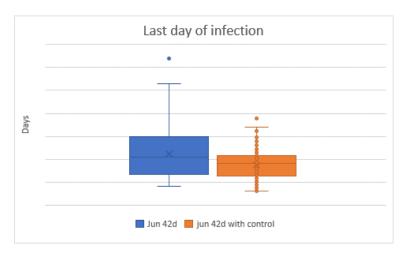


Figure 35. Last day of infection in feral pigs when feral pig control is adopted - Scenario DS1

#### 7.1.3 Discussion

Outbreaks tended to be larger and last longer in June compared to November and this is consistent with the cooler winter months being favourable for virus viability in the environment. Outbreaks were also larger and longer when the time to detection was 60 days compared to 42 as the longer silent spread phase allowed ASF to reach more herds before control measures could be applied.

When outbreaks spilled over into the feral pig population, feral pigs then became a significant source of infection back into domestic pig farms. This is consistent with the high proportion of farms in the study area that are non-commercial and have limited biosecurity measures in place.

Feral pigs were more likely to be infected from other feral pigs in June whereas infection from domestic pigs was relatively more important in November. This is most likely due to the TRF feral pig population in the TRF region peaking in May/June and bottoming out in November (Table 5) and the density-dependent nature of transmission between sounders (Section1.4.2).



#### 7.2 Outbreak scenario DS2

Infected pork products are illegally imported into Queensland, Australia via mail from overseas. An outbreak of ASF (Georgia 2007/II strain) begins in November on a small commercial farm near Laidley and is detected and reported to the authorities 21 days later.

#### **7.2.1** Method

Five small commercial herds near Laidley were selected to represent the primary case (Table 18). ASF was introduced into each herd separately in November and allowed to spread silently for 21 days at which point the default ASF control program (Appendix C) was initiated. The feral pig diffusive spread pathway was enabled and the jump pathway disabled. 100 outbreaks were simulated for each of the five seed herds and all 500 runs were pooled into a single result. The process was repeated for a 42-day silent spread and for a June start date (i.e., there were four scenario variations: Jun 21d, Jun 42d, Nov 21d, Nov 42d).

 Herd type
 Size
 Longitude
 Latitude
 Region

 Small commercial
 1200
 152.275
 -27.7483
 TEF

 Small commercial
 20
 152.507
 -27.5324
 TEF

152.254

152.234

152.277

-27.6339

-27.5558

-27.5153

**TEF** 

TEF

TEF

Table 18. Seed herds - Scenario DS2

17

162

403

#### 7.2.2 Results

# 7.2.2.1 Infection in domestic pigs

Herd ID

52

711

3659

4001

4147

Small commercial

Small commercial

Small commercial

Infection was always detected when the silent spread period was 21 days. With a 42-day silent spread period, there was an 18-22% likelihood of infection dying out before detection (Table 19).

Table 19. Probability of disease not being detected - Scenario DS2

Scenario variation	Number of runs with no detection	%
Jun 21d	0	0.0%
Jun 42d	89	17.8%
Nov 21d	0	0.0%
Nov 42d	109	21.8%

With delayed detection (42-day silent spread compared to 21 days) outbreaks in domestic pigs were larger and lasted longer (Table 14, Figures 36-37). Outbreaks in June tended to be slightly larger than those in November.

Table 20. Number of IPs for Scenario DS2



Jun 21d	4.2	2	1	22
Nov 21d	3.6	2	1	22
Jun 42d	10.7	4	1	55
Nov 42d	9.8	4	1	51

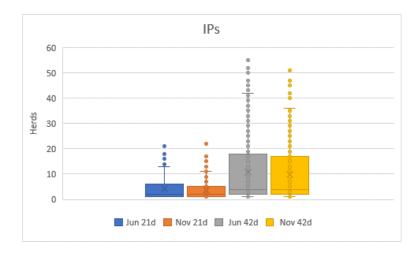


Figure 36. Number of IPs - Scenario DS2

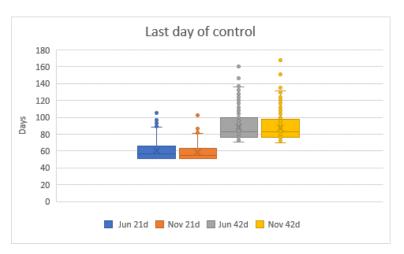


Figure 37. Last day of control - Scenario DS2

# 7.2.2.2 Infection in feral pigs

There was a moderately high likelihood (40-66%) that infection would spread from domestic to feral pigs (Table 21). Infection persisted longer in June compared to November.

*Table 21.* Spread to feral pigs – Scenario DS2

Scenario variation	Runs with spread to feral pigs	%
Jun 21d	267	53.4%
Jun 42d	332	66.4%
Nov 21d	199	39.8%
Nov 42d	321	64.2%



When infection spread to feral pigs, it was more extensive with delayed detection, and in June compared to November. Infection persisted for much longer in June compared to November which can be attributed to reduced virus viability in the hotter summer months. Infection always died out in the feral pig population within 6 months if it was controlled in domestic pigs.

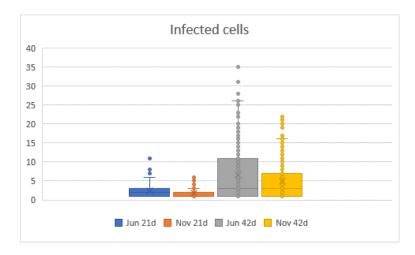


Figure 38. Number of cells with infected feral pigs - Scenario DS2

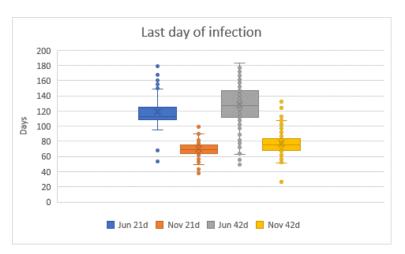


Figure 39. Last day of infection in feral pigs - Scenario DS2

### 7.2.2.3 Source of infection

Where spread of infection occurred in domestic pigs, feral pigs were relatively less important in this scenario, accounting for around 22% of all domestic herd infections. Movement of live pigs (43%) and indirect contacts (25%) were significant contributors to spread between herds.

Table 22. Source of infection for domestic pig herds – Scenario DS2

Scenario variation	local	direct	saleyard	indirect	feral pig
Jun 21d	12.12%	47.32%	0.12%	15.94%	24.49%
Nov 21d	8.23%	59.96%	0.00%	17.22%	14.60%
Jun 42d	9.45%	29.78%	0.27%	32.41%	28.08%
Nov 42d	10.42%	33.50%	0.10%	35.20%	20.78%
Average	10.06%	42.64%	0.13%	25.19%	21.99%



Where ASF spread to the feral population, the source of infection for sounders (infected cells) is shown in Table 23. In this region, feral pigs were more likely to be infected from contact with domestic pigs than from contact with other feral pigs. Infection from other feral pigs is higher in June compared to November.

Table 23. Source of infection for feral pig sounders (cells)

Scenario variation	Feral-to-feral	Farm-to-feral
Jun 21d	12.60%	87.40%
Nov 21d	7.58%	92.42%
Jun 42d	13.85%	86.15%
Nov 42d	7.04%	92.96%

# 7.2.2.4 Effect of applying control to feral pigs

Simulations were run in which pre-emptive feral pig control involving surveillance and population reduction was applied around IPs. This approach was evaluated using the June 21d outbreak simulations.

There was little effect on the number of IPs and the duration of the outbreak which given the lower contribution that feral pigs make to infection of domestic herds in this scenario is not unexpected.

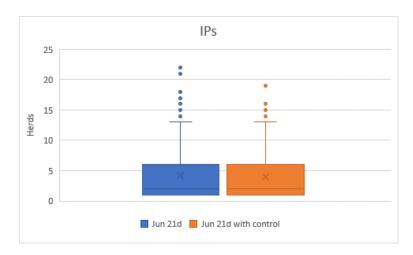


Figure 40. Number of IPs when feral pig control is used - Scenario DS2





Figure 41. Last day of control when feral pig control is adopted - Scenario DS2

Implementing feral pig control had a major impact on infection in the feral pig population. In addition to reducing the proportion of runs with spread to feral pigs from 53.4% to 42.6%, both the number of infected cells and duration of infection in the feral pig population were reduced.

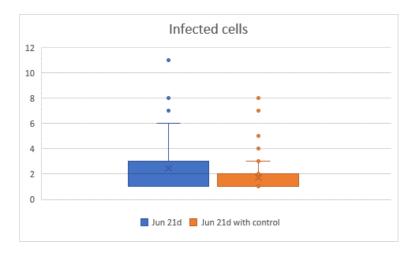


Figure 42. Number of cells with infected feral pigs when feral pig control is used - Scenario DS2

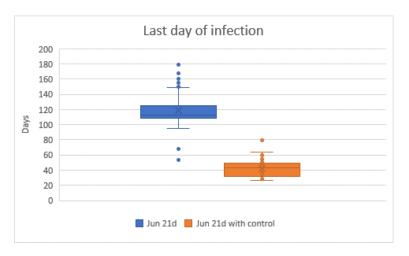


Figure 43. Last day of infection in feral pigs when feral pig control is used - Scenario DS2





#### 7.2.3 Discussion

Outbreaks tended to be larger and last longer in June compared to November and this is consistent with the cooler winter months being favourable for virus viability in the environment. Outbreaks were also larger and longer when the time to detection was 42 days compared to 21 as the longer silent spread phase allowed ASF to reach more herds before control measures could be applied.

Feral pigs played a relatively less important role in outbreaks than in Scenario DS1. Outbreaks tended to be driven more by movement of live pigs and indirect contacts between herds. Feral pigs were more likely to be infected from contact with domestic pigs than from contact with other feral pigs. This is due to the much smaller feral pig population and lower population densities in the TEF region compared to the TRF region (Section 7.2.5).

Augmenting the domestic pig control program with a feral pig control program did not materially reduce the size or duration of outbreaks in domestic pigs. This is reasonable given that transmission from feral pigs was a minor contributor to outbreaks (due to relatively low feral pig densities in the TEF region). Implementing feral pig control did, however, reduce the size and duration of outbreaks in the feral pig population. This may be a consideration for domestic pig outbreaks that occur in areas with high feral pig densities.

#### 7.3 Outbreak scenario DS3

Infected pork products are illegally imported into Queensland, Australia via courier from overseas. An outbreak of ASF (Georgia 2007/II strain) begins in June on a reasonably large commercial farm near Kingaroy and is detected and reported to the authorities 21 days later.

### **7.3.1** Method

Five medium-to-large-scale commercial herds near Kingaroy were selected to represent the primary case (Table 24). ASF was introduced into each herd separately in June and allowed to spread silently for 21 days at which point the default ASF control program (Appendix C) was initiated. The feral pig diffusive spread pathway was enabled and the jump pathway disabled. 100 outbreaks were simulated for each of the five seed herds and all 500 runs were pooled into a single result. The process was repeated for a 42-day silent spread and for a November start date (i.e., there were four scenario variations: Jun 21d, Jun 42d, Nov 21d, Nov 42d).

**Herd ID** Herd type Longitude Latitude Size Region 17 Medium to large commercial 2500 151.796 -26.2812 TEF 26 Medium to large commercial 4000 151.782 -26.5035 TRS 40 Medium to large commercial 7000 151.914 -26.4411 **TEF** 7500 -26.6981 69 Medium to large commercial 151.867 TEF 6500 151.783 Medium to large commercial -26.4238 89 **TEF** 

Table 24. Seed herds - Scenario DS3



### 7.3.2 Results

# 7.3.2.1 Infection in domestic pigs

In this scenario, ASF always established and did not die out before detection. Outbreaks were larger with the longer time to first detection (42 days compared to 21 days). The June outbreaks also tended to be larger than the November ones (Table 26).

Table 25. Number of IPs for Scenario DS3

Scenario variation	mean	median	min	max
Jun 21d	6.6	4	1	29
Nov 21d	5.8	3	1	27
Jun 42d	18.0	12	1	78
Nov 42d	14.8	10	1	62

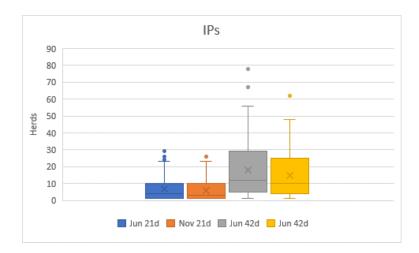


Figure 44. Number of IPs - Scenario DS3

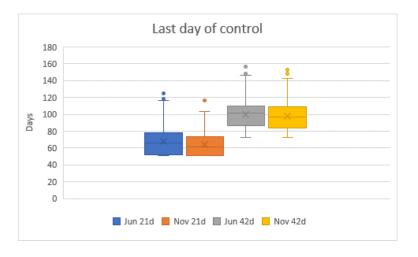


Figure 45. Last day of control - Scenario DS3



# 7.3.2.2 Infection in feral pigs

There was a high likelihood (50-93%) that infection would spread from domestic to feral pigs in this scenario. The likelihood increased with longer delays to detection.

**Scenario variation** Runs with spread to feral pigs Jun 21d 301

Table 26. Spread to feral pigs – Scenario DS3

60.20% Jun 42d 463 92.60% Nov 21d 260 52.00% Nov 42d 433 86.60%

When infection spread to feral pigs, it was more extensive with delayed detection, and for June outbreaks compared to November. Infection persisted for much longer in June compared to November which can be attributed to reduced virus viability in carcases in hotter months. Infection always died out in feral pigs in this scenario if it was controlled in domestic pigs.

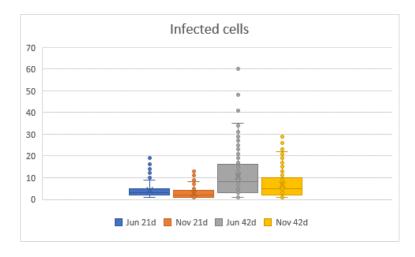


Figure 46. Number of cells with infected feral pigs - Scenario DS3

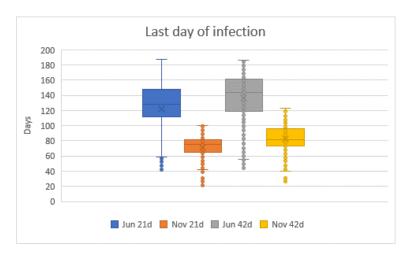


Figure 47. Last day of infection in feral pigs - Scenario DS3



#### 7.3.2.3 Source of infection

Where spread of infection occurred in domestic pigs, feral pigs accounted for around 24% of all domestic herd infections. Movement of live pigs (42%) and indirect contacts (27%) were significant contributors to spread between herds in this scenario.

Table 27. Source of infection for domestic pig herds – Scenario DS3

Scenario variation	local	direct	saleyard	indirect	feral pig
Jun 21d	6.17%	45.09%	0.10%	17.39%	31.25%
Nov 21d	6.52%	57.25%	0.00%	17.89%	18.34%
Jun 42d	7.47%	28.46%	0.31%	34.61%	29.15%
Nov 42d	8.25%	36.43%	0.12%	38.20%	17.01%
Average	7.10%	41.81%	0.13%	27.02%	23.94%

When ASF spread to the feral population, the source of infection for sounders (infected cells) is shown in the table. Similar to Scenario DS2, in this region, feral pigs were more likely to be infected from contact with domestic pigs than from contact with other feral pigs. Infection from other feral pigs is higher in June compared to November.

**Table 28.** Source of infection for feral pig sounders (cells) – Scenario DS3

Scenario variation	Feral-to-feral	Farm-to-feral
Jun 21d	12.23%	87.77%
Nov 21d	5.35%	94.65%
Jun 42d	13.95%	86.05%
Nov 42d	7.63%	92.37%

### 7.3.2.4 Effect of applying control to feral pigs

Simulations were run in which pre-emptive feral pig control involving surveillance and population reduction was applied around IPs. This approach was evaluated using the June 21d outbreak simulations.

In this scenario, pre-emptive feral pig control reduced size and duration of the outbreak in domestic pigs. Compared to DS2, feral pigs contributed more to infection of domestic herds.



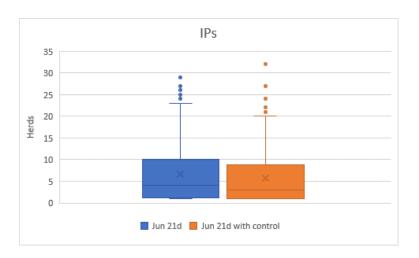


Figure 48. Number of IPs when feral pig control is used - Scenario DS3



Figure 49. Last day of control when feral pig control is adopted - Scenario DS3

Implementing feral pig control had a major impact on infection in the feral pig population. In addition to reducing the proportion of runs with spread to feral pigs from 60.2% to 47.4%, both the number of infected cells and duration of infection in the feral pig population were reduced.

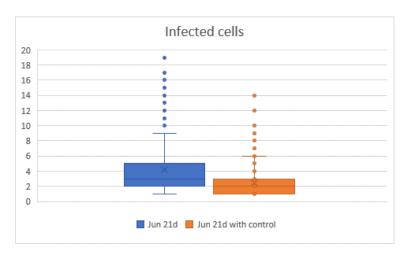


Figure 50. Number of infected cells when feral pig control is used - Scenario DS3



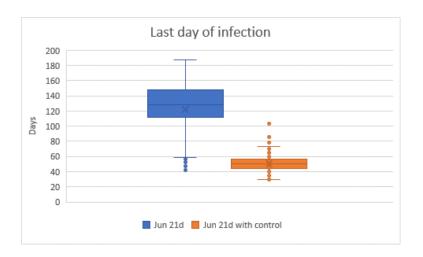


Figure 51. Last day of infection in feral pigs when feral pig control is used - Scenario DS3

### 7.3.2.5 Effect of biosecurity in domestic pig herds

The effect of enhanced or reduced biosecurity in domestic pig herds was simulated by

- (a) increasing a herd's biosecurity risk score for all type 2 (medium-to-large commercial) and Type 3 (small commercial) pig herds, unless they already have the highest score (4).
- (b) Reducing a herd's biosecurity risk score for all type 2 (medium-to-large commercial) and Type 3 (small commercial) pig herds, unless they already have the lowest score (1).

This was applied to the Jun 21 day set of runs and compared to the baseline (with default biosecurity). Note that biosecurity changes only applied to two out of the six domestic pig herd types.

Enhanced biosecurity increased the likelihood that infection did not spread beyond the seed herd from 25% to 36% and reduced the likelihood of infection spreading to feral pigs from 60% to 50%. Conversely, reduced biosecurity reduced the likelihood of ASF not spreading from the seed herd from 25% to 16% and increased the likelihood that it would spread to feral pigs from 60% to 72%

Table 29. Impact of enhanced biosecurity, based on percentage of runs – Scenario DS3

	Jun 21d	Jun 21d	Jun 21d
	baseline	enhanced biosecurity	reduced biosecurity
Did not spread beyond seed herd	25.00%	35.60%	16.00%
Spread to feral pigs	60.20%	49.60%	76.20%

Enhancing biosecurity reduced both the size and duration of the domestic pig outbreaks. Reducing biosecurity increased the size and duration of outbreaks.



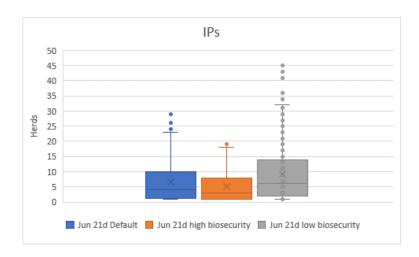


Figure 52. Number of IPs when biosecurity is enhanced/reduced - Scenario DS3

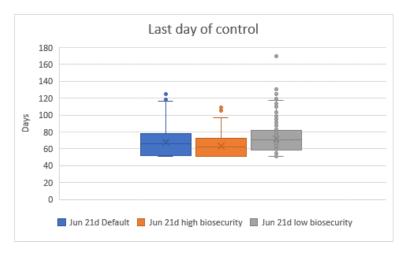


Figure 53. Last day of control when biosecurity is enhanced/reduced - Scenario DS3

In addition to reducing the likelihood of infection getting into feral pigs, enhanced biosecurity reduced the extent of infection in the feral pig population, reducing the number of infected sounders (cells) and slightly reducing the duration of infection. Reduced biosecurity had the opposite effect.

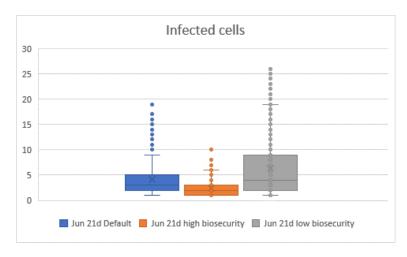


Figure 54. Number of infected cells when biosecurity is enhanced/reduced - Scenario DS3



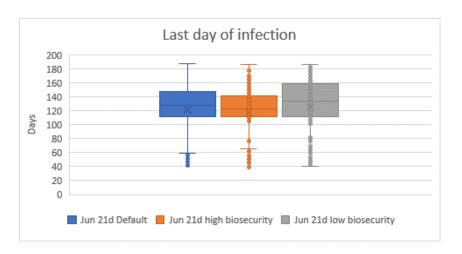


Figure 55. Last day of infection in feral pigs when biosecurity is enhanced/reduced - Scenario DS3

#### 7.3.3 Discussion

As with Scenario DS1 and DS2, outbreaks tended to be larger and last longer in June compared to November and this is consistent with the cooler winter months being favourable for virus viability in the environment., outbreaks were also larger and longer when the time to detection was 42 days compared to 21 as the longer silent spread phase allowed ASF to reach more herds before control measures could be applied. Outbreaks in this scenario (seeded in medium to large commercial farms) were larger and longer than outbreaks in Scenario DS2 (seeded in small commercial farms). As both scenarios were conducted in the TEF region the difference is likely due to medium and commercial farms having higher numbers of direct and/or indirect contacts than small commercial farms.

There was a higher likelihood of spillover of ASF from domestic pigs into feral pigs in this scenario than Scenario DS2. This is likely due to the somewhat higher feral pig density in the Scenario DS3 study area than the Scenario DS2 study area but is also influenced by the proximity of farms to feral pig populations and the biosecurity measures in place. As was the case with Scenario DS2, feral pigs in this scenario played a relatively less important role in outbreaks than in Scenario DS1. Outbreaks tended to be driven more by movement of live pigs and indirect contacts between herds. Feral pigs were more likely to be infected from contact with domestic pigs than from contact with other feral pigs. This is due to the much smaller feral pig population and lower population densities in the TEF region compared to the TRF region (Section 7.2.5).

As was the case with Scenario DS2, augmenting the domestic pig control program with a feral pig control program did not materially reduce the size or duration of outbreaks in domestic pigs. This is reasonable given that transmission from feral pigs was a minor contributor to outbreaks (due to relatively low feral pig densities in the TEF region). Implementing feral pig control did, however, reduce the size and duration of outbreaks in the feral pig population. This may be a consideration for domestic pig outbreaks that occur in areas with high feral pig densities.

Enhancing on-farm biosecurity measures decreased the likelihood of spillover transmission from domestic pigs to feral pigs, reduced the size and duration of domestic pig outbreaks, and reduced the size and duration of feral pig outbreaks. Conversely, reducing on-farm biosecurity measures



increased the likelihood of spillover transmission from domestic pigs to feral pigs, increased the size and duration of domestic pig outbreaks, and increased the size and duration of feral pig outbreaks.

### 7.4 Outbreak scenario FS1

A foreign national yacht lands at Princess Charlotte Bay and illegally dumps rubbish on a beach. The rubbish includes ASFV-contaminated pork products sourced from a country where ASF is present and is subsequently accessed by feral pigs in the area. An outbreak of ASF (Georgia 2007/II strain) begins in December in a group of feral pigs near Princess Charlotte Bay.

#### **7.4.1** Method

Five cells populated with feral pigs near Princess Charlotte Bay were selected to represent the primary case (Table 30). ASF was introduced into each cell separately in June and allowed to spread via the feral pig diffusive spread pathway. 100 outbreaks were simulated for each of the five seed cells and all 500 runs were pooled into a single result. The scenario was run firstly for undetected outbreaks and then with detection after 30, 60 and 180 days. The process was repeated for a December start date.

Cell ID Feral pig population December Feral pig population June Longitude Latitude Region 175226 14 143.5745 -14.0195 TRS 8 17 -14.3805 191036 10 143.2515 **TRS** 196082 13 22 144.1635 -14.4945 **TRS** -14.5325 197732 14 24 143.8595 TRS 203566 11 18 143.9165 -14.6655 TRS

Table 30. Seed cells - Scenario FS1

#### 7.4.2 Results

#### 7.4.2.1 Secondary spread

There was only a moderate likelihood that ASF would spread beyond the seed cell, lower in December compared to June.

Table 31. Spread of ASF beyond seed cell - Scenario FS1

Start date	Number of runs with secondary spread	%
Jun	268	53.60%
Dec	150	30.00%

### 7.4.2.2 Infection in feral pigs

Infection only spread slowly in the feral pig population. In all runs, ASF died out, with the longest outbreak lasting 212 days, with the average time being 72 days. Incursions in December were associated with small outbreaks involving an average of just two infected cells (maximum of 17 cells) and dying out in around 35 days (maximum of 197 days).



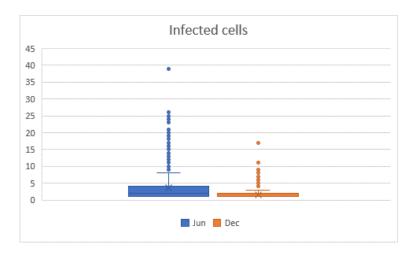


Figure 56. Number of infected cells - Scenario FS1

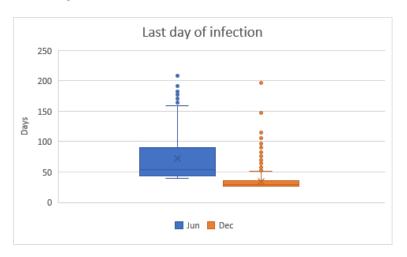


Figure 57. Last day of infection - Scenario FS1

# 7.4.2.3 Infection in domestic pigs

There was no spread to domestic herds. NB there are no domestic pig farms in the vicinity.

### 7.4.2.4 Effect of applying control to feral pigs

Simulations were run assuming ASF was detected in the feral population 30, 60 and 180 days after introduction. Early detection and implementation of control measures in the June simulations reduces the size of the outbreak in feral pigs; later detection and control was less effective. However, the average duration of an outbreak, in the uncontrolled simulations was only 73 days and only 5 runs (1%) lasted >180 days and 123 (24.6%) lasted >90 days. Therefore, it is not surprising that implementing control late in the outbreaks did not have a major impact.

Table 32. Number of infected cells for different feral pig control start days - Scenario FS1 (June)

	No control	Control from day 30	Control from day 60	Control from day 180
mean	3.6	2.6	3.5	3.4
median	2	2	2	2
min	1	1	1	1



max	39	13	25	32
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Table 33. Last day of infection for different feral pig control start days - Scenario FS1 (June)

	No control	Control from day 30	Control from day 60	Control from day 180
mean	72.6	64.3	73.4	72.9
median	54	55	58	60
min	4	5	6	7
max	212	119	174	199

Feral pig control at 30 days is compared with outbreaks with no control in the figures below. Control at 90 days and 180 days gave no improvement over the no control simulations and are not shown.

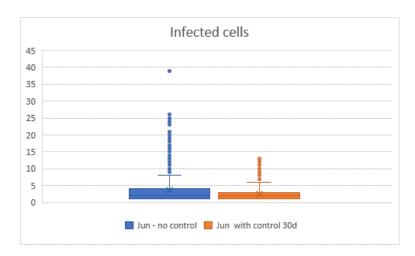


Figure 58. Number of infected cells when feral pig control starts on day 30 - Scenario FS1 (June)

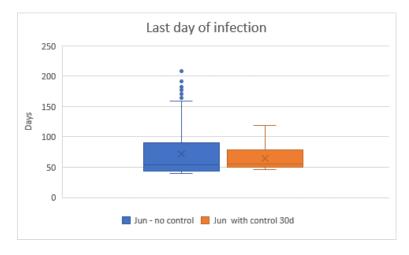


Figure 59. Last day of infection when feral pig control is adopted on day 30 - Scenario FS1 (June)



The results for the December start day simulations are not shown as those outbreaks were generally small and died out quickly, with an average last day of infection of 35 days. Only 27.4% of runs lasted >30 days, 2% lasted >60 days and 0.2% lasted >180 days. Implementing control measures produced no discernible benefit in terms of reducing the size or duration of the outbreaks in feral pigs.

### 7.4.3 Discussion

Outbreaks of ASF in feral pigs in the Princess Charlotte Bay area in June were more likely to involve secondary spread (beyond the seed cell) than those in December. Outbreaks started in June were larger and longer than those started in December. This is consistent with the December (summer) characteristics of the TRS region of lower feral pig population densities, faster carcass decomposition, and reduced viability of ASFV in the environment.

As outbreaks in this scenario tended to fade out the only advantage from feral pig control was for a 30-day time to detection of outbreaks starting in June. Feral pig control might have had a bigger impact if there had have been domestic pigs in the vicinity of the outbreak or if the outbreak had 'jumped' via anthropogenic transmission.

#### 7.5 Outbreak scenario FS2

ASFV-contaminated food brought in by a European backpacker working in the Biloela area of Queensland is discarded at a rubbish tip that is accessible to feral pigs. An outbreak of ASF (Georgia 2007/II strain) begins in November in a group of feral pigs near Biloela.

#### **7.5.1** Method

Five cells populated with feral pigs near Biloela were selected to represent the primary case (Table 34). ASF was introduced into each cell separately in November and allowed to spread via the feral pig diffusive spread pathway. 100 outbreaks were simulated for each of the five seed cells and all 500 runs were pooled into a single result. The scenario was run using passive detection of infection in domestic pigs. The process was repeated for a June introduction date.

Cell ID **Feral pig population December** Longitude Latitude Feral pig population June Region 629577 150.5285 -24.3745 9 15 TRS 9 **TRS** 630411 15 150.5475 -24.3935 631244 9 150.5475 -24.4125 15 TRS 632075 9 15 -24.4315 150.5095 TRS 632907 9 15 150.4905 -24.4505 TRS

Table 34. Seed cells - Scenario FS2

### 7.5.2 Results

### 7.5.2.1 Infection in domestic pigs

There was a high probability that the infection would spread to domestic pigs, particularly in June (Table 35).



Table 35. Transmission of ASF from feral pigs to domestic pigs - Scenario FS2

Start date	Number of runs with spillover into domestic pigs	%
June	484	96.8%
November	415	83.0%

The time from when ASF was first introduced to the feral pig population until it first infected domestic pigs ranged from 1 to 11 days, average 4 days. On average, it took 14.7 (range 7 to 35) days for ASF to be reported after being introduced into a domestic herd. Outbreaks in domestic pigs tended to be larger and last longer for June compared to November (Figures 60 and 61).

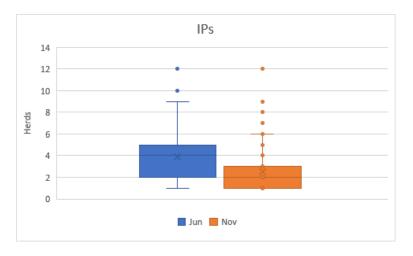


Figure 60. Number of IPs - Scenario FS2



Figure 61. Last day of infection - Scenario FS2

### 7.5.2.2 Infection in feral pigs

ASF was more likely to spread in feral pigs for outbreaks starting in June compared to November. If ASF was controlled in domestic pigs, it always died out in the feral population.



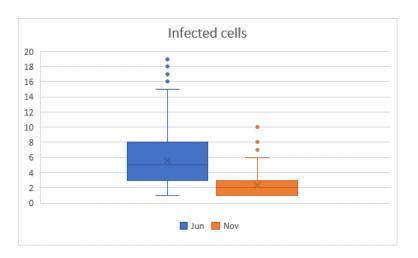


Figure 62. Number of infected cells - Scenario FS2

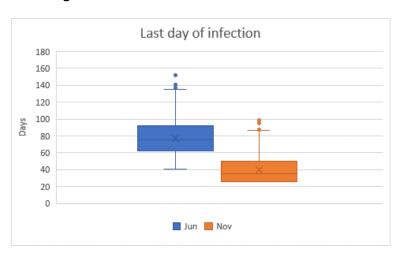


Figure 63. Last day of infection in feral pigs - Scenario FS2

# 7.5.2.3 Spread of infection

When infection spread to domestic pigs, feral pigs were a major source of infection for domestic herds, accounting for 85-90% of infections in this scenario.

**Table 36.** Source of infection in domestic pigs - Scenario FS2

	local	direct	saleyard	indirect	feral pig
June	3.74%	1.21%	0.19%	5.15%	89.70%
November	3.95%	3.95%	0.45%	6.64%	85.01%
Mean	3.84%	2.58%	0.32%	5.90%	87.36%

	Feral-to-feral	Farm-to-feral	
June	51.16%	48.84%	
November	46.23%	53.77%	



In feral pigs, around 50% of infections were due to contact with other feral pigs and 50% to contact with infected domestic herds.

### 7.5.2.4 Effect of applying control to feral pigs

Simulations were run in which feral pig control involving surveillance and population reduction was applied around IPs once infection had been reported. This approach was evaluated using the June outbreak simulations.

Applying feral pig control measures had little effect on the size and duration of outbreaks in domestic pigs in this scenario. However, only small numbers of IPs were involved – on average only 4 per run. Not surprisingly, feral pig control did reduce infection in the feral pig population, reducing both the number of infected cells and the duration of infection.

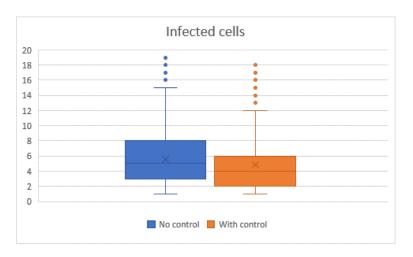


Figure 64. Number of infected cells with and without feral pig control - Scenario FS2

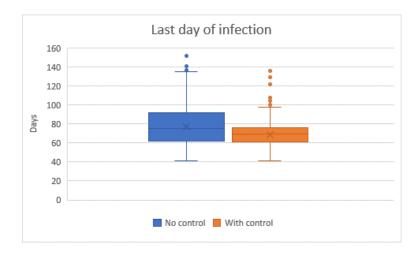


Figure 65. Last day of infection with and without feral pig control - Scenario FS2

### 7.5.3 Discussion

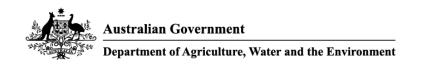
There was a high likelihood of spillover transmission from feral pigs to domestic pigs. Feral pig transmission was the dominant spread pathway in domestic outbreaks. This a consequence of the high feral pig density in the TRS region and the high number of non-commercial farms in the study





area (with low levels of on-farm biosecurity and limited direct/indirect contacts with other pig farms). As with previous scenarios, outbreaks started in June were larger and longer than those started in December. This is consistent with the December (summer) characteristics of the TRS region of lower feral pig population densities, faster carcass decomposition, and reduced viability of ASFV in the environment.

Feral pig control (in response to the declaration of infection in a domestic farm) reduced the size and duration of outbreaks in feral pigs but did not affect domestic outbreaks (which were generally small anyway). Feral pig control might have had a bigger impact if there had have been higher numbers of domestic pigs in the vicinity of the outbreak or if the outbreak had 'jumped' via anthropogenic transmission.





### 8 DISCUSSION

#### 8.1 General

This report describes the development of a new AADIS-ASF model that simulates the spread and control of ASF in domestic pigs, feral pigs, and between domestic and feral pigs in Australia. A systematic review by Hayes et al., (2021) highlights the importance of including wild pigs in ASF simulation models to better understand spread and control. To the authors' knowledge, AADIS-ASF is one of the first models of livestock disease to incorporate a concurrent feral pig component that allows ASF spread to be modelled within domestic pigs, within feral pigs, and between domestic and feral pigs. More importantly, AADIS-ASF simulates a wide range of surveillance and control measures in both domestic and feral pigs and allows detailed experiments on spread, control, and resourcing of ASF outbreaks. Assessing and responding to the threat of an ASF outbreak in feral pigs is an integral part of Australia's response strategy for ASF and a recent Animal Health Committee (AHC) working group concluded that feral pigs are likely to play a significant role in Australia's ability to respond to an ASF incursion (Animal Health Committee, 2020).

AADIS-ASF can simulate the introduction of ASF into feral and/or domestic pig populations at configurable points in time and space. The model simulates ASF transmission through live pig movements, fomite and human movements, and local disease spread in addition to transmission between domestic and feral pigs. Control strategies for ASF in the domestic pig population are based on the AUSVETPLAN Response Strategy for ASF (Animal Health Australia, 2020) and include movement controls, surveillance, tracing, infected premises operations and post-outbreak surveillance to support the regaining of ASF-free status. Control strategies for feral pigs have also been included in the model and include a general destruction operation and surveillance. These options provide the flexibility and scope to help understand how ASF may establish and spread in domestic and feral pigs across different regions and seasons, and the success of using different control strategies in eradicating disease.

# 8.2 Domestic pig outbreaks

The AADIS-ASF model has only been parameterised for Queensland context and will be scaled up to a national model through Biosecurity Innovation Program project 182021. The initial goal of this project was to represent spread at the national scale however it became clear early in the project that acquisition of national level data was not possible in the timeframe needed for the project. This data has since been made available and will be subject to analysis for the follow on BIP project. Consequently, pig premises and movement data were provided by QDAF and reviewed in collaboration with industry representatives.

The case studies presented are based on incursion scenarios provided by QDAF and adapted in consultation with industry and QDAF. They were selected to represent the most likely or highly important routes of introduction, and disease was seeded into areas with high domestic pig populations and/or high feral pig densities. For the study scenarios selected, on average, ASF is likely to be controlled in domestic pigs within 6 months of disease introduction (based on the configured assumptions in the model). The results suggest that the control measures used are sufficient to control disease and resources are adequate to complete all control activities.

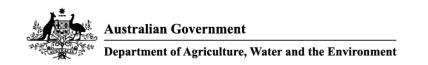




However, the model assumes the declared areas are very large, with a Restricted Area (RA) the size of a local government area (LGA) and the Control Area (CA) the entire state of Queensland. In addition, the model does not consider special permits to allow movements in declared areas, nor does it consider movements from abattoirs that may impact premises classification. Provision of these movements in the model, particularly those from abattoirs, may change the course of disease spread, premises classifications and/or model outputs relating to control. The criteria for determining declared areas is currently an active area of discussion in government and industry. The nature of declared areas for a disease such as ASF, where aerosol spread is not an epidemiological feature, was considered in the latest AUSVETPLAN response strategy review. In a recent movement controls workshop for Exercise Razorback, a DAWE initiative to improve Australia's preparedness to respond to an ASF outbreak, a key recommendation was to retain the flexibility afforded by considering criteria in determining declared areas in AUSVETPLAN rather than applying prescribed sizes of declared areas. Indeed, the latest draft version of the AUSVETPLAN Response Strategy – African swine fever (v5.1) moves away from the prescriptive declared areas described for diseases such as FMD and takes a more risk-based approach into consideration. Criteria such as the biosecurity of the farms, nearby feral and domestic pig populations, business continuity, and animal welfare require consideration in determining the most appropriate geographic extent of declared areas. Given the recent and active nature of these discussions implementing the findings into the model was not possible at the time of this project. This will be an active area of research following this project, and the assessment of small RAs, such as using the IP itself, and modifying the CA size to cover major pig producing areas are examples of some of the options to assess.

Other key findings from the studies include the importance of indirect transmission of disease, the positive influence biosecurity measures play on disease spread, the relatively low likelihood of disease spread from non-commercial (smallholder and pig keeper) properties to other domestic pig properties (compared to commercial operators), and the limited size and duration of outbreaks in areas with a low numbers of pig farms. These results are broadly consistent with other ASF modelling studies with respect to the important contribution indirect spread plays on transmission (Lee at al., 2020) and the influence of high-density farming on disease spread (Andraud et al., 2019). European studies have also shown that smaller scale piggeries with lower levels of biosecurity play an important role in spread (Halasa at al., 2016a; 2016b; Lee at al., 2020). Mur et al (2019) discussed the limited role of smallholders in spreading infection to other farms. ASF tends to fade out in the farm prior to detection (Andraud et al., 2019).

The indirect spread pathway was difficult to parameterise due to the paucity of data available. Indirect spread is a catch all phrase describing the movement of fomites such as equipment, vehicles, semen, and people between pig farms. Whilst these movements were included in the indirect pathway, they were not separately modelled. Rather a risk category was assigned to each, and together with the likely number of movements, was converted into an overall daily number of indirect movements. Similarly, the spread of disease from abattoirs to other farms and areas was not considered in this study. Whilst this pathway is an important risk pathway for spread onto farms there is no available data to parameterise the model. More information on transport company movements and biosecurity practices is needed and is the subject of further research by





industry. Experiences from overseas outbreaks (Lee et al., 2020) suggest indirect routes of transmission are important contributors of disease spread.

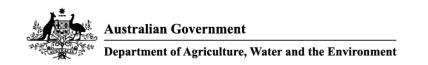
# 8.3 Feral pig outbreaks

The potential influence by feral pigs on the size and duration of an ASF outbreak in Australia was demonstrated in many simulations undertaken during the project. Outbreaks in domestic pigs tended to be larger and longer when disease spread in feral pigs was included in the scenarios (Section 5.3). Disease transmission between domestic and feral pigs is far more likely to occur in the non-commercial sector (smallholders and pig keepers) (Section 6) where farms typically have limited on-farm biosecurity measures in place (Section 3.1). These findings are consistent with Chenais et al. (2019) who concluded that the outbreak in the Caucasus and Russian Federation was largely one of poor biosecurity pig farms with spillover to feral pigs.

Under the study scenarios assessed (using the preliminary default parameterisation of the model), disease is unlikely to establish in feral pig populations for longer than a year assuming the outbreaks are controlled in the domestic sector. These findings held irrespective of whether control measures were used in feral pigs, although implementing a targeted control program in feral pigs reduced the duration of the outbreak in domestic pigs. These are preliminary findings as there is significant uncertainty associated with several parameters that may influence the nature of disease spread both within and between feral and domestic pigs.

Feral pig density, contact rates, and the probability of disease transmission between feral and domestic pigs are highly uncertain given the lack of data available under Australian conditions. A sensitivity analysis was undertaken to assess the influence of these parameters on the nature of an outbreak (Section 5). There was a clear signal that the density of feral pigs, the level of direct/indirect contact between feral pigs, and the level of direct/indirect contact between domestic and feral pigs (proxied by spillover transmission probabilities), strongly influenced the size and duration of ASF outbreaks in both domestic and feral pigs. This reinforces the critical requirement for the model to be informed by reliable localised data on feral pig distribution and abundance, inter-sounder contact rates, and the level of opportunity for direct and indirect contact between feral and domestic pigs. Outbreaks of ASF are likely to be larger and longer in cooler temperatures which are favourable for virus viability in the environment (including feral pig carcasses) (Schulz et al., 2019; Probst et al., 2020). This was observed to be a model outcome throughout the case studies (Section 7), the sensitivity analysis (Section 5) and the validation activities (Section 6).

The natural spread of ASF in feral pigs is simulated by the diffusive pathway described in Section 3.8. Factors driving this are feral pig densities, between-group contact rates, and seasonal influences on the viability of ASFV in carcasses and in the environment. A sensitivity analysis was undertaken to determine the emergent rates of spread in different regions at different times of the year (Section 5.1). Spread rates were generally higher in the high feral pig density temperate southern regions (TEF and TES) than the lower density southern temperate (TEF and TES) and central desert (DES) regions. This is consistent with observations of the spread of ASF in wild boar in Europe (EFSA, 2020). The overall median spread rate was 19 km/year which is consistent with overseas estimates of the non-anthropogenic spread rate of ASF in wild boar of 8-24 km/year





(Śmietanka et al., 2016; EFSA, 2018a; Wozniakowski et al., 2021; Podgórski & Śmietanka, 2018; Taylor et al., 2020). Further work is needed in this area as it is not known how relevant the European spread rates are to Australia. Also, as the focus of this project was Queensland, further work is needed under different seasonal conditions, particularly in cooler southern Australia where virus viability in the environment and in feral pig carcases will be much longer than in the hot climate that is typical of Queensland.

Section 5 demonstrated the density-dependent transmission of ASF between sounders (Podgórski et al., 2020) and the model may thus be useful in assessing how targeted reductions in feral pig density may influence the risk of transmission in the domestic pig population (EFSA, 2018b).

#### 8.4 Limitations & future work

The new model already provides insights into how ASF may spread in the Australian context, but these are preliminary findings only. Further review and validation work is required to gain confidence in model outcomes prior to adoption into decision making and policy. The limitations of the project and potential future work are detailed in this section.

# 8.4.1 Expansion to other jurisdictions

The AADIS-ASF model has only been parameterised for Queensland and will be scaled up to a national model through Biosecurity Innovation Program project 182021. It is expected that the conceptual model, assumptions, and implementation will be broadly suitable for other jurisdictions, however, it is possible that refinements to the conceptual model and/or implementation will be triggered during the expansion to national scale.

### 8.4.2 Within-herd spread

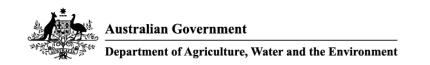
The model considers different herd or farm types and their unique movement patterns and biosecurity practices (Section 3.1). There is a mathematical assumption that disease spreads within a pig herd homogenously at a constant rate, i.e., the epidemiological unit of interest is a well-mixed herd. In reality, the sheds, pens and yards characteristic of pig farming means that disease spread is likely to be multiscale, i.e., fast within a shed and slower between sheds. The model indirectly addresses this issue through modified transmission parameters to reflect the potentially longer and less intensive infectious periods of the farm. The alternative is to explicitly model the spread of disease within individual sheds on a pig farm. A review of this approach will be carried out as part of a follow-on project.

# 8.4.3 Post-outbreak management

The post-outbreak management module is available in AADIS-ASF but was not enabled and parameterised for the case studies. Estimating market damages and trade losses from an ASF outbreak was beyond the scope of this project.

### 8.4.4 Feral pig surveillance and control

The AADIS-ASF-QLD implementation of feral pig passive surveillance, active surveillance, and control is preliminary and needs to be refined. The outcomes of the 2019 AHC ASF task force feral





pig working group surveillance sub-group and the latest AUSVETPLAN ASF response strategy should be considered. This work will allow the model to help assess the potential cost/benefits of feral pig surveillance and control strategies with respect to the domestic pig industry.

# 8.4.5 Feral pig distribution and abundance raster data

The feral pig distribution and abundance raster layer used in the AADIS-ASF-QLD model will be improved to better align with current understanding and literature e.g., Hone et al., 2020. A review is underway of the national feral pig population including the density and distribution of feral pigs considering the more recent research. The current work of the ABARES pest management group and the North Australia Quarantine Strategy will provide more information on feral pig abundance and distribution and will be useful to cross reference and ground truth population estimates in the model.

When expanding AADIS-ASF to a national scale it may be necessary to revisit the cell size chosen for the grid. The 2x2 km grid cell used in the Qld model will not scale well nationally, so either a larger cell size will need to be used (e.g., 5x5 km) or mega-region models created (e.g., WA, QLD+NSW, NSW+VIC, NT+QLD) rather than a single national model. Note that if the cell size changes then the model's assumptions on sounder ranges, feral pig population dynamics and contact rates between adjoining cells and will also need to be revisited. As part of this work, additional raster layers such as land use and terrain accessibility should be derived to better inform passive and active surveillance.

# 8.4.6 Raster vs agent-based modelling approach to representing feral pigs

A decision was made during the development of the AADIS-ASF-QLD model to use a geographic automaton modelling approach for representing the feral pig population (i.e., each (atomic) cell has a count of feral pigs that changes monthly). This approach scales well nationally but has limitations representing the ecology, mobility, and contact patterns of sounders. The alternative is to represent sounders and solitary males as individual agents that organically interact with each other. This approach captures feral pig ecology and epidemiology accurately but doesn't scale up (as it results in millions of extra agents). The AADIS framework could be used to create a local-scale agent-based feral pig model by defining sounder and solitary male agents (i.e., 'herd types') and the localised outcomes of this model could help parameterise the larger scale main model. It might be useful to allow AADIS to represent wild populations as either point-based (e.g., sounder groups) or raster-based (e.g., midges as vectors of livestock disease).

### 8.4.7 Feral pig jump contacts

The model includes an option to simulate longer distance sporadic jumps in disease transmission among feral pigs. This could include anthropogenic spread (e.g., due to feral pig hunter movements), or natural spread (e.g., longer distance movements of lone males). These longer distance movements are not well understood and were not included in the simulation studies. Further information is needed to better understand the nature of these movements and the probability of associated disease transmission. The inclusion of this pathway of transmission in the model may contribute to larger and more lengthy outbreaks of disease, as infection may seed into unexpected areas and make control in feral pigs more problematic and lengthier. It may also result





in increased likelihood of disease spread to domestic pig herds. Further work is needed to parameterise the jump pathway. Can genomic, qualitative (e.g., hunter surveys), or trace elemental studies provide clues on the origins and movement pattern of feral pigs? Is any data available on pigs being purposely relocated for hunting purposes? What is the likelihood of infected carcasses or fomites being moved around by hunters? What sort of distances do solitary males cover in Australia when looking for mates? AADIS supports multiple concurrent jump pathways, so it would be possible to model natural jumps separately to human-mediated jumps.

# 8.4.8 The domestic/feral pig epidemiological interface

The epidemiological interface between domestic and feral pigs was difficult to parameterise in the AADIS-ASF-QLD. There is a shortage of local empirical data available on direct/indirect contacts between feral and domestic pigs. There is a need for local field work (e.g., camera traps, collaring, satellite tracking, genomics, pathogen surveillance, trace elemental studies) on direct/indirect contacts between feral and domestic pigs to directly inform the model. How and under what conditions do we see interactions (contacts)? How does this vary over space and time? How likely is it that different types of contact would result in transmission? Are there situations where ASF becomes established in feral pigs? Under what conditions, if any, do we see large outbreaks that are difficult to control? Government and industry are very interested in better understanding the domestic/feral epidemiological interface and reducing the likelihood of spill-over transmission events.

### 8.4.9 Compartmentalisation

An incursion of ASF in Australia would result in significant damage to the pig industry through closure of international markets for pork and pig products. There is strong interest in the establishment of biosecurity-based compartments during 'peacetime' that may allow business continuity in the face of an ASF outbreak (Ausvet, 2019). It may be useful for AADIS-ASF to be extended to assist investigations into the mechanics, costs, and benefits of compartmentalisation.

Although modelling compartmentalisation was beyond the scope of this project, the AADIS database schema was updated to allow the definition of company-linked producer networks.

- A new *ManagementGroup* database table with interim attributes *groupID* and *groupName*. Other attributes can be defined as needed.
- A new groupID attribute in the Herd database table that indexes into the ManagementGroup table.

This allows each herd to be (optionally) associated with a specific management group and can be used to identify integrated production systems and multi-site piggeries. A future implementation of compartmentalisation could make use of the management linkages to identify herds that conform to specified biosecurity measures (thus qualifying the linked premises to a compartment).

### 8.4.10Indirect contacts

The AADIS-ASF-QLD model represents direct contacts by replaying NLIS movements. Indirect contacts are harder to quantify. The risk of indirect transmission could be better understood





through focussed field studies and/or data analysis. For example, truck movement leaving abattoirs, feed and feed trucks, semen movements, shared trucks (both within and between industries) with inadequate decontamination between consignments. Abattoirs are not currently agents in the AADIS model. Infected consignments sent to an abattoir are logged and are considered a dead-end for disease transmission. By explicitly representing abattoirs as agents, it would be possible to model indirect transmission out of abattoirs via contaminated trucks. This would also facilitate identification of (indirect) trace premises allowing movement controls (and implications thereof) and resolution priorities to be better reported. Trace premises, of which there may be many, are a potential response bottleneck - they will need resourcing and a prioritisation process to assess as negative to be able to move pigs. If not assessed negative, this will very rapidly lead to negative welfare outcomes due to over-stocking.

#### 8.4.11Domestic declared areas

The domestic pig declared areas in AADIS-ASF are largely inherited from the AADIS-FMD model. Declared areas can be jurisdictional (CA=state, RA=LGA) or radial (e.g., CA=10 km, RA=5 km), but cannot be a combination of jurisdictional and radial (e.g., cannot have CA=state, RA=5 km). A more flexible approach may be required in the model, for example, restricting the CA to the South-East area of Qld where most of the pig farms are. A review is needed of the application and designation of movement controls in the model to reflect the current proposals in AUSVETPLAN for declared areas and premises classifications. A new more flexible concept needs to be discussed and developed to allow testing of alternative ASF and pig industry specific control strategies to help understand the potential impact on an outbreak.

#### 8.4.12 Model validation

It's challenging to validate epidemiological models when the subject disease has never been detected in the subject country. One option is to adapt the AADIS-ASF model to a country that has experienced an ASF outbreak (such as Germany or South Korea) and compare the modelled outcomes with field data.

Another option is a comparative validation where multiple ASF models are adapted to a common study area and the simulated outbreak results comparted. The QUADs emergency management group Epiteam, representing the US, Canada, UK, NZ, and Australia, provides technical advice to chief veterinary officers on key animal health policy issues. The group have previously conducted model comparison studies for validation and to assist contingency plans (Sanson et al., 2011; Roche et al., 2015; Rawdon et al., 2018) and could provide a forum for future validation studies for AADIS-ASF.

### 8.4.13User interface and model outputs

A review of the user interface (user guide, database files, configuration files and graphical user interface) and model outputs (report files and visualisation) is required to ensure fitness for purpose.





# 8.4.14Stakeholder engagement

The project focussed on developing a new modelling capability to support Australian preparedness and planning for ASF and this required significant engagement with federal and state governments, and industry. Further stakeholder engagement, including communication of project outcomes, is planned for the follow-on Biosecurity Innovation Program project 182021 which will scale the AADIS-ASF model up to a national context.

#### 8.5 Conclusions

The new AADIS-ASF model is a successful proof of concept that an agent-based domestic pig disease spread model can interoperate with a geographic automata-based feral pig disease spread model. The preliminary findings of the project's simulation studies suggest ASF is likely to be controlled in domestic pigs within 6 months of disease introduction (based on the configured assumptions of the scenarios). Indirect transmission of ASF (such as fomites, trucks, and people movements) is an important aspect of outbreaks and on-farm biosecurity plays a critical role in reducing ASF spread. The simulations suggest feral pigs have the potential to amplify the size and duration of an outbreak, but their influence will depend on the region, the time of year, the density of the feral pig population, and the extent of on-farm biosecurity measures. Spillover between domestic and feral pigs is far more likely to involve non-commercial farms (smallholders and pig keepers) than commercial farms.

The new AADIS-ASF model is a flexible and powerful decision support tool for preparedness for a potential incursion of ASF in Australia. The model will help answer questions about the potential size of an outbreak, the risk of transmission spillover events between domestic and feral pigs, appropriate control and eradication measures, and resource requirements. The model will also help identify knowledge and data gaps, support preparedness and training exercises, and inform strategic decision making.

#### REFERENCES

ACIL Allen Consulting (2019) Economic analysis of African Swine Fever incursion into Australia. Final report to Australian Pork Limited. <a href="http://australianpork.com.au/wp-content/uploads/2019/11/African-Swine-Fever-Final-Report-140819.pdf">http://australianpork.com.au/wp-content/uploads/2019/11/African-Swine-Fever-Final-Report-140819.pdf</a>

Afayoa, M., Atuhaire, D. K., Ochwo, S., Okuni, J. B., Kisekka, M., Olaho-Mukani, W., & Ojok, L. (2016). Haematological, biochemical and clinical changes in domestic pigs experimentally infected with African swine fever virus isolates from Uganda. *Bulletin of Animal Health and Production in Africa*, 62(1), 7–22.

Alkhamis, M. A., Gallardo, C., Jurado, C., Soler, A., Arias, M., & Sánchez-Vizcaíno, J. M. (2018). Phylodynamics and evolutionary epidemiology of African swine fever p72-CVR genes in Eurasia and Africa. *PLoS ONE*, *13*(2), 1–18. <a href="https://doi.org/10.1371/journal.pone.0192565">https://doi.org/10.1371/journal.pone.0192565</a>

Al-Riyami, S., (2021). Decision support tools for vector-borne spread of animal disease. PhD thesis. Faculty of Veterinary and Agricultural Sciences, University of Melbourne.

Andraud, M., Halasa, T., Boklund, A., & Rose, N. (2019). Threat to the French Swine Industry of African Swine Fever: Surveillance, Spread, and Control Perspectives. *Frontiers in Veterinary Science*, *6*, 248. https://doi.org/10.3389/fvets.2019.00248

Andriamanivo, H.R., Randriamananjara, D., Ralalarison, R. A., Nomenjanahary, L. A., Razafindraibe, N. P., Andria-Mananjara, E. D., Rakotomanana, D. O., Fenozara, P. S., Biarmann, M., Halm, A., Razafimandimby, H., Flachet, L., & Cardinale, E. (2019). How could an African swine fever outbreak evolve in an enzootic context? The case of Imerintsiatosika, Madagascar in 2015. *PloS one*, 14(9), e0221928. https://doi.org/10.1371/journal.pone.0221928

Animal Health Australia (2020). Response strategy African swine fever (version 5.0). Australian Veterinary Emergency Plan (AUSVETPLAN), Edition 5, Canberra, ACT. animalhealthaustralia.com.au/download/18226/

Animal Health Australia (2021). Response strategy African swine fever (version 5.1). Australian Veterinary Emergency Plan (AUSVETPLAN), Edition 5, Canberra, ACT.

Animal Health Committee (2020). African swine fever (ASF) feral pig task group report 2020.

Arias, M., Jurado, C., Gallardo, C., Fernández-Pinero, J., & Sánchez-Vizcaíno, J. M. (2018). Gaps in African swine fever: Analysis and priorities. Transboundary and emerging diseases, 65 Suppl 1, 235–247. <a href="https://doi.org/10.1111/tbed.12695">https://doi.org/10.1111/tbed.12695</a>

Artois, M., Depner, K. R., Guberti, V., Hars, J., Rossi, S., & Rutili, D. (2002). Classical swine fever (hog cholera) in wild boar in Europe. *Revue scientifique et technique (International Office of Epizootics)*, 21(2), 287–303. https://doi.org/10.20506/rst.21.2.1332

Australian Bureau of Statistics (2014). Agricultural Commodities, Australia. <a href="http://www.abs.gov.au/ausstats/abs@.nsf/mf/7121.0">http://www.abs.gov.au/ausstats/abs@.nsf/mf/7121.0</a>

Australian Pork (2019). Annual Report 2018-2019. Australian Pork Limited, Barton, ACT.

Ausvet (2019) Technical White Paper: Business Continuity in the Face of African Swine Fever: Compartmentalisation and Company Biosecurity. Ausvet and One Health Scientific Solutions, 5 November 2019. <a href="mailto:onehealthscientific.com/2019/11/15/technical-white-paper-business-continuity-in-the-face-of-african-swine-fever/">onehealthscientific.com/2019/11/15/technical-white-paper-business-continuity-in-the-face-of-african-swine-fever/</a>

Balmoş, OM, Supeanu, A, Tamba, P, Cazan, CD, Ionică, AM, Ungur, A, Motiu, M, Manita, FA, Ancuceanu, BC, Bărbuceanu, F and Mihalca, AD, (2021). Entomological survey to study the possible involvement of arthropod vectors in the transmission of African swine fever virus in Romania. *EFSA supporting publication 2021*: 18(3):EN-6460. 35 pp. doi:10.2903/sp.efsa.2021.EN-6460

Barker, S.C. & Walker, A.R. (2014). Ticks of Australia – the species that infect domestic animals and humans. *Zootaxa*, 3816(1), 001-144. doi.org/10.11646/zootaxa.3816.1.1

Barnes, T. S., Morais, O., Cargill, C., Parke, C. R., & Urlings, A. (2020). First steps in managing the challenge of African Swine Fever in Timor-Leste. *One health (Amsterdam, Netherlands)*, 10, 100151. https://doi.org/10.1016/j.onehlt.2020.100151

Barongo, M. B., Ståhl, K., Bett, B., Bishop, R. P., Fèvre, E. M., Aliro, T., Okoth, E., Masembe, C., Knobel, D., & Ssematimba, A. (2015). Estimating the Basic Reproductive Number (R0) for African Swine Fever Virus (ASFV) Transmission between Pig Herds in Uganda. *PloS one*, 10(5), e0125842. https://doi.org/10.1371/journal.pone.0125842

Barongo, M. B., Bishop, R. P., Fèvre, E. M., Knobel, D. L., & Ssematimba, A. (2016). A Mathematical Model that Simulates Control Options for African Swine Fever Virus (ASFV). *PloS one*, *11*(7), e0158658. <a href="https://doi.org/10.1371/journal.pone.0158658">https://doi.org/10.1371/journal.pone.0158658</a>

Bellini, S., Rutili, D., & Guberti, V. (2016). Preventive measures aimed at minimizing the risk of African swine fever virus spread in pig farming systems. Acta veterinaria Scandinavica, 58(1), 82. https://doi.org/10.1186/s13028-016-0264-x

Beltrán-Alcrudo, D., Arias, M., Gallardo, C., Kramer, S. & Penrith, M.L. (2017). African swine fever: detection and diagnosis – A manual for veterinarians. FAO Animal Production and Health Manual No. 19. Rome. Food and Agriculture Organization of the United Nations (FAO). 88 pages.

Bengsen, A., Gentle, M., Mitchell, J., Oearson, H. & Saunders, G. (2014). Impacts and management of wild pigs *Sus Scrofa* in Australia. *Mammal Review*, 44(2), 135-47. doi.org/10.1111/mam.12011

Bengsen, A. J., P. West and C. R. Krull (2018). Feral Pigs in Australia and New Zealand: Range, Trend, Management, and Impacts of an Invasive Species, Cambridge Univ Press, the Pitt Building, Trumpington St, Cambridge Cb2 1rp, Cambs, Uk.

Blome, S., Gabriel, C., & Beer, M. (2013). Pathogenesis of African swine fever in domestic pigs and European wild boar. *Virus Research*, 173(1), 122–130. https://doi.org/10.1016/j.virusres.2012.10.026 Blome, S., Franzke, K., & Beer, M. (2020). African swine fever - A review of current knowledge. *Virus research*, 287, 198099. <a href="https://doi.org/10.1016/j.virusres.2020.198099">https://doi.org/10.1016/j.virusres.2020.198099</a>

Boklund, A., Toft, N., Alban, L., & Uttenthal, A. (2009). Comparing the epidemiological and economic effects of control strategies against classical swine fever in Denmark. *Prev. vet. Med.*, 90(3-4), 180–193. doi.org/10.1016/j.prevetmed.2009.04.008

Boklund, A., Dhollander, S., Chesnoiu Vasile, T., Abrahantes, J. C., Bøtner, A., Gogin, A., Gonzalez Villeta, L. C., Gortázar, C., More, S. J., Papanikolaou, A., Roberts, H., Stegeman, A., Ståhl, K., Thulke, H. H., Viltrop, A., Van der Stede, Y., & Mortensen, S. (2020). Risk factors for African swine fever incursion in Romanian domestic farms during 2019. *Scientific reports*, 10(1), 10215. https://doi.org/10.1038/s41598-020-66381-3

Boinas, F. S., Hutchings, G. H., Dixon, L. K., & Wilkinson, P. J. (2004). Characterization of pathogenic and non-pathogenic African swine fever virus isolates from Ornithodoros erraticus inhabiting pig premises in Portugal. *The Journal of General Virology*, 85(Pt 8), 2177–2187. https://doi.org/10.1099/vir.0.80058-0

Borca, M. V., Ramirez-Medina, E., Silva, E., Vuono, E., Rai, A., Pruitt, S., Holinka, L. G., Velazquez-Salinas, L., Zhu, J., & Gladue, D. P. (2020). Development of a Highly Effective African Swine Fever Virus Vaccine by Deletion of the I177L Gene Results in Sterile Immunity against the Current Epidemic Eurasia Strain. *Journal of virology*, *94*(7), e02017-19. <a href="https://doi.org/10.1128/JVI.02017-19">https://doi.org/10.1128/JVI.02017-19</a>

Bradhurst R., Roche S., Garner G., Sajeev A.S.M. and Kwan P. (2013). Modelling the spread of livestock disease on a national scale: the case for a hybrid approach. *Proceedings of the 20th International Congress on Modelling and Simulation (MODSIM2013), Modelling and Simulation Society of Australia and New Zealand, December 2013, 345–351.* 

Bradhurst R. (2015). *Modelling the spatiotemporal spread and control of viral disease in livestock using a hybrid equation-based and agent-based approach*. PhD Thesis, University of New England, <a href="https://hdl.handle.net/1959.11/19661">https://hdl.handle.net/1959.11/19661</a>

Bradhurst R., Roche S., Kwan P., and Garner G. (2015) A hybrid modelling approach to simulating foot-and-mouth disease outbreaks in Australian livestock. *Front. Environ. Sci.*, 19 March 2015. https://doi.org/10.3389/fenvs.2015.00017

Bradhurst R, Roche S, East I, Kwan P and Garner G. (2016). Improving the computational efficiency of an agent-based spatiotemporal model of livestock disease spread and control. *Environ Model Softw.* 2016; 77:1–12. https://doi.org/10.1016/j.envsoft.2015.11.015

Bradhurst R, Garner G, East I, Death C, Dodd A, Kompas T (2019) Management strategies for vaccinated animals after an outbreak of foot-and-mouth disease and the impact on return to trade. *PLoS ONE* 14(10): e0223518. <a href="https://doi.org/10.1371/journal.pone.0223518">https://doi.org/10.1371/journal.pone.0223518</a>

Bradhurst R., Spring D., Stanaway M., Milner J., & Kompas T. (2021a). A generalised and scalable framework for modelling incursions, surveillance and control of plant and environmental pests. *Environmental Modelling and Software*. 139. https://doi.org/10.1016/j.envsoft.2021.105004

Bradhurst R, Garner G, Hovari M, de la Puente M, Mintiens K., Yadav S., Federici T., Kopacka I, Stockreiter S, Kuzmanova I, Paunov S, Cacinovic V, Rubin M, Szilagyi J, Szepesine Kokany Z, Santi A, Sordilli M, Sighinas L, Spiridon M, Potocnik M, Sumption K. (2021b). Development of a transboundary model of livestock disease in Europe. *Transboundary and Emerging Diseases*. https://doi.org/10.1111/tbed.14201

Brookes, V. & Hernandez-Jover, M. (2020). Study monitors feral pigs to understand biosecurity risk. The Innovator. Graham Centre for Agricultural Innovation, Charles Sturt University. csu.edu.au/research/grahamcentre/news/newsletters/2020/winter-2020

Bui, N., Nguyen, T.L., Canevari, J., et al. Estimation of a within-herd transmission rate for African swine fever in Vietnam. *Authorea*. August 14, 2020. doi.org/10.22541/au.159741535.57194009

Cadenas-Fernández, E., Sánchez-Vizcaíno, J. M., Pintore, A., Denurra, D., Cherchi, M., Jurado, C., Vicente, J., & Barasona, J. A. (2019). Free-Ranging Pig and Wild Boar Interactions in an Endemic Area of African Swine Fever. Frontiers in veterinary science, 6, 376. https://doi.org/10.3389/fvets.2019.00376

Caley, P. (1993). Population dynamics of feral pigs (Sus scrofa) in a tropical riverine habitat complex. *Wildlife Research* 20(5): 625-636. <a href="https://doi.org/10.1071/WR9930625">https://doi.org/10.1071/WR9930625</a>

Caley, P. (1993). The Ecology and Management of Feral Pigs in the 'Wet-dry' Tropics of the Northern Territory. Master of Applied Science (Resource Management), University of Canberra.

Caley, P. (1997) Movements, Activity Patterns and Habitat Use of Feral Pigs (*Sus scrofa*) in a Tropical Habitat. *Wildlife Research*, **24**, 77-87. doi.org/10.1071/WR94075

Cannon R.M. and Roe R.T. (1982). *Livestock Disease Surveys: A Field Manual for Veterinarians*. Australian Bureau of Animal Health, Epidemiology Branch, Canberra, Australia.

Cannon R.M. (2001). Sense and sensitivity – designing surveys based on an imperfect test. *Preventive Veterinary Medicine* 49(3-4), 141-163. doi:10.1016/S0167-5877(01)00184-2

Chang'a, J. S., Mayenga, C., Settypalli, T., Achenbach, J. E., Mwanandota, J. J., Magidanga, B., Cattoli, G., Jeremiah, M., Kamigwe, A., Guo, S., Kalabi, D., Mramba, F., & Lamien, C. E. (2019). Symptomatic and asymptomatic cases of African swine fever in Tanzania. *Transboundary and emerging diseases*, 66(6), 2402–2410. https://doi.org/10.1111/tbed.13298

Chenais, E., Sternberg-Lewerin, S., Boqvist, S., Liu, L., LeBlanc, N., Aliro, T., Masembe, C., & Ståhl, K. (2017). African swine fever outbreak on a medium-sized farm in Uganda: biosecurity breaches and within-farm virus contamination. *Tropical Animal Health and Production*, 49(2), 337–346. <a href="https://doi.org/10.1007/s11250-016-1197-0">https://doi.org/10.1007/s11250-016-1197-0</a>

Chenais, E., Depner, K., Guberti, V. et al. (2019). Epidemiological considerations on African swine fever in Europe 2014–2018. *Porcine Health Management* 5(6). <a href="https://doi.org/10.1186/s40813-018-0109-2">https://doi.org/10.1186/s40813-018-0109-2</a>

Cho, K. H., Kim, H. J., Kim, D. Y., Yoo, D., Nah, J. J., Kim, Y. J., & Kang, H. E. (2021). Surveillance of ASF-infected pig farms from September to October 2019 in South Korea. Journal of veterinary science, 22(2), e26. <a href="https://doi.org/10.4142/jvs.2021.22.e26">https://doi.org/10.4142/jvs.2021.22.e26</a>

Choquenot, D., McIlroy, J. & Korn, T. (1996). Managing Vertebrate Pests: Feral Pigs. Bureau of Resource Sciences, Australian Government Publishing Service, Canberra.

Choquenot, D., B. Lukins and G. Curran (1997). Assessing Lamb Predation by Feral Pigs in Australia's Semi-Arid Rangelands. *Journal of Applied Ecology* 34(6): 1445-1454. https://doi.org/10.2307/2405260

Choquenot, D., Lukins, B., & Curran, G. (1997). Assessing Lamb Predation by Feral Pigs in Australia's Semi-Arid Rangelands. *Journal of Applied Ecology* 34(6): 1445-1454. https://doi.org/10.2307/2405260

Corn, J. L., Stallknecht, D. E., Mechlin, N. M., Luttrell, M. P., & Fischer, J. R. (2004). Persistence of pseudorabies virus in feral swine populations. *Journal of wildlife diseases*, 40(2), 307–310. https://doi.org/10.7589/0090-3558-40.2.307

Costard, S., Mur, L., Lubroth, J., Sanchez-Vizcaino, J. M., & Pfeiffer, D. U. (2013). Epidemiology of African swine fever virus. *Virus Research*, *173*(1), 191–197. https://doi.org/10.1016/j.virusres.2012.10.030

Costard S., Zagmutt F., Porphyre T. *et al.* (2015). Small-scale pig farmers' behavior, silent release of African swine fever virus and consequences for disease spread. *Sci Rep* **5**, 17074. <a href="https://doi.org/10.1038/srep17074">https://doi.org/10.1038/srep17074</a>

Cowled, B. D., Aldenhoven, J., Odeh, I. O. A., Garrett, T., Moran, C., and Lapidge, S. J. (2008). Feral pig population structuring in the rangelands of eastern Australia: Applications for designing adaptive management units. *Conservation Genetics* **9**, 211-224.

Cowled, B., Giannini, F., Beckett, S., Woolnough, A., Barry, S., Randall, L. & Garner, G. (2009) Feral pigs: predicting future distributions. *Wildlife Research* 36, 242-251. https://doi.org/10.1071/WR08115

Cowled, B. D., Garner, M. G., Negus, K., & Ward, M. P. (2012). Controlling disease outbreaks in wildlife using limited culling: modelling classical swine fever incursions in wild pigs in Australia. *Veterinary research*, 43(1), 3. <a href="https://doi.org/10.1186/1297-9716-43-3">doi.org/10.1186/1297-9716-43-3</a>

Croft, S., Massei, G., Smith, G. C., Fouracre, D., & Aegerter, J. N. (2020). Modelling Spatial and Temporal Patterns of African Swine Fever in an Isolated Wild Boar Population to Support Decision-Making. *Frontiers in veterinary science*, 7, 154. https://doi.org/10.3389/fvets.2020.00154

Cutler R. and Holyoake P. (2007). The structure and dynamics of the pig meat industry. Report prepared for the Department of Agriculture, Fisheries and Forestry, Canberra, ACT, Australia.

Danzetta, M. L., Marenzoni, M. L., Iannetti, S., Tizzani, P., Calistri, P., & Feliziani, F. (2020). African Swine Fever: Lessons to learn from past eradication experiences. A systematic review. *Frontiers in Veterinary Science*, *7*, 296. <a href="https://doi.org/10.3389/fvets.2020.00296">https://doi.org/10.3389/fvets.2020.00296</a>

Dautartas, A., Kenyhercz, M.W., Vidoli, G.M., Meadows Jantz, L., Mundorff, A. and Steadman, D.W., (2018). Differential decomposition among pig, rabbit, and human remains. *Journal of forensic sciences*, 63(6), pp.1673-1683. <a href="https://doi.org/10.1111/1556-4029.13784">https://doi.org/10.1111/1556-4029.13784</a>

Davies, K., Goatley, L. C., Guinat, C., Netherton, C. L., Gubbins, S., Dixon, L. K., & Reis, A. L. (2017). Survival of African Swine Fever Virus in Excretions from Pigs Experimentally Infected with the Georgia 2007/1 Isolate. *Transboundary and Emerging Diseases*, 64(2), 425–431. doi.org/10.1111/tbed.12381

de Carvalho Ferreira, H. C., Backer, J. A., Weesendorp, E., Klinkenberg, D., Stegeman, J. A., & Loeffen, W. L. (2013a). Transmission rate of African swine fever virus under experimental conditions. *Veterinary Microbiology*, 165(3-4), 296–304. doi.org/10.1016/j.vetmic.2013.03.026

de Carvalho Ferreira, H. C., Weesendorp, E., Quak, S., Stegeman, J. A., & Loeffen, W. L. (2013b). Quantification of airborne African swine fever virus after experimental infection. *Veterinary Microbiology*, 165(3-4), 243–251. <a href="https://doi.org/10.1016/j.vetmic.2013.03.007">doi.org/10.1016/j.vetmic.2013.03.007</a>

de Carvalho Ferreira, H. C., Tudela Zúquete, S., Wijnveld, M., Weesendorp, E., Jongejan, F., Stegeman, A., & Loeffen, W. L. (2014). No evidence of African swine fever virus replication in hard ticks. Ticks and tick-borne diseases, 5(5), 582–589. https://doi.org/10.1016/j.ttbdis.2013.12.012

Dehhaghi M, Kazemi Shariat Panahi H, Holmes EC, Hudson BJ, Schloeffel R, Guillemin GJ. Human (2019) Tick-Borne Diseases in Australia. *Frontiers in Cellular and Infection Microbiology*, 28, 9(3).

Dent J.B. and Blackie M.J. (1979). Model-Evaluation. *Systems Simulation in Agriculture*, Springer Netherlands, 94-117. https://doi.org/10.1007/978-94-011-6373-6 5

Department of Sustainability, Environment, Water, Population and Communities (2021). Terrestrial Ecoregions in Australia.

https://www.environment.gov.au/land/nrs/science/ibra/australias-ecoregions

Depner, K. *et al.* Epidemiological analyses of African swine fever in the Baltic States and Poland. *EFSA Journal*, 15 (2017). doi.org/10.2903/j.efsa.2017.5068

Dexter, N. (1990). Population density and management of feral pigs at Aurukun, North Queensland. B. o. R. Resources. Canberra, Department of Agriculture.

Dexter, N. (1996). The effect of an intensive shooting exercise from a helicopter on the behaviour of surviving feral pigs. *Wildlife Research*, 23: 435-441.

Dexter, N. (1998). The influence of pasture distribution and temperature on habitat selection by feral pigs in a semi-arid environment. *Wildlife Research* 25(5): 547-559.

Doran, R.J. and Laffan, S.W. (2005). 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. Preventive Veterinary Medicine, 70, 133–152. doi.org/10.1016/j.prevetmed.2005.03.002 Driels M.R. and Shin Y.S. (2004). Determining the number of iterations for Monte Carlo simulations of weapon effectiveness (No. NPS-MAE-04-005). Naval Postgraduate School, Monterey California, Department of Mechanical and Astronomical Engineering. http://www.dtic.mil/cgi-bin/GetTRDoc?Location=U2&doc=GetTRDoc.pdf&AD=ADA423541

East I.J., Davis J., Sergeant E.S.G. and Garner M.G. (2014). Structure, dynamics and movement patterns of the Australian pig industry. *Australian Veterinary Journal*, 92(3), 52-57. doi:10.1111/avj.12141

Eblé, P. L., Hagenaars, T. J., Weesendorp, E., Quak, S., Moonen-Leusen, H. W., & Loeffen, W. (2019). Transmission of African Swine Fever Virus via carrier (survivor) pigs does occur. Veterinary microbiology, 237, 108345. https://doi.org/10.1016/j.vetmic.2019.06.018

EFSA (2014a). Scientific Opinion on African Swine Fever. *EFSA Journal 2014*, 12(4), European Food Safety Authority Panel on Animal Health and Welfare. doi.org/10.2903/j.efsa.2014.3628

EFSA (2014b). Evaluation of possible mitigation measures to prevent introduction and spread of African swine fever virus through wild boar. *EFSA Journal 2014*;12(3):3616, European Food Safety Authority.

EFSA (2018a). Scientific report on the epidemiological analyses of African swine fever in the European Union (November 2017 until November 2018). *EFSA Journal 2018*, 16(11):5494, 106 pp. European Food Safety Authority. doi.org/10.2903/j.efsa.2018.5494

EFSA (2018b). African swine fever in wild boar. *EFSA Journal*, 16(7):e05344 https://doi.org/10.2903/j.efsa.2018.5344

EFSA (2020). Scientific report on the epidemiological analyses of African swine fever in the European Union (November 2018 to October 2019). *EFSA Journal* 2020;18(1):5996, 107 pp. doi.org/10.2903/j.efsa.2020.5996

Farez, S., & Morley, R. S. (1997). Potential animal health hazards of pork and pork products. *Revue scientifique et technique (International Office of Epizootics)*, 16(1), 65–78. doi.org/10.20506/rst.16.1.992

Froese, J.G., Smith, C.S., Durr, P.A., McAlpine, C.A., van Klinken, R.D. (2017) Modelling seasonal habitat suitability for wide-ranging species: Invasive wild pigs in northern Australia. *PLoS ONE* 12(5): e0177018. <a href="https://doi.org/10.1371/journal.pone.0177018">https://doi.org/10.1371/journal.pone.0177018</a>

Gallardo, C., Soler, A., Nieto, R., Cano, C., Pelayo, V., Sánchez, M. A., Pridotkas, G., Fernandez-Pinero, J., Briones, V., & Arias, M. (2017). Experimental Infection of Domestic Pigs with African Swine Fever Virus Lithuania 2014 Genotype II Field Isolate. *Transboundary and Emerging Diseases*, 64(1), 300–304. https://doi.org/10.1111/tbed.12346

Gallardo, M.C., Reoyo, A.d.I.T., Fernández-Pinero, J. *et al.* (2015) African swine fever: a global view of the current challenge. *Porcine Health Management* 1, 21. <a href="https://doi.org/10.1186/s40813-015-0013-y">https://doi.org/10.1186/s40813-015-0013-y</a>

Gallardo, C., Soler, A., Nieto, R., Cano, C., Pelayo, V., Sánchez, M. A., Pridotkas, G., Fernandez-Pinero, J., Briones, V., & Arias, M. (2017). Experimental Infection of Domestic Pigs with African Swine Fever Virus Lithuania 2014 Genotype II Field Isolate. *Transboundary and Emerging Diseases*, 64(1), 300–304. https://doi.org/10.1111/tbed.12346

Gallardo et al. (2018). Evolution in Europe of African swine fever genotype II viruses from highly to moderately virulent. *Veterinary microbiology*, 219, 70–79.

https://doi.org/10.1016/j.vetmic.2018.04.001

Gaudreault, N. N., Madden, D. W., Wilson, W. C., Trujillo, J. D., & Richt, J. A. (2020). African Swine Fever Virus: An Emerging DNA Arbovirus. *Frontiers in veterinary science*, *7*, 215. https://doi.org/10.3389/fvets.2020.00215

Geard, N., Bradhurst, R., McVernon J., Handley, A. and Bines, J. (2021). An agent-based model of neonatal rotavirus vaccination. In preparation.

Gentle, M., Pople, A., Scanlan, J.C. & Carter, J. (2009). The dynamics of feral pig (*Sus scrofa*) populations in response to food supply, *Wildlife Research*, 46(3), 191-204. doi.org/10.1111/j.1365-2664.2005.01094.x

Gentle, M., J. Speed and D. Marshall (2015). Consumption of crops by feral pigs (*Sus scrofa*) in a fragmented agricultural landscape. *Australian Mammalogy*, 37(2): 194-200. https://doi.org/10.1071/AM15003

Gentle M. and Pople A. (2013). Effectiveness of commercial harvesting in controlling feral pig populations, *Wildlife Research*, 40(6), 459-469, (11 October 2013). https://doi.org/10.1071/WR13100

Gentle M., Pople A., Scanlan J.C. and Carter J. (2019) The dynamics of feral pig (*Sus scrofa*) populations in response to food supply. *Wildlife Research*, 46, 191-204. https://doi.org/10.1071/WR17176

Gibbens J.C., Sharpe C.E., Wilesmith J.W., Mansley L.M., Michalopoulou E., Ryan J.B., Hudson M., 2001. Descriptive epidemiology of the 2001 foot-and-mouth disease epidemic in Great Britain: the first five months. *Veterinary Record*, 149(24), 729–743.

Giles, J. R. (1980). *The ecology of the feral pig in western New South Wales*. Doctor of Philosophy Ph.D. Thesis, University of Sydney.

Gogin, A., Gerasimov, V., Malogolovkin, A., & Kolbasov, D. (2013). African swine fever in the North Caucasus region and the Russian Federation in years 2007-2012. *Virus Research*, 173(1), 198–203. <a href="https://doi.org/10.1016/j.virusres.2012.12.007">https://doi.org/10.1016/j.virusres.2012.12.007</a>

Gonzague, M., Roger, F., Bastos, A., Burger, C., Randriamparany, T., Smondack, S., & Cruciere, C. (2001). Isolation of a non-haemadsorbing, non-cytopathic strain of African swine fever virus in Madagascar. *Epidemiology and Infection*, 126(3), 453–459. https://doi.org/10.1017/s0950268801005465

Green, L. E., & Medley, G. F. (2002). Mathematical modelling of the foot and mouth disease epidemic of 2001: strengths and weaknesses. *Research in veterinary science*, 73(3), 201–205. https://doi.org/10.1016/s0034-5288(02)00106-6

Gulenkin, V. M., Korennoy, F. I., Karaulov, A. K., & Dudnikov, S. A. (2011). Cartographical analysis of African swine fever outbreaks in the territory of the Russian Federation and computer modeling of the basic reproduction ratio. *Preventive Veterinary Medicine*, 102(3), 167–174. https://doi.org/10.1016/j.prevetmed.2011.07.004

Guinat, C., Reis, A. L., Netherton, C. L., Goatley, L., Pfeiffer, D. U., & Dixon, L. (2014). Dynamics of African swine fever virus shedding and excretion in domestic pigs infected by intramuscular inoculation and contact transmission. *Veterinary Research*, *45*(1), 93. https://doi.org/10.1186/s13567-014-0093-8

Guinat, C., Gubbins, S., Vergne, T., Gonzales, J. L., Dixon, L., & Pfeiffer, D. U. (2016a). Experimental pig-to-pig transmission dynamics for African swine fever virus, Georgia 2007/1 strain. *Epidemiology and Infection*, *144*(1), 25–34. <a href="https://doi.org/10.1017/S0950268815000862">https://doi.org/10.1017/S0950268815000862</a>

Guinat, C., Gogin, A., Blome, S., Keil, G., Pollin, R., Pfeiffer, D. U., & Dixon, L. (2016b). Transmission routes of African swine fever virus to domestic pigs: current knowledge and future research directions. *The Veterinary record*, *178*(11), 262–267. <a href="https://doi.org/10.1136/vr.103593">https://doi.org/10.1136/vr.103593</a>

Guinat, C., Porphyre, T., Gogin, A., Dixon, L., Pfeiffer, D. U., & Gubbins, S. (2018). Inferring withinherd transmission parameters for African swine fever virus using mortality data from outbreaks in the Russian Federation. *Transboundary and Emerging Diseases*, 65(2), e264–e271. https://doi.org/10.1111/tbed.12748

Gulenkin, V. M., Korennoy, F. I., Karaulov, A. K., & Dudnikov, S. A. (2011). Cartographical analysis of African swine fever outbreaks in the territory of the Russian Federation and computer modeling of the basic reproduction ratio. *Preventive Veterinary Medicine*, *102*(3), 167–174. https://doi.org/10.1016/j.prevetmed.2011.07.004

Halasa, T., Bøtner, A., Mortensen, S., Christensen, H., Toft, N., & Boklund, A. (2016a). Simulating the epidemiological and economic effects of an African swine fever epidemic in industrialized swine populations. *Veterinary Microbiology*, 193, 7–16. https://doi.org/10.1016/j.vetmic.2016.08.004

Halasa, T., Boklund, A., Bøtner, A., Toft, N., & Thulke, H. H. (2016b). Simulation of Spread of African Swine Fever, Including the Effects of Residues from Dead Animals. *Frontiers in Veterinary Science*, 3, 6. https://doi.org/10.3389/fvets.2016.00006

Halasa, T., Bøtner, A., Mortensen, S., Christensen, H., Wulff, S. B., & Boklund, A. (2018). Modeling the Effects of Duration and Size of the Control Zones on the Consequences of a Hypothetical African Swine Fever Epidemic in Denmark. *Frontiers in Veterinary Science*, 5, 49. <a href="https://doi.org/10.3389/fvets.2018.00049">https://doi.org/10.3389/fvets.2018.00049</a>

Hassall and Associates (2007). A review and analysis of saleyard marketing in Australia. Report prepared for the Department of Agriculture, Fisheries and Forestry, Canberra, ACT, Australia.

https://www.agriculture.gov.au/sites/default/files/sitecollectiondocuments/animal-plant/animal-health/livestock-movement/saleyards-movement-ead.pdf

Hayes, B. H., Andraud, M., Salazar, L. G., Rose, N., & Vergne, T. (2021). Mechanistic modelling of African swine fever: A systematic review. *Preventive veterinary medicine*, 191, 105358. Advance online publication. <a href="https://doi.org/10.1016/j.prevetmed.2021.105358">https://doi.org/10.1016/j.prevetmed.2021.105358</a>

Hayama, Y., Shimizu, Y., Murato, Y., Sawai, K., & Yamamoto, T. (2020). Estimation of infection risk on pig farms in infected wild boar areas-Epidemiological analysis for the reemergence of classical swine fever in Japan in 2018. *Preventive veterinary medicine*, 175, 104873. https://doi.org/10.1016/j.prevetmed.2019.104873

Hernández-Jover, M., Schembri N., Toribio J-A. and Holyoake P.K. (2009). Evaluation of the implementation of new traceability and food safety requirements in the pig industry in eastern Australia. *Australian Veterinary Journal*, 87(10), 387-396. doi:10.1111/j.1751-0813.2009.00483.x

Hernández-Jover M., Hayes L. and Toribo J-A. (2014). Smallholder production in Australia – Final Report. Charles Sturt University. http://www.agriculture.gov.au/SiteCollectionDocuments/animal-plant/pests-diseases/biosecurity/animal/smallholder-report.pdf

Hernández-Jover, M., Schembri, N., Holyoake, P. K., Toribio, J. L., & Martin, P. A. (2016). A Comparative Assessment of the Risks of Introduction and Spread of Foot-and-Mouth Disease among Different Pig Sectors in Australia. *Frontiers in Veterinary Science*, *3*, 85. <a href="https://doi.org/10.3389/fvets.2016.00085">https://doi.org/10.3389/fvets.2016.00085</a>

Heise-Pavlov, P. M. and Heise-Pavlov, S.R. (2003). Feral pigs in tropical lowland rainforest of northeastern Australia: ecology, zoonoses and management. *Wildlife Biology* 9(4): 21-27, 27. https://doi.org/10.2981/wlb.2003.060

Hone, J. (1990). How many feral pigs in Australia? Australian Wildlife Research 17(6): 571-572.

Hone, J. (2002). Feral pigs in Namadgi National Park, Australia: dynamics, impacts and management. *Biological Conservation*, 105(2): 231-242. <a href="https://doi.org/10.1016/S0006-3207(01)00185-9">https://doi.org/10.1016/S0006-3207(01)00185-9</a>

Hone, J. and T. Buckmaster (2015). How many are there? The use and misuse of continental-scale wildlife abundance estimates. *Wildlife Research* 41(6): 473-479. <a href="https://doi.org/10.1071/WR14059">https://doi.org/10.1071/WR14059</a>

Hone J. (2020) How many feral pigs in Australia? An update. *Australian Journal of Zoology* 67, 215-220. https://doi.org/10.1071/ZO20077

Hu, B., Gonzales, J.L. & Gubbins, S. (2017). Bayesian inference of epidemiological parameters from transmission experiments. *Scientific Reports* 7, 16774. <a href="https://doi.org/10.1038/s41598-017-17174-8">https://doi.org/10.1038/s41598-017-17174-8</a>

Huyvaert, K. P., Russell, R. E., Patyk, K. A., Craft, M. E., Cross, P. C., Garner, M. G., Martin, M. K., Nol, P., & Walsh, D. P. (2018). Challenges and Opportunities Developing Mathematical Models of

Shared Pathogens of Domestic and Wild Animals. *Veterinary Sciences*, 5(4), 92. doi.org/10.3390/vetsci5040092

Iglesias, I., Muñoz, M. J., Montes, F., Perez, A., Gogin, A., Kolbasov, D., & de la Torre, A. (2016). Reproductive Ratio for the Local Spread of African Swine Fever in Wild Boars in the Russian Federation. *Transboundary and emerging diseases*, 63(6), e237–e245. doi.org/10.1111/tbed.12337

Jaing, C., Rowland, R.R.R., Allen, J.E. et al. (2017). Gene expression analysis of whole blood RNA from pigs infected with low and high pathogenic African swine fever viruses. *Scientific Reports* 7, 10115. https://doi.org/10.1038/s41598-017-10186-4

Jori, F., A. Relun, B. Trabucco, F. Charrier, O. Maestrini, D. Chavernac, D. Cornelis, F. Casabianca and E. M. C. Etter (2017). Questionnaire-Based Assessment of Wild Boar/Domestic Pig Interactions and Implications for Disease Risk Management in Corsica. *Frontiers in Veterinary Science* 4. https://doi.org/10.3389/fvets.2017.00198

Keeling M.J. and Rohani P. (2008). *Modeling infectious diseases in humans and animals*. Princeton University Press, Princeton and Oxford.

King, K., Chapman, D., Argilaguet, J. M., Fishbourne, E., Hutet, E., Cariolet, R., Hutchings, G., Oura, C. A., Netherton, C. L., Moffat, K., Taylor, G., Le Potier, M. F., Dixon, L. K., & Takamatsu, H. H. (2011). Protection of European domestic pigs from virulent African isolates of African swine fever virus by experimental immunisation. *Vaccine*, 29(28), 4593–4600. https://doi.org/10.1016/j.vaccine.2011.04.052

Kipanyula, M. J., & Nong'ona, S. W. (2017). Variations in clinical presentation and anatomical distribution of gross lesions of African swine fever in domestic pigs in the southern highlands of Tanzania: a field experience. *Tropical Animal Health and Production*, 49(2), 303–310. https://doi.org/10.1007/s11250-016-1193-4

Kompas, T., Bradhurst, R., Garner G., East I., Iglesias R., Al-Riyami S., Stevenson M. (2018). Vector-borne spread of Animal Disease (CEBRA Project 1608B). Technical Report for the Department of Agriculture, Water and Environment.

https://minerva-access.unimelb.edu.au/handle/11343/274363

Korennoy, F. I., Gulenkin, V. M., Gogin, A. E., Vergne, T., & Karaulov, A. K. (2017). Estimating the Basic Reproductive Number for African Swine Fever Using the Ukrainian Historical Epidemic of 1977. *Transboundary and Emerging Diseases*, 64(6), 1858–1866. https://doi.org/10.1111/tbed.12583

Kovalenko J, Sidorov M, Burba L. (1965). Experimental investigations on African swine fever. *Bull. Off. Int. Epizoot.* 63, 169-89.

Kukielka, E., J. A. Barasona, C. E. Cowie, J. A. Drewe, C. Gortazar, I. Cotarelo and J. Vicente (2013). Spatial and temporal interactions between livestock and wildlife in South Central Spain assessed by camera traps. *Preventive Veterinary Medicine*, 112(3-4): 213-221.

https://doi.org/10.1016/j.prevetmed.2013.08.008

Laffan, S.W., Lubarsky, E., Ward, M.P. and Highfield, L.D. (2007). A Geographic Automata System for Modelling Disease Outbreaks, Proceedings of the 17th International Congress on Modelling and Simulation (MODSIM2007), Modelling and Simulation Society of Australia and New Zealand, Wellington, New Zealand, December 2007, 1252-1257.

Lange, M. (2015). Alternative control strategies against ASF in wild boar populations PG EcoEpi Helmholtz Centre for Environmental Research Leipzig / Halle Department Ecological Modelling. July, 1–29.

Lange, M., Kramer-Schadt, S., Blome, S., Beer, M., & Thulke, H. H. (2012). Disease severity declines over time after a wild boar population has been affected by classical swine fever - legend or actual epidemiological process? *Preventive veterinary medicine*, 106(2), 185–195. doi.org/10.1016/j.prevetmed.2012.01.024

Lange, M., & Thulke, H. (2017). Elucidating transmission parameters of African swine fever through wild boar carcasses by combining spatio-temporal notification data and agent-based modelling. *Stochastic environmental research and risk assessment,* 31, 379-391. <a href="https://doi.org/10.1007/s00477-016-1358-8#">doi.org/10.1007/s00477-016-1358-8#</a> blank

Leslie, E., Cowled, B., Garner, G., Toribio, J. A., & Ward, M. P. (2014). Effective surveillance strategies following a potential classical Swine Fever incursion in a remote wild pig population in North-Western Australia. *Transboundary and emerging diseases*, 61(5), 432–442. doi.org/10.1111/tbed.12044

Lewis, J. S., M. L. Farnsworth, C. L. Burdett, D. M. Theobald, M. Gray and R. S. Miller (2017). Biotic and abiotic factors predicting the global distribution and population density of an invasive large mammal. Scientific Reports 7(44152).doi.org/10.1038/srep44152

Lopez, J., Hurwood, D., Dryden, B., and Fuller, S. (2014). Feral pig populations are structured at fine spatial scales in tropical Queensland, Australia. *Plos One* **9**. doi: 10.1371/journal.pone.0091657.

Mačiulskis, P., Masiulis, M., Pridotkas, G., Buitkuvienė, J., Jurgelevičius, V., Jacevičienė, I., Zagrabskaitė, R., Zani, L., & Pilevičienė, S. (2020). The African Swine Fever Epidemic in Wild Boar (*Sus scrofa*) in Lithuania (2014-2018). *Veterinary Sciences*, *7*(1), 15. https://doi.org/10.3390/vetsci7010015

Mazur-Panasiuk, N., Żmudzki, J., & Woźniakowski, G. (2019). African Swine Fever Virus - Persistence in Different Environmental Conditions and the Possibility of its Indirect Transmission. *Journal of Veterinary Research*, *63*(3), 303–310. <a href="https://doi.org/10.2478/jvetres-2019-0058">https://doi.org/10.2478/jvetres-2019-0058</a>

McLeod, R. (2004) *Counting the Cost: Impact of Invasive Animals in Australia 2004*. Cooperative Research. Centre for Pest Animal Control. Canberra

Mellor, P.S., Kitching, R.P. & Wilkinson, P.J. (1987). Mechanical transmission of African swine fever virus and capripox virus by Stomoxys calcitrans. *Research in veterinary science*. 43. 109-12. doi.org/10.1016/S0034-5288(18)30753-7

Milne, G., Fermanis, C., & Johnston, P. (2008). A mobility model for classical swine fever in feral pig populations. *Veterinary research*, 39(6), 53. <a href="https://doi.org/10.1051/vetres:2008029">doi.org/10.1051/vetres:2008029</a>

Mitchell J., Dorney W., Mayer R., McIlroy J. (2009) Migration of feral pigs (*Sus scrofa*) in rainforests of north Queensland: fact or fiction? *Wildlife Research*, **36**, 110-116. doi.org/10.1071/WR06066

Mur, L., Sánchez-Vizcaíno, J. M., Fernández-Carrión, E., Jurado, C., Rolesu, S., Feliziani, F., Laddomada, A., & Martinez Lopez, B. (2016). Understanding African Swine Fever infection dynamics in Sardinia using a spatially explicit transmission model in domestic pig farms. *Transboundary and Emerging Diseases*. https://doi.org/10.1111/tbed.12636

Nielsen, J. P., Larsen, T. S., Halasa, T., & Christiansen, L. E. (2017). Estimation of the transmission dynamics of African swine fever virus within a swine house. *Epidemiology and Infection*, *145*(13), 2787–2796. https://doi.org/10.1017/S0950268817001613

Nigsch, A., Costard, S., Jones, B. A., Pfeiffer, D. U., & Wieland, B. (2013). Stochastic spatio-temporal modelling of African swine fever spread in the European Union during the high-risk period. *Preventive Veterinary Medicine*, *108*(4), 262–275. https://doi.org/10.1016/j.prevetmed.2012.11.003

Nurmoja, I., Mõtus, K., Kristian, M., Niine, T., Schulz, K., Depner, K., & Viltrop, A. (2020). Epidemiological analysis of the 2015-2017 African swine fever outbreaks in Estonia. *Preventive Veterinary Medicine*, 181, 104556. <a href="https://doi.org/10.1016/j.prevetmed.2018.10.001">https://doi.org/10.1016/j.prevetmed.2018.10.001</a>

Oberin, M. (2020). *Mathematical modelling the within-herd spread of African swine fever in Australia*. Honours thesis. Faculty of Veterinary and Agricultural Sciences, University of Melbourne, Australia.

Olesen, A. S., Lohse, L., Boklund, A., Halasa, T., Gallardo, C., Pejsak, Z., Belsham, G. J., Rasmussen, T. B., & Bøtner, A. (2017). Transmission of African swine fever virus from infected pigs by direct contact and aerosol routes. *Veterinary Microbiology*, *211*, 92–102. https://doi.org/10.1016/j.vetmic.2017.10.004

Olesen, A. S., Lohse, L., Hansen, M. F., Boklund, A., Halasa, T., Belsham, G. J., Rasmussen, T. B., Bøtner, A., & Bødker, R. (2018). Infection of pigs with African swine fever virus via ingestion of stable flies (Stomoxys calcitrans). *Transboundary and Emerging Diseases*, 65(5), 1152–1157. doi.org/10.1111/tbed.12918

Oļševskis E, Guberti V, Seržants M, et al. (2016). African swine fever virus introduction into the EU in 2014: Experience of Latvia. *Research in Veterinary Science*. 105:28-30. doi.org/10.1016/j.rvsc.2016.01.006

Olugasa, B. O., & Ijagbone, I. F. (2007). Pattern of spread of African swine fever in south-western Nigeria, 1997-2005. *Veterinaria Italiana*, *43*(3), 621–628.

O'Neill, X., White, A., Ruiz-Fons, F. *et al.* (2020). Modelling the transmission and persistence of African swine fever in wild boar in contrasting European scenarios. *Sci Rep* 10, 5895. doi.org/10.1038/s41598-020-62736-y

Pavlov, P. and J. Hone (1982). The Behaviour of Feral Pigs, Sus scrofa, in Flocks of Lambing Ewes. *Wildlife Research*, 9(1): 101-109.

Pearson, H.E., Toribio, J.-A.L. Hernandez-Jover, M., Marshall, D., Lapidge, S.J. (2014). Pathogen presence in feral pigs and their movement around two commercial pig farms in Queensland, *Australia. Vet. Rec.* 174,1–8.

Pearson, H.E. (2012). Understanding and mitigating the risk of pathogen transmission from wild animals to domestic pigs in Australia, Thesis submitted to the University of Sydney, Veterinary Science. <a href="http://hdl.handle.net/2123/8738">http://hdl.handle.net/2123/8738</a>.

Pearson, H. E., Toribio, J., Lapidge, S. J., & Hernández-Jover, M. (2016). Evaluating the risk of pathogen transmission from wild animals to domestic pigs in Australia. *Preventive veterinary medicine*, 123, 39–51. <a href="https://doi.org/10.1016/j.prevetmed.2015.11.017">https://doi.org/10.1016/j.prevetmed.2015.11.017</a>

Peck, K. M., & Lauring, A. S. (2018). Complexities of Viral Mutation Rates. *Journal of virology*, 92(14), e01031-17. <a href="https://doi.org/10.1128/JVI.01031-17">https://doi.org/10.1128/JVI.01031-17</a>

Penrith M-L and Vosloo W (2009). Review of African swine fever: transmission, spread and control. *Journal of the South African Veterinary Association* 80(2):58–62.

Penrith, M. L., Bastos, A. D., Etter, E., & Beltrán-Alcrudo, D. (2019). Epidemiology of African swine fever in Africa today: Sylvatic cycle versus socio-economic imperatives. *Transboundary and Emerging Diseases*, 66(2), 672–686. <a href="https://doi.org/10.1111/tbed.13117">https://doi.org/10.1111/tbed.13117</a>

Penrith, M.L. Current status of African swine fever. *CABI Agric Biosci* **1,** 11 (2020). https://doi.org/10.1186/s43170-020-00011-w

Pepin KM, Davis AJ, Beasley J, Boughton R, Campbell T, Cooper SM, et al. Contact heterogeneities in feral swine: implications for disease management and future research. *Ecosphere* (2016) 7:e01230. *doi.org/10.1002/ecs2.1230* 

Pershin, A., Shevchenko, I., Igolkin, A., Zhukov, I., Mazloum, A., Aronova, E., Vlasova, N., & Shevtsov, A. (2019). A Long-Term Study of the Biological Properties of ASF Virus Isolates Originating from Various Regions of the Russian Federation in 2013-2018. *Veterinary Sciences*, 6(4), 99. https://doi.org/10.3390/vetsci6040099

Pereira de Oliveira, R., Hutet, E., Paboeuf, F., Duhayon, M., Boinas, F., Perez de Leon, A., Filatov, S., Vial, L., & Le Potier, M. F. (2019). Comparative vector competence of the Afrotropical soft tick Ornithodoros moubata and Palearctic species, O. erraticus and O. verrucosus, for African swine fever virus strains circulating in Eurasia. *PloS one*, 14(11), e0225657. https://doi.org/10.1371/journal.pone.0225657

Pietschmann, J., Guinat, C., Beer, M., Pronin, V., Tauscher, K., Petrov, A., Keil, G., & Blome, S. (2015). Course and transmission characteristics of oral low-dose infection of domestic pigs and European wild boar with a Caucasian African swine fever virus isolate. Archives of virology, 160(7), 1657–1667. https://doi.org/10.1007/s00705-015-2430-2

Pikalo, J., L. Zani, J. Huehr, M. Beer and S. Biome (2019). Pathogenesis of African swine fever in domestic pigs and European wild boar - Lessons learned from recent animal trials. *Virus Research*, 271, 197614. <a href="https://doi.org/10.1016/j.virusres.2019.04.001">https://doi.org/10.1016/j.virusres.2019.04.001</a>

Pittiglio, C., Khomenko, S. & Beltran-Alcrudo, D. (2018). Wild boar mapping using population-density statistics: From polygons to high resolution raster maps. *PloS one*, 13(5), e0193295. doi.org/10.1371/journal.pone.0193295

Plant, J. W., R. Marchant, T. D. Mitchell and J. R. Giles (1978). Neonatal lamb losses due to feral pig predation. *Aust Vet J*, 54(9): 426-429.

Plowright, W., & Parker, J. (1967). The stability of African swine fever virus with particular reference to heat and pH inactivation. *Archiv fur die gesamte Virusforschung*, 21(3), 383–402. doi.org/10.1007/BF01241738

Podgórski, T., Apollonio, M. & Keuling, O. (2018). Contact rates in wild boar populations: Implications for disease transmission. *J. Wildl. Manage*. 82(6), 1210-18 doi.org/10.1002/jwmg.21480

Podgórski, T., & Śmietanka, K. (2018). Do wild boar movements drive the spread of African Swine Fever?. Transboundary and emerging diseases, 65(6), 1588–1596. https://doi.org/10.1111/tbed.12910

Podgórski, T., Borowik, T., Łyjak, M., & Woźniakowski, G. (2020). Spatial epidemiology of African swine fever: Host, landscape and anthropogenic drivers of disease occurrence in wild boar. Preventive veterinary medicine, 177, 104691. https://doi.org/10.1016/j.prevetmed.2019.104691

Post, J., Weesendorp, E., Montoya, M., & Loeffen, W. L. (2017). Influence of Age and Dose of African Swine Fever Virus Infections on Clinical Outcome and Blood Parameters in Pigs. *Viral Immunology*, 30(1), 58–69. <a href="https://doi.org/10.1089/vim.2016.0121">https://doi.org/10.1089/vim.2016.0121</a>

Probst, C., Globig, A., Knoll, B., Conraths, F.J. & Depner, K. (2017). Behaviour of free ranging wild boar towards their dead fellows: potential implications for the transmission of African swine fever. *R. Soc. open sci.* 4(5), 170054. doi.org/10.1098/rsos.170054

Probst, C., Gethmann, J., Amler, S. *et al.* (2019). The potential role of scavengers in spreading African swine fever among wild boar. *Sci Rep* 9, 11450. <a href="https://doi.org/10.1038/s41598-019-47623-5">doi.org/10.1038/s41598-019-47623-5</a>

Probst, C., Gethmann, J., Amendt, J., Lutz, L., Teifke, J.P. & Conraths, F.J. (2020). Estimating the Postmortem Interval of Wild Boar Carcasses. *Veterinary Sciences*, 7(1), 6. <a href="https://doi.org/10.3390/vetsci7010006">doi.org/10.3390/vetsci7010006</a>

Pullar, E. M. (1953). The wild (feral) pigs of Australia: their origin, distribution and economic importance. Memoirs of the National Museum. Melbourne, National Museum. 18: 7-23.

Rawdon, T. G., Garner, M. G., Sanson, R. L., Stevenson, M. A., Cook, C., Birch, C., Roche, S. E., Patyk, K. A., Forde-Folle, K. N., Dubé, C., Smylie, T., & Yu, Z. D. (2018). Evaluating vaccination

strategies to control foot-and-mouth disease: a country comparison study. *Epidemiology and infection*, 146(9), 1138–1150. <a href="https://doi.org/10.1017/S0950268818001243">https://doi.org/10.1017/S0950268818001243</a>

Reis, A. L., Parkhouse, R., Penedos, A. R., Martins, C., & Leitão, A. (2007). Systematic analysis of longitudinal serological responses of pigs infected experimentally with African swine fever virus. *The Journal of General Virology*, 88(Pt 9), 2426–2434. https://doi.org/10.1099/vir.0.82857-0

Ridoutt, C., Lee, A., Moloney, B., Massey, P., Charman, N., & Jordan, D. (2014). Detection of brucellosis and leptospirosis in feral pigs in New South Wales. Australian veterinary journal, 92(9), 343–347. https://doi.org/10.1111/avj.12203

Roche, S. E., Garner, M. G., Wicks, R. M., East, I. J., & de Witte, K. (2014). How do resources influence control measures during a simulated outbreak of foot and mouth disease in Australia?. *Preventive veterinary medicine*, 113(4), 436–446. https://doi.org/10.1016/j.prevetmed.2013.12.003

Roche, S. E., Garner, M. G., Sanson, R. L., Cook, C., Birch, C., Backer, J. A., Dube, C., Patyk, K. A., Stevenson, M. A., Yu, Z. D., Rawdon, T. G., & Gauntlett, F. (2015). Evaluating vaccination strategies to control foot-and-mouth disease: a model comparison study. Epidemiology and infection, 143(6), 1256–1275. https://doi.org/10.1017/S0950268814001927

Sánchez-Cordón, P. J., Montoya, M., Reis, A. L., & Dixon, L. K. (2018). African swine fever: A reemerging viral disease threatening the global pig industry. *Veterinary journal (London, England: 1997), 233*, 41–48. https://doi.org/10.1016/j.tvjl.2017.12.025

Sánchez-Vizcaíno JM (2010). Early detection and contingency plans for African swine fever. 24<sup>th</sup> Conference of the OIE Regional Commission for Europe, World Organisation for Animal Health, Astana, Kazakhstan.

Sánchez-Vizcaíno, J. M., Mur, L., & Martínez-López, B. (2012). African swine fever: an epidemiological update. *Transboundary and emerging diseases*, 59 Suppl 1, 27–35. <a href="https://doi.org/10.1111/j.1865-1682.2011.01293.x">https://doi.org/10.1111/j.1865-1682.2011.01293.x</a>

Sánchez-Vizcaíno, J. M., Mur, L., Gomez-Villamandos, J. C., & Carrasco, L. (2015). An update on the epidemiology and pathology of African swine fever. *Journal of Comparative Pathology*, 152(1), 9–21. <a href="https://doi.org/10.1016/j.jcpa.2014.09.003">https://doi.org/10.1016/j.jcpa.2014.09.003</a>

Sanson R.L. (1994). The epidemiology of foot-and-mouth disease: implications for New Zealand. New Zealand Veterinary Journal, 42(2), 41-53. <a href="http://dx.doi.org/10.1080/00480169.1994.35785">http://dx.doi.org/10.1080/00480169.1994.35785</a>

Sanson, R. L., Harvey, N., Garner, M. G., Stevenson, M. A., Davies, T. M., Hazelton, M. L., O'Connor, J., Dubé, C., Forde-Folle, K. N., & Owen, K. (2011). Foot and mouth disease model verification and 'relative validation' through a formal model comparison. Revue scientifique et technique (International Office of Epizootics), 30(2), 527–540. https://doi.org/10.20506/rst.30.2.2051

Sargent, R. Verification and validation of simulation models. *J Simulation* 7, 12–24 (2013). https://doi.org/10.1057/jos.2012.20 Saunders, G. (1988). The ecology and management of feral pigs in New South Wales. Masters, Macquarie University.

Saunders, G. and H. Bryant (1988). The Evaluation of a Feral Pig Eradication Program During a Simulated Exotic Disease Outbreak. *Wildlife Research* 15(1): 73-81.

Saunders, G. (1993). The Demography of Feral Pigs (Sus Scrofa) in Kosciusko National Park, New South Wales. *Wildlife Research* 20(5): 559-569.

Schembri, N., Hernandez-Jover, M., Toribio, J. A., & Holyoake, P. K. (2015). On-farm characteristics and biosecurity protocols for small-scale swine producers in eastern Australia. *Preventive Veterinary Medicine*, *118*(1), 104–116. https://doi.org/10.1016/j.prevetmed.2014.11.008

Scherer, C., V. Radchuk, M. Franz, H.-H. Thulke, M. Lange, V. Grimm and S. Kramer-Schadt (2020). Moving infections: individual movement decisions drive disease persistence in spatially structured landscapes. *Oikos* 129(5). https://doi.org/10.1111/oik.07002

Schulz, K., Staubach, C. & Blome, S. (2017). African and classical swine fever: similarities, differences and epidemiological consequences. *Vet Res* 48, 84 (2017). https://doi.org/10.1186/s13567-017-0490-x

Schulz, K., Conraths, F. J., Blome, S., Staubach, C. & Sauter-Louis, C. (2019). African Swine Fever: Fast and Furious or Slow and Steady? *Viruses*, *11*(9), 866. <a href="https://doi.org/10.3390/v11090866">doi.org/10.3390/v11090866</a>

Sereda, A. D., Balyshev, V. M., Kazakova, A. S., Imatdinov, A. R., & Kolbasov, D. V. (2020). Protective Properties of Attenuated Strains of African Swine Fever Virus Belonging to Seroimmunotypes I-VIII. Pathogens (Basel, Switzerland), 9(4), 274. https://doi.org/10.3390/pathogens9040274

Siviy J.M., Penn M.L. and Stoddard R.W. (2007). *CMMI and Six Sigma: partners in process improvement*. Addison-Wesley, USA.

Slatyer, R., Hafi, A., Richards, K., Cozens, M., Addai, D., Cao, L., Mornement, C., Keighley, M., and Arthur, T., (2021). A cost-benefit analysis of the potential economic consequences of African swine fever in Australia. ABARES Research Report, in preparation.

Śmietanka, K., Woźniakowski, G., Kozak, E., Niemczuk, K., Frączyk, M., Bocian, Ł., Kowalczyk, A., & Pejsak, Z. (2016). African Swine Fever Epidemic, Poland, 2014-2015. Emerging infectious diseases, 22(7), 1201–1207. https://doi.org/10.3201/eid2207.151708

Smith, D., Cooper, T., Pereira, A., & Jong, J. (2019). Counting the cost: The potential impact of African Swine Fever on smallholders in Timor-Leste. *One health* (Amsterdam, Netherlands), 8, 100109. https://doi.org/10.1016/j.onehlt.2019.100109

Spencer, P. B. S. and J. O. Hampton (2005). Illegal translocation and genetic structure of feral pigs in Western Australia. *Journal of Wildlife Management*, 69(1): 377-384.

Peter B. S. Spencer, & Hampton, J. (2005). Illegal Translocation and Genetic Structure of Feral Pigs in Western Australia. *The Journal of Wildlife Management*, 69(1), 377-384. http://www.jstor.org/stable/3803613

Spickler, A.R. (2018). *African swine fever*, Center for Food Security & Public Health, Iowa State University, Ames, www.cfsph.iastate.edu/DiseaseInfo/factsheets.php.

Stahl, K., Sternberg-Lewerin, S., Blome, S., Viltrop, A., Penrith, M.L & Chenais, E. (2019). Lack of evidence for long term carriers of African swine fever virus - a systematic review *Virus Res.*, 272 (2019), Article 197725. doi.org/10.1016/j.virusres.2019.197725

Stahl, K., Sternberg-Lewerin, S., Blome, S., Viltrop, A., Penrith, M.L & Chenais, E. (2019). Lack of evidence for long term carriers of African swine fever virus - a systematic review *Virus Res.*, 272 (2019), Article 197725. doi.org/10.1016/j.virusres.2019.197725

Strahan, R. (1983). *Complete book of Australian mammals*. Sydney, Australia, Angus and Robertson.

Sur J. H. (2019). How far can African swine fever spread? *Journal of Veterinary Science*, 20(4), e41. https://doi.org/10.4142/jvs.2019.20.e41

Taylor N. (2003). Review of the use of models in informing disease control policy development and adjustment. School of Agriculture, Policy and Development, The University of Reading.

Taylor, R. A., Condoleo, R., Simons, R., Gale, P., Kelly, L. A., & Snary, E. L. (2020). The Risk of Infection by African Swine Fever Virus in European Swine Through Boar Movement and Legal Trade of Pigs and Pig Meat. *Frontiers in Veterinary Science*, *6*, 486. https://doi.org/10.3389/fvets.2019.00486

Taylor, R. A., T. Podgorski, R. R. L. Simons, S. Ip, P. Gale, L. A. Kelly and E. L. Snary (2021). Predicting spread and effective control measures for African swine fever-Should we blame the boars? *Transboundary and Emerging Disea*ses 68(2): 397-416.

Tharle, C. (2021). Modelling the spread of African swine fever in feral pig groups in Australia and the potential role of infectious carcass transmission. Master of Agricultural Science thesis. Faculty of Veterinary and Agricultural Sciences, University of Melbourne, Australia.

Tisdell, C. A. (1982). Wild pigs, environmental pest or economic resources. Sydney, Pergamon Press.

Toger, M., Benenson, I., Wang, Y., Czamanski, D. & Malkinson, D. (2018). Pigs in space: An agent-based model of wild boar (Sus scrofa) movement into cities. *Landscape and Urban Planning*. 173. doi.org/10.1016/j.landurbplan.2018.01.006

Torrens, P.M. and Benenson, I. (2005). Geographic automata systems. International *Journal of Geographical Information Science*, 19(4), 385-412. doi.org/10.1080/13658810512331325139

Truvé, J. & Lemel J. (2003). Timing and distance of natal dispersal for wild boar *Sus scrofa* in Sweden, *Wildlife Biology*, 9(4), 51-57. doi.org/10.2981/wlb.2003.056

Twigg L. E., Lowe T., Martin G., Everett M. (2005) Feral pigs in north-western Australia: basic biology, bait consumption, and the efficacy of 1080 baits. *Wildlife Research* 32, 281-296. https://doi.org/10.1071/WR04110

Vose, D. (2008). Risk analysis: A quantitative guide. Third edition. John Wiley and Sons, Ltd, Chichester, West Sussex, United Kingdom.

Ward, M.P., Laffan, S.W. & Highfield, L.D. Modelling spread of foot-and-mouth disease in wild white-tailed deer and feral pig populations using a geographic-automata model and animal distributions. (2009). *Preventive Veterinary Medicine*. 91(1), 55-63. doi.org/10.1016/j.prevetmed.2009.05.005

Ward, M. P., Garner, M. G., & Cowled, B. D. (2015). Modelling foot-and-mouth disease transmission in a wild pig-domestic cattle ecosystem. *Australian veterinary journal*, 93(1-2), 4–12. doi.org/10.1111/avj.12278

Walczak, M., Żmudzki, J., Mazur-Panasiuk, N., Juszkiewicz, M., & Woźniakowski, G. (2020). Analysis of the Clinical Course of Experimental Infection with Highly Pathogenic African Swine Fever Strain, Isolated from an Outbreak in Poland. Aspects Related to the Disease Suspicion at the Farm Level. *Pathogens* (Basel, Switzerland), 9(3), 237. <a href="https://doi.org/10.3390/pathogens9030237">https://doi.org/10.3390/pathogens9030237</a>

Weitz, J. & Dushoff, J. (2015). Modeling Post-death Transmission of Ebola: Challenges for Inference and Opportunities for Control. *Scientific Reports*, 5, 8751 (2015). doi.org/10.1038/srep08751

West, P. (2008). *Assessing Invasive Animals in Australia 2008*. Invasive Animals Cooperative Research Centre, Australian Government National Land & Water Resources Audit.

Whytlaw, P., W. Edwards and B. Congdon (2013). Marine turtle nest depredation by feral pigs (Sus scrofa) on the Western Cape York Peninsula Australia: Implications for management. *CSIRO Wildlife Research* 40: 377-384.

Wilson, G., N. Dexter, P. O'Brien and M. Bomford (1992). *Pest Animals in Australia: A Survey of Introduced Wild Mammals*, Bureau of Rural Resources by Kangaroo Press.

Wilson, C., Marshall, D., Gentle, M.N. (In preparation). A comparison of feral pig (Sus scrofa) home and core range size and activity levels across four sites in Australia. Biosecurity Queensland, Department of Primary Industries and Fisheries. (Queensland.)

Wishart, J., S. Lapidge, M. Braysher, S. D. Sarre and J. Hone (2015). Observations on effects of feral pig (Sus scrofa) age and sex on diet. *Wildlife Research* 42(6): 470-474.

Wilkinson, P.J., Donaldson, A.I., Greig, A., Bruce, W., (1977). Transmission studies with African swine fever virus – infections of pigs by airborne virus. *J. Comp. Pathol.* 87, 487–495.

Wilkinson, P.J., Donaldson, A.I., (1977). Transmission studies with African swine fever virus. The early distribution of virus in pigs infected by airborne virus. *J. Comp. Pathol.* 87, 497–501.

Woolnough, A. P., P. B. West and G. R. Saunders (2004). Institutional knowledge as a tool for pest animal management. *Ecological Management & Restoration* 5(3): 226-228.

Wozniakowski, G., Pejsak, Z., and Jabłonski, A. (2021). Emergence of African Swine Fever in Poland (2014–2021). Successes and Failures in Disease Eradication. *Agriculture* 2021,11, 738. <a href="https://doi.org/10.3390/agriculture11080738">https://doi.org/10.3390/agriculture11080738</a>

Wu, N., C. Abril, A. Thomann, E. Grosclaude, M. G. Doherr, P. Boujon and M. P. Ryser-Degiorgis (2012). Risk factors for contacts between wild boar and outdoor pigs in Switzerland and investigations on potential Brucella suis spill-over. *Bmc Veterinary Research* 8.

Wyckoff, A. C., S. E. Henke, T. A. Campbell, D. G. Hewitt and K. C. VerCauteren (2009). FERAL SWINE CONTACT WITH DOMESTIC SWINE: A SEROLOGIC SURVEY AND ASSESSMENT OF POTENTIAL FOR DISEASE TRANSMISSION. *Journal of Wildlife Diseases* 45(2): 422-429.

Yadav, S., Olynk Widmar, N.J. & Weng, H.Y. (2016). Modeling Classical Swine Fever Outbreak-Related Outcomes. *Frontiers in Veterinary Science*, 3, 7. <u>doi.org/10.3389/fvets.2016.00007</u>

Yoo, D., Kim, H., Lee, J. Y., & Yoo, H. S. (2020). African swine fever: Etiology, epidemiological status in Korea, and perspective on control. *Journal of Veterinary Science*, *21*(2), e38. https://doi.org/10.4142/jvs.2020.21.e38

Yoon H, Hong SK, Lee I, et al. (2020). Clinical symptoms of African swine fever in domestic pig farms in the Republic of Korea, 2019. *Transboundary and Emerging Diseases*. 2020 Mar. DOI: 10.1111/tbed.13552.

Zhao, D., Liu, R., Zhang, X., Li, F., Wang, J., Zhang, J., Liu, X., Wang, L., Zhang, J., Wu, X., Guan, Y., Chen, W., Wang, X., He, X., & Bu, Z. (2019). Replication and virulence in pigs of the first African swine fever virus isolated in China. *Emerging microbes & infections*, 8(1), 438–447. https://doi.org/10.1080/22221751.2019.1590128

# APPENDIX A DOMESTIC PIG WITHIN-HERD EBM PARAMETERISATION

Table 37. Domestic pig within-herd EBM parameterisation for ASFV Georgia 2007/1

EBM parameter (Figure 4)	VLC	MLC	SC	SGT	SH	PK	References		
transmission rate (β)	1.5	1.5	1.2	0.6	0.8	0.8	Adapted from Gulenkin et al., 2011; Guinat et al., 2016; Nielsen et al., 2017; Guinat et al., 2018; Oberin 2020		
average latent period $(1/\sigma)$			4 da	ays			Penrith & Vosloo, 2009; Blome et al., 2013; Guinat et al., 2014; Pietschmann et al., 2015; Guinat et al., 2016a; Beltrán-Alcrudo et al.,		
average infectious period $(1/\gamma)$			5 da	ays			2017		
carcass tx rate (B <sub>D</sub> )	0						It is assumed that carcasses are removed promptly from domestic pig herds and do not		
carcass infectious period (1/ε) days			0				play a role in transmission		
R <sub>0</sub> (derived)	7.5	7.5	6.0	3.0	4.0	4.0	Pietschmann et al., 2015; Guinat et al., 2019; Schultz et al., 2019		
probability of death after infection (m)			0.9	)5			Gallardo et al., 2015; Halasa et al., 2016a		
average incubation period (1/λ)	5 days						Costard et al., 2015; Guinat et al., 2016a Gallardo et al., 2018; Walczak et al., 2020		
proportion clinical (c)	1.0						Spickler, 2018		
average clinical period (1/φ)	7 days						Gallardo et al., 2018		
natural immunity	Be	taPert <sup>1</sup>	(120,	180, 36	50) da	ys	Sereda et al., 2020		

<sup>&</sup>lt;sup>1</sup>variant of the Beta distribution with parameters minimum, most likely and maximum values (Vose, 2008)

### APPENDIX B DOMESTIC PIG BETWEEN-HERD SPREAD PATHWAY PARAMETERISATION

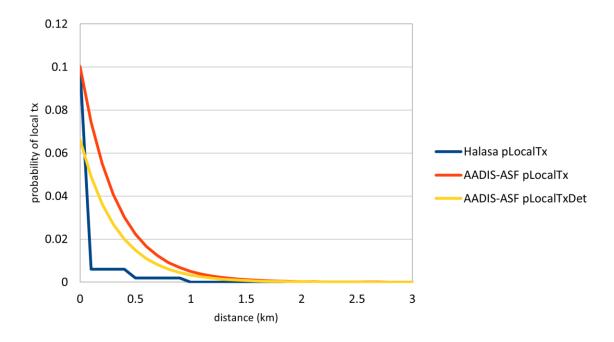
Table 38. Selected parameterisation for the AADIS-ASF local spread pathway

Local spread parameter (Equations 1-3)	Value
average daily probability of local spread transmission between an infectious herd and a neighbouring herd 0 km apart (defined per mega- region)	pastoral = 0.027 north-east = 0.031 south-east = 0.031 south-west = 0.031
local spread kernel radius	3 km
local spread decay mode	exponential
decay constant	-6
infectivity power	0.3
susceptibility power	0.3
risk category (reflects on-farm biosecurity measures)	herd-dependent
biosecurity weight (dampens the probability of local/indirect transmission)	see Table 2
seasonal weight (reflects environmental influence on ASFV viability)	see Table 11
Detection weight (reflects reduced probability of local spread once an outbreak has been declared)	0.66
number of transmissions from an effective contact	BetaPERT <sup>1</sup> (1, 2, 5)

<sup>&</sup>lt;sup>1</sup>variant of the Beta distribution with parameters minimum, most likely and maximum values (Vose, 2008)

The red curve in Figure 66 depicts the daily probability of silent local spread from an averagely infectious average-sized small commercial pig herd in north-east Queensland in May, to a neighbouring susceptible average-sized small commercial pig herd. The yellow curve depicts the dampened probability once an outbreak has been declared. The blue curve depicts the local spread probability curve used by Halasa and colleagues (2016a). Note that the Halasa curve is over a 2 km spatial kernel radius and the AADIS-ASF curve has been temporarily scaled down from a 3 km radius to 2 km radius for comparative purposes. Note that the AADIS-ASF probability of local spread is far more complex than the Halasa probability as it considers the dynamic (daily)

prevalence of the infectious herd, infectivity of the infectious herd (dependent on herd size), susceptibility of the susceptible herd (dependent on herd size), biosecurity measures in place on the susceptible farm, seasonal environmental effects, and distance between the infectious and susceptible herds relative to 3 km spatial kernel. In contrast, the Halasa and colleagues' probabilities are from a simple static distance-based step function that aggregates contributing risk factors across the kernel radius. Comparisons are also difficult given (i) the generally higher density of pig farms in Europe than Australia, and (ii) cultural-based smallholdings of pigs in Europe will have quite different direct/indirect contact profiles than hobbyist smallholdings of pigs in Australia.



**Figure 66.** Example of AADIS-ASF probability of local spread between small commercial pig farms during the silent spread phase (red) and during the control program (yellow)

**Table 39.** Seasonal weights reflecting environmental influence on ASFV transmission

Mega region	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
PL	0.7882	0.8641	0.9767	1.1419	1.1936	1.2057	1.1994	1.1845	1.0692	0.8443	0.7526	0.7798
NE	0.9648	0.9870	0.9987	1.0190	1.0191	1.0279	1.0367	1.0272	1.0273	1.0009	0.9549	0.9364
SE	0.9262	0.9713	1.0014	1.0306	1.0243	1.0211	1.0211	1.0243	1.0180	1.0125	0.9744	0.9748
sw	0.8645	0.8911	0.9458	1.0431	1.0581	1.0440	1.0581	1.0581	1.0581	1.0390	0.9984	0.9415

References: adapted from Mazur-Panasiuk et al., 2019

Table 40. Selected parameterisation for the AADIS-ASF indirect spread pathway

Indirect spread parameter	VLC	MLC	sc	SGT	SH	PK		
Poisson distribution of average daily number of indirect contacts	1.166	0.544	0.339	0.190	0.177	0.177		
distribution of distance in km of indirect contacts BetaPert <sup>1</sup> (minimum, most likely, maximum)	3 100 350	100 70 70 50 70						
destination herd type for an indirect movement	contact	matrix (de	pendent o		and dest	ination		
probability of infection from an indirect contact	Pastoral = 0.010 North-east = 0.011							
risk category (reflects on-farm biosecurity measures)	herd-dependent							
biosecurity weight (dampens the probability of local/indirect transmission)	See Table 2							
seasonal weight (reflects environmental influence on ASFV viability)	See Table 39							
number of transmissions resulting from an effective indirect contact		В	etaPERT <sup>1</sup>	(1, 2, 5)				

<sup>&</sup>lt;sup>1</sup>variant of the Beta distribution with parameters minimum, most likely and maximum values (Vose, 2008)

References: QDAF

**Table 41.** Selected parameterisation for the AADIS-ASF direct spread pathway. This only applies to pig keepers as the direct movements for all other herd types are driven by historical NLIS movements.

Direct spread parameter (pig keepers only)	Value
daily number of direct contacts	Poisson (0.0082)
distance of a direct movement	BetaPERT <sup>1</sup> (3, 65, 600) km
destination premises type probabilities	farm=0.1108, saleyard=0.2429, abattoir=0.6463
destination mega-region for a direct movement	contact matrix (dependent on source mega-region and herd type)
destination herd type for a direct movement	contact matrix (dependent on source and destination herd types)
consignment size (proportion of herd size)	BetaPERT <sup>1</sup> (0.002, 0.059, 1.0)

<sup>&</sup>lt;sup>1</sup>variant of the Beta distribution with parameters minimum, most likely and maximum values (Vose, 2008)

# APPENDIX C DOMESTIC PIG CONTROL MEASURES PARAMETERISATION

 Table 42. Selected parameterisation for domestic pig control measures

Component	Parameterisation
movement controls	• state-wide CA for 28 days followed by 10 km radial CAs. Saleyard movements stopped. Direct movements dampened by 95%. Indirect movements dampened by 50%.
	• LGA-based RAs for 28 days followed by 3 km radial RAs. Saleyard movements stopped.  Direct movements dampened by 98%. Indirect movements dampened by 85%.
	declared areas lifted 28 days after all enclosed IP operations have completed.
IP Operations	destruction of all susceptible animals on IPs, followed by disposal and decontamination of premises.
	no ring destruction or preemptive destruction.
	1 destruction team ramping up to 25 between days 3 and 28 of the response
	4 disposal teams ramping up to 28 between days 7 and 30 of the response
	the number of decontamination teams were configured to not be a limiting constraint
tracing	direct and indirect movements onto and off all IPs (15-day tracing window)
	• tracing effectiveness: direct = 0.95, indirect = 0.80
surveillance	investigation of all reported SPs, DCPs and TPs
	periodic visits to at-risk premises (ARPs) inside RAs
	lab test required for confirmation of infection
	2 false positive reports of clinical signs for every true positive report
	5 surveillance teams ramping up to 40 between days 3 and 21 of the response

References: AUSVETPLAN, 2020; estimates based on discussions with QDAF

Table 43. Duration and costing parameterisation for domestic pig control measures

Parameter	VLC	MLC	SC	SGT	SH	PK
surveillance duration (days)	1	1	1	1	1	1
direct tracing duration (days)			BetaPert <sup>1</sup>	(1, 2, 3)		
indirect tracing duration (days)			BetaPert <sup>1</sup> (	(1, 3, 3)		
destruction duration (days)	4.2	0.9	0.6	0.6	0.5	0.5
disposal duration (days)	4.9	0.9	0.6	0.6	0.6	0.5
decontamination duration (days)	8.0	1.2	0.6	0.6	0.6	0.6
surveillance cost (A\$ per herd)	\$1725	\$1150	\$850	\$850	\$625	\$625
destruction cost (A\$ per pig)	\$4	\$4	\$4	\$4	\$4	\$4
disposal cost (A\$ per pig)	\$9	\$9	\$9	\$9	\$9	\$9
decontamination cost (A\$ per herd)	\$116,847	\$77,898	\$15,580	\$15,580	\$7790	\$7790
compensation cost (A\$ per pig)	\$223	\$223	\$223	\$223	\$150	\$150

<sup>&</sup>lt;sup>1</sup>variant of the Beta distribution with parameters minimum, most likely and maximum values (Vose, 2008)

References: Adapted from Slayter et al., 2021.

# APPENDIX D FERAL PIG WITHIN-GROUP EBM PARAMETERISATION

 Table 44. Feral pig within-group EBM parameterisation for ASFV Georgia 2007/1 per region

EBM parameter (Figure 12)	DES (1)	TEF (4)	TES (5)	TRS (6)	TRF (7)	References
transmission rate $(\beta_i)$			0.6		ı	Penrith & Vosloo, 2009; Blome et al., 2013; Guinat et al., 2014; Pietschmann
latent period (1/ $\sigma$ )			4 days			et al., 2015; Guinat et al., 2016a; Beltrán-Alcrudo et al., 2017
infectious period (1/γ)			5 days			
carcass transmission rate (B <sub>D</sub> )			0.6			Taylor et al., 2020
carcass infectious period (1/ε) days summer autumn winter spring	10 15 10 7 10 12 30 30 10 10 14 60 60 10 10 12 30 30 7 10				10 10	Adapted from Twigg et al., 2005; Dautartas et al. 2018; Blome et al., 2020; Carlson et al., 2020; Fischer et al., 2020; Tharle, 2021;
R <sub>0</sub> (including carcasses) summer autumn winter spring	6 9 6 4.2 6 7 18 18 6 6 8 36 36 6 6 7 18 18 4.2 6				6	Penrith & Vosloo, 2009; Blome et al., 2013; Guinat et al., 2014; Pietschmann et al., 2015; Guinat et al., 2016a; Beltrán-Alcrudo et al., 2017
probability of death after infection (m)			0.90			Gallardo et al., 2015; Halasa et al., 2016a; Guinat et al., 2016a; Gallardo et al., 2018
incubation period (1/ $\lambda$ )			5 days			
proportion clinical (c)			1.0			Spickler, 2018
clinical period (1/φ)			7 days			Gallardo et al., 2018
natural immunity (days)	Bet	taPert¹	(120,	180, 3	60)	Sereda et al., 2020
recovery period R <sub>p</sub> (days)			730			Adapted from Giles, 1980; Caley, 1993; Saunders 1993; Dexter
recovery lag Rı (days)			365			(1998); Gentle et al. (2019)
recovery gradient				tumn : ring 1.	_	

#### **APPENDIX E**

### FERAL PIG BETWEEN-GROUP SPREAD PATHWAY PARAMETERISATION

**Table 45.** Parameterisation of the feral pig between-group diffusive spread pathway

Parameter (Equation 7)	DES (1)	TEF (4)	TES (5)	TRS (6)	TRF (7)	References
contact rate summer autumn winter spring	0.07 0.14 0.14 0.07	0.07 0.14 0.14 0.07	0.07 0.14 0.14 0.07	0.07 0.07 0.07 0.07	0.07 0.07 0.07 0.07	Adapted from Podgorski et al., 2018; Taylor et al., 2020; Taylor et al., 2021; J. Vicente pers. comms 2021
diffusion mode (Moore or radial)			Moore	Э		
diffusion range (radial distance in km or Moore neighbourhood range)			r=1			Pepin et al., 2016 Podgorski et al., 2018 Scherer et al., 2020
contact choice (most suitable, most populated, random)			randor	n		
probability of effective contact			0.75	Adapted from Cowled et al., 2012		
seasonal weight summer autumn winter spring	0.8 1.0 1.2 0.9	0.8 1.0 1.2 0.9	0.8 1.0 1.2 0.9	0.8 1.0 1.2 0.9	1.0 1.0 1.0 1.0	Adapted from Mazur- Panasiuk et al., 2019

Table 46. Parameterisation of the feral pig between-group jump spread pathway (<u>disabled</u>)

Parameter (Equation 8)	DES (1)	TEF (4)	TES (5)	TRS (6)	TRF (7)	References
contact rate summer autumn winter	0.00 0.00 0.00	0.00 0.00 0.00	0.00 0.00 0.00	0.00 0.00 0.00	0.00 0.00 0.00	N/A
spring	0.00	0.00	0.00	0.00	0.00	
jump mode (directed or random)			randor	m		N/A
jump distance (km)		Beta	Pert¹ (0	0, 0, 0)		N/A
catchment radius (km)			0			N/A
contact choice (most suitable, most populated, random)			randor	N/A		
probability of effective contact			0.0	N/A		
seasonal weight			N/A			N/A

<sup>&</sup>lt;sup>1</sup>variant of the Beta distribution with parameters minimum, most likely and maximum values (Vose, 2008)

# APPENDIX F DOMESTIC PIG AND FERAL PIG SPREAD PATHWAY PARAMETERISATION

 Table 47. Parameterisation of the domestic pig to feral pig spread pathway

Parameter (Equation 9)	DES (1)	TEF (4)	TES (5)	TRS (6)	References	
spatial kernel radius			5 km			N/A
average daily probability of transmission	0.025	0.025	0.025	0.025	0.025	Adapted from Cadenas-Fernández et al., 2019; Taylor et al., 2020; Taylor et al., 2021
decay mode (linear, exponential, gaussian, Hayama)			Hayama			Hayama et al., 2020
decay alpha			2.81			Adapted from Hayama et al., 2020
decay r0			2.5			Adapted from Hayama et al., 2020
biosecurity weight (dampens the probability of transmission)		S	ee Table	2	N/A	
seasonal weight		Se	ee Table	45		N/A

 Table 48. Parameterisation of the feral pig to domestic pig spread pathway

Parameter (Equation 10)	DES (1)	TEF (4)	TES (5)	TRS (6)	TRF (7)	References		
spatial kernel radius			5 km			N/A		
average daily probability of transmission	0.05	0.05	0.05	0.05	0.05	Adapted from Cadenas-Fernández et al., 2019; Taylor et al., 2020; Taylor et al., 2021		
decay mode		ı	Hayama	9		Hayama et al., 2020		
decay alpha			2.81			Adapted from Hayama et al., 2020		
decay r0			2.5			Adapted from Hayama et al., 2020		
biosecurity weight (dampens the probability of transmission)		Se	e Table	2				
seasonal weight		See	e Table	45				

# APPENDIX G FERAL PIG SURVEILLANCE AND CONTROL PARAMETERISATION

Table 49. Parameterisation of the feral pig surveillance and control components (test data only)

Parameter	Passive		Active	Post control
(Section 3.5)	surveillance	Control	surveillance	surveillance
trigger	ongoing	domestic IP or detection in feral pigs	domestic IP or detection in feral pigs	completion of control
treatment/ surveillance area	Qld	0 km inner radius 5 km outer radius	5 km inner radius 10 km outer radius	0 km inner radius 5 km outer radius
duration	ongoing	21 days	21 days	21 days
effectiveness	sensitivity 0.25	70% knockdown	sensitivity 0.98 specificity 1.0	sensitivity 0.98 specificity 1.0
cost per area A\$	\$0	\$60,000	\$60,000	\$30,000
number of actions	ongoing	1	1	1
resources required per action	N/A	1	1	1
resource pool initial size	N/A	15	15	15
resource pool final size	N/A	60	60	60
resource pool ramp lag (days)	N/A	5	5	5
resource pool ramp duration (days)	N/A	14	14	14

# APPENDIX H VISUALISATION AND GRAPHICAL USER INTERFACE

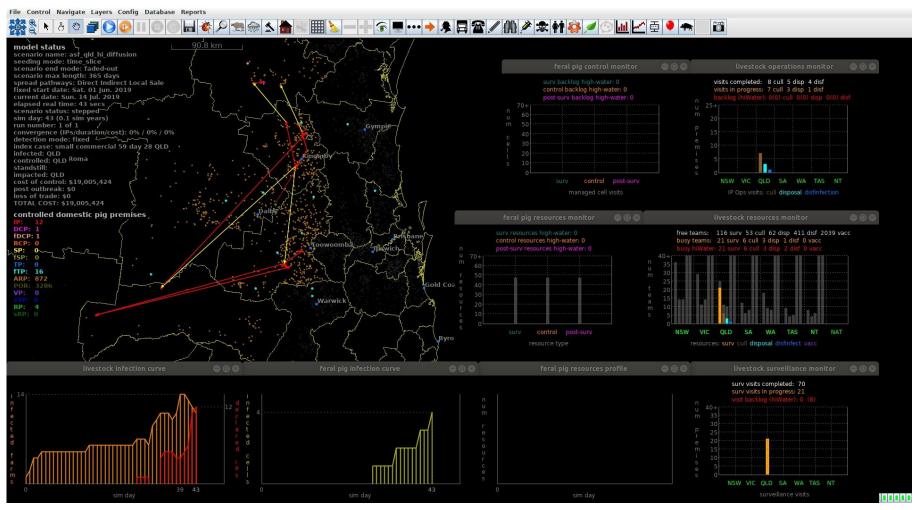


Figure 67. AADIS-ASF-QLD domestic pig infection visualisation

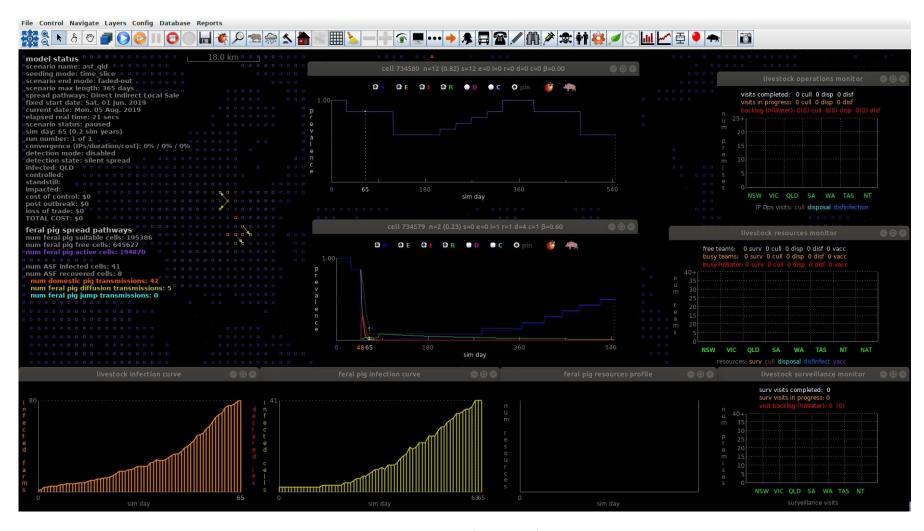


Figure 68. AADIS-ASF-QLD feral pig infection visualisation

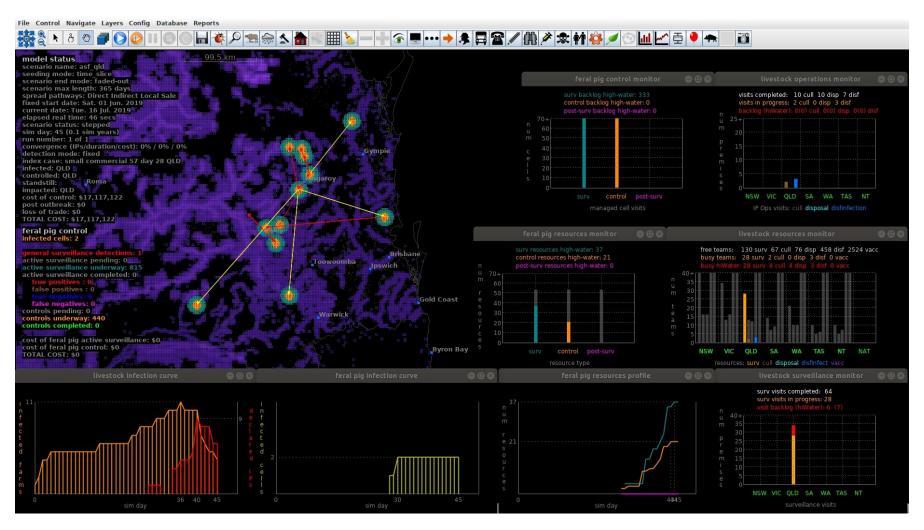
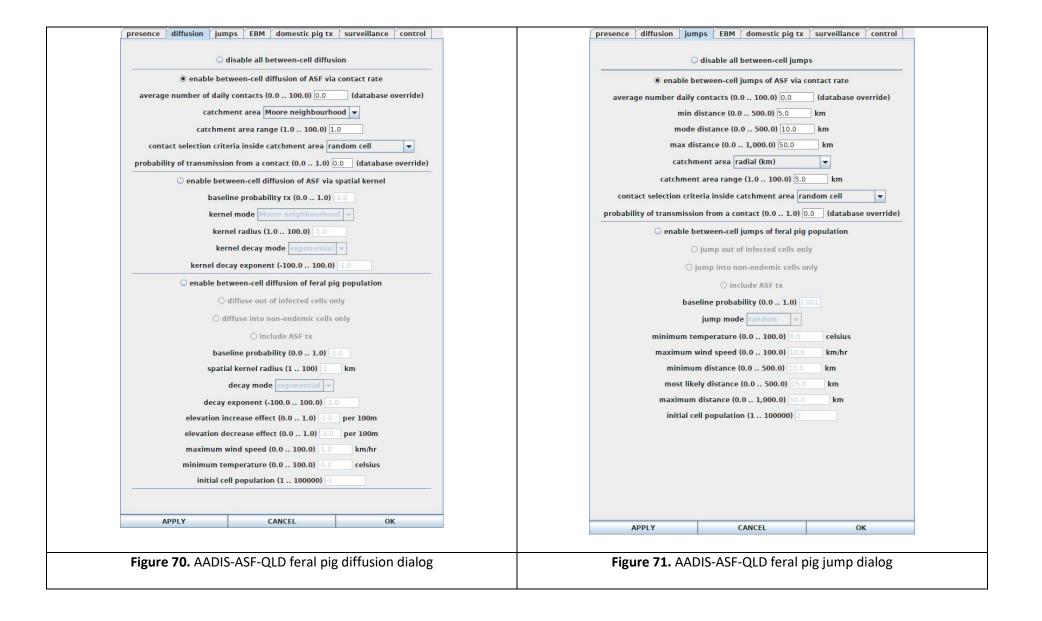


Figure 69. AADIS-ASF-QLD feral pig surveillance and control visualisation



presence diffusion jumps EBM domestic pig tx surveillance control	presence diffusion jumps EBM domestic pig tx surveillance control
presence diffusion jumps EBM domestic pig tx surveillance control    feral pig within-cell EBM overrides	enable transmission of ASF from feral pig to domestic pig spatial kernel radius (0.0 100.0) 5.0 km spatial kernel decay mode Hayama  exponential decay exponent (-100.0 100.0) 3.45388 Gaussian decay sigma (0.0 100.0) 2.81 Hayama decay alpha (0.0 100.0) 2.5 probability of tx (0.0 1.0) 0.0 (database override)  enable transmission of ASF from domestic pig to feral pig spatial kernel radius (0.0 100.0) 5.0 km spatial kernel decay mode Hayama  exponential decay exponent (-100.0 100.0) 3.45388 Gaussian decay sigma (0.0 100.0) 0.65 Hayama decay alpha (0.0 100.0) 2.81 Hayama decay r0 (0.0 100.0) 2.81 Hayama decay r0 (0.0 100.0) 2.5 probability of tx (0.0 10.0) 0.0 (database override)
APPLY CANCEL OK	APPLY CANCEL OK
Figure 72. AADIS-ASF-QLD feral pig EBM dialog	Figure 73. AADIS-ASF-QLD feral & domestic transmission dialog

