MATH5872M Dissertation in Data Science and Analytics

An Analysis of the Statistical Models Developed after the 2001 Foot and Mouth Epidemic

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Supervisor: [redacted]

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The candidate confirms that the work submitted is his/her own and that appropriate credit has been given where reference has been made to the work of others.

Abstract

The 2001 foot and mouth epidemic was catastrophic to England affecting every aspect of the country including farming, tourism and even the military operations.

The disease spread over the country in under two weeks and caused many animals to be culled and farms were left empty. The Government had to take drastic action which involved paying out billions of pounds in compensation and calling in the military to help control the operation.

As many animals were affected including those not being reared on a farm such as deer, nature reserves were also closed and residents were asked to avoid taking public footpaths through the countryside to prevent the risk of further spread. This caused local businesses and tourism to take a large hit on revenue.

Many statistical models were developed to aid in controlling and understanding the epidemic so that in the future the Government and the public can be more prepared for such an outbreak. Models differed in the techniques and their overall accuracy and this paper aims to compare the different techniques and model an example area using the farms on Fair Isle Island. It delves further into the components of the models to construct a sensitivity analysis to conclude on which are the key factors in determining how likely a farm is to catch the disease and what are the ideal conditions for it to do so.

Contents

1 Introduction

1.1 History of the 2001 epidemic

The 2001 foot and mouth epidemic was a viral disease highly infectious to livestock which broke into 44 countries and caused over 2000 UK premises to be affected. This epidemic lasted seven months before the UK was confirmed to be FMD (foot and mouth disease) clear. FMD affected many cloven-hoofed animals including cattle, pigs and sheep and cost the UK Government over £8million over the course of the outbreak.

The disease is usually found in Asian and African countries and speculation has been made as to how the UK acquired this virus. Everysite (2002) investigated the cause of the first outbreak confirmed in the UK and this was from a pig farm in Northumberland. This was reported and confirmed on 23rd February 2001 and was likely the consequence of the pig farm being licensed to feed its livestock processed waste. This was thought to be the likely carrier of the disease. The time at which the disease became infectious was unknown as the virus can be carried for up to 14 days without showing symptoms so the outbreak could have happened as early as late January. This made it difficult for researchers and veterinary professionals to know the likely time of infection and therefore try and identify contaminated objects and people to mitigate the spread of the disease. As the farmer was unaware of the disease the pigs were taken to the abattoir in the second week of February when the disease was contagious, and due to the virus infectiousness it spread to other farms in Essex and Kent. This was spread to sheep and then to other livestock farms through their attendance and contact with farmers markets where farmers from various locations in the country would attend, spreading the disease across the UK. Within two weeks, the foot and mouth disease was nationwide.

In an attempt to control the disease measures were put in place such as movement bans on livestock and culling policies, many statistical models aimed to measure the performance of the control measures. The idea being improvements could be made on these control measures if ever an epidemic were to happen again.

Countries such as Asia and South Africa where the FMD is still very much apparent have different policies for controlling the disease. They do not have a slaughter policy like here in the UK as the disease is not fatal. The UK implemented this policy so they could have a foot and mouth disease free country.

Figure 1 shows the effect the epidemic had on the number of pigs, cattle and sheep held in the UK. These totals were taken from the December of each year and therefore the 2001 figure is lower, especially for the sheep. This is due to the animals being culled

for being infected and also being culled under suspicion of being infected. The reduction in sheep is clearer in figure1 due to the significantly higher amount of animals held.

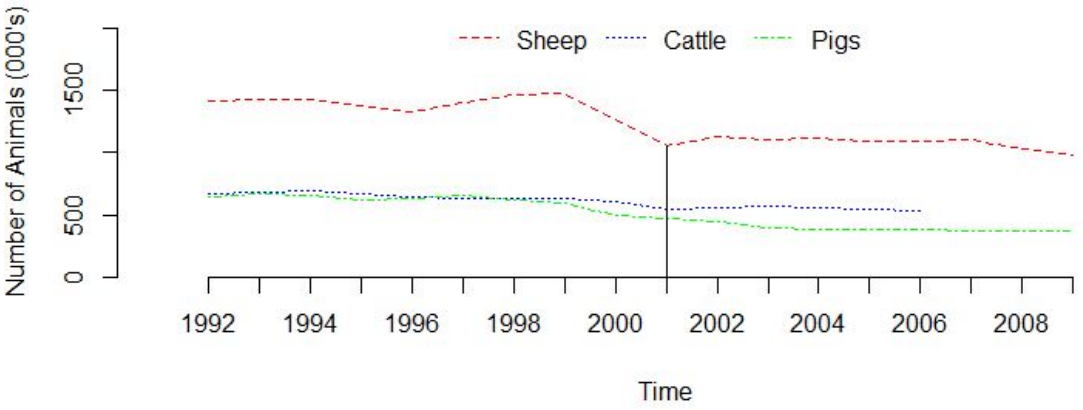
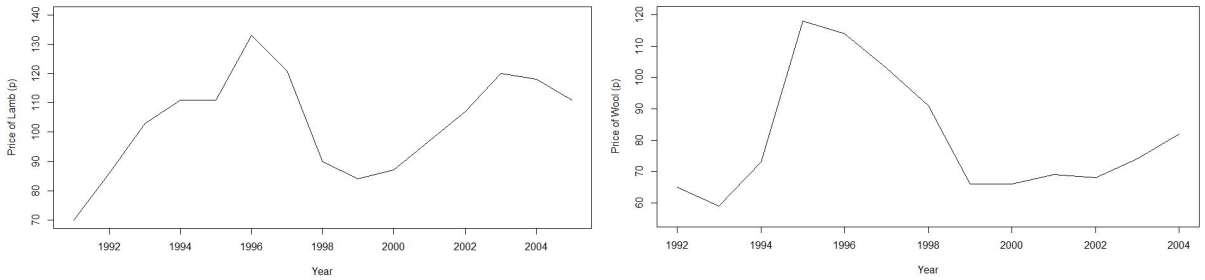


Figure 1: The number of animals in UK holdings. Source: gov.co.uk (2017).

One of the reasons that the number of sheep holdings decreased from 1999 onwards was the price of lamb and the price of wool. As these took a drastic decline in 1998 the price of wool came down and even though the price of lamb decreased, the slump in the prices of wool forced the smaller farmers out of the business. The revenue that the farmers are making from the wool did not amount to enough to cover the medical costs of the animals alone, before food and housing costs were considered. These prices are shown in figure 2.



(a) (b)

Figure 2: Figure (a) shows the price per kilo of lamb and figure (b) shows the price per kilo of wool. Source: The Telegraph (2006).

Table 1 shows the order of events that happened in February 2001.

Monday 19th	The first outbreak was suspected, an investigation was undertaken at Cheale Meats abattoir in Essex after suspicions were raised in 27 pigs.
Tuesday 20th	The outbreak is confirmed by the Ministry of Agriculture. Tests were undertaken confirming the pigs caused the outbreak of the disease. A 5 mile exclusion zone was put on the two farms that had supplied the pigs to the abattoir.
Wednesday 21st	A ban on all exports of live animals, dairy and meat products is announced by the Government, active until 1st March. The exclusion zone is widened to 10 miles. A couple of other farms are investigated.
Thursday 22nd	Another case is confirmed, this farm supplied meat to the abattoir where the outbreak happened. Local residents are advised to avoid the countryside to help prevent further spread of the disease. Local events such as the point to point and hunting are suspended and postmen are told to leave the post at farm gates.
Friday 23rd	The sixth outbreak is confirmed in Northumberland. The Government enforces a movement ban of livestock for the next 7 days. Nature reserves and safari parks are closed to prevent the disease spreading.
Saturday 24th	The slaughter of cattle and pigs on 8 farms begins.
Sunday 25th	The seventh outbreak is confirmed in Devon. Animals were culled at a site in Kent under "dangerous contact". No access to parks in London for the public to protect the deer.
Monday 26th	An 11th case is confirmed. Investigators are tracing all the animals that have passed through the same farmers markets. The rest of the European Union are briefed on the crisis.
Tuesday 27th	An extension on the ban on exports is made until 9th March. Belgium cancels all livestock markets and enforces a movement ban. Many European countries cull imports from Britain. 16 outbreaks confirmed. The movement ban is extended another two weeks.
Wednesday 28th	102 farmers are now under investigation with 50 believed to be clear of the disease. 26 cases have now been confirmed.

Table 1: The timeline of the foot and mouth epidemic for February 2001. Source: *The Daily Telegraph*, 1st March 2001.

The National Audit Office has stated that even before the FMD was confirmed in the UK, 57 farms were already infected which indicated how contagious and easily the disease spread as by this point it was already around the country. This also highlights how discrete the symptoms of the virus can be as the infected farms were unaware.

Figure 3: The spread of the foot and mouth disease in the UK. 1. 19/02; 2. 23/02; 3. 24/02; 4. 27/02; 5. 01/03; 6. 03/03. Source: *The BBC*, 18th February 2002.

Date	Event
19th February	The initial outbreak in Essex
23rd February	The disease spreads in Tyne and Wear
24th February	The virus moves back south to Devon
27th February	Wales receives the next outbreak
1st March	The epidemic reaches Cumbria and Scotland
3rd March	The FMD spreads back down to Cornwall

Table 2: Further information regarding the events happened from Figure 3.

Figure 3 was taken from the BBC article in 2002 and highlights how the disease spread across the country in less than two weeks.

Figure 4 below was taken from Scott *et al.* (2004), a table of the number of animals slaughtered in England, Scotland and Wales at the time of the outbreak. As is evident from the graph, the species that were culled the most were sheep and the most animals were culled when they had contact with non-contagious premises within 3km of an infected premises, this does not mean that they tested positive for the disease. This was a precautionary procedure to mitigate the spread of the disease further implemented by the Government. The disease was so contagious that they had no time to test the animals, they had to be culled as a precaution.

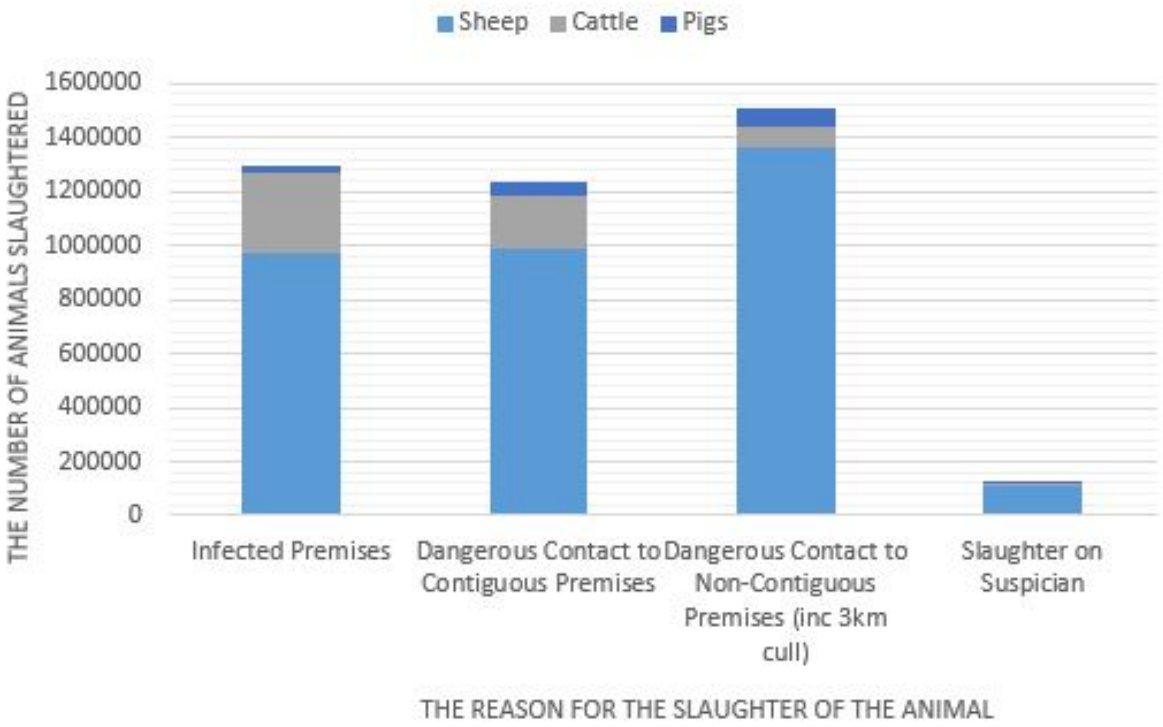
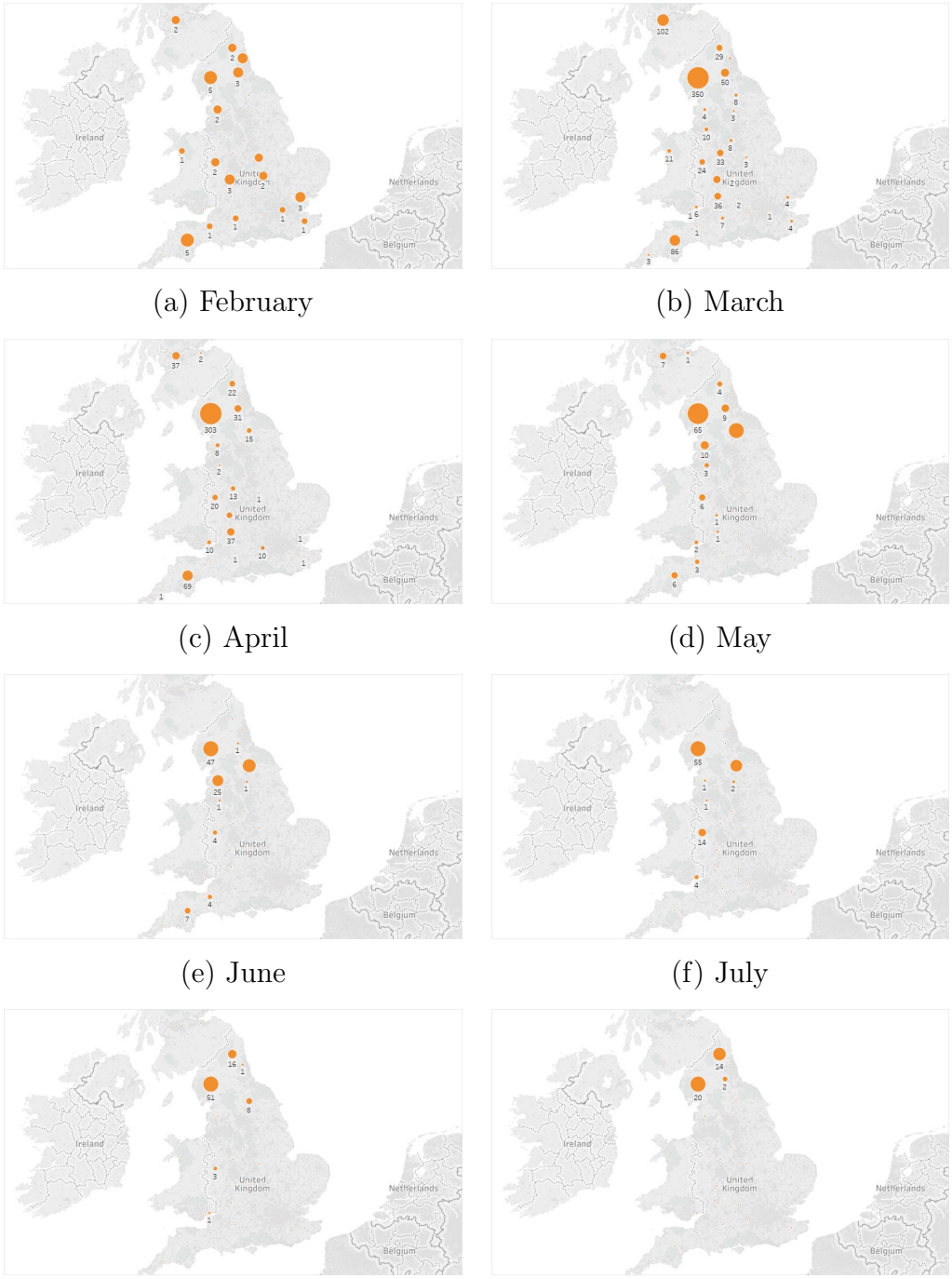


Figure 4: The number of animals slaughtered by classification. Source: Scott *et al.* (2004).

Animals on the infected premises had tested positive for FMD whereas the other three categories had not tested positive but had come into contact with a contaminated area or had some other means to be under suspicion of holding the disease. Although not certain the animal has the disease they needed to be culled as the spread of the disease was so quick and there was no known cure for the disease. This meant the Government had to take drastic action to try and minimise further spread.



(a) February (b) March

(c) April (d) May

(e) June (f) July

(g) August (h) September

Figure 5: The number of new outbreaks each month during 2001. Source: GOV.co.uk.

Figure 5 shows the months that the outbreak happened and the number of new confirmed infected premises with their geographical location. Cumbria had the most outbreaks and

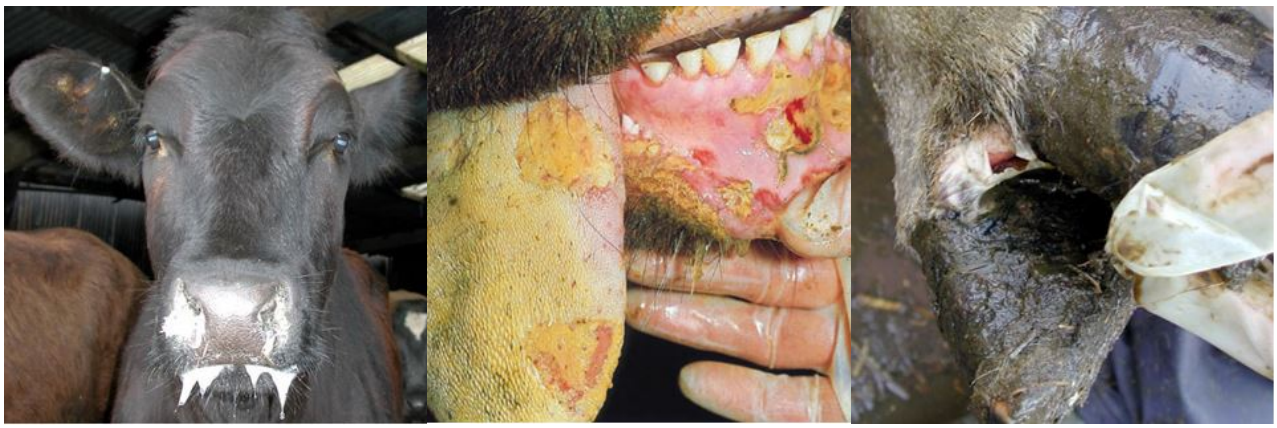
had confirmed infectious premises in every month of the outbreak, even in September. This area suffered the most and this was to be as expected as Cumbria was heavily populated with farms.

1.2 The symptoms of foot and mouth disease

The foot and mouth disease has an incubation period between 2 and 10 days. This means that the infected animals could be showing symptoms only after 10 days of becoming infected. This makes it difficult for farmers and veterinary surgeons to diagnose the virus early enough to prevent the spread to the other livestock at risk in the area. As the disease is so contagious and can take a few days to show symptoms, the whole herd of animals can be infected before any farmer or veterinary surgeon becomes suspicious.

The main symptoms for the disease are

- Blisters around the mouth and on the tongue.
- Blisters and sores on the teats and between the hooves.
- Excess salivation which is foamy and stringier than usual.
- Lameness.
- The animal gets more lethargic as a result of sore hooves.



(a) (b) (c)

Figure 6: Figure (a) shows a cow suffering with excess saliva, (b) shows the tongue blisters and (c) shows the sores the animals get on their hooves. Source: *National Animal Disease Information Service (2011).*

The disease also causes symptoms that are harder to identify, for example the animals will lose their appetite as their mouths will be sore. This will also cause them to lose weight. Due to the sores on their hooves, the animals will be less active and will struggle to stand depending at what stage they are at and how developed their symptoms are.

Arrowquip (2017) states similar symptoms can relate to other diseases such as Bovine respiratory disease complex which reduces the appetite of cattle and causes excess saliva. Similar symptoms of other diseases may delay the farmer in reporting the disease as they may misjudge the symptoms for something else.

Female livestock can find it difficult to conceive. If they are already pregnant, the disease can cause the animal to abort the unborn, or when giving birth the disease increases the chances of the offspring being a still born. In the 2001 epidemic, the livestock would not have made it this far due to the culling control measures being enforced. The livestock in Asia where the disease is common that are not culled as a result of the disease would be affected by this symptom.

1.3 The social implications on farmers

In the 2001 epidemic farmers were faced with changes to their day to day routines and were having to take extra precaution with the fear of catching the disease. This caused mental health issues for the farmers, especially those unfortunate to have all their livestock culled.

Van Haaften *et al.* (2004) studied the effects on the farmers and categorized the three key contributors to the psychological change as stress, marginalization and depression.

Farmers throughout the outbreak incurred higher stress levels due to factors such as the fear of catching the disease, the unknown financial consequences and the change in movement of livestock procedures. Van Haaften *et al.* (2004) highlighted that this was a common factor throughout other times when farmers incurred difficult times such as the decline in meat prices in Australia 1974.

Culture loss, or marginalization occurred throughout the outbreak as normal day to day procedures were disrupted, this can vary from person to person. Farmers feeling lost with no sense of belonging is a result of marginalization, this occurring more in culled farms where farmers no longer have any livestock to maintain.

Depression can be caused by dramatic social changes. This is the result of trying to cope with the stress and marginalization of the outbreak and the consequences of the disease.

The report discussed the thoughts on the Governments ideas that farmers do not have

any bonds with their livestock however the study shows the behavioral effects on the farmers are not just economically driven. One of the main contributors to stress was the Governments top-down system where by farmers had no input in the policies. This lead to the farmers feeling hopeless which added to the depression levels.

Figure 7 shows the daily activities that the farmers in Cumbria were prevented from engaging in. As is evident 96% of the farmers were prevented from visiting their friends during the epidemic. This epidemic lasted 6 months therefore this impacted not just the work life of the farmers, but their social activities too. These figures were taken from Bennett *et al.* (2002).

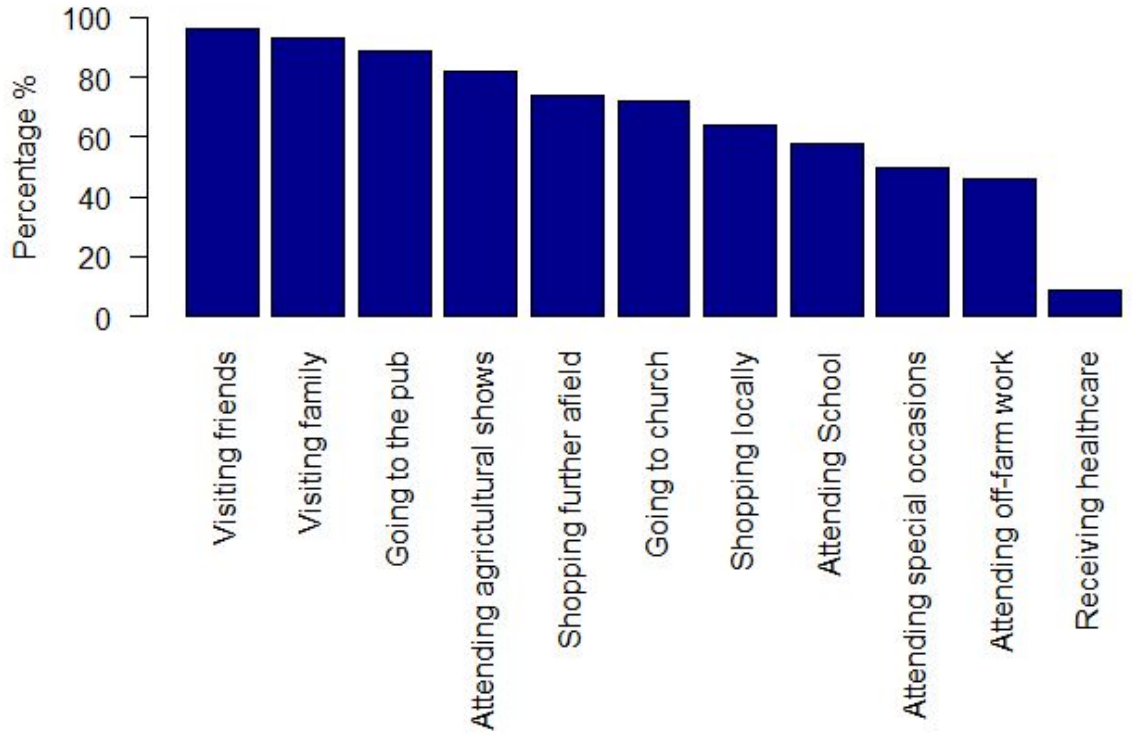


Figure 7: The percentage of farmers in Cumbria who were prevented from engaging in their usual activities. Source: Bennett *et al.* (2002).

The psychological effects of farmers and their families should be considered when forecasting the cost of future epidemics and they should be investigated when looking into the control measures for the disease. The Government needs to appreciate the long term effects of the people witnessing their whole lifestyle being affected and put actions in place to support these farms throughout any future crisis.

Mort *et al.* (2004) discussed further the consequences of the disease by stating that

the reduced confidence in the farming industry, something that these farmers have done all their lives were affecting the sleeping patterns and general well being of the individuals. Trauma is defined as the inability to fight or have control of a situation, this is what the effected farmers incurred.

Another important fact to consider are the long term implications on the social factors relating to the disease. Farmers have a lack of confidence in the Government to control any future epidemics and a fear of another outbreak.

Many staff hired to help with the epidemic were untrained and had to learn as they worked. This caused stress to the individuals but also to the farmers who reared the livestock. This was due to the Government not having the contingency plan underway for such a crisis and this added to the lack in confidence the UK residents had.

Franks *et al.* (2002) explains how the disease drastically impacted farmers household incomes forcing them into a new career path, even when they have farmed all their life. This adds to stress levels as Van Haaften *et al.* (2004) stated. Some of the farmers in Franks *et al.* (2002) survey had experience in other fields however some did not and were working another job to provide for their families due to lack of confidence in the agriculture industry as they felt they were not supported by the Government in ways they should have been.

1.4 The cost of the epidemic to the UK

The National Audit Office produced a report in 2002 containing information relating to the cost borne by the UK government relating to the 2001 FMD epidemic.

Cost Incurred	£(billions)
Compensation and other costs associated to farmers	1.4
Goods and services purchased to eradicate the disease	1.3
Public Sector Costs	0.3
Private Sector Costs	0.6
Lost Revenue from Tourism	4.5
Total Cost	8.1

Table 3: The costs associated with the FMD 2001. Source: *The National Audit Office.* (2002).

As is evident in table 3, the Government incurred a heavy cost of £8.1 billion for the 32 weeks of the outbreak. There has been speculation on the Government's contingency

planning and the reasons why these were so high and why there did not seem to be any contingency plan in place for such an event to happen. The Daily Telegraph (2016) added that the Governments response to the outbreak was just too slow.

Compensation and other costs associated to farmers: Farmers who had to have their livestock culled as a result of the foot and mouth epidemic were compensated from the Government for their loss of assets and also earnings. The costs of disinfecting the premises and removal of any contaminated objects were also included in this figure. The Guardian (2001) expressed the National Farmers Unions views that some farmers compensation was too high which caused controversy in the epidemic. It was said that over 40 farmers were made millionaires as a result of the epidemic.

Goods and services purchased to eradicate the disease: Goods such as disinfectant and culling equipment were needed to be purchased to attempt to control the epidemic. Veterinary staff, soldiers and field team were recruited to help the Government with diagnosis and the culling of the infected livestock. All this came at a cost to the Government.

Lost revenue from tourism: Tourists from around the world were restricted access to many footpaths in the countryside as were local residents which was a loss for country pubs and cafes. Airlines and transport companies also incurred a loss as the media coverage for the FMD deterred visitors from overseas, along with the security checks that were put in place when crossing the border. Nature reserves and national parks were closed to prevent the disease spreading to animals such as deer.

1.5 Control measures implemented

In an attempt to control the epidemic and also prevent further contamination and spread of the virus, the Government implemented a number of control measures. These control measures were criticized by the public as the contingency plans the Government had in place for such an event were not able to cope with the scale of this outbreak.

The Government implemented the following control measures:

- Restrictions on the movement of livestock
- Fencing was erected to ensure that contamination between wild and domestic animals was to a minimum

- Import and export bans on livestock were put in place to stop the spread of the disease to other countries
- Severe and fast culling of infected animals

The public criticized the time it took the Government to make a call to the military for support. It took them three weeks to make this decision and by this time the spread had already reach most parts of the UK. This is a similar situation with the veterinary staff, in the early stages of the epidemic there were not enough veterinary staff to put enough control on the disease and this contributed to the scale of the epidemic. If the Government had known the transmission rates of the disease then more vets could have tested farms all around the UK and could have culled the infected ones in the first few days and this would have prevented the disease spreading to such a scale.

Most of the statistical models that will be discussed were developed to assess the efficiency of the control measures that were implemented as they have to be used retrospective. These models can help the Government to improve their contingency plans for the future so that if an epidemic similar was to happen again, they would be much better prepared.

1.6 Other epidemics in animals

Animals cause epidemics similarly to the way that humans cause epidemics such as the swine flu and the plague however when a human being is not feeling very well, they know to see a professional. Animals cannot do this so they rely on humans to identify the symptoms and take action. When this is not done, or the symptoms are not visible at the time when the infection or disease is spreading, this is when epidemics start as more and more animals become infected and the disease is harder to control.

The bovine spongiform encephalopathy (BSE) is also known as mad cow disease and was a disease that was first confirmed in 1986. It is a disease of the nervous system of cattle. The epidemic lasted years and by 1993 more than one thousand cases were being reported each week with the disease affecting more than half of the UK's population of dairy herds. The symptoms of this include nervousness and aggression and the cattle generally acting different which is where it gets its name from.

Another similar epidemic that hit the farming industry in 2000 was swine fever. This outbreak caused nearly 75,000 pigs to be slaughtered with the fear of the disease spreading. This outbreak caused export bans which was worth a total of £126million per year, The

Guardian (2000). The main symptoms of this disease are blotchy skin, constipation and coughing, Defra (2005).

There are other diseases that broke out in the UK such as bovine tuberculosis and avian influenza, also known as bird flu. These diseases are still very much present in the UK with the latest confirmed case of bird flu occurring in February 2015. However as these diseases are still active in the UK it is categorized as an endemic not an epidemic.

1.7 Overview of the project

This report studies the statistical models developed in the 2001 foot and mouth epidemic. It looks at the accuracy of the models and the differences in the way the components are calculated for example the distance measures and the infection hazard rates.

The report starts by introducing the epidemic and briefs on the cost to the Government and the timeline of the disease. The effects on farmers and the industry are considered and other epidemics from animals are briefed. The advantages and disadvantages for using statistical models for the foot and mouth epidemic are stated before looking at the mathematics of the models and the components used. A sensitivity analysis on the models is undertaken which identifies the drivers of these models and the ideal farm types that will spread the disease even further.

An example of how the models can be used in reality is demonstrated using the Fair Isle off the coast of Scotland and this is modelled using three different models to make a comparison, The discrete time mode, the baseline model and the true road distance model. The main focus on these models with the Fair Isle example is to identify the difference in the models for calculating the how likely susceptible farms are to catching the disease.

The report then concludes with a discussion on what control measures and actions need to be improved on to help prevent and control any future epidemics with the view to minimal disruption in the country. This is whilst considering the social effects on farmers and their residents. The difficulty in estimating the parameters are discussed and the report is concluded with the overall influence statistical models have on any future epidemics.

The program `R` was used to compute graphs

1.8 Useful definitions

The following terms will be used throughout this report.

IP (Infected Premises)	Any plot of land and/or farm that have been contaminated and therefore are now infectious to other farms.
Culling	The controlled slaughter of animals for management purposes. These animals are specifically selected due to them containing the disease or are suspected of having the disease.
Epidemic	The occurrence of a virus or disease at a specific time that is spread across communities.
Susceptible	How likely the animal is to carry the disease after it has come into contact with an infected object and/or animal.
Infectious	How likely the animal is to pass on the disease to another animal and/or object for that to become infected.
Livestock	The term used to describe the farm animals, these can be either cattle, sheep or pigs in this paper.
Euclidean distance	The straight line distance between two points.
Latency Period	The difference in time between the animal becoming infected, and then becoming infectious to other animals.
Transmission Rate	The contact rate of a disease and the subject measured in effective contacts per unit of time.

2 Analysis of Statistical Models

Throughout the foot and mouth epidemic, various statisticians produced models that could predict the prevalence of the disease within the farms. These were produced in as much detail as going to individual farms and analysing case specific examples.

Many of the statistical models were highly accurate considering the variables which were questioned to have contributed to the spread of the disease, with most of the models stating that the minor lack in accuracy was due to the human influence in the decision to classify a farm with foot and mouth disease for those specific cases.

2.1 The advantage of making statistical models

Statistical models have been proven to help many epidemics including the 2001 foot and mouth epidemic. The underlying reason is that it allows the country to be more prepared when should an outbreak happen; this can be from allocation of resources to seeing how well the current control measures are performing. It can also help to identify areas which may need to be improved if ever the outbreak was to happen again. Below are numerous other benefits of the development of statistical models:

- Contribute to the gain in understanding of the epidemic.
- Are able to challenge the model with different scenarios to help with contingency planning.
- Aims to minimise disruption.
- Simplifying complex situations making them easier to interpret for non-experts.
- Identify the efficiency of the current control policy and help to improve for the future.
- Ability to trial new strategies.
- Allocation of resources for the different situations.
- Surveillance targeting.

These ideas were taken from Taylor (2003).

2.1.1 Understanding the epidemic

Statistical models can aid an understanding of the epidemic. It can help to identify the biggest contributions to the spread of the disease, for example a certain species of animal may be more susceptible to picking up the disease. This could help to prevent the spread of the disease if there was another outbreak ad farms containing that species could be targeted for investigation first. It also enables the Government to help protect the areas most vulnerable to the disease and thus helps prevent the spread in that way.

2.1.2 Challenging different scenarios

The models can be tested in different situations that are likely to occur in real life, this can then be used to identify how the model will react to certain scenarios. This can help to see if the current strategy is efficient, for example the control measures that were put in place in the 2001 epidemic such as the livestock movement ban. Alternatively, it can highlight any areas of improvement that are needed to be reviewed before any potential crisis occurs in real life.

Sensitivity analysis methods can also help to put contingency planning into place, this is using different parameters for potential situations and gaining an understanding of how the epidemic would react and therefore being able to plan for many situations, Ferguson *et al.* (2003). This planning could include prevention methods but also the allocation of resources that would need to be on standby for that particular occurrence. As with any crisis such as natural disasters and epidemics it is vital to have a plan of action if these occur and using sensitivity analysis allows the plans to be more efficient and also error tests them first.

2.1.3 Disruption minimisation

In a epidemic such as the 2001 foot and mouth epidemic that affected thousands of farms, the Government need to put measures in place that will reduce the disruption caused throughout the country. The development of statistical models can contribute to this by identifying the disruption caused through the trial of different scenarios stated in section 2.1.2 and then modifying their control policies accordingly to minimise this disruption, for example the minimisation of compensation paid out, the resources such as staff members used and also the prevention of the epidemic and its consequences in general.

2.1.4 Simplifying the epidemics

Statistical models can help people who are not experts in that field to understand the epidemic, Huppert and Katriel (2013). The models simplify the real situation and put it into a simplified model which can be dissected to understand each component. For example, the foot and mouth epidemic needed control policies implemented by the Government; these needed to be approved by people who may not have been biologists and therefore developing statistical models which can break down the event, help to spread the understanding.

The models break the different components down, for example the probability distance measure and the rate of infection and then put them all together at the end. The break down helps to understand the way the models are used and developed but understanding the components can also help identify the most influential components and this can help put together more efficient control measures.

2.1.5 Identification of the efficiency of the control policy

The foot and mouth epidemic had many control policies, some of which were implemented during the outbreak such as the livestock movement ban. The development of the statistical models can demonstrate the performance of these methods being implemented by testing the model before and after using the real data depending on the model. The model can also be tested against new control measures that are due to be implemented to test to see if they have a positive impact. This can help prevention in the future as they can identify areas of improvement and can also prevent the Government spending money on a control measure that did not help the epidemic.

There were a lot of speculation about the Governments delay in recruiting more people to help with the epidemic. With statistical models problems like that will be mitigated as the model will predict the worst case scenario so the Government can be prepared and already have the tools in place.

2.1.6 Trialling new strategies

New strategies for preventing such an epidemic or controlling an epidemic can be trialled and tested in the model. This can identify any opportunities for improvement before implementing this in real life where it can impact numerous people and organizations. This can help to save on resources for potential strategies that do not work and also help with protecting the entities reputation that put the strategy in place.

2.1.7 Allocation of resources

Through the 2001 epidemic many scientists did not predict the disease to spread as far and as quickly as it did. Many resources were used throughout the months that the outbreak was ongoing and this caused much disruption to the UK as a whole but cost the UK Government billions of pounds as shown in table 3. When trialling the models with different scenarios this allows for prior preparation in allocating the resources needed in worse case scenarios, Taylor (2003). This can go from getting the veterinary support that would be needed to confirm the disease in the livestock, to getting together the military that would be needed to help cull the animals.

Knowing this information prior to the outbreak of the disease would enable the Government to be more organised and would also allow them to have a stock of resources such as the vast amount of disinfectant and equipment, so that when the time came they could help prevent the disease by disinfecting as soon as possible.

2.1.8 Surveillance targeting

Surveillance targetting is another use of the models to control the disease, it allows the identification of priority areas that need special care, this could go from extra allocation of resources to a geographical region or a certain area of the disease control such as the resources allocated to farmers compensation.

For example in the 2001 foot and mouth epidemic, the Cumbria region would have needed special care. This would have been identified by Surveillance targeting as it would have noted that the region is heavily populated with farms and they are more prone to the disease. This was identified in figure 5.

2.2 The limitations to making statistical models

Lack of data is the key limitation for developing the models, the model accuracy can only be as accurate and current as the data being used and this is a fundamental flaw, Chis Ster *et al.* (2012). With the foot and mouth epidemic, there were many gaps in the data and this lead to uncertainty in the conclusions and less confidence in the models. For example within the foot and mouth epidemic a key thing to know would be how each farm became infected, whether it was air borne or direct contact however this information is almost impossible to obtain as every animal would have had to be tested which is unrealistic. Information regarding where the animals were kept and the field boundaries would have been useful too, however this would have to be collected by each individual farm and

animals escape and are moved regularly.

Statistical models relating to virus spread are also difficult to interpret and have confidence in as there will always be assumptions made relating to what farm infected other farms in relation to distance. This is due to it being incredibly difficult if not impossible to confirm which farm infected another, especially if there are more than one infected farm in the surrounding area.

Although the models were developed and used, they do need to be validated and kept up to date. The models used in the development in the contiguous culling policy that was made in the first month of the epidemic was actually contradicted in the future months and was concluded to be unnecessary in controlling the epidemic, meaning the model was inaccurate or misused. This was due to the lack of field data around in the early stages of the epidemic. If more accurate data had been available and other models had been developed, then the contiguous culling may have been avoided which would have in turn saved the Government costly compensation payouts.

The real world varies whereas statistical models generally stay constant. For example certain animals may spread the disease faster than others, this will be shown in Figure 9 in this report. The real epidemic is subject to change. For example the model may predict using probabilities the number of animals to be infected in a group exposed to the disease, however some conditions may mean that a significantly different number of animals are infected. This is something that the model would not pick up and therefore leads to uncertainty. This all depends on what type of model the expert has assigned to the problem and the components of the model.

Sometimes events that occur happen as a result of another event occurring. For example, the probability of a farmer reporting their own farm being infected will be less if they have acquired an animal as a result of an illegal movement when the movement ban was implemented. This increases the complexity of the model and can cause problems when trying to develop a model that non-experts can understand.

Events and behavior may change over time. This could be due to the media exposure and more people are aware of the event or in this case the epidemic. For example, throughout the epidemic farmers were becoming increasingly aware of the symptoms of the foot and mouth disease and were taking extra care and precautions. This would change the probabilities of the farmer reporting suspicion of the disease, which many models take into account. This implies that parameters should be changing as time progresses for the model to be as accurate as possible.

2.3 Susceptibility vs Infectivity

Susceptibility is a measure used to describe an animals sensitivity to catching a disease or infection.
Infectivity represents the risk borne by the animal to spread the disease to others.

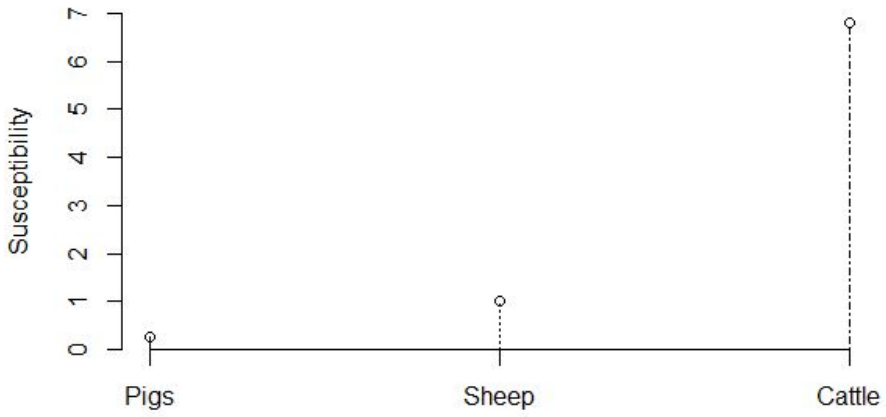


Figure 8: The susceptibility levels for each species.

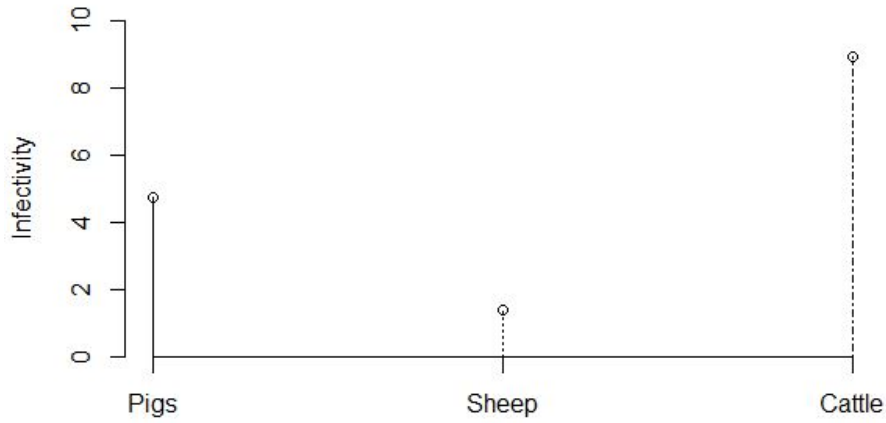


Figure 9: The infectivity levels for each species.

Chis Ster and Ferguson (2007) gave evidence with the results of their statistical model that cattle were the most susceptible species in the 2001 epidemic as the cattle to sheep susceptibility ratio was 6.8. The pig to sheep susceptibility ratio was 0.27; this gives the order of Figure 8. As the ratios were given in the paper, the value for pigs and cattle were plotted on the assumption that the sheep susceptibility was 1 and the cattle and pigs were calculated with respect to this.

Figure 9 shows the cattle have the greatest infectivity. Pigs are more likely to spread the disease than sheep. This is also supported by a BBC news article stating that the National Farmers Union were very concerned for the disease to spread to pig herds as they are more infectious than sheep. This is due to the viruses they produce.

These susceptibility and infectivity rates are crucial when using statistical models as they can determine how much at risk the farm is to catching the disease. These figures will be used in section 7 for modelling purposes. Farms containing more than one species are more susceptible to the disease than others however for modelling purposes separate susceptibility and infectivity rates for each species will be used.

2.4 Challenges incurred

Producing statistical models with any epidemic incurs challenges. These can be from acquiring and cleaning the data collected to knowing what parameters need to be estimated to give the most accurate model at that time.

2.4.1 Lack of data on farm-level transmission

Statistical models from the 2001 foot and mouth epidemic have mainly consisted of the prediction of the prevalence of the disease between farms, predicting if a farm had the disease using the transmission rate of the distance between local farms and then using the number of animals and species in the model to formulate a probability that the farm was infected. There has been little focus on the models that look at the infectivity within farms and this is due to the lack of data. Chis Ster *et al.* (2012) highlighted this difficulty. For this data to be available, the vet would have to predict which animal caught the virus first and then look at the chain of which animals infected which and this is nearly impossible. For this to happen the vet would need to test each individual animal and this is unrealistic as the disease was spreading that quickly, there were not enough vets to visit each case let alone test all the animals.

There is substantial data available relating to each farm as the observation, however there is little information regarding each farms animal infection date and the number and type of animals that were directly affected, this makes any models computed by academics to be sufficiently validated. This prevents certain conclusions being made and thus leaves questions to be answered such as why one farm was infected over the other.

Within Chis Ster *et al.* (2012) studies there were two questions posed that were difficult to answer with the lack of data available for each farm. These questions were also difficult to answer due to the nature and the extensive testing on each animal that

would have to be undertaken. The first question being how many animals in the farm are initially infected. Knowing this would then allow for further investigation on the speed of the infection spreading throughout the uninfected livestock. As the infection is highly contagious and there is an incubation period knowing which animals got the disease first would be nearly impossible.

The second question is the question of at what point do farmers report the infection and what is the probability of the farmer reporting the infection at certain levels of infection. This is hard to predict as it is very subjective and can depend on the farmers awareness of the disease and the symptoms that the livestock are showing. Some animals will show symptoms after 2 days but some may show after 10 therefore it may not fully depend on the farmer reporting the disease but being made aware that their animals are infected. This is why many of the statistical models are criticised as each animal will show symptoms at different stages of being infected. Some of the symptoms of the disease can be caused by other illnesses of the animals and therefore could be misinterpreted by the farmer as something else.

2.4.2 Parameterization

The first challenge that is observed when looking at statistical models is defining the parameters as demonstrated by Tildesley *et al.* (2008). There are many and some unmeasurable when defining a statistical model for the prevalence of foot and mouth disease.

Chis Ster *et al.* (2012) used a range of techniques to estimate their parameters. They embedded previous models and made an assumption to estimate their parameters. However their main method for estimating the model parameters was using a sensitivity analysis and identifying which value fits best to the model.

A need for statistical approaches that can estimate the parameters of non-linear models was identified by Chis Ster and Ferguson (2007).

Parameter estimation is a difficult task that can change the results of a model to the extent that the ideal solution for the objective is not used and therefore consequences are incurred.

The models developed in the foot and mouth disease have parameters that need estimating before any real data from the epidemic can be applied. For example susceptibility and infectivity values, latent and infection periods and the time it takes for the farmers to report any suspicion in their herds.

Kinsley *et al.* (2016) explain the importance of estimating parameters and the caution

that needs to be taken when using other disease data within the method of estimation. Each disease is different and reacts differently to channels of transmission, different hosts and the latent periods therefore using one disease to estimate the parameters of another can sway the results and therefore cause a control measure to be analysed incorrectly. Alexanderson *et al.* (2002) use this method.

The statistical models all require certain parameters to be estimated. The method used can be the difference between choosing one model over another when analysing the control measures therefore the method used to calculate these estimates is crucial.

2.4.3 Markov Chain Monte-Carlo (MCMC)

Markov Chain Monte-Carlo methods are known as one of the most popular parameter estimation methods for statistical models. The methods benefit of not having to define the distribution before anything can be evaluated, this means that the method is flexible. The posterior summaries are easily calculated such as the mean and confidence intervals. This makes it easier when seeing how well each parameter fits the model and choosing which to use going forward.

Chis Ster and Ferguson (2007) used MCMC methods to fit to the data and to obtain parameter estimates.

2.4.4 Fitting the model to data

Tidsley *et al.* (2008) fitted their model to the time series data they had collected to fit their unknown parameters. The real data from the epidemic can be used to fit the model to the events that happened in real life and then using maximum likelihood methods. Althaus (2014) also used maximum likelihood methods to estimate their parameters, however had the difficulty of using this method as the disease he was modelling, EBOV was still ongoing and there were very little data to be used. In such cases assumptions will have to be made to help estimate the other parameters.

2.4.5 Sensitivity analysis

Chis Ster and Ferguson (2012) used sensitivity analysis to establish unknown parameters such as a latent period or an infectious period. This was explored in section 3.1.2. For parameters such as those that are fixed and relatively simple to calculate, this method of modelling using different values and then choosing the one closest to the pattern of the real data can be used.

Haydon *et al.* (1997) used this method to select their latent and infectious time periods.

Chis Ster and Ferguson (2012) stated that infectivity rates for each of the species were taken from other diseases reported. These were then used in the foot and mouth modelling.

2.4.6 Human conclusions

One of the main challenges when producing statistical models in this field is the fact that some of the farms in the analysis were culled through the decision of a surveyor and not because the farms tested positive for the disease. These were for reasons such as they were in a 3km proximity to an infected farm or they had been transported over what the surveyors thought were contaminated areas and therefore a decision was made to cull the livestock. This was also noted in Chis Ster and Ferguson (2007).

As the animals were not tested as positive, the models would therefore not have predicted some of these special cases and that would have therefore reduced the accuracy of these models. With any decision such as this, no model will be able to predict such cases and therefore some caution should be used when using the models.

The decision from one veterinary professional to another will vary as the matters are very subjective. This makes it difficult for any model to duplicate this decision and therefore there will always be farms in the models that are modelled incorrectly to the real data.

Due to the rate at which the disease was spreading across the country the severe culling policies ensured that any animal under suspicion of being infected were culled within 48 hours. This left little room for second opinions from other professionals therefore when a decision was made, no matter what the reasons these animals were culled. The statistical models did not always pick those up as the conditions stated in the models may have been different from the reasons they were culled. The surveyors and veterinary surgeons were all trained in the disease and therefore their judgment was valued and trusted to make the correct decision to help control the disease.

The psychological effects of making the decisions to cull thousands of animals were explained in section1.3.

3 Models from the 1967-1968 epidemic

In November 1967 an outbreak of foot and mouth disease was confirmed at a pig farm in Shropshire. Within a month the UK had over 746 new outbreaks making the outbreak the biggest in the previous ten years. It took until June 1968 to bring the outbreak under control, overall costing the UK Government £27 million. The total of 400,000 animals were culled due to the epidemic, The Guardian (2001).

The 1967 BBC article stated that the Ministry of Agriculture issued advice to farmers to keep their animals under cover, split the livestock into smaller sections, examine their animals daily and also keep as many people and vehicles off the farm as possible. These restrictions are no comparison for the movement bans in the 2001 epidemic. Eventually the Government had to tighten import policies and improve the standards for animal hygiene.

This model planned to assess the efficiency of the control policies by measuring and predicting the spread of the disease through large herds. It looks at the average number of herds the virus infects over a certain time period.

One result of Millers (1976) model was that the 'Dissemination rate', the rate of the average number of herds infected decayed exponentially over the time of the epidemic. This is due to farmers putting the control measures advised by the Ministry of Agriculture.

3.1 The discrete time model

Haydon *et al.* (1997) estimated a set of transmission rates between herds in the outbreak. This model used a discrete time, state transition model and produced a reproduction number for the virus. The results concluded that the transmission rate was high at first, and then declined towards the end of the epidemic, which is to be expected. This was believed to be due to the weather around that time, the weather was perfect for airborne transmission of the virus at the start of the epidemic but not at the end.

Haydon *et al.* (1997) used the parameter $\beta(t)$ to estimate the probability that a infectious herd at time t will infect any susceptible herd at farm t. These herds can be assumed as farms.

A total of 16,506 farms were included in the model and these probabilities were calculated over 80 days of the epidemic including the following parameters

- $S(t)$ as the number of susceptible herds at time t.
- $L(t)$ as the number of latent herds at time t. These are the animals that are infected but are not yet infectious so can not infect any other herd.

- $I(t)$ as the number of infectious herds at time t.
- $R(t)$ as the number of removed/culled herds at time t.
- σ is the fixed latent period. This is the time between an animal becoming infected and it becoming infectious to other animals.
- Δ represents the change in the values from t and $(t+1)$.

The components of this model include

$$\Delta L(t) = \beta(t)S(t)I(t) - \beta(t-\sigma)S(t-\sigma)I(t-\sigma)$$

This represents the change in the Latent herds. The latent herds are the animals that are infected but are not yet infectious to other animals. This states that the change in the number of latent herds at time t multiplies the probability of a infected farm infecting any susceptible farms at time t, $\beta(t)$ multiplied by the number of susceptible farms at time t. This is then multiplied by the number of infected farms at time t. The difference between the probability of a farm infecting a susceptible at the latent period, $\beta(\sigma)$ is subtracted from the probability at time t. The difference in the number of susceptible farms at time σ and t are subrtacted from each other, similarly with the number of infected farms at time σ and t. These three differences are then numtiplies together and subtracted from The product of the first three components.

The model used to calculate the transmission rates in figure 11 is

$$\beta(t) = \frac{\Delta L(t) + \beta(t-\sigma)S(t-\sigma)I(t-\sigma)}{S(t)I(t)}.$$

A disadvantage of this model is that it does not take into consideration any topology or any spatial aspects of the spread of the disease. For example it would assume that farms located next to each other have the same chance of catching the disease as farms miles apart. This makes the model unrealistic. This model also excludes incorporating how many animals in each herd and also the different species. As very little data was collected for this epidemic it made developing more complex models harder. Data for the size of infected herds was collected however data for uninfected herds was not. This makes it difficult to see the reason for why the herds that were infected were infected over the herds that were not.

This model was used to identify the efficiency of the control measures that were implemented and concluded that improved culling methods in an appropriate time would be needed to control future epidemics. The control measures that were used in the 1967 epidemic were successful in reducing the transmission rates but the enviromental factors did change, if the wind had kept like it did initially when most of the disease was spread it would have spread to more farms.

Howard and Donnelly (2000) based their model on Haydon *et al.* (1997), but added a stochastic nature. Their model aimed to estimate the rate of transmission of the virus between an infected farm, and a susceptible one. Their conclusion targeted the effect of the delay in slaughtering animals on the infected farm from them catching the disease, from looking at the the transmission rate per day. The model was then put into different scenarios relating to different delays in slaughter and overall confirmed that if all the animals were slaughtered on the same day as they were confirmed to be infected it would have reduced the epidemic by up to 60% and would therefore reduced the disruption and scale of the epidemic.

3.1.1 The discrete time model in use

In the following example the fixed latent period, σ, will be set at five days. The fixed infectious period, ν, will be set to four days. The probability β will also be assumed as constant, this is for modelling purposes.

At $t = 0$ the following values are

- $S(0) = 2160$
- $I(0) = 0$
- $L(0) = 1$
- $R(0) = 0$

Table 4 shows an example of how the model can be used. Each column represents a day for example L_1 is how many animals are in their first latency day. This is the number of animals in the latent herd. As latent animals are not infectious until five days, this is when they pass into the infectious period of four days.

Each day that there are infectious animals, these animals infect other herds and this is when more susceptible animals fall into the latent herds. These are animals that have been infected but are not yet showing symptoms or are infectious to other animals. They

then move along the days until they become infectious and then are eventually removed or culled.
An explanation of what is happening in table 4 in key times t is explained below:

$t = 0$: Only one animal is infected. As this animal is classified as a latent animal, they are infected but can not infect other animals for the latent period of five days, it can not infect any other animals until day six when it enters the infectious period. There are no animals in the infectious period and therefore no animals will be infected at $t = 1$.

$t = 6$: The infected animal entered the infectious period at $t = 5$ therefore at $t = 6$ it has infected another herd of animals and therefore more animals enter the latent period. As there is one animal in the infectious period, for the subsequent days that there are animals in the infectious period there will be new infected animals entering the latent period. This is due to new animals being infected each day.

$t = 10$: There are no new infected animals due to there being no infectious animals in the infectious period at $t = 9$. The one initial infected animal was removed and therefore there will be no more newly infected animals until there is at least one in the infectious period. This occurs at $t = 12$.

$t = 21$: There are now no more susceptible animals. In reality it would be very unlikely for a disease to infect every single animal on the island but for modelling purposes it is appropriate. The model will just carry on until those last infected animals go through to the removed stage at $t = 30$. Haydon *et al.* (1997) used real data in their model and therefore they did not come to a point where there were no more susceptible animals. If this were to happen then every single animal that were susceptible to the disease including cows, sheep, pigs and deer would be culled and there would be no more left in the UK.

$t = 30$: There are no animals that are susceptible to the disease or infected. They have all been removed and therefore in this example it would mean that there are no cattle or sheep on Fair Isle at all. The disease has infected every single cow and sheep and they have all been culled.

		Latent					Infectious				
t	S	L_1	L_2	L_3	L_4	L_5	I_1	I_2	I_3	I_4	R
0	2159	1	0	0	0	0	0	0	0	0	0
1	2159	0	1	0	0	0	0	0	0	0	0
2	2159	0	0	1	0	0	0	0	0	0	0
3	2159	0	0	0	1	0	0	0	0	0	0
4	2159	0	0	0	0	1	0	0	0	0	0
5	2159	0	0	0	0	0	1	0	0	0	0
6	2009	150	0	0	0	0	0	1	0	0	0
7	1899	110	150	0	0	0	0	0	1	0	0
8	1859	40	110	150	0	0	0	0	0	1	0
9	1654	205	40	110	150	0	0	0	0	0	1
10	1654	0	205	40	110	150	0	0	0	0	0
11	1654	0	0	205	40	110	150	0	0	0	0
12	1334	320	0	0	205	40	110	150	0	0	0
13	1204	130	320	0	0	205	40	110	150	0	0
14	654	550	130	320	0	0	205	40	110	150	0
15	549	105	550	130	320	0	0	205	40	110	150
16	479	70	105	550	130	320	0	0	205	40	110
17	379	100	70	105	550	130	320	0	0	205	40
18	264	115	100	70	105	550	130	320	0	0	205
19	134	130	115	100	70	105	550	130	320	0	0
20	114	20	130	115	100	70	105	550	130	320	0
21	0	114	20	130	115	100	70	105	550	130	320
22	0	0	114	20	130	115	100	70	105	550	130
23	0	0	0	114	20	130	115	100	70	105	550
24	0	0	0	0	114	20	130	115	100	70	105
25	0	0	0	0	0	114	20	130	115	100	70
26	0	0	0	0	0	0	114	20	130	115	100
27	0	0	0	0	0	0	0	114	20	130	115
28	0	0	0	0	0	0	0	0	114	20	130
29	0	0	0	0	0	0	0	0	0	114	20
30	0	0	0	0	0	0	0	0	0	0	114
31	0	0	0	0	0	0	0	0	0	0	0

Table 4: An example of the model used in 1967.

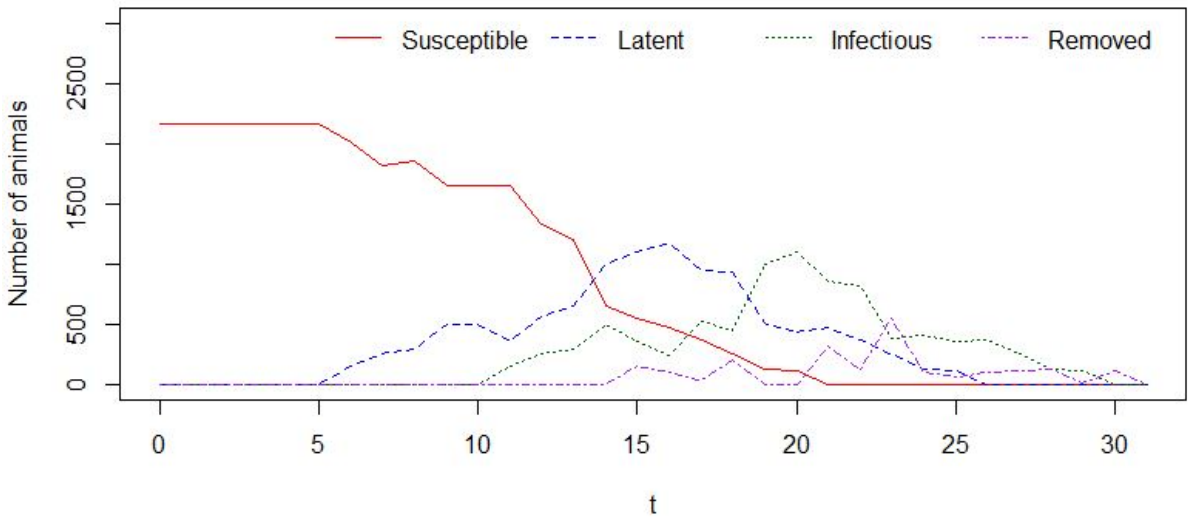


Figure 10: The number of animals in each stage of the disease cycle at each time t.

This example was based on the Fair Isle farms in section 7 and the order of farms becoming infected came from a random selection using R using the infection hazard rates as probabilities for each of the farms after I and H have been infected.

As is evident in the model, if the first animal that was infected were removed in any of the first five days of it being in the latent herd category then none of the other animals would have become infected. This is because the animal would not be in the infectious stage and can therefore not infect any others. This would have prevented the epidemic in Fair Isle, considering any contaminated objects were disinfected.

Figure 11 shows the transmission rates for each of the Fair Isle example days. As is evident when there are no more susceptible animals the transmission rate starts to even out the the rate at the fixed latent period day, σ. At day 10 the transmission rate is very high as there are no animals in the infectious period. The dotted line represents the fixed latent period σ.

Haydon *et al.* (1997) plotted their transmission rates on a log scale where as figure 11 is plotted on a linear scale. The difference in these plots correspond to this, but also correspond to the difference in number of farms that were modelled. Haydon *et al.* (1997) modelled 16,507 herds whereas in the Fair Isle example, the number of animals used were 2,160 with 14 farms being used. The difference in the number of data points used will be a big factor in figure 11 as the data pattern is not as established as in Haydon *et al.* (1997).

Haydon *et al.* (1997) shows that in the first stages of the epidemic the replication rate,

R_0 is over one, this means that for every animal that is infected, it then infects more than one animal. From around day 30 this decreases so that each animal is less infectious and it infects less than one animal for every animal infected.

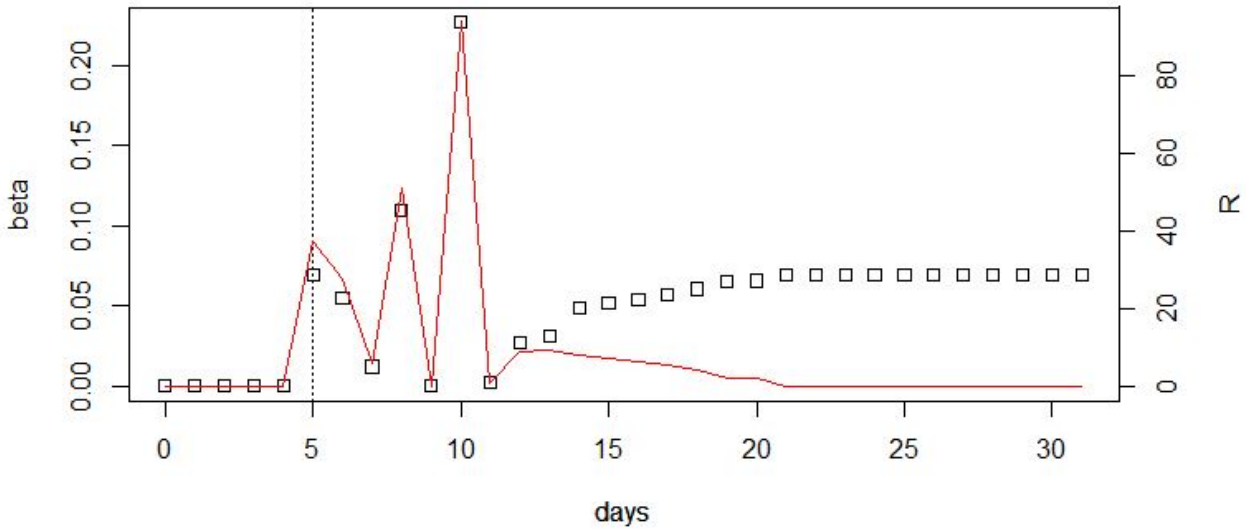


Figure 11: The transmission rates $\beta(t)$ for each of the days in the Fair Isle example.

The replication rate R is calculated by

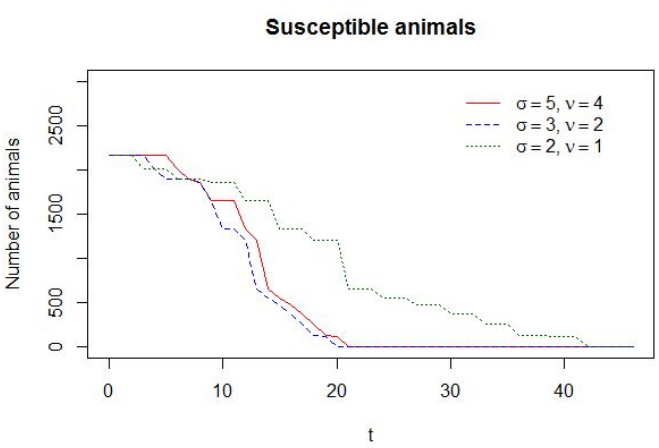
$$R_0 = \frac{\beta(0)N}{\nu}.$$

The red line on figure 11 represents the replication rate. Haydon *et al.* (1997) had a steady declining replication rate but due to the significantly reduced amount of data in the Fair Isle example, the replication rate is volatile. As the different days infect different numbers of animals the replication rate increases and then towards the end the number of susceptible animals comes to zero therefore the infected animals are no longer infecting any others. This makes the replication rate zero.

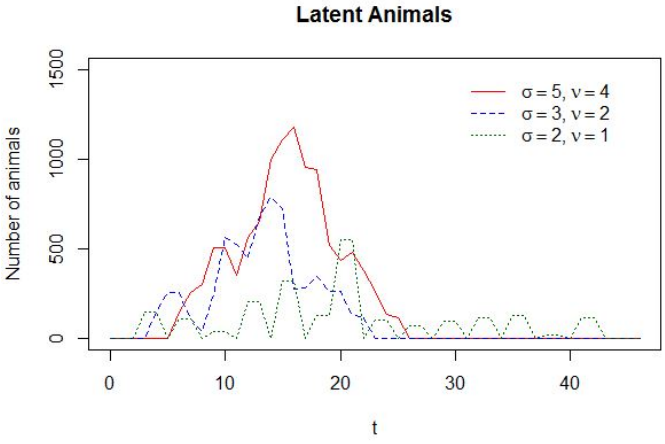
Haydon *et al.* (1997) replication rate never reaches zero, this is due to not having all the animals in the country infected therefore there are still live susceptible animals. For this rate to reach zero, every cow, sheep and pig would have needed to be removed, as in the Fair Isle example.

3.1.2 Sensitivity analysis

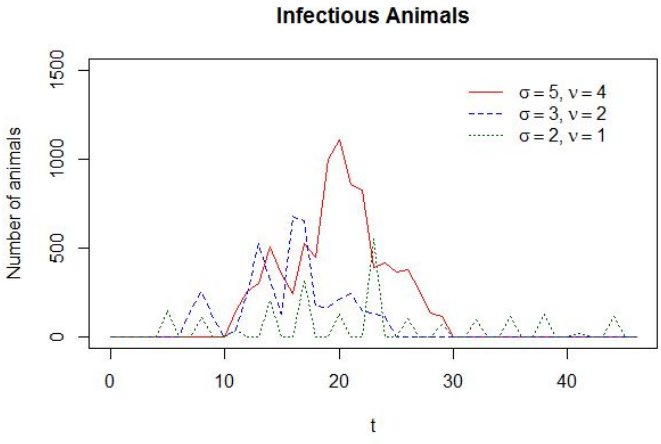
Figure 12 shows the results of a sensitivity analysis on the fixed latency and infection periods, σ and ν. Overall, the results showed that using $\sigma = 3$ and $\nu = 2$ got every susceptible animal to the removal stage the quickest, in 26 days. When using $\sigma = 5$ and $\nu = 4$ got to the same stage at 31 days, and $\sigma = 2$ and $\nu = 1$ were 46 days. This value was the largest as there were only one day where susceptible animals could be infected by the infectious animal. There is a two day gap between the first latent day and the only infectious day therefore there is always a two day wait before any more animals can be infected.



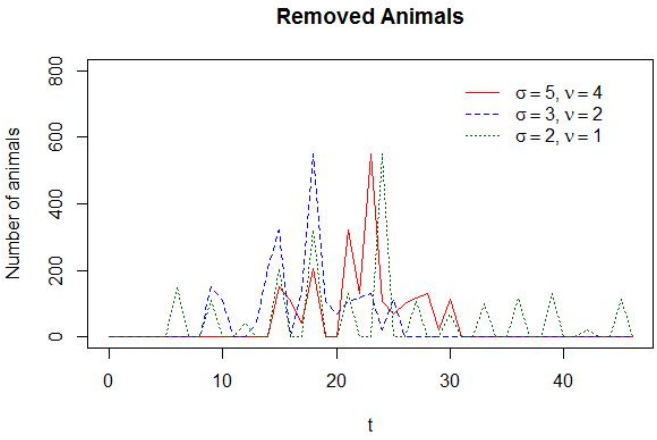
(a) Susceptible animals



(b) Latent animals



(c) Infectious animals



(d) Removed animals

Figure 12: The results of the sensitivity analysis on changing the fixed latent period σ and the fixed infection period ν.

Figure 12 (a) shows the difference in the changing σ and ν values to the decrease in the susceptible animals in the model. Smaller latency and infection periods cause the disease to take longer to infect all the susceptible animals. This is the case for when the latency period was 2 days and the infection period was 1. The reason for this taking so long is animals could only be infected when animals were in the infection day, therefore there were always going to be gaps. Figure 5 explains this as there are always two days when the latent herds are zero. An example of this would be at $t = 2, 5, 8, 11$.

		Latent		Infectious	
t	S	L_1	L_2	I_1	R
0	2159	1	0	0	0
1	2159	0	1	0	0
2	2159	0	0	1	0
3	2009	150	0	0	1
4	2009	0	150	0	0
5	2009	0	0	150	0
6	1899	110	0	0	150
7	1899	0	110	0	0
8	1899	0	0	110	0
9	1859	40	0	0	110
10	1859	0	40	0	0
11	1859	0	0	40	0
12	1654	205	0	0	40

Table 5: Progress of the epidemic for days 0-12 using $\sigma = 2$ and $\nu = 1$.

Figure 12 (b) shows the sum of the animals in the latent herds at each day. As was said previously, when using an infectious period of one day there are days when the latent herds are zero. This explains the pattern for $\nu = 1$. The pattern for the largest $\sigma = 5$ value peaks around the middle at $t = 16$, this is due to the larger herds becoming infected at that time and each latency day is filled with animals, there were no delays in infections at this time. Using $\sigma = 3$ has a similar pattern however as there are not as many latency days, the number of animals classified as latent herds are fewer. Figure 12 (c) shows a similar trend.

Figure 12 (d) shows the animals that have been removed or culled. As there is always only one day for the animals to be removed the trend reflects the amount of animals in each herd being removed. Using $\nu = 1$ the trend is that the value goes to zero and only

increased every two days when there was not a delay in animals being infected.

This model could be improved by using a different parameter to determine a separate latency for the different animals. In reality different species and individual animals will show symptoms and become infectious at different times. It will also depend on the way the animal caught the disease to what its latency period is. This model assumes that all animals have a fixed latent period σ. This is the same for the infectious period ν. Animals are said to be able to carry the disease for two to ten days before they show signs of symptoms therefore every animal is different and making the assumption that they are all the same decreases the accuracy of the model.

A point to discuss when using this model is the transmission rates are not always constant in reality and in the height of an epidemic such as $t = 10$ it is unlikely that the number of newly infected animals will be zero. In the height of an epidemic more cases are confirmed and more animals are being infected. Each individual animal is unique with its latency and infectious period and species to species are very different and model does not consider this.

3.2 Change in the models from the 1967 to the 2001 epidemic

The main difference in the development of the two models is the data that was available in 2001. Data on the number of each species on a farm was available and due to the technological advancements in programming and even online maps have allowed more detailed analysis on such epidemics.

Computer programmes that can model millions of data subjects have become more available and because of this more detailed analysis can be undertaken and conclusions as to how accurate the models are can be made. Improvements can then be justified and for an example such as the foot and mouth epidemic, control measures can be evaluated and it may help the Government prevent if not control any future epidemics. This can help to save disruption and also money.

Sensitivity analysis and simulations can be easily calculated and this helps to model the data in different scenarios, something that would have been too time consuming in 1967.

4 Baseline Model

This section is based on the work of Chis Ster and Ferguson (2007). The following definitions will be used throughout this model;

- Susceptibility relates to the species sensitivity to catching the disease from infected animals or contaminated objects.

- Infectivity represents the risk of the animal passing the disease onto other susceptible animals.

This model describes the probability $k(d)$ of contact between farms a distance d apart. The assumption that the transmission of the virus decreases as the distance between farms increases is applied using a power law.

The equation below models the probability of farms i and j coming into contact when scaled with Euclidean distance d_{ij},

$$k(d_{ij}) = \left(1 + \frac{d_{ij}}{a}\right)^{-\gamma}$$

where

- j represents the infected farm.
- i represents the farm susceptible to the infection.
- d_{ij} is the Euclidean distance between the two.
- a is the kernel offset.
- γ is the kernel power.

These parameters are a simplified representation of the different types of contacts that can occur between farms. If the model were to specify every type of contact such as direct contact between the animals and contact through a contaminated vehicle to name but a few it would become over specified and due to the lack of ability to get this information the model would be inaccurate as it could not be validated.

An example of the how the probability function above changes as the distance changes can be viewed in the graphs below. The two kernel parameters a and γ are changed to show the difference this would make in the overall pattern of the graph.

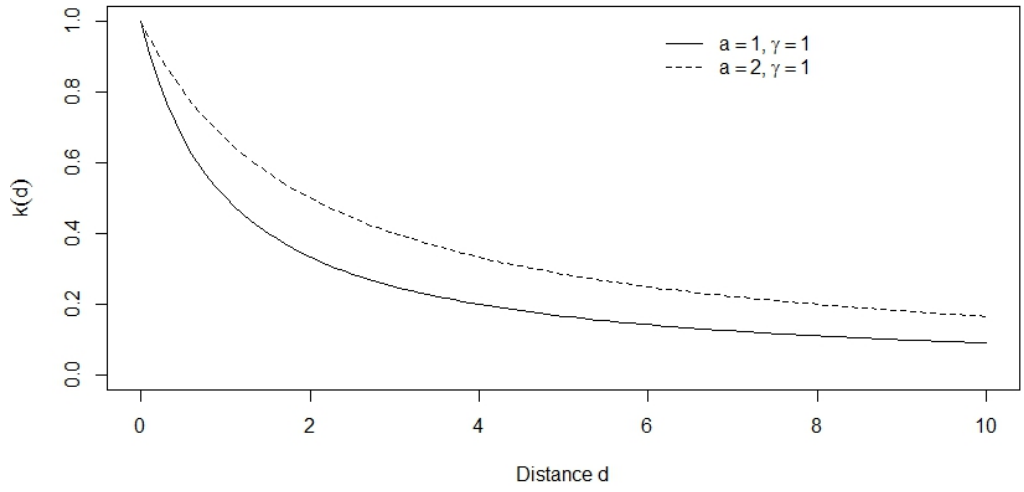


Figure 13: The probability of the contact between farms, $k(d)$ a distance d apart with two measures of the a parameter.

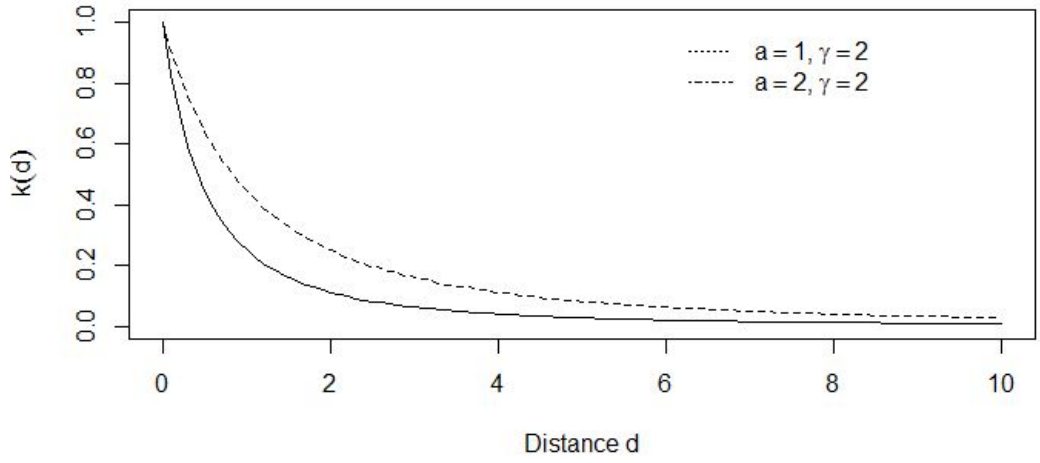


Figure 14: The probability of the contact between farms, $k(d)$ a distance d apart with two measures of the γ parameter.

Figure 13 and figure 14 demonstrate the difference in the pattern adjusting the two

parameters can make.

Figure 13 shows the effect of changing the kernel offset a when $\gamma = 1$. This shows the difference in the shape of the curve. As a increases, the curve becomes smoother indicating that as the distance between the farm increases, compared to a=1 the farm is more likely to become infected.

When comparing figure 13 and figure 14, it is evident that by increasing the γ value it shifts the probabilities further down the scale of the graph. The curve in figure 14 is steeper when the distance between the two farms is very small. Altering the y parameter is very important as this can be the difference between predicting infection in a farm and whether to cull the livestock or not. Not only would the livestock be at risk from the accuracy of the model, but this conclusion could also cost the Government a very large amount of compensation.

The infection hazard rate β is the risk at which the susceptible farm can be infected by the infectious farm. Therefore the higher the value, the higher the chance of the susceptible farm becoming infected. This is a different β to that shown in the 1967 model, that was a probability, this is an infection hazard rate.

The infection hazard rate from an infected farm j to a susceptible farm i is calculated below

$$\beta_{ij} = (S_c n_i^c + S_s n_i^s)\Big(I_c n_j^c + I_s n_j^s\Big)k(d_{ij})$$

where

- S_c and S_s is the susceptibility of cattle and sheep respectively.
- n_i^c and n_i^s are the number of cattle and sheep in farm i.
- I_c and I_s is the infectivity of cattle and sheep respectively.
- n_j^c and n_j^s are the number of cattle and sheep in farm j.
- $k(d_{ij})$ represents the probability of farms i and j coming into contact in relation to their Euclidean distance d_{ij}. Where d_{ij} is the Euclidean distance apart.

As the model is over specified due to the amount of values that need to be estimated, an assumption is made that S_c is a representation of cattle-to-sheep susceptibility meaning that $S_s = 1$. This is because S_c is a ratio that is compared to sheep therefore if $S_s = 1$, S_c is a multiple of S_s. This makes the model easier to use and more applicable to data that is different to the case data used to develop the model. Another assumption of this

model is that the susceptibility and infectivity parameters scale linearly with the number of each species on the farm. In reality this may not be accurate as having more animals on a farm does not necessarily ensure that the farm will be more susceptible to the virus. The cases are more complex in reality but for modeling purposes, an assumption that more livestock held equates to greater risk of catching the infection is implemented.

This equation calculates the susceptibility of farm i by multiplying the susceptibility of each species with the number of each species of animal held on the farm. The infectivity of farm j is calculated in a similar way, by multiplying the infectivity of each species by the number of each species in that farm. To finally get the infection hazard rate, the susceptibility of farm i, the infectivity of farm j and the probability of the farm coming into contact as calculated above $k(d_{ij})$ are multiplied together.

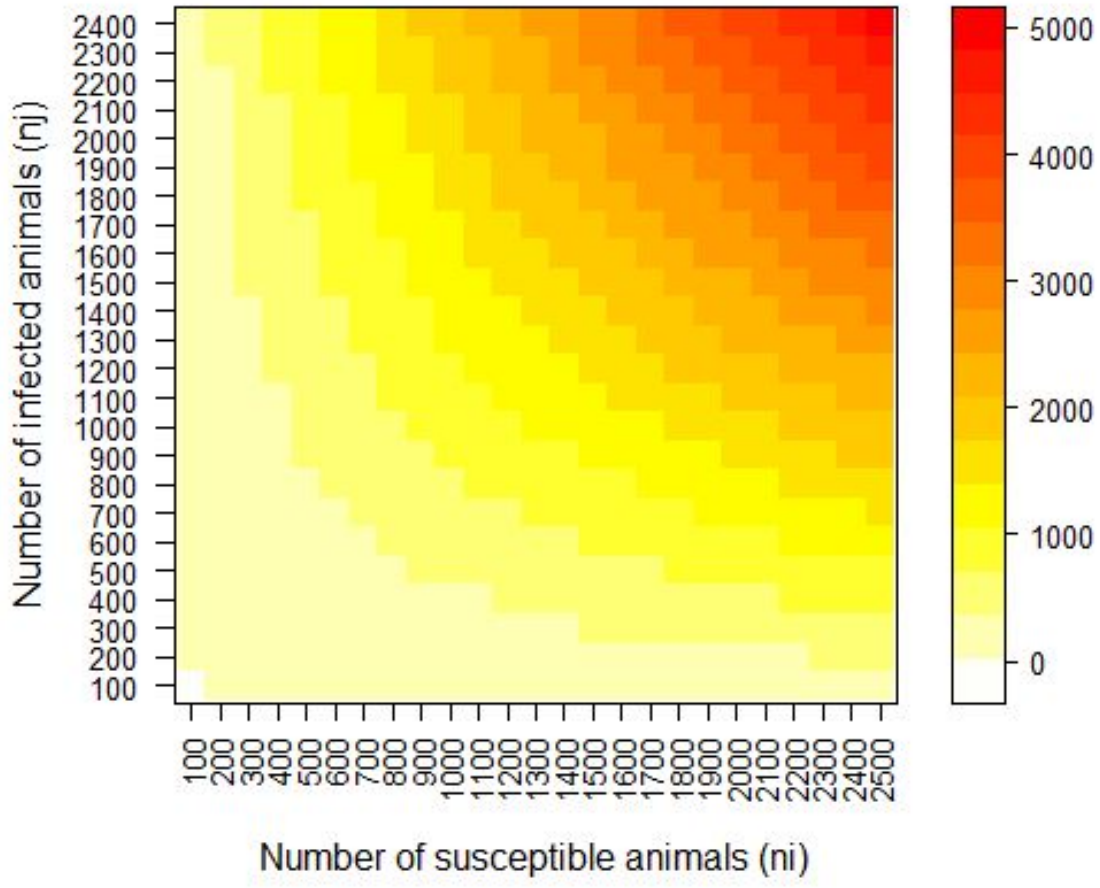


Figure 15: The affect on $\beta/1000$ when changing the number of animals in the susceptible farm n_i and the infected farm n_j.

Figure 15 shows how the change in number of animals held on an susceptible farm and infected farm can change the value of the infection hazard. The higher the number of both farms, the higher the infection hazard is. This was made under the assumption that each farm had the same number of sheep as they do cattle. As this is rarely the case, the heat map was made for demonstration purposes. The following susceptibility and infectivity values were used: $S_c = 6.8$, $S_s = 1$, $I_c = 8.94$ and $I_s = 1.38$. These values were taken from Chis Ster and Ferguson (2007) and were discussed in section 2.3. The β value was scaled down by 1000 to enable the graph easier to interpret.

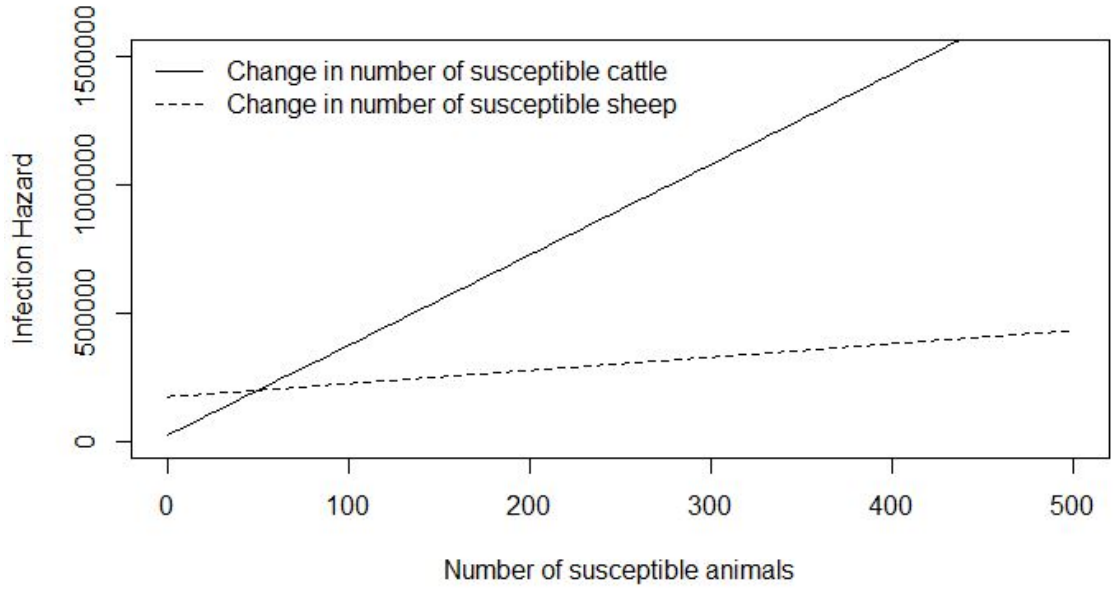


Figure 16: The change in the infection hazard rate when the number of species changes.

Figure 16 shows the difference in the change in number of animals in a species can change the infection hazard to the farm. As is evident by the graph a higher number of cattle held on the farm is significantly more dangerous than having more sheep. This calculation assumes the other numbers of animals such as number of sheep and the number of infected animals are constant. The distance between the susceptible farm and infected farm is also constant at a distance of 1 mile.

The force of infection λ_i measures the strength of the infection infecting the susceptible farm. This is calculated by taking the infection hazard rate for the susceptible farm with respect to the infected farm and multiplying this by the latent period, then these are summed together with all the infected farms to get the force of infection.

For a susceptible farm the force of the infection depends on the event and the specifics of that farm for example the history and location however this has been simplified and the result are as follows.

The force of the infection of a susceptible farm i at time t is

$$\lambda_i(t) = \sum_j \beta_{ij} L_{ij}(t)$$

where

$$L_{ij}(t) = \begin{cases} 1 & \text{, if farm } i \text{ is susceptible and farm } j \text{ is} \\ & \text{infectious at time } t \\ 0 & \text{, otherwise.} \end{cases}$$

Here L represents the latency, in this model the assumption is one day, so the farms are infectious within one day of them becoming infected. This gives them the ability to infect susceptible farms within a day of them being infected.

The probability density function of a farm i is infected at time t is

$$p_i^{infected}(t) = \lambda_i(t) \exp\left[-\int_0^t \lambda_i(\tau) d\tau \right].$$

This gives the log-likelihood

$$l_i^{infected} = \log(\lambda_i(t)) - \int_0^t \lambda_i(t) d\tau.$$

As the model needs to take into consideration the farms that escape being infected, the log-likelihood of the farm not being infected is

$$l_i^{noninfected} = - \int_0^{min(r_i, T)} \lambda_i(\tau) d\tau.$$

where

- r_i is the time at which the animals are culled.

- T is the duration of the epidemic.

This then gives the total log-likelihood of the model

$$l = \sum_i l_i^{infected} + \sum_i l_i^{noninfected}.$$

The model needed to understand to what extent the transmission of the virus was affected between species so an additional parameter was added. The different species mixes were identified and then a parameter p was added which determines the degree to which mixing the species is assortative with $p < 1$ being assortative mixing and $p > 1$ being disassortative.

Assortative mixing within farms is the mixing of species that have similar characteristics, in this context this means that the animals held on each farm are mixed together and the different species, cattle and sheep have contact.

This model makes the assumption that the transmission parameters were constant over time and although this was not the case, this needed to be assumed as if not, the model would be over-specified and would not be relatable to farms other than those that the model were developed on. This also presents the problems of parameter identification explained in section 2.4.2.

The equations above also change in accordance with the new parameter p. The first was 23rd February when the movement ban was put in place. The second change in parameter was the 31st March when the control measures were intensified. The models were fitted with those parameters and the individual case data.

4.1 Results of the Baseline model

The models fitted by Chis Ster and Ferguson (2007) were separated into time varying and non time varying models.

The time varying models were as follows:

1. Cattle infectivity model.
2. Sheep infectivity model.
3. Cattle and sheep infectivity model.
4. Time varying kernel model.

The non time varying models were as follows:

1. Time varying kernel and interaction model.

The posterior deviance is used to test the models fit to the data and the adequacy of the model. This should be traded off with the level of complexity of the model.

The time varying models demonstrated higher accuracy than the models that were solely focused on the infectivity of individual species at that point in time. This is to be as expected as over time, the control measures were intensified the time varying models take into account the changes in the control measures and this is likely to change the transmission rate.

This is seen from the posterior deviance in table 6 and table 7 the time varying kernel model and the time varying kernel and interaction model have a posterior deviance of 29,555 and 29,529 respectively. Making this lower than the model with the next lowest posterior deviance which is the interaction model at 29,662. The lower the posterior deviance, the better the model fits the case data. These models are the most complex and accurate of the models in question.

The Deviance Information Criterion (DIC) is used as an additional comparison for the fit of the models. This is in the situation when two models posterior deviance's are very similar. It is computed with an MCMC algorithm.

The results of the models show that after the movement ban implemented 23rd February the kernel decays faster that before the ban. This gives evidence to suggest that the movement ban assisted in controlling the transmission between the farms and this helped when it came to the transmission distance of the disease as this was restricted.The kernel power before the ban was 1.72 and post ban was 2.68.

The susceptibility ratio, S_c is more informative than the individual species specific infectivity ratio as this compares cattle to sheep in one measure. The time varying kernel with interaction model states that cattle are 5.7 times more susceptible than sheep. Aftosa (2014) states that cattle are the most susceptible animal to the disease and that there were questions raised as to if the outbreak would have occurred without the presence of cattle. As this paper was aimed at the outbreaks worldwide, such as the 1929 outbreak in the US this question could be relevant. However as the UK outbreak was caused by a pig farm, this argument could be dismayed.

Table 7 shows that the models with cattle time varying infectivity are the models most fitted to the cases. The surprising conclusion out of the results was that after the 31st March, when the control measures were intensified, the cattle infectivity actually rose. This is due to the incidence reports decreasing, however they were nearly all cattle

farms. Chis Ster and Ferguson (2007) conclude to state that the reason for this could be the relaxing of the regulations as fewer farms are reported, and therefore farmers are not taking the movement ban precautions as serious as they had when the outbreak was the most topical.

Model		1	2	3	4	5	6	7
Susceptibility Ratio		6.86	5.95	6.83	6.81	6.80	6.74	5.71
		(0.44)	(0.57)	(0.44)	(0.46)	(0.45)	(0.45)	(0.55)
Cattle	Infectivity	8.60	11.9	8.29	8.55	8.29	8.31	11.7
$(I_c)(\text{x}10^8)$		(1.06)	(1.88)	(0.99)	(1.07)	(0.99)	(0.98)	(1.89)
Sheep	Infectivity	1.43	2.16	1.37	2.47	1.34	1.30	2.00
$(I_s)(\text{x}10^8)$		(0.18)	(0.31)	(0.17)	(0.55)	(0.17)	(0.17)	(0.28)
Posterior deviance		29,687	29,662	29,668	29,684	29,672	29,555	29,529
Complexity		4.6	5.1	5.7	5.7	5.7	6.5	7.2
DIC		29,691	29,667	29,674	29,689	29,678	29,561	29,536

Table 6: The models applied on the first set of infection and on 23rd February for the time varying models. Source:*Chis Ster and Ferguson (2007).*
1. Baseline Model 2. Interaction Model 3. Cattle Infectivity Model 4. Sheep Infectivity Model 5. Cattle and Sheep Infectivity Model 6. Time Varying Kernel Model 7. Time Varying Kernel and Interaction Model.

Model		1	2	3	4	5	6	7
Susceptibility Ratio		7.24	6.36	7.35	7.34	5.95	6.08	7.35
		(0.47)	(0.57)	(0.49)	(0.49)	(0.54)	(0.6)	(0.54)
Cattle	Infectivity	7.64	10.30	8.9	8.41	12.1	13.5	7.81
$(I_c)(\text{x}10^8)$		(1.0)	(1.60)	(1.17)	(1.1)	(1.81)	(2.21)	(0.54)
Sheep	Infectivity	1.31	1.93	1.32	1.43	2.24	2.27	1.33
$(I_s)(\text{x}10^8)$		(0.18)	(0.27)	(0.18)	(0.19)	(0.3)	(0.3)	(0.17)
Posterior deviance		28,144	28,122	28,128	28,136	27,981	27,968	28,140
Complexity		4.6	4.9	5.6	5.4	6.3	6.5	6.9
DIC		28,149	28,128	28,134	28,142	27,987	27,975	28,148

Table 7: The models applied on 23rd February and 31st March for the time varying models. Source:*Chis Ster and Ferguson (2007).*
1. Baseline Model 2. Interaction Model 3. Cattle Infectivity Model 4. Cattle and Sheep Infectivity Model 5. Cattle and Sheep Infectivity Interaction Model 6. Cattle Infectivity and Interaction Model 7. Time Varying Kernel Model.

5 Using Road Networks to Measure Distance

Most statistical models that were made during and post the 2001 foot and mouth epidemic had an element where by the distance between an infected farm and the susceptible farm was calculated using the Euclidean distance. This does not take into account of any rivers, lakes or roads. Savill *et al.* (2006) uses road networks as a means of calculating the distance between the two farms as the road networks are the most likely method of transport for the livestock and therefore could be said to be the more accurate way to predict the spread of disease.

Road networks were chosen because they are largely influenced by geographical characteristics such as lakes, mountains and rivers which act as direct barriers for the transmission of the disease. Savill *et al.* (2006) wanted to see if these distances altered the accuracy of the predictor model.

The nodes used in the calculations are road intersections taken from the Digimap Meridian 2 Database, the link is the distance between two nodes. The database can distinguish between different types of roads but does not include private lanes and minor roads, for example farm lanes.

Savill *et al.* (2006) create their own network of nodes and links and then ensure that the Euclidean distance is calculated between each node. The nearest node to the farmhouse is assigned in the network by assuming that the nearest node is that nearest the farm entrance.

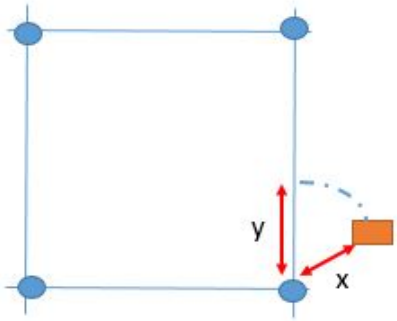


Figure 17: The difference between true road distance y and Euclidean distance x.The orange rectangle represents the farmhouse and the blue circles represent the nearest road intersection (node) that the farmhouse is assigned to. The dotted line is the private farm lane that is not picked up on the Digimap Meridian 2 Database.

Figure 17 represents the difference between the two measures of distance used in Figure

18. The x value us the Euclidean distance and the y value is the true road distance.

Figure 18 represents the true road distances between the farmhouses and the farm entrance which joins a road. There are two types of farms categorized here, farmhouses less than 200m from the road network and farmhouses that are far enough away that their track is not identified on the road network from the original database. An assumption that the farmhouses close to the road network are 0m away from the road. This explains the observations recorded as 0 in Figure 18.

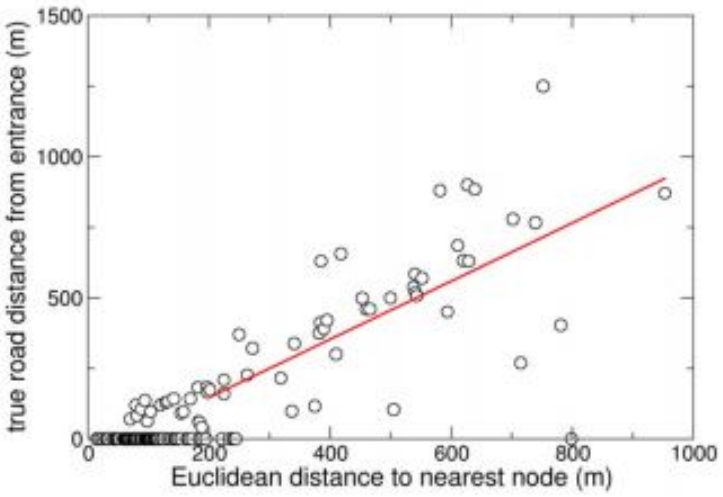


Figure 18: The relationship between the Euclidean distance between two farms and the true road distance.

Figure 18 demonstrates that on average, the true road distance between the susceptible farm and the infected premises is generally higher than the Euclidean distance. The points on the graph that have a true road distance of 0 are those farms that their farm track leads directly to a node. This does not mean that the farmhouse is on the node.

The conclusion made from this analysis is that Euclidean distance is adequate and more appropriate for many reasons for each case is different. For example an analysis along the M6 shows farms can be accessed through other roads and tunnels going underground therefore, although the M6 is categorized as a barrier for the spread of the foot and mouth disease, as there are other means to access the farm, Euclidean distance provides a better method for calculating the distance. However to contradict this statement, another test was carried out with two farms that were separated by a estuary, the conclusion of this test was that using the road networks as a method of calculating the distance was the most accurate method as the disease could not be transferred over the estuary. This

however was a rare case.

Possible reasons for this could be that private roads and tracks are most likely to be used to move livestock locally and are not recorded on the Digimap Meridian 2 Database, that was used as the map for the road networks. Another potential reason for Euclidean distance being better suited as a distance measure is that disease can be spread through the social networks. Using road networks can also be seen as unrealistic as if animals are being moved from field to field, it is likely they will be moved through a gate rather than taken on the road.

Previous models were correct in using the Euclidean distance in general but in regions where there are many geographical factors that could act as barriers for the spread of disease, the shortest distance on the road networks should be used. This would help prevention methods for future outbreaks.

These tests did however have their challenges and assumptions. It is not possible to know which farm infected which other farm so the test had to assume the closest farm infected the one being observed. This might not have always been the case yet there is no evidence to show otherwise. An additional problem when looking at this specific model is that this test was used for regions. A farm in another region may be close to the border and in actual fact be closer to a farm in another region that it caught the disease from. This test would not identify this as the farms are tested within their regions no matter how close to the border they are located.

6 Results from the Statistical Models

The statistical models proved to be successful in predicting if the farm was contaminated and even in cases where they were at incredibly high risk. These were retrospective predictions.

6.1 Accuracy of the statistical models

The statistical models for the 2001 foot and mouth epidemic were seen to be fairly accurate. Tildsley *et al.* (2008) explains that the models that were developed all had a purpose and had to take view to model on those regions where the outbreaks were more dominant than the individual cases.

To begin with the accuracy of the models that were predicting the proportion of farms that were reported as susceptible was extremely high, so the model was changed to separate those farms that were classified as reported cases, and those that were culled. The accuracy of predicting these cases is around 10-15%, this is relatively high as it does account for the stochastic variability between different epidemics. The accuracy rate is also considered as high due to the subjectivity of the decision being made by the veterinarian. It is difficult to predict a human decision.

Increasing the accuracy of the models can be done through multiple simulations, although initially this risks increasing the number of false positives meaning that farms will be predicted to be infected when they are not. This improvement can be done which will increase the sensitivity of the model so that it is more likely to pick up any infected farms, however this will reduce the specificity of the model.

To improve the accuracy of the models, additional transmission kernels can be used to represent the separate species rather than one to represent them all. As seen in previous models, cattle transfer the disease more than sheep so using one transmission kernel for both species is inaccurate.

In general, models that are used to predict the farms that have been culled tend to have a lower accuracy of the models that predict infected premises. This is due to the culling process and decision being much more complex. Usually in cases like these a professional will make the decision based on their own judgment and it is very difficult to develop a model that can model individuals judgment.

Overall Tildsley *et al.* (2008) concludes to say that these 2001 foot and mouth disease models are very useful in determining the efficiency of the control measures that were implemented, however these models can only be used from a retrospective angle.

6.2 Pigs contributed less to the results

Pig farms seemed to be relatively rare compared to cattle and sheep farms and were therefore omitted from the statistics in Chis Ster and Ferguson (2007). This was due to fewer than 3% of farms being pigs only.

A sensitivity analysis that included pigs after their initial findings were presented, gave evidence to show that including pig farms in the analysis did not give any additional findings and the conclusions were not affected. Omitting the pig farms therefore simplified the model and allowed for more in depth analysis of the cattle and sheep farms.

This was interesting as pigs are known to shed their hair at least once a year, this contaminates everything from the animal pens to the clothes the farmer is wearing. However due to the small number of pig farms affected in the country, Chis Ster and Ferguson (2007) disregarded these farms.

Steinfeldt *et al.* (2016) states in their report that statistical models such as the ones used in Chis Ster and Ferguson (2007) are poor at predicting the prevalence of foot and mouth disease in areas that are densely populated with pig farms.

Pigs could also be more susceptible to the transmission and infectivity of foot and mouth disease due to their gastrointestinal tract. The virus is spread through cattle mainly through respiratory channels; however foot and mouth disease is more likely spread orally through swine. This is not taken into consideration in the Chis and Ster (2007) study as pigs were omitted and therefore this gives evidence to show that the use of their statistical model to predict the prevalence of foot and mouth disease on a pig farm is weak.

Alexanderson *et al.* (2002) concluded in their experiment that overall pigs were the species that contributed most to the airborne transmission of the foot and mouth disease and therefore concluded that pigs should be included in the development of the statistical models.

7 Modeling the epidemic using R

To model the epidemic, farms were plotted on the Fair Isle island in Great Britain. There were 14 farms located on this island and the baseline model is applied to identify which farms had the highest risk of infection. This model takes into consideration the number of animals in each species held on the farm and also the distance between the susceptible and infected farm.

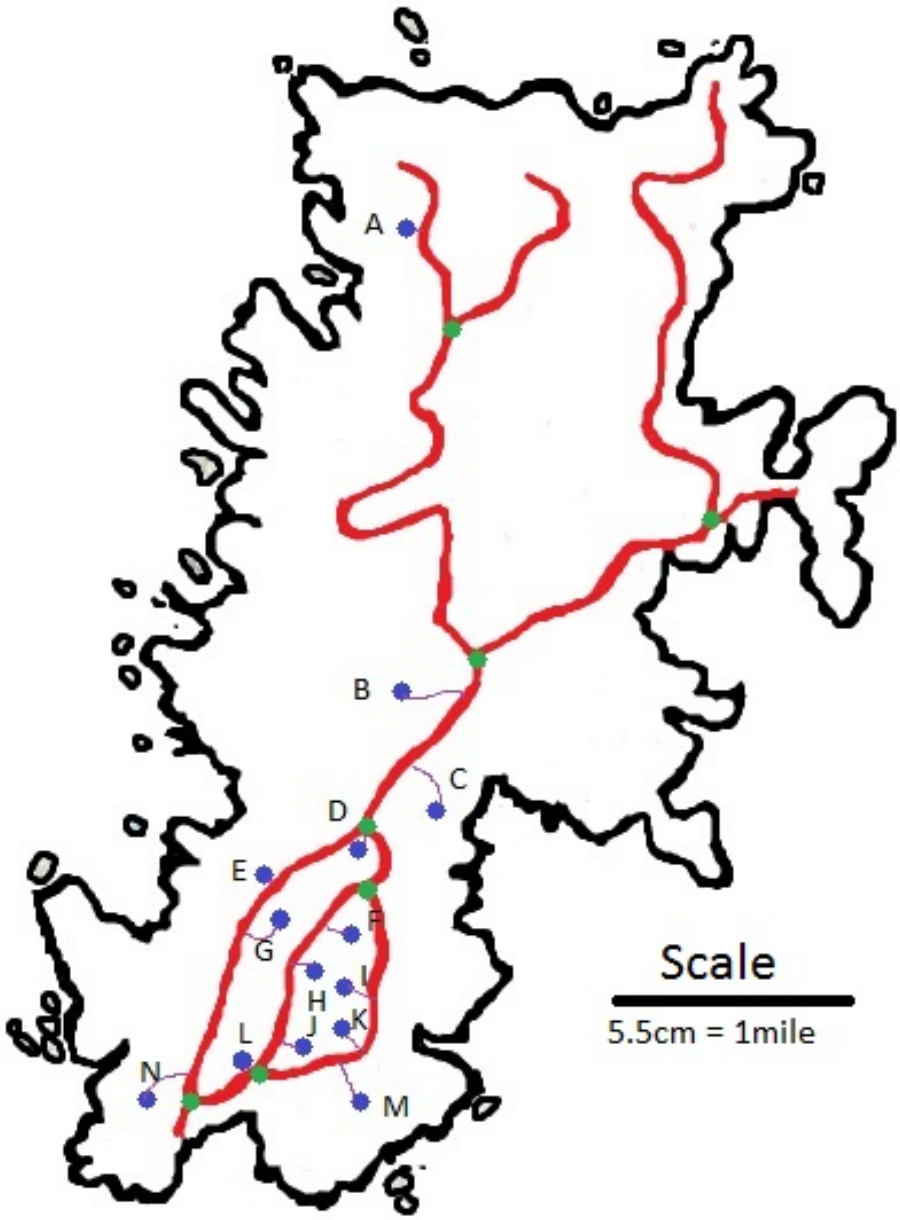


Figure 19: A map of the farms A-N located on Fair Isle and the associated road network.

In both the tests the susceptibility rate for cattle used was 6.8 and the susceptibility for sheep was 1. The infectivity for cattle was 8.946 and for sheep it was 1.38. These were all values taken from section 2.3.

In figure 19 the blue dots represent the farmhouse labelled A-N, the red lines are the main roads, the green dots represent the nodes that were added in place of the road intersections and the thin purple lines represent the farm tracks to the main road.

7.1 Infected farm I

The following farms will be modelled with the assumption that only farm I is infected at time t. When looking at the infection hazard of the other farms, these are all calculated at time t, and at time t the only farm that is infected is farm I.

Farm	No. cattle	No. sheep	dist from I	$k(d_{ij})$	Infection Hazard
A	200	350	1.4	0.416	553848.8
B	120	200	0.65	0.6061	478466.2
C	105	100	0.44	0.6944	437665.3
D	40	60	0.37	0.7299	188156.5
E	0	130	0.29	0.7752	81355.81
F	50	20	0.2	0.8333	231513.7
G	20	0	0.24	0.8065	84409.84
H	60	50	0.13	0.8850	313601.4
I	75	75	NA	NA	NA
J	35	70	0.22	0.8197	196459.3
K	15	100	0.15	0.8697	138461.9
L	0	40	0.31	0.7634	24650.38
M	10	120	0.27	0.7874	117488.3
N	100	15	0.54	0.6494	347695.1

Table 8: The simulated farms information that will be used to model the epidemic.

Table 8 shows the probability $k(d_{ij})$ of the farms coming into contact and the infection hazard β_{ij} of each farm with respect to the infected farm I. These components were taken from the baseline model and applied to the Fair Isle farms.

The number of cattle and sheep for each farm were simulated as no real data was available for these farms. The Euclidean distance from each susceptible farm to the infected farm I was calculated by using the map and the scale and physically measuring

the distance and then converting this to miles. This method does not take into account the topology of the island including streams and hills.

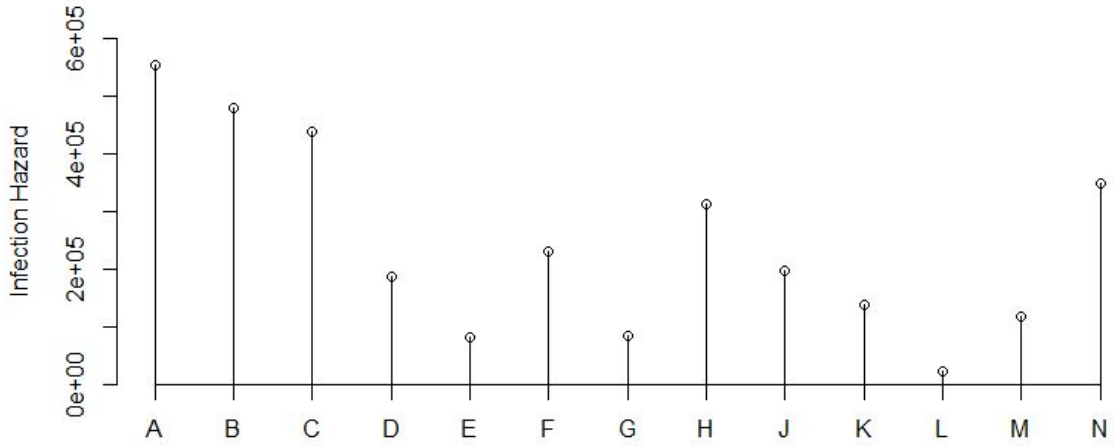


Figure 20: The infection hazard.

Figure 20 shows the infection hazard for each farm with respect to infected farm I. Farm A has the highest infection hazard, this is due to the high number of animals held on the farm. Cattle have the highest susceptibility therefore if a farm has less cattle than another, their infection hazard rate will be lower, even after taking into consideration distance. Farms E and L both have no cattle held on the farm and therefore have the lowest infection hazard rate even though they hold sheep and are located closer to farm I than farms with higher infection hazard rates.

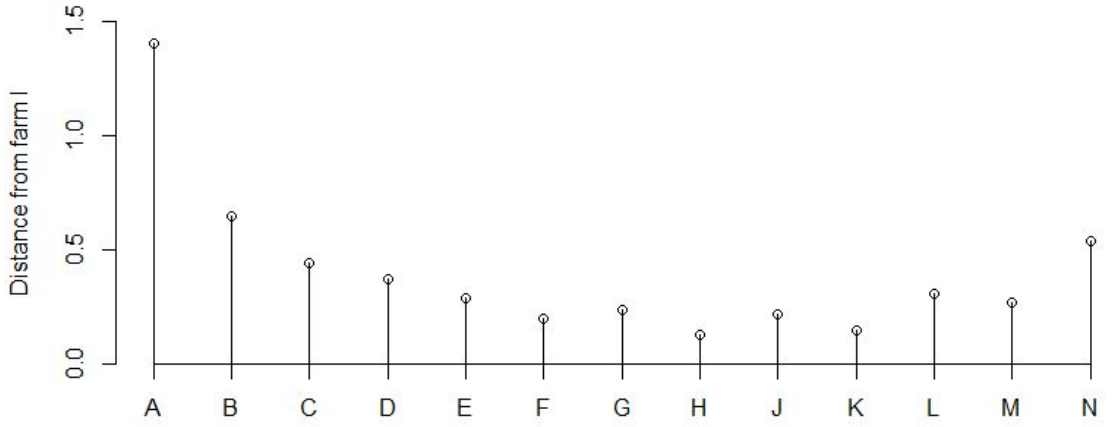


Figure 21: The Euclidean distance from farm I.

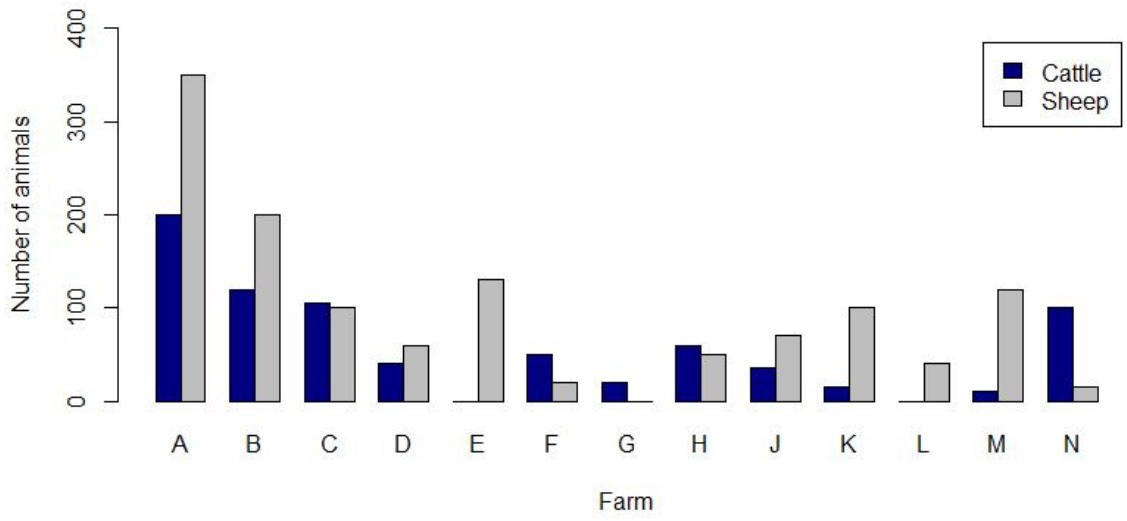


Figure 22: The number of each species on each farm.

Figure 21 and figure 22 show the components that make up the infection hazard rate. As stated previously, in general the higher the number of cattle on the farm, the higher the infection hazard rate.

In this test the force of infection is the same as the infection hazard rate. This is because we use 1 day as the latency period and there is only one infected farm therefore the force of infection is only summed over one farm. In section 7.2 an additional farm is infected and therefore the infection rate is different as will be shown in section 7.2.

7.2 Infected farms I and H

In theory infecting two farms at time t would increase the risk of infection for every farm as there is more risk of the neighboring farms coming into contact with infection. The force of infection is the infection hazard rates for each infected farm summed together and multiplied by the latency period of which in this case is 1 day.

Table 9 shows the infection hazard rate calculated for the susceptible farms if farm H was infected. This has not been calculated for farm I as this farm is not susceptible, it already has the disease.

Farm	No. cattle	No. sheep	dist from H	$k(d_{ij})$	Infection Hazard
A	200	350	1.4	0.4167	433611.5
B	120	200	0.63	0.6135	379190.3
C	105	100	0.44	0.6944	342650.8
D	40	60	0.33	0.7519	151739.2
E	0	130	0.22	0.8197	67348.52
F	50	20	0.18	0.8474	184325.6
G	20	0	0.13	0.8850	72518.02
H	60	50	NA	NA	NA
I	75	75	0.11	NA	NA
J	35	70	0.2	0.8333	156372.6
K	15	100	0.2	0.8333	103885.8
L	0	40	0.27	0.7874	19906.77
M	10	120	0.29	0.7752	90556.19
N	100	15	0.47	0.6803	285175.1

Table 9: The simulated farms infection rates if farm H is infected.

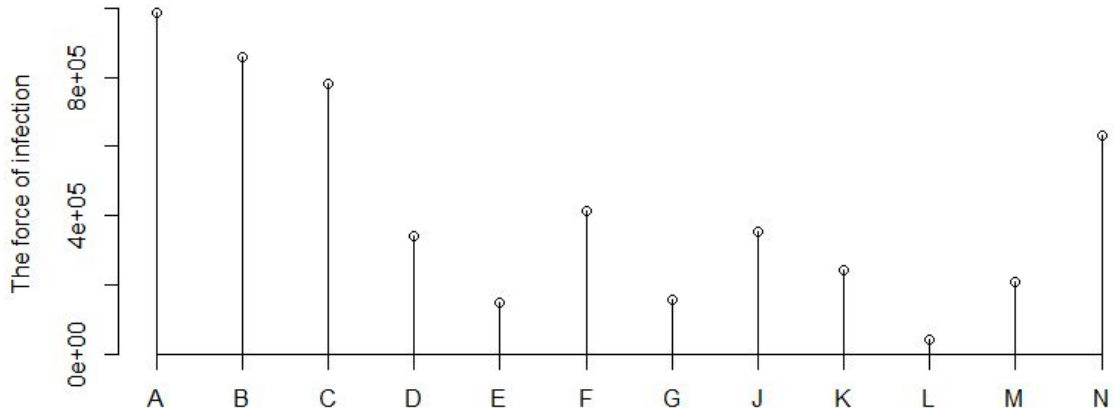


Figure 23: The force of infection if farms H and I are infected.

As is evident from figure 23 the farms most susceptible to catching the infection have not changed from if only farm I was infected. The chance of catching the disease is higher but the order of most susceptible farms is exactly the same. This demonstrates the importance of the number of animals on the farm rather than the distance. In both tests, only farm I being infected and both farm H and I infected the number of animals on the susceptible farms do not change. This is the reason for the similarities between both tests.

7.3 Using the true road distance

This section uses the true road distances as a measure of the distance between farms rather than Euclidean distance. The theory being that the transfer of animals from farm to farm is more than likely through the road networks. This section is based on the theories of Savill *et al.* (2006).

From the Fair Isle map, nodes were added to each road intersection. These nodes will be used throughout the model. The Euclidean distance from each farmhouse to their nearest node is calculated and then the distance from which the private farm lanes meet the main road, and the node are also calculated.

Figure 24 represents how different the two methods of calculating the distance can be. For example the blue line represents $y = x$. The points above the line such as I and C have entrances to farm lanes that are further away than if the euclidean distance would be calculated. Figure 17 shows this.

Farms L and H have equal distances, this is due to farm L being located on the node, and farm H having the same distances as a coinscidence. The dashed red line represents the regression line of $y = 0.4938 + 0.0693x$ and the solid blue line represents the line $x = y$, if both distances were the same.

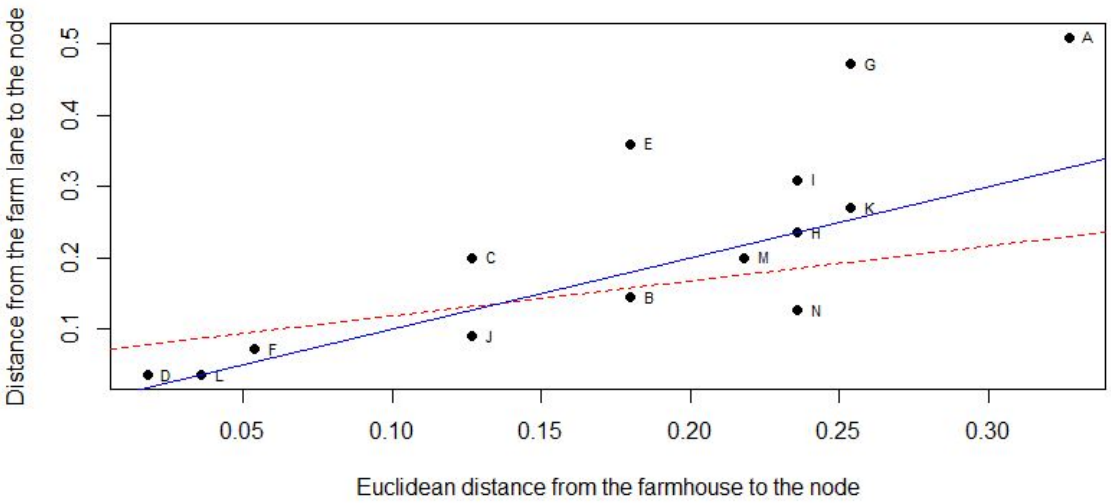
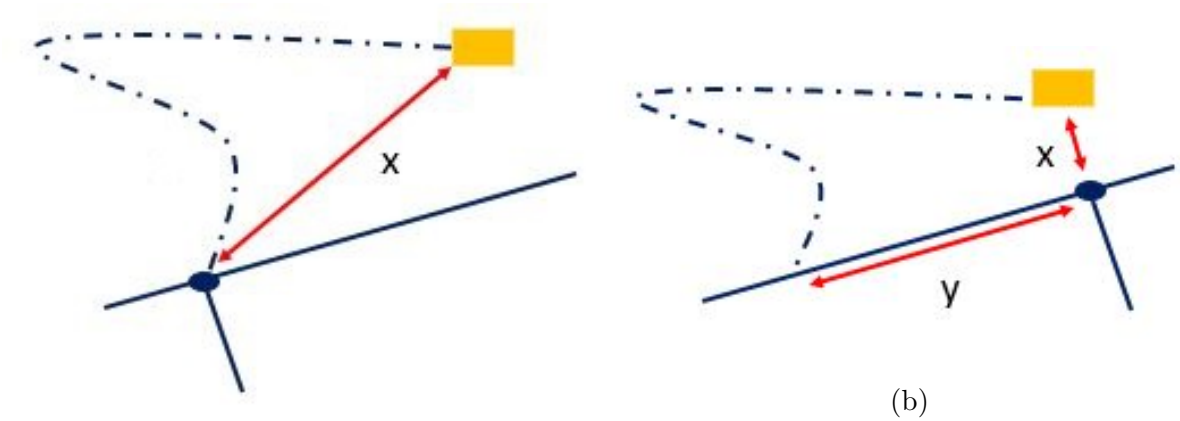


Figure 24: The differences in the two methods of calculating the distance between farms.

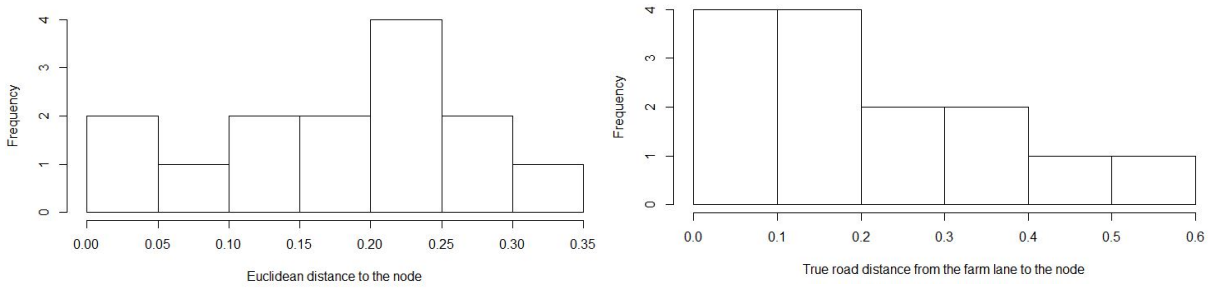
Figure 26 shows the spread of the distances for both methods. Using the true road distances, there are farm that have a far higher true road distance than Euclidean. This could be due to the farm being at the end of a main road and the intersection is further away. Examples of these can be seen in figure 25. It could also be due to the farm having

a long farm lane that is further down the road than the farmhouse. As is evident the true road distances are positively skewed, most of the distances between the nodes and the farm entrance are small.



(a) (b)

Figure 25: Figure (a) shows how a farm could have road distance, y of 0. Figure (b) shows how a farm can have a large road distance but a short Euclidean distance.



(a) (b)

Figure 26: Figure (a) shows a histogram of Euclidean distances to the nearest node, Figure (b) shows the histogram of the true road distances between the farm entrance and the nearest node.

Looking at the histograms in figure 26 it seems as though the means of the Euclidean distance and also the true road distance are not the same. A paired t-test confirms this with the mean of the differences being -0.0414 with a p value of 0.1246 and a 95% confidence interval of -0.09577156 and 0.01305728.

The following example will use the distances between the infected farm and susceptible farms private farm tracks, this is the true road distances between the two. This will then highlight the difference in the infection hazard between this method, and using the Euclidean distance as in the example above.

Figure 27 shows a diagram of the distance that will be used instead of the Euclidean distance between the farm houses. These are the true road distances between the infected farm track and the susceptible farm track.

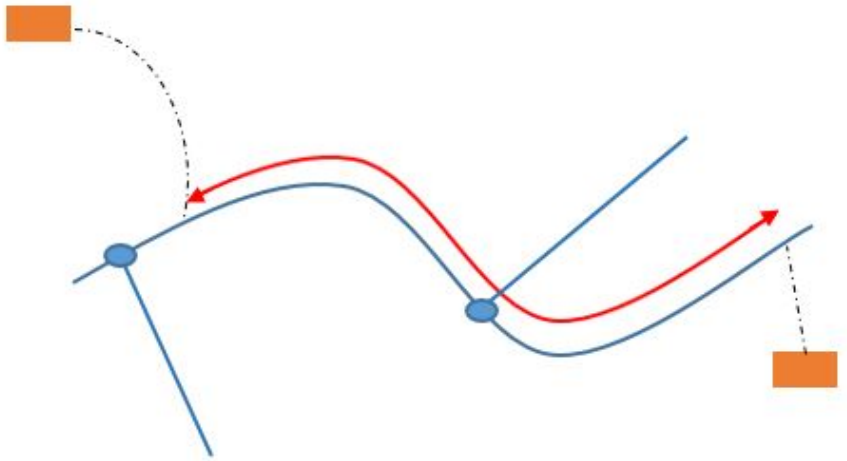


Figure 27: The true road distance between the two farm tracks.

These have been measured from the infected farm I to the susceptible farms for each farm. This is a test to see how using this distance, as it would be the distance that would be travelled by vehicles and the route used that does not incur any geographical barriers for the disease. The test aims to see if using the true road distance between the infected farms changes the infection hazard rates of the farms.

Savill *et al.* used the nodes as a means to calculate the true road distance however in this experiment the closest node to each farm was not the same as the closest node to the infected farm therefore a better way of calculating the distance would be to use the distance between the farm tracks as in figure 27.

Farm	No. cattle	No. sheep	dist from I	$k(d_{ij})$	Infection Hazard
A	200	350	1.709	0.3691	490674.4
B	120	200	1.054	0.4869	384356.9
C	105	100	0.901	0.5260	331529.7
D	40	60	0.709	0.5851	150833.5
E	0	130	0.636	0.6112	64149.75
F	50	20	0.381	0.7244	201170.5
G	20	0	0.763	0.5672	59369.37
H	60	50	0.545	0.6472	229365.4
I	75	75	NA	NA	NA
J	35	70	0.6	0.6250	149800.2
K	15	100	0.163	0.8598	136914.1
L	0	40	0.436	0.6964	22487.46
M	10	120	0.218	0.8210	122504.2
N	100	15	0.654	0.6046	323730.6

Table 10: The simulated farms information that will be used to model the epidemic using the true road distance between farm tracks.

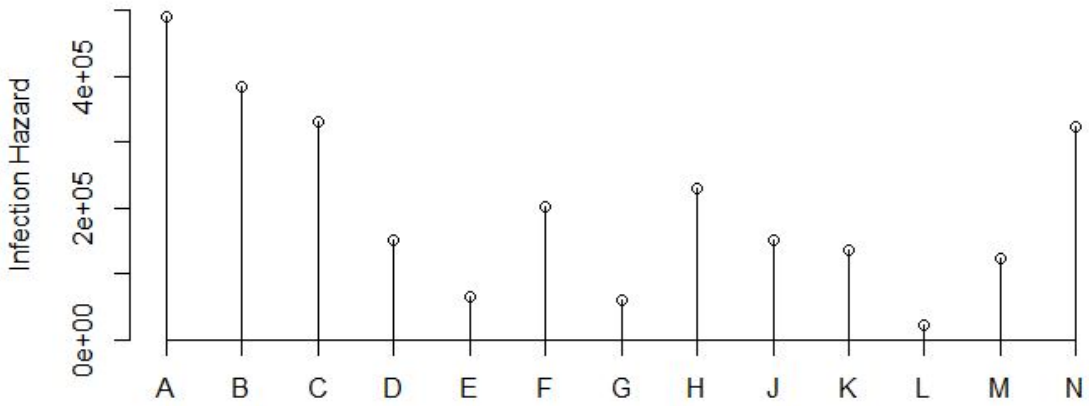


Figure 28: The infection hazard when using the true road distance between the farm tracks.

As was said above, figure 28 confirms that even the change in distance between the two nodes is not enough to drastically change the infection hazard rates. It is the number

of animals that mainly control those values. Savill *et al.* (2006) confirms that using the Euclidean distance for most cases is the most appropriate measure.

7.4 The log-likelihood of farm B being infected

This section looks at modelling the likelihood of infection to a farm B on Fair Isle. The previous sections looked at different methods to calculate the distances from the susceptible and infected farms and the infection hazards however this section takes it one step further and calculated the log-likelihood of a certain farm being infected at certain times.

The order the farms are infected have been taken from the discrete time model in section 3.1.

$t = 0$ to 4: No animals are infectious, but one animal is infected in the latent period.

$t = 5$: One animal is in the infectious period but it takes one day to become infected therefore no farms have been infected yet.

$t = 6$: Farm I becomes infected.

$t = 7$: Farm H becomes infected.

$t = 8$: Farm L becomes infected.

$t = 9$: Farm C becomes infected.

$t = 10$ to 11: No more farms are infected as there are no farms in the infectious period.

Figure 29 shows the change in the log-likelihood that farm B is infected over time. The red line shows the force of infection at time t. As is evident when it gets to $t = 9$, the force of infection stays constant. This is due to no new animals are infected in those times however as time continues the likelihood that the farm will become infected increases. This is due to the infected animals not being removed or culled yet so the infection is still spreading.

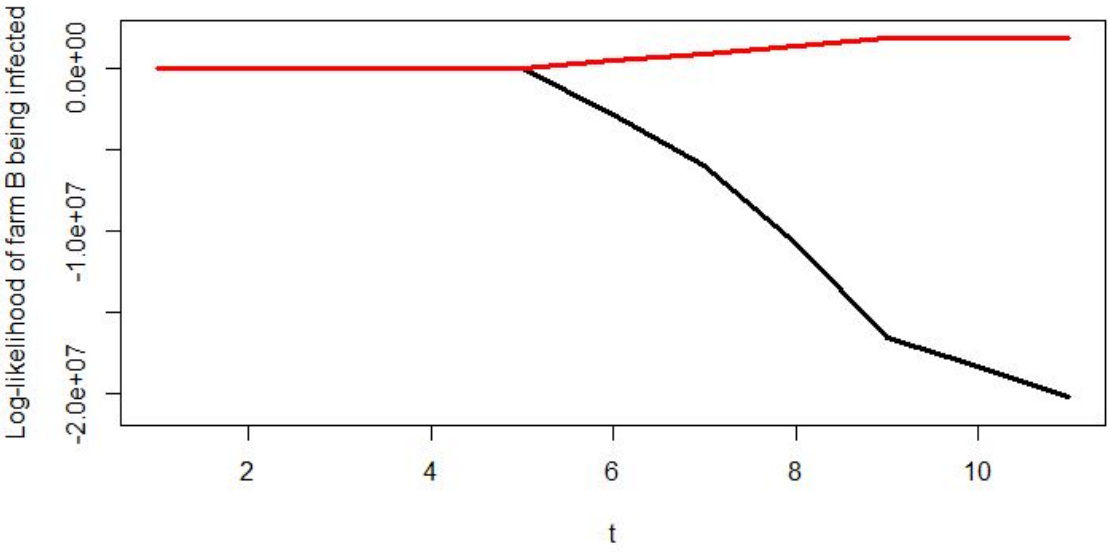


Figure 29: The log-likelihood of farm B being infected at time t.

8 The Future

Section 5 concluded that using Euclidean distance as a measure of the distance between a infectious and susceptible farm when calculating the transmission risk was a much better measure than using the shortest road distance. This being said Savill *et al.* (2006) state that for future epidemics and the modeling of disease transmission geographically,the shortest distance on road networks should be used. This is due to geographical barriers such as rivers and mountains that will change the way that the disease is spread for certain parts of the country.

8.1 Is proactive culling the future?

The 2001 foot and mouth epidemic was a tragic outbreak that forced 10 million animals to be culled, an article in *The Daily Telegraph* (2002) states that this is a much larger number than the Government initially predicted. This is due to reports of holding the infection and also certain radius policies that ensured all animals susceptible and in the danger zone of catching the disease were culled.

The BBC produced an article in 2011 stating if an epidemic like the 2001 outbreak happened again, proactive culling would not be the Governments preferred method. The epidemic in 2001 cost the public and private sector a total of over 8 billion, this was due to compensation for the farmers and increase in staff and costs of burial pits.

Chis Ster and Ferguson (2007) stated that there was no evidence to suggest that proactive culling did not help the epidemic and the BBC in 2011 agreed, but said the Government would take a different approach to the outbreak if it were to happen again. In the future they would use a more targeted approach and closely monitor the animals health and only slaughter when the symptoms started occurring. This is because the rate of infection they have studied is actually less than two days where previously it was between 4 and 8 days. This changes things and had enabled scientists to come to the realisation that until the animals are showing symptoms, they are not infectious.

8.2 Improvements on statistical models

To improve the accuracy of statistical models an idea would be to look at the field boundaries between farms to calculate the probability of farms coming into contact. Although this information will be time consuming to obtain it will allow further accuracy when calculating the transmission rates. Currently most of the models use the distance between farmhouses, however the animals are more likely to be in a field some distance

away from the farmhouse and in fact could be next to another farms livestock making the probability of contact close to 1. This would completely change the models result.

8.3 Future developments of this project

To develop this project further, analysis relating to the within farm disease spread would be interesting to look at. This would require extensive data for each farm but would enable investigations on the different transmission channels for the disease.

To develop some of the models from this report, further parameters could be used to show the different ways the animals can catch the disease, for example airborne and direct contact. Each of these will have a different transmission rate but using this would incur further parameter estimation for these and would also require data of how the animals caught the disease so that validation of the model could take place. This would be very useful however it would be difficult not to over specify the model with the increase in number of parameters used.

Comparing the parameters such as latency and infectious days could be compared to other epidemic such as the mad cow disease. This way would enable identification on what species are the most prone to diseases and how much the latency and infectious periods last for each disease.

Researching other foot and mouth epidemics outside of the UK could help to know how much the geography of the country did contribute to the spread. In countries such as Africa where some parts of the country are not as densely populated the spread of the disease may be slower due to less contact between farms.

Collecting data regarding the social implications on farmers throughout the epidemic would also be an interesting topic to explore. There are data regarding the effects the epidemic had on farmers post epidemic however it would be interesting to see how these changed throughout the months of the outbreak.

Finally data is needed for further analysis of this project. The data collected from the epidemic is highly confidential and therefore very difficult to gain access to even on a between farm level. Any new models to be developed will need the data for validation purposes and this is restricted access.

9 Discussion

Many statistical models were developed after both the 1967 and 2001 foot and mouth epidemic. All with different objectives and methods for analysing the spread of the epidemic and the efficiency of the control measures implemented.

9.1 The difference in the models

Haydon *et al.* (1997) used the data collected from the 1967 epidemic to model the transmission rates between the herds throughout the epidemic. This model was useful at the time as it allowed identification of the very high transmission rates in the first ten days of the epidemic. This was investigated and concluded to be due to the environment having the perfect conditions for wind borne disease spread for that time period.

When compared to more recent models it seems to be lacking complexity. The more recent models of Chis Ster and Ferguson (2007) contain more parameters such as susceptibility and infectivity rates and also make the distinction between species. This makes the model more realistic as cattle are much more susceptible than sheep so it would be inaccurate to calculate the transmission of them together without any differentiation. Although many papers have a difference in susceptibility and infectivity rates such as Chis Ster and Ferguson (2007) having a cattle susceptibility of 6.8 and Bravo de Rueda *et al.* 2014 use 2.2. These estimations can be vital when modelling so they need to be used consistently through any model.

Chis Ster and Ferguson (2007) indicated the importance of the number of animals held on both the susceptible and infectious farm over the distance between them both. The Fair Isle example demonstrated this to highlight that the farm furthest away had the highest infection hazard because it had the most animals held. This goes to show that the control measures in the future need to be prioritising testing the larger farms over the smaller holdings.

Using the true road distance as well as the Euclidean distance proved that changing the distance had little effect on the overall infection hazard of the susceptible farms and that the number of animals held on the farm was much more influential than changing the distance by a mile or two.

9.2 Reality of the epidemic

When analysing epidemics it is easy to get lost in the mathematics and the logic behind the models however it is important to take into consideration the reality of the event.

Most of these models have the assumption that each animal has a fixed susceptibility or infectivity rate and that they will all show symptoms at the same time. In reality this is not the case. For example with people, when a person gets ill one person could show symptoms a couple of days into catching the virus where as the next person could be well for a week after catching the virus. Animals are the same. Applying this to the models would make them far too complex and tailored to the farm being analysed however it is something that needs to be understood.

Another factor that the models do not consider is the different types of ways the disease could be spread. This is through the air, contaminated premises and direct contact between contaminated animals. Each of these channels of transmission will have different transmission rates. The data that is collected for analysis does not include the way in which the livestock caught the disease. Although the vet could have an idea for example if the livestock had been taken to the same abbotoir as an infected animal such as the initial outbreak in Essex, nothing is certain and therefore modelling the different types of transmission is difficult as the data is merely impossible to obtain.

9.3 Modelling difficulties

The lack of data available from the epidemics causes difficulty in the analysis. The lack of information on farm boundaries and the topology of the farms makes it difficult to know where the animals have crossed and potentially contaminated other areas. The Ordanace survey does show rivers and streams but it is difficult to know if these streams are ditches that the animals cannot cross, in that case the disease would stop there. Or if it is a light stream they walk through and can contaminate the other side.

The other difficulty is modelling when the animals have had direct contact. Two farms could have connecting fields both with livestock contained. The farm houses could be miles apart so the models would predict that the animals may not come into contact but in actual fact they can contaminate each herd over one fence. This proves difficulty in developing a model which is completely accurate as there would be too many parameters to consider with every field boundary.

Some models do estimate the within farm epidemic such as Thornley and France (2007) however this parameter is not as critical as the between far, epidemic. This is due to the disease spreading so quickly and the culling policy of removing all the animals on the farm even if they show no symptoms. Therefore even if half of the animals in the herd were infected and half were not, they would all be removed anyway regardless of the estimation.

9.4 Social implications

The psychological effects on farmers and their families have to be taken into consideration. Even though many people, including the National Farmers Union had strong disagreements on some of the valuation of the culled cattle. They thought the Government was paying too much to farmers as compensation and they should have been using the money to improve control measures instead of culling animals that were not confirmed as infected.

Stress and depression levels were something that increased throughout the farming industry and these had permanent impact on UK farming. Many farms were closed after the outbreak as farmers were left heartbroken and without business. Some never bought any livestock again and broke away from farming for good.

It was not only farmers that were effected by the outbreak but the staff helping control the disease were also affected psychologically. Vets making the decisions to cull hundreds of animals were highly stressed due to the pressure and responsibility they were handed. The military that were culling and burning the thousands of animals at a time were being largely affected too. The disease reached people across the region and due to the scale of the disease and its publicity, the general public were largely affected.

Overall the main findings from this report were that the number of animals in the herds were much more influential than the distance from the susceptible to the infected farms. Although pigs contributed to the epidemic as it was them that were infected in the first confirmed case, they did not contribute as much to the spread of the disease than cattle and sheep, this is a reason for them not being included in the statistical models. Most of the statistical models were analysing the effectiveness of the control policies implemented by the Government. The main conclusion with the models and also the UK residents is that the Government did not act quick enough and they did not have the necessary resources put in place for such an epidemic.

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A Appendix

Figure 1

```
sheep<-c(1415,1427,1427,1376,1326,1406,1465,1481,1267,1049,1129,
1101,1118,1094,1092,1104,1026,978)
cattle<-c(672,681,689,666,645,632,627,635,606,541,563,570,563,545,529)
pigs<-c(650,664,663,617,638,662,625,595,496,465,441,391,386,379,383,377,
370,375)

layout(rbind(1,2), heights=c(7,1))
plot(0,0,type="n",axes=FALSE,xlab="Time",
ylab="Number of Animals (000's)",xlim=c(0,20),ylim=c(0,2000))
axis(1,at=c(1:18),labels=c("1992","1993","1994","1995","1996","1997","1998",
"1999","2000","2001","2002","2003","2004","2005","2006","2997","2008",
"2009"),pos=0)
axis(2)
lines(sheep,lty=2,col="red")
lines(cattle,lty=3,col="blue")
lines(pigs,lty=4,col="green")
lines(c(10,10),c(0,1049),lty=1)

par(mar=c(0, 0, 0, 0))
llegend('top','groups',c("Sheep","Cattle","Pigs"), lty = c(2,3,4),
col=c('red','blue','green'),ncol=3,bty ="n")
```

Figure 2

```
x<-c(1991,1992,1993,1994,1995,1996,1997,1998,1999,2000,2002,2003,2004,2005)
y<-c(70,86,103,111,111,133,121,90,84,87,107,120,118,111)
plot(x,y,type="n",xlab="Year",ylab="Price of Lamb (p)",ylim=c(70,140))
lines(x,y)

x<-c(1992,1993,1994,1995,1996,1997,1998,1999,2000,2001,2002,2003,2004)
y<-c(65,59,73,118,114,103,91,66,66,69,68,74,82)
plot(x,y,type="n",xlab="Year",ylab="Price of Wool (p)",ylim=c(55,120))
lines(x,y)
```

Figure 7

```
x<-0:10
y<-c(96,93,89,82,74,72,64,58,50,46,9)
par(mar=c(12,5,2,5))
names(y)<-c("Visiting friends","Visiting family",
            "Going to the pub","Attending agrictultural shows",
            "Shopping further afield","Going to church","Shopping locally",
            "Attending School","Attending special occasions","Attending
             off-farm work",
            "Receiving healthcare")
barplot(y,ylim=c(0,100),las=2,col="darkblue",ylab="Percentage \%")
```

Figure 8

```
x<-c(1,2,3)
y<-c(0.27,1.0,6.8)
plot(x,y,ylim=c(0,7),ylab="Susceptibility",axes=FALSE,xlab="")
axis(2)
axis(1,at=c(1,2,3),labels=c("Pigs","Sheep","Cattle"),pos=0)
lines(c(2,2),c(0,1.0),lty=3)
lines(c(3,3),c(0,6.8),lty=4)
lines(c(1,1),c(0,0.27),lty=1)
```

Figure 9

```
x<-c(1,2,3)
y<-c(4.7334,1.38,8.946)
plot(x,y,ylim=c(0,10),ylab="Infectivity",axes=FALSE,xlab="")
axis(2)
axis(1,at=c(1,2,3),labels=c("Pigs","Sheep","Cattle"),pos=0)
lines(c(2,2),c(0,1.38),lty=3)
lines(c(3,3),c(0,8.946),lty=4)
lines(c(1,1),c(0,4.7334),lty=1)
```

Figure 10

```
t<-0:46
s1<-c(2159,2159,2159,2159,2159,2159,2009,1899,1859,1654,
```

```
     1654,1654,1334,1204,654,549,479,379,264,134,114,0,
     0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0)
l1<-c(1,1,1,1,1,0,150,260,300,505,505,355,565,655,1000,
     1105,1175,955,940,520,435,479,379,264,134,114,0,
     0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0)
i1<-c(0,0,0,0,0,1,1,1,1,0,0,150,260,300,505,355,245,525,
     450,1000,1105,855,825,390,415,365,379,264,134,114,
     0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0)
r1<-c(0,0,0,0,0,0,0,0,0,0,1,0,0,0,0,0,150,110,40,205,0,0,
     320,130,550,105,70,100,115,130,20,114,0,0,0,0,0,0,
     0,0,0,0,0,0,0,0,0,0)

plot(t,s1,ylab="Number of animals",type="n",ylim=c(0,3000))
lines(t,s1,col="red",lty=1)
lines(t,l1,col="blue",lty=2)
lines(t,i1,col="darkgreen",lty=3)
lines(t,r1,col="purple",lty=4)
legend('topright','groups',c("Susceptible","Latent","Infectious",
       "Removed"), lty = c(1,2,3,4),
       col=c('red','blue','darkgreen',"purple"),ncol=4,bty ="n")
```

Figure 11

```
days<-0:31
beta<-c(0,0,0,0,0,0.0695,0.0548,0.0121,0.11,0,0.2275,0.0022,0.0267,
        0.0312,0.0485,0.0519,0.0536,0.0573,0.0606,0.0651,0.0658,0.0694,
        0.0694,0.0693,0.0695,0.0694,0.0695,0.0695,0.0695,0.0695,0.0695,
        0.0695)
R<-c(0,0,0,0,0,37.512,27.523,5.744,51.122,0,94.071,0.910,8.904,
     9.391,7.929,7.123,6.419,5.429,3.999,2.180,1.875,0,0,0,0,0,
     0,0,0,0,0,0)
par(mar=c(5,5,5,5))
plot(days, beta,pch=0)
abline(v=5,lty=3)
par(new=TRUE)
plot(days,R,type="l",axes=FALSE,bty="n",col="red",ylab="")
axis(side=4,at=pretty(range(R)))
```

```
mtext("R",side=4,line=3)
```

Figure 12

```
t<-0:46
s1<-c(2159,2159,2159,2159,2159,2159,2009,1899,1859,1654,
      1654,1654,1334,1204,654,549,479,379,264,134,114,0,
      0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0)
l1<-c(1,1,1,1,1,0,150,260,300,505,505,355,565,655,1000,
      1105,1175,955,940,520,435,479,379,264,134,114,0,
      0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0)
i1<-c(0,0,0,0,0,1,1,1,1,0,0,150,260,300,505,355,245,525,
      450,1000,1105,855,825,390,415,365,379,264,134,114,
      0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0)
r1<-c(0,0,0,0,0,0,0,0,0,0,1,0,0,0,0,0,150,110,40,205,0,0,
      320,130,550,105,70,100,115,130,20,114,0,0,0,0,0,0,
      0,0,0,0,0,0,0,0,0,0,0)

s2<-c(2159,2159,2159,2159,2009,1899,1899,1899,1859,1654,
      1334,1334,1204,654,549,479,379,264,134,114,0,
      0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0)

l2<-c(1,1,1,0,150,260,260,110,40,245,565,525,450,680,
      785,725,275,285,345,265,264,134,114,0,0,0,0,0,0,0,
      0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0)

i2<-c(0,0,0,1,1,0,0,150,260,110,0,40,245,525,320,130,680,
      655,175,170,215,245,150,134,114,0,0,0,0,0,0,0,0,0,
      0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0)

r2<-c(0,0,0,0,0,0,1,0,0,0,150,110,0,0,40,205,320,0,130,550,
      105,70,108,115,130,20,114,0,0,0,0,0,0,0,0,0,0,0,0,0,0,
      0,0,0,0,0,0,0,0,0)

s3<-c(2159,2159,2159,2009,2009,2009,1899,1899,1899,1859,1859,1859,
      1654,1654,1654,1334,1334,1334,1204,1204,1204,654,654,654,549,549,
      549,479,479,479,379,379,379,264,264,264,134,134,134,114,114,114,
```

```
      0,0,0,0,0)
l3<-c(1,1,0,150,150,0,110,110,0,40,40,0,205,205,0,320,320,0,130,130,
      550,550,0, 105,105,0,70,70,0,100,100,0,115,115,0,130,130,0,20,20,
      0,114,114,0,0,0,0)

i3<-c(0,0,1,0,0,150,0,0,110,0,0,40,0,0,205,0,0,320,0,0,130,0,0,550,0,0,
       105,0,0,70,0,0,100,0,0,115,0,0,130,0,0,20,0,0,114,0,0)
r3<-c(0,0,0,1,0,0,150,0,0,110,0,0,40,0,0,205,0,0,320,0,0,130,0,0,550,0,
       0,105,0,0,70,0,0,100,0,0,115,0,0,130,0,0,20,0,0,114,0)

plot(t,s1,ylab="Number of animals",main="Susceptible animals",type="n",
      ylim=c(0,3000))
lines(t,s1,col="red",lty=1)
lines(t,s2,col="blue",lty=2)
lines(t,s3,col="darkgreen",lty=3)
legend(30,3000,c(expression(paste(sigma==5,", ",nu==4),paste(sigma==3,
       ", ",nu==2),paste(sigma==2,", ",nu==1))),lty=c(1,2,3),col=c("red",
       "blue","darkgreen"),bty="n")

plot(t,s1,ylab="Number of animals",main="Latent Animals",type="n",
       ylim=c(0,1500))
lines(t,l1,col="red",lty=1)
lines(t,l2,col="blue",lty=2)
lines(t,l3,col="darkgreen",lty=3)
legend(30,1500,c(expression(paste(sigma==5,", ",nu==4),paste(sigma==3,",
        ",nu==2),paste(sigma==2,", ",nu==1))),lty=c(1,2,3),col=c("red",
        "blue","darkgreen"),bty="n")

plot(t,s1,ylab="Number of animals",main="Infectious Animals",type="n",
        ylim=c(0,1500))
lines(t,i1,col="red",lty=1)
lines(t,i2,col="blue",lty=2)
lines(t,i3,col="darkgreen",lty=3)
legend(30,1500,c(expression(paste(sigma==5,", ",nu==4),
        paste(sigma==3,", ",nu==2),paste(sigma==2,", ",nu==1))),
        lty=c(1,2,3),col=c("red","blue","darkgreen"),bty="n")
```

```
plot(t,s1,ylab="Number of animals",main="Removed Animals",type="n",
        ylim=c(0,800))
lines(t,r1,col="red",lty=1)
lines(t,r2,col="blue",lty=2)
lines(t,r3,col="darkgreen",lty=3)
legend(30,800,c(expression(paste(sigma==5,", ",nu==4),paste(sigma==3,
        ", ",nu==2),paste(sigma==2,", ",nu==1))),lty=c(1,2,3),
        col=c("red","blue","darkgreen"),bty="n")
```

Figure 13

```
curve((1+x/1)\^(-1),0,10,ylim=c(0,1),xlab="Distance d",
ylab=expression(k(d)))

curve((1+x/2)\^(-1),0,10,add=TRUE,lty=2)

legend(6,1,c(expression(paste(a==1,", ",gamma==1),paste(a==2,", ",
gamma==1))),lty=c(1,2),bty="n")
```

Figure 14

```
curve((1+x/1)\^(-2),0,10,ylim=c(0,1),xlab="Distance d",
ylab=expression(k(d)),lty=3)

curve((1+x/2)\^(-2),0,10,add=TRUE,lty=4)

legend(6,1,c(expression(paste(a==1,", ",gamma==2),paste(a==2,", ",
gamma==2))),lty=c(3,4),bty="n")
```

Figure 15

```
data<-as.matrix(data)

rownames(data) <- c("100","200","300","400","500","600","700","800","900",
 "1000","1100","1200","1300","1400","1500","1600","1700", "1800","1900",
"2000","2100","2200","2300","2400","2500")

colnames(data) <- c("100","200","300","400","500","600","700","800","900",
```

```
 "1000","1100","1200","1300","1400","1500","1600","1700","1800","1900",
"2000","2100","2200","2300","2400")

levelplot(data,col.regions = heat.colors(100),xlab = 'Number
of susceptible animals', ylab = 'Number of infected animals',
scales=list(tck = c(1,0),
x=list(rot=90)))
```

Figure 16

```
curve((((6.8*x)+(1*50))*((8.94*50)+(1.38*50))),from=0, to=500 ,
ylim=c(0,1500000),xlab="Number of susceptible animals",
ylab="Infection Hazard")

curve((((6.8*50)+(1*x))*((8.94*50)+(1.38*50))),from=0, to=500
,add=TRUE,lty=2)

legend("topleft",c(expression(paste("Change in number of susceptible
 cattle"),paste("Change in number of susceptible sheep"))),
lty=c(1,2),bty="n")
```

Figure 20

```
x<-c(1,2,3,4,5,6,7,8,9,10,11,12,13)
y<-c(553848.8,478466.2,437665.3,188156.5,81355.81,231513.7,
84409.84,313601.4,196459.3,138461.9,24650.38,117488.3,347695.1)
plot(x,y,ylim=c(0,600000),ylab="Infection Hazard",axes=FALSE,xlab="")
axis(2)
axis(1,at=c(1,2,3,4,5,6,7,8,9,10,11,12,13),labels=c("A","B",
       "C","D","E","F","G","H","J","K","L","M","N"),pos=0)
lines(c(1,1),c(0,553848.8),lty=1)
lines(c(2,2),c(0,478466.2),lty=1)
lines(c(3,3),c(0,437665.3),lty=1)
lines(c(4,4),c(0,188156.5),lty=1)
lines(c(5,5),c(0,81355.81),lty=1)
lines(c(6,6),c(0,231513.7),lty=1)
lines(c(7,7),c(0,84409.84),lty=1)
lines(c(8,8),c(0,313601.4),lty=1)
```

```
lines(c(9,9),c(0,196459.3),lty=1)
lines(c(10,10),c(0,138461.9),lty=1)
lines(c(11,11),c(0,24650.38),lty=1)
lines(c(12,12),c(0,117488.3),lty=1)
lines(c(13,13),c(0,347695.1),lty=1)
```

Figure 21

```
x<-c(1,2,3,4,5,6,7,8,9,10,11,12,13)
y<-c(1.4,0.65,0.44,0.37,0.29,0.2,0.24,0.13,0.22,0.15,0.31,0.27,0.54)
plot(x,y,ylim=c(0,1.5),ylab="Distance from farm I",axes=FALSE,xlab="")
axis(2)
axis(1,at=c(1,2,3,4,5,6,7,8,9,10,11,12,13),labels=c("A","B","C","D","E",
"F","G","H","J","K","L","M","N"),pos=0)
lines(c(1,1),c(0,1.4),lty=1)
lines(c(2,2),c(0,0.65),lty=1)
lines(c(3,3),c(0,0.44),lty=1)
lines(c(4,4),c(0,0.37),lty=1)
lines(c(5,5),c(0,0.29),lty=1)
lines(c(6,6),c(0,0.2),lty=1)
lines(c(7,7),c(0,0.24),lty=1)
lines(c(8,8),c(0,0.13),lty=1)
lines(c(9,9),c(0,0.22),lty=1)
lines(c(10,10),c(0,0.15),lty=1)
lines(c(11,11),c(0,0.31),lty=1)
lines(c(12,12),c(0,0.27),lty=1)
lines(c(13,13),c(0,0.54),lty=1)
```

Figure 22

```
sheep<-c(350,200,100,60,130,20,0,50,70,100,40,120,15)
cows<-c(200,120,105,40,0,50,20,60,35,15,0,10,100)
farms<-c("A","B","C","D","E","F","G","H","J","K","L","M","N")
table<-(rbind( cows, sheep))
barplot(table,
        xlab="Farm",ylab="Number of animals", col=c("navyblue","grey"),
       legend =c("Cattle","Sheep"), beside=TRUE, names.arg=c("A","B",
       "C","D","E","F","G","H","J","K","L","M","N")
      ,ylim=c(0,400))
```

Figure 23

```
x<-c(1,2,3,4,5,6,7,8,9,10,11,12)
y<-c(987460.3,857656.5,780316.1,339895.7,148704.33,415839.3,
156927.86,352831.9,242347.7,44557.15,208044.49,632870.2)
plot(x,y,ylim=c(0,1000000),ylab="The force of infection ",axes=FALSE,
xlab="")
axis(2)
axis(1,at=c(1,2,3,4,5,6,7,8,9,10,11,12),labels=c("A","B","C","D","E",
"F","G","J","K","L","M","N"),pos=0)
lines(c(1,1),c(0,987460.3),lty=1)
lines(c(2,2),c(0,857656.5),lty=1)
lines(c(3,3),c(0,780316.1),lty=1)
lines(c(4,4),c(0,339895.7),lty=1)
lines(c(5,5),c(0,148704.33),lty=1)
lines(c(6,6),c(0,415839.3),lty=1)
lines(c(7,7),c(0,156927.86),lty=1)
lines(c(8,8),c(0,352831.9),lty=1)
lines(c(9,9),c(0,242347.7),lty=1)
lines(c(10,10),c(0,44557.15),lty=1)
lines(c(11,11),c(0,208044.49),lty=1)
lines(c(12,12),c(0,632870.2),lty=1)
```

Figure 24

```
x<-c(0.327,0.18,0.127,0.018,0.18,0.054,0.254,0.236,0.236,0.127,0.254,
0.036,0.218,0.236)
y<-c(0.509,0.145,0.2,0.036,0.36,0.072,0.472,0.236,0.309,0.09,0.27,
0.036,0.2,0.127)
lab<-c("A","B","C","D","E","F","G","H","I","J","K","L","M","N")
plot(x,y,xlab="Euclidean distance from the farmhouse to the node",
ylab="Distance from the farm lane to the node",pch=16)
text(x, y, labels=lab, cex= 0.7,pos=4)
lm(x~y)
abline(lm(x~y),col="red")
abline(0,1,col="blue")
```

Figure 26

```
hist(x,xlab="Euclidean distance to the node",main=" ")
hist(y,xlab="True road distance from the farm lane to the node",main=" ")
```

Figure 29

```
x<-1:11
y<-c(0,0,0,0,0,-2870791.52,-6003589.56,-10812050.59,-16543071.85,
-18311911.64,-20219311.43)
plot(x,y,xlab="t", ylab="Log-likelihood of farm B being infected",type="l"
,lwd=3,ylim=c(-21000000,2000000))
z<-c(0,0,0,0,0,478466.2,857656.5,1351507.09,1838119.79,
1838119.79,1838119.79)
lines(x,z,col="red",pch=16,lwd=3)
```