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In this thesis we create and analyze a model for the spread of Newcastle disease in a farm population. Our model takes the stable structure of Swedish farms into account, allowing the virus to spread both inside the stable and between stables. We derive the basic reproduction number R_0 for the setting with only a single stable along with the next-generation matrix of which the dominant eigenvalue is the basic reproduction number for more than a single stable. With programmed simulations in R we analyze the final size of the epidemic for varying values of parameters and R_0. We draw conclusions about how various parameters affect R_0 on their own and look at the effect of the free parameters on the probability that the disease never leaves a stable where it started. We find that without knowing more about the infectivity and mortality of the virus the only way that stable separation is a sufficent way to safeguard against further spread is if infected stables are unable to contact other stables and the farmer is able to quickly recognize symptoms of NDV.

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Contents

1 Introduction

Newcastle Disease (ND) was first discovered on Java in Indonesia but received its name from an outbreak in Newcastle-upon-Tyne in England, both during the 1920's (World organization for animal health). It is a contagious virus disease which mainly infect different species of birds leading to respiratory issues, thin-shelled eggs, reduced egg production and often ultimately to death. There is no known treatment. Mortality in infected birds depends on the strain of the virus which is either lentogenic (negligible mortality rate), mesogenic (low mortality rate) or velogenic (high mortality rate). Different strains of NDV occurs in various parts of the world to differing extent. Lentogenic strains are for instance worldwide while velogenic strains are widely spread in Africa, the Middle East and Asia (World organization for animal health).

Since the first identified outbreak in 1926 there have been several devastating outbreaks where infection in poultry farms have led to grave economic losses as well as the death of millions of birds in attempts to eradicate the disease amongst commercial chickens. Noteable outbreaks have been those in California in 1971 and in 2002 where the estimated loss has been 12 million repsectively 3.16 million birds (California department of food and agriculture). Sweden has so far been able to avoid disastrous outbreaks as that in California but several small scale outbreaks have been reported, the most recent (as of the time of writing) being in November 2017 where Newcastle disease was found in a farm of 26 000 laying hens (Sveriges Radio, Utbrott av Newcastlesjuka på skånsk gård).

ND is highly contagious and spreads from birds to birds through direct contact, either via feces of the infected animal or through secretions from nose and mouth. The virus survives for longer durations in feces and can be picked up on tools, clothing or machines by interacting humans thus spreading the disease further if cleanliness routines are not respected. No treatment exists for the disease and neither does a vaccine offering comeplete immunity (World organization for animal health).

In this thesis we construct a model based around NDV in a farm setting. Our model is a generalization of a Reed-Frost model containing extensions for an animal population. We analytically derive the basic reproduction number for our model and use simulations coded in R to answer questions relating to the model and the farm setting. Our goal is to find out whether or not dividing poultry into stables is an effective way to safeguard against the spread of NDV. Normally when NDV - or any other type of harmful epizootic - is discovered in a farm population, the population is immediately culled to avoid further spread of the disease. We wish to look into what the possibilities for spread between stables are and especially with what certainty a farmer may need to fully cull a herd when cases of NDV appear on the farm.

2 Model

2.1 Population and contacts

To model the spread of NDV in a farm population of laying hens we have opted for a model based around the four types of poultry farms existent in Sweden. These four types are **free-roaming indoors**, **free-roaming outdoors**, **ecological** and **caged**. As we will later see, under certain restrictions, the first three models will behave similarily to each other while the model for the caged poultry will remain unique. We will only be working with laying hens and ignoring various types of broiler farms. This simplifies the model and allows us to disregard the life-times of birds.

In all four farms birds are first and foremost kept in stables for the majority of their lives. In both free-roaming farms, birds are allowed to roam without restriction inside their habitat without the constraints of cages. In the indoors farm they are confined to their stables exclusively while in the outdoors farm the birds also receive a couple of hours outdoors in an enclosure. Both free-roaming models come with further population and size constraints set by Jordbruksverket (department of agriculture.) In the ecological farm birds live identical lives to the birds in the free-roaming outdoors farm where the main difference is in sharper population constraints. Lastly, the birds on the caged farms are housed in cages arranged in grids. These too are situated inside stables (Jordbruksverket, SJVFS).

We assume that poultry farms consist of N laying hens making up the entire population. These N hens are then separated into k unique stables where H_i is the total population of stable i for $i = 1, ..., k$ such that

$$\sum_{i=1}^{k} H_i = N.$$

Further restrictions on H_i will depend on the type of farm in question.

We consider the poultry population to be of the same *type*, meaning that we do not differentiate between the birds on the farm. They all share the same traits and characteristics in our model - the population is *homogeneous*. Furthermore, we also assume that N remains constant throughout the duration of the epizootic, that is we allow for no in- or outflow of chickens into the farm, i.e., the population is *closed*.

Besides the traditional in-stable-interaction we allow for contact to happen between populations of different stables. Since NDV is good at surviving outside the body of the birds in for instance infected feces we find it reasonable to assume that contact, via human interaction, may happen between two given stables i and j. If cleanliness routines are not properly respected there is a slight probability that infection successfully spreads. We assume that farmers are aware of potential NDV and very careful with respect to cleanliness routines making the probability of spread from stable i to stable j low for $i \neq j$.

Free-roaming indoors: The simplest farm is the one where the hens are allowed to roam freely indoors. The population N is divided into k unique stables with the only upper bound on H_i being due to the restrictions made by Jordbruksverket. For the free-roaming indoors farm this restriction is of each hen having at least $0.11m^2$ to move upon, or equivalently, each stable having no more than 9 hens per square meter available area (Jordbruksverket, SJVFS).

In the previous subsection we made an assumption of the population of hens being homogeneous and seeing as the H_i hens roam about freely inside the stables it seems fair to assume that we in this model would have a uniformly mixing population. That is, all birds have the same rate of contact with eachother inside the stables.

Free-roaming outdoors: The free-roaming outdoors farm is simply an extension of the indoors farm and uniform mixing still applies by the same argument as for the previous model. The constraints for the stables are the same as before with the additional constraint on the outside area having at least $4m^2$ available area per chicken (Jordbruksverket, SJVFS). To simplify the model we assume that the outdoor enclosures are paired with the stables, meaning that stable i has enclosure i which has no contact with either stable j or the enclosure of stable j for $i \neq j$. We also make no difference in contact rates when birds are in- or outdoors.

Ecological: As the outdoors farm was an extension of the indoors farm we find the ecological model to simply be an extension of the outdoors-farm. In the ecological model stables are allowed a maximum of 3000 chickens meaning that for stable i, $H_i \leq 3000$. Past this constraint the two farms behave in the same fashion (Jordbruksverket, SJVFS).

Caged: The last of the four types of farms is the setting where the birds are kept in cages for the entirety of the epidemic. This gives way to a quite different contact between the birds in the stable. First of all we again assume that the N birds are split into k unique stables where the birds are housed inside cages. Cages contain a maximum of 16 birds and require $0.06m^2$ of available area per bird living in the cage (giving a minimum size of $0.96m^2$ for a cage with 16 individuals in). Cages are placed in a $I \times J$-grid as in the example presented in figure 1 (Jordbruksverket, SJVFS).
Since birds no longer roam freely we are unable to assume that there is uniform mixing in the population of a caged farm and we must instead respect the grid arrangement of the cages. Adding to this, we must also respect the fact that susceptible individuals in a cage with an infected bird must run a higher risk of being infected themselves than a susceptible individual in a cage further along the grid would be. The grid in figure 1 is an example of how cages may be connected in this model (for $I = 3$, $J = n$) where nodes represent cages and edges represent the connections between these.

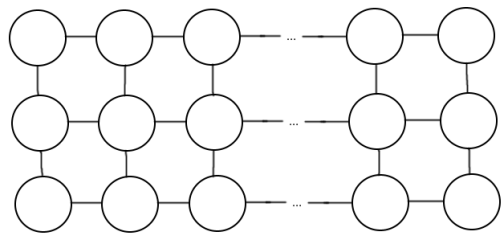

Figure 1: $3 \times n$-grid of cages

2.2 Model description

The four above farms have been the basis for our theoretical model. Instead of modelling all four we have chalked them down to a single concise model, flexible enough to represent the three first models. Since there is too big of a difference between the caged model and the other three we have decided to ignore deeper analysis of it and leaving the problem of spread in a caged population with a few brief comments outlined in section 2.5.

For the free-roaming indoors, free-roaming outdoors and ecological farms we opt for a traditional stochastic SIR compartment model to describe the spread of NDV in these populations. This model classifies the population N into 3 different states: susceptible, infectious and removed where the three states correspond to the clinical states of the individuals in the population. Removed translates to diseased individuals which no longer spread the disease which, in our case, means individuals which have died from the virus.

The SIR-model only allows for individuals to move from one state to the other in the direction S→I→R. Removed individuals may not get the disease as a result of already having suffered through the disease and those who enter the R state will remain there for the rest of the epidemic. Infected individuals will remain infectious for a while before entering the absorbing removed state. Susceptible individuals either remain susceptible for the full duration of the epidemic or become infected, eventually dying and entering the removed state. Usually the epidemic is stopped once the number of infectious individuals reach zero.

We let t denote the time of the epidemic, choosing $t = 0$ as the time of the start of the epidemic where we in our model have chosen to utilize discrete time. Next we let $S(t)$, $I(t)$ and $R(t)$ denote the number of susceptibles, infectious respectively removed at time t of the epidemic. Now at time $t = 0$ the population consists of N individuals inside k stables where stable i contains H_i individuals for $i = 1, 2, ..., k$. Since the population is constant we note that $N = S(t) + I(t) + R(t)$ throughout the whole epidemic. Furthermore, we choose $S(0) = n$ and $I(0) = m$ as our initial number of susceptibles and infectious with $R(0) = 0$. To extend upon this we may look at the epidemic at stable level where for stable i we write $S_i(t)$ for the number of susceptible individuals in stable i at time t and $I_i(t)$ for the number of infectious individuals in stable i at time t. For the

initial number of susceptibles in stable i we write n_i and accordingly for m_i such that

$$n = \sum_{i=1}^{k} n_i, \quad m = \sum_{i=1}^{k} m_i$$

We assume that the number of initial infectives m is small implying that it is possible that $m \leq k$ i.e. we allow for the possibility that $m_i = 0$ for some i.

At $t = 0$ the epidemic starts. Throughout the duration of the epidemic, infected individuals have contact with other individuals at a constant rate $\lambda > 0$. Contacted individuals are picked randomly from any of the three states and if an infected individual contacts a susceptible individual then this susceptible individual immediately becomes infected and will furthermore spread the disease in the same way. The individuals in the infected state remain infected and continue to attempt to infect others for a random duration I known as the infections period. After this time, the infected individuals enter the removed state (they die) and stop infecting others. This random variable I follows a specified distribution F_I such that $\mathbb{E}[I] = 1/\gamma$ where γ is the recovery rate parameter.

Normally an epidemic runs until the time $t = T$ where

$$T = \min\{t \geq 1 | I(t) = 0\},$$

that is, until the number of infectious individuals have reached 0. In our model we have added a second stopping criterion. When NDV is discovered in a farm the main preventive measure to avoid further spread is to cull the entire population of the farm. This means that if Newcastle disease is confirmed in the population of birds upon a farm, the individuals must be destroyed, naturally stopping the epidemic. We thus add another relevant parameter in the form of the detection of the disease which we consider a random variable D which follows a specified distribution F_D such that $\mathbb{E}[D] = 1/\alpha$ much like with the infectious times where $\alpha = \alpha(R(t))$ depends on the total number of removed individuals at time t.

For contact *between* stables our main parameter of interest is in the probability of eventual disesase transmission from one stable to another. Following in line with the already established SIR structure of the model and individuals we define a stable i to be infected at time t if $I_i(t) > 0$. At every time step t each infected stable contacts all the remaining stables at a rate β where $\beta = \beta(I_i(t))$ depends on the number of infectives in stable i at time t. If the contacted stable j is susceptible ($S_j(t) > 0$) then further infection is possible while if the contacted stable is not susceptible ($S_j(t) = 0$) nothing will happen.

This establishes a traditional SIR-model for which there are many well known results applicable to it. Further details on the parameters of the model will be given in the following subsection.

2.3 Parameters

We let farms consist of a total of N individuals, in our case N laying hens who inhabit the farms. These N individuals are then divided into k unique stables where every stable i has a total of H_i individuals inhabiting it for $i = 1, ..., k$. The total population N is then divided into the three SIR-states where we let $S(t)$, $I(t)$, $R(t)$ denote the total number of individuals in each state at each time step t. The time t is discrete where each time step amounts to a day in the life of each individual. For $t = 0$ we have an initial distribution according to

$$S(0) = n,\ I(0) = m,\ R(0) = 0,$$

where $N = n + m$. We may further divide the population of the stables in the same fashion, letting $S_i(t)$, $I_i(t)$, $R_i(t)$ denote the number of individuals in each state for each stable $i = 1, ..., k$. We have made no assumptions on how the total population is divided into stables and the only thing we may say about the distribution of the population into stables is

$$\sum_{i=1}^{k} H_i = N, \sum_{i=1}^{k} S_i(0) = n, \sum_{i=1}^{k} I_i(0) = m, \sum_{i=1}^{k} R_i(0) = 0,$$

$$S_i(t) + I_i(t) + R_i(t) = n_i + m_i.$$

The most important parameters for this model and the upcoming simulations are the probability parameters that govern the transition probabilities (probability of going from one state to another). Firstly we have the infection parameter. We let π_I be the probability that a given susceptible individual is infected by a given infective individual. Since the population is homogeneous this probability is the same throughout all k stables. Writing i_1 for a given susceptible individual and i_2 for a given infective individual we find that, in stable j,

$$\mathbb{P}(i_1 \text{ is infected by } i_2 \text{ at time } t) = \pi_I,$$

$$\mathbb{P}(i_1 \textit{ avoids} \text{ infection by } i_2 \text{ at time } t) = 1 - \pi_I,$$

$$\mathbb{P}(i_1 \text{ avoids infection by } \textit{all} \text{ infectives in stable } j \text{ at time } t) = (1 - \pi_I)^{I_j(t)}.$$

However, this is not the only pressure that is exerted on stable j at a given time t. We must also take into account the potential added pressure from the other infected stables. For now, we simply say that the infection pressure that stable i adds onto stable j at time t is a random variable $\iota_{ij}(t)$ which we detail further down in this subsection. Simply taking $\iota_{ij}(t)$ for granted as the added pressure we get that

$$\mathbb{P}(i_1 \text{ avoids infection in stable } j \text{ at time } t) = (1 - \pi_I)^{I_j(t) + \sum_i \iota_{ij}(t)}.$$

Due to assumptions of homogenity and uniform mixing this holds for all stables. Taking the complement lets us arrive at the desired probability.

$$\mathbb{P}(i_1 \text{ in stable } j \text{ is infected at time } t) = 1 - (1 - \pi_I)^{I_j(t) + \sum_i \iota_{ij}(t)} = p_{I,j}(t).$$

This gives us the probability that a single, given individual in stable j is infected at time t. Every attempt at infecting a susceptible is a Bernoulli trial with probability $p_{I,j}(t)$ which by independence of individuals gives that the total number of susceptibles in stable j that become infected at time t is binomially distributed according to

$$T_{I,j}(t) \sim \text{Bin}(S_j(t), p_{I,j}(t)),\ t = 1, 2, ...$$

This gives us both the probability of transitioning from state S to state I at any time t and in any stable i as well as the total number of transitions for this time step.

For transitions from I to R things are less complicated. We let π_R denote the probability that a given infective individual recovers, that is, π_R is the probability that a given infective moves from the infective state into the removed state at any time t. This parameter is kept constant and does not depend on any other parameter of the model. Indeed, if we let I denote the infectious periods then for given time τ

$$\mathbb{P}(\text{Recover at time } \tau | \text{Infected since } t) = (1 - \pi_R)^{\tau - t - 1}\pi_R, \quad \tau = t + 1, t + 2, ...,$$

which we recognize as the Geometric distribution with parameter π_R. Naturally, for $I \sim \text{Geom}(\pi_R)$ we have that $\mathbb{E}[I] = 1/\pi_R$ which thus checks out with the assumptions made about the infectious periods in the SIR-model. Furthermore, since infective individuals are removed independently with probability π_R we can see these as Bernoulli trials. Thus, with the same arguments as before, we find that the total number of transitions from the infective state to the removed state in stable j at time t is given by

$$T_{R,j}(t) \sim \text{Bin}(I_j(t), \pi_R).$$

At this point we have introduced a generalized chain-binomial Reed-Frost type model. The traditional Reed-Frost model employes a deterministic recovery of $\pi_R = 1$ which we have generalized to a random variable based on the probability $\pi_R > 0$.

We have yet to explain the extra pressure added from a different stable i onto a given stable j at time t. We have only said that this is given by a random variable $\iota_{ij}(t)$. We let π_S denote the probability that a given infective individual in a given stable i spreads the disease into another stable j for $i, j = 1, ..., k$ and $i \neq j$. In a real world setting this would amount to infective substance from a given infective in stable i being attached to tools or clothing from human interaction which would later move into a different stable j. If this stable i is susceptible then added infection will be possible. Through similar arguments as for $p_{I,j}(t)$ we get that

$$\mathbb{P}(\text{Infective substance from given } i_1 \text{ fails to spread into given stable}) = 1 - \pi_S,$$

$$\mathbb{P}(\text{Inf. subs. fails to spread from stable } i \text{ at time } t) = (1 - \pi_S)^{I_i(t)},$$

$$\mathbb{P}(\text{Inf. subs. spreads from stable } i \text{ at time } t) = 1 - (1 - \pi_S)^{I_i(t)} = p_{S,i}(t).$$

This gives us the probability that a given stable spreads the disease into another stable. Now this is another case of a Bernoulli variable which means that the total number of stables contacted by stable i at time t is another Binomial, namely,

$$T_{S,i}(t) \sim \text{Bin}(k-1, p_{S,i}(t)),$$

where which stables will be contacted is drawn randomly from the $k-1$ available stables with no stable being contacted twice at a single time step t.

What we must ask ourselves now is to what extent we shall add pressure upon successful contact. If stable i contacts stable j at time t then infective substance has been transmitted from stable i to stable j. We let $\iota_{ij}(t)$ be the random variable which quantifies the amount of pressure added upon successful transmission from stable i into stable j. In our model we find it realistic to let $\iota_{ij}(t) \sim \text{Poisson}(\mu)$ i.i.d. for $i, j = 1, ..., k$ whenever stable i successfully contacts stable j. Here μ is set to represent the infectivity of the substance as well as the contact rate that individuals have with a contaminated site. However one may just as well argue for a completely different distribution and one is not necessarily limited to discrete distributions.

This added pressure is what we find in our $p_{I,i}(t)$ parameter from above where a susceptible stable is subjected to the pressure exerted from all infectious individuals inside the stable as well as the possible added pressure from external stables. Note that modeling the between-stable-spread this way upholds our previous assumptions: if $I_i(t) = 0$ then the probability of spread will be zero; if $S_i(t) = 0$ then $T_{I,i} \sim \text{Bin}(0, p_{I,i}(t))$ and nothing will happen.

The last parameter is relating to the discovery rate of the virus. We let π_D denote the probability of recognizing the symptoms of the virus in a given removed individual which will thus lead to finding, by the same reasoning as above, that

$$p_D(t) = 1 - (1 - \pi_D)^{R(t)},$$

where $p_D(t)$ is thus the probability of discovering the disease in the full population at time t. Modeling the discovery rate amounts to modeling the recognition of the virus by the farm hand. This amounts to recognizing the symptoms of the disease in the population. Normally, symptoms would be possible to discover in the infectious population too and not exclusively in the deceased population. However we have chosen to ignore these since adding these in makes the model far more complicated as we must also quantify the weight infective symptoms has compared to finding a deceased individual. Instead we simply consider the removed individuals to be the sole increasing factor in the discovery rate.

Since an attempt at discovering the disease is made only once per day and these attempts are independent of each other we find that

$$\mathbb{P}(\text{Discover NDV at time } \tau) = \left(\prod_{t=1}^{\tau-1} (1 - p_D(t)) \right) p_D(\tau), \quad \tau = 1, 2, ...,$$

This leaves us with five varying parameters out of which four are probability parameters $\{\pi_I, \pi_R, \pi_S, \pi_D\}$ and our main source of interest. The fifth parameter is the expectation μ of added pressure upon successful between-stable-spread. The two first in combination, π_I and π_R are what control the infectivity and spread of the disease in the population. These control the reproduction number R_0 in a non-trivial way and varying these will decide if an outbreak of the disease, according to our model, ends in a major or minor outbreak.

2.4 Reproduction number R_0

One of the most crucial values to infer in any SIR-model is the basic reproduction number R_0. This value is defined to be the expected number of cases produced by a typical infected individual during its infectious period, i.e. the mean number of successful contacts a given infective has before being removed. Now R_0 immediately tells us something about the average number of infected by any given infective but more importantly, well known results can be applied to R_0 to tell us something about the severity of the outbreak. It holds that

$$\mathbb{P}(\text{A major outbreak occurs } | R_0 \leq 1) = 0,$$

$$\mathbb{P}(\text{A major outbreak occurs } | R_0 > 1) > 0.$$

This means that when $R_0 \leq 1$ a major outbreak is impossible while when $R_0 > 1$ a major outbreak is possible (Ball, Pellis, Trapman, 2011, page 4; Britton (in press), 2010, page 4). We will derive this R_0 for our model below.

Consider first the single-type population, i.e., the setting where $k = 1$ with a single stable and no contact between stables. Setting the initial number of infectives to 1 we argue that since the population is homogeneous and there is no difference in susceptibility or infectivity amongst individuals, any infective individual behaves as a typical infective. Therefore we may let this initial infective be this typical infective which then gives that the expected number of cases produced by this infective during its infectious period is given by

$$\mathbb{E}[T_{I,1}(1) \cdot I] = \mathbb{E}[T_{I,1}(1)] \cdot \mathbb{E}[I].$$

We remind the reader that we have defined $T_{I,i}(t) \sim \text{Bin}(S_i(t), p_{I,i}(t))$ where $p_{I,i}(t) = 1 - (1 - \pi_I)^{I_i(t) + \sum_j \iota_{ij}(t)}$ using the assumption that $m_1 = 1$. Since there is no other stable there will be no added infection pressure $\iota_{ij}(t)$ and thus the parameter $p_{I,1}(1) = \pi_I$. This makes R_0 immediately available to us in this setting as,

$$R_0 = \mathbb{E}[T_{I,1}(1)] \cdot \mathbb{E}[I] = \frac{(N-1)\pi_I}{\pi_R}. \tag{1}$$

Looking at the more complex setting of $k > 1$ quickly makes things more difficult to derive. Consider the matrix A_k such that

$$A_k = \begin{bmatrix} \mu_{11} & \mu_{12} & \dots \mu_{1k} \\ \mu_{21} & \mu_{22} & \dots \mu_{2k} \\ \vdots & \ddots & \vdots \\ \mu_{k1} & \mu_{k2} & \dots \mu_{kk} \end{bmatrix}, \tag{2}$$

where μ_{ij} is defined to be the expected number of cases in stable j produced by a typical infective in stable i during its infectious period for $i, j = 1, ..., k$. This makes every μ_{ij} essentially a reproduction number of its own for a sub part of the total population. Results give us that R_0 is the dominant eigenvalue of the matrix A_k (Andersson and Britton, 2000, page 54).

We will derive this matrix A_k and the subsequent reproduction number here for a general $k > 1$ in the model derived in section 2 with the assumption of $\pi_D = 0$, by deriving μ_{ij} for $i, j = 1, ..., k$ in two different cases. We may use the same trick as before, setting the initial number of infectives in stable i to $m_i = 1$ where $m_j = 0$ for all $j \neq i$. Taking infectious periods into account we reason that

$$\mu_{ij} = \mathbb{E}[T_{I,j}(1) \cdot I] = \mathbb{E}[T_{I,j}(1)] \cdot \mathbb{E}[I] = \frac{\mathbb{E}[T_{I,j}(1)]}{\pi_R}. \tag{3}$$

The catch here is in computing $\mathbb{E}[T_{I,j}(1)]$ which depends on the random variables $\iota_{ij}(t)$ of added infectious pressure which we have yet to assume anything about. We argue that two different cases of μ_{ij} will occur, the first, and simplest, being the case when $i = j$, i.e., the case of μ_{ii} for $i = 1, ..., k$ of interior spread. We then find that

$$\mathbb{E}[T_{I,i}(1)] = (N_i - 1)(1 - (1 - \pi_I)^1) = (N_i - 1)\pi_I,$$

where we used that $T_{I,i}(1) \sim \text{Bin}(N_i - 1, p_{I,i}(1))$. Since $\mathbb{P}(\iota_{ii}(t) = 0) = 1$ and since $T_{S,j}(1) \sim \text{Bin}(k - 1, p_{S,j}(1))$ where $p_{S,j}(1) = 1 - (1 - \pi_S)^{m_j} = 0$ we have that $\mathbb{P}(T_{S,j}(1) = 0) = 1$ which implies that $\mathbb{P}(\iota_{ji}(1) = 0) = 1$ for all $j = 1, ..., k$. We may thus conclude that

$$\mu_{ii} = \frac{(N_i - 1)\pi_I}{\pi_R}. \tag{4}$$

For the case when $i \neq j$ we get again

$$\mu_{ij} = \frac{\mathbb{E}[T_{I,j}(1)]}{\pi_R}.$$

However this time around we have that $T_{I,j}(1) \sim \text{Bin}(N_j, p_{I,j}(1))$ where $p_{I,j}(1) = 1 - (1 - \pi_I)^{\iota_{ij}(1)}$ and $\iota_{ij}(1)$ is a random variable which we do not yet know. It is still possible to derive μ_{ij} although a bit more tedious than the previous case.

We begin by expanding with the tower property,

$$\begin{aligned}
&\mathbb{E}[T_{I,j}(1)] = \mathbb{E}[\mathbb{E}[T_{I,j}(1)|\iota_{ij}(1)]] \\
&= \sum_{x=0}^{\infty} \mathbb{E}[T_{I,j}(1)|\iota_{ij}(1) = x]\mathbb{P}(\iota_{ij}(1) = x) \\
&= \sum_{x=0}^{\infty} (N_j)(1 - (1 - \pi_I)^x)\mathbb{P}(\iota_{ij}(1) = x) \\
&= N_j \left(\sum_{x=0}^{\infty} \mathbb{P}(\iota_{ij}(1) = x) - \sum_{x=0}^{\infty} (1 - \pi_I)^x \mathbb{P}(\iota_{ij}(1) = x) \right) \\
&= N_j \left(1 - \sum_{x=0}^{\infty} (1 - \pi_I)^x \mathbb{P}(\iota_{ij}(1) = x) \right) \qquad (5)
\end{aligned}$$

To continue we must study the behavior of $\iota_{ij}(1)$ which is a random variable dependent on the outcome of other random variables. For instance we have that $\mathbb{P}(\iota_{ij}(t) = 0|T_{S,i}(t) = 0) = 1$ since if we make no contacts from stable i at all then stable j in particular has not been contacted and thus the added pressure from i into j must be zero with probability one. Furthermore we have, by definition, that $\iota_{ij}(t)|T_{S,i}(t) = k - 1 \sim \text{Poisson}(\mu)$ since if stable i contacts all $k - 1$ available stables then it must contact stable j too and thus the size of $\iota_{ij}(t)$ will be a Poisson random variable with unspecified mean μ.

We expand further with the law of total probability.

$$\begin{aligned}
&\mathbb{P}(\iota_{ij}(1) = x) = \sum_{y=0}^{k-1} \mathbb{P}(\iota_{ij}(1) = x|T_{S,i}(1) = y)\mathbb{P}(T_{S,i}(1) = y) = \\
&= \sum_{y=0}^{k-2} \mathbb{P}(\iota_{ij}(1) = x|T_{S,i}(1) = y)\mathbb{P}(T_{S,i}(1) = y) \\
&+ \mathbb{P}(\iota_{ij}(1) = x|T_{S,i}(1) = k - 1)\mathbb{P}(T_{S,i}(1) = k - 1) \\
&= \sum_{y=0}^{k-2} \mathbb{P}(\iota_{ij}(1) = x|T_{S,i}(1) = y)\mathbb{P}(T_{S,i}(1) = y) + \frac{\mu^x e^{-\mu}}{x!}\mathbb{P}(T_{S,i}(1) = k - 1) \qquad (6)
\end{aligned}$$

We need further tools to continue. The unknown terms that we need to investigate are the $\mathbb{P}(\iota_{ij}(1) = x|T_{S,i}(1) = y)$ for $y \in \{0, 1, ..., k - 2\}$. Now, with an arbitrary amount of stables $k > 2$ it is possible that $T_{S,i}(t) = y$ such that $y < k - 1$. If stable i contacts $y < k - 1$ stables we have to take the event of "i contacts j" for given j into account. We thus construct $C_{ij}(t)$ which is defined as

$$C_{ij}(t) = \begin{cases} 1, & \text{if stable } i \text{ contacts stable } j \text{ at time } t, \\ 0, & \text{otherwise} \end{cases},$$

a binary random variable functioning as an indicator for the event of stable i

contacting stable j. Repeating the law of total probability we then get

$$\begin{aligned}
&\mathbb{P}(\iota_{ij}(1) = x | T_{S,i}(1) = y) = \\
&= \mathbb{P}(\iota_{ij}(1) = x | T_{S,i}(1) = y, C_{i,j}(1) = 0)\mathbb{P}(C_{ij}(1) = 0 | T_{S,i}(1) = y) \\
&+ \mathbb{P}(\iota_{ij}(1) = x | T_{S,i}(1) = y, C_{i,j}(1) = 1)\mathbb{P}(C_{ij}(1) = 1 | T_{S,i}(1) = y) \\
&= \mathbb{P}(\iota_{ij}(1) = x | T_{S,i}(1) = y, C_{i,j}(1) = 0)\mathbb{P}(C_{ij}(1) = 0 | T_{S,i}(1) = y) \\
&+ \frac{\mu^x e^{-\mu}}{x!}\mathbb{P}(C_{ij}(1) = 0 | T_{S,i}(1) = y) \qquad (7)
\end{aligned}$$

To continue we need to find the distribution of $C_{ij}(t) = z | T_{S,i}(t) = y$ for $z \in \{0, 1\}$, $y \in \{0, 1, ..., k-2\}$. Essentially, $\mathbb{P}(C_{ij}(t) = z | T_{S,i}(t) = y)$ is the probability of drawing stable z from a population of size $k-1$ in y draws where no stable can be drawn twice. We thus conclude $C_{ij}(t) | T_{S,i}(t) = y$ to be a hypergeometric random variable such that

$$\mathbb{P}(C_{ij}(t) = z | T_{S,i}(t) = y) = \frac{\binom{1}{z}\binom{k-2}{y-z}}{\binom{k-1}{y}}.$$

In fact, for any $t = 1, 2, ...$ we get that

$$\mathbb{P}(C_{ij}(t) = 0 | T_{S,i}(t) = y) = ... = 1 - \frac{y}{k-1}, \qquad (8)$$

$$\mathbb{P}(C_{ij}(t) = 1 | T_{S,i}(t) = y) = ... = \frac{y}{k-1} \qquad (9)$$

This is the last tool we need to fully calculate $\mathbb{E}[T_{I,j}(1)]$. Plugging this into (7) we get

$$\begin{aligned}
&\mathbb{P}(\iota_{ij}(1) = x | T_{S,i}(1) = y) \\
&= \mathbb{P}(\iota_{ij}(1) = x | T_{S,i}(1) = y, C_{i,j}(1) = 0)\mathbb{P}(C_{ij}(1) = 0 | T_{S,i}(1) = y) \\
&+ \frac{\mu^x e^{-\mu}}{x!}\mathbb{P}(C_{ij}(1) = 0 | T_{S,i}(1) = y) \\
&= \mathbb{P}(\iota_{ij}(1) = x | T_{S,i}(1) = y, C_{i,j}(1) = 0)\left(1 - \frac{y}{k-1}\right) + \frac{\mu^x e^{-\mu}}{x!}\frac{y}{k-1} \qquad (10)
\end{aligned}$$

Moving backwards, we may plug results in (10) into equation (6), arriving at

$$
\begin{aligned}
&\mathbb{P}(\iota_{ij}(1) = x) = \qquad (11)\\
&= \sum_{y=0}^{k-2} \mathbb{P}(\iota_{ij}(1) = x | T_{S,i}(1) = y)\mathbb{P}(T_{S,i}(1) = y) + \frac{\mu^x e^{-\mu}}{x!}\mathbb{P}(T_{S,i}(1) = k-1)\\
&= \sum_{y=0}^{k-2} \left(\mathbb{P}(\iota_{ij}(1) = x | T_{S,i}(1) = y, C_{i,j}(1) = 0) \left(1 - \frac{y}{k-1}\right) + \frac{\mu^x e^{-\mu}}{x!}\frac{y}{k-1} \right) \mathbb{P}(T_{S,i}(1) = y)\\
&+ \frac{\mu^x e^{-\mu}}{x!}\mathbb{P}(T_{S,i}(1) = k-1)\\
&= \sum_{y=0}^{k-2} \mathbb{P}(\iota_{ij}(1) = x | T_{S,i}(1) = y, C_{i,j}(1) = 0) \left(1 - \frac{y}{k-1}\right) \mathbb{P}(T_{S,i}(1) = y)\\
&+ \frac{\mu^x e^{-\mu}}{x!}\frac{1}{k-1} \left(\sum_{y=0}^{k-2} y\mathbb{P}(T_{S,i}(1) = y) + (k-1)\mathbb{P}(T_{S,i}(1) = k-1) \right)\\
&= \sum_{y=0}^{k-2} \mathbb{P}(\iota_{ij}(1) = x | T_{S,i}(1) = y, C_{i,j}(1) = 0) \left(1 - \frac{y}{k-1}\right) \mathbb{P}(T_{S,i}(1) = y)\\
&+ \frac{\mu^x e^{-\mu}}{x!}\frac{1}{k-1}((k-1)(1-(1-\pi_S)^1))\\
&= \sum_{y=0}^{k-2} \mathbb{P}(\iota_{ij}(1) = x | T_{S,i}(1) = y, C_{i,j}(1) = 0) \left(1 - \frac{y}{k-1}\right) \mathbb{P}(T_{S,i}(1) = y) + \frac{\mu^x e^{-\mu}}{x!}\pi_S
\end{aligned}
$$

Moving on, we may now finally enter the result of (11) into the original (5),

$$
\begin{aligned}
&\mathbb{E}[T_{I,j}(1)] = N_j \left(1 - \sum_{x=0}^{\infty}(1-\pi_I)^x \mathbb{P}(\iota_{ij}(1) = x)\right) = \\
&= N_j \left(1 - \mathbb{P}(\iota_{ij}(1) = 0) - \sum_{x=1}^{\infty}(1-\pi_I)^x \mathbb{P}(\iota_{ij}(1) = x)\right) \\
&= N_j \left(1 - \sum_{y=0}^{k-2} 1 \cdot \left(1 - \frac{y}{k-1}\right) \mathbb{P}(T_{S,i}(1) = y) - \pi_S e^{-\mu} - \sum_{x=1}^{\infty}(1-\pi_I)^x \mathbb{P}(\iota_{ij}(1) = x)\right) \\
&= N_j \left(1 - 1 + \mathbb{P}(T_{S,i}(1) = k-1) + \frac{1}{k-1}\left((k-1)(\pi_S) - (k-1)\mathbb{P}(T_{S,i}(1) = k-1)\right)\right) \\
&- N_j \left(\pi_S e^{-\mu} + \sum_{x=1}^{\infty}(1-\pi_I)^x \mathbb{P}(\iota_{ij}(1) = x)\right) \\
&= N_j \left(\pi_S - \pi_S e^{-\mu} - \sum_{x=1}^{\infty}(1-\pi_I)^x \left(\sum_{y=0}^{k-2} 0 \left(1 - \frac{y}{k-1}\right) \mathbb{P}(T_{S,i}(1) = y) + \frac{\mu^x e^{-\mu}}{x!}\pi_S\right)\right) \\
&= N_j \left(\pi_S - \pi_S e^{-\mu} - \sum_{x=1}^{\infty}(1-\pi_I)^x \frac{\mu^x e^{-\mu}}{x!}\pi_S\right) \\
&= N_j \left(\pi_S - \pi_S e^{-\mu} - \pi_S e^{-\mu}\left(e^{\mu - \mu\pi_I} - 1\right)\right) \\
&= N_j \left(\pi_S - \pi_S e^{-\mu\pi_I}\right) = N_j \pi_S (1 - e^{-\mu\pi_I})
\end{aligned} \tag{12}
$$

Returning back to our original problem in equation (3) we find that, for $i \neq j$ and with the assumption that $m_i = 1$ and $m_j = 0$ for all $j \neq i$ we get

$$
\mu_{ij} = \mathbb{E}[T_{I,j}(1) \cdot I] = \frac{N_j \pi_S}{\pi_R}(1 - e^{-\mu\pi_I}). \tag{13}
$$

We thus conclude that the next generation matrix $A_k(\mu_{ij})_{i,j=1,\ldots,k}$ consists of elements of the form

$$
\mu_{ij} = \begin{cases} \dfrac{(N_i - 1)}{\pi_R}\pi_I, & \text{for } i = j \\ \dfrac{N_j \pi_S}{\pi_R}(1 - e^{-\mu\pi_I}), & \text{for } i \neq j \end{cases} \tag{14}
$$

We stop here, referencing back to the fact that R_0 is now the largest eigenvalue of the next generation matrix $A_k = (\mu_{ij})_{i,j=1,\ldots,k}$ (Andersson and Britton, 2000, page 54). Computing R_0 analytically from this matrix essentially boils down to solving the resulting k-dimensional polynomial, i.e., the characteristic equation of A_k. Efficiently doing so may provide useful insight into the structure of R_0 as a function of our parameters but even computing a general solution to the cubic equation obtained from the determinant of $(A_3 - \lambda I_3)$ is an ardous task. Luckily, we may instead utilize computational power to provide us with the resulting R_0 for varying values of parameters.

2.5 Some comments on the caged model

In the setting for the caged model, things are not nearly as nice to us as before. We may still employ an SIR-model. In fact, most of our assumptions made in section 2.2 are still applicable in this setting however any further comparison is met with contradictions. Firstly, since individuals spend their entire lives in cages arranged in an $I \times J$-grid we can no longer assume that the population is mixing uniformly since it is no longer possible for an individual in cage $(1, 1)$ to directly contact an individual in cage (I, J) and at a given time t all susceptible individuals can not necessarily be contacted by all infective individuals. Secondly, due to the arrangement in grids of cages, one ought to consider some kind of spatial structure where individuals in cage (i, j) for $i, j \geq 2$ perhaps only may contact individuals in cages directly connecting to their own (cage i, j contacts cage $(i + 1, j)$; $(i, j + 1)$; $(i + 1, j + 1)$ and so forth) which introduces another complicated element to the model. Lastly, cages rarely house a single individual and may contain up to 16 individuals per cage. This means that if a single individual is infected in a given cage (i, j) the remaining 15 susceptibles will face a higher infection pressure than individuals in adjacent cages. This essentially makes the model a variant of a household model where cages are households limited to a certain kind of spatial structure.

3 Simulations and results

To get a better understanding of the movemenets of our modeled epizootic we implement various simulations. Simulations are written in code using the program R and no exterior packages have been used.

Four different types of simulations have been written up. Each simulation builds on the other one making the last one the only relevant one for actual simulations. The first simulation implements the epizootic in a single stable setting ($k = 1$) and runs the simulation until the number of infectives reaches zero. It does not utilize the alternate stopping criterion detailed in the previous section and uses only two parameters π_I and π_R.

The second simulation simply takes the previous simulation but allows k to be greater than 1. Still, no between-stable-spread is introduced and neither is the alternate stopping. It runs until $I(t) = 0$ for some t and only takes parameters π_I and π_R.

The next extension allows for between-stable-spread to occur. It thus includes a third parameter π_S as argument but still no alternate stopping, meaning it stops as soon as $I(t) = 0$.

Lastly, the final simulation is the main simulation. It builds upon all previous simulations but also contains the alternative stopping criterion of discovering the disease. As a result it contains four parameters $\pi_I, \pi_R, \pi_S, \pi_D$ and runs until either the total number of infectives $I(t)$ reaches 0 or stops when the specified criteria for the discovery of the disease are met.

The code itself uses results from theory to simulate the infection spread based on what has been derived and written in section 2 of this paper. Two main functions make up the simulation. The first one implements the initial distribution at time $t = 0$ of the disease. As arguments it takes k, the number of stables, H_k a vector of length k consisting of the number of individuals in each stable and m the total number of initially infected. It creates a matrix of dimension $N \times 3k$ where $N = \sum_k H_k$ consisting of zero's and one's such that every column triple describes the state of the individual in that particular stable with 1 for TRUE and 0 for FALSE where rows indicate individuals. It then randomly spreads out m infected inidividuals amongst the stables and returns the full matrix. Simple changes in the code would allow for the number of initially infected to work differently. One may easily make a change such that the given number m of total initially infective are all in the same stable at time $t = 0$.

This function sets up the simulation of the full epizootic. The full simulation takes as arguments a matrix received from the previously mentioned starting function, our four probability parameters $\pi_I, \pi_R, \pi_S, \pi_D$ and the expected amount of added pressure μ.

It sums up the columns in the starting matrix, storing them inside a $1 \times 3k$-matrix which thus contains the number of inidividuals in each state in each stable. It then runs the simulation according to what has been described in section 2. As a time step t is entered we begin by simulating a binomial following

the distribution of $T_{S,i}(t)$ for all stables $i = 1, ..., k$ which gives the total number of stables contacted by stable i. We then simulate a Poisson(μ)-variable for every successful hit, representing the $\iota_{ij}(t)$-values. We then run through all the stables, simulating binomials for $T_{I,i}(t)$ and $T_{R,i}(t)$ for every stable i and ultimately updating the stable distributions according to the results. Before exiting the loop we allow for the disease to be discovered by simulating a uniform number and checking if it is lower than $p_D(t)$. If it is we immediately exit the loop and return the state distributions at every time step t. If it is not then we increment t and repeat the process until we either succeed or $I(t) = 0$.

Simple modifications have been made to the code to return things such as the final sizes or other properties of the epidemic. Some simple functions have also been written to simplify the running of simulations as well as the gathering of data.

This is the structure that we have deemed reasonable and realistic. We have tried to combine efficiency with realisism as far as the spread of the virus goes and extensions are undoubtedly possible to make. The assumption of discrete time steps may be flawed but we have failed to find a better implementation when it comes to these simulations. One may also question whether or not the initially infected individuals at time step $t = 0$ should be randomized into the k different stables. It may indeed be better to simply choose infectives just as we choose sizes of stables but since we are mostly interested in running the simulations for $m = 1$ we find this to not be a problem.

3.1 Final size - Z

These simulations are powerful tools since they implement our model in a setting where we can observe its behavior for any possible combination of parameters. At its core, one run of the simulation for a set of values of $\{N, m, \pi_I, \pi_R, \pi_S, \pi_D, \mu\}$ provides us with the distribution into states S, I, R for every time step t until the epidemic is stopped. While this is indeed very useful, it is a lot of information to take in for all the possible combinations of parameters and we may instead focus our attention on the result of the epidemic, i.e., the final size of a simulation. We thus define the random variable Z to be the final size of the epidemic, that is, the number of individuals who have become infected during the run of the epidemic. More rigorously we write

$$Z = R(T) - m, \tag{15}$$

where $T = \min\{t \geq 1 | \{I(t) = 0\} \cup \{D(t) = 1\}\}$ where $D(t)$ is an indicator variable for the event that the virus is discovered in the population.

We previously mentioned that when our basic reproduction number is greater than 1, a major outbreak is possible but it was never quite clear what defined a major as well as minor outbreak. This variable Z does not give us a clear line to draw either but it at least, in a sense, lets us assess and quantify the severity of an outbreak. In the subsections that follow we will thoroughly use Z as a way to illustrate the changing behavior of R_0 and the various free parameters in the

model. Additionally, theory gives us that if N is large enough the distribution of Z is bimodal, where one point will be 0 and the other point will be a quantity $\zeta > 0$, something we will observe through simulations in the coming subsections (Britton (in press), 2010, page 13).

3.2 Univariate case - $k = 1$

One of the simplest settings to consider is the case where the farm consists of only a single stable, i.e., when $k = 1$. In this setting we allow no between-stable-spread and thus $\pi_S = 0$. To simplify it even further we also choose to ignore the discovery aspect of the model by simply setting $\pi_D = 0$. The basic reproduction number takes on a quite simple form here as $R_0 = \mu_{ij} = \mu_{11} = \frac{(N-1)\pi_I}{\pi_R}$. As stated in section 2.4 we may immediately say something about the infectivity of an outbreak based only on the size of R_0 which here depends on the 3 parameters which we allow to vary. It is not clear what values of our free variables that are interesting to study so we will let these vary throughout our simulations where it is initially only assumed that $m_1 = m = 1$.

In this subsection we wish to check that our simulations match our analytic derivations of R_0. Additionally, we want to use these simulations to further existing theoretical results which apply to our model by deriving properties, analyzing conjectures and understanding the behavior of our model as parameters change. We begin by analyzing the resulting final size Z for simulations where N is fixed to $N = 3000$ and where either π_I or π_R is allowed to vary with the other fixed to a pre-specified value. We have chosen $N = 3000$ because we deem it to be a realistic number for many Swedish farms as it is the upper bound for the population of a stable inside an ecological farm as well as allowing for relatively short computational times as opposed to for larger numbers. Our results are displayed in figure 2.

We have chosen to present trajectories of Z for intervals of R_0 such that the highest R_0 we see is $R_0 = 30$. One may argue that this is a particulary high value for the basic reproduction number but as a counterargument we claim that it is unclear what the true R_0 is for NDV. Indeed, it may be the case that the disease is infectious enough that an R_0 of 30 is not impossible but even if this is not the case, illustrating the trajectories of Z up until this point seems reasonable enough to illustrate the converging behavior of Z as parameters, and subsequently R_0, grows.

Analytic results tell us that, for $R_0 \leq 1$, major outbreaks should be impossible. This property is indeed apparent in figure 2 as we have this region to the left of the vertical line in A and B and to the right of it in plots C and D. In the case when π_I ranges from 0 towards 1 we see clearly in B a period where outbreaks result in $Z = 0$ followed by a steep ascent as soon as π_I passes the vertical line, only to have convergence of $Z \to N - m$ as π_I continues to 1.

Similar behavior is noticeable in C and D, where π_I is kept fixed at $\pi_I = 0.0001$ and we instead let π_R vary freely. One striking difference between A, B and C, D is the point to which Z converges as the parameters grow. In the case of π_R we have $Z \to 0$ as $\pi_R \to 1$. This is due to the structure of R_0 as a function

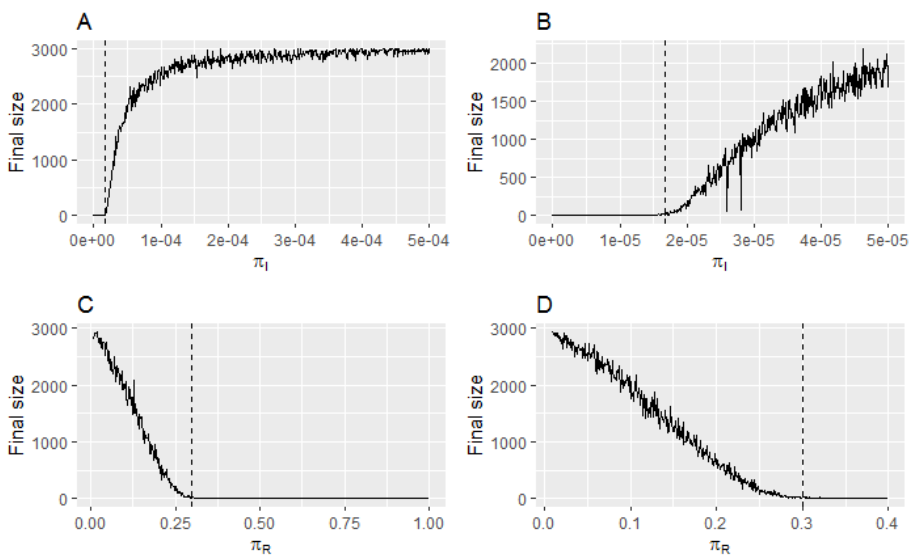

Figure 2: All four plots have been created by setting $N = 3000$ and running the simulations for a grid of values of length 500 where we for every value run 100 iterations, taking the mean of the final size. Plots A and B have had π_R fixed to $\pi_R = 0.05$ whereas plots C and D has $\pi_I = 0.0001$. In A, the grid is chosen so that $R_0 \in [0, 30]$ as π_I grows while in B it is chosen so that $R_0 \in [0, 3]$. In C, the grid is chosen so that $R_0 \in [30, 0.0299]$ while in D $R_0 \in [30, 0.75]$. The vertical line is the point where $R_0 = 1$.

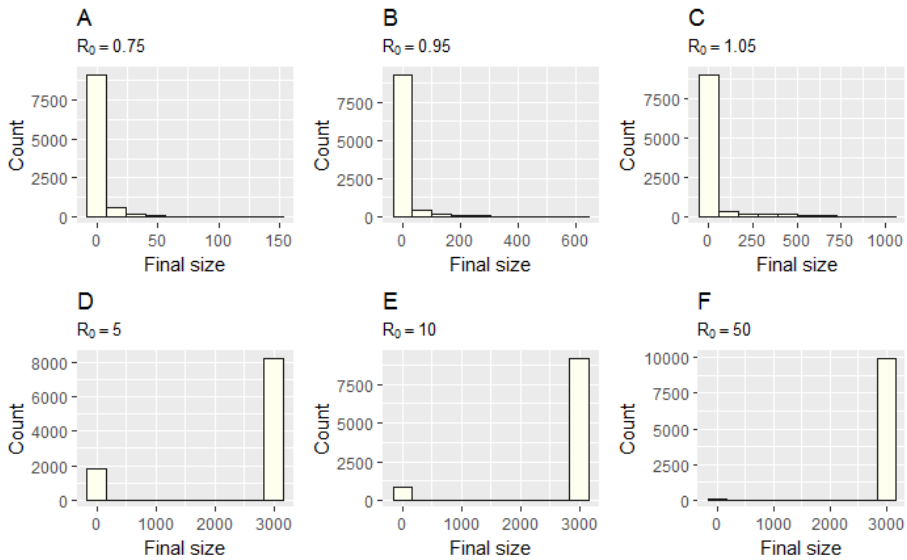

Figure 3: Histograms are created through simulations with $N = 3000$, $\pi_R = 0.05$ and π_I chosen so that R_0 is what is given in the titles of the plots. For every plot, 5000 iterations have been run with the final size being presented.

of these parameters. In the case when π_I is free we have $R_0(\pi_I) = c \cdot \pi_I$ which grows linearly from 0 to c as π_I ranges between 0 and 1. However, for π_R we have that $R_0(\pi_R) = c/\pi_R$ for which $R_0(\pi_R) \to c$ as $\pi_R \to 1$ and which tends to infinity as $\pi_R \to 0$. Obviously, as the probability of infection grows, the number of infected must also grow while as the probability of being removed grows we get shorter infectious periods meaning that $I(t)$ is not allowed to grow as large which in turn lowers the pressure on the susceptibles.

In figure 2 we saw trajectories of Z which made it plausible to reason that as the probability parameters π_I and π_R tend to their boundaries, Z converges either to 0 or $N - m$. In fact it holds that, under certain conditions, that the distribution of Z is bimodal (Britton (in press), 2010, page 13). In figure 3 we give histograms of the final sizes for set values of N, π_R and π_I such that the underlying R_0 will be of varying size.

In A, B and C we note almost exclusively outbreaks resulting in $Z = 0$ with a few minor outliers which together fails to make up even a 10'th of the total outbreaks. We note that as R_0 grows and eventually passes 1 onto $R_0 = 1.05$ we see a similar distribution where the size of the outlying final sizes grow larger and larger. As R_0 gets even bigger we see how Z clearly moves into this bimodality mentioned above as evidenced in D, E and F.

Indeed, at this point Z seems to take either the value $Z = 0$ of a minor outbreak or $Z = N - m$ of a major outbreak. The conclusion we may draw from this is that when R_0 is large, the best chance a population has of surviving is

when the initial infective is removed before it successfully contacts any susceptible. We also note that the previously mentioned property of R_0 as informing us about the potential size of the outbreak seems to hold. While we have not defined how large Z ought to be for us to consider the outbreak to be a major one, we can easily see that for the two histograms where $R_0 < 1$, no major outbreak has occured and at most, 600 individuals have been removed where the virus failed to become epidemic and disappeared from the population with the removed individuals.

3.3 Multitype case - $k > 1$

In this subsection we allow for a farm to have more than one stable corresponding to the assumption of $k > 1$. As a result we find it natural to introduce the parameters π_S, the probability of disease spread, and μ, the average pressure added upon successful spread, into our simulations. Much like in the previous subsection we simplify the model by putting π_D, the probability of discovery, to 0.

In the multitype case we are still interested in for what set of parameter values we may achieve a minor and conversely major outbreak. We are also interested in the ability of the disease to spread from stable to stable, both in a setting where the reproduction number is less than 1 but also in the setting where we let R_0 grow farther past one.

Much like in the previous subsection, we note that major outbreaks may only occur if $R_0 > 1$ and we will thus be interested in the trajectory of the disease for the cases when $R_0 \leq 1$ as well as $R_0 > 1$. As opposed to the univariate case, the structure of R_0 is not obvious. It is the dominant eigenvalue of the next-generation matrix, found by solving the characteristic equation $\det(A_k - \lambda I_k) = 0$ which makes the characteristic equation a polynomial in degree k which as Abel famously proved there is no general solution for when $k > 4$. However, since the structure of the elements μ_{ij} of A_k are simple enough we may still draw some asymptotic conclusions as we let the free variables tend to their boundaries. In figure 4 we find four plots of R_0 as we let the parameters π_I, π_R, π_S and μ vary throughout their domain, keeping the other parameters fixed.

We remind the reader that we in uncovered that we in (13) found that

$$\mu_{ij} = \begin{cases} \dfrac{(N_i - 1)}{\pi_R}\pi_I, \text{ for } i = j \\ \dfrac{N_j \pi_S}{\pi_R}(1 - e^{-\mu\pi_I}), \text{ for } i \neq j \end{cases}$$

Simply looking at the size of R_0 we reason that our parameters from the univariate case π_I and π_R has the strongest control over the resulting R_0. This is not unreasonable since they are the only probability parameters to occur in all expressions μ_{ij}. In the case of π_I we note that, much like in the univariate case, R_0 grows linearly with π_I. Keeping all other parameters fixed we have that $\mu_{ii}(\pi_I) = c \cdot \pi_I$ which is indeed linear, converging to c as $\pi_I \to 1$ and to 0 as π_I tends to 0. Furthermore, $\mu_{ij}(\pi_I) = c(1 - e^{-d\pi_I})$ which converges to

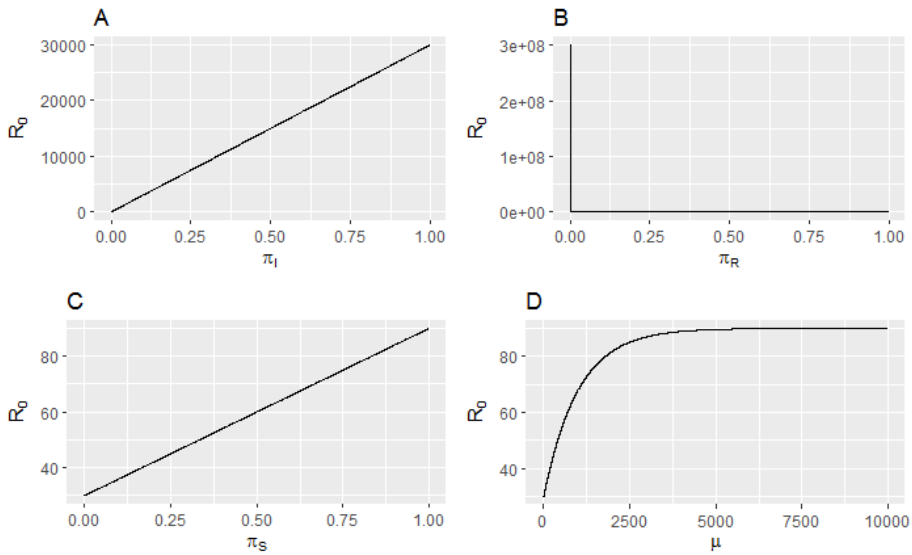

Figure 4: R_0 in a population with $k = 3$ stables and total population $N_i = 3000$ for all stables. In A, B and C probability parameters take values over a grid from $(0, 1]$ of length 1000 while in D, μ varies in $\{0, 1, ..., 1000\}$. Fixed parameters are set to $\pi_I = 0.001$, $\pi_R = 0.1$, $\pi_S = 0.001$ and $\mu = 1$.

$c(1 - e^{-d})$ as π_I tends to 1 and which goes to 0 as $\pi_I \to 0$. Since all μ_{ij} tends to 0 as $\pi_I \to 0$ it is not unreasonable that R_0 is 0 in this case, while we in this setting, when $\pi_I \to 1$, get $R_0 = 300027$.

Looking at the expectations μ_{ij} but for π_R instead we arrive at a very different structure. Obviously, $\mu_{ii}(\pi_R) = c_1/\pi_R$ much like in the univariate case which tends to infinity as $\pi_R \to 0$ and simply tends to c_1 as $\pi_R \to 1$. In the same vein we see $\mu_{ij}(\pi_R) = c_2/\pi_R$ which behaves much in the same way. Here, for $\pi_R \in (0, 1]$ we get $R_0 \in (\infty, 3]$.

For π_S and μ, the R_0 behaves a bit differently. In both cases, the parameters do not occur in the interior of stables and only affect the between-stable-contact. Indeed, as a result of this μ_{ii} is independent of both and so these parameters may not affect this part of the R_0 in any way. We note that $\mu_{ij}(\pi_S) = c \cdot \pi_S$ is a linear function, as is the effect π_S has on R_0 as a whole. Still, the magnitude of the effect of π_S is not nearly as strong as π_I and π_R has. Even when we let $\pi_S \to 0$, making between-stable-spread an impossibilty and $\mu_{ij} = 0$, the R_0 is still quite big at $\frac{(N_i-1)\pi_I}{\pi_R} = 29.99$. For the converse, we note that letting π_S grow towards 1, meaning that a given stable i *always* contacts the other $k - 1$ stables, does not make R_0 explode and while it in this setting grows towards $R_0 = 89$, this is a very minor increase when compared to the changes possible as π_I or π_R is allowed to vary. While it is interesting to note the comparibly minor effect of π_S it is not too surprising. Even if $\pi_S = 1$ successful spread still depends

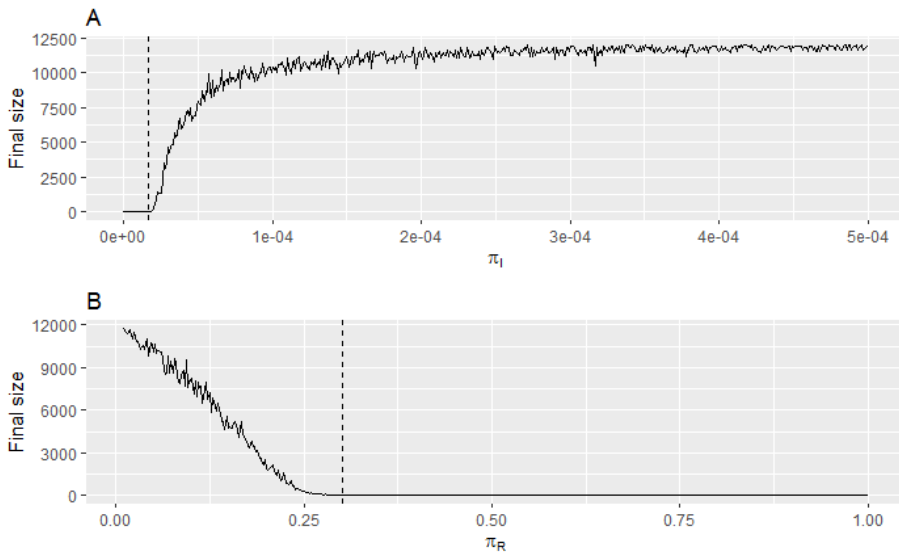

Figure 5: The final size Z as a result of the mean of 100 iterations for every parameter value. In both plots, $k = 4$ and $N_i = 3000$ for $i = 1, ..., k$ with $\pi_S = 0.001$ and $\mu = 1$. The vertical line indicates the point where $R_0 = 1$. In A, π_I is chosen over a grid of length 500 such that $R_0 \in [0, 30]$. In B, π_R is chosen over a grid of length 500 such that $R_0 \in [30, 0]$.

on $\iota_{ij}(t)$ which for $\pi_S = 1$ is simply Poisson(μ) and for low enough μ may fail to add any kind of pressure at all to stable j. Additionally, if $\iota_{ij}(t) > 0$ we may still fail to infect susceptibles in stable j. While stable contact probability is indeed relevant for the model, the many further controls it has to pass through to successfully lead to infection lowers its effect on the basic reproduction number considerably. We add that, as expected, computing R_0 again with the same values for parameters but letting $k > 3$ leads to larger effect on R_0 for π_S.

Lastly, for μ we note similar results to those regarding the impact of π_S on R_0. Interior spread (μ_{ii}) is independent of the size of μ and its only effect is in the amount of pressure added upon successful exterior stable contact (i.e., the size of $\iota_{ij}(t)$). We have that $\mu_{ij}(\mu) = c(1 - e^{-d\mu})$ which is the same form as for $\mu_{ij}(\pi_I)$ although the domain of μ is quite different. As $\mu \to \infty$ we get $\mu_{ij}(\mu) \to c$ and when $\mu \to 0$ we get $\mu_{ij}(\mu) \to 0$. Much like for π_S we note that even though μ grows we see little impact on R_0. In fact, letting μ increase even further we note that R_0 converges (in this case to $R_0 = 90$) as $\mu \to \infty$.

Having these in mind we may easily check the transitioning behavior of the final size Z as we let parameters vary freely. Figure 5 gives plots containing the trajectories for Z as π_I and π_R are allowed to vary throughout a grid of pre-specified values.

Essentially, we note the same behavior as we did in the univariate case.

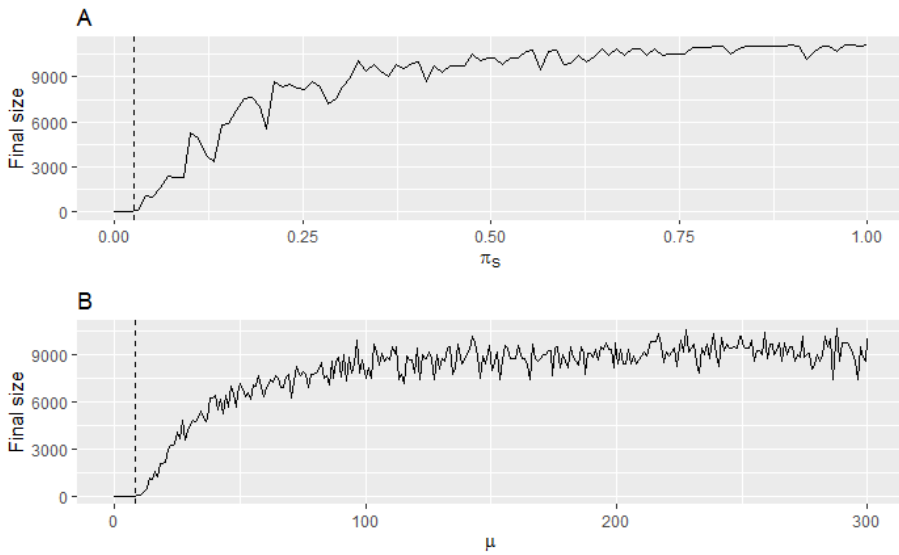

Figure 6: The final size Z as a result of the mean of 25 in A, and 50 in B, iterations for every parameter value. In both plots, $k = 4$ and $N_i = 3000$ for $i = 1, ..., k$ with $\pi_I = 0.00001$ and $\pi_R = 0.1$. The vertical line indicates the point where $R_0 = 1$. In A, π_S is chosen over a grid of length 100 such that $R_0 \in [0.29, 27]$. In B, μ is chosen over a grid of length 100 such that $R_0 \in [0.29, 27]$.

Despite having introduced two more parameters, the behavior of the final size is more or less the same. In A we note a slow start while $R_0 < 1$ where a majority of outbreaks finish with $Z = 0$ after which we get a transitional period as the curve slowly converges to a point where outbreaks result in $Z = N - m$. In B we also see something quite similar to what we saw in the univariate case when we allowed π_R to vary. As $\pi_R \to 0$ we again get $Z \to N - m$ much like we saw in figure 2. Again, we note the difference in directions as the probability parameters tend to 1 and point out that this is due to the structure of R_0 as a function of π_I versus as a function of π_R as we noted in Figure 4 with the trajectories of R_0 as functions of these.

For the two newly introduced parameters, things work out a bit differently. While the trajectory is similar, convergence seems to happen much more slowly as we note in the range on the x-axis, more so between π_I and π_S. In fact, while both curves tend towards the limit of $Z = N - m$, none of them succeed in hitting a final size this large. While it is not quite clear from the plots themselves, A only reaches to about 10000 while B reaches 11000 at their highest. In comparison with the plots in figure 5, this gives a great illustration on the impact of these four variables on the final size Z. Both π_I and π_R control the outcome of the epidemic more strongly than π_S and μ does. This was visible already in the

derivations of R_0 where these two parameters appeared in all the terms μ_{ij} in the next-generation matrix and these plots in figure 6 give us more evidence of this. Indeed, even if $\pi_S = 1$, this only tells us that contact will occur repeadetely throughout the epidemic. Still, if π_I is low enough to make infection difficult, no matter how often exterior infection pressure is added, it will fail to gain traction due to the small size of π_I.

One of our main questions of interest is of to what degree confinement of individuals into stables is a safeguarding measure against major outbreaks of the virus. We wish to study this by running simulations and counting the number of runs in which the initially infected stable successfully infects another non-infected stable. It is not clear for what parameters we wouldd like to analyze this since all free parameters play an important part in this event. Luckily, we may actually derive the correct probability with the caveat that it requires knowing the time T when the epidemic is stopped along with knowledge of $I_i(t)$ where i is such that $m_i = 1$. We give the result below and refer to appendix for the full proof. Now if we know the time T when the epidemic is stopped and assuming $m_i = 1$ with $m_j = 0$ for all $j \neq i$,

$$\begin{aligned}
&\mathbb{P}(\text{No spread occurs}) = \\
&= \prod_{t=1}^{T-1} \mathbb{P}(\text{No spread occurs at time } T-t | \text{No spread occurs at time } T-t-1, ..., 1) \\
&= \prod_{t=1}^{T-1} \left(\prod_{j \neq i} \left(1 - p_{S,i}(t) \left(1 - e^{-\mu}\right) \left(1 - ((1-\pi_I)^{\iota_{ij}(t)})^{N_j}\right)\right)\right) = \mathcal{P} \qquad (16)
\end{aligned}$$

where the epidemic is known up to every previous time step (so $I_i(t)$ is always known) and with $\iota_{ij}(t) > 0$. We will refer to this as equation (equation number here) or simply as $\mathcal{P}$.

Writing the inner product as

$$\begin{aligned}
\mathcal{P}^* &= 1 - p_{S,i}(t) \left(1 - e^{-\mu}\right) \left(1 - ((1-\pi_I)^{\iota_{ij}(t)})^{N_j}\right) \\
&= 1 - f_1(\pi_S, I_i(t)) \cdot f_2(\mu) \cdot f_3(\pi_I, \iota_{ij}(t), N_j) \qquad (17)
\end{aligned}$$

we may more easily see the structure of the equation. This expression $\mathcal{P}^*$ may be easily interpretated as a product of the probability of stable contact, the probability of infection and the probability of a Poisson(μ) variable being greater than 0. These are the steps needed to pass through to successfully spread the virus into another stable. It is easy to see that for N_j and $\iota_{ij}(t)$ fixed, if $\pi_I \to 0$ then the probability of spread not occuring will be 1 ($f_3 \to 0$) which makes sense since if it is impossible to infect then no matter how much exterior pressure is placed on a stable no individual will ever contract the disease from the pressure. Similar results hold for π_S: with $I_i(t)$ fixed and $\pi_S \to 0$ then $\mathcal{P}^*$ will again be 1. If π_S is 0 then it will be impossible for a stable to contact another stable, making it impossible to spread the virus. Lastly, if $\mu \to 0$ then $f_2(\mu) = 0$ and as a result the probability $\mathcal{P}^*$ becomes 1 in this case too. We

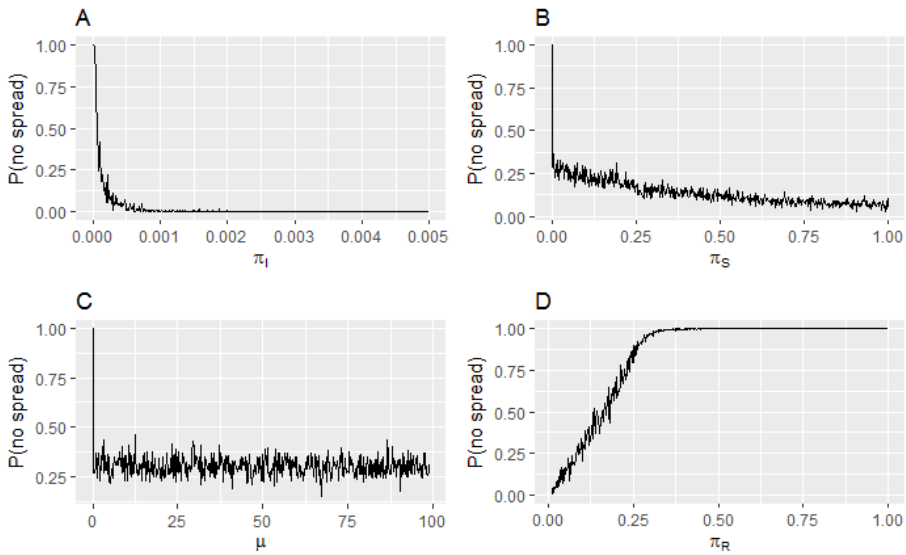

Figure 7: Plots are created to estimate the probability of no spread occuring. Fixed parameters are $N_j = 3000$, $k = 4$, $\pi_I = 0.0001$, $\pi_R = 0.1$, $\pi_S = 0.001$ and $\mu = 1$. In A, π_I varies over a sequence from 0 to 0.005. In B, π_S varies over a sequence from 0 to 1. In C, μ varies over a sequence from 0 to 100. In D, π_R varies over a sequence from 0.01 to 1. All sequences are of length 500 and values for $\mathcal{P}$ have been obtained by running 100 simulations for each value in the sequence and taking the mean.

may interpret this as that if the added pressure is on average 0 then regardless of whether contacts are successful or not, the added pressure will not add any infectivity to a stable.

The other free parameters also play a part but not quite an as pivotal role. The size of N_j and $\iota_{ij}(t)$ plays an important part in the speed of convergence of f_3 towards 1 as $\pi_I \to 1$ which accelerates as both variables grow. The same holds for $I_i(t)$ which too works as an accelerator for the convergence of f_1. For both parameters this behavior comes with no surprises.

Perhaps most important to notice is the behavior when parameters π_I, π_S and μ tend to their upper boundaries. When this happens, the part of the product which contains the parameter will converge to 1 and thus disappear from the equation - removing one of the obstacles the disease needs to pass through to successfully spread. We note that this convergence happens quite fast and is dependent on the other parameters of the model. For instance, the speed of convergence to 1 of $p_{S,i}(t)$ is heavily dependent on the size of $I_i(t)$ and as a result, the larger $I_i(t)$ is, the faster the convergence.

While there are difficulties in analytically deriving the probability of no spread occuring any further than we have done in Proposition 1 (see appendix),

we may use the simulations available as data in order to estimate the probability of no spread occuring. In the plot in figure 7 we have created 4 plots for this probability for sequences of parameters as the others are kept fixed.

In plot A we see the probability nosedive towards 0 as π_I runs towards 1. Using a relative convergence criterion and $\epsilon = 1e - 6$ as error limit we get that $f_3(\pi_I, 3000, 1)$ converges to 1 at around $\pi_I = 0.001$ essentially removing one of the obstacles that the disease has to pass through to successfully spread. This illustrates the impact that π_I has on the probability of spread occuring - π_I needs to be very close to 0 for us to be certain that no spread will occur throughout the epidemic. In the other plots we see far less stability and a larger variance.

For B, where π_S is allowed to vary we note a similar behavior to that in A but with a much slower convergence. Variation is also an issue and even though the trajectory settles in as we near 1 we still note a quite unstable curve. An explanation for this is in the randomness apparent in $p_{S,i}(t)$ which depends on $I_i(t)$, our results in every step of the simulation. Stability may improve for a larger number of iterations. We note that despite the fact that the probability of no spread occuring is still low even for small values of π_S, it takes a much longer time for the probability to converge to 0 as opposed to what we see in A. Indeed, this further tells us something about the impact π_I has on the probability compared to the impact of π_S or μ. We already saw something similar in figure 4 of the effect of π_S and μ on R_0 as weak compared to the control that both π_I and π_S exerts over it. This is further strengthened by the results in C where $\mu \to \infty$. Converges settles in at around $\mu = 14$ (by checking convergence with the relative convergence criterion and $\epsilon = 1e-6$) and anything after that seems to be similar behavior. Variation is large here too but perhaps most important to note is that after convergence the probability is above 0. In fact, the lowest probability found through our simulations were of $\mathcal{P} = 0.16$.

Lastly, we observe the behavior of the probability as we allow π_R to change. While π_R does not occur in the probability $\mathcal{P}^*$ it affects the random variable T, the first time the epidemic is stopped. As $\pi_R \to 0$, T grows larger and if we specify $\mu > 0$, $\pi_I < 1$ and $\pi_S < 1$ then $\mathbb{P}(\text{No spread occurs}) \to 0$ as $\pi_R \to 0$ and as a result, as $T \to \infty$.

We have yet to say much about the y-axis of figure 7 since taking these in conjuncture with the trajectories for granted is somewhat naive. What we may state for certain is that, in our model, the probabilities of no spread occuring for the given sets of parameters, is what is portrayed in the plots in figure 7. However, this is far from the whole truth about the probability of no spread occuring. Its dependance on all free parameters is more complicated than what we may infer from the trajectories in figure 7 and still no analysis has been performed on the behavior of $\mathcal{P}$ as more than one parameter is allowed to vary. The presented results are still very dependent on the fixed variables and with the small knowledge we have of the true parameter values in the modeled setting of NDV, it gets difficult to relay information about what the actual real-life probability would be. We leave this discussion for later and return to this problem in subsection 3.4 where we extend the model with the discovery

probability.

3.4 Introducing discovery - $\pi_D > 0$

As we introduce the discovery property of the model, $\pi_D > 0$, the model changes abruptly again. The univariate model has received extensive studies throughout the past century with extra attention having been given to the Reed-Frost-version where the recovery probability π_R is constant and set to 1. The multi-type epidemic has received its fair share of attention as well and theoretical results and approximations exist in this setting too. However, little has been formalized relating to alternative stopping conditions and once this property is entered into our model, many of our previous results are not as easily employed.

We still have our results relating to R_0, that for $R_0 \leq 1$, a major outbreak is impossible. This holds with discovery in place too, since either the disease is stopped before the first time $I(t) = 0$ and at this point $Z_d < Z$ (where Z_d denotes the final size in the same model but where $\pi_D > 0$) or the disease is stopped once $I(t) = 0$ and thus $Z_d = Z$. When $R_0 > 1$ we have that a major outbreak may occur and that it occurs with a certain probability. With the same argumentation we reason that here too, either $Z_d < Z$ or $Z_d = Z$ if the epidemic is not prematurely stopped due to discovery of the disease. We thus conclude that, with Z_d denoting the final size of an epidemic with discovery included and Z denoting the final size of the same epidemic without discovery ($\pi_D = 0$) we always have

$$Z_d \leq Z.$$

Obviously this is assuming that Z_d is produced with the same values for parameters as Z has been.

In subsections 3.2 and 3.3 we created trajectories of the final size Z as parameters were allowed to vary throughout their domain. We repeat this analysis here for different values of π_D.

Just as we stated above, one would expect the new final sizes Z_d to be smaller than their counterparts Z. If we set all parameters but π_D fixed and consider the final size as a function of Z we note that $Z_d(\pi_D) \to Z$ as $\pi_D \to 0$. As a converse, we note that when $\pi_D \to 1$ we get $Z_d \to 0$.

We note that the behavior of the trajectories of Z in figure 8 is still similar to what we saw in figures 2 and 5. Minor outbreaks for $R_0 \leq 1$ followed by a sharp transitional period and ultimately convergence. It is however no longer true that it converges to a point where $Z \to N - m$ as is evidenced in the y-axis in both plots. In the case of π_R, simulations fail to reach the point $Z = N - m$ even with $R_0 = 30$ which is not too surprising since for larger π_R we note larger infectious periods and as a result, an epidemic which lasts longer. This gives the model more opportunities to discover the disease and immediately stop the epidemic since discovery happens independently once every time step.

Discovery only depends on the total number of removed individuals at time

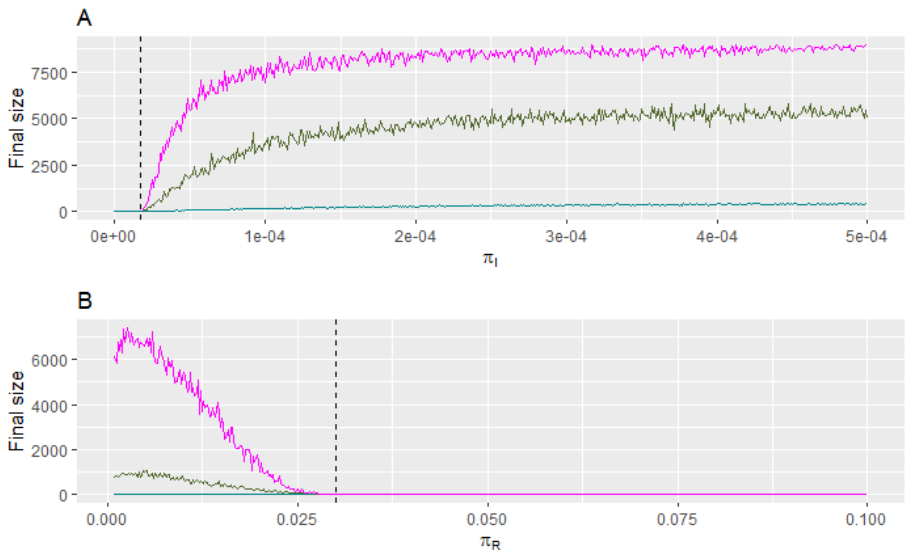

Figure 8: Plots are created with $N = 3000$, $k = 4$, $\pi_S = 0.001$, $\mu = 1$. Both plots have been produced by running the free variable over a sequence of values of length 500, running 100 iterations for each value and taking the mean. In A, π_I ranges from 0 to 0.0005 coresponding to $R_0 \in [0, 30]$ with $\pi_R = 0.05$. In B, π_R ranges from 0.001 to 0.1 corresponding to $R_0 \in [30, 0.3]$ with $\pi_I = 0.00001$. The vertical line is set at the point where $R_0 = 1$, turquoise represents $\pi_D = 0.001$, olive represents $\pi_D = 1e - 5$ and magenta for $\pi_D = 1e - 7$.

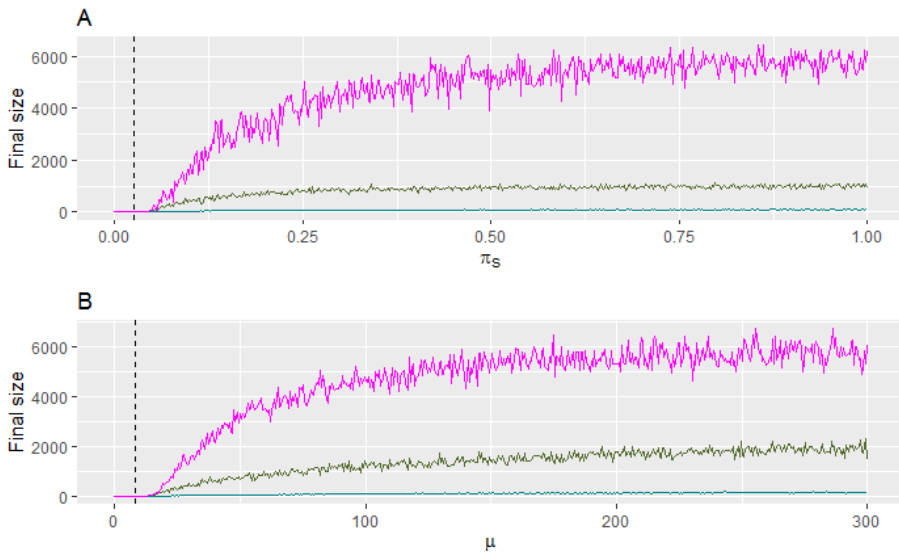

Figure 9: Plots are created with $N = 3000$, $k = 4$, $\pi_I = 0.00001$, $\pi_S = 0.1$. Both plots have been produced by running the free variable over a sequence of values of length 500, running 100 iterations for each value and taking the mean. In A, π_S ranges from 0 to 1 corresponding to $R_0 \in [0.299, 27]$ with $\mu = 30$. In B, μ ranges from 0 to 300 corresponding to $R_0 \in [30, 0.3]$ with $\pi_S = 0.1$. The vertical line is set at the point where $R_0 = 1$, turquoise represents $\pi_D = 0.001$, olive represents $\pi_D = 1e-5$ and magenta for $\pi_D = 1e-7$.

t and we thus find that

$$\begin{aligned}
&\mathbb{P}(\text{Fail to discover disease}|T) = \\
&= \mathbb{P}(\text{Fail to discover disease at time } T-1, ..., 1|T) \\
&= \prod_{t=1}^{T-1} (1 - p_D(t)) = \prod_{t=1}^{T-1} (1 - \pi_D)^{R(t)} \\
&= (1 - \pi_D)^{\sum_{t=1}^{T-1} R(t)} \leq (1 - \pi_D)^{(T-1)R(T-1)}
\end{aligned} \tag{18}$$

This bound is quite crude and somewhat uninformative since $R(T-1)$ may be quite large in comparison to $R(t)$ for $t < T-1$ however it does nicely prove how the probability of never discovering the disease in the population tends to 0 both as π_R shrinks towards 0 and when the size of the population N is large.

In figure 9 we observe similarily created plots but this time with π_S and μ varying instead of π_I and π_R. Much like we noticed when comparing figure 8 with its predecessor figure 5, we note that for $\pi_D = 1e-7$ (line in magenta), there is barely any difference between the trajectories in figures 9 and 6. For π_S in A, there is a slight difference in the maximum size of Z compared to the

maximum Z observed in 6A while for μ in B, the magnitude of the final size seems to be the same for both 6B and 9B.

While both π_S and μ play a part in the probability of discovering the disease we theorize that their impact is minor when compared to that of π_I and π_S. We know the structure of the probability of failing to discover, i.e., $\mathbb{P}(Z_d = Z)$, and the form it has makes it simple enough to estimate by using data from our simulations, but the difficulty of talking about the impact various parameters has is contained in the difficulty of saying how $R(t)$ is affected by the same parameters. However, based on what we have seen previously, the final size is heavily dependent on the basic reproduction number R_0 which we in subsection 3.3 illustrated for varying parameters. There we illustrated the minor impact that both π_S and μ have on the full R_0 and their dependance on parameters π_I and π_R.

While we see that here, our assumptions about properties of Z dependent on the size of R_0 remains true we may still keep a bit of skepcisism towards the quantity. In section 2.4 where the next-generation matrix, the basis for the multi-type R_0, was derived, we did not take discovery of the disease into account. As a result we may be careful with interpreting R_0 as the actual basic reproduction number in the model with $\pi_D > 0$ and instead simply use it for comparison with identical simulations where $\pi_D = 0$, much like in the comparison of Z_d and Z.

Another important property of Z that was briefly mentioned in section 3.1 and observed in 3.2 is the bimodality inherent in the variable as $R_0 > 1$. In 3.2 we confirmed this property and noted that the number of outbreaks resulting in $Z = 0$ quickly shrank towards 0 as the reproduction number grew larger. We have already stated that when $\pi_D > 0$ we will have $Z_d \leq Z$ and as such we theorize that this property of bimodality will be unavailable in this setting.

Despite having complicated the structure of the model since section 3.2 we still get quite similar results in final sizes when comparing figure 10 with figure 3. The main thing that changes the distribution of Z is obviously the discovery property. We reasoned that since $Z_d \leq Z$, prematurely stopping the epidemic would only make the final sizes lower than their counterpart where alternate stopping is impossible. This is quite apparent in all six histograms in figure 10. Looking at the three upper histograms we note a distribution of Z very similar to those exhibited in figure 3. In the histograms in figure 3, a significant majority of outbreaks result in $Z = 0$, something which is true in figure 10 too. However, the main difference lies in the severity of the outbreaks. In the histogram for $R_0 = 0.75$ in figure 3, the largest outliers of final sizes lies around 200 while in figure 10 this fails to break past 100. For the bottom three histograms the distributions differ a lot more between those in figure 3 and those in figure 10. As theorized, bimodality is no longer inherent for Z_d but there is a hint of this property left in all three of the bottom histograms in figure 10. When $R_0 = 5$, the two main outcomes of Z are $Z = 0$ and $Z = N - m$ where their ratio seems to correspond to a similar ratio observed in the same setting in figure 3. The same seems to ring true for $R_0 = 10$ even if there are now a larger middle ground than there are outcomes $Z = 0$ while for $R_0 = 50$ the number of $Z = 0$ cases

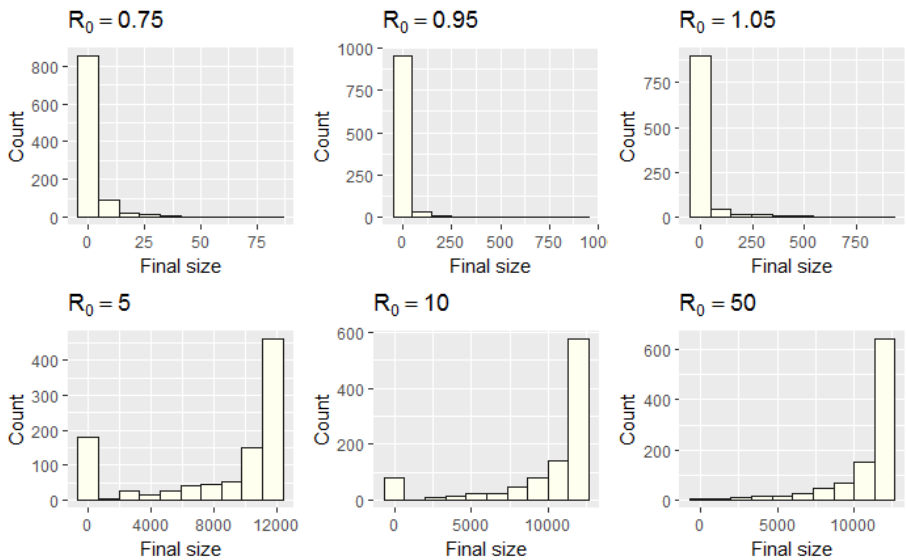

Figure 10: Histograms are created through simulations with $N = 3000$, $\pi_R = 0.05$, $\pi_S = 0.1$, $\mu = 1$ and π_I chosen such that R_0 is what is given in the titles of the separate plots. Data has been obatined by running 1000 iterations and collecting the final sizes Z. Probability of discovery is $\pi_D = 1e - 6$ for all six plots.

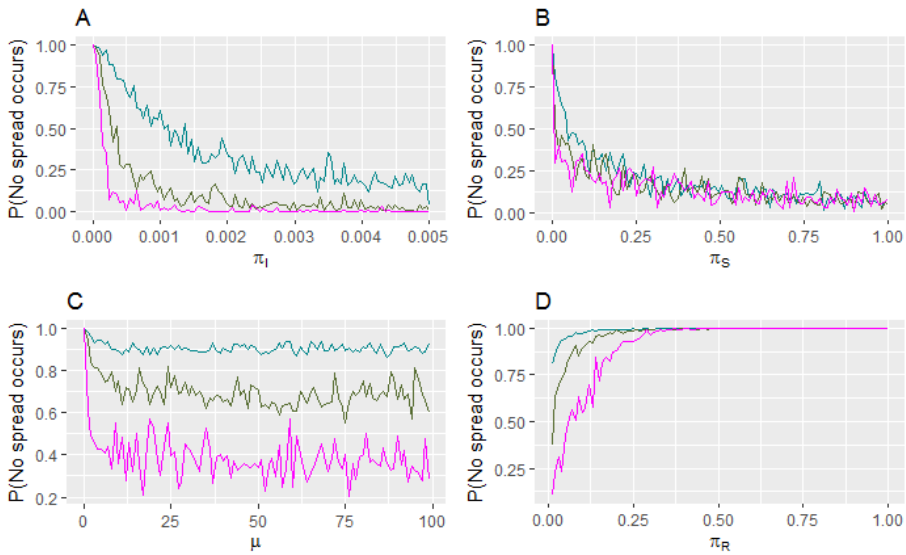

Figure 11: Estimation of the probability of spread occuring. Fixed parameters are $N_j = 3000$, $k = 4$, $\pi_I = 0.0001$, $\pi_R = 0.1$, $\pi_S = 0.001$ and $\mu = 1$. In A, π_I varies over a sequence from 0 to 0.005. In B, π_S varies over a sequence from 0 to 1. In C, μ varies over a sequence from 0 to 100. In D, π_R varies over a sequence from 0.01 to 1. All sequences are of length 100 and values for $\mathcal{P}$ have been obtained by running 100 simulations for each value and taking the mean. Turquoise corresponds to $\pi_D = 0.1$, olive to $\pi_D = 0.01$ and magenta to $\pi_D = 0.001$.

are barely noticeable.

We theorize that even when $\pi_D > 0$, Z converges to some distribution as R_0 grows which is not necessarily bimodal but where the the distribution of $Z = 0$ and $Z = N - m$ outcomes is similar to that of when $\pi_D = 0$ albeit allowing for more than simply two outcomes.

In section 3.3 we briefly opened up for analysis of the probability that the virus fails to spread from the initially infected stable ($m = m_i = 1$) during the run of the epidemic. We will here extend upon this, repeating the analysis in a setting where $\pi_D > 0$ and discovery is thus allowed.

We note that the derivation of the probability of no spread occuring is still sound in the setting where discovery is included. The only assumption made was that we knew the time T when the epidemic is first stopped (no spread is possible once this has occured) but this includes any alternative stopping criterion we add to our model. The main difference lies in the data used for estimation of the probability. Since the probability depends not only on the time T when the epidemic is stopped but also on the full history of the epidemic, most importantly on the $I_i(t)$'s where i is the stable of the initial infective.

Parameters are used identical to those used in the construction of figure 7 with the main difference being that we now have included the discovery parameter which will change the data available. The results are available in figure 11.

The magenta lines are the ones most closely resembling the trajectories observed in figure 7. For these, $\pi_D = 0.001$ and we may see this as the trajectory stabilizing into the multi-type result as π_D tends to 0. It is however surprising how quickly this stabilisation occurs. Contrast with the trajectories of the final size Z_d. We know that $\pi_D \to 0 \Leftrightarrow Z_d \to Z$ and in figures 8 and 9 this holds true when we set $\pi_D = 1e-7$. However in this setting we may take π_D nearly 10000 times larger and still achieve a considerable stabilization.

In A we notice quick stabilisation as π_D gets smaller but also higher probability that non-infected stables escape the infection for larger π_D. Naturally, this behavior ought to appear in all plots - as π_D gets larger we have an increasingly large probability of prematurely stopping the epidemic which here hopefully happens before the epidemic jumps into a new stable.

This is most clear in plot C where we see large differences in the probability of the disease failing to spread for the different π_D. As $\mu \to \infty$ the trajectory converges quickly. This is noticeable from the structure of the inner part of the probability $\mathcal{P}^*$ where,

$$\mathcal{P}^* = 1 - p_{S,i}(t)(1 - e^{-\mu})(1 - ((1 - \pi_I)^{\iota_{ij}(t)})^{N_j}).$$

We have that, as $\mu \to \infty$, $(1 - e^{-\mu})$ converges fast to 1. In addition to this, for $N = 3000, \pi_I = 0.001$ and approximating $\iota_{ij}(t) \approx \mu$ we get that $\mathcal{P}^* \to 1 - p_{S,i}(t)$ for $\mu \geq 14$ as evidenced by C. At this point the only variation left in the equation is in $I_i(t)$ and the change in variation noticeable between the three trajectories is as such a result of the variation in $I_i(t)$ which comes with lower values of π_D.

In D we see something similar too, although for a much shorter duration. As π_R tends to 1 we see the probability of never spreading goes along with it. The reasoning behind this is that when π_R is large we see an increased probability in individuals immediately recovering which includes the initially infected and as such, an increased probability of immediately stopping the epidemic. As such, π_D quickly stops playing a part when π_R tends to 1 since individuals will be removed before they are able to spread into other stables.

In plot B we see that when π_S tends to 1 we converge to a single unified point independent of the discovery probability. In fact, when π_S tends to one we find that

$$\lim_{\pi_S \to 1} \mathcal{P}^* = 1 - (1 - e^{-\mu}) \left(1 - ((1 - \pi_I)^{\iota_{ij}(t)})^{N_j}\right),$$

which with the parameters we have used for figure 11, along with assuming $\iota_{ij}(t) \approx \mu$ gives us that $\mathcal{P}^* \approx 1 - 0.16 = 0.84$. Now this number is not the entire probability of spread not occuring since we still need to raise it to the power of $k - 1$ as well as further raising it to the power of $T - 1$. With the values we have specified in figure 11 we will wind up with something akin to $(0.6)^{T-1}$ which indeed tends to 0 quickly but still has a fair chance of remaining large if the disease is stopped quickly.

Similar results hold when we take the limit as $\mu \to \infty$ or $\pi_I \to 1$ but these are more difficult to describe since they will still be dependent on $p_{S,i}(t)$ and thus in turn on $I_i(t)$.

To clearly say something about stable isolation as a preventive measure against a full outbreak we would need more information about several of the parameters. The size of the population N is of great importance but for this we are able to chose reasonable bounds by the restrictions set by Jordbruksverket and due to the scarcity of NDV in Sweden we may set $m = m_i = 1$ as a way to represent this. What is more difficult to estimate are the parameters relating to the infectivity of the disease. The parameter π_I depends both on actual infectivity of the virus but depends also on the behavior of individuals in the population and the amount of contact they have with each other. Adding to the uncertainty relating to infectivity, it is difficult to say what impact infective substance μ would have when added into the live population. If we consider the added pressure $\iota_{ij}(t)$ to be infective substance left inside a stable after human contact then this would behave differently than an infective individual would: it remains fixed in place and behaves more as a contaminated site than it does as an infective individual. Regardless, we have often used $\mu = 1$ to signify that the average added pressure functions as another infective individual (due to the large population size, the impact of an extra infective would be minor when the number of infectives is great but important when the number of infectives is low). The probability of being removed is equally difficult to estimate. The world organization for animal health reports that for velogenic strains mortality rates may approach 100% but does not say anything about how long infective individuals survive.

Lastly, we have the parameters relating to human interaction. These are even more difficult to estimate, but for the sake of prevention of spread, these can at the very least be controlled. Stable contact from stable i at time t occurs according to the binomial random variable $T_{S,i}(t) \sim \text{Bin}(k-1, p_{S,i}(t))$ where $p_{S,i}(t)$ is random and dependent on the number of infective individuals at time t, $I_i(t)$. We have incorporated this property of the model with the argument that when farm staff enters the stable, infective substance may follow the staff upon exit. If cleanliness routines are not fully respected at this point, this infective substance may follow into a susceptible stable where it is picked up by a susceptible individual and allowed to begin the spread inside this stable. Luckily, this probability can be controlled and as ordered for by the world organization for animal health, important routines for the sake of preventing outbreaks are, among others, "proper carcass disposal", "bird-proofing houses" and "control of human traffic". We theorize that through proper routines and preventive measures against the disease, one may reduce the probability of stable contact which as such reduces the probability of between-stable-spread occuring.

In addition to this, by setting the probability of discovery large enough one may immediately stop the disease upon infection after which one may remove the infective and save the remaining population from a greater outbreak. The problem here is that this is much more easily done in theory than it is done in practice. Firstly, immediately recognizing NDV in a large population would be

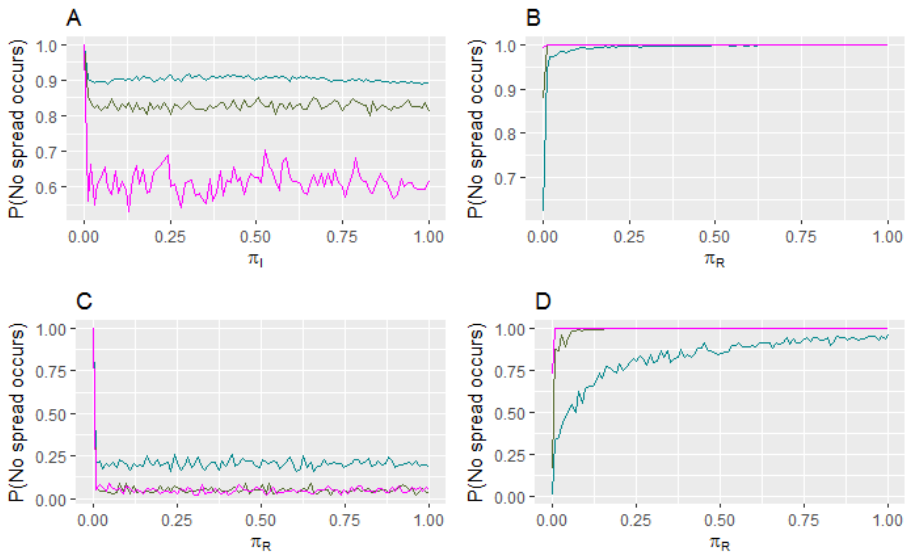

Figure 12: We set $N = 3000$, $k = 4$, and $\mu = 1$. We let π_I vary along a grid from 0 to 1 of length 100 whereas π_R varies along a grid from $1e-4$ to 1 also of length 100. In plots A and B, $\pi_S = 1e-5$ and $\pi_D = 0.1$ while in C and D $\pi_S = 1e-4$ and $\pi_D = 0.01$. When π_I is fixed, turqouise corresponds to $\pi_I = 0.001$ with olive being $1e-4$ and magenta $\pi_I = 1e-5$. When π_R is fixed, turquoise corresponds to $\pi_R = 0.01$, with olive being 0.001 and magenta $\pi_R = 1e-4$.

difficult and secondly, even if the initially infective is immediately removed, this does not necessarily stop the disease as infective substance may remain inside the stable, further infecting the remaining susceptibles after an individual has been removed.

We close this section of with a plot created under these assumptions. Letting $\pi_S \approx 0$ and with π_D large, we let π_I and π_R vary and estimate the probability of no spread occuring during the epidemic. In plots A and B we have set π_S and π_D to what we consider optimistic values. Both adhere to the assumptions made on the parameters. The results are also optimistic. As π_I varies throughout its entire domain we only ever reach a probability of 0.5 and this is when we set $\pi_R = 1e-4$ corresponding to infectious periods of 10000 days. In B we note the lowest probability at around a similar value of π_R as its trajectory starts. Here the probability almost immediately rises and converges to 1, estimating it as nearly impossible to spread into another stable. The lowest R_0 in A is obviously at the point where $\pi_I = 0$ but looking at how the probability converges early on regardless of the size of π_I we may take more or less any point arriving at R_0 huge which is also promising. Looking at B and especially at the magenta line we have that $R_0 = 300$ at the point where the magenta line starts its trajectory

which is similarily large. Remember that, as $\pi_R \to 1$, R_0 becomes smaller which, with our previous results about the outcome of an outbreak for large π_D from figure 11, leads us to theorize that the probability of no spread occuring is independent of R_0 for large π_D.

In C and D we have allowed ourselves to be less optimistic which unfortunately leads to less positive results. In the plot for D the trajectories converge to 1 although slowly with the turquoise line where $\pi_I = 0.001$ barely even reaching 1 before π_R reaches its maximum value. In the plot for C things are even less certain where the probability immediately dives down, stabilizing around 0.25 or even lower and tending to 0 as we pick successively lower π_R.

Our conclusion here is that one can prevent major outbreaks regardless of how infective the virus is by making sure that cleanliness routines are withheld and by being vigilant and alert for potential outbreaks. In practice this would amount to strangling between-stable-contact while also keeping an eye out for deceased individuals and unnatural behavior. If this is done to the correct degree (which in our model corresponds to the level of $\pi_S \leq 1e-5$ and $\pi_D \geq 0.1$) then there is a low, but still positive, probability that spread between stable occurs and upon discovered infection isolated stables may be considered safe. However, if these routines are not kept to the appropriate degree, it will take very little for an infectious enough disease to contact all stables and wipe out the entire population on its own.

4 Discussion

The objective of this thesis was to create a stochastic model for the spread of Newcastle disease in a farm population. Several assumptions have been made to cater to this situation and our limited knowledge of both the Newcastle disease virus as well as the routines and procedures apparent on a typical poultry farm have led to approximations and simplifications to aid the mathematical model in both simplicity and analytic tractability. Realism has remained a constant through the definition of the model but not a main priority. In fact, while it has been nearly a century since the discovery of NDV, due to the variation inherent in the virus, both in different strains but also in manifestation amongst different types of fowl, little can be stated as to how long infected individuals survive, how infectious the disease is or to what extent infected substance compares to direct contact.

This leads us into one of the main complications with the goal of this thesis and that is in its usefulness. The model allows for a large variation of values and the structure, for instance when it comes to between-stable-spread, is complex enough to take multiple factors into account. However, when there is no good way to estimate parameters it is difficult to use either the equation for the reproduction number, the simulations or proposition 1 to give results applicable to a real-life farm setting.

Our goals were to create a model, which we have succeeded with, but also to use this model to say something about how well isolation of birds into stables

would work. This has proven to be a more difficult task than expected and we have only been able to look at simple cases where single variables have been allowed to vary. We have written a program for estimation of this probability using the coded simulation of the epidemic but this again runs into the issue of requiring parameter input to be useful.

As a consequence of this, our obtained results mostly relate to certain analytic derivations along with a closer study of the final size Z. We successfully derive an equation for the basic reproduction number in the case of $k = 1$ stables along with deriving the next-generation matrix A_k which can be used to derive R_0 in a multi-type setting ($k > 1$) either numerically or analytically for k low. For R_0, the perhaps most interesting result is the one given in figure 4 where we illustrate the size of R_0 as the involved parameters are allowed to vary. While we are unable to derive a general formula for R_0 when $k > 1$ we may still use these results to get an understanding of to what degree various parameters impact the R_0. The main surprise here is how little effect the probability of spread π_S and the expected amount of added pressure upon successful stable contact μ has on the reproduction number and final size. With more time we would have liked to extend the computation of R_0 to include the discovery parameter as well as being able to conclude something more about the structure of R_0 with more than only a single parameter varying.

Due to time limitations we were also unable to extend the caged model into more than simple comments and initial observations. An interesting question would be to compare the ability of both interior spread between caged birds and the free-roaming birds and to look into whether cages further enable the spread of NDV or acts as a neutralizer, slowing the speed of the spread down. We conjecture that the caged model would see less extensive spread than the other models, leaving comments on which one is ultimately *better* to be unsaid.

As a model for NDV we argue that this model is both realistic and appropriate. The only questionable assumption is whether taking Swedish farms as a setting for the virus is appropriate. Not only is it an odd source of inspiration due to NDV not being as widespread in Sweden as it is in other countries but adding to this we are also assuming that the strain of the virus which we are modeling is of the velogenic kind, i.e, the most infectious and deadly kind (otherwise the SIR-model would be inappropriate) however this virus strain is not said to be found in Europe (World organization for animal health). Despite this, the reasoning behind the various parts of the model are sound and reasonable and the model in its general form (as detailed in sections 2.2 and 2.3) can be applied to a wide range of similar diseases in similar settings. An example of such a disease would be foot-and-mouth disease which spreads primarily through direct contact but where the virus may also be carried via humans through clothing, vehicles or equipment (Jordbruksverket). Foot-and-mouth disease infects cloven hoofed animals and as such assumptions detailed in section 2.1 on farm structure along with population and contact would have to be looked over and changed from poultry to cleft-hoofed be able to apply the model.

As we have repeated throughout this thesis, our goal with the creation of this model has been to find an equilibrium between simplification and realism as is

often the goal with any model. The statistician George Box famously said that "all models are wrong but some are useful" and this statement indeed rings true for our model too. Several extensions which we have glossed over could be implemented, making the model no less wrong but perhaps slightly more useful. An immediate example would be to correct the discovery probability and extending it to also depend on the infected population. In a real-world setting, we assume that the farm hand is not blind to symptoms in the farm population and as such a more realistic definition would be

$$p_D(t) = 1 - (1 - \pi_D)^{cI(t)+R(t)},$$

where c is used to quantify the weight that symptoms in live individuals have on the discovery probability. This idea was mentioned in section 2.4 but it was dismissed due to the difficulties in estimating c as quantifying such a behavior is not easy.

Another interesting extension would be to look at different distributions of the added exterior pressure, $\iota_{ij}(t)|C_{ij}(t) = 1$. We have chosen this to be Poisson(μ) but there is no set limitation on this distribution and it may very well be chosen to be deterministic which might simplify the structure of the model.

Other properties that have been considered but ultimately dismissed relate to spatial structure inside a stable, implementing site contamination for infective substance and making the contact parameter λ vary between λ_{in} of contact rate inside stables and λ_{out} of contact rates when outdoors birds are allowed outside.

Appendix

Proposition 1: If the time T when the epidemic is stopped is known and we assume that $m_i = 1$ with $m_j = 0$ for all $j \neq i$ then

$$\begin{aligned}&\mathbb{P}(\text{The disease never spreads}) = \\ &= \prod_{t=1}^{T-1} \left(\prod_{j \neq i} \left(1 - p_{S,i}(t) \left(1 - e^{-\mu}\right) \left(1 - ((1-\pi_I)^{\iota_{ij}(\tau)}\right)^{N_j} \right) \right) \end{aligned} \tag{19}$$

Proof: For the proof we use several variables and results from subsection 2.4. We have that

$$\begin{aligned}&\mathbb{P}(\text{The disease never spreads}) = \\ &= \mathbb{P}(\text{Disease fails to spread at time } T-1, ..., \text{disease fails to spread at time } 1) \\ &= \mathbb{P}(\text{Fails to spread at time } T-1 | \text{Fails at } T-2, ..., 1)\mathbb{P}(\text{Fails at } T-2, ..., 1) \\ &= \prod_{t=1}^{T-1} \mathbb{P}(\text{Fails to spread at time } T-t | \text{Fails at } T-t-1, ..., 1) \end{aligned} \tag{20}$$

For $\tau \in \{1, ..., T-1\}$ we have that, using that we know that the disease has failed to spread at every previous timestep $t < \tau$,

$$\begin{aligned}&\mathbb{P}(\text{Fails to spread at time } \tau) = \\ &= \mathbb{P}(\text{Stable 1 fails to spread into stable } 1, ..., \text{Stable } k \text{ fails to spread into stable } k) \\ &= \prod_{j=1}^{k} \prod_{i=1}^{k} \mathbb{P}(\text{Stable } i \text{ fails to spread into stable } j \text{ at time } \tau) \\ &= \prod_{j \neq i} \mathbb{P}(\text{Stable } i \text{ fails to spread into stable } j \text{ at time } \tau) \end{aligned} \tag{21}$$

Here we have used that the events that a given stable spreads into another given stable are independent along with the assumption of $m_i = 1$ with $m_j = 0$ for all $j \neq i$. The last assumptions gives us that $\mathbb{P}(C_{ji}(t) = 0) = 1$ along with how we have defined that $\mathbb{P}(C_{ii}(t) = 0) = 1$ for all t. Continuing we get that

$$\begin{aligned}&\mathbb{P}(\text{Stable } i \text{ fails to spread into stable } j \text{ at time } \tau) = \\ &= \mathbb{P}(C_{ij}(\tau) = 0 \ \cup \ \{C_{ij}(\tau) = 1, \iota_{ij}(\tau) = 0\} \ \cup \ \{C_{ij}(\tau) = 1, \iota_{ij}(\tau) > 0, T_{I,j}(\tau) = 0\}) \\ &= \mathbb{P}(C_{ij}(\tau) = 0) + \mathbb{P}(C_{ij}(\tau) = 1, \iota_{ij}(\tau) = 0) + \mathbb{P}(C_{ij}(\tau), \iota_{ij}(\tau) > 0, T_{I,j}(\tau) = 0) \\ &= A + B + C. \end{aligned} \tag{22}$$

We continue by looking at the probabilities of these events separately.

$$
\begin{aligned}
A = \mathbb{P}(C_{ij}(\tau) = 0) &= \sum_{y=0}^{k-1} \mathbb{P}(C_{ij}(\tau) = 0 | T_{S,i}(\tau) = y) \mathbb{P}(T_{S,i}(\tau) = y) \\
&= \sum_{y=0}^{k-1} \left(1 - \frac{y}{k-1}\right) \mathbb{P}(T_{S,i}(\tau) = y) = 1 - \frac{1}{k-1}(k-1) p_{S,i}(\tau) = 1 - p_{S,i}(\tau)
\end{aligned} \tag{23}
$$

$$
\begin{aligned}
B &= \mathbb{P}(C_{ij}(\tau) = 1, \iota_{ij}(\tau) = 0) = \mathbb{P}(\iota_{ij}(\tau) = 0 | C_{ij}(\tau) = 1) \mathbb{P}(C_{ij}(\tau) = 1) \\
&= e^{-\mu} \left(1 - \mathbb{P}(C_{ij}(\tau) = 0)\right) = e^{-\mu} p_{S,i}(\tau)
\end{aligned} \tag{24}
$$

$$
\begin{aligned}
C &= \mathbb{P}(C_{ij}(\tau) = 1, \iota_{ij}(\tau) > 0, T_{I,j}(\tau) = 0) \\
&= \mathbb{P}(T_{I,j}(\tau) = 0 | C_{ij}(\tau) = 1, \iota_{ij}(\tau) > 0) \mathbb{P}(C_{ij}(\tau) = 1, \iota_{ij}(\tau) > 0) \\
&= \mathbb{P}(T_{I,j}(\tau) = 0 | C_{ij}(\tau) = 1, \iota_{ij}(\tau) > 0) \mathbb{P}(\iota_{ij}(\tau) > 0 | C_{ij}(\tau) = 1) \mathbb{P}(C_{ij}(\tau) = 1) \\
&= \left(1 - (1 - (1 - \pi_I)^{I_j(\tau) + \iota_{ij}(\tau)})\right)^{S_j(t)} \left(1 - \mathbb{P}(\iota_{ij}(\tau) = 0 | C_{ij}(\tau) = 1)\right) \left(1 - \mathbb{P}(C_{ij}(\tau) = 0)\right) \\
&= \left((1 - \pi_I)^{\iota_{ij}(\tau)}\right)^{N_j} \left(1 - e^{-\mu}\right) \left(p_{S,i}(\tau)\right)
\end{aligned} \tag{25}
$$

where $\iota_{ij}(\tau) > 0$.

$$
\begin{aligned}
\Rightarrow A + B + C &= 1 - p_{S,i}(\tau) + e^{-\mu} p_{S,i}(\tau) + \left((1 - \pi_I)^{\iota_{ij}(\tau)}\right)^{N_j} \left(1 - e^{-\mu}\right) \left(p_{S,i}(\tau)\right) \\
&= 1 - p_{S,i}(\tau) \left(1 - e^{-\mu} - \left((1 - \pi_I)^{\iota_{ij}(\tau)}\right)^{N_j} \left(1 - e^{-\mu}\right)\right) \\
&= 1 - p_{S,i}(\tau) \left(1 - e^{-\mu}\right) \left(1 - ((1 - \pi_I)^{\iota_{ij}(\tau)})^{N_j}\right)
\end{aligned} \tag{26}
$$

Plugging this back into equation 21 gives us that

$$
\begin{aligned}
&\mathbb{P}(\text{The disease never spreads}) = \\
&\prod_{t=1}^{T-1} \left(\prod_{j \neq i} \left(1 - p_{S,i}(t) \left(1 - e^{-\mu}\right) \left(1 - ((1 - \pi_I)^{\iota_{ij}(t)})^{N_j}\right)\right)\right),
\end{aligned} \tag{27}
$$

where $\iota_{ij}(t) > 0$. This completes the proof.

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