# Modelling the transmission dynamics and the effect of different control strategies for African swine fever virus in East Africa

By

MIKE BARONGO BITAMALE

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# DECLARATION

This thesis is my original work and has not been presented to any other university or institution for any award.

**MIKE BARONGO BITAMALE** (BSc, MPhil)

Sign:  Date: 7th July 2018

# DEDICATION

I dedicate this thesis to my family especially my wife and children who stood by me, encouraged me and prayed for me all through this journey. Secondly, I dedicate it to the Almighty for sustaining me through the entire process by His grace.

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# Table of Contents

# LIST OF FIGURES

# LIST OF TABLES

# LIST OF ABBREVIATIONS

| | |
|---|---|
| ASF | African Swine Fever |
| ASFV | African Swine Fever Virus |
| CF | Curve Fitting |
| CISA-INIA | Centro De Investigacion en Sanidad Animal |
| CSF | Classical Swine Fever |
| DFE | Disease Free Equilibrium |
| FMD | Food and Mouth Disease |
| GIS | Geographical Information System |
| LRM | Linear Regression Model |
| MAAIF | Ministry Of Agriculture Animal Industry and Fisheries |
| MUIENR | Makerere University Institute of Environment and Natural Resources |
| NAADS | National Agricultural Advisory Services |
| NADDEC | National Animal Disease Diagnostic and Epidemiology Centre |
| ODE | Ordinary Differential Equations |
| OIE | World Organisation for Animal Health |
| PDA | Personal Digital Assistant |
| PDNS | Porcine Dermatitis and Nephropathy Syndrome |
| PRRS | Porcine Reproductive and Respiratory Syndrome |

# SUMMARY

## MODELLING THE TRANSMISSION DYNAMICS AND THE EFFECT OF DIFFERENT CONTROL STRATEGIES FOR AFRICAN SWINE FEVER VIRUS IN EAST AFRICA

By

MIKE BARONGO BITAMALE

Promoter: Prof D Knobel

Co-promoters: Dr. RP Bishop, Dr. A Ssematimba

Department: Veterinary Tropical Diseases

Degree: PhD

African swine fever (ASF) is a highly contagious, lethal and economically devastating haemorrhagic disease of domestic pigs. Knowledge of the epidemiology of the disease is important for the design of improved control measures. Such insights of the dynamics of virus can be obtained from mathematical constructs. In this study, we used two methods to estimate the basic reproduction number ($R_0$) from field data. Our estimates predicted persistence of ASF in pig populations and recommended enhanced biosecurity measures. We developed a stochastic model to assess the relative impact of the timing of the implementation of different control strategies on disease-related mortality. The results showed that intervention within 14 days of the outbreak and using a combination of strategies was the best control option. The modelling approach was particularly valuable in that it determined an optimal timing for implementation of interventions.

A between-village spatial-deterministic model was developed. The model simulations showed that there were intervention windows of 30 days from the onset of the outbreak to reduce ASFV spread between villages. The study also analysed cross sectional data collected in a survey conducted in the study area to identify key parameters of low input production systems. We found out that farmers mostly kept local pig breeds by tethering. They fed the pigs on farm crop residues and household food leftovers or swill. We recommend timely intervention by authorities during outbreaks; the use of a cocktail of control strategies; restriction of free movement of animals; and improving the supply of affordable pig feeds to incentivize adoption of better husbandry and health practices and increasing pig productivity.

# CHAPTER 1

## Introduction

## 1.1 Background

African swine fever (ASF) is a devastating disease of domestic pigs caused by a large double stranded DNA (dsDNA) virus which is the sole member of the family, *Asfarviridae* [1–3]. The virus is ancestrally associated with and maintained either through a sylvatic cycle involving warthogs (*Phacochoerus aethiopicus*) and soft ticks in the genus *Ornithodoros* or in a domestic cycle that involves pigs of local breeds, with or without tick involvement [4]. The ASF virus is stable over a wide range of temperatures and pH which enables it to persist in excretions, carcasses and pork products from infected pigs [5].

The virus is contagious and can spread rapidly in naïve pig populations by direct or indirect contact. Susceptible pigs in most contemporary production systems become infected mainly through the direct oro-nasal route after contact with infected pigs or indirectly through feeding of virus contaminated products (swill and garbage), contact with fomites and sometimes by tick vectors, although the latter route of transmission has seldom been demonstrated for domestic pigs [5]. The incubation period of ASF is typically less than 7 days before infected pigs present a range of syndromes varying from per-acute, acute to chronic disease and apparently healthy but sub-clinically infected depending on the virulence of the strain and perhaps also the pig genotype [6]. The virus causes acute haemorrhagic disease with high morbidity and mortality rates in domestic pigs that can be close to 100% in naïve populations but does not cause clinical disease in warthogs or other wild African suids [1].

ASF was confined to eastern and southern Africa in its reservoir hosts (soft ticks and wild suids) until the introduction into Africa of susceptible domestic pigs. The virus was first described in Kenya in the early twentieth century by Montgomery as an acute haemorrhagic fever of domestic pigs with clinical and pathological resemblances to classical swine fever (CSF) but differing

epidemiologically and immunologically. CSF is, as far as is known, absent from sub-Saharan Africa with the exception of the island of Madagascar, from which sporadic incursions into the island of Mauritius have occurred and been eradicated. Two incursions over a period a century apart have been reported from South Africa, both of which were eradicated [2]. The virus was ancestrally maintained through a sylvatic cycle of infection between soft ticks and young warthogs in the warthog burrows. The virus also circulated within the tick population by trans-stadial and trans-ovarial routes of transmission [7]. A domestic (pig-to-pig) cycle has become increasingly important, and is the only mechanism of transmission, particularly in West and Central Africa. There is currently no vaccine and ASF control is mainly by diagnosis, and slaughter to eradicate infected animals, combined with quarantine in areas where the outbreaks have occurred. The disease has spread to previously uninfected countries in Africa in the last two decades. Recent studies have described two novel ASF p72 genotypes from eastern Africa: Genotype XXIII from Ethiopia [8] and genotype XXIV from Mozambique [9].

Pig populations in many sub-Saharan African countries have been increasing with Uganda and Nigeria topping the list with estimated populations over 3.2 million and over 7 million respectively [10–12]. It has recently been established that the rate of increase of demand for pig products is higher than that of other types of meat [13–15]. For most sub-Saharan countries this increase in pork production and consumption can be attributed to growth in smallholder or backyard husbandry systems. However, the full potential of the small-scale pig-keeping sector has been hampered by a number of constraints, including ASF, lack of institutional credit facilities, together with shortage of suitable feeds [16,17]. ASF is widely regarded as the major disease constraint to pig production and enhancement of pork value chains on the African continent [1]. The majority of smallholder farmers who supply pork products do not observe strict biosecurity measures, thus

exacerbating the challenge of disease prevention and control as the sector grows [5]. Increased awareness of biosecurity combined with vigilance is required to protect pig populations, the associated pig value chains and ultimately livelihoods.

The economies of most sub-Saharan African countries are reliant on agriculture, with over 60% of their populations (especially those living in the rural areas) deriving their livelihoods directly from agriculture. The pig production sector has been growing rapidly in the recent past due to increased meat consumption in most sub-Saharan countries [18]. Pigs grow fast, and do not require a large amount of capital investment or space to rear them. Therefore, many smallholder farmers in rural areas have adopted pig production as a viable income generating enterprise. However, ASF inflicts economic and personal losses on farmers, resulting in loss of valuable protein and reduced income. Nonetheless, due to the flexibility of the pig keeping system and ease of sale, farmers have come to regard pigs as easily-liquidated assets. However, the risk of disease, particularly ASF, can prevent investment in the pig sector.

Research efforts on ASF have focused on development of improved diagnostics [1,3] and on the unique biology of virus-host-macrophage interaction [19,20]. Several publications and reviews provide insight to guide formulation and implementation of control strategies in the region [21]. None of the research to date has explored the use of mathematical modelling to generate insight to guide formulation of improved control strategies in smallholder pig-keeping systems, where stamping out is not an economically viable option.

Although ASF has been controlled and even eradicated in some countries outside the African continent [7], the control measures used are logistically impractical and too expensive to implement in the African context. It is therefore important to design control measures that are

appropriate in a regional context, cost effective and acceptable to various stakeholders. To attain this, a better understanding of the epidemiology of ASFV and evaluation of the effectiveness of different control options is required. Mathematical models provide an avenue for generating such knowledge.

Mathematical models have been used to understand and predict the dynamics of infectious diseases. They play a central role in the design of disease control strategies by guiding the identification of critical intervention points [22], and quantifying the magnitude, duration and cost of disease outbreaks [23]. Models have also been used to assess benefits that accrue from given interventions and the probable changes to disease dynamics as a result of these interventions [22,24].

## 1.2 Problem statement

ASF is known to be endemic in many rural communities in Africa affecting household livelihoods [1]. However, there has been virtually no significant effort to understand ASF epidemiology from the modelling perspective, especially in areas where it is endemic in Africa. Mathematical models can be used to better understand the disease, its infection dynamics and to assess the efficacy of different control programs [25–29]. In spite of significant efforts to control ASF in Africa, to date the role of ASF modelling in identifying the potential impact of control strategies has been under-researched. Research has focused on improved diagnostics, molecular epidemiology and experimental studies primarily focused on preventing the incursion of the disease outside of the African continent.

In the current project, we model the transmission dynamics of ASF in the East African context with the aim of better understanding epidemiology and the potential effect of different control

measures. We focus on measures intended to limit the spread of disease and minimize the social-economic impact on smallholder pig farmers. Specifically, this entails the development and use of modelling tools capable of generating insight into ASFV dynamics, persistence in domestic pig population and evaluation of the effect of various control strategies.

## 1.3 Significance of the study

It is important to undertake this study because:

1. Formal mathematical models for ASFV dynamics have value in identifying knowledge gaps in the literature;
2. These models will improve our quantitative understanding of the biology of ASFV;
3. Models will provide a framework for description of the impact of ASF on smallholder pig farmers and their livelihoods;
4. The research outputs can be used to inform and guide policy development for ASF control.

## 1.4 Study Objectives

The overall objective of this study is to model the transmission dynamics of the ASF virus in smallholder pig farmer production systems, involving free ranging pigs in order to enhance our understanding of the epidemiology and inform the design of appropriate control measures.

Specifically we aim at:

1. Analysing key parameters of low input pig production systems
2. Determining the rate of spread of the virus at a village level through estimating the between-village reproduction ratio using field data,

3. Developing compartment models for ASF to provide a framework for description of the dynamics of ASFV,
4. Analysing models representing different states of disease equilibrium.
5. Evaluating the potential effects of different control options,

## 1.5 Structure of the thesis

This thesis is based on articles that have been published (Chapter 3 and Chapter 4) [25,30] and others submitted for publication (Chapter 5 and Chapter 6). These articles are based on research that was conducted while I was registered as a doctoral candidate at the University of Pretoria. The thesis is structured in such a way that we provide a general introduction to ASF, ASFV and the objectives of the study in Chapter 1. Chapter 2 gives a review of ASF literature in relation to the study objectives. We estimate R0 for ASF from field data in Chapter 3, and we develop a stochastic model and use it to simulate the effect of different control options for ASF in Chapter 4. In Chapter 5 we describe the production system practiced in the study area. A spatial-deterministic model is developed to assess effect of time for introduction of control measures on the spatial transmission of ASFV in Chapter 6, and Chapter 7 sums up the thesis with a general discussion and conclusion. There were some modifications to published/submitted papers on the position of figures and tables, and formatting of references.

# CHAPTER 2

## Literature Review

## 2.1 African swine fever and its causative virus

African swine fever (ASF) is a contagious and lethal viral disease of domestic pigs. ASF has one of the highest mortality rates of any pig disease, frequently almost 100 percent of infected pigs in naïve herds [11,31,32]. It is classified as a notifiable disease by the World Organization of Animal Health (OIE), and its presence leads to immediate restrictions to trading in pigs and pig products. The disease can have a major impact on animal health, people's livelihoods, global trade in pigs and pig products, and poses a threat to global food security, through its proven record of 'escape' from the African continent [33,34]. The causative agent, African swine fever virus (ASFV), is a double-stranded DNA virus in the genus *Asfivirus*. It is the sole member of the family Asfarviridae (although recently a related superfamily of viruses has been discovered) [2,35–37]. All ASFV strains are considered to be belong to a single serotype, with 24 genotypes identified to date [9]. The genome varies in length from about 170 to 193 kilo base pairs (kbp) depending on the isolate, and contains hairpin loops and terminal inverted repetitions [32]. ASFV is maintained either in a sylvatic cycle between warthogs and tampans (soft ticks) that live in warthog burrows, or in a domestic cycle that may, or more frequently may not, involve soft ticks [1,13,38]. As in the case of warthogs, other African suids such as bush pigs and giant forest hogs (*Hylochoerus meinertzhageni*) are also susceptible to ASFV infection though resistant to its pathogenic effects [2,11]. ASF disease usually has different clinical presentations and pathological lesions depending on the virus strain, how long the strain has been circulating in the area, route and dose of infection, and the host characteristics [2,34,39]. According to Sanchez-Vizcaino et al., [34,40], ASFV strains have been classified as highly virulent, moderately virulent or low virulence strains that are responsible for peracute, acute or sub-acute to chronic forms of ASF respectively. ASFV is notably stable across a wide range of temperatures and pH. The virus is capable of surviving in

serum at room temperature for 18 months, in blood at 4 $^{0}$C for six years, and at temperatures as high as 55 $^{0}$C for 20 minutes [2].

## 2.2 Distribution of ASF

### 2.2.1 Global impact of ASF

ASF was first reported outside of the African continent in Portugal in 1957 and again in 1960, probably from Angola through airline meals that were discarded and consumed by local pigs [1]. Subsequently, ASFV was introduced to other parts of Europe, the Caribbean and Latin America but was ultimately eradicated in these areas through test and slaughter by 1995, save for Sardinia where it has remained endemic [1,32]. As already mentioned, outbreaks were reported in Portugal in 1957 and 1960 before the virus established itself in the Iberian Peninsula. From there the virus spread to other European countries including France (1964, 1967, 1977), Italy (1967, 1980), Malta (1978), Spain (1960-95), Belgium (1985) and the Netherlands (1986) [1,2,41]. Recently, following eradication in the Iberian Peninsula, a different virus genotype (p72 genotype II) was introduced into the Caucasus region. This incursion into the Caucasus occurred in 2007 when ASF was confirmed in the Republic of Georgia, before spreading to Armenia and Azerbaijan. ASF was later confirmed in Chechnya (in dead wild boars) and a number of regions in the southern Russian Federation [42,43]. By 2014, ASFV had reached Belarus and Ukraine within the eastern sector of the European Union [31,32]. It spread further west into Poland and Lithuania, mainly through the movement of wild boars, and threatens the pig industry throughout Europe [32]. Prevention of further geographic expansion of ASF will require enhanced surveillance and biosecurity measures especially in small-scale private holdings in the endemic zones in order to protect pig farmers and their associated businesses and livelihoods in other parts of Europe [5,29,43].

### 2.2.2 The African impact of ASF

In Africa, ASF was first reported in Kenya in 1921 [44]. Several studies demonstrated that a virus existed that caused a disease that differed epidemiologically and immunologically from classical swine fever, which was prevalent in Europe and Asia at the time ASF was first reported in Kenya [2]. These studies reported that outbreaks were related to contacts between free-ranging domestic pigs and wild suids. Following its recognition in Kenya, ASF outbreaks were reported in sub-Saharan African countries [2]. The disease has since been described from most countries in eastern, western, central and southern Africa. By 1954, it had been diagnosed in domestic pigs in Angola and Mozambique [45]. It was reported in Guinea Bissau and Senegal by 1959 [38]. ASF has also been reported in the Democratic Republic of Congo, Republic of the Congo, Central African Republic and Cameroon. It made its first entrance in Chad in 2010 [46–48] while in Nigeria, it was first reported in Lagos state in 1997 and has continued to seriously impact the pig industry causing heavy socio-economic losses to pig farmers [49–51].

In the east African region, ASFV has remained endemic since its first detection in Kenya. In 1994, ASF outbreaks were reported in commercial farms in Kenya, after an apparent absence since 1963. These outbreaks were attributed to movements of domestic pigs from areas of high endemicity [2]. Recently two ASF outbreaks occured in western Kenya between October 2006 and February 2007 and between December 2010 and March 2012, resulting in the death of 82 pigs and 163 pigs respectively [52]. In Uganda, the virus is maintained within the pig populations across the country [53]. It was estimated that at least 40 outbreaks occurred in the district of Gulu alone in a period of 18 months (October 2010 – March 2012) [30,54]. Furthermore, surveillance data from the Ministry of Agriculture, Animal Industry and Fisheries (MAAIF) annual reports indicate that Uganda had up to 143 ASF outbreaks between 2006 and 2014.

## 2.3 ASF Maintenance Cycles and Transmission routes

ASFV is known to have three maintenance cycles: (1) a sylvatic cycle occurring in east and southern Africa involving soft ticks of the genus *Ornithodoros* and warthogs, (2) a cycle between domestic pigs and soft ticks, and (3) a domestic cycle involving naïve pigs and infected pigs [53,55]. In the sylvatic cycle, warthogs are natural reservoir hosts of ASFV, although they show no sign of disease. Their role in the epidemiology of ASF is well described [56,57]. Soft ticks that inhabit warthog burrows feed on neonatal warthogs infecting them in the process and they develop sufficiently high viraemia to infect new ticks [41]. Within the soft tick population, the virus can be transmitted from one tick to another by trans-stadial, trans-ovarial or sexual passage. Soft ticks have a life span of 15-20 years and are reported to live for at least five years without feeding and are known to retain and transmit the virus to susceptible hosts for periods of at least two years [1,58–60]. The maintenance of the virus in the sylvatic cycle is therefore dependent on the interaction between warthogs and soft ticks and transmission does not occur vertically or horizontally in warthog populations[1,61–63]. This cycle is limited to eastern and southern African countries where argasid ticks of the genus *Ornithodoros* are distributed.

The pig-tick cycle has been documented in studies done in Malawi, Madagascar and Mozambique [64–68] but may be more widespread as evidenced by the high level of pig exposure to tick bites in the study region [69]. This could explain the sporadic nature of ASF outbreaks in the study region.

The third and probably the most common cycle (domestic cycle) involves only domestic pigs without either wild suids or soft ticks. In this cycle, the virus is maintained among domestic pigs or from pig products to domestic pigs establishing a cycle of viral circulation. The development

of a higher degree of resistance to viruses of similar virulence by pigs in the study area seem to serve as a source of infection for naive pigs [52,70].

Since the introduction of ASF in the European Union in 2014, a potential fourth maintenance cycle in wild boars in EU member states has been suggested. A recent study has described an epidemiological pattern that suggests a cycle that focuses on the wild boars and their habitat as a virus reservoir [71]. It is proposed that this cycle be named the 'wild boar–habitat' cycle.

Transmission can occur by direct contact between domestic pigs for up to 30 days after infection or indirectly by contact with infected pig products and fomites for a considerably long period [1]. The relative stability of the virus under varying environmental conditions presents a potential risk of introduction to places previously free of ASF. In 2011, an ASF outbreak was reported in the coastal town of Mombasa, Kenya and virus genotyping confirmed that the virus had spread from the Kenya-Uganda border (unpublished data; Gallardo, Okoth and Bishop). According to [1,72–74], the spread and maintenance of ASFV in the domestic pigs is governed by several factors including; (1) level of adoption of biosecurity practices, (2) production systems employed, (3) frequency of farmer contact with infected farms, other farmers, pig traders or veterinary officials, (4) use of swill and/or food left overs, (5) introduction of new asymptomatic animals on the farm, and (6) presence of an abattoir/ slaughter slabs in the community. Other practices that increase risk of transmission of ASF include (a) pig agistment, which involves sharing of animals between close neighbours, (b) mass pig sales during suspected ASF outbreaks, (c) delayed diagnostic confirmation of outbreaks, and (d) non-reporting of suspected outbreaks to authorities [73].

Recent studies on ASF in the study area such as the "People, Animals & their Zoonoses" (PAZ) project in western Kenya aimed at investigating the presence of ASFV viral DNA in domestic pigs

presented to slaughter during a time period with no officially reported outbreaks of ASF found an apparent increased prevalence of the virus at slaughter slabs, relative to the population at large suggesting that sub-clinical, chronically infected or recovered pigs may be responsible for persistence of the virus in endemic areas [52]. Similarly, the recurring nature of small-scale ASF outbreaks in domestic pigs was investigated by testing for the virus in tissues in a proportion of animals. The study revealed that healthy pigs carrying ASFV exist in the swine population and it is hypothesized that these carrier pigs may play a role in sporadic disease outbreaks, although their trigger remains unclear [70].

## 2.4 ASF prevention and control strategies

There is currently no vaccine or chemotherapeutic available for ASF. Control therefore depends on preventing contact between pigs and the virus by adopting best practice pig husbandry [52]. It has been suggested that

> *"eradication of ASFV from its natural hosts and vectors in Africa is not an option, eradication of ASFV in domestic pigs may theoretically be achievable, provided that pigs are managed in a way that excludes contact with the sylvatic sources of infection and prevents maintenance of the virus in domestic pig populations"* [21].

In most endemic areas in Africa, ASF spread is often (although not exclusively) associated with free-ranging pig husbandry, unrestricted pig movements especially when ASF is suspected, and lack of - or improper implementation of - basic biosecurity measures. Pig traders often move between villages, slaughter slabs and live animal markets collecting and delivering pigs without use of protective clothing or disinfectants [73,75,76]. The nature of the pig trade suggests that traders may contribute to a rapid dissemination of the virus between villages [76]. Well-established

pig traders on the other hand have self-serving interests in eliminating the disruption and financial losses caused by market closures or quarantine imposed by veterinary authorities due to outbreaks. This highlights the possibility of future cooperation between authorities and pig traders in developing and implementing effective approaches to reduce the risk of ASF outbreaks [76].

Due to the lack of established pig breeders in the majority of rural areas, pig farmers tend to buy piglets or weaners for restocking from neighbours, relatives, other famers or from live animal markets. These new pigs may be exposed to the virus at source or while in transit. Worse still, it is believed that some pig farmers sell off their pigs if they suspect them to have been exposed [73]. This was evidenced in a study on the presence of African swine fever virus in an endemic region of western Kenya where viral DNA was detected in a relatively high number of pigs delivered for slaughter in the absence of reported outbreaks [52]. Farmers who purchase pigs for restocking from live animal markets or pig traders stand a high risk of introducing the disease to their household. As a preventive measure, new pigs must be isolated for a period of at least two weeks before they are integrated in the herd [77,78]. For farmers who feed their pigs on swill or food leftovers, it is recommended that swill is boiled for 30 minutes to ensure that it is free of viral contaminants. Additionally, there should be a physical barrier to restrict people from having direct contact with the pigs unless they use protective clothing and walk through a foot bath after all solid material has been removed from the footware [73,79]. This restriction and use of disinfectants also applies to veterinary authorities when they visit a farm, pig traders as well as family members and other visitors. Pig pens should be cleaned frequently. There is a need to sensitise pig farmers on the proper use of biosecurity practices to prevent or minimise the risk of ASF introduction and spread on and from their farms [80].

Several ASF control strategies have been proposed and implemented with varying degrees of success in different parts of the world. Early disease reporting is the first step towards ASF control. To enhance effective reporting, farmers or pig traders should be trained to recognize ASF based on high mortality in pigs of all ages and the typical clinical signs and lesions [2,81]. The higher prevalence in slaughter slabs might indicate that many farmers do require this kind of training. Stakeholders in the early reporting phase are usually not incentivised to report because of the consequences they face when veterinary authorities act by imposing quarantine and closing markets. Although it is very important to put in place mechanisms for rapid laboratory confirmation of suspected outbreaks, rapid quarantine responses and pig market closures at community level, these measures have frequently resulted in cases where market closures have lasted for more than a year severely impacting the livelihood of the stakeholders along the pig value chain [73]. The effect of these measures at the household level (including those households that are not directly affected by the outbreak) serves as a disincentive to report the disease next time it is suspected.

When ASF is suspected, rapid laboratory diagnosis is required to confirm the outbreak. This is because ASF should be differentiated from other swine diseases with similar clinical signs such as classical swine fever (CSF), porcine dermatitis and nephropathy syndrome (PDNS), porcine reproductive and respiratory syndrome (PRRS) and erysipelas as well as bacterial septicaemia. Laboratory confirmation requires rapid, reliable, sensitive and specific detection methods [32]. Currently available diagnostic methods in the literature are based on virological, molecular and serological techniques to detect ASFV [81,82]. Recently, effort has been put in developing pen side kits to provide tools for faster and more inexpensive ASF diagnosis on the farm, providing results within hours [83]. However, such assays have yet to be validated in endemic areas, which

are characterised by regular sporadic outbreaks and animals that carry virus in tissues other than blood.

Once an ASF outbreak is confirmed, the first step is theoretically to notify the OIE, although it is unclear what proportion of outbreaks are actually reported in practice, then assess OIE recommended control measures for suitability and feasibility of implementation in the affected area. Some of the measures include proper disposal of carcasses or litter, thorough cleaning and disinfection of pig pens and houses [77,84]. Other measures to be performed routinely as a form of prevention even in the absence of an outbreak are pig confinement and efficient sterilisation of garbage and/ or food leftovers by boiling for 30 minutes before feeding to pigs. Areas with confirmed ASF cases should be designated, with control of pig movements, restriction of farm visits by stakeholders in the production chain, and closure of live pig markets, slaughter slabs and butchers. Farmers should be encouraged to confine their pigs, apply basic biosecurity measures and authorities must outlaw free movement of pigs at least for the period while the outbreak continues [1,85]. However, veterinary authorities lack resources to ensure compliance with these regulations, and such measures require changes in production systems and marketing habits. Furthermore pig farmers are more likely to comply if they perceive some benefits from these control measures and are involved in their development, which is currently not typically the case [1].

Many developed countries that suffered ASF incursions from 1958-1995, were able to eradicate it by ‘stamping out’ of all infected and in-contact pigs, with proper disposal of the carcasses. The culling was extended to include all of the pigs in a defined area, whether or not they were infected or in-contact [1]. In developing countries such swine depopulation programmes cannot be sustained due to inadequate resources in terms of funds to compensate affected stakeholders, and

veterinary staff and infrastructure to apply the measures [73,75]. The veterinary services should therefore develop more effective surveillance systems for improved reporting, early detection and rapid response to all suspected ASF outbreaks before spread has occurred.

The use of mathematical models as tools for guiding the design and testing of control strategies for infectious diseases is well established [26,27,86–89]. Mathematical modelling is recognised as an important piece in the scientific toolbox for its ability to describe and structure biological phenomena, give insight into complex processes and predict future outcomes [28]. Models are useful in estimating important disease parameters and in assessing the effectiveness of control options [25,30]. They can be used to generate information for estimating the required effort and resources for implementing control strategies as well as assessing their efficacy. Modelling has played a pivotal role in providing policy support and enhanced decision making for infectious disease control [87,89]. Models have been applied to study livestock diseases and were insightful in aiding understanding of disease transmission and factors driving epidemic behaviour [90–92]. They are helpful in explaining and communicating fundamental principles of disease epidemiology. However, there have been few attempts in modelling transmission dynamics of ASFV for purposes of enhancing our understanding of its epidemiology and informing the design of appropriate control strategies in endemic situations in Africa. This thesis is an attempt to address this knowledge gap.

# CHAPTER 3

## Estimating the basic reproductive number ($R_0$) for African swine fever virus (ASFV) transmission between pig herds in Uganda

Mike B. Barongo[a,g], Karl Ståhl[d], Bernard Bett[a], Richard P. Bishop[a], Eric M. Fèvre[a,b], Tony Aliro[c], Edward Okoth[a], Charles Masembe[e], Darryn Knobel[f h], Amos Ssematimba[a]

*[a]International Livestock Research Institute, P.O. Box 30709-00100, Nairobi, Kenya*
*[b]Institute of Infection and Global Health, University of Liverpool, Leahurst Campus, Neston, CH64 7TE, UK*
*[c]Ministry of Agriculture, Animal Industry and Fisheries, Entebbe, Uganda*
*[d]Department of Disease Control and Epidemiology, National Veterinary Institute, Uppsala, Sweden*
*[e]Department of Biological Sciences, College of Natural and applied Sciences, Makerere University, Uganda*
*[f]Department of Veterinary Tropical Diseases, Faculty of Veterinary Science, University of Pretoria, South Africa*
*[g]Department of Academic Registrar (ICT Division), Makerere University, Uganda*
*[h]Center for Conservation Medicine and Ecosystem Health, Ross University School of Veterinary Medicine, Basseterre, St Kitts*

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## 3.1 Abstract

African swine fever (ASF) is a highly contagious, lethal and economically devastating haemorrhagic disease of domestic pigs. Insights into the dynamics and scale of virus transmission can be obtained from estimates of the basic reproduction number ($R_0$). We estimate $R_0$ for ASF virus in small holder, free-range pig production system in Gulu, Uganda. The estimation was based on data collected from outbreaks that affected 43 villages (out of the 289 villages with an overall pig population of 26,570) between April 2010 and November 2011. A total of 211 outbreaks met the criteria for inclusion in the study. Two methods were used, specifically; (i) Epidemic doubling time and (ii) a compartmental susceptible-infectious (SI) model. For implementation of the SI model, three approaches were used namely; curve fitting (CF), a linear regression model (LRM) and the *SI/N* proportion. The $R_0$ estimate from epidemic doubling time method was 1.63. Estimates from the SI-based method were 1.58 for the CF approach, 1.90 for the LRM, and 1.77 for the *SI/N* proportion. Since all these values were above one, they predict the observed persistence of the virus in the population. We hypothesize that the observed variation in the estimates is a consequence of the data used. Higher resolution and temporally better defined data would likely reduce this variation. This is the first estimate of $R_0$ for ASFV in a free range smallholder pig keeping system in sub-Saharan Africa and highlights the requirement for more efficient application of available disease control measures.

**Key words:** African swine fever; basic reproductive number; mathematical modelling; Uganda.

## 3.2 Introduction

African Swine Fever (ASF) is a highly contagious, lethal and economically devastating haemorrhagic fever of domestic pigs. The disease is of high economic importance both globally and in sub-Saharan Africa where demand for animal protein including pork has greatly increased in the last two decades [15,93].

The disease is caused by African Swine Fever virus (ASFV), a large double-stranded DNA-virus and sole member of the family *Asfarviridae* [94]. ASFV isolates vary in their virulence, from highly virulent isolates that kill up to 100% of the pigs to moderately or low virulence viruses with mortalities ranging between 30-70% [34,95]. ASF produces clinical signs that range from peracute, acute, sub-acute and chronic forms depending on the virulence of the strain, intensity of exposure and pig breed [2,4]. The disease is characterised by high fever, loss of appetite, haemorrhages in the skin and internal organs, and death. Pigs that apparently recover from the disease become virus carriers [34].

ASF has spread and is now established in many sub-Saharan countries since its discovery in Kenya in 1921 [44]. Initially it was reported from countries in East and Southern Africa but has now spread through Central and West Africa, and Indian Ocean islands with Chad becoming the most recent country to be affected [21]. The disease first spread outside the African continent to Portugal in 1957. From 1968-1995, ASFV in the p72 genotype I was present in European countries including Malta, Sardinia, Italy, France, Belgium and the Netherlands. The prevalent genotype in Gulu district is genotype IX. It was eradicated in all these countries except Sardinia where it remains endemic and poses a continuous risk of re-introduction and spread in Europe [1,39]. ASF was accidentally introduced into the Caucasus in 2007 from where it spread rapidly and widely

within the Russian Federation. Outbreaks were reported more recently in Poland, Lithuania, Latvia and Estonia [96,97].

This virus is stable at a wide range of temperatures and pH and is capable of remaining infective in faeces, tissue and environment for many days [1]. The incubation period in domestic pigs varies from 5 to 15 days depending on the virus genotype [34]. ASFV is maintained in two main cycles: a sylvatic cycle that involves natural hosts, namely warthogs and soft ticks (*Ornithodoros moubata*) and a domestic cycle that may not involve the soft ticks [41]. In the domestic cycle, ASFV can be transmitted by direct contact with infected animals, indirect contact through fomites, and tick vectors. Transmission in the domestic cycle is exacerbated by sociocultural factors such as pig movement networks (traders, butchers, boar service), superstition and beliefs (e.g. that a carcass cannot be buried), use of untreated swill, lack of confinement of pigs and low biosecurity adoption [73,98].

There is currently no available vaccine against ASF and the available control strategies focus on preventing and controlling the spread of the virus although in better-resourced parts of the world, "stamping out" of pigs within infected farms and surrounding areas is used [1,99].

This study was based on data from confirmed outbreaks that occurred in Gulu district, northern Uganda, in the period 2010-2011. The main economic activity in the district is subsistence agriculture which engages up to 90% of the population, with 9% of the households involved in pig farming. The pig production systems practiced in the study area are predominantly traditional free ranging and tethering, supplemented by very limited semi-intensive and intensive farming with virtually no biosecurity measures in regular use. In addition to the roaming of free ranging pigs, movements in the area occur for purposes of restocking, breeding, and trading [15,73]. Moving of

apparently asymptomatic pigs to neighbouring villages when an outbreak is suspected is an additional factor that may promote virus transmission.

Estimates of the basic reproductive number ($R_0$) are fundamental in underpinning rational control strategies based on disease modelling. $R_0$ is the average number of secondary cases arising from a single infectious individual in a wholly susceptible population throughout its infectious period [100–105]. This parameter can be estimated using a variety of mathematical techniques [99,102,106]. This estimate provides a means to better understand the dynamics of infectious disease outbreaks and to assess the potential efficacy of disease control measures [107]. It is frequently used as a threshold parameter to quantify the spread of disease and is therefore a quantitative indicator of both the risk of an epidemic and the effort required to control it in a particular population. In order to control an infectious disease, it is necessary to reduce $R_0$ to below unity (refer to appendix I) [107,108]. This parameter can predict the speed and scale of disease spread and the level of herd immunity required to contain the disease [99].

In many resources-constrained small holder communities, such as in East Africa, information on transmissibility of diseases like ASF is often lacking and usually limited to daily counts of new cases [109,110]. Additionally, decisions about the best control strategies to implement during an epidemic are complex, usually involving technical, political, sociocultural and economic issues. ASF outbreaks are not reported quickly enough to allow collection of all the required empirical data for the estimation of disease parameters. To overcome this constraint, indirect methods can be used to estimate these parameters, including $R_0$. There are a number of approaches available for estimating $R_0$. Some methods are purely analytical, and not very reliable [111], while others are mathematical expressions involving multiple population parameters that have to be estimated separately [107,112] using outbreak data [86,102,106]. Ward et al. [102], Bett et al. [106] and Li

et al. [111] describe different methods for estimating $R_0$ from outbreak data for a number of diseases across different geographical regions. In this study, we estimate $R_0$ for ASF transmission between herds of pigs based on data from confirmed outbreaks using some of these methods as described next.

## 3.3 Materials and methods

This is to certify that any sampling of live or dead pigs described within this chapter on Estimating the basic reproductive number ($R_0$) for African swine fever virus (ASFV) transmission between pig herds in Uganda by Barongo et al, was conducted in close collaboration between the District veterinary office in Gulu District and scientists from Uganda, Kenya and Sweden as part of disease investigations for African swine fever, and funded through a collaborative research project. Disease surveillance and disease investigations lie within the mandate of the District veterinary office. The district veterinarian thus holds a general permission to sample animals for this purpose. Data from these disease investigations were reported to the National Animal Disease Diagnostic and Epidemiology Centre, NADDEC, under the ministry of Agriculture Animal Industry and Fisheries in Entebbe. The data also formed part of the basis for international reporting to the OIE.

### 3.4 Data source

We used data collected during previous research activities from villages in Gulu District with laboratory confirmed outbreaks of ASF (material described in [113,114]). The two hundred eleven outbreaks included in this study occurred between April 2010 and November 2011. In brief, villages that reported outbreaks of disease characterized by fever and mortality in pigs to the district veterinary authorities were visited. Within each village, samples (blood and serum) were collected from clinically diseased and/or apparently healthy pigs from all affected households.

Samples were kept cool awaiting transportation to the Molecular Biology Laboratory at Makerere University Institute of Environment and Natural Resources (MUIENR) in Kampala for storage at -20°C until further processing. In the laboratory, outbreaks were confirmed by detection of ASFV nucleic acids using a commercially available real-time PCR (Tetracore Inc., Rockville, Maryland) in accordance with the instructions of the manufacturer [115]. During a second visit to all villages with laboratory confirmed outbreaks, additional data was collected using semi-structured questionnaires from a total of 211 households. The data collected included farm location (GPS coordinates), start month of the confirmed outbreak, number of pigs that had died, number that were still alive, disposal mechanism of carcasses, feed source and production system practiced.

A herd, here defined as a collection of all pigs in a pig-keeping household, was taken to be the epidemiological unit of interest [102]. Thus, our estimates of $R_0$ reflect spread between herds. All outbreaks in the district during the period of study are assumed to have been reported. Additionally, it was assumed that all herds in the district were susceptible during the study period and the pigs from different herds were homogeneously mixing [15]. The exact number of herds present during the period of the study could not be directly determined and we estimated from the National Livestock Census Report (2008) on distribution of livestock in Uganda that there were 6,200 pig herds (mean herd size of 4.3) distributed over the 289 villages in Gulu district.

## 3.5 Data Analysis

Two methods, adapted from previous studies, were used in the estimation of $R_0$. These methods are epidemic doubling time and compartmental susceptible-infectious (SI) method [102].

### 3.5.1 Epidemic doubling time method

During the initial phase of an epidemic, the number of secondary cases increases exponentially, with each infection producing $R_0$ new infections per generation assuming a constant doubling time (td) [102]. Anderson and May [103] defined a relationship between doubling time (td) and $R_0$ as $R_0 = 1 + (\mathrm{T}/\mathrm{td}) * \log_e 2$ where T is the herd infectious period. For the outbreaks studied here, the herd from which the first case of death was reported was considered the index case. Each herd that was subsequently infected was considered to present a new outbreak. Outbreaks were ordered by month and the average time for the number of outbreaks to double (td) for all possible combinations during this phase were computed using Microsoft Excel® 2010. We assumed an infectious period of one month because data was aggregated at a monthly scale and as mentioned there is evidence from the literature that herds can remain infectious for a prolonged period [116]. We then used the doubling time and infectious period to estimate $R_0$ from the above equation.

### 3.5.2 SI modelling method

This method has been described and used in a number of studies [86,102,106,107]. We describe three approaches for estimating $R_0$ using a simple deterministic SI model of the epidemic process. First we estimated the transmission rate, $\beta$, from epidemic data using a linear regression model (LRM) following an approach as described by Eblé et al. [117] and Gulenkin et al. [99]. The regression model was defined as $log\ (-log\ (1-E(C)/S)) = log\ (\beta) + log\ (I\Delta t/N)$, where $C, S, I$ are respectively the number of newly-infected herds, susceptible herds and infectious herds

at the start of the time interval Δt. We used Microsoft Excel® 2010 to run the regression model. These estimates were bootstrapped and their mean taken as an estimate of $\beta$. $R_0$ was then estimated from the product of $\beta$ and T where T is the infectious period of the herd.

Secondly, a curve fitting (CF) approach was used to fit a Susceptible-Infectious-Removed (SIR) model to the epidemic data in order to estimate $\beta$ and the removal rate $\gamma$. This approach was used as described by Gulenkin et al. [99]. Curve fitting was implemented using a modelling software package *Berkeley Madonna* ver. 8.3.18. These two parameters were then used to compute an estimate for $R_0$ from $(S_0 * \beta) / \gamma$, where $S_0$ is the size of the susceptible population.

Lastly, we estimated $\beta$ using an approach that describes disease transmission between epidemiological units in a Susceptible-Infectious (SI) model [102,106]. Here we assumed that all newly infected herds *(C)* were infected via contact with infectious herds *(I)* during the period of interest. Repeated infections reported from the same herd were considered to represent distinct outbreaks if they occurred in a period of more than two months of each other. Stegeman et al. [86] and Bett et al. [106] have shown that the number of new cases/outbreaks $C$ is given by $\beta SI/N$ from which $\beta$ can be estimated given $N$ as the total number of herds. The basic reproductive number $R_0$ is calculated as the product $\beta$T, where T is the infectious period. Microsoft Excel® 2010 was used to estimate the monthly $\beta$ (Table 2), which was then analysed using bootstrapping techniques [106].

Sensitivity analysis was performed to assess whether the initial number of susceptible herds ($N$) had an effect on the estimate of $R_0$, assuming $N$ lies between (3 100 – 12 400) [118]. Due to the poor temporal resolution of the data arising from the reporting timescale, it was not possible to perform a sensitivity analysis of $R_0$ to variation in the infectious period.

## 3.6 Results

During the study period, ASF resulted in a total of 1141 deaths in 211 herds in 43 villages in Gulu district. We present the distribution of infected herds per month in Fig.3.1. Table 3.1 summarises all the parameters obtained using each of the method.

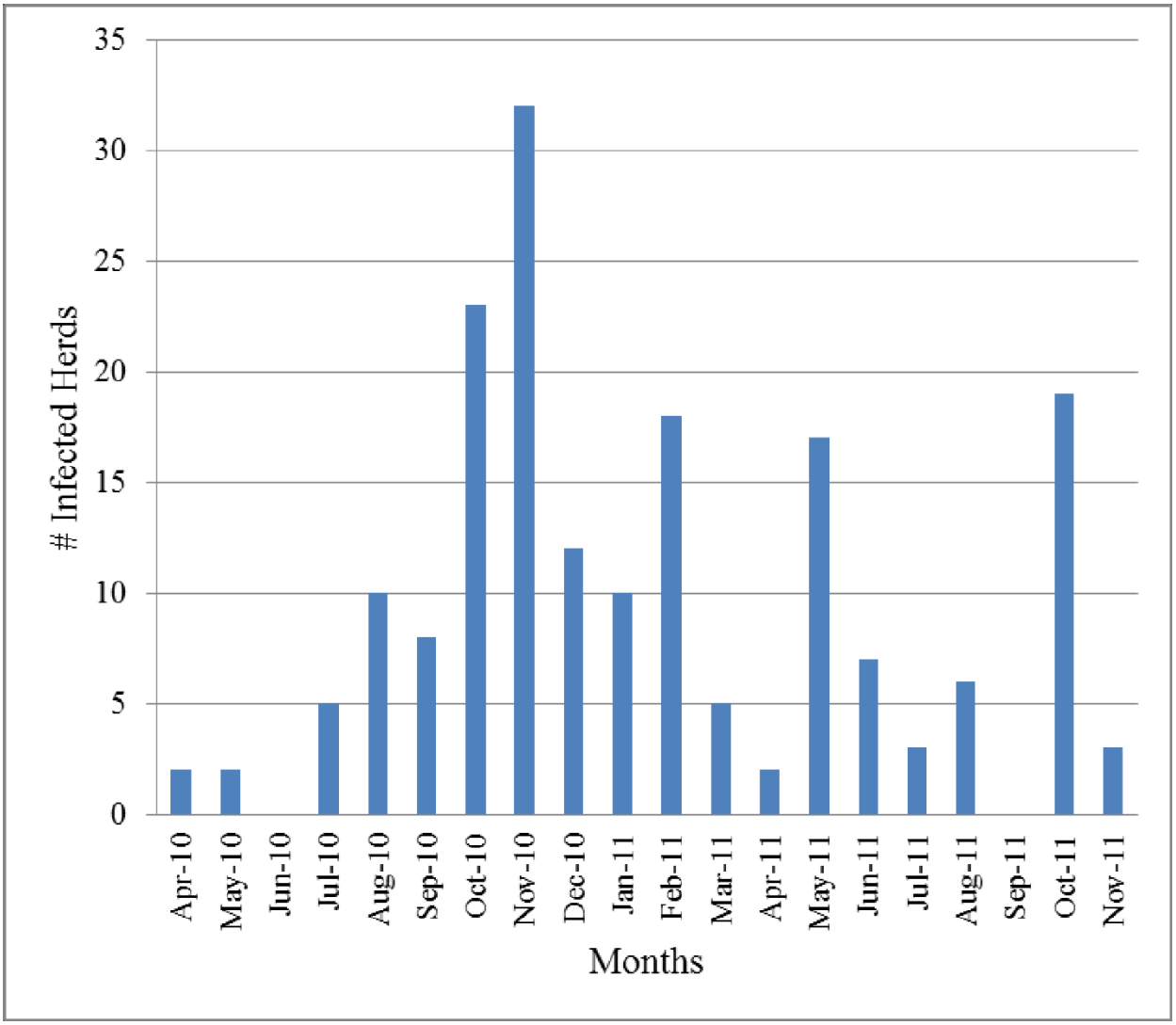


Fig. 3.1. Number of African swine fever infected herds per month in Gulu District, Uganda, April 2010 – November 2011.

Table 3.1. Summary of the parameters estimated using the different methods

| Method | | Parameter estimates | | | | Confidence Interval | |
|---|---|---|---|---|---|---|---|
| | | β | td | γ | $R_0$ | LB | UB |
| Epidemic doubling time | | - | 1.106 | - | 1.63 | 1.56 | 1.72 |
| SI model | LRM | 1.90 | - | - | 1.90 | 1.87 | 1.94 |
| | CF | 0.0059 | - | 0.8236 | 1.58 | - | - |
| | SI/N | 1.77 | - | - | 1.77 | 1.74 | 1.81 |

### 3.6.1 Estimate of $R_0$ from the epidemic doubling time method

During the initial period of study (April - November 2010), the number of outbreaks increased exponentially as depicted in Fig 3.2. The computed average doubling time (td) during the initial phase was 1.106 (95%CI: 0.97-1.25) months. Using this doubling time, we estimated $R_0$ to be 1.63 (95%CI: 1.56-1.72).

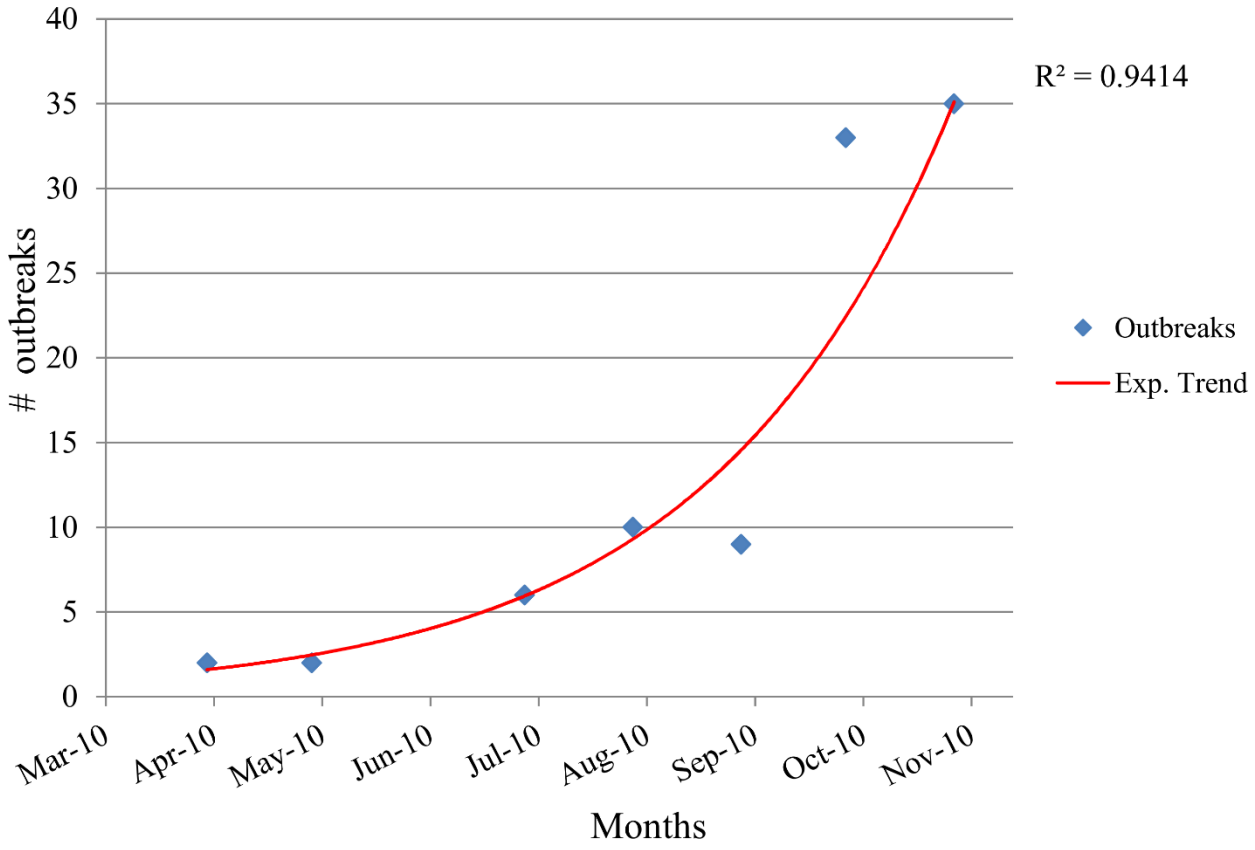


Fig. 3.2. Exponential curve fitted to the data from the first phase (April – November 2010) of the African swine fever outbreaks in Gulu District, Uganda.

### 3.6.2 Estimate of $R_0$ from the SI modelling method

*Linear regression approach*

Using the linear regression model approach the estimate for $\beta$ was 1.90 (95% CI: 1.87- 1.94) herds per infected herd per month resulting in an $R_0$ of 1.90 (95%CI: 1.87-1.94) since the infectious period is one month.

*Curve fitting approach*

Using the curve fitting approach an SI model was fitted to the epidemic data and the results are as shown in Fig S2 in appendix I. Here $\beta$ was estimated to be 0.0059 herds per infected herd per

month while $\gamma$ was 0.8236 per herd per month. We were not able to compute the confidence interval using this approach. Nonetheless these two parameters were used to estimate $R_0 = 1.58$.

*SI/N proportion approach*

In Table 3.2, we show how the proportion SI/N, the number of newly infected households *(C)*, the number of infected households *(I)* and the transmission rate ($\beta$) varied during the period of study. The monthly $\beta_i$ estimates were bootstrapped (Fig S3 in appendix I) giving an overall $\beta$ of 1.77 herds per infected herd per month and using this $\beta$ value; $R_0$ was estimated to be 1.77 (95%CI: 1.74-1.81). Monthly $R_0$ estimates were found to be robust with regard to variation in the initial number of susceptible herds (Fig S4 in appendix I).

Table 3.2. Estimated monthly SI/N, β and $R_0$ during African swine fever outbreaks

| Month | # Herds | *C* | *I* | *SI/N* | *β(CN/SI)* | $R_0$ |
|---|---|---|---|---|---|---|
| Apr-10 | 6198 | 2 | 0 | 0 | - | - |
| May-10 | 6196 | 2 | 2 | 2 | 1.00 | 1.00 |
| Jul-10 | 6190 | 6 | 2 | 2 | 3.00 | 3.00 |
| Aug-10 | 6180 | 10 | 6 | 6 | 1.67 | 1.67 |
| Sep-10 | 6171 | 9 | 10 | 10 | 0.90 | 0.90 |
| Oct-10 | 6138 | 33 | 9 | 9 | 3.70 | 3.70 |
| Nov-10 | 6103 | 35 | 33 | 32 | 1.08 | 1.08 |
| Dec-10 | 6091 | 12 | 35 | 34 | 0.35 | 0.35 |
| Jan-11 | 6079 | 12 | 12 | 12 | 1.02 | 1.02 |
| Feb-11 | 6061 | 18 | 12 | 12 | 1.53 | 1.53 |
| Mar-11 | 6056 | 5 | 18 | 18 | 0.3 | 0.28 |
| Apr-11 | 6054 | 2 | 5 | 5 | 0.41 | 0.41 |
| May-11 | 6034 | 20 | 2 | 2 | 10.28 | 10.28 |
| Jun-11 | 6027 | 7 | 20 | 19 | 0.36 | 0.36 |
| Jul-11 | 6024 | 3 | 7 | 7 | 0.44 | 0.44 |
| Aug-11 | 6016 | 8 | 3 | 3 | 2.75 | 2.75 |
| Oct-11 | 5992 | 24 | 8 | 8 | 3.10 | 3.10 |
| Nov-11 | 5989 | 3 | 24 | 23 | 0.13 | 0.13 |

## 3.7 Discussion

In this study, two methods were used to estimate $R_0$ from ASF epidemic data in a predominantly free-ranging pig production system in northern Uganda. The mean estimates for $R_0$ ranged between 1.58 and 1.90. Considering the estimates from the methods used, doubling time method yielded the highest estimate of $R_0$.

The assumed period of infectiousness of herds is plausible given the existing factors that may favour prolonged infectiousness specifically pig agistment, increased sales and home slaughtering of sick animals [73]. Increased survival of some of the shedding animals may also favour a prolonged infectious period, as does the likely survival of the pathogen, which is a highly stable DNA virus, in the environment outside its host [39,116].

Under-reporting of outbreaks has been reported to influence transmission parameters estimates specifically leading to underestimation of $R_0$, yet in most epidemics, a significant fraction of outbreaks may go unreported [102]. However, for the purposes of this analysis, we assumed that all outbreaks during the study period were reported. This assumption is supported by the fact that, in the study area, farmers were primed to report outbreaks due to the ongoing research activities. There were frequent information dissemination exercises by the research team which we expect to have minimized the rate of reporting failures. In the event that some outbreaks were unreported, then our analyses may have underestimated $R_0$.

Since $R_0$ is known to be both population- and pathogen- specific [119] due to its sensitivity to production system, contact structure and environmental factors, it is interesting that our $R_0$ estimates from doubling time method are in close agreement with those of Gulenkin et al. [99] and Iglesias et al. [120] who estimated $R_0$ to range from 2 to 3 and 1.58 respectively at the between-

farm level. This could be just a matter of coincidence since, for example, estimation approaches that ignore the latent period of an infection tend to underestimate its $R_0$ [121–123]. Therefore comparison of estimates from different studies and geographical areas should be made with caution. The true value of $R_0$ for most epidemics may be difficult to quantify for a number of reasons. The source of each outbreak is usually unknown, reporting time-scales are frequently inconsistent and obtaining good contact tracing data is further complicated by the existence of multiple indirect routes of infection, farming systems and the role of human behaviour in transmission of ASFV and other pathogens [110]. Human behavioural factors such as poor handling and processing of pork and pork products at slaughter slabs, butchers and pork joints (i.e., makeshift kiosks where pork is roasted and eaten), farmers' attitudes and cultural beliefs regarding handling of sick and dead animals, and use of swill are known risk factors for ASF transmission that may have influenced our estimates [73].

Gulenkin et al. [99] have identified road network density and pig density as significant risk factors for disease spread. The spatial distribution of ASF infected herds (April 2010 - November 2011) shown in Figure S5 in appendix I confirms that road network density and pig population density are key risk factors that may have also influenced our estimates. Their effect on our estimates needs to be investigated further and quantified. Despite these uncertainties, empirical data from epidemics can be a valuable source for estimating epidemiological parameters.

De Carvalho Ferreira et al. [39] assert that controlling an ASF outbreak is highly dependent on measures implemented by veterinary authorities, such as 'stamping out' (slaughter) of infected herds and quarantining affected areas. However, such measures are only feasible in countries which have economic means to compensate farmers. In resource constrained countries such as

Uganda, the only feasible measures focus on preventive mitigation, including enhanced biosecurity, and early detection and response. Estimates of $R_0$ can inform the efficient application of these measures.

Generally, few if any attempts have been made to estimate $R_0$ from field data in the endemic regions of Africa. Here we have estimated $R_0$ for ASF in a predominantly free-ranging production system, a system that is common in many parts of East Africa. All the mean estimates were above one which is consistent with the observed persistence of disease in the population. Though $R_0 > 1$, ASF endemicity can be due other factors than $R_0 = 1$. Maintenance cycles like sylvatic and tick-pig can cause ASF to be endemic even when $R_0 <> 1$. It is plausible that we can have a backward bifurcation which would have consequences on equilibrium behaviour where endemicity can exist even when $R_0 < 1$. It also been noted that bifurcation can be a consequence of host-related factors such as induced immunity and differential susceptibility [124].

This is indicative of the inadequacy of the existing control measures in curbing ASF dissemination thereby requiring enhanced effort in devising new strategies or improving adherence to existing ones. In conclusion, we recommend that more epidemiological studies be designed to collect daily outbreak data from the field this will enable the relaxation of several assumptions made in this work and result in more accurate estimates of $R_0$.

# CHAPTER 4

## A mathematical model that simulates control options for African swine fever virus (ASFV)

Mike B. Barongo[1,#1], Richard P. Bishop[1], Eric M. Fèvre[1, 2], Darryn L. Knobel[3,#2], Amos Ssematimba[1,4]

[1]*International Livestock Research Institute, P.O. Box 30709, Nairobi 00100, Kenya*

[2]*Institute of Infection and Global Health, University of Liverpool, Leahurst Campus, Neston, CH64 7TE, UK*

[3]*Department of Veterinary Tropical Diseases, Faculty of Veterinary Science, University of Pretoria, South Africa*

[4]*Department of Mathematics, Faculty of Science, Gulu University, P.O. Box 166, Gulu, Uganda*

[#1]*Department of Academic Registrar, Makerere University, P.O. Box 7062, Kampala, Uganda*

[#2]*Center for Conservation Medicine and Ecosystem Health, Ross University School of Veterinary Medicine, P.O. Box 334, Basseterre, St Kitts*

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## 4.1 Abstract

A stochastic model designed to simulate transmission dynamics of African swine fever virus (ASFV) in a free-ranging pig population under various intervention scenarios is presented. The model was used to assess the relative impact of the timing of the implementation of different control strategies on disease-related mortality. The implementation of biosecurity measures was simulated through incorporation of a decay function on the transmission rate. The model predicts that biosecurity measures implemented within 14 days of the onset of an epidemic can avert up to 74% of pig deaths due to ASF while hypothetical vaccines that confer 70% immunity when deployed prior to day 14 of the epidemic could avert 65% of pig deaths. When the two control measures are combined, the model predicts that 91% of the pigs that would have otherwise succumbed to the disease if no intervention was implemented would be saved. However, if the combined interventions are delayed (defined as implementation from > 60 days) only 30% of ASF-related deaths would be averted. In the absence of vaccines against ASF, we recommend early implementation of enhanced biosecurity measures. Active surveillance and use of pen-side diagnostic assays, preferably linked to rapid dissemination of this data to veterinary authorities through mobile phone technology platforms are essential for rapid detection and confirmation of ASF outbreaks. This prediction, although it may seem intuitive, rationally confirms the importance of early intervention in managing ASF epidemics. The modelling approach is particularly valuable in that it determines an optimal timing for implementation of interventions in controlling ASF outbreaks.

**Keywords:** African swine fever; stochastic model; simulation; biosecurity; transmission rate

## 4.2 Introduction

African swine fever (ASF) is a devastating disease in domestic pigs caused by a DNA virus of the *Asfarviridae* family [65,94]. This disease is a significant constraint to pig production, causing economic losses to pig farmers and posing a threat to food security. ASF is endemic in most parts of Africa and its recent introduction into Georgia and subsequent spread to Russia and the European Union [99] renders it a global animal health problem that needs to be dealt with urgently [95]. It is a highly contagious disease transmitted by either direct contact between infected and susceptible pigs or indirectly through contact with infectious material in the environment and on fomites [11]. African swine fever virus (ASFV) is a resistant and stable virus capable of persisting in the environment and in pig products over a wide range of temperatures and pH for a prolonged period of time thereby enabling its transmission over long distances [65]. Clinical forms of the disease vary across a spectrum from peracute through acute to chronic and in some cases, apparently healthy virus carriers arise [34]. Peracute and acute syndromes are characterised by high fever, loss of appetite, haemorrhages and cyanosis on the skin and internal organs with mortality rates of up to 100% in naïve pig herds [55,65,95,125].

ASF has no cure or vaccine and its control depends on proper use of biosecurity measures, pig confinement and movement restriction plus culling of pigs on infected farms and in surrounding areas [126]. However, movement restriction is challenging to be effectively implemented in developing countries due to limited funding for public veterinary services. Likewise, pig confinement is not widely used in resource-poor countries where a large number of pigs are free-ranging due to limited access to and high cost of quality feeds. For other livestock diseases, vaccination is a key component of control strategies. In the case of ASF, research to develop and test vaccines is ongoing and a few experimental vaccines are promising candidates but need wider

evaluation before being commercialised [127–129]. In the absence of vaccines and/or chemotherapy and a lack of funds to compensate farmers in the event of culling, enhanced biosecurity remains the main ASF control measure in resource-poor countries.

In these resource-poor countries, there is limited information about animal movement patterns, factors that favour persistence of transmissible virus as well as the role of farmer behaviour in maintaining the endemic status of the disease. These factors, together with the limited knowledge about the disease's transmission pathways renders the design of improved ASF control strategies even more difficult [1].

Mathematical models may provide insight into the epidemiology of infectious diseases and the design of control strategies. They can be used to guide the identification of critical intervention points aimed at minimising disease-related mortality (hereafter referred to as disease burden) [22]. In addition, they can be used as tools for quantifying the magnitude, duration and cost of disease epidemics [23]. Models also provide an environment to assess how interventions may change the dynamics of the disease and how benefits may accrue from these interventions [22,24]. Therefore, integrating mathematical modelling benefits the design of ASF control strategies. In this study, we develop and parameterise a mathematical model to simulate the transmission of ASFV. We use the model to assess the relative impact of different intervention scenarios as well as to determine the optimum response time to suspected ASF epidemics.

Due to data limitation, the scope of the current study was limited to simulations with the aim of using the outcomes to inform further studies. For example, the outcomes from this study provide

a means to; 1) guide the design of the required experimental studies and, 2) help improve field data collection during future epidemics. In a commensurate interaction, results from these studies will in turn further refine future modelling attempts.

## 4.3 Materials and Methods

### 4.3.1 Geographical Study Area

The pattern of ASF outbreaks in Eastern Africa is different from that reported in Eastern Europe. In our study area some ASF outbreaks have been confirmed in areas with no wild pigs or argasid ticks (R. Bishop and E. Okoth, unpublished). Wilkinson [130] & OIE [125] have reported cases of pigs that survive ASF infection to become persistently infected (i.e., appear healthy while still shedding the ASFV virus) but when stressed they reactivate to infectious state.

The production system in the study area is characterized by low input pig husbandry practices where pigs are mainly free ranging and occasionally tethered [73]. Pigs in this kind of production system are known to cover an area within a radius of about 3km per day scavenging for food [15]. We therefore assume that pigs are homogeneously mixing due to the wide area they cover per day. Our study unit was a Parish consisting of 9 villages. This unit covers a geographical area of over 20 square Kilometers.

### 4.3.2 Model formulation and assumptions

Our model consists of five compartments categorising animals based on their status with respect to the disease: susceptible (S), infected but not yet infectious (E), infectious (I), carrier (i.e. persistently infected and asymptomatic animals, C) and the disease-induced deaths (D). The model incorporates population demographics as described by [103] and [131]. The model structure is

shown in Fig. 4.1 and events, parameter definitions, data sources and estimates are presented in Tables 4.1 and 4.2. The total population is given by N = S + E + I + C + D.

Table 4.1. Events defining the effect of transition between compartments and the rate at which they occur.

| **Event** | **Effect** | **Transition rate** |
|---|---|---|
| Exposure | (S, E, I, C) $\rightarrow$ (S-1, E+1, I, C) | $\beta S\left(I+\varepsilon C\right)$ |
| Infection | (S, E, I, C) $\rightarrow$ (S, E-1, I+1, C) | $\sigma E$ |
| Disease mortality | (S, E, I, C) $\rightarrow$ (S, E, I-1, C) | $\gamma\rho I$ |
| Recruitment | (S, E, I, C) $\rightarrow$ (S+1, E, I, C) | $\mu N$ |
| To Carrier | (S, E, I, C) $\rightarrow$ (S, E, I-1, C+1) | $\gamma(1-\rho)I$ |
| Carrier reactivation | (S, E, I, C) $\rightarrow$ (S, E, I+1, C-1) | $\kappa C$ |
| Natural death in Susceptible | (S, E, I, C) $\rightarrow$ (S-1, E, I, C) | $\mu S$ |
| Natural death in Exposed | (S, E, I, C) $\rightarrow$ (S, E-1, I, C) | $\mu E$ |
| Natural death in Infectious | (S, E, I, C) $\rightarrow$ (S, E, I-1, C) | $\mu I$ |
| Natural death in Carriers | (S, E, I, C) $\rightarrow$ (S, E, I, C-1) | $\mu C$ |

Several assumptions are made to allow for this formulation. New animals are born into the susceptible (S) class at a constant per capita birth rate equal to the natural mortality rate $\mu$. This assumption is vital to ensure that any system dynamics that we observe are likely to be disease-related. The movement of susceptible pigs from S to the Exposed (E) class is governed by the transmission rate parameter $\beta$. After a latent period $\sigma^{-1}$ days, exposed pigs transit to a state of infectiousness (I). A proportion $\rho$ of infectious pigs succumb to the disease while those that survive beyond the infectious period ($\gamma^{-1}$ days) are assumed to become carrier pigs at a rate $\gamma(1-\rho)$ [4,55,125,130]. Carrier pigs are also assumed to contribute to the infection pressure though at a reduced rate ($\beta\varepsilon$) and may occasionally reactivate and transition back to the infectious class (I) at a rate $\kappa$ [39]. Natural mortality occurs in all classes and additional disease-specific mortality

occurs in the infectious class at a rate ( $\gamma\rho$ ).We assume density-dependent transmission because the pigs freely interact and infection can occur when contact happens.

Table 4.2. The Minimum, Mode and Maximum estimates used in the Pert distributions for the parameters of the model (day $^{-1}$)

| | **Definition** | **Min** | **Mode** | **Max** | **Key data source** |
|---|---|---|---|---|---|
| $\mu$ | Non-specific mortality/ crude birth rate* | 0.0020 | 0.0027 | 0.0035 | User defined# |
| $\beta$ | Transmission rate$ | 0.200 | 0.300 | 0.500 | [116] |
| $\gamma$ | ASF-specific mortality rate | 0.080 | 0.125 | 0.250 | [116] |
| $\rho$ | Proportion of infectious that die | 0.600 | 0.700 | 0.800 | [95] |
| $\sigma$ | Transition rate from exposed to infectious class | 0.120 | 0.250 | 0.350 | [2,95,96,125] |
| $\kappa$ | Rate of reactivation of carriers* | 0.040 | 0.060 | 0.080 | [125] |
| $\varepsilon$ | Scale-down factor on effective contact rate for carrier animals* | 0.250 | 0.300 | 0.350 | User defined |

* User defined for purposes of this simulation, # Based on observed average life expectancy of 370 days, $ The minimum $\beta$ estimate of [116] is taken as the Max value for the Pert distribution in estimating $\beta$. We scaled down by a factor of $(1.5)^{-1}$ and $(1.5)^{-2}$ respectively for the mode and minimum values

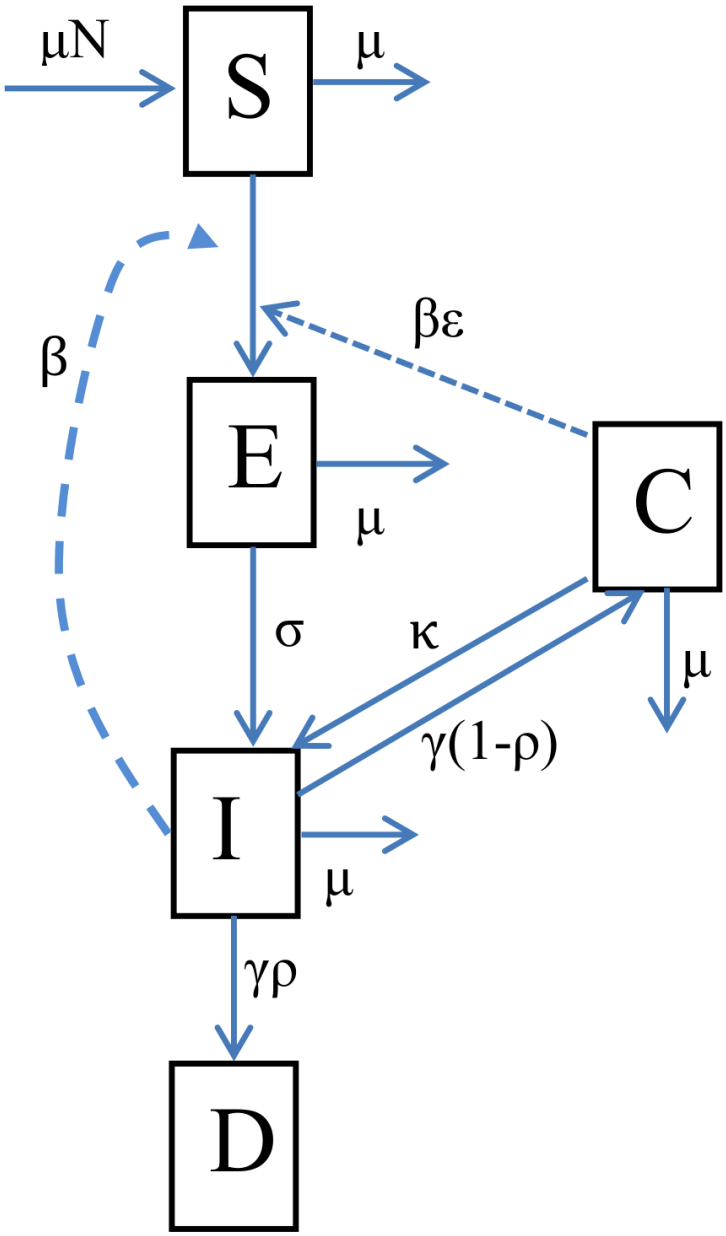


Fig. 4.1 The schema shows the transition pathways between epidemiological classes of the ASF model

The schema shows the transition pathways between epidemiological classes of the ASF model. The transition from class (S) to class (E) was governed by transmission rate ( $\beta$ ) while the transition from class (E) to class (I) was dependent on latent period ( $\sigma$ ). The infectious animals either die at a rate ( $\gamma\rho$ ) and enter class (D) or enter the carrier class (C) at a rate $\gamma(1-\rho)$. Carriers also transmit at a reduced rate ( $\varepsilon\beta$ ) and can re-activate to infectiousness at a rate ( $\kappa$ ). There is natural mortality that occurs in each class at a rate μ. New recruits enter the S class at a rate μN

The dynamics of the system described and presented in Fig. 4.1 are captured by the differential equations:

$$\frac{dS}{dt} = -\beta S\left(I + \varepsilon C\right) + \mu N - \mu S,$$

$$\frac{dE}{dt} = \beta S\left(I + \varepsilon C\right) - (\sigma + \mu)E,$$

$$\frac{dI}{dt} = \sigma E + \kappa C - \gamma\rho I \; - \gamma(1-\rho)I - \mu I,$$

$$\frac{dC}{dt} = \gamma(1-\rho)I - (\kappa + \mu)C,$$

$$\frac{dR}{dt} = \gamma\rho I$$

The system's events were implemented stochastically using Gillespie's direct algorithm [26,132] and 1000 simulations were run per scenario described in the section of intervention scenarios.

### 4.3.3 Model Parameters

In Table 4.2 we present estimates for some model parameters obtained from literature. Generally, there is limited fields and /or experimental data to quantify model parameters and those accessed varied widely. We chose to use the Pert distribution to randomly generate parameter estimates over the extracted parameter ranges. The Pert distribution is best suited to situations when information available to estimate parameters is limited but sufficient to extract the Minimum, Maximum and Mode (i.e., most likely) estimate. The non-specific mortality ($\mu$) was estimated as the reciprocal of the mean life expectancy of pigs in the study region (i.e. 280 - 500 days). The transmission rate

parameter β(t) has been estimated from the literature [116] taking into account the difference in pig interactions between those under experimental conditions and those in the natural setting. The scale-down factor (ε) on the transmission rate for carrier animals and the rate of reactivation of carriers (κ) to infectious state are user-defined for simulation purposes. These parameters have been set to values between zero and one due to lack of information on them pending further studies to give them appropriate values. For purposes of our exploration we set them in a low range of 0.3 and 0.06 respectively.

### 4.3.4 Intervention scenarios modelled

Using scenario analysis approach, we assess the effect of different interventions on the cumulative number of pigs that succumb to ASF over a simulation period of 200 days. As a reference point for the assessment of impact of interventions, we started by simulating the dynamics of the disease in the population of 500 pigs without any intervention. Thereafter two categories of intervention scenarios were simulated, with each being initiated at various time points after the onset of the epidemic. The first category consisted of implementation of biosecurity measures which were modelled as a change in the time-dependent transmission rate parameter β(t) according to

$$\beta(t) = \begin{cases} \beta_1 & t < \tau \\ \beta_0 + (\beta_1 - \beta_0)e^{-(t-\tau)} & t \geq \tau \end{cases}$$

where τ is the time at which biosecurity interventions start [133]. β(t) is modelled to gradually reduce following an exponential decay from a baseline value $\beta_1$ to a value that asymptotically approaches $\beta_0$ (set to 0.05 in this study) [133,134]. The value of $\beta_0$ can be set to zero if the biosecurity measures put in place are perceived to be able to stop all further transmission.

The second category of interventions modelled the potential effect of using hypothetical vaccines with varying efficacies and coverage. The vaccine-protected proportion is obtained from the product of vaccine efficacy and coverage levels. In this study, we modelled vaccination at three protection levels; 30%, 50% and 70%. Vaccination was modelled as a single pulse event during the course of the simulation. The effect of time to intervention was assessed by implementing the above interventions at 14, 30 and 60 days after the start of the epidemic. As examples the naming of the intervention scenarios was as follows: "Vac_7030" is vaccine intervention with effect of 70% and day 30 after onset of epidemic whereas "Bio_30" is a biosecurity intervention 30 days after the onset of the epidemic. Baseline is an intervention-free scenario while "Bio_Vac_7014" is a combination of biosecurity and vaccination strategy (with 70% effect) at day 14 and "Ctns_Vac_7014" is an intervention scenario where 70% are effectively vaccinated at day 14 and all new recruits thereafter are also vaccinated.

We present results of a few of the many permutations of intervention scenarios that could be compared. Day 14 and day 60 were of particular interest because they represented the earliest practical date to implement an intervention and a representation of a date not very long into the complete course of an outbreak, yet too late for an intervention to cause the desired mitigating effect. The model simulations were run in Wolfram Mathematica 9.

## 4.4 Results

Model predictions of the impact of the different intervention strategies on the cumulative number of pigs that succumbed to the disease are presented. The results are presented as boxplots depicting the median, lower and upper quartiles of predicted disease burden from the 1000 simulations per intervention scenario. Fig. 4.2 depicts the disease burden for different times of introduction of

intervention scenarios while Fig. 4.3 presents a comparison of the potential impact of delaying the start of intervention strategies (scenarios for day 14 and day 60).

### 4.4.1 Effect of enhanced biosecurity measures on the disease burden

In Fig. 4.2a, the model predicts a 74% reduction on the disease burden if biosecurity measures are implemented 14 days after the onset of the epidemic (Bio_14) compared to the baseline scenario which predicts a median of 535 fatalities. Implementing biosecurity measures 30 and 60 days after the onset of the epidemic decreases the disease burden by 41% and 13.5% from the baseline scenario, respectively.

### 4.4.2 Effect of vaccination interventions on the disease burden

Fig. 4.2b shows the disease burden under pulse vaccination where 50% of the animals at risk are vaccine-protected at 14, 30 or 60 days after onset of the epidemic. Vaccinating 50% after 14 days of the epidemic onset reduced the burden by 44% while waiting for 60 days reduced the burden by 16%. Fig. 4.2c shows the impact of the different proportions protected by the vaccine when intervening at day 14, either singly or in combination with biosecurity measures. The model predicts a 65% reduction in cumulative pig deaths when the vaccine protects 70% of the animals at risk (i.e., Vac_7014). With a delayed intervention, i.e. when intervening at day 60 post epidemic onset, there is a minimal reduction (ranging from 4% to 14 %) in the disease burden across all simulated intervention scenarios (Fig. 4.2d). Among the simulated vaccine intervention scenarios, Vac_7014 is predicted to avert the highest number of pig deaths (only 185 deaths) compared to the baseline scenario.

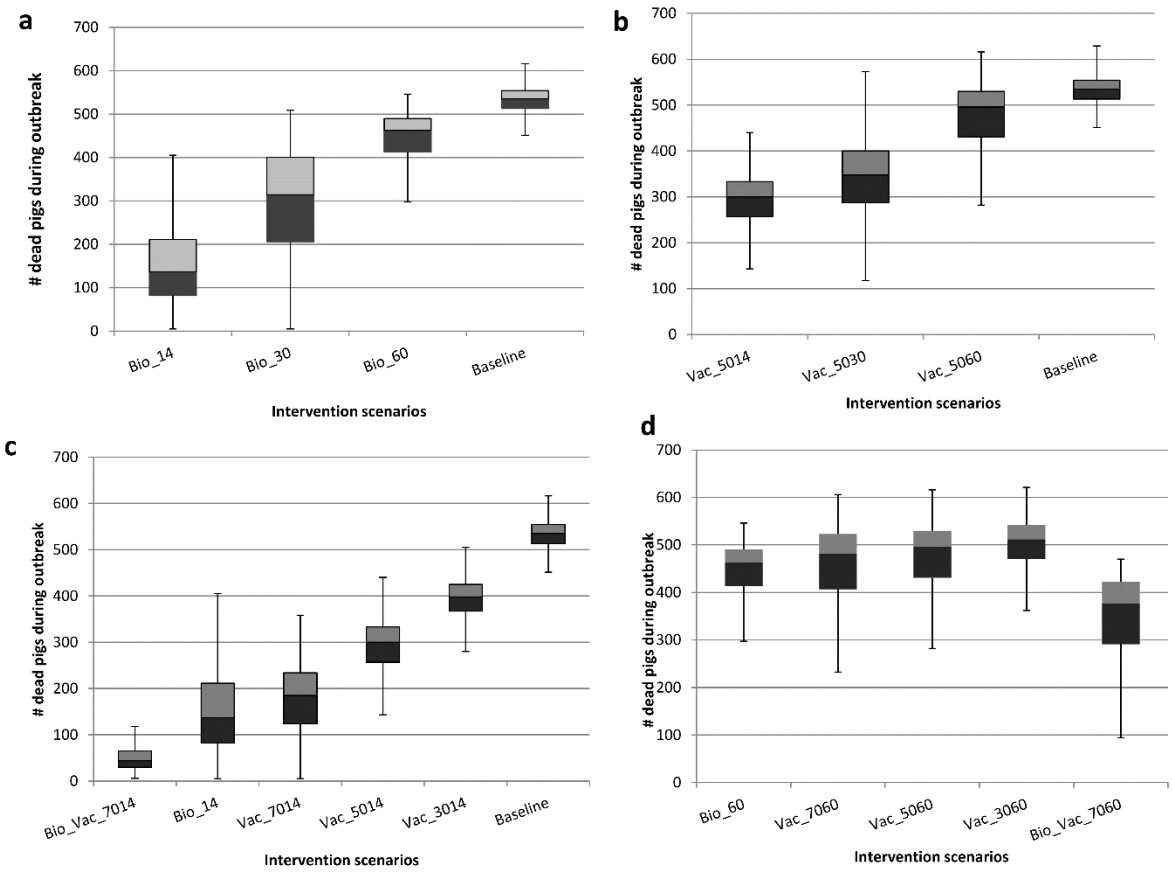


Fig. 4.2 Box plots showing the effect of timing of introduction of different intervention scenarios on disease burden.

The baseline box represents an intervention-free scenario. Panel (a) shows the effect of the timing of introduction of biosecurity measures after the onset of the epidemic (where Bio_xx = Biosecurity strategy implemented at day xx). Panel (b) depicts effects of vaccination (protecting 50%) implemented at day 14, 30 and 60 days on disease burden (i.e. Vac_50xx = Vaccination conferring 50% protection at day xx). Panel (c) compares the effects of different vaccine efficacies and a combination intervention strategy on disease burden when intervention is started at day 14 (Bio_Vac_7014 = Combination of biosecurity and 70% Vaccine efficacy implemented at day 14). Panel (d) depicts the effects of delayed intervention on disease burden across different strategies of vaccine efficacies and combination scenarios (i.e. Vac_yyxx = Pulse Vaccination of efficacy yy% implemented at day xx while Bio_Vac_7060 is a combination strategy of Biosecurity measures and 70% efficacy vaccine implemented at day 60).

In Fig. 4.3a we compare the impact of interventions implemented at day 14 involving protection of 70% of the pigs at risk through vaccination under different vaccination and biosecurity schemes. An intervention scenario involving a pulse vaccination at day 14 coupled with a continuous vaccination (and protection) of 70% of the new recruits (i.e., Ctns_Vac_7014) reduces the disease burden by 82%. A strategy combining intensified biosecurity measures and pulse vaccination and

protecting 70% of the pigs at risk (i.e., Bio_Vac_7014) could avert up to 91% of pig deaths, yet the same strategy, when implemented after 60 days post epidemic onset could only save 30% (Fig. 4.3b).

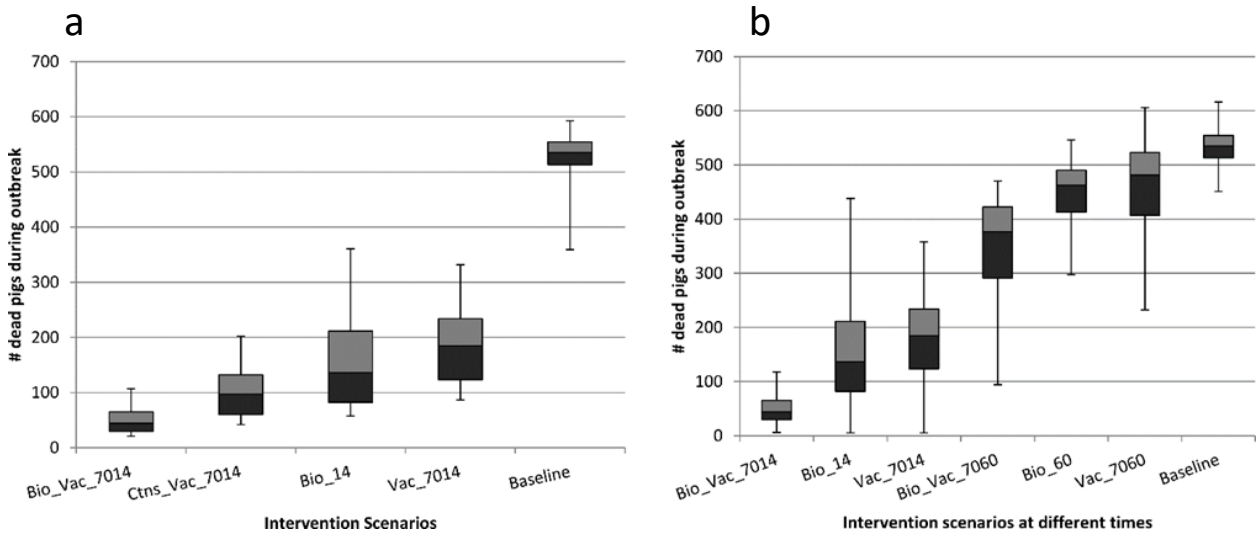


Fig. 4.3 Box plots comparing different intervention scenarios at day 14 and day 60.

Panel (a) shows relative impact of the intervention scenarios at day 14. Bio_Vac_7014 is a combination of biosecurity measures and vaccination with 70% effect at day 14. Box Ctns_Vac_7014 is a scenario of pulse vaccination at day 14 followed by continuous vaccination programme of new recruits. Panel (b) compares the effects similar intervention scenarios at day 14 and day 60 to show the effect of timing of intervention on disease burden irrespective of the strategy implemented.

## 4.5 Discussion

ASF continues to be a major constraint to the growth of the pig industry in sub-Saharan Africa and poses a significant risk to established pig industries in the developed world mainly the European Union and China. There is a need to continuously refine and update both our knowledge of its epidemiology and control measures. In the present study, we used a stochastic compartmental mathematical model to assess the potential impact of different intervention scenarios on the disease burden.

In the absence of treatment or vaccines against ASF, control strategies primarily rely on biosecurity measures [34,126]. It has been noted that smallholder pig farmers find it difficult to fully comply with biosecurity measures for a prolonged period of time because of the nature of their production systems [73]. We investigated the benefits of compelling small holder farmers to adopt and intensify biosecurity measures specifically in times of ASF outbreaks, in order to minimize the disease burden. When compared to the baseline scenario of not intervening at all, our model predicts that if biosecurity measures are enhanced within a fortnight of an epidemic onset, the disease burden can be reduced by up to 74%. This finding emphasises the need to hasten the intensification of biosecurity measures in the event of suspicion. This can be achieved through measures such as improved hygiene, isolation of sick or new pigs, movement control, treatment of swill, use of disinfectants, proper confinement and disposal of dead pigs as soon as ASF is suspected [73]. This is achievable if resources are available to implement this intervention however if left entirely in the hands of farmers, they may not have the resources or incentive to meet these costs. Above all, farmers in the settings in question need to be highly aware of ASF and of the tools available to them to quickly upscale their on-farm biosecurity. This is in agreement with standard ASF control protocols, which emphasises that biosecurity is essential in the control of ASF [95,135].

In addition, we modelled three protection levels of hypothetical vaccines and three intervention time points for their use. The greatest vaccine impact (of 65% reduction in disease burden) was predicted at the highest simulated vaccine protection level of 70% when implemented at day 14 post epidemic onset. The impact of this pulse vaccination on disease burden is likely to be affected by the continuous influx of susceptible new recruits that enter the system. In an ideal situation, vaccination schemes should be designed in such a way as to include newly-recruited animals on a

continuous basis. We capture this scenario by simulating interventions where vaccination is continuous and conclude that continuous vaccination reduces the disease burden by 82%.

The most effective simulated intervention strategy (with 91% of deaths averted) is a combination of pulse vaccination (protecting 70% of the pigs at risk) together with enhanced biosecurity measures implemented by day 14. However, vaccines are still a long way from being commercially available, let alone accessible and affordable to the rural pig farmers. Early attempts to develop conventional vaccines against ASFV achieved partial protection or could not be scaled up for commercial production [128]. Nonetheless, these results, although theoretical at this point, illustrate the potential impact of vaccines on disease burden and how they could improve control efficacy when combined with biosecurity measures. They also help in identifying the levels of protection that any eventual vaccine would need to attain in order to be effective in preventing epidemics.

The predicted effect of intervention strategies on the disease burden was found to be dependent on time to intervention with delayed intervention reducing the impact of intervention scenarios. For example, intervening 60 days post epidemic onset reduced the impact of all scenarios, with only 4% to 30% of baseline deaths averted as compared to reductions of 65% to 91% when intervening at day 14. This prediction, although intuitive, emphasises the importance of early intervention in managing ASF epidemics, and our modelling approach provides a means to determine appropriate and feasible intervention moments in controlling ASF, in this case found to be 14 days post epidemic onset.

ASFV varies in virulence with some strains causing 100% mortality while less virulent ones allow some pigs to recover from either sub-acute or chronic infections to become persistently infected

or carrier pigs. These carrier animals are assumed to play a role in maintaining the disease in the domestic cycle and pose a major challenge to its control [35,125]. However, there is no sufficient evidence to quantify the contribution of carrier pigs to infection pressure and what proportion reactivate to an infectious state.

We recommend that further studies be carried out to more reliably quantify these model parameters using empirical data from field activities. Our studies [4,136] on ASFV p72 genotypes IX and X in East African smallholder systems indicates that transmission data is particularly hard to collect in the field with the currently available techniques, since anthropogenic effects (rapid selling to butchers, or pig farmers in distant villages) complicate collection and interpretation of transmission data. In this study we have relied on using random parameter choice based on the Pert distribution informed by available data to improve reliability of the study's outcomes. We envisage that in future projects involving appropriately designed experimental infections will be used to refine ASFV transmission parameters.

Although our model predicts a combination of vaccination and enhanced biosecurity as the best intervention scenario, the only currently feasible strategy is implementation of enhanced biosecurity measures. We therefore recommend intensification of active surveillance and use of pen-side diagnostic assays for rapid detection and confirmation of ASF to allow for timely implementation of enhanced biosecurity. However, we also recommend continued research on the development of a vaccine against ASFV to allow for deployment of a hybrid intervention strategy. Most importantly, given the importance of the time to implementing biosecurity measures, we recommend that veterinary services in ASF outbreak risk areas work to educate farmers on the most feasible biosecurity measures to adopt in a time efficient manner [73]. Finally, we also

suggest that further work on cost-benefit analyses should be performed to compare the simulated interventions from an economic perspective.

# CHAPTER 5

## An Analysis of Key Parameters of Low Input Pig Production Systems along the Kenya-Uganda Border

Mike B. Barongo[1], Richard P. Bishop[2], Eric M. Fèvre[3,4],

Amos Ssematimba[5], Darryn L. Knobel[6]

[1]*Department of Academic Registrar, Makerere University, P.O. Box 7062, Kampala, Uganda*

[2]*Department of Veterinary Microbiology and Pathology, Washington State University, PO Box 647040, Pullman, WA.*

[3]*International Livestock Research Institute, P.O. Box 30709, Nairobi 00100, Kenya*

[4]*Institute of Infection and Global Health, University of Liverpool, Leahurst Campus, Neston, UK*

[5]*Department of Mathematics, Faculty of Science, Gulu University, P.O. Box 166, Gulu, Uganda*

[6]*Current address: Center for Conservation Medicine and Ecosystem Health, Ross University School of Veterinary Medicine, P.O Box 334, Basseterre, St Kitts*

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## 5.1 Abstract

Livestock production systems influence disease spread dynamics. For example, in pig production, economically important diseases such as African swine fever and Foot- and- mouth Disease are largely driven by the nature of production systems practiced. Hence, deeper knowledge on the factors that determine production systems especially in low income countries are key parameters if better disease control strategies are to be developed. The main purpose of this cross-sectional research was to identify the key parameters governing low input production systems in smallholder pig farmers in four districts along the Kenya-Uganda border. Data were collected by administering questionnaires to 640 households selected in the study area. These data were analyzed to obtain information pertaining demography of the producers, pig breeds kept, farm-inputs, and the husbandry types practiced among others. Analysis results showed that 86% of the households were headed by males though activities relating to pig farming were primarily performed by females. Approximately 43% of pig keepers had been educated to secondary level, but a low proportion of these were female. Over 90% of the households surveyed kept local pig breeds with the weaner pig category constituting 48% of the pigs on the farm. The predominant husbandry system was tethering (71%). The farmers feed their pigs mainly on crop residues/grass (40%) and Swill/household leftovers (40%). The main challenge faced by these pig farmers was access to affordable and reliable feed supplies. A significant proportion of small scale pig farmers were willing to accept advice from government extension workers. The study therefore strongly suggests that improving the supply of affordable pig feeds would be a key step in incentivizing adoption of better husbandry and health practices and increasing productivity in the small pig sector.

**Keywords**: Low input systems; Small holder Pig farmers; pig husbandry systems; pig breeds

## 5.2 Introduction

Many developing countries in the sub-Saharan Africa are reliant on agriculture as their main economic activity and source of livelihood. According to recent studies, agriculture is the most important sector of the Ugandan economy, employing over one third of the work force and 69% of the population, mainly women, in the rural areas [137]. Livestock farming as a subsector of agriculture has continued to play an important role in meeting the ever increasing demand for animal proteins as a result of an increase in urbanization and also the general increase in population [138,139]. It has been noted that pigs are important livestock capable of providing much needed animal protein due to their high productive rates, quick maturation and ease of conversion to cash to meet urgent household needs that have a direct impact on smallholder farmers' livelihoods and food security [16,21,73].

Different breeds of pigs tend to be reared across the farmers' economic profile with the more economically empowered farmers keeping both pure and cross breed pigs from European domestication centers in commercial farms while the small holder farmers keep African genotype pigs [16,140]. African genotype pigs are the predominant breeds in areas where low input systems are used [141]. Pigs require minimal family labour and feeding if allowed to roam freely, although it is illegal according to the animal disease act (Section 9 and Rules 15–28) of Kenya allowing pigs to roam freely scavenging for food. This free-range practice is risky in relation to spread of diseases such as African Swine Fever (ASF) and Foot and mouth Disease (FMD) within the pig populations. Hence, there is need to conduct studies to assess the pros and cons of such a practice with an underlying aim of devising means on how to minimize it. The first step towards achieving this is through understanding the factors that influence the choice of a pig husbandry system.

Such knowledge can, among others, be derived from conducting interview studies in the geographical areas of interest. To this effect, previously, studies focusing on farmer perceptions [141], characteristics of free-range systems [16] and risk factors associated with small holder pig production systems on the Kenya-Uganda border [75] have been conducted. The current study builds on the study of [75] and focuses on gaining deeper understanding of the factors that favour the predominantly practiced pig husbandry system along the Kenya-Uganda border. The main study aim was to analyse the key parameters of low input pig production system in the selected geographical area. The other objective was to capture data on the diversity of pig keeping practices, to understand pig keeping systems in relation to ASF prevalence. It is expected that the findings of this study will provide subsidies for public sector agencies to assist farmers by developing good production practices with pigs, reducing rates of disease infestation, and improving the health status of farms.

## 5.3 Materials and Methods

### 5.3.1 Data source

The data were collected through a household cross-sectional survey using structured questionnaires. The study was conducted along the Kenya-Uganda border between July and November 2012, targeting farmers operating on either side of the shared border. Primarily, households with pigs on farm were targeted for interview and any adult household member conversant with the pig farming activities was interviewed. A total of 640 pig keeping households participated in the study.

Details on the selection and inclusion criteria of respondents was earlier described in [73,75] and are only briefly described here. The study was conducted in two districts in Uganda (Busia and

Tororo) and two in Kenya (Teso and Busia, Figure 5.1). The sampling frame was designed in such a way that eight sub-counties on the Ugandan side that lie along the border were selected (four from each district) and eight locations on the Kenyan side of the border were selected (four from each district) with the help of GIS software. In each sub-county in Uganda (and corresponding location in Kenya), two parishes (and corresponding sub-locations in Kenya) were randomly selected. Subsequently, a list of villages in each selected parish in Uganda (corresponding sub-location in Kenya) was obtained with the help of district veterinary staff and local leaders. Two villages were randomly sampled from each list and a list of all pig keeping households in the selected village was generated with assistance from the local leaders.

Twenty households were systematically sampled from the list of all pig keeping households. Where villages had less than 20 pig keeping households, the selection was extended to pig keeping households from adjoining villages. The sampling frame generated 32 villages and 640 households that were involved in the study. The questions were set with an underlying objective of capturing data on the diversity of pig husbandry practices and to further relates this information to ASF prevalence. The data collected included household information, pig production systems, phenotypic characteristics of pigs on farm, socioeconomic indicators, ASF awareness, biosecurity practices, access to advisory services and pig farmers social networks.

### 5.3.2 Study Area

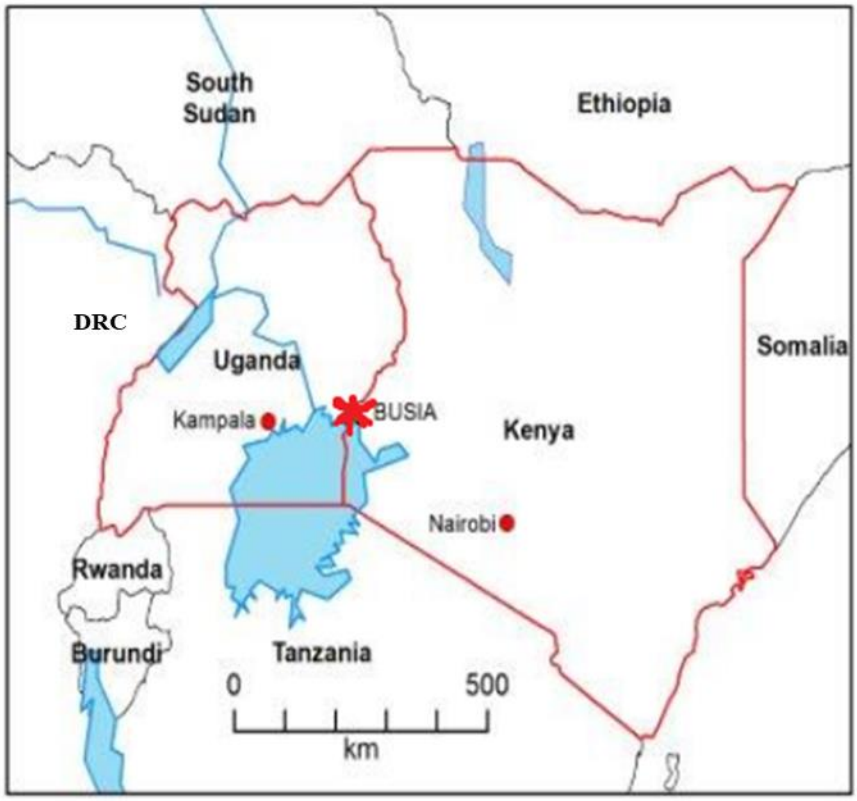


Fig. 5.1. Map showing the study area along the Busia/Tororo (Uganda) and Busia/ Teso (Kenya) border.

### 5.3.3 Data Analysis

The data were captured using Palm Digital Assistants (PDA) running on Pendragon forms 5.1 and was downloaded into a Microsoft Access 2010 database. Descriptive Statistical analyses of the data were performed using Microsoft Access 2010 and Statistical Package for the Social Sciences (SPSS) Ver. 18 software.

## 5.4 Results

### 5.4.1 Household Characteristics

The descriptive results from the study are presented in Table 5.1. The age group of the respondents ranged from 16 to 84 years with the highest proportion (48%) in the 30-50 age group. About half of all household heads had only primary level education and 43% had attained a secondary education. Of the respondents, 65% had primary level education only, and 22% had attained at least secondary level education. The main occupation in the study area is farming (51%) followed by those engaged in some form of business (17%), civil service employment (15%) while casual labor and others constituted (17%). The average pig keeping household size was seven people even though overall the average household size in the region is officially between 4 to 5 people (2014 Uganda census and Kenya population data sheet 2011).

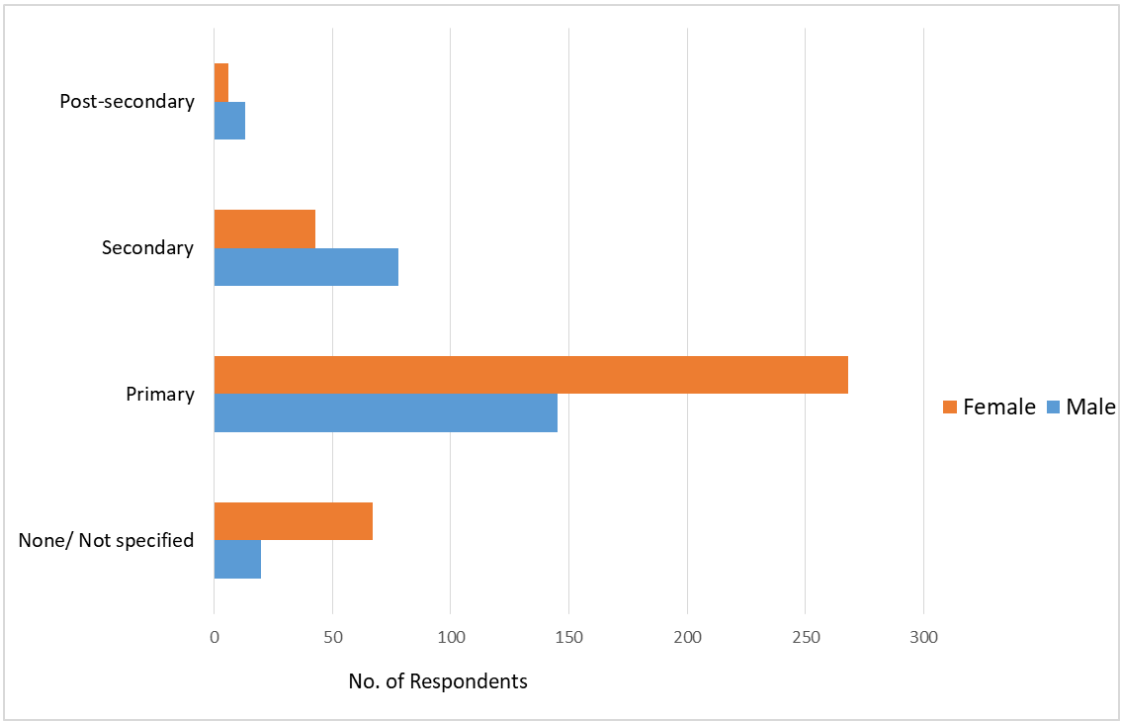


Fig. 5.2 Education characteristics of respondents by gender

In figure 5.2 we characterize education level by gender of respondent. The majority of respondents (60%) were female of which 70% had primary level education while 17 % had no formal education. For the males, only 8% had no formal education while 56% had attained primary level education. There were only 3% of respondents who had post-secondary education. Table 5.2 presents education level by gender of respondent

Table 5.1. Summary descriptive results for selected Household (HH) characteristics from the study area

| | **Number of HHs** | | | |
|---|---|---|---|---|
| **Characteristics** | **Overall** | **Kenya** | **Uganda** | **Overall %** |
| **HH head Gender** | | | | |
| Male | 553 | 276 | 277 | **86.4** |
| Female | 87 | 44 | 43 | **13.6** |
| **Respondent's Gender** | | | | |
| Male | 256 | 117 | 139 | **40.0** |
| Female | 384 | 203 | 181 | **60.0** |
| **Respondent's age (years)** | | | | |
| Below 30 | 172 | 87 | 85 | **26.9** |
| 30-49 | 307 | 158 | 149 | **48.0** |
| 50 Above | 161 | 75 | 86 | **25.2** |
| **Education Level of HH head** | | | | |
| No Education | 24 | 19 | 5 | **6.9** |
| Primary | 174 | 111 | 63 | **49.7** |
| Secondary | 109 | 43 | 66 | **31.1** |

| | | | | |
|---|---|---|---|---|
| Post-secondary | 43 | 15 | 28 | **12.3** |
| **Respondent's Education** | | | | |
| None | 87 | 45 | 42 | **13.6** |
| Primary | 413 | 214 | 199 | **64.5** |
| Secondary | 121 | 48 | 73 | **18.9** |
| Post-secondary | 19 | 13 | 6 | **3.0** |
| **Occupation of HH head** | | | | |
| Business | 58 | 35 | 23 | **16.5** |
| Casual Laborers | 15 | 10 | 5 | **4.3** |
| Farmers | 179 | 96 | 83 | **51.0** |
| Civil servants | 54 | 21 | 33 | **15.4** |
| Others | 45 | 27 | 18 | **12.8** |
| **Household Size** | | | | |
| 1-5 | 207 | 120 | 87 | **32.3** |
| 6-10 | 363 | 178 | 185 | **56.7** |
| above 10 | 70 | 22 | 48 | **10.9** |

**Table 5.2 . Education characteristics of respondents by gender**

| Education Level | Gender of Respondents | | | |
|---|---|---|---|---|
| | Male | % | Female | % |
| None/ Not specified | 20 | 23 | 67 | 77 |
| Primary | 145 | 35 | 268 | 65 |
| Secondary | 78 | 64 | 43 | 36 |
| Post-secondary | 13 | 68 | 6 | 32 |

### 5.4.2 Household level production system parameters

Table 5.3 presents results on the identified key system parameters in the study area. One important observation is that 92% of the 640 pig keeping households surveyed had local African pig breeds on the farm while only 23 households (3.6%) had pure European-domestication centre breeds and 16% kept crosses. The majority of the pigs on the farms were weaners (48%). There were marginally more weaners on Ugandan side (51.8%) than on the Kenyan side of the border. The most frequently purchased pig category by the farmers was piglets (71.8%) followed by weaners (21.7%). We observed that for all pig categories purchased, other than piglets, the Kenyan side accounts for most such purchases with 64% for weaners, 75% for sows and 76.5% for boars. The most common sources for pigs that were introduced into the households during the previous year were from acquaintances such as friends, relatives and neighbors (47.4%) and other farmers (29.4%) while only 18.1% were born in the household. There is more in-house breeding in Uganda (54.2%) compared to Kenya (45.8%).

**Table 5.3** Characterizing Household (HH) level pig systems parameters on the Kenya – Uganda border

| Parameter | | Total | | Ugandan side | | Kenyan side | |
|---|---|---|---|---|---|---|---|
| | | No. HH | % | No. HH | % | No. HH | % |
| **Breed kept** | Local | 590 | 92.0 | 282 | 48.0 | 308 | 52.0 |
| | Cross | 105 | 16.0 | 33 | 31.0 | 72 | 69.0 |
| | Grade/Exotic | 23 | 3.6 | 12 | 52.0 | 11 | 48.0 |
| **Pig Categories** | Piglets | 114 | 12.6 | 51 | 44.7 | 63 | 55.3 |
| | Weaners | 436 | 48.1 | 226 | 51.8 | 210 | 48.2 |
| | Sows | 227 | 25.1 | 113 | 49.8 | 114 | 50.2 |
| | Boars | 129 | 14.2 | 61 | 47.2 | 68 | 52.8 |
| **Pigs purchased** | Piglets | 322 | 71.8 | 163 | 50.6 | 159 | 49.4 |
| | Weaners | 97 | 21.7 | 35 | 36.0 | 62 | 64.0 |
| | Sows | 12 | 2.7 | 3 | 25.0 | 9 | 75.0 |
| | Boars | 17 | 3.8 | 4 | 23.5 | 13 | 76.5 |
| **Pig source** | Pig farmer | 234 | 29.4 | 102 | 43.6 | 132 | 56.4 |
| | In house | 144 | 18.1 | 78 | 54.2 | 66 | 45.8 |
| | Friend/relatives/ neighbor | 378 | 47.4 | 159 | 42.1 | 219 | 57.9 |
| | Market/ pig traders | 36 | 4.5 | 32 | 88.9 | 4 | 11.1 |
| | Other (NAADS, NGOs) | 5 | 0.6 | 2 | 40 | 3 | 60 |
| **Management system** | Free range | 238 | 27.1 | 122 | 51.3 | 116 | 48.7 |
| | Tether | 626 | 71.4 | 309 | 49.4 | 317 | 50.6 |
| | Housed | 13 | 1.5 | 7 | 53.9 | 6 | 46.1 |
| **Feed source** | Crop Residue/ Grass | 617 | 39.8 | 314 | 50.9 | 303 | 49.1 |
| | Swill/HH leftovers | 615 | 39.7 | 309 | 50.2 | 306 | 49.8 |
| | Commercial feeds | 316 | 20.5 | 152 | 48.1 | 164 | 51.9 |
| **Advisory services** | HH member/friend | 180 | 29.5 | 100 | 55.6 | 80 | 44.4 |
| | Local Leaders | 80 | 13.1 | 39 | 48.7 | 41 | 51.3 |
| | Other farmers | 64 | 10.5 | 35 | 54.7 | 29 | 45.3 |
| | Pig Traders | 8 | 1.3 | 3 | 37.5 | 5 | 62.5 |
| | Vet/NAADS officers | 278 | 45.6 | 154 | 55.4 | 124 | 44.6 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| **Pig agisting** | On-farm | 46 | 31.1 | 28 | 60.1 | 18 | 39.9 |
| | Off-Farm | 102 | 68.9 | 64 | 62.7 | 38 | 37.3 |

The pig husbandry systems practiced in the study area were a mix of tethering, housing and free range. The majority of the households tethered (71.4%) their pigs, although not all the time while 27.1% of the farms surveyed practiced free- range with occasional tethering. Only 1.5% of the farmers housed their pigs. The farmers interviewed feed their pigs on Crop Residues/ Grass (39.8%) and Swill/household leftovers (39.7%) and commercial feeds were only used by 20.5% of the farmers. From the households surveyed, feeding practices were found to be similar on both sides of the border. For advice on issues pertaining to pig keeping, it was found that 45.6% of the farmers relied on Veterinary or National Agricultural Advisory Services (NAADS) officers, and 29.5% on a Household member/friend and only 1.3% relied on pig traders.

In the study area, there is a practice of keeping some pigs away from the household farm (agisting). This is intended to provide a fall-back position in the event of a tragedy hitting the farm. This acts as a form of insurance. Of the households visited 23% used agisting, and of these, 31.1% kept a friend's pig (on-farm) while and 68.9 % had someone else keep their pig (on a different farm).

### 5.4.3 Challenges of these production systems

Some of the challenges faced by low input pig production systems are shown in Table 5.4. Animal feeds were found to be the most important constraint that low input farmers face. The price of feed was considered high by 45.4% of farmers interviewed while 47.8% of the farmers reported that feeds are scarce or that the supply is unreliable. Surprisingly very few households (6.8%) were concerned about feed quality. The second major challenge farmers identified was pig health.

Approximately 11% of the households (71 among 640) reported health related issues that resulted in pig death on the farm. Some of these deaths were reported to be sudden and African swine fever was suspected.

Table 5.4. Challenges faced by low input pig production systems on the Kenya-Uganda border

| **Parameter** | | **Total** | | **Ugandan side** | | **Kenyan side** | |
|---|---|---|---|---|---|---|---|
| | | Number | % | Number | % | Number | % |
| **Feeds** | High Prices | 227 | 45.4 | 113 | 49.8 | 114 | 50.2 |
| | Quality | 34 | 6.8 | 25 | 73.5 | 9 | 26.5 |
| | Supply/access | 239 | 47.8 | 95 | 39.8 | 144 | 60.2 |
| **Pig Health** | Pig death | 71 | 70.3 | 56 | 78.9 | 15 | 21.1 |
| | Disinfectants | 30 | 29.7 | 30 | 100. | 0 | 0.0 |
| **Market issues** | Poor prices | 2 | 28.6 | 0 | 0.0 | 2 | 100.0 |
| | Market disruptions | 2 | 28.6 | 1 | 50.0 | 1 | 50.0 |
| | Others (No pig buyers) | 3 | 42.8 | 2 | 67.0 | 1 | 33.0 |

Of the surveyed households, 5% occasionally used disinfectants as a way to mitigate pig health related issues. A handful of farmers (1.1%) expressed challenges with market related issues such as poor prices, market disruptions and transportation of pigs to the market. The relatively small number of pig farmers reporting market related challenges could be due to lack of a pig supply relative to demand hence a potential for increase in pig production.

## 5.5 Discussion

Outbreaks involving most economically important pig diseases for example ASF and FMD are known to be partly driven by the pig production systems practiced in the area of interest [73,74,79].

This current study was conducted among the pig producers on the Kenya-Uganda border with the aim of capturing data on the diversity of husbandry practices.

From the survey, it was found that males were the majority household heads, yet most of the respondents were female. The low male attendance for interviews could be an indication that they are engaged in other economic activities other than pig farming [75] or that they attach less economic value to the pigs. Nantima et al. (2015) found that males owned the majority of the pigs, even though much of the day to day care of the pigs is given by females. This finding was in agreement with those from a study carried out in Kakamega in Western Kenya in which it was found that women took the lead in the management of family pigs since men were rarely at home [141]

The dominant age-group of respondents was 30 to 49 years, age range that is still active and capable of receiving technical training in managing animals. A study conducted in rural villages of Western Kenya on indigenous pig management practices reported that farmers in pig production were of a similar age range [140]. Importantly, most respondents in this study had at least primary level education which creates an opportunity for the relevant authorities to train and disseminate appropriate technologies for improved and sustainable production of pig products. This is convenient especially in this situation where 51% of the sampled households state that farming is their main occupation.

Among the key parameters of low input pig production systems investigated, pig breed kept was the most important and almost every household (92%) in the study area kept local African genotype pigs. A few farmers kept a mix of local and cross or local and pure European breed pigs. This finding confirms that local pig genotypes remain predominant in rural areas despite numerous

calls to introduce exotic breeds for better outputs. It has been pointed out that lack of knowledge regarding different pig breeds, their growth properties and their ability to cope in local environments are among the most important impediments to breed improvement [141].

The predominant management system was tethering (71%) while free ranging was 27% with only 1.5% housing their pigs. For those practicing a mixture of systems, tethering was mainly during the day and when crops were still in the gardens while free-range was at night and almost all the time when there are no crops in the gardens that pigs could destroy. Therefore when the farmers think there are no crops that could be destroyed by pigs, they free-range them, which is a significant risk factor for disease spread. The distribution of pig-type kept in the households provides some insight into the factors that inform management decisions on what to stock and the producer's valuation of their stock. For example, nearly half of the pigs found in the households were weaners and only 25% were sows. This high proportion of weaners could be attributed to the fact that some farmers look at a pig as an easily-liquidated asset, in some ways comparable to operating a bank account. The weaner pigs category is the most marketable and is easily disposed of without having to wait for a market day or having to transport them to the market which would require obtaining a movement permit [141].

With regard to restocking during one-year period preceding the study, piglets dominated (72% of households) the number purchased with only 22% purchasing weaners. It is worth noting that Kenyan farmers were more engaged in the purchase of older categories of pigs than Ugandan farmers. This trend could be attributed to the higher consumption of pork in Uganda [142,143], leading to most of the pigs going to the market for slaughter rather than being sold to fellow pig farmers. On the source of pigs purchased, friends/relatives/ neighbours led with 47%. Although

this provides an ‘easy to reach’ source for pig restocking, the pig safety and health cannot be ascertained and therefore pose a great risk of ASF introduction on the farm. In-house breeding constituted only 18% of all the pigs on the farms. This low number can be attributed to the cost and risks involved in rearing a sow to maturity, yet purchasing of piglets that reach market weight easily, provides faster returns [141].

On the role of NAADS and other Non-Governmental Organizations in pig farming, it was surprising that close to 95% of the farmers did not buy pigs and/or piglets from these organizations or some other established breeders. Over 45% of households reported that NAADS’ played a primarily role in providing advisory services. Friends and household members too were a source of advice to 29.5% of the households but the least trusted, as a source of advice were the pig traders at 1.3%. The demonstrated lack of trust in pig traders is likely due to the fact that traders have previously been found to originate rumours of suspected diseases outbreak in order to set off an avalanche of pig sales by farmers at giveaway prices for fear of losing their animals [73].

Smallholder farmers in low input production systems are faced with several challenges. Farmers are faced with constraints such as high costs of feeds coupled with poor quality and unreliability of feed supplies. In an effort to cut production costs, pig farmers choose farming approaches that cost them as little as possible. For example, it was found that scavenging in the neighbourhood for food accounts for 40% of the feed intake. Household leftovers and swill (including by products from food processing and brew by-products) are also used to feed pigs in 40% of the farms surveyed and only 20% of the interviewed households used commercial feeds and only as a supplement to other sources of feed. On the other hand, given its relatively low cost, there were some farmers that relied almost entirely on either household leftovers/ crop residues from the farm

or forage for feed needs and majority of the farmers used some combination of at least two of the above sources [73,75,84].

The other approach that farmers use to mitigate risks and also reduce costs associated with rearing pigs was agistment. The proportion of farmers agisting pigs was 23% of which 69% kept some of their pig(s) with a separate pig keeping household and 31% agisted on farm (i.e., they kept some pigs in their own household that never belonged to them). Among the risks attributable to the cost-cutting husbandry systems is that of disease introduction and spread in pig populations. It was reported that free ranging pigs are capable of roaming as far away from the household as 4 km in search for food in a single night [15] and this could present a huge risk of disease introduction and spread. Agistment too, as does free-ranging, may pose its own risk of disease spread.

The high costs of farm inputs like feeds have perpetuated these practices since the only other (non-appealing) alternative is the selloff of their pigs at very low profits [16,75]. Besides cost of inputs, another important issue is pig health. In this context, farmers highlighted the inability to diagnose disease due to lack of veterinarians. Conflict with neighbors and market issues such as poor prices offered by pig traders (usually by under estimating the live weight of the pigs) were additional issues of concern. A further problem was market disruptions during suspected ASF outbreaks. These findings are in agreement with challenges identified in previous studies [75,140,144].

## 5.6 Conclusion

This study has highlighted the key parameters constraining low input pig production systems in the Kenya-Uganda border area. Factors such as the cost of inputs like feeds as well as farmers' awareness pertaining disease diagnosis training are known to impact the choice of husbandry practices. Therefore the most important area would be to improve the supply of pig feed which

would leverage adoption of husbandry practices designed to improve production efficiency and promote sustainable pork production to meet the increasing demand for animal protein from the increasing human population, as well as improving pig farmers' incomes. Better management systems could also have a significant impact on reducing pig diseases like African swine fever, thus mitigating animal health risks.

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# CHAPTER 6

## Spatial-Deterministic Model for the Transmission Dynamics of African swine fever virus

Mike B. Barongo[1], Richard P. Bishop[2], Eric M. Fèvre[3,4], Darryn L. Knobel[5], Amos Ssematimba[6]

*[1]Department of Academic Registrar, Makerere University, P.O. Box 7062, Kampala, Uganda*

*[2]Department of Veterinary Microbiology and Pathology, Washington State University, PO Box 647040, Pullman, WA.*

*[3]International Livestock Research Institute, P.O. Box 30709, Nairobi 00100, Kenya*

*[4]Institute of Infection and Global Health, University of Liverpool, Leahurst Campus, Neston, UK*

*[5]Current address: Center for Conservation Medicine and Ecosystem Health, Ross University School of Veterinary Medicine, P.O Box 334, Basseterre, St Kitts*

*[6]Department of Mathematics, Faculty of Science, Gulu University, P.O. Box 166, Gulu, Uganda*

In preparation

## 6.1 Abstract

African swine fever virus (ASFV) causes a highly contagious disease of domestic pigs in Africa and Europe. This virus spreads through aerosols and close contact with infectious pigs, as well as pig products, fomites and soft ticks. For improved control of the spread of the virus, knowledge on its spatial transmission is needed. A spatial-deterministic compartmental model for the epidemiology of ASFV was developed to simulate the effects of pig interaction on the spatial spread of the disease. The model tracks the spread of the disease across various villages from one village where it was introduced. The model was parameterised using data from existing literature and expert opinion. The results show that restricting pig interactions does not only contain an outbreak within the village of introduction but also helps create a window to allow for implementation of other control measures after the onset of the outbreak. The study recommends prompt interventions after onset of reported outbreaks and development of programmes that will enable smallholder pig farmers to access affordable and reliable pig feed which in turn may lead to improved compliance of farmers to animal confinement regulations.

**Keywords:** African swine fever virus, spatial-deterministic model, spatial transmission; mathematical modelling

## 6.2 Introduction

African swine fever (ASF) is one of the most devastating diseases in domestic pigs. It is caused by African swine fever virus (ASFV), a DNA virus of the Asfarviridae family. It results in acute haemorrhagic fever in susceptible animals with mortalities of up to 100% [65]. Currently, ASF has no treatment or vaccine and its control is based primarily on preventive sanitary measures, which

rely on knowledge of the epidemiological patterns [1,126]. This disease spreads through several pathways, including through aerosols and close contact with infectious pigs. Knowledge of its spatial transmission is therefore vital for the design of improved control measures. Modelling techniques that incorporate spatial aspects of the disease provide a useful approach to understand spatial transmission and the impact of various control measures.

Mathematical models are increasingly being used as valuable tools for studying the magnitude, duration and cost of disease outbreaks, and for comparing control strategies [23]. These models have been applied in the study of livestock diseases and play a fundamental role in aiding our understanding of underlying processes of disease transmission, factors driving epidemic behaviour and assessment of interventions on the epidemiology of the disease [22,24]. Models have been used to explain and communicate the fundamental principles of disease transmission. Insight gained through mathematical modelling can be valuable for the decision-making process in the management and control of livestock diseases like ASF.

There have been a few attempts in modelling ASF transmission dynamics for purposes of informing control strategies. Recently, Barongo et al. [25] conducted a modelling study to assess the effects of control measures and their timing on the burden of ASF in a pig herd. In another study [30], the same authors used outbreak field data to compute the basic reproductive number ($R_0$) for ASF. Iglesias et al. [120] recently conducted a study to estimate $R_0$ for ASF albeit in wild boars in the Russian Federation. On the role of modelling in informing policy, Halasa et al. [145], conducted a simulation study to assess the epidemiological and economic effects of an ASF epidemic in Denmark and predicted ASF-induced median direct costs and export losses of €12 and €349 million respectively. Spatial transmission of ASF has received limited attention in modelling

efforts to date. Recently, Mur et al. [146] conducted a modelling study to understand ASF infection dynamics in Sardinian pig farms. One of their aims was to identify geographic areas at highest risk of infection in Sardinia. They used a spatially explicit transmission model and concluded that ASF epidemiology and infection dynamics in Sardinia created a complex and multifactorial disease situation.

In this study, we demonstrate the use of spatial-deterministic models to reproduce the transmission dynamics of ASF in the context of smallholder pig farmers in East Africa. This was implemented using a deterministic compartmental model that simulated the epidemiology of ASFV in a homogenously mixing pig population within a village, modelled its transmission between villages using a probabilistic Monte Carlo function, and tracked its spread across villages. The study findings show that restricting pig interactions does not only contain an outbreak within the village of introduction but also helps create a window to allow for implementation of other control measures after the onset of the outbreak.

## 6.3 Methods

### 6.3.1 Model formulation and assumptions

In this study, we extend the susceptible, exposed, infected and resistant (SEIR) model to include a carrier state C, which comprises pigs that are capable of transmitting the disease at a lower rate, based on previous works [131,147]. The other compartments that make up the SEICR model are: the susceptible compartment (S) which contains individuals who do not have any type of immunity to the disease and can become infected upon contact with adequate infectious material; the exposed compartment (E) that contains individuals who have experienced adequate contact with an infective pig (such that transmission has occurred) but who are not yet infectious; the infectious

compartment (I) comprising individuals who have transited from the exposed compartment after the latent period and who are now infectious. A proportion of the infectious individuals who survive beyond the infectious period transit to the carrier compartment (C) while the infectious individuals who succumb to the disease are removed from the system through the Removed compartment (R).

Let S, E, I, C and R be the number of individuals in the susceptible, exposed, infectious, carrier and removed compartments at a given time. The total population at that time is given by N = S + E + I + C. Assuming a homogeneously mixing population of pigs within each village and further assuming that the residency time in the various states are exponentially distributed, the following disease and/or population related processes are considered: 1) there is recruitment of susceptible pigs into the village at a constant rate $\Lambda$ pigs per day through pig birth, purchases, gifts and loaning/agistment (pigs brought into the village and kept on behalf of someone else) during the period of the outbreak; 2) there is disease transmission at a per capita rate $\beta$ pigs per day; 3) once infected, exposed pigs spend a latent period $\sigma^{-1}$ days before becoming infectious, 4) once infectious, they remain so for a period $\gamma^{-1}$ days, after which a proportion $\rho$ of typically infectious pigs transits to carrier state while the rest (1- $\rho$ ) are removed from the system as a result of disease-induced death; 5) the infectiousness of carriers pigs is reduced by a factor $\varepsilon$; 6) carriers pigs remain so for an average period $\alpha^{-1}$ days and finally, 7) there is natural (non-ASF) mortality in all four compartments (S, E, I, C) at per capita rate $\mu$.

The processes are summarized in the compartmental diagram in Figure 6.1 and the ordinary differential equations for the SEICR model are presented in Equations 2.1. This model was adopted from [25] by incorporating the infection pressure from carrier pigs.

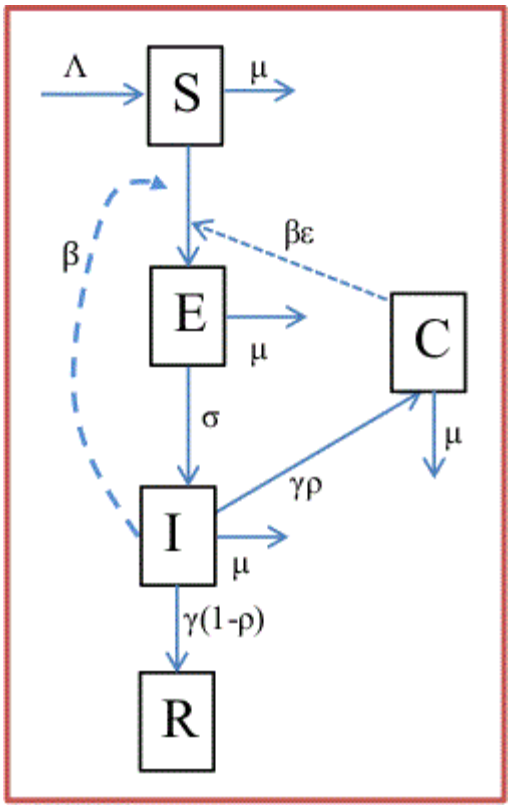


Fig. 6.1. Schematic representation of the SEICR Model for this study (Adopted from[25])

The above process is depicted by the following system of ordinary differential equations (ODEs) as:

$$\frac{dS}{dt} = \Lambda - \beta S\left(I + \varepsilon C\right) - \mu S,$$

$$\frac{dE}{dt} = \beta S\left(I + \varepsilon C\right) - (\sigma + \mu)E,$$

$$\frac{dI}{dt} = \sigma E - (\gamma + \mu)I,$$

$$\frac{dC}{dt} = \gamma\rho I - (\alpha + \mu)C,$$

$$\frac{dR}{dt} = \gamma(1-\rho)I + \alpha C \tag{2.1}$$

### 6.3.2 Qualitative analysis of the system

A qualitative analysis of the model was performed, which specifically entailed investigations of the existence and stability of the system's equilibrium states to infer the probable course of an infection introduced within the village. We use the analysis methods as described in [148], [149] and elsewhere. Briefly, let $(S^*, E^*, I^*, C^*)$ be the equilibrium states of the system described by the Equations (2.1). At an equilibrium state, we have $\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dC}{dt} = 0$. For establishment of the existence of equilibrium points, we note that, by definition, a trivial equilibrium state is that where all individuals go extinct in the village, while for the disease-free equilibrium, we require that all $E^* = I^* = C^* = 0$, and for the endemic equilibrium, at least one of these three variables should be above zero. Stability analyses are conducted based on the well-documented stability criteria analyses in dynamical systems theory [148,149].

### 6.3.3 Quantitative analysis: model formulation and assumptions

Under the quantitative analysis of the model, we used simulation techniques to achieve our goals. For the between-villages disease dynamics model, Nambuku Sub-location in Busia County in Kenya was randomly selected from the list of all administrative units that participated in an ASF epidemiology study on the Uganda-Kenya border in 2012-2013 [73]. Using ArcGIS software, a 5x5 cell grid was overlaid on Nambuku. The individual cells in Figure 2 are taken to represent the villages in Nambuku Sub-location. This grid overlay made it possible to define the SEICR transmission model in Equations (2.1) on each village and to track the spatial spread of the disease if it was introduced in one of the villages. A two-dimensional array (coupled lattice-based) was used to define this overlay grid in System dynamics software, Stella ver. 8.1.1. This method is based on those previously described by [150] and [151].

The grid is numbered from top left row-wise with the first cell as cell(1, 1) and the last bottom right cell as cell(5, 5). The cells (1, 1), (1, 2), (1, 5), (4, 1), (4, 5), (5, 1), (5, 4), (5, 5) are assumed to be outside the model boundary and are therefore not affected by the disease dynamics (see Fig 2). We assumed that cells are not infected by diagonal infectious neighbours and we modelled the within-village dynamics using the deterministic model (Equations (2.1)) but assuming a stochastic process for transmission between adjacent villages.

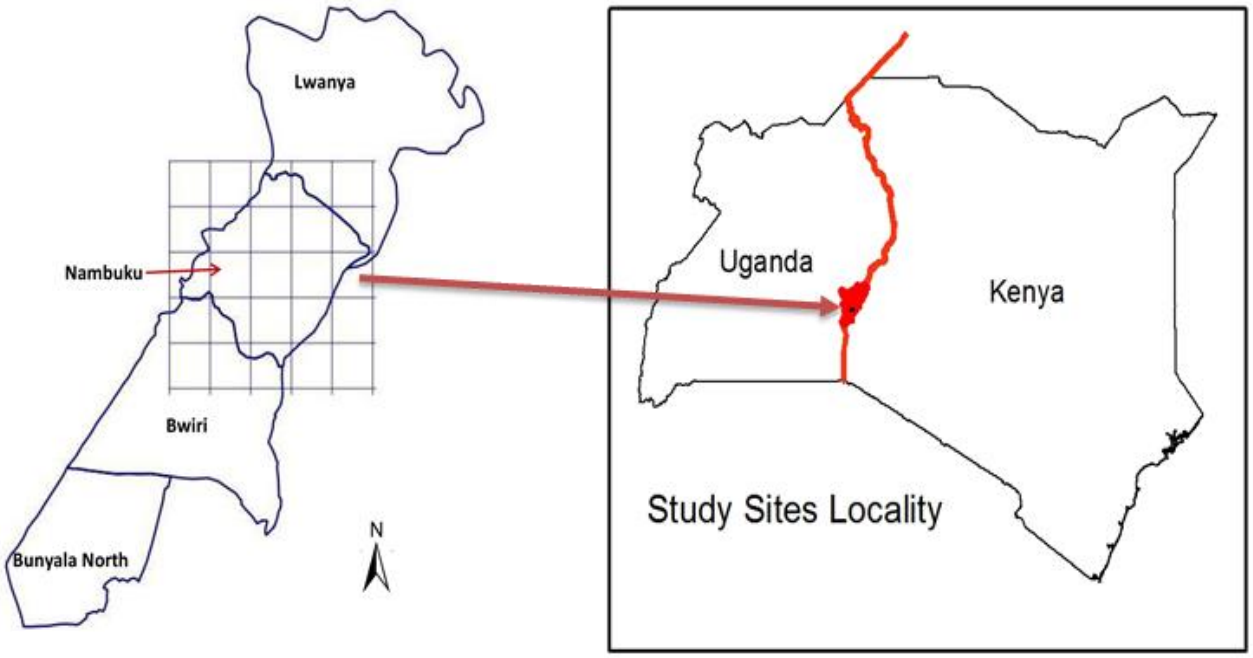


Fig. 6.2 Grid overlay on Nambuku Sub-location to represent the villages modelled

If **A** represents the system of ordinary differential Equations (2.1), then for an arbitrary village represented by C(i, j), the dynamics of the system will be governed by the equation:

$$A_{i,j} = \sum_{b=j-1}^{b=j+1} \sum_{a=i-1}^{a=i+1} f_{a,b}(x) * A_{a,b}$$

$$i, j >= 1,$$

where $f_{a,b}(x)$ is a System Dynamics Monte Carlo function that samples a random number equal to 1 for $x$ percent of the time in each iteration of the model. When the function returns 1, then this village C(i, j) is at risk of infection by any of the four villages adjacent to it (i.e. C(i-1, j), C(i, j-1), C(i, j+1) and C(i+1, j)) that are within the grid. The System Dynamics formulation is

given in appendix II and has also been previously presented by Neuwirth et al. [150]. The schematic representation of this model formulation is given in Fig 6.3.

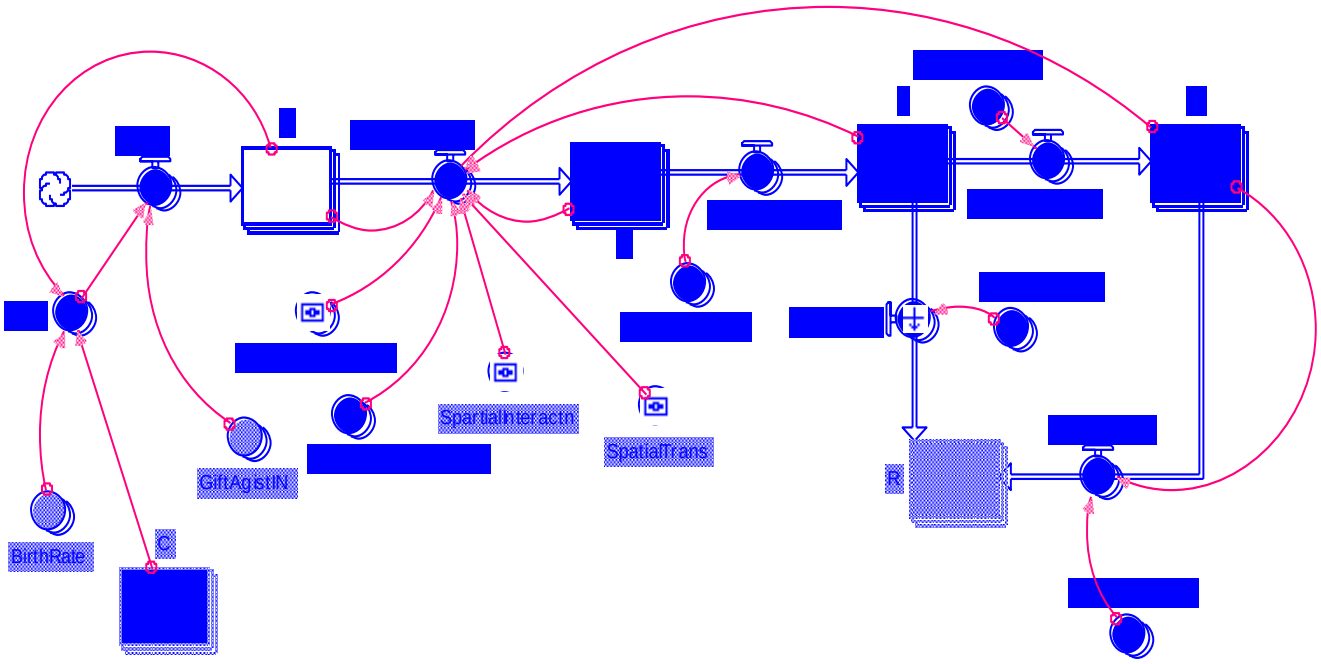


Fig. 6.3. Schematic representation of SEICR model in System Dynamics Software.

In Table 6.1, we map model parameters as defined in Equations (2.1) to their corresponding definitions in the System Dynamics formulation and also define the added parameters that relate to the between-village spatial formulation.

Table 6.1. Mapping the deterministic ordinary DE parameters and System Dynamics parameters

| **Parameter** | **DE formulation** | **System Dynamics formulation equivalent** | **Estimate and Citation** |
|---|---|---|---|
| Transmission rate (per day) | $\beta$ | Transmission rate | 0.3 [116] |
| Recruitment rate (per day) | $\Lambda$ | Birth | 0.001* |
| | | GiftAgistIN | Normal(2,1)* |

| | | | |
|---|---|---|---|
| Incubation rate (per day) | $\sigma$ | IncubationRate | 3 [116] |
| Infection Period (days) | $\gamma^{-1}$ | RecoveryTime | 3 [116] |
| Fraction dying | $\rho$ | Fraction dying | 0.8** |
| Duration in carrier state (days) | $\alpha^{-1}$ | Carrier duration | 45[39,116] |
| Infectivity reduction | $\varepsilon$ | Infectivity Reduction | 0.35* |
| Spatial Interaction between villages | | SpartialInteractn | 0.1** |
| Transmission on spatial contact | | SpatialTrans | 20%** |

*The estimates are from published literature and data collected during a cross-sectional study in the area [73,75]

**Parameters for scenario setting based on expert opinion and observations during field studies.

# 6.4 Results

### 6.4.1 Qualitative Analysis of the SEICR model

The ordinary differential equations model (Equation (2.1)) was analysed to identify the equilibrium states and investigate their stability. Due to analytical intractability, we analysed only the disease free equilibrium (DFE) state where there are no infectious, exposed or carriers (i.e. $S = S_0 = \Lambda/\mu$, $E^* = I^* = C^* = 0$) and while the properties of the endemic equilibrium state (i.e. either E, I or C >0) are often times inferred from the findings of the stability properties of the DFE. This inference is largely informed by the relationship between the stability conditions for the DFE and the parameter restrictions that ensure $R_0 < 1$ and the endemic equilibrium being stable if $R_0 > 1$ [148,149].

### 6.4.2 Existence of the trivial equilibrium state

For as long as the recruitment rate$\Lambda > 0$, the population of the pigs will never go extinct in the village, implying that, the trivial equilibrium point cannot exist.

### 6.4.3 Existence and stability of endemic equilibrium state

Following the methods described in [148] and [149], we first determine the Jacobian at an equilibrium state and assess the stability of the state using given criteria, for example the Routh-Hurwitz conditions or any other dynamical systems criteria based on the roots of the characteristic equation.

At the Disease free equilibrium state,

$$\frac{dS}{dt} = \Lambda - \beta S^* \left( I^* + \varepsilon C^* \right) - \mu S^* = 0,$$

$$\frac{dE}{dt} = \beta S^* \left( I^* + \varepsilon C^* \right) - (\sigma + \mu) E^* = 0,$$

$$\frac{dI}{dt} = \sigma E^* - (\gamma + \mu) I^* = 0,$$

$$\frac{dC}{dt} = \gamma \rho I^* - (\alpha + \mu) C^* = 0,$$

$$\frac{dR}{dt} = \gamma (1 - \rho) I^* + \alpha C^* = 0$$

At DFE, $I^*= E^*= C^*=0$ which implies $S^* = \Lambda/\mu$. Therefore the Jacobian matrix for the system is given as:

$$J_{DFE} = \begin{bmatrix} -\mu & 0 & -\frac{\beta\Lambda}{\mu} & -\frac{\beta\varepsilon\Lambda}{\mu} \\ 0 & -(\sigma+\mu) & \frac{\beta\Lambda}{\mu} & \frac{\beta\varepsilon\Lambda}{\mu} \\ 0 & \sigma & -(\gamma+\mu) & 0 \\ 0 & 0 & \gamma\rho & -(\alpha+\mu) \end{bmatrix}$$

and the characteristic equation from the matrix above is of fourth degree, derived by evaluating $\text{Det}(\mathbf{J}_{DFE} - r\mathbf{I}) = 0$ where r is an eigenvalue of the matrix. Upon simplification, the characteristic equation for the DFE is obtained as:

$$(\mathbf{r}+\mu)[\mathbf{r}^3 + (a+b+c)\mathbf{r}^2 + (ab+bc+ac+\sigma\beta S_0)\mathbf{r} + (abc + c\sigma\beta S_0 + \sigma\gamma\rho\beta\varepsilon S_0)] = 0$$

where $a = (\sigma+\mu),\ b = (\gamma+\mu),\ c = (\alpha+\mu)$.

The roots of this equation are the eigenvalues to the Jacobian matrix and we use them to determine the stability of DFE. Note that stability is ensured if all eigenvalues have a negative real part. In this case, the first solution is $r_1 = -\mu$ which is has a negative real part since $\mu > 0$. For the other three eigenvalues, we applied the Routh-Hurwitz conditions to determine the stability of this equilibrium point [148,149]. The Routh-Hurwitz conditions guarantee that all eigenvalues of a characteristic equation of the form $r^3 + a_1r^2 + a_2r + a_3 = 0,$ have negative real parts if and only if $a_1 > 0$, $a_3 > 0$ and $a_1a_2 - a_3 > 0$).

For the equation

$$\mathbf{r}^3 + \left(a+b+c\right)\mathbf{r}^2 + \left(ab+bc+ac+\sigma\beta S_0\right)\mathbf{r} + \left(abc+c\sigma\beta S_0+\sigma\gamma\rho\beta\varepsilon S_0\right) = 0,$$

we have that

$$a_1 = \left(a+b+c\right),\ a_2 = \left(ab+bc+ac+\sigma\beta S_0\right) \text{ and } a_3 = \left(abc+c\sigma\beta S_0+\sigma\gamma\rho\beta\varepsilon S_0\right).$$

In this case, all the parameters in expressions for $a_i$ (i=1, 2, 3) are positive; this satisfies the first two Routh-Hurwitz conditions. Thus, we only need to further investigate whether $a_1a_2 - a_3 > 0$ and from this requirement, we establish the condition for the stability of the DFE as

$[(\sigma+\mu)+(\gamma+\mu)+(\alpha+\mu)] \times [(\sigma+\mu)(\gamma+\mu)+(\gamma+\mu)(\alpha+\mu)+(\sigma+\mu)(\alpha+\mu)+\sigma\beta S_0] > [(\sigma+\mu)(\gamma+\mu)(\alpha+\mu)+(\alpha+\mu)\sigma\beta S_0+\sigma\gamma\rho\beta\varepsilon S_0]$.

Rearranging the stability condition DFE, we get;

$$a_1a_2 - a_3 > 0$$

$$\frac{a_1a_2}{a_3} > 1 \text{ or } \frac{a_3}{a_1a_2} < 1$$

Comparing to $R_0$ <1 for stability of DFE,

$$R_0 = \frac{a_3}{a_1a_2}$$

$$R_0 = \frac{[(\sigma+\mu)(\gamma+\mu)(\alpha+\mu)+(\alpha+\mu)\sigma\beta S_0+\sigma\gamma\rho\beta\varepsilon S_0]}{[(\sigma+\mu)+(\gamma+\mu)+(\alpha+\mu)]\times[(\sigma+\mu)(\gamma+\mu)+(\gamma+\mu)(\alpha+\mu)+(\sigma+\mu)(\alpha+\mu)+\sigma\beta S_0]}$$

If the expression is less than one, the DFE is stable and any disease introduction will not be established in the village.

### 6.4.4 Numerical Simulation of SEICR model

The model in Section 2.3 was calibrated using parameter estimates from Table 6.1. Due to a lack of empirical data, we assumed different degrees of interaction between pigs in adjacent villages for disease transmission to take place. It was further assumed that there is a 20% probability of contracting the disease as a result of between village interactions. Numerical simulations were performed for three different scenarios. For each scenario, we introduced an infection in the village represented by cell (3, 2) and we traced the spatial transmission of the disease to other villages over time under the different scenarios. We extracted results for the disease burden in cell (3, 2) as well as in the adjacent cell (3, 3) and a far off border cell (5, 4).

*Scenario 1: Assuming no pig interaction between villages*

Fig 6.4 shows the disease burden on cell (3, 2) and there is no spread to other cells because there is no pig interaction allowed between villages. Therefore the infection is contained within the village but it progresses to an endemic state.

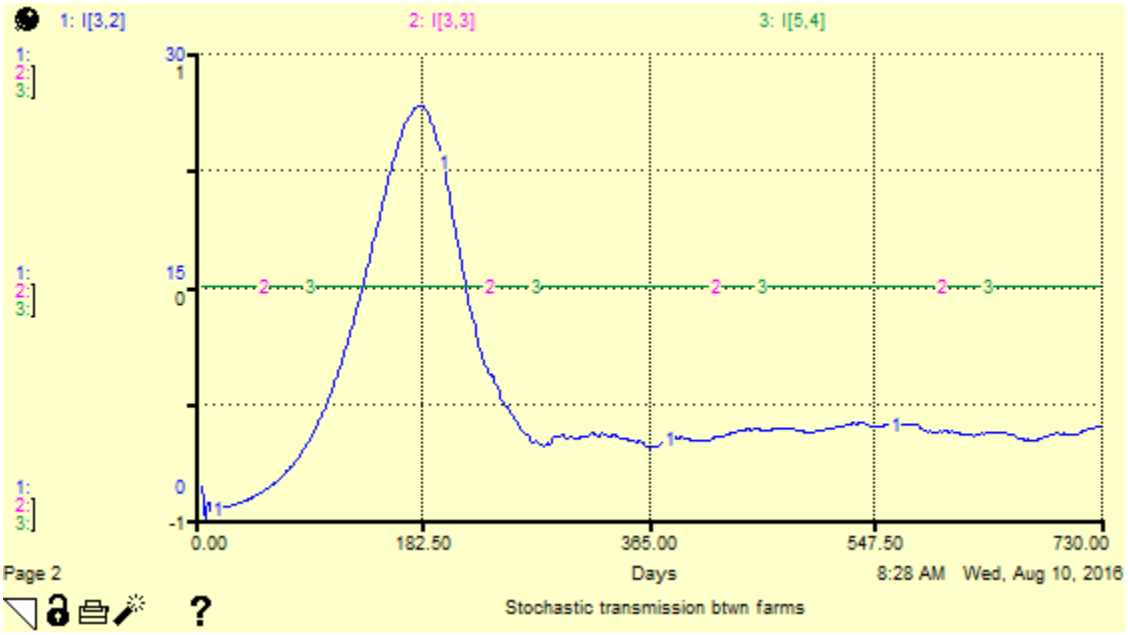


Fig. 6.4. Simulation run for the dynamics in the village represented by cell (3, 3) assuming no interaction between villages

*Scenario 2: Pig interaction between villages set at 10%*

Next we modelled a scenario where the infection is introduced into the same village, cell (3, 2) and the interaction with pigs from neighbouring villages is set at 10%. The results showed a gradual but lagged spread of the infection to adjacent villages as shown in Fig 6.5. The graphical estimate of the lag between epidemic curves for two adjacent villages (cells) is 30 days.

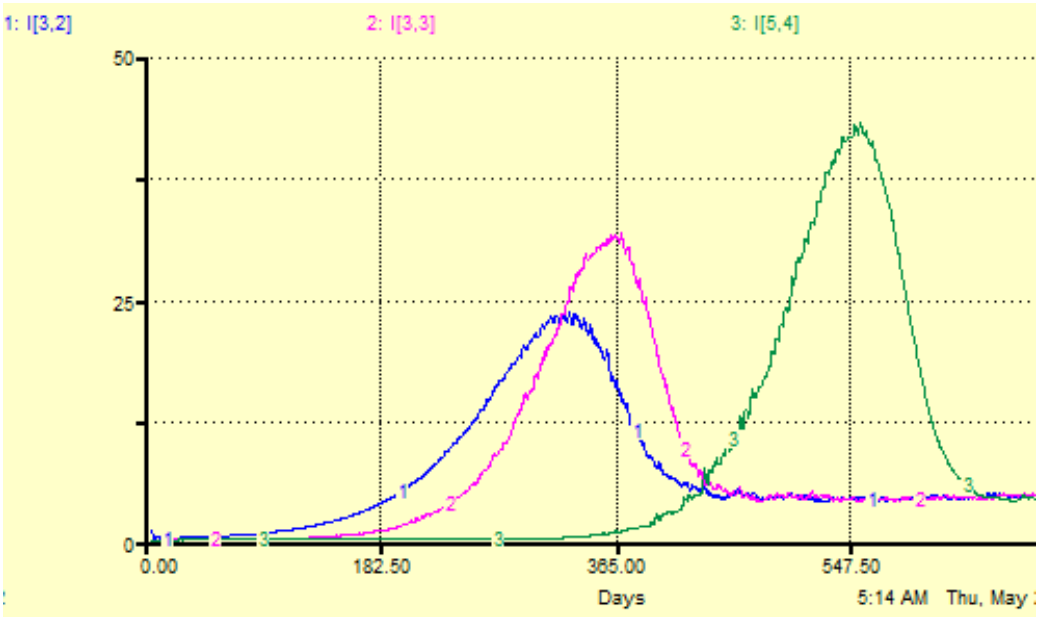


Fig. 6.5. Infection burden in villages represented by cells (3, 2), (3, 3) and (5, 4) assuming a 10% between-village interaction

*Scenario 3: Pig interaction between villages set at 30%*

When the interaction between pigs of adjacent villages was increased from 10% to 30%, the epidemic curves developed marked spikes and the lag between adjacent villages was almost wiped away as shown in figure 6.6.

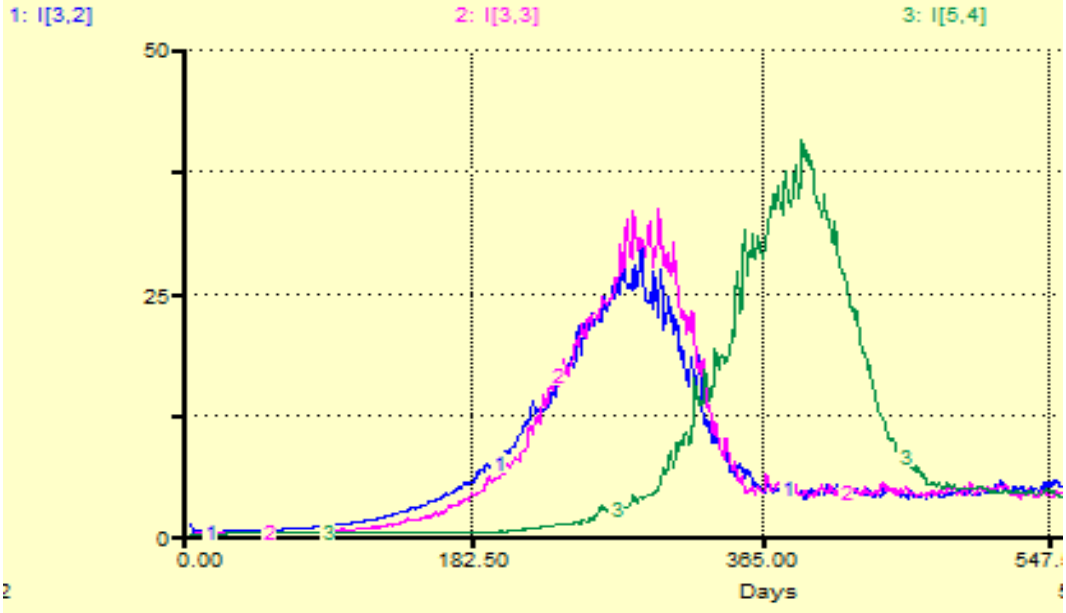


Fig. 6.6. Infection burden in villages represented by cells (3, 2), (3, 3) and (5, 4) assuming a 30% between-village interaction

## 6.5 Discussion

We formulated a deterministic model to characterize the transmission of ASFV within a village and probabilistic spatial spread of the virus as a result of pig interaction between villages. Since there is currently no treatment or vaccine for ASF, control is dependent on preventing contact between domestic pigs and sources of the virus [2]. We assessed the effect of herd interaction between villages on the disease burden at a village level. The results showed that restricting pig interaction between villages creates a lag (time from when the outbreak in the first village is first

'detectable' to the time of the first case in the next village) of 30 days before the start of an outbreak in the neighbouring village and this lag may accord outbreak response teams an opportunity to contain the outbreak with minimal damage.

The model incorporated a carrier state, which caters for pigs that have been exposed to the virus but recovered from the disease. Previous studies have shown the presence of African swine fever virus in sub-clinical, chronically infected or recovered pigs [52,53]. These carrier pigs are thought to be responsible for the persistence of the virus thereby extending circulation of ASFV within the herd [32]. It is possible that repeated ASF outbreaks may not necessarily be due to carrier pigs reactivating to fully blown infectiousness but rather dependent on pork processing and handling procedures/activities (i.e. slaughter slabs, pork joints, use of swill and food leftovers) that involve these carrier pigs that have persistent virions in blood or tissues [32].

We derived an expression for $R_0$ based on the dynamics of ASF. This expression can be used to quantify the force of invasion of ASF for a particular outbreak if the key parameters are known. One of the key parameter in the expression for $R_0$ is $\beta$. An effective way to reduce $\beta$ has been found to be the implementation of biosecurity measures that minimize direct pig to pig contact such as tethering, housing pigs and isolation of new pig recruits on the farm[73,75]. On the other hand, previous studies have emphasized quick isolation, quarantining and/or culling of infected animals as a means to curb disease transmission [73,152]. However, in developing countries early detection and isolation remain big challenges, let alone culling without compensation mechanisms in place. In many rural areas where smallholder pig farmers live, veterinary officials lack the basics facilities to confirm suspected ASF cases, the means to reach these rural areas in time.

Besides the deterministic model, we also used a spatial-deterministic model to investigate the spatial spread of ASFV between villages in the study area resulting from interactions of pigs from neighbouring villages. A previous study on the spatial ecology of free-ranging domestic pigs in western Kenya found that pigs could roam up to 3 km away from the household in a single night [15]. There are a number of pathways through which ASF is spread which include direct contact between domestic pigs, contact with infectious material, contact with wild suids and exposure to soft ticks [1]. Transmission via these routes is exacerbated by pig movements due to the free-ranging production system practiced in the study area making it difficult to eliminated between-village pig contacts and exposures. The results of this study show that by applying pig movement restrictions between villages, it is possible to prevent ASF from spreading from village to village once it is accidentally introduced in one of the villages. A closely related study by Mur et al., (2017)[146] concluded that spatial transmission of the pathogen further complicated the dynamics of the disease, thereby rendering control efforts insufficient.

To further understand the effect of restricting pig movements/interaction on the spatial spread of ASF, we allowed for up to 30% interaction. The simulation results showed that the disease could spread to adjacent villages after some time. This lag in disease invasion to the neighbouring village provides a window of 30 days. With proactive and incentivized veterinary authorities, this window provides an opportunity to contain the outbreak within a village before it progresses to ravage the next village. However, if pig interaction of up to 30% is allowed, results show that this level of pig interaction can greatly curtail the effectiveness of the instituted control measures. This finding though with a relatively relaxed time interval, agrees with findings of Barongo *et al.* [25] who found that the optimal timing for the introduction of control measures was within 14 days of the onset of an outbreak.

The findings of this study strengthen the case for authorities to implement animal movement restrictions for a reasonable period and confinement in order to curb repeated outbreaks of ASF. This may require sensitizing pig farmers and getting their buy in on how long movement restrictions will be in place. In events of reported ASF outbreaks, quick implementation of some biosecurity measures such as tethering or use of thorny vegetation to provide reasonable pig-proof pens and avoiding use of swill are recommended in order to contain the disease within the area of the reported outbreak. We strongly recommend that governments (local or national) should develop programmes that focus on tackling the factors that prevent small pig farmers from adopting more modern husbandry methods such as lack of awareness and the required investment. Top on the list are the identification of locally available safe feeds and building pig pens to house the pigs.

# CHAPTER 7

## Summarising Discussion and Recommendations

## 7.1 Summarising Discussion

We have modelled the transmission dynamics of ASF disease in East Africa in the context of smallholder farming systems, with a view to understanding ASFV epidemiology in order to optimise control strategies that minimise the spread of the virus and mitigate social-economic impact on small-scale pig farmers. Several modelling tools were used to generate insight into ASF transmission mechanisms, probable causes of persistence in domestic pig populations and most importantly evaluation of various control strategies [25,30]. Additionally, we analysed key parameters of low input system husbandry practices among the pig producers on the Kenya-Uganda border that could facilitate ASF spread among pigs in the study area.

We used three methods to estimate $R_0$ from ASF epidemic data in a predominantly free-ranging pig production system in northern Uganda. These were the nearest infectious neighbour, the doubling time and the SI model methods. The mean estimates for $R_0$ ranged between 1.58 and 3.24. Considering that the estimates from all the methods are above the critical threshold of one, the disease is bound to be established when it is introduced in naïve pig populations, consistent with the observed persistence of disease. These estimates may have been affected by underestimation of infectious period and under-reporting of outbreaks, leading to underestimation of $R_0$ [102]; however, for the purposes of this analysis, we assumed that all outbreaks during the study period were reported. This assumption is supported by the fact that, in the study area, farmers were primed to report outbreaks due to the on-going research activities.

Though $R_0$ is known to be sensitive to production system, contact structure and environmental factors, our $R_0$ estimates from the nearest infectious neighbour and doubling time methods were in close agreement with those of Gulenkin et al. [99] and Iglesias et al. [120] who estimated $R_0$ to range from 2 to 3 and 1.58 respectively at the between-farm level. Comparison of estimates from

different studies and geographical areas should be made with caution. The true value of $R_0$ for most epidemics may be difficult to quantify for a number of reasons such as unknown source of outbreaks, inconsistent reporting time-scales, complication of obtaining good contact tracing data from the multiple indirect routes of infection, farming systems and the role of human behaviour in transmission of ASFV. When reporting is on a higher resolution time-scale, the parameter estimates are better and more generalizable. With high-resolution data, we would recommend the use of the doubling time method to estimate a more realistic figure for $R_0$. Despite these uncertainties, empirical data from epidemics can be a valuable source for estimating epidemiological parameters. This was the first attempt to estimate $R_0$ from field data for ASF in a predominantly free-ranging production system in an endemic region of East Africa.

In order to formalize questions relating to ASF epidemiology and the impact of control measures, we formulated a stochastic compartmental mathematical model to assess the potential impact of different intervention scenarios on disease burden. We investigated the benefits of compelling smallholder farmers to adopt and intensify biosecurity measures during ASF outbreaks, in order to minimize the disease burden. When compared to the baseline scenario of no intervention, our model predicted that if biosecurity measures are enhanced within a fortnight from the time ASF is first suspected, the disease burden can be significantly reduced. However, this typically takes too long given current reporting mechanisms and infrastructure. We believe this is achievable if resources are made available to veterinary services to support and guide the implementation of these interventions and control so that it is not left entirely in the hands of resource-poor pig farmers, which is currently the prevailing scenario in our study area on the Uganda-Kenya border.

Three protection levels of hypothetical vaccines and three intervention time points for their use were assessed using our models. The greatest vaccine impact was predicted at a simulated vaccine protection level of 70% when implemented at day 14 post-epidemic onset, although if continuous vaccination of susceptible new recruits that enter the system is included, this is predicted to reduce the disease burden by as much as 82%. The most effective simulated intervention strategy (with 91% of deaths averted) is a combination of pulse vaccination (protecting 70% of the pigs at risk) together with enhanced biosecurity measures implemented by day 14. Unfortunately, vaccines are still a long way from being commercially available, even in the developed world let alone accessible and affordable to rural pig farmers in sub-Saharan Africa. Initial attempts to develop experimental live attenuated vaccines against ASFV have achieved in some instances good level of protection against homologous infection. However the current constraint is lack of suitable cells for enabling scale up for commercial production at a realistic cost in relation to the value of the animals [128].

There was a clear pattern between the disease burden and timing of intervention, whereby delayed implementation of control reduced the positive impact of intervention scenarios. This prediction, although intuitive, emphasizes the importance of early intervention in managing ASF epidemics, and our modelling approach provides a means to determine timescales beyond which interventions were of limited value in controlling ASF. Although our model predicts a combination of vaccination and enhanced biosecurity as the best intervention scenario, the only currently feasible strategy is implementation of enhanced biosecurity measures. We therefore recommend intensification of active surveillance and use of pen-side diagnostic assays, where these are available, for rapid detection and confirmation of ASF to allow for timely implementation of enhanced biosecurity. Given the importance of the time to implementation of biosecurity

measures, we recommend that veterinary services in ASF outbreak risk areas work to educate farmers on the most feasible basic biosecurity measures to prevent ASF and additional measures to adopt, as soon as ASFV infection is suspected [73].

Outbreaks of pig diseases such as ASF are known to be entirely driven by the pig production systems that allow pigs access to sources of infection in the area of interest. We assessed key parameters of low input production systems among the pig producers on the Kenya-Uganda border to identify practices that could contribute to propagation of ASF spread among pigs in the area. It was found that pigs are mainly looked after by women and that the dominant age-group of respondents was 30 to 49 years. Most of the respondents had at least primary level education, which creates an opportunity for training on best practices and disseminating appropriate technologies for improved and sustainable pig production.

Among the key parameters of low input pig production systems investigated, the pig genotype and phenotype were the most important. Therefore knowledge of different pig types, which may ultimately be defined as breeds, their growth properties and their ability to cope in local environments are among the most important factors for fostering increased pig production [153], although there are arguments against it in the absence of markets for more and better quality pig products. Secondly, the type of management system used was found to be another key element. Although tethering was the predominant type of management, farmers stated that tethering was mainly used during the day and when crops were still in the gardens, but pigs would be allowed to free-range at night and almost all the time when there were no crops in the gardens.

It was noted that pig restocking and the source of pigs purchased were also key parameters in determining how rapidly and over what range ASF could spread. Recent studies have shown a

high prevalence of ASFV in slaughter animals in the absence of reported outbreaks [52]. When farmers purchase pigs from pig traders or live animal markets, there is a high possibility of introducing the disease on the farm. It is therefore vital that NAADS and NGOs play a key role in providing farmers with piglets from ASF-free sources or help to support local pig breeders, employing efficient husbandry practices.

Smallholder farmers are faced with several challenges ranging from high costs of feeds coupled with poor quality and unreliability of feed supplies. In an effort to cut production costs, pig farmers choose farming approaches that cost them as little as possible. For example, allowing pigs to scavenge in the neighbourhood for food accounts for 40% of the feed intake while swill and commercial feeds are the remaining sources of feed. On the other hand, given its relatively low cost, there were some farmers that relied almost entirely on either household leftovers/ crop residues from the farm or forage for feed needs. The majority of the farmers used some combination of at least two of the above sources.

The other approach that farmers use to mitigate risks and also reduce costs associated with rearing pigs was agistment, which involves sharing of animals between close neighbours or relatives. Agistment may pose its own risk of disease spread due to pig movement between households. The high costs of farm inputs, particularly feeds have perpetuated these practices since the only other (non-appealing) alternative is the sale of pigs at very low prices [16,75]. Besides cost of inputs, another important issue is pig health. The farmers highlighted their inability to diagnose disease in the absence of veterinarians. Market issues such as poor prices offered by pig traders (usually by under-estimating the live weight of the pigs) were of additional concern. A further problem was market disruptions during suspected ASF outbreaks [75,140,144].

We formulated a deterministic model to characterize the transmission of ASFV within a village and probabilistic spatial spread of the virus as a result of pig interaction between villages. We assessed the effect of herd interactions between villages on the disease burden at a village level. The results showed that restricting pig interaction between villages creates a lag of 30 days before the start of an outbreak in the neighbouring village and this lag may afford outbreak response teams an opportunity to contain the outbreak with minimal damage.

There are a number of pathways through which ASF is spread, including direct contact between domestic pigs, contact with infectious material, contact with wild suids and exposure to soft ticks [1]. Transmission via these routes is exacerbated by pig movements due to the free-ranging production system practiced in the study area, making it difficult to eliminate between-village pig contacts and exposures. The modelling research frameworks described in this thesis emphasize that there is lack of quantitative data for most of these parameters and basic information still needs to be collected to provide more realistic predictions. However, one clear conclusion is that by applying pig movement restrictions between villages significantly lowered the chance of spreading to the next village.

The findings of this study strengthen the case for authorities to implement animal movement restrictions and confinement in order to curb repeated outbreaks of ASF. In the event of reported ASF outbreaks, quick implementation of some basic biosecurity measures such as tethering/confinement and avoiding use of swill are recommended in order to contain the disease within the area of the reported outbreak. We strongly recommend that governments (local or national) and NGOs should develop programmes that focus on tackling the factors that inhibit small pig farmers entering the potentially profitable pig market. Key areas include biosecurity

education, innovative solutions to providing reliable low cost pig feed and loans to subsidize the building of pig pens to house the pigs.

## 7.2 Recommendations

Based on the findings of this research, we recommend the following measures and further studies in order to implement these findings or further this work. We therefore recommend:

1. Gender-targeted training on improved husbandry practices through strengthened advisory services, focused on women's groups. Governments need to develop the entire pig value chain in particular supporting input suppliers to work with researchers to support production of cost-effective pig feeds.
2. More epidemiological studies be designed to confirm whether ticks are involved in maintaining the virus in the study area, because confining the pigs in housing with the ticks is not helpful in preventing infection.
3. Intensification of active surveillance and use of pen-side diagnostic assays for rapid detection and confirmation of ASF to allow for timely implementation of enhanced biosecurity.
4. Continued research on the development of a vaccine against ASFV to allow for deployment of a hybrid intervention strategy.
5. Early implementation of animal movement restrictions and confinement especially during suspected ASF outbreaks.

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# Appendix I

## Philosophical underpinning of $R_0$

Consider a 'closed' and 'naive' pig population that is in one way or the other invaded by a disease-causing organism. For simplicity of derivations, the following assumptions are made:

- There is homogeneous mixing of individuals
- That all subsequent infections are a result of contacts between susceptible (S) and the infectious (I) individuals
- That every infection results into either death or immunity (R)

Under these assumptions, the following state-transition SIR (density-dependent) model is formulated:

$$\frac{dS}{dt} = -\beta SI, \qquad (1)$$

$$\frac{dI}{dt} = \beta SI - \gamma I, \qquad (2)$$

$$\frac{dR}{dt} = \gamma I. \qquad (3)$$

Subject to the initial conditions: $S(0) = S_0 > 0$; $I(0) = I_0 > 0$ and $R(0) = 0$. (4)

Where β is the transmission term (a product of probability of transmission and contact rate) and is the removal rate ($1/\gamma$ = average infectious period). Give the initial conditions and parameters, we are interested in knowing if the infection will spread or not and how it will develop over time. Initially, it follows from equation (2), that:

$$\frac{dI}{dt} = (\beta S_0 - \gamma) I_0$$

If $\frac{dI}{dt} > 0$, $(\beta S_0 - \gamma) > 0$, then I(t) starts to increase and there is likelihood of an epidemic. This implies $\beta S_0 > \gamma$ or $\beta S_0 / \gamma > 1$. The expression $\beta S_0 / \gamma$ is the reproduction number ($R_0$) of the infection. If $\frac{dI}{dt} \leq 0$, then $(\beta S_0 - \gamma) \leq 0$, then I(t) remains below $I_0$ implying the epidemic does not occur. If $R_0 < 1$, the disease dies out, whereas if $R_0 \geq 1$, the disease persists in the population (Li et al., 2011).

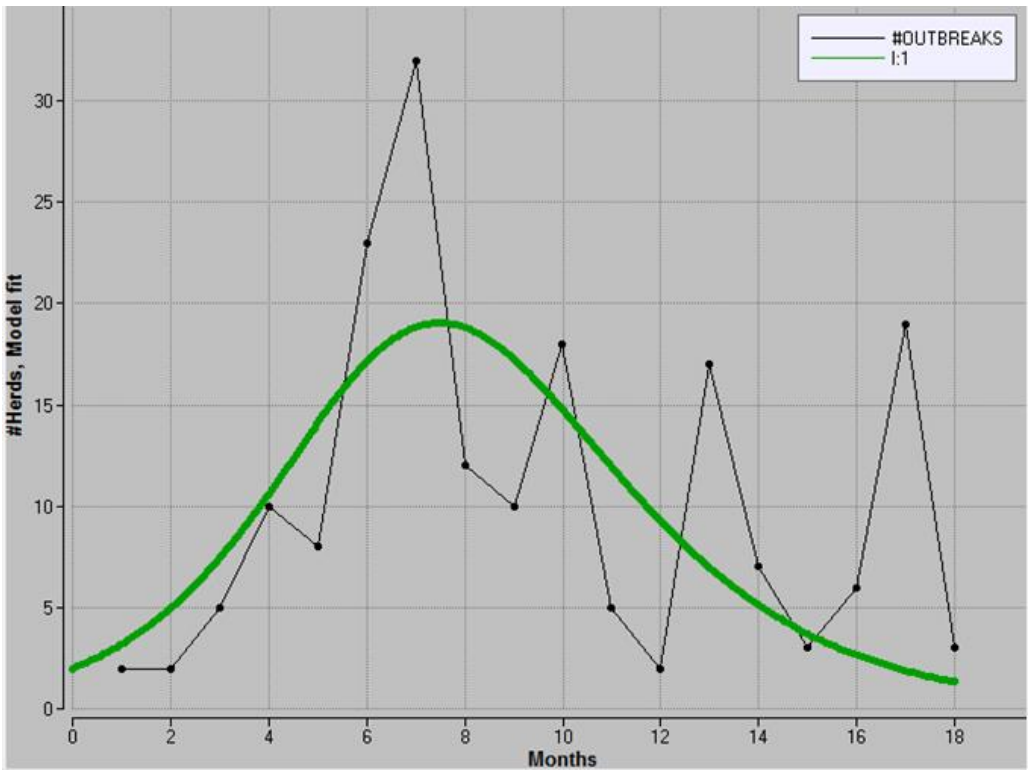


Fig S2: SIR model used to simulate outbreak data of African swine fever, Gulu District, Uganda, April 2010 - November 2011

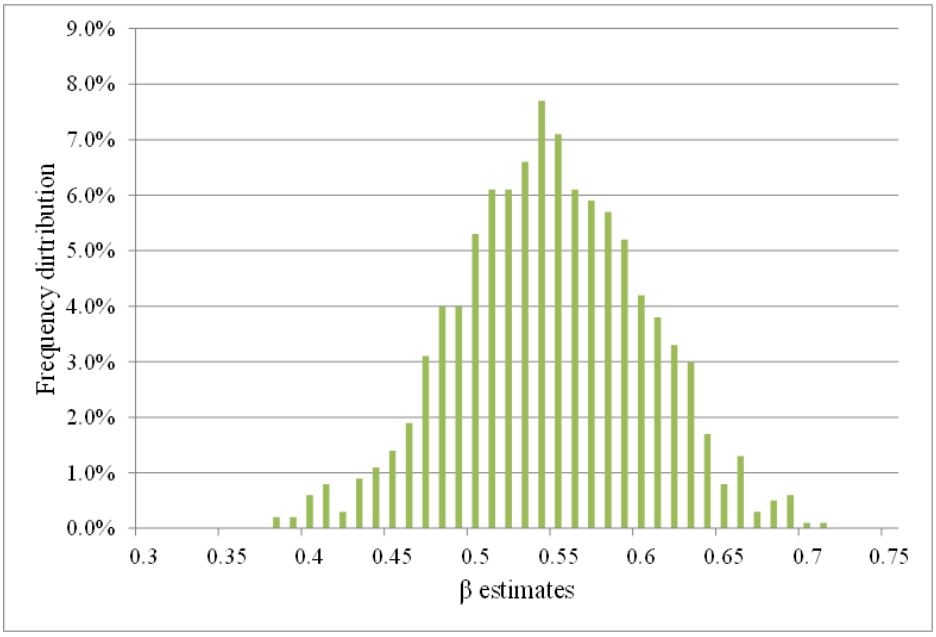


Fig S3: Distribution of bootstrapped monthly transmission rate coefficient β estimates

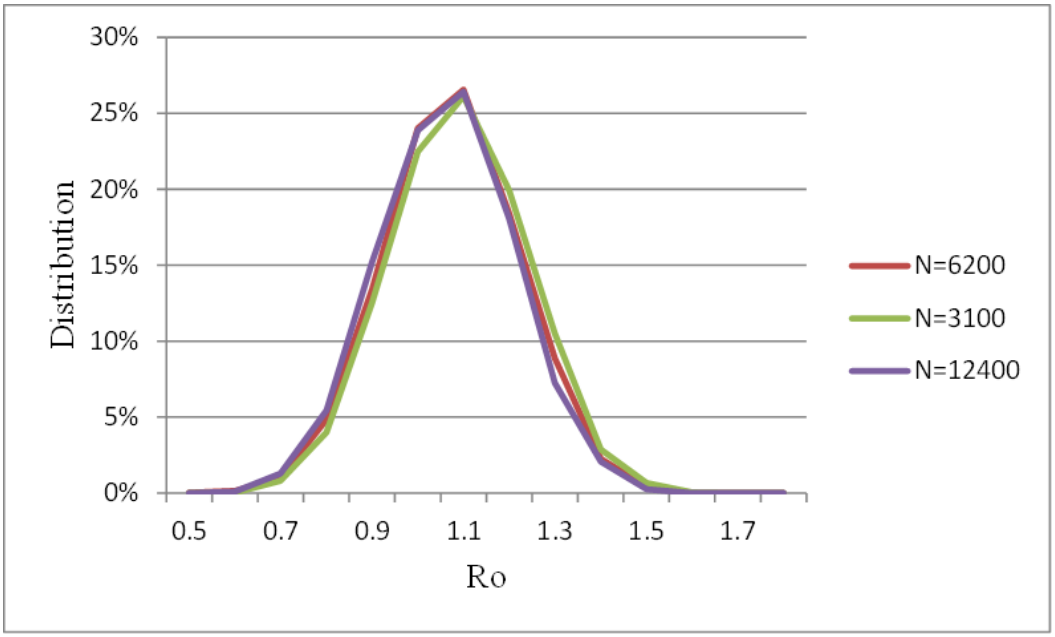


Fig S4: Sensitivity of basic reproduction number $R_0$ to variation in initial number of herds

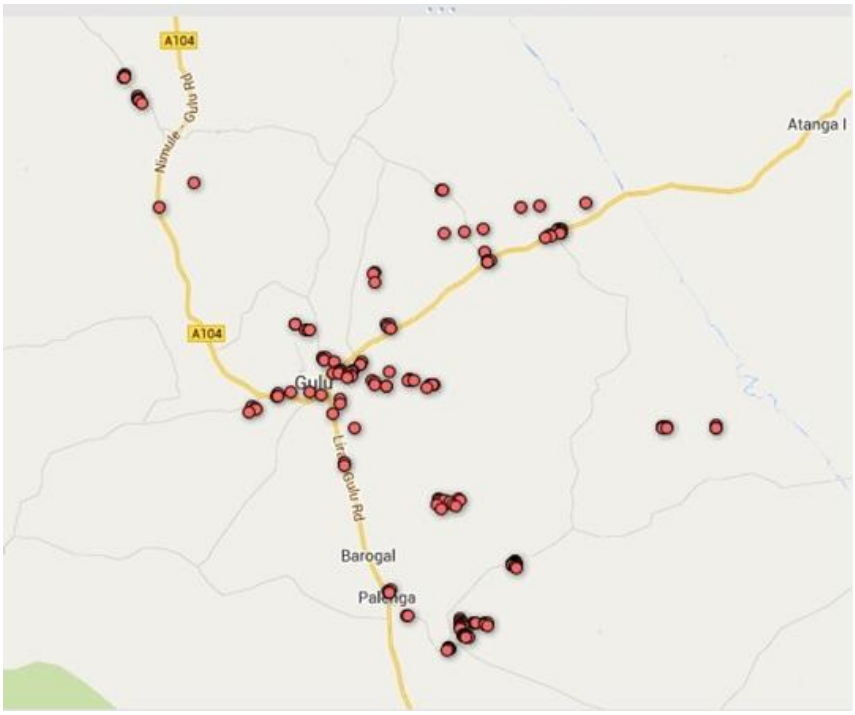


Fig S5. Spatial distribution of ASF infected herds (April 2010 - November 2011)

## **Appendix II**: System Dynamics formulation

C[West,North](t) = C[West,North](t - dt) + (Recovery_Rate[West,North] -

CDeathRate[West,North]) * dt

INIT C[West,North] = 0

INFLOWS:

Recovery_Rate[West,North] = CONVEYOR OUTFLOW

TRANSIT TIME = RecoveryTime[West,North]

OUTFLOWS:

CDeathRate[West,North] = C[West,North]/CarrierDuration[West,North]

E[West,North](t) = E[West,North](t - dt) + (Infection_Rate[West,North] -

Infectious_Rate[West,North]) * dt

INIT E[West,North] = 0

INFLOWS:

Infection_Rate[1,1] =

Transmission_Rate[1,1]*S[1,1]*I[1,1]/(S[1,1]+I[1,1]+E[1,1]+C[1,1])+Infectivity_Reduction[1,1

]*C[1,1]*S[1,1]*Transmission_Rate[1,1]/(S[1,1]+I[1,1]+E[1,1]+C[1,1])+0*SpartialInteractn*M

ONTECARLO(SpatialTrans)

Infection_Rate[1,2] =

Transmission_Rate[1,2]*S[1,2]*I[1,2]/(S[1,2]+I[1,2]+E[1,2]+C[1,2])+Infectivity_Reduction[1,2

]*C[1,2]*S[1,2]*Transmission_Rate[1,2]/(S[1,2]+I[1,2]+E[1,2]+C[1,2])

+0*SpartialInteractn*MONTECARLO(SpatialTrans)

Infection_Rate[1,3] =

Transmission_Rate[1,3]*S[1,3]*I[1,3]/(S[1,3]+I[1,3]+E[1,3]+C[1,3])+Infectivity_Reduction[1,3

]*C[1,3]*S[1,3]*Transmission_Rate[1,3]/(S[1,3]+I[1,3]+E[1,3]+C[1,3])
+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[1,3]*S[1,3]*I[2,3]/(S[1,3]+I[1,3]+E[1,3]+C[1,3])+Infectivity_Reduction[1,3]*C[2,3]*S[1,3]*Transmission_Rate[1,3]/(S[1,3]+I[1,3]+E[1,3]+C[1,3]))
+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[1,3]*S[1,3]*I[1,4]/(S[1,3]+I[1,3]+E[1,3]+C[1,3])+Infectivity_Reduction[1,3]*C[1,4]*S[1,3]*Transmission_Rate[1,3]/(S[1,3]+I[1,3]+E[1,3]+C[1,3]))
Infection_Rate[1,4] =
Transmission_Rate[1,4]*S[1,4]*I[1,4]/(S[1,4]+I[1,4]+E[1,4]+C[1,4])+Infectivity_Reduction[1,4]*C[1,4]*S[1,4]*Transmission_Rate[1,4]/(S[1,4]+I[1,4]+E[1,4]+C[1,4]) +
SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[1,4]*S[1,4]*I[1,3]/(S[1,4]+I[1,4]+E[1,4]+C[1,4])+Infectivity_Reduction[1,4]*C[1,3]*S[1,4]*Transmission_Rate[1,4]/(S[1,4]+I[1,4]+E[1,4]+C[1,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[1,4]*S[1,4]*I[2,4]/(S[1,4]+I[1,4]+E[1,4]+C[1,4])+Infectivity_Reduction[1,4]*C[2,4]*S[1,4]*Transmission_Rate[1,4]/(S[1,4]+I[1,4]+E[1,4]+C[1,4]))
Infection_Rate[1,5] =
Transmission_Rate[1,5]*S[1,5]*I[1,5]/(S[1,5]+I[1,5]+E[1,5]+C[1,5])+Infectivity_Reduction[1,5]*C[1,5]*S[1,5]*Transmission_Rate[1,5]/(S[1,5]+I[1,5]+E[1,5]+C[1,5])+0*SpartialInteractn*MONTECARLO(SpatialTrans)
Infection_Rate[2,1] =
Transmission_Rate[2,1]*S[2,1]*I[2,1]/(S[2,1]+I[2,1]+E[2,1]+C[2,1])+Infectivity_Reduction[2,1]*C[2,1]*S[2,1]*Transmission_Rate[2,1]/(S[2,1]+I[2,1]+E[2,1]+C[2,1])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,1]*S[2,1]*I[2,2]/(S[2,1]+I[2,1]+E[2,1]+C[2,

1])+Infectivity_Reduction[2,1]*C[2,2]*S[2,1]*Transmission_Rate[2,1]/(S[2,1]+I[2,1]+E[2,1]+C[2,1]))+MONTECARLO(SpatialTrans)*SpartialInteractn*(Transmission_Rate[2,1]*S[2,1]*I[3,1]/(S[2,1]+I[2,1]+E[2,1]+C[2,1])+Infectivity_Reduction[2,1]*C[3,1]*S[2,1]*Transmission_Rate[2,1]/(S[2,1]+I[2,1]+E[2,1]+C[2,1]))

Infection_Rate[2,2] =

Transmission_Rate[2,2]*S[2,2]*I[2,2]/(S[2,2]+I[2,2]+E[2,2]+C[2,2])+Infectivity_Reduction[2,2]*C[2,2]*S[2,2]*Transmission_Rate[2,2]/(S[2,2]+I[2,2]+E[2,2]+C[2,2])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,2]*S[2,2]*I[2,2]/(S[2,2]+I[2,1]+E[2,2]+C[2,2])+Infectivity_Reduction[2,2]*C[2,1]*S[2,2]*Transmission_Rate[2,2]/(S[2,2]+I[2,2]+E[2,2]+C[2,2]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,2]*S[2,2]*I[2,3]/(S[2,2]+I[2,2]+E[2,2]+C[2,2])+Infectivity_Reduction[2,2]*C[2,3]*S[2,2]*Transmission_Rate[2,2]/(S[2,2]+I[2,2]+E[2,2]+C[2,2]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,2]*S[2,2]*I[3,2]/(S[2,2]+I[2,2]+E[2,2]+C[2,2])+Infectivity_Reduction[2,2]*C[3,2]*S[2,2]*Transmission_Rate[2,2]/(S[2,2]+I[2,2]+E[2,2]+C[2,2]))

Infection_Rate[2,3] =

Transmission_Rate[2,3]*S[2,3]*I[2,3]/(S[2,3]+I[2,3]+E[2,3]+C[2,3])+Infectivity_Reduction[2,3]*C[2,3]*S[2,3]*Transmission_Rate[2,3]/(S[2,3]+I[2,3]+E[2,3]+C[2,3])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,3]*S[2,3]*I[2,2]/(S[2,3]+I[2,3]+E[2,3]+C[2,3])+Infectivity_Reduction[2,3]*C[2,2]*S[2,3]*Transmission_Rate[2,3]/(S[2,3]+I[2,3]+E[2,3]+C[2,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,3]*S[2,3]*I[1,3]/(S[2,3]+I[2,3]+E[2,3]+C[2,3])+Infectivity_Reduction[2,3]*C[1,3]*S[2,3]*Transmission_Rate[2,3]/(S[2,3]+I[2,3]+E[2,3]+C[2,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,3]*S[2,3]*I[2,4]/(S[2,3]+I[2,3]+E[2,3]+C[2,3])+Infectivity_Reduction[2,3]*C[2,4]

*S[2,3]*Transmission_Rate[2,3]/(S[2,3]+I[2,3]+E[2,3]+C[2,3]))+MONTECARLO(SpatialTrans)*SpartialInteractn*(Transmission_Rate[2,3]*S[2,3]*I[3,3]/(S[2,3]+I[2,3]+E[2,3]+C[2,3])+Infectivity_Reduction[2,3]*C[3,3]*S[2,3]*Transmission_Rate[2,3]/(S[2,3]+I[2,3]+E[2,3]+C[2,3]))

Infection_Rate[2,4] =

Transmission_Rate[2,4]*S[2,4]*I[2,4]/(S[2,4]+I[2,4]+E[2,4]+C[2,4])+Infectivity_Reduction[2,4]*C[2,4]*S[2,4]*Transmission_Rate[2,4]/(S[2,4]+I[2,4]+E[2,4]+C[2,4])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,4]*S[2,4]*I[2,3]/(S[2,4]+I[2,4]+E[2,4]+C[2,4])+Infectivity_Reduction[2,4]*C[2,3]*S[2,4]*Transmission_Rate[2,4]/(S[2,4]+I[2,4]+E[2,4]+C[2,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,4]*S[2,4]*I[1,4]/(S[2,4]+I[2,4]+E[2,4]+C[2,4])+Infectivity_Reduction[2,4]*C[1,4]*S[2,4]*Transmission_Rate[2,4]/(S[2,4]+I[2,4]+E[2,4]+C[2,4]))+MONTECARLO(SpatialTrans)*SpartialInteractn*(Transmission_Rate[2,4]*S[2,4]*I[2,5]/(S[2,4]+I[2,4]+E[2,4]+C[2,4])+Infectivity_Reduction[2,4]*C[2,5]*S[2,4]*Transmission_Rate[2,4]/(S[2,4]+I[2,4]+E[2,4]+C[2,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,4]*S[2,4]*I[3,4]/(S[2,4]+I[2,4]+E[2,4]+C[2,4])+Infectivity_Reduction[2,4]*C[3,4]*S[2,4]*Transmission_Rate[2,4]/(S[2,4]+I[2,4]+E[2,4]+C[2,4]))

Infection_Rate[2,5] =

Transmission_Rate[2,5]*S[2,5]*I[2,5]/(S[2,5]+I[2,5]+E[2,5]+C[2,5])+Infectivity_Reduction[2,5]*C[2,5]*S[2,5]*Transmission_Rate[2,5]/(S[2,5]+I[2,5]+E[2,5]+C[2,5])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,5]*S[2,5]*I[2,4]/(S[2,5]+I[2,5]+E[2,5]+C[2,5])+Infectivity_Reduction[2,5]*C[2,4]*S[2,5]*Transmission_Rate[2,5]/(S[2,5]+I[2,5]+E[2,5]+C[2,5]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,5]*S[2,5]*I[3,5]/(S[2,5]+I[2,5]+E[2,5]+C[2,5])+Infectivity_Reduction[2,5]*C[3,5]*S[2,5]*Transmission_Rate[2,5]/(S[2,5]+I[2,5]+E[2,5]+C[2,5]))

Infection_Rate[3,1] =

Transmission_Rate[3,1]*S[3,1]*I[3,1]/(S[3,1]+I[3,1]+E[3,1]+C[3,1])+Infectivity_Reduction[3,1]*C[3,1]*S[3,1]*Transmission_Rate[3,1]/(S[3,1]+I[3,1]+E[3,1]+C[3,1])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,1]*S[3,1]*I[2,1]/(S[3,1]+I[3,1]+E[3,1]+C[3,1])+Infectivity_Reduction[3,1]*C[2,1]*S[3,1]*Transmission_Rate[3,1]/(S[3,1]+I[3,1]+E[3,1]+C[3,1]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,1]*S[3,1]*I[3,2]/(S[3,1]+I[3,1]+E[3,1]+C[3,1])+Infectivity_Reduction[3,1]*C[3,2]*S[3,1]*Transmission_Rate[3,1]/(S[3,1]+I[3,1]+E[3,1]+C[3,1]))

Infection_Rate[3,2] =

Transmission_Rate[3,2]*S[3,2]*I[3,2]/(S[3,2]+I[3,2]+E[3,2]+C[3,2])+Infectivity_Reduction[3,2]*C[3,2]*S[3,2]*Transmission_Rate[3,2]/(S[3,2]+I[3,2]+E[3,2]+C[3,2])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,2]*S[3,2]*I[3,1]/(S[3,2]+I[3,2]+E[3,2]+C[3,2])+Infectivity_Reduction[3,2]*C[3,1]*S[3,2]*Transmission_Rate[3,2]/(S[3,2]+I[3,2]+E[3,2]+C[3,2]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,2]*S[3,2]*I[2,2]/(S[3,2]+I[3,2]+E[3,2]+C[3,2])+Infectivity_Reduction[3,2]*C[2,2]*S[3,2]*Transmission_Rate[3,2]/(S[3,2]+I[3,2]+E[3,2]+C[3,2]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,2]*S[3,2]*I[3,3]/(S[3,2]+I[3,2]+E[3,2]+C[3,2])+Infectivity_Reduction[3,2]*C[3,3]*S[3,2]*Transmission_Rate[3,2]/(S[3,2]+I[3,2]+E[3,2]+C[3,2]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,2]*S[3,2]*I[4,2]/(S[3,2]+I[3,2]+E[3,2]+C[3,2])+Infectivity_Reduction[3,2]*C[4,2]*S[3,2]*Transmission_Rate[3,2]/(S[3,2]+I[3,2]+E[3,2]+C[3,2]))

Infection_Rate[3,3] =

Transmission_Rate[3,3]*S[3,3]*I[3,3]/(S[3,3]+I[3,3]+E[3,3]+C[3,3])+Infectivity_Reduction[3,3]*C[3,3]*S[3,3]*Transmission_Rate[3,3]/(S[3,3]+I[3,3]+E[3,3]+C[3,3])+SpartialInteractn*MO

NTECARLO(SpatialTrans)*(Transmission_Rate[3,3]*S[3,3]*I[3,2]/(S[3,3]+I[3,3]+E[3,3]+C[3,3])+Infectivity_Reduction[3,3]*C[3,2]*S[3,3]*Transmission_Rate[3,3]/(S[3,3]+I[3,3]+E[3,3]+C[3,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,3]*S[3,3]*I[2,3]/(S[3,3]+I[3,3]+E[3,3]+C[3,3])+Infectivity_Reduction[3,3]*C[2,3]*S[3,3]*Transmission_Rate[3,3]/(S[3,3]+I[3,3]+E[3,3]+C[3,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,3]*S[3,3]*I[3,4]/(S[3,3]+I[3,3]+E[3,3]+C[3,3])+Infectivity_Reduction[3,3]*C[3,4]*S[3,3]*Transmission_Rate[3,3]/(S[3,3]+I[3,3]+E[3,3]+C[3,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,3]*S[3,3]*I[4,3]/(S[3,3]+I[3,3]+E[3,3]+C[3,3])+Infectivity_Reduction[3,3]*C[4,3]*S[3,3]*Transmission_Rate[3,3]/(S[3,3]+I[3,3]+E[3,3]+C[3,3]))

Infection_Rate[3,4] =

Transmission_Rate[3,4]*S[3,4]*I[3,4]/(S[3,4]+I[3,4]+E[3,4]+C[3,4])+Infectivity_Reduction[3,4]*C[3,4]*S[3,4]*Transmission_Rate[3,4]/(S[3,4]+I[3,4]+E[3,4]+C[3,4])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,4]*S[3,4]*I[3,3]/(S[3,4]+I[3,4]+E[3,4]+C[3,4])+Infectivity_Reduction[3,4]*C[3,3]*S[3,4]*Transmission_Rate[3,4]/(S[3,4]+I[3,4]+E[3,4]+C[3,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,4]*S[3,4]*I[2,4]/(S[3,4]+I[3,4]+E[3,4]+C[3,4])+Infectivity_Reduction[3,4]*C[2,4]*S[3,4]*Transmission_Rate[3,4]/(S[3,4]+I[3,4]+E[3,4]+C[3,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,4]*S[3,4]*I[3,5]/(S[3,4]+I[3,4]+E[3,4]+C[3,4])+Infectivity_Reduction[3,4]*C[3,5]*S[3,4]*Transmission_Rate[3,4]/(S[3,4]+I[3,4]+E[3,4]+C[3,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,4]*S[3,4]*I[4,4]/(S[3,4]+I[3,4]+E[3,4]+C[3,4])+Infectivity_Reduction[3,4]*C[4,4]*S[3,4]*Transmission_Rate[3,4]/(S[3,4]+I[3,4]+E[3,4]+C[3,4]))

Infection_Rate[3,5] =

Transmission_Rate[3,5]*S[3,5]*I[3,5]/(S[3,5]+I[3,5]+E[3,5]+C[3,5])+Infectivity_Reduction[3,5

]*C[3,5]*S[3,5]*Transmission_Rate[3,5]/(S[3,5]+I[3,5]+E[3,5]+C[3,5])+SpartialInteractn*MO
NTECARLO(SpatialTrans)*(Transmission_Rate[3,5]*S[3,5]*I[3,4]/(S[3,5]+I[3,5]+E[3,5]+C[3,
5])+Infectivity_Reduction[3,5]*C[3,4]*S[3,5]*Transmission_Rate[3,5]/(S[3,5]+I[3,5]+E[3,5]+C
[3,5]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,5]*S[3,5]*I[2,5
]/(S[3,5]+I[3,5]+E[3,5]+C[3,5])+Infectivity_Reduction[3,5]*C[2,5]*S[3,5]*Transmission_Rate[
3,5]/(S[3,5]+I[3,5]+E[3,5]+C[3,5]))

Infection_Rate[4,1] =

Transmission_Rate[4,1]*S[4,1]*I[4,1]/(S[4,1]+I[4,1]+E[4,1]+C[4,1])+Infectivity_Reduction[4,1
]*C[4,1]*S[4,1]*Transmission_Rate[4,1]/(S[4,1]+I[4,1]+E[4,1]+C[4,1])+0*SpartialInteractn*M
ONTECARLO(SpatialTrans)

Infection_Rate[4,2] =

Transmission_Rate[4,2]*S[4,2]*I[4,2]/(S[4,2]+I[4,2]+E[4,2]+C[4,2])+Infectivity_Reduction[4,2
]*C[4,2]*S[4,2]*Transmission_Rate[4,2]/(S[4,2]+I[4,2]+E[4,2]+C[4,2])+SpartialInteractn*MO
NTECARLO(SpatialTrans)*(Transmission_Rate[4,2]*S[4,2]*I[3,2]/(S[4,2]+I[4,2]+E[4,2]+C[4,
2])+Infectivity_Reduction[4,2]*C[3,2]*S[4,2]*Transmission_Rate[4,2]/(S[4,2]+I[4,2]+E[4,2]+C
[4,2]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[4,2]*S[4,2]*I[4,3
]/(S[4,2]+I[4,2]+E[4,2]+C[4,2])+Infectivity_Reduction[4,2]*C[4,3]*S[4,2]*Transmission_Rate[
4,2]/(S[4,2]+I[4,2]+E[4,2]+C[4,2]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmi
ssion_Rate[4,2]*S[4,2]*I[5,2]/(S[4,2]+I[4,2]+E[4,2]+C[4,2])+Infectivity_Reduction[4,2]*C[5,2]
*S[4,2]*Transmission_Rate[4,2]/(S[4,2]+I[4,2]+E[4,2]+C[4,2]))

Infection_Rate[4,3] =

Transmission_Rate[4,3]*S[4,3]*I[4,3]/(S[4,3]+I[4,3]+E[4,3]+C[4,3])+Infectivity_Reduction[4,3
]*C[4,3]*S[4,3]*Transmission_Rate[4,3]/(S[4,3]+I[4,3]+E[4,3]+C[4,3])+SpartialInteractn*MO

NTECARLO(SpatialTrans)*(Transmission_Rate[4,3]*S[4,3]*I[4,2]/(S[4,3]+I[4,3]+E[4,3]+C[4,3])+Infectivity_Reduction[4,3]*C[4,2]*S[4,3]*Transmission_Rate[4,3]/(S[4,3]+I[4,3]+E[4,3]+C[4,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[4,3]*S[4,3]*I[3,3]/(S[4,3]+I[4,3]+E[4,3]+C[4,3])+Infectivity_Reduction[4,3]*C[3,3]*S[4,3]*Transmission_Rate[4,3]/(S[4,3]+I[4,3]+E[4,3]+C[4,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[4,3]*S[4,3]*I[4,4]/(S[4,3]+I[4,3]+E[4,3]+C[4,3])+Infectivity_Reduction[4,3]*C[4,4]*S[4,3]*Transmission_Rate[4,3]/(S[4,3]+I[4,3]+E[4,3]+C[4,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[4,3]*S[4,3]*I[5,3]/(S[4,3]+I[4,3]+E[4,3]+C[4,3])+Infectivity_Reduction[4,3]*C[5,3]*S[4,3]*Transmission_Rate[4,3]/(S[4,3]+I[4,3]+E[4,3]+C[4,3]))

Infection_Rate[4,4] =

Transmission_Rate[4,4]*S[4,4]*I[4,4]/(S[4,4]+I[4,4]+E[4,4]+C[4,4])+Infectivity_Reduction[4,4]*C[4,4]*S[4,4]*Transmission_Rate[4,4]/(S[4,4]+I[4,4]+E[4,4]+C[4,4])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[4,4]*S[4,4]*I[4,3]/(S[4,4]+I[4,4]+E[4,4]+C[4,4])+Infectivity_Reduction[4,4]*C[4,3]*S[4,4]*Transmission_Rate[4,4]/(S[4,4]+I[4,4]+E[4,4]+C[4,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[4,4]*S[4,4]*I[3,4]/(S[4,4]+I[4,4]+E[4,4]+C[4,4])+Infectivity_Reduction[4,4]*C[3,4]*S[4,4]*Transmission_Rate[4,4]/(S[4,4]+I[4,4]+E[4,4]+C[4,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[4,4]*S[4,4]*I[5,4]/(S[4,4]+I[4,4]+E[4,4]+C[4,4])+Infectivity_Reduction[4,4]*C[5,4]*S[4,4]*Transmission_Rate[4,4]/(S[4,4]+I[4,4]+E[4,4]+C[4,4]))

Infection_Rate[4,5] =

Transmission_Rate[4,5]*S[4,5]*I[4,5]/(S[4,5]+I[4,5]+E[4,5]+C[4,5])+Infectivity_Reduction[4,5]*C[4,5]*S[4,5]*Transmission_Rate[4,5]/(S[4,5]+I[4,5]+E[4,5]+C[4,5])+0*SpartialInteractn*MONTECARLO(SpatialTrans)

Infection_Rate[5,1] =

Transmission_Rate[5,1]*S[5,1]*I[5,1]/(S[5,1]+I[5,1]+E[5,1]+C[5,1])+Infectivity_Reduction[5,1]*C[5,1]*S[5,1]*Transmission_Rate[5,1]/(S[5,1]+I[5,1]+E[5,1]+C[5,1])+0*SpartialInteractn*MONTECARLO(SpatialTrans)

Infection_Rate[5,2] =

Transmission_Rate[5,2]*S[5,2]*I[5,2]/(S[5,2]+I[5,2]+E[5,2]+C[5,2])+Infectivity_Reduction[5,2]*C[5,2]*S[5,2]*Transmission_Rate[5,2]/(S[5,2]+I[5,2]+E[5,2]+C[5,2])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[5,2]*S[5,2]*I[4,2]/(S[5,2]+I[5,2]+E[5,2]+C[5,2])+Infectivity_Reduction[5,2]*C[4,2]*S[5,2]*Transmission_Rate[5,2]/(S[5,2]+I[5,2]+E[5,2]+C[5,2]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[5,2]*S[5,2]*I[5,3]/(S[5,2]+I[5,2]+E[5,2]+C[5,2])+Infectivity_Reduction[5,2]*C[5,3]*S[5,2]*Transmission_Rate[5,2]/(S[5,2]+I[5,2]+E[5,2]+C[5,2]))

Infection_Rate[5,3] =

Transmission_Rate[5,3]*S[5,3]*I[5,3]/(S[5,3]+I[5,3]+E[5,3]+C[5,3])+Infectivity_Reduction[5,3]*C[5,3]*S[5,3]*Transmission_Rate[5,3]/(S[5,3]+I[5,3]+E[5,3]+C[5,3])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[5,3]*S[5,3]*I[5,2]/(S[5,3]+I[5,3]+E[5,3]+C[5,3])+Infectivity_Reduction[5,3]*C[5,2]*S[5,3]*Transmission_Rate[5,3]/(S[5,3]+I[5,3]+E[5,3]+C[5,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[5,3]*S[5,3]*I[4,3]/(S[5,3]+I[5,3]+E[5,3]+C[5,3])+Infectivity_Reduction[5,3]*C[4,3]*S[5,3]*Transmission_Rate[5,3]/(S[5,3]+I[5,3]+E[5,3]+C[5,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[5,3]*S[5,3]*I[5,4]/(S[5,3]+I[5,3]+E[5,3]+C[5,3])+Infectivity_Reduction[5,3]*C[5,4]*S[5,3]*Transmission_Rate[5,3]/(S[5,3]+I[5,3]+E[5,3]+C[5,3]))

Infection_Rate[5,4] =

Transmission_Rate[5,4]*S[5,4]*I[5,4]/(S[5,4]+I[5,4]+E[5,4]+C[5,4])+Infectivity_Reduction[5,4]*C[5,4]*S[5,4]*Transmission_Rate[5,4]/(S[5,4]+I[5,4]+E[5,4]+C[5,4])

+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[5,4]*S[5,4]*I[5,3]/(S[5,4]+I[5,4]+E[5,4]+C[5,4])+Infectivity_Reduction[5,4]*C[5,3]*S[5,4]*Transmission_Rate[5,4]/(S[5,4]+I[5,4]+E[5,4]+C[5,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[5,4]*S[5,4]*I[4,4]/(S[5,4]+I[5,4]+E[5,4]+C[5,4])+Infectivity_Reduction[5,4]*C[4,4]*S[5,4]*Transmission_Rate[5,4]/(S[5,4]+I[5,4]+E[5,4]+C[5,4]))

Infection_Rate[5,5] =

Transmission_Rate[5,5]*S[5,5]*I[5,5]/(S[5,5]+I[5,5]+E[5,5]+C[5,5])+Infectivity_Reduction[5,5]*C[5,5]*S[5,5]*Transmission_Rate[5,5]/(S[5,5]+I[5,5]+E[5,5]+C[5,5])+0*SpartialInteractn*MONTECARLO(SpatialTrans)

OUTFLOWS:

Infectiuos_Rate[West,North] = CONVEYOR OUTFLOW

TRANSIT TIME = IncubationRate[West,North]

I[1,1](t) = I[1,1](t - dt) + (Infectiuos_Rate[1,1] - Recovery_Rate[1,1] - DeathRate[1,1]) * dt

INIT I[1,1] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[1,2](t) = I[1,2](t - dt) + (Infectiuos_Rate[1,2] - Recovery_Rate[1,2] - DeathRate[1,2]) * dt

INIT I[1,2] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[1,3](t) = I[1,3](t - dt) + (Infectiuos_Rate[1,3] - Recovery_Rate[1,3] - DeathRate[1,3]) * dt

INIT I[1,3] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[1,4](t) = I[1,4](t - dt) + (Infectiuos_Rate[1,4] - Recovery_Rate[1,4] - DeathRate[1,4]) * dt

INIT I[1,4] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[1,5](t) = I[1,5](t - dt) + (Infectiuos_Rate[1,5] - Recovery_Rate[1,5] - DeathRate[1,5]) * dt

INIT I[1,5] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[2,1](t) = I[2,1](t - dt) + (Infectiuos_Rate[2,1] - Recovery_Rate[2,1] - DeathRate[2,1]) * dt

INIT I[2,1] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[2,2](t) = I[2,2](t - dt) + (Infectiuos_Rate[2,2] - Recovery_Rate[2,2] - DeathRate[2,2]) * dt

INIT I[2,2] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[2,3](t) = I[2,3](t - dt) + (Infectiuos_Rate[2,3] - Recovery_Rate[2,3] - DeathRate[2,3]) * dt

INIT I[2,3] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[2,4](t) = I[2,4](t - dt) + (Infectiuos_Rate[2,4] - Recovery_Rate[2,4] - DeathRate[2,4]) * dt

INIT I[2,4] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[2,5](t) = I[2,5](t - dt) + (Infectiuos_Rate[2,5] - Recovery_Rate[2,5] - DeathRate[2,5]) * dt

INIT I[2,5] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[3,1](t) = I[3,1](t - dt) + (Infectiuos_Rate[3,1] - Recovery_Rate[3,1] - DeathRate[3,1]) * dt

INIT I[3,1] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[3,2](t) = I[3,2](t - dt) + (Infectiuos_Rate[3,2] - Recovery_Rate[3,2] - DeathRate[3,2]) * dt

INIT I[3,2] = 2

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[3,3](t) = I[3,3](t - dt) + (Infectiuos_Rate[3,3] - Recovery_Rate[3,3] - DeathRate[3,3]) * dt

INIT I[3,3] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[3,4](t) = I[3,4](t - dt) + (Infectiuos_Rate[3,4] - Recovery_Rate[3,4] - DeathRate[3,4]) * dt

INIT I[3,4] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[3,5](t) = I[3,5](t - dt) + (Infectiuos_Rate[3,5] - Recovery_Rate[3,5] - DeathRate[3,5]) * dt

INIT I[3,5] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[4,1](t) = I[4,1](t - dt) + (Infectiuos_Rate[4,1] - Recovery_Rate[4,1] - DeathRate[4,1]) * dt

INIT I[4,1] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[4,2](t) = I[4,2](t - dt) + (Infectiuos_Rate[4,2] - Recovery_Rate[4,2] - DeathRate[4,2]) * dt

INIT I[4,2] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[4,3](t) = I[4,3](t - dt) + (Infectiuos_Rate[4,3] - Recovery_Rate[4,3] - DeathRate[4,3]) * dt

INIT I[4,3] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[4,4](t) = I[4,4](t - dt) + (Infectiuos_Rate[4,4] - Recovery_Rate[4,4] - DeathRate[4,4]) * dt

INIT I[4,4] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[4,5](t) = I[4,5](t - dt) + (Infectiuos_Rate[4,5] - Recovery_Rate[4,5] - DeathRate[4,5]) * dt

INIT I[4,5] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[5,1](t) = I[5,1](t - dt) + (Infectiuos_Rate[5,1] - Recovery_Rate[5,1] - DeathRate[5,1]) * dt

INIT I[5,1] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[5,2](t) = I[5,2](t - dt) + (Infectiuos_Rate[5,2] - Recovery_Rate[5,2] - DeathRate[5,2]) * dt

INIT I[5,2] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[5,3](t) = I[5,3](t - dt) + (Infectiuos_Rate[5,3] - Recovery_Rate[5,3] - DeathRate[5,3]) * dt

INIT I[5,3] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[5,4](t) = I[5,4](t - dt) + (Infectiuos_Rate[5,4] - Recovery_Rate[5,4] - DeathRate[5,4]) * dt

INIT I[5,4] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

I[5,5](t) = I[5,5](t - dt) + (Infectiuos_Rate[5,5] - Recovery_Rate[5,5] - DeathRate[5,5]) * dt

INIT I[5,5] = 0

TRANSIT TIME = varies

INFLOW LIMIT = INF

CAPACITY = INF

INFLOWS:

Infectiuos_Rate[West,North] = CONVEYOR OUTFLOW

TRANSIT TIME = IncubationRate[West,North]

OUTFLOWS:

Recovery_Rate[West,North] = CONVEYOR OUTFLOW

TRANSIT TIME = RecoveryTime[West,North]

DeathRate[West,North] = LEAKAGE OUTFLOW

LEAKAGE FRACTION = Fraction_dying[West,North]

NO-LEAK ZONE = 20.8333%

R[West,North](t) = R[West,North](t - dt) + (DeathRate[West,North] +

CDeathRate[West,North]) * dt

INIT R[West,North] = 0

INFLOWS:

DeathRate[West,North] = LEAKAGE OUTFLOW

LEAKAGE FRACTION = Fraction_dying[West,North]

NO-LEAK ZONE = 20.8333%

CDeathRate[West,North] = C[West,North]/CarrierDuration[West,North]

S[West,North](t) = S[West,North](t - dt) + (Inflow[West,North] - Infection_Rate[West,North]) *

dt

INIT S[West,North] = 500

INFLOWS:

Inflow[West,North] = Birth[West,North]+GiftAgistIN[West,North]

OUTFLOWS:

Infection_Rate[1,1] =

Transmission_Rate[1,1]*S[1,1]*I[1,1]/(S[1,1]+I[1,1]+E[1,1]+C[1,1])+Infectivity_Reduction[1,1]*C[1,1]*S[1,1]*Transmission_Rate[1,1]/(S[1,1]+I[1,1]+E[1,1]+C[1,1])+0*SpartialInteractn*MONTECARLO(SpatialTrans)

Infection_Rate[1,2] =

Transmission_Rate[1,2]*S[1,2]*I[1,2]/(S[1,2]+I[1,2]+E[1,2]+C[1,2])+Infectivity_Reduction[1,2]*C[1,2]*S[1,2]*Transmission_Rate[1,2]/(S[1,2]+I[1,2]+E[1,2]+C[1,2])

+0*SpartialInteractn*MONTECARLO(SpatialTrans)

Infection_Rate[1,3] =

Transmission_Rate[1,3]*S[1,3]*I[1,3]/(S[1,3]+I[1,3]+E[1,3]+C[1,3])+Infectivity_Reduction[1,3]*C[1,3]*S[1,3]*Transmission_Rate[1,3]/(S[1,3]+I[1,3]+E[1,3]+C[1,3])

+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[1,3]*S[1,3]*I[2,3]/(S[1,3]+I[1,3]+E[1,3]+C[1,3])+Infectivity_Reduction[1,3]*C[2,3]*S[1,3]*Transmission_Rate[1,3]/(S[1,3]+I[1,3]+E[1,3]+C[1,3]))

+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[1,3]*S[1,3]*I[1,4]/(S[1,3]+I[1,3]+E[1,3]+C[1,3])+Infectivity_Reduction[1,3]*C[1,4]*S[1,3]*Transmission_Rate[1,3]/(S[1,3]+I[1,3]+E[1,3]+C[1,3]))

Infection_Rate[1,4] =

Transmission_Rate[1,4]*S[1,4]*I[1,4]/(S[1,4]+I[1,4]+E[1,4]+C[1,4])+Infectivity_Reduction[1,4

]*C[1,4]*S[1,4]*Transmission_Rate[1,4]/(S[1,4]+I[1,4]+E[1,4]+C[1,4]) +
SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[1,4]*S[1,4]*I[1,3]/(S[1,4
]+I[1,4]+E[1,4]+C[1,4])+Infectivity_Reduction[1,4]*C[1,3]*S[1,4]*Transmission_Rate[1,4]/(S[
1,4]+I[1,4]+E[1,4]+C[1,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_
Rate[1,4]*S[1,4]*I[2,4]/(S[1,4]+I[1,4]+E[1,4]+C[1,4])+Infectivity_Reduction[1,4]*C[2,4]*S[1,4
]*Transmission_Rate[1,4]/(S[1,4]+I[1,4]+E[1,4]+C[1,4]))
Infection_Rate[1,5] =
Transmission_Rate[1,5]*S[1,5]*I[1,5]/(S[1,5]+I[1,5]+E[1,5]+C[1,5])+Infectivity_Reduction[1,5
]*C[1,5]*S[1,5]*Transmission_Rate[1,5]/(S[1,5]+I[1,5]+E[1,5]+C[1,5])+0*SpartialInteractn*M
ONTECARLO(SpatialTrans)
Infection_Rate[2,1] =
Transmission_Rate[2,1]*S[2,1]*I[2,1]/(S[2,1]+I[2,1]+E[2,1]+C[2,1])+Infectivity_Reduction[2,1
]*C[2,1]*S[2,1]*Transmission_Rate[2,1]/(S[2,1]+I[2,1]+E[2,1]+C[2,1])+SpartialInteractn*MO
NTECARLO(SpatialTrans)*(Transmission_Rate[2,1]*S[2,1]*I[2,2]/(S[2,1]+I[2,1]+E[2,1]+C[2,
1])+Infectivity_Reduction[2,1]*C[2,2]*S[2,1]*Transmission_Rate[2,1]/(S[2,1]+I[2,1]+E[2,1]+C
[2,1]))+MONTECARLO(SpatialTrans)*SpartialInteractn*(Transmission_Rate[2,1]*S[2,1]*I[3,1
]/(S[2,1]+I[2,1]+E[2,1]+C[2,1])+Infectivity_Reduction[2,1]*C[3,1]*S[2,1]*Transmission_Rate[
2,1]/(S[2,1]+I[2,1]+E[2,1]+C[2,1]))
Infection_Rate[2,2] =
Transmission_Rate[2,2]*S[2,2]*I[2,2]/(S[2,2]+I[2,2]+E[2,2]+C[2,2])+Infectivity_Reduction[2,2
]*C[2,2]*S[2,2]*Transmission_Rate[2,2]/(S[2,2]+I[2,2]+E[2,2]+C[2,2])+SpartialInteractn*MO
NTECARLO(SpatialTrans)*(Transmission_Rate[2,2]*S[2,2]*I[2,2]/(S[2,2]+I[2,1]+E[2,2]+C[2,
2])+Infectivity_Reduction[2,2]*C[2,1]*S[2,2]*Transmission_Rate[2,2]/(S[2,2]+I[2,2]+E[2,2]+C

[2,2]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,2]*S[2,2]*I[2,3]/(S[2,2]+I[2,2]+E[2,2]+C[2,2])+Infectivity_Reduction[2,2]*C[2,3]*S[2,2]*Transmission_Rate[2,2]/(S[2,2]+I[2,2]+E[2,2]+C[2,2]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,2]*S[2,2]*I[3,2]/(S[2,2]+I[2,2]+E[2,2]+C[2,2])+Infectivity_Reduction[2,2]*C[3,2]*S[2,2]*Transmission_Rate[2,2]/(S[2,2]+I[2,2]+E[2,2]+C[2,2]))

Infection_Rate[2,3] =

Transmission_Rate[2,3]*S[2,3]*I[2,3]/(S[2,3]+I[2,3]+E[2,3]+C[2,3])+Infectivity_Reduction[2,3]*C[2,3]*S[2,3]*Transmission_Rate[2,3]/(S[2,3]+I[2,3]+E[2,3]+C[2,3])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,3]*S[2,3]*I[2,2]/(S[2,3]+I[2,3]+E[2,3]+C[2,3])+Infectivity_Reduction[2,3]*C[2,2]*S[2,3]*Transmission_Rate[2,3]/(S[2,3]+I[2,3]+E[2,3]+C[2,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,3]*S[2,3]*I[1,3]/(S[2,3]+I[2,3]+E[2,3]+C[2,3])+Infectivity_Reduction[2,3]*C[1,3]*S[2,3]*Transmission_Rate[2,3]/(S[2,3]+I[2,3]+E[2,3]+C[2,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,3]*S[2,3]*I[2,4]/(S[2,3]+I[2,3]+E[2,3]+C[2,3])+Infectivity_Reduction[2,3]*C[2,4]*S[2,3]*Transmission_Rate[2,3]/(S[2,3]+I[2,3]+E[2,3]+C[2,3]))+MONTECARLO(SpatialTrans)*SpartialInteractn*(Transmission_Rate[2,3]*S[2,3]*I[3,3]/(S[2,3]+I[2,3]+E[2,3]+C[2,3])+Infectivity_Reduction[2,3]*C[3,3]*S[2,3]*Transmission_Rate[2,3]/(S[2,3]+I[2,3]+E[2,3]+C[2,3]))

Infection_Rate[2,4] =

Transmission_Rate[2,4]*S[2,4]*I[2,4]/(S[2,4]+I[2,4]+E[2,4]+C[2,4])+Infectivity_Reduction[2,4]*C[2,4]*S[2,4]*Transmission_Rate[2,4]/(S[2,4]+I[2,4]+E[2,4]+C[2,4])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,4]*S[2,4]*I[2,3]/(S[2,4]+I[2,4]+E[2,4]+C[2,4])+Infectivity_Reduction[2,4]*C[2,3]*S[2,4]*Transmission_Rate[2,4]/(S[2,4]+I[2,4]+E[2,4]+C[2,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,4]*S[2,4]*I[1,4

]/(S[2,4]+I[2,4]+E[2,4]+C[2,4])+Infectivity_Reduction[2,4]*C[1,4]*S[2,4]*Transmission_Rate[2,4]/(S[2,4]+I[2,4]+E[2,4]+C[2,4]))+MONTECARLO(SpatialTrans)*SpartialInteractn*(Transmission_Rate[2,4]*S[2,4]*I[2,5]/(S[2,4]+I[2,4]+E[2,4]+C[2,4])+Infectivity_Reduction[2,4]*C[2,5]*S[2,4]*Transmission_Rate[2,4]/(S[2,4]+I[2,4]+E[2,4]+C[2,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,4]*S[2,4]*I[3,4]/(S[2,4]+I[2,4]+E[2,4]+C[2,4])+Infectivity_Reduction[2,4]*C[3,4]*S[2,4]*Transmission_Rate[2,4]/(S[2,4]+I[2,4]+E[2,4]+C[2,4]))

Infection_Rate[2,5] =

Transmission_Rate[2,5]*S[2,5]*I[2,5]/(S[2,5]+I[2,5]+E[2,5]+C[2,5])+Infectivity_Reduction[2,5]*C[2,5]*S[2,5]*Transmission_Rate[2,5]/(S[2,5]+I[2,5]+E[2,5]+C[2,5])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,5]*S[2,5]*I[2,4]/(S[2,5]+I[2,5]+E[2,5]+C[2,5])+Infectivity_Reduction[2,5]*C[2,4]*S[2,5]*Transmission_Rate[2,5]/(S[2,5]+I[2,5]+E[2,5]+C[2,5]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[2,5]*S[2,5]*I[3,5]/(S[2,5]+I[2,5]+E[2,5]+C[2,5])+Infectivity_Reduction[2,5]*C[3,5]*S[2,5]*Transmission_Rate[2,5]/(S[2,5]+I[2,5]+E[2,5]+C[2,5]))

Infection_Rate[3,1] =

Transmission_Rate[3,1]*S[3,1]*I[3,1]/(S[3,1]+I[3,1]+E[3,1]+C[3,1])+Infectivity_Reduction[3,1]*C[3,1]*S[3,1]*Transmission_Rate[3,1]/(S[3,1]+I[3,1]+E[3,1]+C[3,1])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,1]*S[3,1]*I[2,1]/(S[3,1]+I[3,1]+E[3,1]+C[3,1])+Infectivity_Reduction[3,1]*C[2,1]*S[3,1]*Transmission_Rate[3,1]/(S[3,1]+I[3,1]+E[3,1]+C[3,1]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,1]*S[3,1]*I[3,2]/(S[3,1]+I[3,1]+E[3,1]+C[3,1])+Infectivity_Reduction[3,1]*C[3,2]*S[3,1]*Transmission_Rate[3,1]/(S[3,1]+I[3,1]+E[3,1]+C[3,1]))

Infection_Rate[3,2] =

Transmission_Rate[3,2]*S[3,2]*I[3,2]/(S[3,2]+I[3,2]+E[3,2]+C[3,2])+Infectivity_Reduction[3,2]*C[3,2]*S[3,2]*Transmission_Rate[3,2]/(S[3,2]+I[3,2]+E[3,2]+C[3,2])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,2]*S[3,2]*I[3,1]/(S[3,2]+I[3,2]+E[3,2]+C[3,2])+Infectivity_Reduction[3,2]*C[3,1]*S[3,2]*Transmission_Rate[3,2]/(S[3,2]+I[3,2]+E[3,2]+C[3,2]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,2]*S[3,2]*I[2,2]/(S[3,2]+I[3,2]+E[3,2]+C[3,2])+Infectivity_Reduction[3,2]*C[2,2]*S[3,2]*Transmission_Rate[3,2]/(S[3,2]+I[3,2]+E[3,2]+C[3,2]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,2]*S[3,2]*I[3,3]/(S[3,2]+I[3,2]+E[3,2]+C[3,2])+Infectivity_Reduction[3,2]*C[3,3]*S[3,2]*Transmission_Rate[3,2]/(S[3,2]+I[3,2]+E[3,2]+C[3,2]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,2]*S[3,2]*I[4,2]/(S[3,2]+I[3,2]+E[3,2]+C[3,2])+Infectivity_Reduction[3,2]*C[4,2]*S[3,2]*Transmission_Rate[3,2]/(S[3,2]+I[3,2]+E[3,2]+C[3,2]))

Infection_Rate[3,3] =

Transmission_Rate[3,3]*S[3,3]*I[3,3]/(S[3,3]+I[3,3]+E[3,3]+C[3,3])+Infectivity_Reduction[3,3]*C[3,3]*S[3,3]*Transmission_Rate[3,3]/(S[3,3]+I[3,3]+E[3,3]+C[3,3])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,3]*S[3,3]*I[3,2]/(S[3,3]+I[3,3]+E[3,3]+C[3,3])+Infectivity_Reduction[3,3]*C[3,2]*S[3,3]*Transmission_Rate[3,3]/(S[3,3]+I[3,3]+E[3,3]+C[3,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,3]*S[3,3]*I[2,3]/(S[3,3]+I[3,3]+E[3,3]+C[3,3])+Infectivity_Reduction[3,3]*C[2,3]*S[3,3]*Transmission_Rate[3,3]/(S[3,3]+I[3,3]+E[3,3]+C[3,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,3]*S[3,3]*I[3,4]/(S[3,3]+I[3,3]+E[3,3]+C[3,3])+Infectivity_Reduction[3,3]*C[3,4]*S[3,3]*Transmission_Rate[3,3]/(S[3,3]+I[3,3]+E[3,3]+C[3,3]))+SpartialInteractn*MONTECA

RLO(SpatialTrans)*(Transmission_Rate[3,3]*S[3,3]*I[4,3]/(S[3,3]+I[3,3]+E[3,3]+C[3,3])+Infectivity_Reduction[3,3]*C[4,3]*S[3,3]*Transmission_Rate[3,3]/(S[3,3]+I[3,3]+E[3,3]+C[3,3]))

Infection_Rate[3,4] =

Transmission_Rate[3,4]*S[3,4]*I[3,4]/(S[3,4]+I[3,4]+E[3,4]+C[3,4])+Infectivity_Reduction[3,4]*C[3,4]*S[3,4]*Transmission_Rate[3,4]/(S[3,4]+I[3,4]+E[3,4]+C[3,4])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,4]*S[3,4]*I[3,3]/(S[3,4]+I[3,4]+E[3,4]+C[3,4])+Infectivity_Reduction[3,4]*C[3,3]*S[3,4]*Transmission_Rate[3,4]/(S[3,4]+I[3,4]+E[3,4]+C[3,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,4]*S[3,4]*I[2,4]/(S[3,4]+I[3,4]+E[3,4]+C[3,4])+Infectivity_Reduction[3,4]*C[2,4]*S[3,4]*Transmission_Rate[3,4]/(S[3,4]+I[3,4]+E[3,4]+C[3,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,4]*S[3,4]*I[3,5]/(S[3,4]+I[3,4]+E[3,4]+C[3,4])+Infectivity_Reduction[3,4]*C[3,5]*S[3,4]*Transmission_Rate[3,4]/(S[3,4]+I[3,4]+E[3,4]+C[3,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,4]*S[3,4]*I[4,4]/(S[3,4]+I[3,4]+E[3,4]+C[3,4])+Infectivity_Reduction[3,4]*C[4,4]*S[3,4]*Transmission_Rate[3,4]/(S[3,4]+I[3,4]+E[3,4]+C[3,4]))

Infection_Rate[3,5] =

Transmission_Rate[3,5]*S[3,5]*I[3,5]/(S[3,5]+I[3,5]+E[3,5]+C[3,5])+Infectivity_Reduction[3,5]*C[3,5]*S[3,5]*Transmission_Rate[3,5]/(S[3,5]+I[3,5]+E[3,5]+C[3,5])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,5]*S[3,5]*I[3,4]/(S[3,5]+I[3,5]+E[3,5]+C[3,5])+Infectivity_Reduction[3,5]*C[3,4]*S[3,5]*Transmission_Rate[3,5]/(S[3,5]+I[3,5]+E[3,5]+C[3,5]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[3,5]*S[3,5]*I[2,5]/(S[3,5]+I[3,5]+E[3,5]+C[3,5])+Infectivity_Reduction[3,5]*C[2,5]*S[3,5]*Transmission_Rate[3,5]/(S[3,5]+I[3,5]+E[3,5]+C[3,5]))

Infection_Rate[4,1] =

Transmission_Rate[4,1]*S[4,1]*I[4,1]/(S[4,1]+I[4,1]+E[4,1]+C[4,1])+Infectivity_Reduction[4,1]*C[4,1]*S[4,1]*Transmission_Rate[4,1]/(S[4,1]+I[4,1]+E[4,1]+C[4,1])+0*SpartialInteractn*MONTECARLO(SpatialTrans)

Infection_Rate[4,2] =

Transmission_Rate[4,2]*S[4,2]*I[4,2]/(S[4,2]+I[4,2]+E[4,2]+C[4,2])+Infectivity_Reduction[4,2]*C[4,2]*S[4,2]*Transmission_Rate[4,2]/(S[4,2]+I[4,2]+E[4,2]+C[4,2])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[4,2]*S[4,2]*I[3,2]/(S[4,2]+I[4,2]+E[4,2]+C[4,2])+Infectivity_Reduction[4,2]*C[3,2]*S[4,2]*Transmission_Rate[4,2]/(S[4,2]+I[4,2]+E[4,2]+C[4,2]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[4,2]*S[4,2]*I[4,3]/(S[4,2]+I[4,2]+E[4,2]+C[4,2])+Infectivity_Reduction[4,2]*C[4,3]*S[4,2]*Transmission_Rate[4,2]/(S[4,2]+I[4,2]+E[4,2]+C[4,2]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[4,2]*S[4,2]*I[5,2]/(S[4,2]+I[4,2]+E[4,2]+C[4,2])+Infectivity_Reduction[4,2]*C[5,2]*S[4,2]*Transmission_Rate[4,2]/(S[4,2]+I[4,2]+E[4,2]+C[4,2]))

Infection_Rate[4,3] =

Transmission_Rate[4,3]*S[4,3]*I[4,3]/(S[4,3]+I[4,3]+E[4,3]+C[4,3])+Infectivity_Reduction[4,3]*C[4,3]*S[4,3]*Transmission_Rate[4,3]/(S[4,3]+I[4,3]+E[4,3]+C[4,3])+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[4,3]*S[4,3]*I[4,2]/(S[4,3]+I[4,3]+E[4,3]+C[4,3])+Infectivity_Reduction[4,3]*C[4,2]*S[4,3]*Transmission_Rate[4,3]/(S[4,3]+I[4,3]+E[4,3]+C[4,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[4,3]*S[4,3]*I[3,3]/(S[4,3]+I[4,3]+E[4,3]+C[4,3])+Infectivity_Reduction[4,3]*C[3,3]*S[4,3]*Transmission_Rate[4,3]/(S[4,3]+I[4,3]+E[4,3]+C[4,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[4,3]*S[4,3]*I[4,4]/(S[4,3]+I[4,3]+E[4,3]+C[4,3])+Infectivity_Reduction[4,3]*C[4,4]

*S[4,3]*Transmission_Rate[4,3]/(S[4,3]+I[4,3]+E[4,3]+C[4,3]))+SpartialInteractn*MONTECA
RLO(SpatialTrans)*(Transmission_Rate[4,3]*S[4,3]*I[5,3]/(S[4,3]+I[4,3]+E[4,3]+C[4,3])+Infec
tivity_Reduction[4,3]*C[5,3]*S[4,3]*Transmission_Rate[4,3]/(S[4,3]+I[4,3]+E[4,3]+C[4,3]))

Infection_Rate[4,4] =

Transmission_Rate[4,4]*S[4,4]*I[4,4]/(S[4,4]+I[4,4]+E[4,4]+C[4,4])+Infectivity_Reduction[4,4
]*C[4,4]*S[4,4]*Transmission_Rate[4,4]/(S[4,4]+I[4,4]+E[4,4]+C[4,4])+SpartialInteractn*MO
NTECARLO(SpatialTrans)*(Transmission_Rate[4,4]*S[4,4]*I[4,3]/(S[4,4]+I[4,4]+E[4,4]+C[4,
4])+Infectivity_Reduction[4,4]*C[4,3]*S[4,4]*Transmission_Rate[4,4]/(S[4,4]+I[4,4]+E[4,4]+C
[4,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[4,4]*S[4,4]*I[3,4
]/(S[4,4]+I[4,4]+E[4,4]+C[4,4])+Infectivity_Reduction[4,4]*C[3,4]*S[4,4]*Transmission_Rate[
4,4]/(S[4,4]+I[4,4]+E[4,4]+C[4,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmi
ssion_Rate[4,4]*S[4,4]*I[5,4]/(S[4,4]+I[4,4]+E[4,4]+C[4,4])+Infectivity_Reduction[4,4]*C[5,4]
*S[4,4]*Transmission_Rate[4,4]/(S[4,4]+I[4,4]+E[4,4]+C[4,4]))

Infection_Rate[4,5] =

Transmission_Rate[4,5]*S[4,5]*I[4,5]/(S[4,5]+I[4,5]+E[4,5]+C[4,5])+Infectivity_Reduction[4,5
]*C[4,5]*S[4,5]*Transmission_Rate[4,5]/(S[4,5]+I[4,5]+E[4,5]+C[4,5])+0*SpartialInteractn*M
ONTECARLO(SpatialTrans)

Infection_Rate[5,1] =

Transmission_Rate[5,1]*S[5,1]*I[5,1]/(S[5,1]+I[5,1]+E[5,1]+C[5,1])+Infectivity_Reduction[5,1
]*C[5,1]*S[5,1]*Transmission_Rate[5,1]/(S[5,1]+I[5,1]+E[5,1]+C[5,1])+0*SpartialInteractn*M
ONTECARLO(SpatialTrans)

Infection_Rate[5,2] =

Transmission_Rate[5,2]*S[5,2]*I[5,2]/(S[5,2]+I[5,2]+E[5,2]+C[5,2])+Infectivity_Reduction[5,2

]*C[5,2]*S[5,2]*Transmission_Rate[5,2]/(S[5,2]+I[5,2]+E[5,2]+C[5,2])+SpartialInteractn*MO
NTECARLO(SpatialTrans)*(Transmission_Rate[5,2]*S[5,2]*I[4,2]/(S[5,2]+I[5,2]+E[5,2]+C[5,
2])+Infectivity_Reduction[5,2]*C[4,2]*S[5,2]*Transmission_Rate[5,2]/(S[5,2]+I[5,2]+E[5,2]+C
[5,2]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[5,2]*S[5,2]*I[5,3
]/(S[5,2]+I[5,2]+E[5,2]+C[5,2])+Infectivity_Reduction[5,2]*C[5,3]*S[5,2]*Transmission_Rate[
5,2]/(S[5,2]+I[5,2]+E[5,2]+C[5,2]))

Infection_Rate[5,3] =

Transmission_Rate[5,3]*S[5,3]*I[5,3]/(S[5,3]+I[5,3]+E[5,3]+C[5,3])+Infectivity_Reduction[5,3
]*C[5,3]*S[5,3]*Transmission_Rate[5,3]/(S[5,3]+I[5,3]+E[5,3]+C[5,3])+SpartialInteractn*MO
NTECARLO(SpatialTrans)*(Transmission_Rate[5,3]*S[5,3]*I[5,2]/(S[5,3]+I[5,3]+E[5,3]+C[5,
3])+Infectivity_Reduction[5,3]*C[5,2]*S[5,3]*Transmission_Rate[5,3]/(S[5,3]+I[5,3]+E[5,3]+C
[5,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[5,3]*S[5,3]*I[4,3
]/(S[5,3]+I[5,3]+E[5,3]+C[5,3])+Infectivity_Reduction[5,3]*C[4,3]*S[5,3]*Transmission_Rate[
5,3]/(S[5,3]+I[5,3]+E[5,3]+C[5,3]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmi
ssion_Rate[5,3]*S[5,3]*I[5,4]/(S[5,3]+I[5,3]+E[5,3]+C[5,3])+Infectivity_Reduction[5,3]*C[5,4]
*S[5,3]*Transmission_Rate[5,3]/(S[5,3]+I[5,3]+E[5,3]+C[5,3]))

Infection_Rate[5,4] =

Transmission_Rate[5,4]*S[5,4]*I[5,4]/(S[5,4]+I[5,4]+E[5,4]+C[5,4])+Infectivity_Reduction[5,4
]*C[5,4]*S[5,4]*Transmission_Rate[5,4]/(S[5,4]+I[5,4]+E[5,4]+C[5,4])
+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_Rate[5,4]*S[5,4]*I[5,3]/(S[5,
4]+I[5,4]+E[5,4]+C[5,4])+Infectivity_Reduction[5,4]*C[5,3]*S[5,4]*Transmission_Rate[5,4]/(S
[5,4]+I[5,4]+E[5,4]+C[5,4]))+SpartialInteractn*MONTECARLO(SpatialTrans)*(Transmission_

Rate[5,4]*S[5,4]*I[4,4]/(S[5,4]+I[5,4]+E[5,4]+C[5,4])+Infectivity_Reduction[5,4]*C[4,4]*S[5,4]*Transmission_Rate[5,4]/(S[5,4]+I[5,4]+E[5,4]+C[5,4]))

Infection_Rate[5,5] =

Transmission_Rate[5,5]*S[5,5]*I[5,5]/(S[5,5]+I[5,5]+E[5,5]+C[5,5])+Infectivity_Reduction[5,5]*C[5,5]*S[5,5]*Transmission_Rate[5,5]/(S[5,5]+I[5,5]+E[5,5]+C[5,5])+0*SpartialInteractn*MONTECARLO(SpatialTrans)

Birth[West,North] = MIN(BirthRate[West,North]*(C[West,North]+S[West,North]),1)

BirthRate[1,1] = 0

BirthRate[1,2] = 0

BirthRate[1,3] = 0.001

BirthRate[1,4] = 0.001

BirthRate[1,5] = 0

BirthRate[2,1] = 0.001

BirthRate[2,2] = 0.001

BirthRate[2,3] = 0.001

BirthRate[2,4] = 0.001

BirthRate[2,5] = 0.001

BirthRate[3,1] = 0.001

BirthRate[3,2] = 0.001

BirthRate[3,3] = 0.001

BirthRate[3,4] = 0.001

BirthRate[3,5] = 0.001

BirthRate[4,1] = 0

BirthRate[4,2] = 0.001

BirthRate[4,3] = 0.001

BirthRate[4,4] = 0.001

BirthRate[4,5] = 0

BirthRate[5,1] = 0

BirthRate[5,2] = 0.001

BirthRate[5,3] = 0.001

BirthRate[5,4] = 0.001

BirthRate[5,5] = .001

CarrierDuration[West,North] = 45

Fraction_dying[West,North] = .8

GiftAgistIN[1,1] = NORMAL(2,1)

GiftAgistIN[1,2] = 0*NORMAL(2,1)

GiftAgistIN[1,3] = NORMAL(2,1)

GiftAgistIN[1,4] = NORMAL(2,1)

GiftAgistIN[1,5] = 0*NORMAL(2,1)

GiftAgistIN[2,1] = NORMAL(2,1)

GiftAgistIN[2,2] = NORMAL(2,1)

GiftAgistIN[2,3] = NORMAL(2,1)

GiftAgistIN[2,4] = NORMAL(2,1)

GiftAgistIN[2,5] = NORMAL(2,1)

GiftAgistIN[3,1] = NORMAL(2,1)

GiftAgistIN[3,2] = NORMAL(2,1)

GiftAgistIN[3,3] = NORMAL(2,1)

GiftAgistIN[3,4] = NORMAL(2,1)

GiftAgistIN[3,5] = NORMAL(2,1)

GiftAgistIN[4,1] = 0*NORMAL(2,1)

GiftAgistIN[4,2] = NORMAL(2,1)

GiftAgistIN[4,3] = NORMAL(2,1)

GiftAgistIN[4,4] = NORMAL(2,1)

GiftAgistIN[4,5] = 0*NORMAL(2,1)

GiftAgistIN[5,1] = 0*NORMAL(2,1)

GiftAgistIN[5,2] = NORMAL(2,1)

GiftAgistIN[5,3] = NORMAL(2,1)

GiftAgistIN[5,4] = NORMAL(2,1)

GiftAgistIN[5,5] = 0*NORMAL(2,1)

IncubationRate[West,North] = 3

Infectivity_Reduction[West,North] = .35

RecoveryTime[West,North] = 3

SpartialInteractn = 0.1

SpatialTrans = 20

Transmission_Rate[West,North] = 0.3

UNIVERSITEIT VAN PRETORIA
UNIVERSITY OF PRETORIA
YUNIBESITHI YA PRETORIA

Faculty of Veterinary Science

14 June 2017

To whom it may concern
Study Administration
University of Pretoria

Dear Sir/Madam

**RE: Ethics Approval for Mr M Bitamale (Student no 12368980)**

The letter serves to confirm that Mr M Bitamale, under the supervision of Prof Darryn Knobel, has applied to the Office of Dean, Faculty of Veterinary Science) for exemption of the requirements to obtain ethics clearance for finalisation of his PhD project. After evaluating the documentation and discussions with senior academic personnel, a waiver from this requirement was granted as the candidate only made use of data collected as part of another study that has previously been approved by the Ethics Committee of the Ross University.

This letter thus serves as confirmation that the candidate has complied with the ethical requirements for the conducting of research projects at the University of Pretoria.

If you have any concerns, please feel free to contact me.

Yours sincerely

**Prof Vinny Naidoo**
**Deputy Dean: Research and Postgraduate Studies**

Office of the Deputy Dean: Research and Postgraduate Studies
Room 6-7, Arnold Theiler Building, Onderstepoort
University of Pretoria, Private Bag X04
Onderstepoort 0110, South Africa
Tel +27 (0)12 529 8082
Fax +27 (0)12 529 8321
Email vinny.naidoo@up.ac.za
www.up.ac.za