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# **Stochastic epidemic models on random networks: casual contacts, clustering and vaccination**

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## **Abstract**

There has been considerable recent interest in models for epidemics on networks describing social contacts. This thesis considers a stochastic SIR (Susceptible - Infective - Removed) model for the spread of an epidemic among a population of individuals, with a random network of social contacts, that is partitioned into households and in which individuals also make casual contacts, i.e. with people chosen uniformly at random from the population. The behaviour of the model as the population tends to infinity is investigated. A threshold parameter that governs whether or not the epidemic with an initial infective can become established is obtained, as is the probability that such an outbreak occurs and, if so, how large it will become. The behaviour of this model is then compared to that of a finite population using Monte Carlo simulations. The effect of the different transmission routes on the final outcome of an epidemic and the effect of introducing social contacts and clustering to the network on the performance of various vaccination strategies are also investigated.

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# 1. Introduction

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## 1.1 Motivation and early epidemic models

History is filled with examples of epidemics infecting large numbers of people, such as the 1918 influenza epidemic or the Black Death which both killed millions. Indeed, recent notable events often include epidemics, such as swine flu and the Ebola virus. Therefore there is a lot of interest in both understanding diseases and mathematically modelling the spread of epidemics. A mathematical model can give insight into effective vaccination strategies or the required healthcare infrastructure in a community. One of the earliest accounts of mathematical modelling of epidemics is by Daniel Bernoulli (1760) (see Hethcote (2000)), who created a mathematical model to investigate the impact of a vaccination strategy on the spread of smallpox. Since Bernoulli's work, the mathematical modelling of epidemics has continued, with drastically increased popularity from the early 20th century with the differential equation model of malaria by Ross (1911) and the widely cited deterministic epidemic model by Kermack and McKendrick (1927).

In most epidemic models, individuals in the population are assigned to different subgroups, each representing a stage of the epidemic. At any given time an individual belongs to a single subgroup, but individuals can change subgroup over time. One of the most common epidemic models, the SIR model, involves splitting the population into three subgroups: susceptible, infective and removed. The susceptible subgroup represents the individuals not yet infected by the disease, the infective subgroup represents the individuals that are currently infected and the removed subgroup represents the individuals that are no longer affected by the disease (e.g. due to death or natural immunization). Removed individuals are unable to be infected again or transmit the infection. An epi-

demic is said to end when there are no individuals in the infective subgroup. Another example of a popular epidemic model is the SIS epidemic model, which is achieved from the SIR model by assuming that individuals are immediately susceptible again once they have recovered from infection.

Indeed, a special case of the model of Kermack and McKendrick (1927) considers a finite fixed population with an SIR epidemic model, assuming that individuals are homogeneously mixing, i.e. that an infected individual is equally likely to infect any other susceptible individual in the population. One finding of Kermack and McKendrick is that an epidemic often comes to an end before the susceptible population is exhausted, leading to the question of what proportion of the population will be in the removed subgroup when the epidemic ends.

## 1.2 Threshold theorems and early stochastic epidemic models

Kermack and McKendrick comment that a threshold density of the population exists, in which no large epidemic can occur if the population density is below this threshold value, although Heesterbeek (2002) cites the work of En'ko (1889*a*), (1889*b*), as possibly being the first work which implies the existence of a threshold determining whether large epidemics can occur. However, it is after the work of Bailey (1953), who defines a stochastic SIR epidemic with infection and recovery rates, that threshold parameters become better understood. Bailey uses the ratio of the infection and recovery rates to give a distribution for the final size of an epidemic, i.e. the number of individuals in the removed subgroup when the epidemic ends. Building on the work of Bailey, Whittle (1955) simplifies the calculation of the final size distribution and compares the ratio of the infection and recovery rates to the total population size as a threshold parameter, showing that if this ratio exceeds the population size then a large epidemic cannot occur. However, it is the basic reproduction number,  $R_0$ , which has become the most popular threshold parameter in mathematical epidemic modelling. A key contribution towards understanding  $R_0$  is the work of Bartoszyński (1967), who equates epidemic models to branching processes. By relating births in the branching process to individuals becoming infected in the epidemic process, branching process theory provides a natural framework for the study of epidemics. For example, Ball (1983) shows a strong convergence of the number of infectives in a stochastic homogeneously mixing epidemic model

containing  $N$  individuals to the number of individuals in a birth-and-death process as  $N \rightarrow \infty$ . Since the branching process approximation assumes an asymptotic population size, we define the relative final size of a major outbreak to be the number of individuals in the removed subgroup at the end of the epidemic divided by the total population size. Branching process theory also clarifies our understanding of  $R_0$ , which can be interpreted as the mean number of offspring of a single individual in the branching process. Thus  $R_0$  has a clear biological interpretation as the total number of individuals infected by a single infective. Furthermore, if  $R_0 < 1$  or  $R_0 = 1$  then the branching process is subcritical or critical respectively, almost surely becoming extinct (i.e. a large outbreak cannot occur in the epidemic), whereas if  $R_0 > 1$  then the branching process is supercritical and there is a positive probability of non-extinction (i.e. a large outbreak can occur). Indeed, if the branching process avoids extinction then a significant proportion of the population in the approximated epidemic is infected and we say that a major outbreak has occurred. Branching processes are key to understanding many more complicated epidemic models, including household and network models, as discussed in the following sections.

One of the earliest stochastic epidemic models is the chain binomial model (later known as the standard Reed-Frost model) discussed by Reed and Frost in lectures given in 1928, although its origins can be traced back to En'ko (1889*a*), (1889*b*). The standard Reed-Frost model considers a closed, homogeneously mixing population, and is a discrete time SIR model. In the standard Reed-Frost model, each infected individual in generation  $t$ ,  $t = 1, 2, \dots$ , independently infects each susceptible individual in the population with some probability  $p$ . Any individual that becomes infected by an infected individual in generation  $t$  belongs to generation  $t + 1$ , and individuals in generation  $t$  become removed. The epidemic ends when there are no individuals in some generation. Von Bahr and Martin-Löf (1980) extend the standard Reed-Frost model to the randomised Reed-Frost process, allowing each individual in the population to have a different probability of infecting others. Indeed, Martin-Löf (1986) shows that the randomised Reed-Frost process is a special case of iterative symmetric sampling procedures, giving more direct and intuitive proofs of the previous results. Furthermore, Ludwig (1974) shows that the threshold theorem for the randomised Reed-Frost process is analogous to the threshold theorem for the deterministic SIR model discussed by Kermack and McKendrick (1927). Picard and Lefèvre (1990) suggest that the probability an infective infects a

set of individuals is a function of the size of the set, rather than the number of susceptibles in the population, and introduce the collective Reed-Frost process. Finally both Bartlett (1955) and Kendall (1956), have also made important contributions to analysing epidemics while they are in progress and exploring how they reach their final size.

### 1.3 Household epidemic models

Although assuming that a population is homogeneously mixing simplifies the calculations involved in epidemic modelling, it is clear that such an assumption is usually unfounded in practice. For example, individuals at opposite ends of the UK are unlikely to directly infect each other. Therefore interest has spread to investigating epidemic models that allow for population heterogeneities, such as households. Watson (1972) introduces a stochastic SIR epidemic model which splits the population into homogeneously mixing communities, and uses the idea of considering the spread of the epidemic based on the propagation of infected communities rather than individuals. However, Watson (1972) assumes that each community contains a large number of individuals. Using a deterministic model, Bartoszyński (1972) considers a population split into smaller communities, such as households, by assuming that in the early stages of the epidemic all infectious contacts made by the infected members of a household are with individuals in fully susceptible households. Indeed, this assumption is key to many of the later household epidemic models. Unlike Watson (1972), who considers a fixed number of large communities, Bartoszyński (1972) investigates a large number of groups of fixed size (with no minimum group size requirement). This idea is extended further by Becker and Dietz (1995) and Ball et al. (1997), who successfully investigate an SIR epidemic on models which can incorporate small households. Becker and Dietz (1995) and Ball et al. (1997) consider the spread of the epidemic through a ‘clump’, i.e. a household, and then consider the number of global contacts made by each clump, leading to a clump-based threshold parameter  $R_*$ . Furthermore, Ball et al. (1997) discuss the final size distribution of an SIR households model. Later, Ball and Neal (2002) introduce susceptibility sets to calculate the final size of major outbreaks in several models, including the households model, in simpler ways than Ball et al. (1997). Adapting the household model to further reflect current epidemics is still a key area of research. For example, Neal (2016) investigates a model in which an individual mixes within a different household or community depending

on the time of day. For example, individuals infect workplace colleagues during the day and household neighbours at night.

Although the clump-based threshold parameter  $R_*$  is a powerful tool in considering the spread of the epidemic, there is substantial interest in extending the definition of  $R_0$  to household-based epidemic models, i.e. calculating a threshold parameter which, if the approximating branching process avoids extinction, represents the ratio of individuals infected in the next generation of the epidemic to the current generation. An attempt to define  $R_0$  in household models is given by Goldstein et al. (2009), who also introduce an ordering of several reproduction numbers. However, Pellis et al. (2012) refine this definition of  $R_0$ , and Ball et al. (2016) provide an alternative method of calculating  $R_0$ . Although this thesis primarily addresses the reproduction numbers  $R_*$  and  $R_0$ , there are many other reproduction numbers in the literature. Indeed, Ball et al. (2016) consider more than 10 reproduction numbers and extend the ordering of reproduction numbers initiated by Goldstein et al. (2009).

Another parameter with clear biological interpretation is the early real-time exponential growth rate of the epidemic. Indeed, Pellis et al. (2011) note that the early real-time exponential growth rate is one of the first and simplest pieces of information available in emerging epidemics, and show its relationship to other parameters of the epidemic. Furthermore, there has been an increasing interest in estimating the real-time growth rate for heterogeneous epidemic models and then calculating other parameters of interest, for example the works of Pellis et al. (2015) and Trapman et al. (2016).

## 1.4 Random networks and epidemics

The probabilistic construction of a graph, i.e. a random graph, was first introduced independently by Erdős and Rényi (1959) and Gilbert (1959), and begins with a network with a fixed number of nodes, in which each pair of nodes is connected independently with probability  $p$ . Although this model is not a realistic interpretation of many applicable networks, as noted by Erdős and Rényi themselves in 1960, their work sparked further investigation into the properties of networks, including social networks. For example, Milgram (1967) conducted an experiment suggesting that realistic models of social networks should contain short chains between individuals, often referred to as

the ‘small-world’ effect. Furthermore, social networks often incorporate a large amount of clustering, i.e. subgroups of connected individuals, with Watts and Strogatz (1998) discussing several real-life examples of small-world networks with clustering. Watts and Strogatz (1998) also introduce both the clustering coefficient, a method of quantifying the amount of clustering in a network, and a method to construct a clustered small-world network. However, Watts and Strogatz (1998) assume that every vertex has the same degree, whereas Albert et al. (1999) show that the degree distribution of some real life networks, such as an actor collaboration network and the World Wide Web, follow a power law for large vertex degree  $k$ . Note that a distribution  $X$  has a power law tail if, for large  $k$ ,  $P(X = k) \approx k^{-\gamma}$ ,  $\gamma > 0$ . Another common property of social networks, discussed by Newman (2002*b*), is that they are assortatively mixed, i.e. vertices in the network tend to be connected to other vertices with similar degrees. Therefore models for social networks often aim to have tuneable degree distribution, clustering and assortativity, and also have small-world properties.

Epidemics on small-world or clustered networks are generally analytically difficult to investigate owing to the existence of multiple connections between two individuals, i.e. multiple routes of transmission of the epidemic between two individuals. Diekmann et al. (1998) construct a deterministic model for the spread of an SIR epidemic on a random network by assuming a constant degree distribution and that, in the early stages of the epidemic, the neighbours of an infected individual, except the source of the infection, are susceptible. Assuming that all neighbours of an infected individual are susceptible, other than their parent in the epidemic process, turns out to be a powerful tool in modelling the spread of epidemics on networks, and is key to the analysis of many recent epidemic models. Andersson (1998) introduces a stochastic Reed-Frost epidemic model on a network in which individuals have variable degree (i.e. the ‘configuration model’). Under the configuration model, each individual in the population is independently assigned a number of ‘half-edges’ from a specified distribution, and these half-edges are then paired uniformly at random to form the edges of the network. Andersson (1998) shows that the early stages of the epidemic are well approximated by a branching process, however Andersson uses a deterministic approximation to calculate the relative final size of a major outbreak. Newman et al. (2001) use generating function arguments to calculate the size of a giant connected component in the configuration model, i.e. the total number of vertices in the largest connected component in the

graph divided by the total number of vertices in the graph. Similar generating function arguments are applied by Newman (2002*a*) to calculate the expected relative final size of a major outbreak on the configuration model. Meanwhile, Ball and Neal (2002) use susceptibility set arguments to calculate the expected relative final size of a major outbreak on the great circle model, which is closely related to the model of Watts and Strogatz (1998). The great circle model involves a population of individuals equally spaced around a circle, and infected individuals can make both local contacts (i.e. contacts with individuals near them in the circle) and global contacts (i.e. contacts with individuals chosen uniformly at random from the population) with different rates. Indeed, Ball and Neal (2003) give a central limit theorem for the relative final size of a major outbreak on the great circle model given restrictions on the local and global contact process. Using the susceptibility set arguments introduced by Ball and Neal (2002), Ball and Neal (2008) extend the configuration model to include global contacts, so an infected individual can make contacts with both network neighbours and individuals chosen uniformly at random from the population, and prove that the expected relative final size of a major outbreak is equal to the probability that a ‘backwards’ branching process avoids extinction.

The configuration model has proven to be a popular model for networks with arbitrary but specified degree distributions, and allows for the calculation of many properties of epidemics on the network, such as threshold parameters and the expected relative final size of a major outbreak. However, the methods used to calculate these properties rely on the assumption that there are not multiple routes of transmission of the epidemic between two individuals (note that the configuration model has negligible clustering and is not a small-world network). Although an important assumption for the coupling theorems between branching processes and epidemic processes on networks, the lack of clustering in the configuration model hinders its applications to real-world networks, and is therefore an important restriction to relax. One method of introducing clustering to the configuration model is introduced by Trapman (2007), who replaces some vertices in the network with households (i.e. complete graphs), and each member of a household has a single neighbour outside of its household. By considering the spread of the epidemic through household clumps, similarly to Becker and Dietz (1995) and Ball et al. (1997), and assuming that each household infects individuals in disjoint households, Trapman (2007) uses a branching process to approximate the early stages of the epidemic. A similar model proposed

by Ball et al. (2009), (2010), involves considering a population already split into households and then constructing a global network using the configuration model. Ball et al. (2009), (2010), formally couple the epidemic process and an approximating branching process, and calculate the expected relative final size of a major outbreak (and other key properties of the model). Meanwhile, Gleeson (2009) introduces a similar network model to Ball et al. (2009), based on creating a network with complete subgraphs, although Gleeson constructs the network beginning with a target clustering coefficient. Gleeson also considers the bond percolation problem, rather than the spread of an epidemic, which involves deleting each edge in the network independently with a given probability and then considering the size of the giant connected component of the graph. The bond percolation problem for a network is very similar to studying the spread of an epidemic on the network, where the probability an edge is deleted is related to the average transmissibility of the disease, and the size of the giant connected component is related to the expected relative final size of a major outbreak.

In contrast, Newman (2009) introduces a clustered network by modifying the configuration model to allow individuals to also belong to a given number of corners of triangles, which are then randomly connected to form triangles in the network. Newman then considers the percolation problem on the network to calculate the final size of a major outbreak applying a generating function argument. Karrer and Newman (2010) extend this model to allow for the introduction of arbitrary subgraphs, rather than limiting the model to an introduction of triangles. Note that if each individual is assigned at most one subgraph ‘corner’ and the subgraph consists of a complete graph then we recover the model of Ball et al. (2009).

In this thesis we are primarily focused on the random graph models based on the configuration model. However, there are many alternative random graph models and extensions emerging in the literature. For example, Ball and Sirl (2012) extend the model of Ball et al. (2009) to include multiple types of individuals. Another commonly studied model is the random intersection graph, introduced by Singer (1995) and Karoński et al. (1999), in which individuals belong to groups and an infected individual can infect individuals within the groups they belong to. Barabási and Albert (1999) introduce a preferential attachment random graph model, in which individuals that are added to the network are more likely to be connected with high degree individuals in the



network, and the spread of a stochastic SIS epidemic model on the Barabási-Albert random network model is studied by Berger et al. (2005).

## 1.5 The effect of heterogeneity on the spread of epidemics

With the addition of many types of heterogeneity to epidemic models there is naturally an interest in quantifying the effect of heterogeneity on the properties of the epidemic, such as the expected relative final size of a major outbreak. Newman (2003) heuristically argues that higher clustering makes it easier for epidemics to take off, and Britton et al. (2008) support this conjecture by comparing random intersection graphs with different amounts of clustering. Furthermore, Newman (2009) compares models with different clustering coefficients in the configuration model with triangles to yield the same result. However, Miller (2009) introduces a rewiring process on the configuration model with triangles to compare epidemics on clustered networks, and shows that the results found in Newman (2009) are largely caused by the change in degree correlation between models. Instead, Miller (2009) suggests that the addition of clustering actually decreases the expected relative final size of major outbreaks in the configuration model with triangles. Ball et al. (2013) introduce a network model that has tuneable clustering, degree correlation and degree distribution, achieving the tuneable clustering by generalising the rewiring process introduced by Miller (2009), and find that clustering introduced by households decreases the expected relative final size of major outbreaks. Coupechoux and Lelarge (2014), investigating the model of Trapman (2007), also find that in general clustering decreases the expected relative final size of a major outbreak. Similarly, Gleeson et al. (2010) show that clustering increases the threshold parameter for a giant connected component to occur (i.e. increases  $R_*$ ) in several models, including the model of Miller (2009) and special cases of the model of Gleeson (2009), and suggest that clustering decreases the size of the giant connected component.

Britton and Trapman (2012) investigate an analogous problem to that of maximising the expected relative final size of a major outbreak on the configuration model, and show that for a fixed mean degree the final size of a major outbreak is maximised when the network degree is homogeneous. In contrast to the monotonic results previously discussed in this section, Clancy and Pearce (2013) investigate the effect of heterogeneity on a multi-type stochastic

epidemic model (introduced by Watson (1972)) and give conditions on the heterogeneity which determine whether the expected relative final size of a major outbreak will increase or decrease when  $R_0$  is held fixed.

## 1.6 Vaccination strategies

A key application of many mathematical epidemic models is to inform health policy to reduce the impact of diseases, and a key method of reducing the impact of diseases is vaccination. Furthermore, there has been a lot of interest given to modelling the effect of vaccination strategies in the literature. One of the first such examples is the work by Neyman and Scott (1964), who show that immunisation can reduce the expected size of a stochastic discrete time epidemic using a Galton-Watson process. Expanding on this work, Becker (1972) investigates the problem of minimising the number of individuals vaccinated to prevent the spread of the epidemic (i.e. calculating the critical vaccination coverage), motivated by reducing the costs of vaccination. Effectively, Becker tries to reduce the threshold parameter to below 1 using the fewest possible vaccinations, and thus prevent a major outbreak from occurring. Indeed, in epidemic models with basic population structure the critical vaccination coverage is given by  $1 - 1/R_0$ , where  $R_0$  is the basic reproduction number. Becker (1975) refines this idea by using a branching process to approximate the early stages of an epidemic in a model including vaccination, and consider the vaccination coverage required to prevent a major outbreak. However, these early vaccination models assume that vaccinated individuals always become immune to the disease and suffer no ill effects, an often unrealistic assumption noted by Taylor (1968). One extension of epidemic models to allow for a variable reaction to vaccination is given by Becker and Starczak (1998), who introduce a generalised vaccine reaction model which allows for vaccination to result in partial immunity, or even no immunity at all.

With the introduction of heterogeneity to epidemic models there is also interest in investigating how the heterogeneity affects vaccination strategies. For example, we might be interested in calculating the optimal vaccination strategy on an epidemic model, i.e. the vaccination strategy with the smallest critical vaccination coverage. Indeed, the optimal vaccination strategies on both the configuration model and households model of Ball et al. (1997) are well-understood. As we might intuitively expect, the optimal vaccination strat-

egy on the configuration model is to vaccinate individuals with large degrees (see, for example, Dezső and Barabási (2002) or Lloyd-Smith et al. (2005)). In contrast, the optimal vaccination strategy in the model of Ball et al. (1997) is the equalisation strategy, which involves choosing individuals for vaccination to homogenize the number of susceptibles in each household. Becker and Starczak (1997) and Ball and Lyne (2002), (2006), further extend the analysis of vaccination in the households model of Ball et al. (1997), investigating the change in optimal vaccination strategy with the addition of an imperfect vaccine.

However, the optimal vaccination strategy in both the households model of Ball et al. (1997) and the configuration model require global knowledge of the network, i.e. to know which large household or high degree individuals to vaccinate we must know the household size or degree of every individual in the population. To relax this restriction, Cohen et al. (2003) propose a network vaccination strategy which works by sampling a fraction of individuals in the population and, each time an individual is sampled, vaccinating a single neighbour of that individual chosen independently and uniformly at random from its set of neighbours. Therefore, the vaccination strategy introduced by Cohen et al. (2003) tends to vaccinate larger degree individuals than the uniform vaccination strategy (the degree of an individual sampled at random in the population is on average lower than the degree of its network neighbours, see Feld (1991)) while only requiring local knowledge of the network (i.e. the neighbours of a sampled individual). Thus this acquaintance vaccination strategy requires less knowledge of the network and performs better than the uniform vaccination strategy, which involves vaccinating individuals chosen uniformly at random from the population. The acquaintance vaccination scheme introduced by Cohen et al. (2003) is further developed and put into a more rigorous framework by Britton et al. (2007). However, Ball and Sirl (2013) find that this acquaintance vaccination scheme does not admit a simple closed form expression for the vaccination coverage, and may require analysis of a branching process with infinite type space when extended to the generalised vaccine reaction of Becker and Starczak (1998), due to sibling dependence (cf. Olofsson (1996)). Thus Ball and Sirl (2013) modify this acquaintance vaccination strategy so that sampled individuals then choose each neighbour independently for vaccination with a given probability, and show that the early stages of an epidemic on the configuration network model under this modified acquaintance vaccination strategy with generalised vaccine reaction can be approximated by a 6-type

branching process which does not suffer from sibling dependence. Furthermore, Ball and Sirl (2013) show that their acquaintance vaccination performs similarly to Cohen et al.'s acquaintance vaccination strategy. Ball and Sirl (2017) further extend the acquaintance vaccination strategy introduced by Ball and Sirl (2013) to the household and network model, discussed in Ball et al. (2009), and show that their acquaintance vaccination strategy can outperform the household-based vaccination schemes if the degree distribution has heavy tails.

## 1.7 The effect of heterogeneity on vaccination strategies

In addition to investigating the effect of heterogeneity on the final size of a major outbreak, there is interest in the literature in investigating the effect of heterogeneity on the vaccination coverage required to prevent the spread of an epidemic. May and Anderson (1984) show that falsely assuming a homogeneously mixing population can lead to undervaccination if the vaccine is allocated randomly. Similarly, Becker and Utev (1998) consider a stochastic household model, parameterised by final size data and assuming a saturated household infection rate (i.e. each household neighbour of an individual is infected with probability 1), and show that assuming a homogeneously mixing population may yield an underestimate of the critical vaccination coverage. Investigating a deterministic clustered network epidemic model, House and Keeling (2011) find that fixing  $R_0$  and varying the clustering coefficient causes a negligible change in the critical vaccination coverage under the uniform vaccination strategy.

## 1.8 Research opportunities identified

Based on the literature there are many possible research opportunities. We are particularly drawn to examine how the incorporation of heterogeneity affects epidemic models, not only in the transmission of the epidemic but also in the application of vaccination strategies. This results in the following three research areas which form the basis of this thesis.

With the addition of epidemic models including households to the literature several different methods of transmitting the epidemic between households have been examined. For example, in the households model of Ball et al. (1997)

infections occur between households via casual contacts whereas in the model of Ball et al. (2009) infections occur between households via a configuration model network structure. However, there has been little analytic investigation into the difference between these two methods of transmitting the epidemic between households on the final outcome of the epidemic. In this thesis we study the effect of these two different transmission routes on the final outcome of an epidemic.

With the introduction of acquaintance vaccination strategies, e.g. Cohen et al. (2003) and Ball and Sirl (2013), there has been research into comparing their performance to previously known vaccination strategies, such as the uniform and optimal vaccination strategies (e.g. Britton et al. (2007) and Ball and Sirl (2013)). In this thesis we study the robustness of these vaccination strategies when the model contains additional routes of transmission. For example, what is the effect on the expected relative final size of a major outbreak if we apply a vaccination strategy assuming that the epidemic spreads along a random network when it is more appropriate to apply a homogeneously mixing model, and how do these vaccination strategies perform in finite populations?

The current analysis of acquaintance vaccination strategies often assumes that the network has zero clustering, however this is an unrealistic assumption in many social networks. Current literature suggests that clustering often has a large impact on the transmission of the epidemic and reduces the final size of a major outbreak. However, there is no consensus on the best way to construct a clustered network. Two common methods of constructing clustered networks are to include edge-disjoint triangles, e.g. the model of Newman (2009), and to include households, e.g. the model of Ball et al. (2009). In this thesis we investigate the effect of these two types of clustering on the performance of vaccination strategies, especially the vaccination strategy of Ball and Sirl (2013).

## 1.9 Layout of thesis

In Chapter 2 we define the clustering coefficient and degree correlation of a network, discuss the final size of a major outbreak in closed finite populations, give background information on branching processes and introduce some relevant notation. In Chapter 3 we formally introduce an epidemic model with a household and network structure which includes casual contacts, which can be considered as an amalgamation of the models introduced by Ball and Neal

(2008) and Ball et al. (2009). We prove a limit theorem for approximating the early stages of the epidemic with a branching process, calculate the threshold parameters  $R_*$  and  $R_0$  and finally discuss the expected relative final size of a major outbreak by relating the total number of individuals infected in an epidemic to the total progeny of a branching process.

We split Chapter 4 into two segments, firstly using the model of Ball and Neal (2008) to discuss the effect of network heterogeneity on the final size of a major outbreak when  $R_0$  is fixed, and then using the model of Ball et al. (1997) to discuss the effect of household heterogeneity on the final size of a major outbreak when  $R_0$  is fixed. We show that casual contacts and contacts made via a network structure can have different effects on the final outcome of the epidemic by proving conditions which determine whether introducing network heterogeneity to the homogeneously mixing model will increase or decrease the expected relative final size of a major outbreak. Furthermore, we prove that the effect of introducing a small amount of network heterogeneity to the homogeneously mixing model is not necessarily the same as the effect of introducing more network heterogeneity to an already heterogeneous model. We also prove conditions which determine whether introducing household heterogeneity to the homogeneously mixing model will increase or decrease the expected relative final size of a major outbreak.

In Chapter 5 we investigate the effect of casual contacts on the performance of the uniform, optimal and acquaintance vaccination strategies on the configuration network model. Note that we only consider the acquaintance vaccination strategy introduced by Ball and Sirl (2013). Importantly, we prove that if the degree distribution of the network has a small variance then the acquaintance vaccination strategy can underperform compared to the uniform vaccination strategy. Furthermore, we show that the asymptotic expected relative final size of a major outbreak under the acquaintance vaccination strategy is often a lower bound on the final size of a major outbreak in finite populations. Our numerical investigations find that the addition of global contacts while fixing  $R_0$  reduces the performance of the acquaintance and optimal vaccination strategies and increases the critical vaccination coverage.

In Chapter 6 we extend the model of Newman (2009) to include a rewiring process and explore the effect of introducing clustering via edge-disjoint trian-

gles on the performance of the uniform, acquaintance and optimal vaccination strategies. Our numerical investigations show that fixing  $R_0$  and changing the clustering in the network via the rewiring process generally causes a negligible change in the critical vaccination coverage of the uniform, acquaintance and optimal vaccination strategies. However, if the total degree distribution of the network has a small variance then increasing the edge-triangle clustering can increase the critical vaccination coverage of the acquaintance vaccination strategy. In contrast, fixing the expected relative final size of a major outbreak and increasing the clustering in the network can decrease the critical vaccination coverages of the vaccination strategies.

In Chapter 7 we modify the model of Ball et al. (2013), by including a general rewiring process, and explore the effect of introducing clustering via households on the performance of the uniform and acquaintance vaccination strategies. We prove that rewiring large households can drastically affect the performance of the epidemic on the network, for example requiring the uniform vaccination strategy to vaccinate every individual in the population to prevent a major outbreak. Furthermore, our numerical investigations find that fixing the expected relative final size of a major outbreak and increasing the clustering in the network via the rewiring process will generally increase the critical vaccination coverages of the uniform and acquaintance vaccination strategies. Finally, in Chapter 8 we give our concluding remarks and comment on questions arising from this research.

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## 2. Background theory

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In this chapter we introduce some of the definitions and preliminary work required for this thesis. In Section 2.1 we introduce some of the notation used throughout this thesis. We consider the spread of the epidemic within a single household in Section 2.2. Finally, in Section 2.3 we give a brief overview of some common results in branching process theory.

### 2.1 Notation

Firstly, we define  $\mathbb{R}$  and  $\mathbb{Z}$  to be the set of real and integer numbers respectively. Similarly, we define  $\mathbb{R}^+$  and  $\mathbb{Z}^+$  to be the set of positive real and integer numbers respectively. Let  $\mathbf{1}_A$  be the indicator function for the event  $A$  and for  $x \in \mathbb{R}$  let  $\lfloor x \rfloor = \max \{y \in \mathbb{Z} : y \leq x\}$ . For  $x \in \mathbb{R}$ , denote by  $\text{sgn}(x)$  the signum function of  $x$ , so

$$\text{sgn}(x) = \begin{cases} -1 & \text{if } x < 0, \\ 0 & \text{if } x = 0, \\ 1 & \text{if } x > 0. \end{cases} \quad (2.1)$$

For a set  $Y$  we say that  $|Y|$  is the cardinality of  $Y$ . For  $n \in \mathbb{Z}^+ \cup \{0\}$  let  $[0; n] = \{0, 1, \dots, n\}$ . For a vector  $\mathbf{x}$ , we say that  $|\mathbf{x}|$  is the  $\ell^2$ -norm of  $\mathbf{x}$ , so  $|(x_1, x_2)| = \sqrt{x_1^2 + x_2^2}$ . Let  $\delta_{x,y}$  be the Kronecker delta function, so

$$\delta_{i,j} = \begin{cases} 0 & \text{if } i \neq j, \\ 1 & \text{if } i = j. \end{cases} \quad (2.2)$$

For suitable vectors  $\mathbf{x} = (x_1, x_2, \dots, x_l)$  and  $\mathbf{y} = (y_1, y_2, \dots, y_l)$  we say that  $\mathbf{x} \leq \mathbf{y}$  if the inequality holds componentwise, and that  $\mathbf{x} < \mathbf{y}$  if all componentwise inequalities are strict. We say that  $\mathbf{x}^{\mathbf{y}} = (x_1^{y_1}, x_2^{y_2}, \dots, x_l^{y_l})$ . We write  $\infty$



and  $\mathbf{0}$  for vectors of appropriate dimensions with all entries  $\infty$  and  $0$  respectively. Similarly, for suitable matrices  $A$  and  $B$  we say that  $A \leq B$  if the inequality holds componentwise, and that  $A < B$  if all componentwise inequalities are strict.

For a random variable  $X$ , we denote its mean by  $\mu_X$  and its variance by  $\sigma_X^2$ . Unless otherwise specified, we denote its probability generating function by  $f_X(s) = \mathbb{E}[s^X]$  or  $b_X(s) = \mathbb{E}[s^X]$ ,  $s \in [0, 1]$ , with the choice of  $f_X(s)$  or  $b_X(s)$  generally determined by whether we are calculating the spread of an epidemic or susceptibility set (see Section 3.3). For a function  $f(x)$ , we write  $f'(x)$  for the derivative of  $f(x)$  with respect to  $x$ . For a random variable  $X$ , with  $\mathbb{P}(X = k) = p_k$ ,  $k = 0, 1, 2, \dots$ , and  $\mu_X < \infty$ , we define the size-biased distribution  $\tilde{X}$ , such that  $\mathbb{P}(\tilde{X} = k) = \tilde{p}_k = kp_k/\mu_X$ . It follows from this definition of  $\tilde{X}$  that  $\mu_{\tilde{X}} = \mu_X + \sigma_X^2/\mu_X$  and  $f_{\tilde{X}-1}(s) = f'_X(s)/\mu_X$ .

For two random variables  $X$  and  $Y$  we write  $X \stackrel{\mathcal{D}}{=} Y$  if  $X$  and  $Y$  are equal in distribution, i.e.  $\mathbb{P}(X \leq x) = \mathbb{P}(Y \leq x)$  for all  $x$ . Similarly, for a sequence of real-valued random variables  $X_1, X_2, \dots$  and a random variable  $X$  we write  $X_n \xrightarrow{\mathcal{D}} X$  if  $X_1, X_2, \dots$  converge in distribution to  $X$ , i.e. the cumulative density function of  $X_n$  at  $x$  converges to the cumulative density function of  $X$  at  $x$  as  $n \rightarrow \infty$  for every  $x \in \mathbb{R}$  at which the cumulative distribution function of  $X$  is continuous. We give notation for the majority of random variables used throughout this thesis in Table 2.1, and applying the definition of the size-biased distribution to a selection of these random variables yields the probability generating functions for their size-biased distributions given in Table 2.2. Note that for a non-negative integer  $n$ ,  $\text{Const}(n) \stackrel{\mathcal{D}}{=} \text{Bin}(n, 1)$ . The random variable  $\text{Pow}(k^*, \alpha)$  is investigated in Ball et al. (2010), and is an example of a distribution with a power law tail. We use this random variable to numerically investigate the effect of distributions with heavy tails on our models.

## 2.2 The final size of a household epidemic

We begin this section by defining an SIR epidemic in a homogeneously mixing closed finite population. In an SIR epidemic every individual in the population at any given time is either susceptible, infected or removed. An infected individual is infectious for the length of its infectious period, and each individual in the population has an infectious period sampled independently from a non-negative random variable  $I$ , with mean  $\mu_I < \infty$ , which we specify by its Laplace-Stieltjes

Notation	Parameters	Support	Probability mass function
Const( $d$ )	$d \in \mathbb{Z}^+$	$k = d$	1
Bin( $n, p$ )	$n \in \mathbb{Z}^+, p \in [0, 1]$	$k \in \{0, 1, \dots, n\}$	$\binom{n}{k} p^k (1-p)^{n-k}$
Poi( $\alpha$ )	$\alpha \in \mathbb{R}^+$	$k \in \mathbb{Z}^+ \cup \{0\}$	$\frac{\alpha^k e^{-\alpha}}{k!}$
Poi <sup>+</sup> ( $\alpha$ )	$\alpha \in \mathbb{R}^+$	$k \in \mathbb{Z}^+$	$\frac{\alpha^k}{(e^\alpha - 1)k!}$
Geo( $p$ )	$p \in (0, 1)$	$k \in \mathbb{Z}^+ \cup \{0\}$	$(1-p)^k p$
Geo <sup>+</sup> ( $p$ )	$p \in (0, 1)$	$k \in \mathbb{Z}^+$	$(1-p)^{k-1} p$
NB( $r, p$ )	$r \in \mathbb{Z}^+, p \in (0, 1)$	$k \in \mathbb{Z}^+ \cup \{0\}$	$\binom{k+r-1}{k} (1-p)^r p^k$
Log( $p$ )	$p \in (0, 1)$	$k \in \mathbb{Z}^+$	$\frac{-p^k}{k \log(1-p)}$
Pow( $k^*, \alpha$ )	$k^* \in \mathbb{Z}^+, \alpha \in \mathbb{R}^+$	$k \in \mathbb{Z}^+$	$\begin{cases} k^{*- \alpha} & \text{if } k < k^*, \\ k^{-\alpha} & \text{if } k \geq k^* \end{cases}$

Table 2.1: Table of notation for random variables.

Random variable $X$	$\mu_X$	$f_X(s)$	$f_{\tilde{X}-1}(s)$
$X \sim \text{Const}(d)$	$d$	$s^d$	$s^{d-1}$
$X \sim \text{Bin}(n, p)$	$np$	$(1-p+ps)^n$	$(1-p+ps)^{n-1}$
$X \sim \text{Poi}(\alpha)$	$\alpha$	$e^{-\alpha(1-s)}$	$e^{-\alpha(1-s)}$
$X \sim \text{NB}(r, p)$	$\frac{pr}{1-p}$	$\left(\frac{1-p}{1-ps}\right)^r$	$\left(\frac{1-p}{1-ps}\right)^{r+1}$
$X \sim \text{Log}(p)$	$\frac{-p}{(1-p)\log(1-p)}$	$\frac{\log(1-ps)}{\log(1-p)}$	$\frac{1-p}{1-ps}$

Table 2.2: The probability generating functions of a selection of random variables  $X$  and  $\tilde{X} - 1$ .

transform  $\phi_I(\theta) = \mathbb{E} [e^{-\theta I}]$  ( $\theta \geq 0$ ). Throughout an individual's infectious period it makes contact with any other given individual in the population at the points of a Poisson process with rate  $\lambda_H$  (i.e. for each infective and individual in the population we consider a Poisson process on the real line, corresponding to time, for which the points correspond to times at which infectious contacts occur from the infective to the individual), before it becomes removed and plays no further part in the epidemic. A susceptible individual becomes infected when it is contacted by an infected individual. The epidemic terminates when there are no infectives remaining in the population. All infectious periods and Poisson processes are assumed to be mutually independent.

Gontcharoff polynomials can be used to express the final size distribution of an SIR epidemic in a homogeneously mixing closed finite population. They often appear in calculations throughout this thesis because we often consider the spread of the epidemic through a single household of size  $n$ , ignoring network and global contacts, which can be considered as a homogeneously mixing model with a single infective,  $n - 1$  susceptibles and infection rate  $\lambda_H$ . Given a parameter sequence of real numbers  $U = (u_i, i = 0, 1, \dots)$ , the Gontcharoff polynomials  $G_k(x|U)$  (introduced by Gontcharoff (1937)) are defined by  $G_0(x|U) = 1$  and the recurrence

$$G_k(x|U) = \frac{x^k}{k!} - \sum_{j=0}^{k-1} \frac{u_j^{k-j}}{(k-j)!} G_j(x|U), \quad k = 1, 2, \dots \quad (2.3)$$

Consider the spread of the epidemic through a homogeneously mixing population containing a single initial infective and  $n - 1$  susceptibles, i.e. a household of size  $n$ . We say that the final size of a household epidemic is the total number of removed individuals in the household when there are no infectives remaining in the household (excluding the initial infective). Let  $T^{(n)}$  be the final size of the household epidemic amongst the initially susceptible individuals within the household of size  $n$ , so  $T^{(n)} + 1$  is the total number of removed individuals in the household when there are no infectives remaining in the household. To calculate the probability generating function of  $T^{(n)}$ ,  $f_{T^{(n)}}(s)$ , we restrict ourselves to the case of a constant infectious period (we discuss the reasons behind this restriction in Section 3.2.5). So, assuming a constant infectious period of length 1, from Corollary 3.3 of Lefèvre and Picard (1990) (or Ball (1986), Theorem

2.6) the probability generating function for  $T^{(n)}$ , is given by

$$f_{T^{(n)}}(s) = \sum_{k=0}^{n-1} \frac{(n-1)!}{(n-1-k)!} q_{k+1}^{n-1-k} G_k(1|V) s^k, \quad n = 1, 2, \dots, \quad (2.4)$$

where  $q_k = \phi_I(k\lambda_H)$  and  $V = (q_{k+1}, i = 0, 1, \dots)$ . Furthermore,

$$\mu_{T^{(n)}} = n - 1 - \sum_{k=1}^{n-1} \frac{(n-1)!}{(n-1-k)!} q_k^{n-k} G_{k-1}(1|V). \quad (2.5)$$

Let  $\tilde{H}$  be a distribution with support in the non-negative integers and, for  $n = 1, 2, \dots$ , let  $P(\tilde{H} = n) = \tilde{\rho}_n$ . Then let

$$\mu_T = \sum_{n=1}^{\infty} \tilde{\rho}_n \mu_{T^{(n)}}, \quad (2.6)$$

be the mean final size, excluding the initial infective, of a household epidemic in which the household size is sampled according to  $\tilde{H}$ .

## 2.3 Branching process theory

We now give a brief overview of common results in discrete time multi-type branching process theory. This section is an amalgamation of Karlin and Taylor (1975) (Section 8.6), Mode (1971) (Chapters 1 and 2) and Feller (1968) (Chapter 12), and covers the key results related to branching processes required for this thesis. We first define and describe the process and then define the probability generating functions describing the offspring of the various types of individual in the branching process. We then give the probability of extinction of the branching process and finally we define the total progeny of the branching process and give an implicit formula determining the probability generating function of the total progeny of a single-type branching process.

Consider a population containing  $p$  types of individuals. Individuals of each type will produce offspring of possibly any of the  $p$  types, and the offspring of a given individual is independent of the offspring of any other individual in the population. Let  $Z_i^{(m)}$ ,  $i = 1, 2, \dots, p$ ,  $m = 0, 1, 2, \dots$ , be the number of individuals of type  $i$  in the  $m$ th generation of the branching process. Then, for

$m = 0, 1, \dots$  and  $i = 1, 2, \dots, p$ ,

$$Z_i^{(m+1)} = \sum_{j=1}^p \sum_{k=1}^{Z_j^{(m)}} \zeta_k^{ji},$$

where, for  $j = 1, 2, \dots, p$ ,  $k = 1, 2, \dots, Z_j^{(m)}$ ,  $(\zeta_k^{j1}, \zeta_k^{j2}, \dots, \zeta_k^{jp})$  are independent and identically distributed random vectors with distribution

$$P(\zeta_k^{j1} = l_1, \zeta_k^{j2} = l_2, \dots, \zeta_k^{jp} = l_p) = p_j(l_1, l_2, \dots, l_p), \quad l_1, l_2, \dots, l_p = 0, 1, 2, \dots$$

So  $p_j(l_1, l_2, \dots, l_p)$  is the probability that a single individual of type  $j$  produces  $l_i$  offspring of type  $i$ ,  $i = 1, 2, \dots, p$ . Let  $\mathbf{s} = (s_1, s_2, \dots, s_p) \in [0, 1]^p$  and, for  $j = 1, 2, \dots, p$ , let

$$f_j(s_1, s_2, \dots, s_p) = \sum_{i=1}^p \sum_{l_i=0}^{\infty} p_j(l_1, l_2, \dots, l_p) s_1^{l_1} s_2^{l_2} \dots s_p^{l_p},$$

be the corresponding probability generating functions and we define the vector of probability generating functions  $\mathbf{f}(\mathbf{s}) = (f_1(\mathbf{s}), f_2(\mathbf{s}), \dots, f_p(\mathbf{s}))$ .

Let  $\mathbf{e}_i$  denote the vector with 1 in the  $i$ th component and zero otherwise. Then we say that a multi-type branching process is irreducible if and only if, for every pair of types  $i, j$ , there exists some natural number  $m$  such that

$$P(Z_j^{(m)} \geq 1 \mid \mathbf{Z}^{(0)} = \mathbf{e}_i) > 0, \quad (2.7)$$

where  $\mathbf{Z}^{(0)} = (Z_1^{(0)}, Z_2^{(0)}, \dots, Z_p^{(0)})$ . Furthermore, we say that the branching process is positively regular if for some  $m$  equation (2.7) holds for all  $i$  and  $j$ . We say that a multi-type branching process is singular if each individual has exactly one offspring, and otherwise the branching process is non-singular.

We say that the branching process becomes extinct if there exists some natural number  $N$  for which if  $m > N$  then  $\sum_{i=1}^p Z_i^{(m)} = 0$ , i.e. in generation  $m$  there are no individuals of any type. To determine whether a branching process can avoid extinction, we consider the next-generation matrix  $M$ , where  $m_{ij} = \frac{\partial f_i(1, 1, \dots, 1)}{\partial s_j}$ . So  $m_{ij}$  is the expected number of type- $j$  offspring of a single type- $i$  individual. Let  $R$  be the largest eigenvalue in absolute value of  $M$ . Then if  $R < 1$  then the branching process almost surely becomes extinct and we say the branching process is subcritical, if  $R = 1$  then the branching process

almost surely becomes extinct and we say the branching process is critical and if  $R > 1$  then the branching process has a strictly positive probability of not becoming extinct and we say the branching process is supercritical (see, for example, Karlin and Taylor (1975, Section 8.6)).

We now consider the probability generating function for the number of individuals in the  $m$ th generation of the branching process and the probability of extinction of the branching process. For  $i = 1, 2, \dots, p$ ,  $m = 1, 2, \dots$ , let

$$f_i^{(m)}(\mathbf{s}) = \sum_{i=1}^p \sum_{l_i=0}^{\infty} \mathbf{P} \left( Z_1^{(m)} = l_1, Z_2^{(m)} = l_2, \dots, Z_p^{(m)} = l_p \mid \mathbf{Z}^{(0)} = \mathbf{e}_i \right) s_1^{l_1} s_2^{l_2} \dots s_p^{l_p},$$

be the probability generating function of the state of the process in the  $m$ th generation of the branching process, conditioned on the initial generation containing a single individual of type  $i$ . Note that, for  $i = 1, 2, \dots, p$ ,  $f_i^{(1)}(\mathbf{s}) = f_i(\mathbf{s})$  and, since the offspring of any individual is independent of the offspring of any other individual,

$$f_i^{(n+m)}(\mathbf{s}) = f_i^{(m)} \left( f_1^{(n)}(\mathbf{s}), f_2^{(n)}(\mathbf{s}), \dots, f_p^{(n)}(\mathbf{s}) \right). \quad (2.8)$$

Before giving the probability of extinction of the branching process we define the smallest and largest solutions to a set of simultaneous equations. We say that  $\boldsymbol{\pi} = (\pi_1, \pi_2, \dots, \pi_p)$  is the smallest non-negative solution of the set of simultaneous equations  $\boldsymbol{\pi} = \mathbf{g}(\boldsymbol{\pi})$  (with  $\mathbf{g}(\boldsymbol{\pi}) = (g_1(\boldsymbol{\pi}), g_2(\boldsymbol{\pi}), \dots, g_p(\boldsymbol{\pi}))$ ) when  $\boldsymbol{\pi}$  is such that, for any other nonnegative solution  $\boldsymbol{\pi}^*$  of the equation  $\mathbf{s} = \mathbf{g}(\mathbf{s})$ , we have that  $\boldsymbol{\pi} < \boldsymbol{\pi}^*$ . Similarly, we say that  $\mathbf{z}$  is the largest solution of the set of simultaneous equations  $\mathbf{z} = \mathbf{g}(\mathbf{z})$ , when  $\mathbf{z}$  is such that, for any other nonnegative solution  $\mathbf{z}^*$  of the equation  $\mathbf{s} = \mathbf{g}(\mathbf{s})$ , we have that  $\mathbf{z} < \mathbf{z}^*$ .

Let  $\boldsymbol{\pi}$  be the extinction probabilities for this Galton-Watson branching process, i.e.  $\pi_i$  is the probability of eventual extinction of the branching process initiated by a single type- $i$  individual. Then  $\boldsymbol{\pi}$  is the smallest solution of the set of simultaneous equations  $\mathbf{s} = \mathbf{f}(\mathbf{s})$ , and this smallest solution is guaranteed to exist. Furthermore, for  $i = 1, 2, \dots, p$  and any  $\mathbf{s} \in [0, 1]^p$ ,  $\mathbf{s} \neq (1, 1, \dots, 1)$ ,

$$\lim_{m \rightarrow \infty} f_i^{(m)}(\mathbf{s}) = \pi_i. \quad (2.9)$$

We say that the total progeny of the branching process,  $\hat{\mathbf{Z}} = (\hat{Z}_1, \hat{Z}_2, \dots, \hat{Z}_p)$ , is the total number of individuals of each type it contains over its lifetime, or

$\infty$  if the branching process does not become extinct. So

$$\hat{\mathbf{Z}} = \sum_{m=0}^{\infty} \left( Z_1^{(m)}, Z_2^{(m)}, \dots, Z_p^{(m)} \right).$$

Finally, consider a single-type branching process (i.e.  $p = 1$ ) and let  $\hat{f}_Z(s)$  be the probability generating function of the total progeny of the branching process  $\hat{Z}_1$ . Then  $\hat{f}_Z(s)$  is the unique positive solution in  $[0, 1]$  of the implicit equation

$$\hat{f}_Z(s) = s f_1 \left( \hat{f}_Z(s) \right).$$

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### 3. A model of a stochastic SIR epidemic with three levels of mixing

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In this chapter we consider an SIR (Susceptible - Infective - Removed) epidemic model for the spread of an epidemic among a population of individuals, with a random network of social contacts, that is partitioned into households and in which individuals also make casual contacts, i.e. with individuals chosen uniformly at random from the population. This is an extension of previous models such as Ball et al. (1997), the standard households model; Ball and Neal (2008), who consider the spread of an epidemic on a network structure with casual contacts; and Ball et al. (2009), who consider the spread of an epidemic on a network with additional household structure.

We approximate the early stages of the epidemic with a household-based Galton-Watson branching process and prove that as the number of households in the population tends to infinity the total number of individuals infected in the epidemic process converges in distribution to the total progeny of a branching process. Furthermore, we give a limiting theorem that allows us to define a major outbreak in a finite population.

This branching process is then used to calculate a household-based threshold parameter,  $R_*$ , that determines whether a major outbreak can occur or not, and the probability of a major outbreak. We calculate a second threshold parameter, an individual-based reproduction number  $R_0$ , following the definition introduced by Pellis et al. (2012), using a individual-based branching process, and the expected relative final size of a major outbreak using a backward Galton-Watson branching process and the concept of susceptibility sets similarly to Ball and Neal (2002). We investigate the convergence of the probability of a major



outbreak and the final size of a major outbreak in finite populations to their asymptotic limits given by the branching process approximation and then consider the effect on the final size of a major outbreak of changing the infection rates while keeping  $R_0$  constant.

This chapter is organised as follows. We formally introduce the epidemic model in Section 3.1. Approximation of the early stages of an epidemic with a Galton-Watson branching process is described in Section 3.2. We give a heuristic description of the approximating branching process and limit theorems in Section 3.2.1. We discuss threshold parameters  $R_*$  and  $R_0$  in Sections 3.2.2 and 3.2.3 respectively, along with an ordering of  $R_0$  and  $R_*$  in Section 3.2.4. Section 3.2 concludes by considering the probability of a major outbreak with a constant infectious period in Section 3.2.5. Section 3.3 discusses the final size of a major outbreak. In Section 3.4.1 we numerically explore the model, including the accuracy of our asymptotic results in finite populations in Section 3.4.1 and numerical investigations of the model in Section 3.4.2. Finally, we give details of the Galton-Watson branching process described in Section 3.2.1 and proofs of the limit theorem, before giving concluding remarks in Section 3.6 and tables of common notation introduced in this chapter in Section 3.7.

## 3.1 Model

We study a model consisting of a finite, closed population of  $N$  individuals split into  $m$  households, of which  $m_n$  are of size  $n$  (for  $n = 1, 2, \dots$ ). We construct the network structure according to the ‘configuration model’ (see, for example, Newman (2002a)). This means that we assign each individual in the population a number of ‘half-edges’ according to independent samples from an arbitrary but specified distribution  $D$  with  $P(D = k) = p_k$  ( $k = 0, 1, \dots$ ). Conditional on the total number of half-edges being even, these half-edges are then paired uniformly at random to form the edges of the network. We say that an individual’s degree is equal to the number of half-edges it has been assigned. So an individual chosen uniformly at random from the population has degree distributed according to  $D$ .

Next we consider the evolution of the epidemic. We consider an SIR epidemic which means that every individual in the population at any given time is either susceptible, infected or removed. A susceptible individual becomes infected when

it is contacted by an infected individual. An infected individual is infectious for the length of its infectious period during which it makes infectious contacts with other individuals, before it becomes removed and plays no further part in the epidemic. The epidemic terminates when there is no infective remaining in the population.

The epidemic starts with a single infectious individual chosen uniformly at random from the population which is otherwise susceptible. The infectious period of each individual is independently sampled from a non-negative random variable  $I$ , with mean  $\mu_I < \infty$ , which we specify by its Laplace-Stieltjes transform  $\phi_I(\theta) = \mathbb{E}[e^{-\theta I}]$  ( $\theta \geq 0$ ).

Throughout an individual's infectious period it can make infectious contact with other individuals in three ways.

- An infected individual makes contact with any given household neighbour at the points of a Poisson process with rate  $\lambda_H$ .
- An infected individual makes contact with any given network neighbour at the points of a Poisson process with rate  $\lambda_N$ .
- An infected individual makes global infectious contact with individuals chosen uniformly at random from the  $N$  members of the population at the points of a Poisson process with rate  $\lambda_G$ .

Note that  $\lambda_H$  and  $\lambda_N$  are per-pair rates but the per-pair rate for global infectious contact is  $\lambda_G/N$ . So an infectious individual of degree  $d$  in a household of size  $n$  makes infectious contacts at total rate  $d\lambda_N + (n-1)\lambda_H + \lambda_G$ . All infectious periods, network degrees, Poisson processes and uniform samplings for global contacts are assumed to be mutually independent, and these are independent of an individual's household size. We denote the marginal probability that an infected individual infects a given susceptible network and household neighbour by  $p_N = 1 - \phi_I(\lambda_N)$  and  $p_H = 1 - \phi_I(\lambda_H)$  respectively.

It is important to note that our results are asymptotic as the number of households  $m \rightarrow \infty$ . Although we assume that the epidemic starts with a single initial infective chosen uniformly at random from the population, our results are easily modified for several initial infectives, given that the number of initial infectives remains finite as  $m \rightarrow \infty$ . We require that  $D$  has finite

variance and that, as  $m \rightarrow \infty$ ,

$$m_n/m \rightarrow \rho_n \quad (n = 1, 2, \dots),$$

where  $(\rho_1, \rho_2, \dots)$  is a proper probability distribution. We set  $H$  to be the asymptotic household size distribution given by  $P(H = n) = \rho_n$ ,  $n = 1, 2, \dots$ , and assume that  $H$  has finite variance.

The requirement that  $D$  has finite variance ensures that any multiple edges and self-loops amongst individuals becomes sparse in the network as  $m \rightarrow \infty$ , i.e. the total number of multiple edges and self-loops per individual tends to 0 as  $m \rightarrow \infty$  (see Durrett (2006), Theorem 3.1.2). We say that a multiple edge between households occurs when two households have multiple network edges between individuals in them and a household self-loop occurs when two individuals in the same household have a network edge connecting them. Then the condition that  $H$  has a finite variance ensures that multiple edges between households and household self-loops also become sparse as  $m \rightarrow \infty$  (see Ball and Sirl (2012), Section 6.1).

Note that we can recover several other epidemic models discussed in previous literature. Setting  $\lambda_H = 0$  or  $\rho_1 = 1$  effectively removes the households, and recovers the model of Ball and Neal (2008). Setting  $\lambda_G = 0$  removes the global infection and recovers the model of Ball et al. (2009). If  $\lambda_N = 0$  or  $p_0 = 1$  we effectively remove the network structure and recover the household model of Ball et al. (1997). There are several ways of recovering the usual homogeneously mixing stochastic SIR epidemic model by removing the household and network structure, i.e. setting  $\lambda_H = \lambda_N = 0$ , or  $\rho_1 = 1$  and  $p_0 = 1$ . Setting  $\lambda_H = \lambda_G = 0$ , or  $\rho_1 = 1$  and  $\lambda_G = 0$  effectively removes the household structure and global infections, so recovers the network SIR model, Newman (2002a).

## 3.2 Early stages of an epidemic

### 3.2.1 Heuristic description of the approximating branching process

We begin this section by giving an informal description of the discrete time two-type Galton-Watson branching process used to approximate the early stages of the epidemic. We are interested in the final outcome of the epidemic and not its precise evolution, so we can think of the process evolving in the following way (see, for example, Ludwig (1975) or Pellis et al. (2008)). First consider the epidemic spreading only within the household containing the initial infective, ignoring global and network contacts, which we call the household epidemic. Next we consider the number of network and global contacts made by each member of the household epidemic. In the early stages of the epidemic it is likely that each of these network and global contacts will be made with uninfected individuals that are in distinct uninfected households, thus each network or global contact made will result in a single infected household with a single initial infective.

We let each newly infected household proceed in the same manner, considering the spread of the epidemic through the household first and then considering the number of global and network contacts made by the members of the household epidemic. This can then be viewed as a two-type branching process of infected households. ‘Individuals’ in this branching process correspond to single-household epidemics with one initial infective and their type is determined by whether the initial infective is globally contacted (type-1) or contacted via the network (type-2). We call the initial infective in a household the primary infective or case and any subsequent infected individuals in the household secondary infectives. We assume that the epidemic process is started by an individual chosen uniformly at random from the population, so the branching process has a single type-1 ancestor.

Further to the moment conditions on the household size and degree distributions, i.e.  $\sigma_H^2 < \infty$  and  $\sigma_D^2 < \infty$ , we assume that the branching process is irreducible, positively regular and nonsingular. Note that the irreducible and positively regular conditions are satisfied when  $\lambda_G > 0$ ,  $\lambda_N > 0$  and  $p_0 < 1$ . Relaxing any of these conditions recovers either the model of Ball et al.

(1997) or Ball et al. (2009). The non-singular condition rules out the possibility that each individual in the branching process gives rise to precisely one offspring.

Our main result concerning this branching process approximation is an approximation theorem for the total number of type-1 and type-2 infected households in the epidemic, whose proof is given in Section 3.5. For an epidemic on  $m$  households,  $\mathbf{E}^{(m)}$  say, let  $\hat{\mathbf{E}}^{(m)}$  be the total number of (type-1, type-2) infected households infected, including the initial infective. Let  $\mathbf{Z}$  be the branching process described above, with total progeny  $\hat{\mathbf{Z}}$ .

**Theorem 3.1.**

(i) For  $k, l = 0, 1, \dots$ ,

$$\lim_{m \rightarrow \infty} \left| \mathbb{P} \left( \hat{\mathbf{E}}^{(m)} = (k, l) \right) - \mathbb{P} \left( \hat{\mathbf{Z}} = (k, l) \right) \right| = 0. \quad (3.1)$$

(ii) For  $\gamma \in (0, 1/2)$ ,

$$\lim_{m \rightarrow \infty} \left| \mathbb{P} \left( \hat{\mathbf{E}}^{(m)} < (\lfloor m^\gamma \rfloor, \lfloor m^\gamma \rfloor) \right) - \mathbb{P} \left( \hat{\mathbf{Z}} < \infty \right) \right| = 0. \quad (3.2)$$

Theorem 3.1 suggests that, in the limit  $m \rightarrow \infty$ , we say that a major outbreak occurs if the limiting branching process  $\mathbf{Z}$  does not become extinct. Therefore, whether a major outbreak can occur is determined by whether or not the limiting branching process  $\mathbf{Z}$  is supercritical (i.e. whether  $R_* > 1$ , see Section 3.2.2). If  $R_* > 1$ , we can then calculate the probability of a major outbreak (see section 3.2.5), and the expected relative final size of a major outbreak (see Section 3.3). Furthermore, Theorem 3.1 part (ii) naturally leads to a definition of a major outbreak in a finite population, i.e. that, for fixed  $\gamma \in (0, 1/2)$  and a finite population, a major outbreak occurs if it contains at least  $m^\gamma$  type-1 or type-2 individuals.

Our definition of a major outbreak in a finite population is similar to Ball et al. (2009), who define a major outbreak as one which infects at least  $\log(m)$  households. Furthermore, Ball et al. show that this is almost surely the same as the stronger definition of requiring at least  $\log(m)$  infected households in generation  $\lfloor 2 \log \log m / \log R_* \rfloor$ , and that the infected households in generation  $1 + \lfloor 2 \log \log m / \log R_* \rfloor$  contains less than  $(\log(m))^\beta$  half-edges, for some  $\beta \in (1, \infty)$ .

It is worth noting that there are many limit theorems for epidemic processes and branching processes in the literature. For example, Ball and Neal (2002) couple a branching process to an epidemic with local and global structure, e.g. the households model of Ball et al. (1997), and prove that, as the number of individuals in the population tends to infinity, the total number of individuals infected by the epidemic converges in distribution to the total progeny of their approximating branching process. Furthermore, Ball and Neal show that with probability 1 the relative final size of a major outbreak tends to either 0 or a positive constant as the population size tends to infinity. Ball and Neal (2008) give a central limit theorem for the final size of a major outbreak in the network and global model when the degree distribution is constant. We hypothesise that similar results hold for our epidemic model, however the details are difficult and not the focus of this thesis. We note that the proofs of our limit theorems use similar methods to the proofs given by Ball and Neal (2002), Ball and Neal (2008) and Ball et al. (2009).

We refer to  $\mathbf{Z}$  as the forward branching process as it is constructed by considering who an individual will infect forward in time. This is in contrast to the backward Galton-Watson branching process, discussed in Section 3.3, which in some sense is constructed by considering the backwards evolution of the epidemic.

### 3.2.2 Threshold parameter $R_*$

Before giving  $R_*$  we introduce some notation. For  $i, j \in \{1, 2\}$ , let  $m_{ij}$  be the mean number of type- $j$  offspring made by a typical type- $i$  individual in the forward Galton-Watson branching process. So  $m_{11}$  is the mean number of globally contacted individuals infected by the members of a single-household epidemic in which the primary individual was globally contacted. Let  $M$  be the mean next-generation matrix, given by

$$M = \begin{bmatrix} m_{11} & m_{12} \\ m_{21} & m_{22} \end{bmatrix}. \quad (3.3)$$

Let  $R_*$  be the largest eigenvalue of the next-generation matrix  $M$ . So

$$R_* = \frac{m_{11} + m_{22} + \sqrt{(m_{11} - m_{22})^2 + 4m_{12}m_{21}}}{2}. \quad (3.4)$$

Recall that, in the limit  $m \rightarrow \infty$ , a major outbreak occurs in the asymptotic population if the total progeny of the two-type Galton-Watson forward branching process is infinite, so whether the epidemic can become a major outbreak is determined by whether or not the Galton-Watson forward branching process is supercritical. Standard multi-type branching process theory says that an irreducible two-type branching process is supercritical if and only if the largest eigenvalue of the next-generation matrix  $M$  is greater than one (see Section 2.3). If  $R_*$  is less than 1 then the Galton-Watson forward branching process is subcritical and will almost surely become extinct, therefore the probability that a major outbreak occurs is zero. Similarly, if  $R_* = 1$ , then the Galton-Watson forward branching process is critical, and will almost surely become extinct. Thus  $R_*$  is a threshold parameter for whether or not a major outbreak can occur.

If we remove either the global infections, e.g. substituting  $\lambda_G = 0$ , or the network spread of the epidemic, e.g. substituting  $\lambda_N = 0$ , then  $R_*$  is the expected number of infectious contacts made by a single household epidemic. However, if  $\lambda_G > 0$  and  $\lambda_N > 0$  then an intuitive description for  $R_*$  is the expected total number of contacts made by a typical household, by which we mean a weighted average of the offspring of type-1 and type-2 household epidemics. The weighting is given by the proportion of type-1 and type-2 individuals throughout the lifetime of the branching process.

**Theorem 3.2.** *The next-generation matrix  $M$  is given by*

$$M = \begin{bmatrix} \lambda_G \mu_I (\mu_T + 1) & p_N \mu_D (\mu_T + 1) \\ \lambda_G \mu_I (\mu_T + 1) & p_N \left( \mu_D (\mu_T + 1) + \frac{\sigma_D^2}{\mu_D} - 1 \right) \end{bmatrix}, \quad (3.5)$$

with  $\mu_T$  given in equation (2.6).

*Proof.* This proof uses similar arguments to the work in Ball et al. (2010).

First consider the expected number of global contacts made by type-1 and type-2 households and note that the number of global contacts made by an individual is independent of its network degree. This means that the expected number of global contacts made by a single household epidemic does not depend on the type of its primary infective, so  $m_{11} = m_{21}$ .

Let  $C_{GG}$  be the number of global contacts made by a single household epidemic where the primary infective was contacted globally. We firstly condition

on the size of the household that the globally infected individual is in. Note that although the proportion of households of size  $n$  is given by  $\rho_n$ , the probability that an individual chosen uniformly at random from the population is in a household of size  $n$  is proportional to  $n\rho_n$ , leading to the size-biased household size distribution  $\tilde{H}$  (see Section 2.1). Thus

$$\mathbb{E}[C_{GG}] = \sum_{n=1}^{\infty} \tilde{\rho}_n \mathbb{E}[C_{GG}^{(n)}],$$

where  $C_{GG}^{(n)}$  is the random variable  $C_{GG}$  conditional on the household being of size  $n$ .

We then decompose  $C_{GG}^{(n)}$  into the number of global contacts emanating from each member of the household, so

$$C_{GG}^{(n)} = C_{GG}(0) + \sum_{j=1}^{n-1} \chi_j C_{GG}(j), \quad (3.6)$$

where we have labelled the individuals in the household  $0, 1, \dots, n-1$ , with individual 0 being the primary infective in the household,  $\chi_j$  is the indicator of the event that individual  $j$  is infected by the household epidemic (i.e.  $\chi_j = 1$  if  $j$  is infected and 0 otherwise) and  $C_{GG}(j)$  is the number of global infections made by individual  $j$ , conditioned on individual  $j$  becoming infectious.

The event that an individual is infected is independent of how many contacts they would make if they became infected, as whether an individual  $j$  is infected in the household epidemic is independent of individual  $j$ 's infectious period, so  $\chi_j$  and  $C_{GG}(j)$  are independent for  $j = 1, 2, \dots, n-1$ . Furthermore,  $(C_{GG}(j), \chi_j)$ ,  $j = 1, 2, \dots, n-1$ , have the same distribution and  $C_{GG}(0)$  has the same distribution as  $C_{GG}(1)$ . So taking expectation of equation (3.6) yields

$$\mathbb{E}[C_{GG}^{(n)}] = \mathbb{E}[C_{GG}(0)] + \mathbb{E}[T^{(n)}] \mathbb{E}[C_{GG}(0)] = \mathbb{E}[C_{GG}(0)] (\mu_{T^{(n)}} + 1), \quad (3.7)$$

where  $T^{(n)} = \sum_{j=1}^{n-1} \chi_j$  is the final size of the household epidemic amongst the secondary individuals expressed in equation (2.5).

An infectious individual makes global contacts at rate  $\lambda_G$  throughout an infectious period  $I$  with mean  $\mu_I$ . In the limit  $m \rightarrow \infty$ , each global contact is made to a distinct individual. Thus the number of global contacts an



infectious individual makes has a Poisson distribution with parameter  $\lambda_G \mu_I$  and  $E[C_{GG}(0)] = \lambda_G \mu_I$ . Thus, using  $T$  as defined in equation (2.6),

$$m_{11} = m_{21} = \sum_{n=1}^{\infty} \tilde{\rho}_n \lambda_G \mu_I (\mu_{T(n)} + 1) = \lambda_G \mu_I (\mu_T + 1). \quad (3.8)$$

Next we consider the number of infectious network contacts. Let  $C_{GN}$  be the total number of network contacts made by the members of a single household epidemic in which the primary infective has been globally contacted and  $C_{NN}$  be the number of network contacts made by the members of a single household epidemic in which the primary infective has been contacted through the network.

Similarly to the argument for  $C_{GG}$  above, we first condition on the size of the household that the primary infective is in. Recall that the probability that an individual chosen uniformly at random from the population is in a household of size  $n$  is given by  $\tilde{\rho}_n$ , and now consider the probability that an individual contacted via the network is in a household of size  $n$ . We assign each individual in the population  $D$  half-edges independent of the individual's household size and these half-edges are then paired uniformly at random to construct the network. So the probability that a given network neighbour is in a household of size  $n$  is independent of its degree and is equal to the probability that an individual chosen uniformly at random in the population is in a household of size  $n$ ,  $\tilde{\rho}_n$ . So, for  $A \in \{G, N\}$ ,

$$E[C_{AN}] = \sum_{n=1}^{\infty} \tilde{\rho}_n E[C_{AN}^{(n)}],$$

where  $C_{GN}^{(n)}$  and  $C_{NN}^{(n)}$  are the random variables  $C_{GN}$  and  $C_{NN}$  conditional on the household being of size  $n$ .

We then decompose  $C_{GN}^{(n)}$  and  $C_{NN}^{(n)}$  into the number of network contacts emanating from each member of the household. So, for  $A \in \{G, N\}$ ,

$$C_{AN}^{(n)} = C_{AN}(0) + \sum_{j=1}^{n-1} \chi_j C_{AN}(j), \quad (3.9)$$

where, as before, we have labelled the individuals in the household  $0, 1, \dots, n-1$ , with individual 0 being the primary infective in the household,  $\chi_j$  is the indicator of the event that individual  $j$  is infected by the household epidemic and  $C_{GN}(j)$  ( $C_{NN}(j)$ ) is the number of network infections made by individual  $j$  in a type-1

(type-2) household, conditioned on individual  $j$  becoming infected.

Using analogous arguments to the work leading to equation (3.7),  $\chi_j$  and  $C_{AN}(j)$  are independent for  $j = 1, 2, \dots, n-1$ , and  $(C_{AN}(j), \chi_j)$ ,  $j = 1, \dots, n-1$  have the same distribution by symmetry. So considering the expected value of equation (3.9) yields

$$\mathbb{E}[C_{AN}^{(n)}] = \mathbb{E}[C_{AN}(0)] + \mathbb{E}[T^{(n)}] \mathbb{E}[C_{AN}(1)]. \quad (3.10)$$

The expectations of both  $C_{GN}(j)$  and  $C_{NN}(j)$ ,  $j = 0, 1$ , can be determined by conditioning on individual  $j$ 's infectious period,  $I_j$ , and the number of uninfected neighbours  $j$  has in the network, which we denote here by  $K_j$  if the primary infective was globally infected and  $\tilde{K}_j$  otherwise. All infectious periods have the same distribution,  $I$  and  $K_0$ ,  $K_1$  and  $\tilde{K}_1$  will have the same distribution as  $D$ . However  $\tilde{K}_0$  will have the same distribution as  $\tilde{D} - 1$ , where  $\tilde{D}$  is the size-biased distribution of  $D$  (see Section 2.1). We require the size-biased distribution because half-edges are paired uniformly at random, so the probability that a given half-edge is linked to an individual with degree  $k$  is proportional to  $kp_k$ . The  $-1$  arises from one of the individual's neighbours, its parent in the branching process, already having been infected.

In this paragraph we use  $K_j$  to also mean  $\tilde{K}_j$ . For  $j = 0, 1$ , conditioned on  $I$  and  $K_j$ , individual  $j$  makes contact with each of its  $K_j$  susceptible neighbours independently at the points of independent Poisson processes with rate  $\lambda_N$  for a time  $I_j$ . Thus  $(C_{GN}(j)|I_j, K_j) \sim \text{Bin}(K_j, 1 - e^{-\lambda_N I_j})$  and  $(C_{NN}(j)|I_j, K_j) \sim \text{Bin}(K_j, 1 - e^{-\lambda_N I_j})$ , since a Poisson process with rate  $\lambda_N$  has no points before a given time  $I_j$  with probability  $e^{-\lambda_N I_j}$ . Therefore, recalling that  $\phi_I(\theta) = \mathbb{E}[e^{-\theta I}]$  and that an individual's infectious period is independent of its degree distribution,

$$\mathbb{E}[C_{AN}(j)] = \mathbb{E}[\mathbb{E}[C_{AN}(j)|I_j, K_j]] = \mathbb{E}[K_j(1 - e^{-\lambda_N I_j})] = \mathbb{E}[K_j](1 - \phi_I(\lambda_N)).$$

Recall that  $p_N = 1 - \phi_I(\lambda_N)$  and  $\mu_{\tilde{D}} = \mu_D + \sigma_D^2/\mu_D$ , so

$$\mathbb{E}[C_{GN}(0)] = \mathbb{E}[C_{GN}(1)] = \mathbb{E}[C_{NN}(1)] = \mu_D p_N, \quad (3.11a)$$

$$\mathbb{E}[C_{NN}(0)] = (\mu_D + \sigma_D^2/\mu_D - 1) p_N. \quad (3.11b)$$

Substituting equations (3.11) into equation (3.10) yields

$$\begin{aligned} \mathbb{E} \left[ C_{GN}^{(n)} \right] &= \mu_D p_N (\mu_{T^{(n)}} + 1), \\ \mathbb{E} \left[ C_{NN}^{(n)} \right] &= \left( \mu_D (\mu_{T^{(n)}} + 1) + \sigma_D^2 / \mu_D - 1 \right) p_N. \end{aligned}$$

Thus

$$\begin{aligned} m_{12} &= \sum_{n=1}^{\infty} \tilde{\rho}_n \mu_D p_N (\mu_{T^{(n)}} + 1) \quad \text{and} \\ m_{22} &= \sum_{n=1}^{\infty} \tilde{\rho}_n \left( \mu_D (\mu_{T^{(n)}} + 1) + \sigma_D^2 / \mu_D - 1 \right) p_N. \end{aligned}$$

Furthermore, substituting  $\mu_T$  as expressed in equation (2.6),

$$m_{12} = \mu_D p_N (\mu_T + 1) \quad \text{and} \quad (3.12a)$$

$$m_{22} = \left( \mu_D (\mu_T + 1) + \sigma_D^2 / \mu_D - 1 \right) p_N. \quad (3.12b)$$

Substituting equations (3.8) and (3.12) together into the next-generation matrix  $M$  yields

$$M = \begin{bmatrix} \lambda_G \mu_I (\mu_T + 1) & p_N \mu_D (\mu_T + 1) \\ \lambda_G \mu_I (\mu_T + 1) & p_N \left( \mu_D (\mu_T + 1) + \frac{\sigma_D^2}{\mu_D} - 1 \right) \end{bmatrix}.$$

□

### 3.2.3 Basic reproduction number $R_0$

As we discuss in the literature review, early definitions of  $R_0$  can be biologically interpreted as the expected total number of individuals infected by a typical individual in the early stages of the epidemic, during their entire infectious period. However, because of the possible small household size, the primary case in a household epidemic is likely to infect more individuals in the household, leading to a larger total number of infectious contacts made, compared to that of an individual infected later in the household epidemic. Therefore there is not an obvious choice of a typical infectious case, and such a definition needs to be carefully considered. We use the definition of  $R_0$  introduced by Pellis et al. (2012).

We now introduce the notation and nomenclature needed to discuss the

methodology used in Pellis et al. (2012). We say that the initial infective belongs to global generation 0. Let global generation 1 consist of those individuals with whom the initial infective has at least one infectious contact through all types of contact. So global generation 1 consists of all individuals directly contacted by the initial infective through the household, via the network or via global contact. Global generation 2 consists of those individuals that are contacted by at least one global-generation-1 individual, through all types of contact, but not by the initial infective, and so on. For  $n = 0, 1, \dots$ , let  $X_n^{(N)}$  denote the number of global generation- $n$  infectives, where  $N$  is the population size. Then  $R_0$  is defined by

$$R_0 = \lim_{n \rightarrow \infty} \lim_{N \rightarrow \infty} \left( \mathbb{E} [X_n^{(N)}] \right)^{\frac{1}{n}}.$$

This definition of  $R_0$  can be interpreted to be approximately the ratio of the number of individuals in the  $k + 1$ th global generation of the epidemic to the number of individuals in the  $k$ th global generation, for large  $k$ .

Similarly to  $R_*$ , in the limit  $m \rightarrow \infty$ , if  $R_0 \leq 1$  then a major outbreak will not occur and if  $R_0 > 1$  there is a positive probability that a major outbreak will occur. Therefore, similarly to  $R_*$ ,  $R_0$  is a limiting threshold parameter as  $m \rightarrow \infty$ .

Before we discuss the forward individual-based branching process used to calculate  $R_0$  in our model we introduce rank generation numbers and calculate the mean number of infection cases in each rank generation of a single household epidemic in the next section.

### **Mean number of cases in each rank generation of a single household epidemic**

We compute the rank generations of infection in the household epidemic as in Ball et al. (2016). Consider a household epidemic in a household of size  $n$ . We label the initial infective 0 and label the  $n - 1$  remaining susceptibles in the household  $1, 2, \dots, n - 1$ . For each individual  $i$ ,  $i = 0, 1, \dots, n - 1$ , we then construct a list containing the individuals that  $i$  would infect, if it became infected itself. Then construct a directed graph,  $\mathcal{G}_f^{(n)}$ , with vertices labelled  $0, 1, \dots, n - 1$ , in which for any ordered pair of distinct vertices  $(i, j)$ , there is a directed edge from  $i$  to  $j$  if and only if individual  $j$  is in individual  $i$ 's list of attempted infections. We say that the initial infective, i.e. individual

0, has a household generation 0. Those individuals that are in 0's list are then said to have household generation 1. Individuals not in individual 0's list, or individual 0, but who are in a household generation-1 infective's list have household generation 2 and so on. The set of individuals ultimately infected by the epidemic comprises those individuals in  $\mathcal{G}_f^{(n)}$  that have a chain of directed edges leading to them from individual 0 and the household generation number of such an infected individual,  $i$  say, is the length of the shortest chain joining 0 to  $i$ , where the length of a chain is the number of edges in it. Following Ludwig (1975) we call these generation numbers *rank* generation numbers.

Denote by  $\mu_i^{(n)}$  the mean number of infectives in generation  $i$  of a single household epidemic with a household size of  $n$ . We follow the calculations made in Pellis et al. (2012) to calculate  $\mu_i^{(n)}$ . We denote the probability of  $m$  susceptibles out of  $s$  escaping direct infection from  $a$  infectives by  $P_a(m, s)$ ,  $a = 1, 2, \dots, s = 1, 2, \dots, m = 0, 1, \dots, s$ . We also denote by  $i = s - m$  the number of infectives in the following generation. Label the  $s$  susceptibles  $1, 2, \dots, s$  and the  $a$  infectives  $-1, -2, \dots, -a$ . We define the indicator random variables  $J_i$ ,  $i = 1, 2, \dots, s$ , to be

$$J_i = \begin{cases} 1 & \text{if individual } i \text{ escapes infection from } a \text{ infectives,} \\ 0 & \text{otherwise,} \end{cases}$$

and  $X = \{i : J_i = 1\}$ , so  $X$  is the set of individuals that escape infection from the  $a$  infectives. Then  $P_a(m, s) = P(|X| = m)$ .

Next consider the inclusion probability

$$R(Y) = P(X \supseteq Y) = E \left[ \prod_{i \in Y} J_i \right] = E \left[ \prod_{i \in Y} \prod_{j \in A} J_i(j) \right],$$

where  $Y \subseteq \{1, 2, \dots, s\}$ ,  $A = \{-1, -2, \dots, -a\}$  and

$$J_i(j) = \begin{cases} 1 & \text{if individual } i \text{ escapes infection from infective } j, \\ 0 & \text{otherwise.} \end{cases}$$

Let  $I_j$  be the infectious period of individual  $j$ . For  $l = -1, -2, \dots, -a$ ,  $k = -1, -2, \dots, -a$ ,  $k \neq l$  and  $i = 1, 2, \dots, s$ , the probability that individual  $l$  makes infectious contact with  $i$  is independent of the probability that individual  $k$

makes infectious contact with  $i$ . Thus, conditioning on the infectious periods of the infected individuals,

$$\begin{aligned} R(Y) &= E \left[ \prod_{i \in Y} \prod_{j \in A} J_i(j) \right] = E \left[ E \left[ \prod_{i \in Y} \prod_{j \in A} J_i(j) \middle| I_{-1}, I_{-2}, \dots, I_{-a} \right] \right] \\ &= E \left[ \prod_{j \in A} E \left[ \prod_{i \in Y} J_i(j) \middle| I_{-1}, I_{-2}, \dots, I_{-a} \right] \right] \\ &= E \left[ \prod_{j \in A} E[J_Y(j) | I_{-1}, I_{-2}, \dots, I_{-a}] \right], \end{aligned}$$

where

$$J_Y(j) = \begin{cases} 1 & \text{if all individuals in } Y \text{ escape infection from infective } j, \\ 0 & \text{otherwise.} \end{cases}$$

Infectious contacts through the household from an individual  $j$ , with given infectious period  $I_j$ , to a given household neighbour  $i$  are made at the points of a Poisson process with rate  $\lambda_H$ . So, given  $I_j$ , the probability that no infectious contacts are made is  $e^{-\lambda_H I_j}$ . All Poisson processes are independent, so

$$R(Y) = E \left[ \prod_{j \in A} E[J_Y(j) | I_{-1}, I_{-2}, \dots, I_{-a}] \right] = E \left[ \prod_{j \in A} e^{-\lambda_H I_j | Y|} \right].$$

The infectious periods are independent and distributed according to  $I$ . Thus

$$R(Y) = E \left[ \prod_{i \in Y} \prod_{j \in A} J_i(j) \right] = \prod_{j \in A} E \left[ e^{-\lambda_H I | Y|} \right] = \phi_I(\lambda_H | Y|)^a.$$

Therefore we see that the probability a given set,  $Y$  say, of individuals from  $\{1, 2, \dots, s\}$  is infected depends only on the size of the set  $Y$ . Then we can use Martin-Löf (1986) Equation 9, based on the Möbius inversion formula, to see that

$$P_a(m, s) = \binom{s}{m} \sum_{k=m}^s (-1)^{k-m} \binom{s-m}{k-m} q_H(k)^a, \quad m = 0, 1, \dots, s,$$

where  $q_H(k) = \phi_I(k\lambda_H)$ .

Next we denote  $Y_{a,s,k}$ ,  $a = 1, 2, \dots, s = 1, 2, \dots, k = 1, 2, \dots, s$ , to be the number of cases in generation  $k$  of the epidemic and let  $\mu_{a,s,k} = E[Y_{a,s,k}]$  be its

expectation. For  $a = 1, 2, \dots, s = 1, 2, \dots, k = 1, 2, \dots, s$ , conditioning on the number of infectives in the first generation yields

$$\mu_{a,s,k} = E[E[Y_{a,s,k}|Y_{a,s,1}]] = \sum_{i=1}^{s-k+1} E[Y_{a,s,k}|Y_{a,s,1} = i] P(Y_{a,s,1} = i).$$

Now  $P(Y_{a,s,1} = i)$ ,  $i = 1, 2, \dots, s - k + 1$ , is the probability that precisely  $i$  out of  $s$  susceptibles are directly infected by  $a$  infectives. We can consider this as the probability  $s - i$  individuals out of  $s$  escape infection from  $a$  infectives, so  $P(Y_{a,s,1} = i) = P_a(s - i, s)$ .

Next consider  $E[Y_{a,s,k}|Y_{a,s,1} = i]$ . This is the same as the expected number of cases in generation  $k - 1$ , when generation 0 has  $i$  initial infectives and  $s - i$  susceptibles, so  $E[Y_{a,s,k}|Y_{a,s,1} = i] = E[Y_{i,s-i,k-1}] = \mu_{i,s-i,k-1}$ . Thus

$$\mu_{a,s,k} = \sum_{i=1}^{s-k+1} P_a(s - i, s) \mu_{i,s-i,k-1},$$

with  $\mu_{a,s,0} = a$  and  $\mu_{a,0,k} = 0$ ,  $a = 1, 2, \dots, s = 1, 2, \dots$ , and  $k = 1, 2, \dots, s$ .

Finally, we conclude by noting that, for  $i = 0, 1, \dots, n - 1$ ,

$$\mu_i^{(n)} = \mu_{1,n-1,i}. \quad (3.13)$$

Note that if we sum over the mean number of infected individuals in each generation conditioned on the household size  $n$ , we get the mean size of a single household epidemic in a household of size  $n$ . So using the definition of  $T^{(n)}$  from equation (2.6) we see that

$$\sum_{i=0}^{n-1} \mu_i^{(n)} = \mu_{T^{(n)}} + 1. \quad (3.14)$$

### The forward individual-based branching process

To calculate  $R_0$ , we construct a discrete-time two-type individual-based branching process, different to the branching process introduced in Section 3.2.1. In the forward Galton-Watson branching process discussed in Section 3.5, each ‘individual’ in the branching process is an infected household and the households containing the global and network contacts made by the infected members of this infected household are the next generation in the branching process, so one

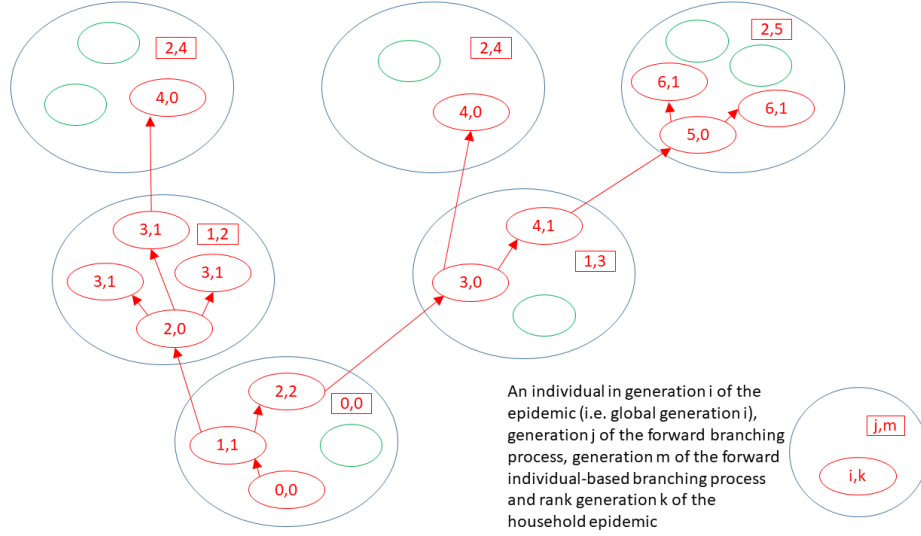


Figure 3.1: Comparing the spread of the epidemic, the individuals in the forward branching process and the individuals in the forward individual-based branching process.

time period corresponds to a household epidemic. Similarly, each ‘individual’ in the forward individual-based branching process consists of an infected household. For the rest of this section, we refer to an ‘individual’ in the forward individual-based branching process as a H-individual, to differ from the individuals in the population. In the contrast to the forward Galton-Watson branching process, in the forward individual-based branching process we consider the infections in the household epidemic occurring in multiple generations as the infection spreads throughout the household. A time period corresponds to a new generation being infected, so an H-individual’s age is which generation of the household epidemic it is in. Thus an H-individual in this branching process will have offspring at multiple time points, as the epidemic spreads through the household. Note that the distribution of the number of offspring of a H-individual over its entire lifespan in this forward individual-based branching process will be the same as the distribution of the number of offspring of an ‘individual’ in the forward Galton-Watson branching process. We hypothesise that, as  $m \rightarrow \infty$ , the total progeny of the forward individual-based branching process will converge in distribution to the total number of (type-1, type-2) infected households infected in the epidemic process on  $m$  households, similarly to the forward Galton-Watson branching process in Section 3.2.1.

We construct this forward individual-based branching process similarly to the single type process used in Ball et al. (2016). As already mentioned, each



H-individual in the branching process represents an infected household. A type-1 individual is a household in which the initial infective was contacted globally, and a type-2 individual is a household in which the initial infective was contacted via the network. Recall from the beginning of Section 3.2.3 that the global generation of an infective is its generation in the epidemic at large. So the initial infective in the epidemic,  $i^*$ , has global generation 0. Given that  $i^*$  became infected, individuals that  $i^*$  would contact, through either the household, the network or globally, are then global generation 1. Similarly, given that individuals in generation 1 became infected, individuals which are not members of a previous global generation that members of generation 1 would infect, through either the household, the network or globally, are then generation 2 and so on. We say that an H-individual's time of birth is given by the global generation of the corresponding household primary case in the epidemic process (see Figure 3.1). An H-individual in this branching process may reproduce at ages  $1, 2, \dots$ . For a type-1 individual, we denote the mean number of type-1 offspring at age  $i + 1$  by  $\nu_{GG}^{(i)}$  and the mean number of type-2 offspring at age  $i + 1$  by  $\nu_{GN}^{(i)}$ . Similarly, for a type-2 individual, we denote the mean number of type-1 offspring at age  $i + 1$  by  $\nu_{NG}^{(i)}$  and the mean number of type-2 offspring at age  $i + 1$  by  $\nu_{NN}^{(i)}$ . Then  $R_0$  is given by the asymptotic (Malthusian) geometric growth rate of the forward individual-based branching process, which is the value of  $\lambda \in \mathbb{R}^+$  such that the maximal eigenvalue of  $V(\lambda)$  is 1, where  $V(\lambda)$  is given by

$$V(\lambda) = \sum_{i=1}^{\infty} \begin{bmatrix} \nu_{GG}^{(i-1)}/\lambda^i & \nu_{GN}^{(i-1)}/\lambda^i \\ \nu_{NG}^{(i-1)}/\lambda^i & \nu_{NN}^{(i-1)}/\lambda^i \end{bmatrix}. \quad (3.15)$$

See, for example, Haccou et al. (2005) Section 3.3.2, adapted to the discrete-time setting.

### Calculation of $R_0$

Recall  $\mu_i^{(n)}$ ,  $i = 0, 1, \dots, n-1$ , the mean number of infectives in rank generation  $i$  of a single household epidemic in a household size of  $n$ , which were calculated in Section 3.2.3. Furthermore, let  $\mu_{i-1} = \sum_{n=i}^{\infty} \tilde{\rho}_n \mu_{i-1}^{(n)}$ , so  $\mu_{i-1}$ ,  $i = 1, 2, \dots$ , is the mean number of cases in rank generation  $i-1$  of a typical single-household epidemic.

**Theorem 3.3.** *The threshold parameter  $R_0$  is the largest positive real root of the equation*

$$1 - \nu_{GG}(\lambda) - \nu_{NN}(\lambda) + \nu_{GG}(\lambda)\nu_{NN}(\lambda) - \nu_{GN}(\lambda)\nu_{NG}(\lambda) = 0, \quad (3.16)$$

*under the constraint*

$$2 - \nu_{GG}(\lambda) - \nu_{NN}(\lambda) \geq 0, \quad (3.17)$$

*where*

$$\nu_{GG}(\lambda) = \nu_{NG}(\lambda) = \lambda_G \mu_I \sum_{i=1}^{\infty} \frac{1}{\lambda^i} \mu_{i-1}, \quad (3.18a)$$

$$\nu_{GN}(\lambda) = \mu_D p_N \sum_{i=1}^{\infty} \frac{1}{\lambda^i} \mu_{i-1}, \quad (3.18b)$$

$$\nu_{NN}(\lambda) = \frac{\mu_{\tilde{D}-1} p_N}{\lambda} + \mu_D p_N \sum_{i=2}^{\infty} \frac{1}{\lambda^i} \mu_{i-1}. \quad (3.18c)$$

*Proof.* We begin by calculating the elements of the matrix  $V(\lambda)$  given in equation (3.15). First note that global and network contacts are both made with individuals in households of size  $\tilde{H}$ . So the mean rank generation sizes of a typical single-household epidemic are given by  $\sum_{n=i}^{\infty} \tilde{\rho}_n \mu_{i-1}^{(n)} = \mu_{i-1}$ ,  $i = 1, 2, \dots$ .

Now consider  $\nu_{GG}^{(i-1)}$  and  $\nu_{NG}^{(i-1)}$ . Note that whether the primary infective was infected globally or via the network does not affect how the epidemic spreads through the household or the number of global contacts made by an individual, so  $\nu_{GG}^{(i-1)} = \nu_{NG}^{(i-1)}$ . As in the proof of Theorem 3.2, the mean number of global contacts made by an infected individual is  $\lambda_G \mu_I$ , so

$$\nu_{GG}^{(i-1)} = \lambda_G \mu_I \mu_{i-1} \quad \text{for } i = 1, 2, \dots,$$

and

$$\sum_{i=1}^{\infty} \nu_{GG}^{(i-1)} / \lambda^i = \lambda_G \mu_I \sum_{i=1}^{\infty} \frac{1}{\lambda^i} \mu_{i-1} = \nu_{GG}(\lambda) = \nu_{NG}(\lambda). \quad (3.19)$$

Next we consider  $\nu_{GN}^{(i-1)}$ . As in the proof of Theorem 3.2, the mean number of network contacts made by a secondary individual in the household epidemic, or an individual infected globally, is  $p_N \mu_D$ . Thus

$$\nu_{GN}^{(i-1)} = p_N \mu_D \mu_{i-1} \quad \text{for } i = 1, 2, \dots,$$

and

$$\sum_{i=1}^{\infty} \nu_{GN}^{(i-1)} / \lambda^i = p_N \mu_D \sum_{i=1}^{\infty} \frac{1}{\lambda^i} \mu_{i-1} = \nu_{GN}(\lambda). \quad (3.20)$$

Finally we consider  $\nu_{NN}^{(i-1)}$ . As mentioned in the proof of Theorem 3.2, the mean number of network contacts made by a secondary individual in the household epidemic is  $p_N \mu_D$  and the mean number of network contacts made by a primary individual infected via the network is  $p_N \mu_{\tilde{D}-1}$ . So the expected network offspring of an individual in generation 0 is  $p_N \mu_{\tilde{D}-1}$  and the expected network offspring of an individual in a subsequent generation is  $p_N \mu_D$ . Therefore

$$\nu_{NN}^{(i-1)} = \begin{cases} p_N \mu_{\tilde{D}-1} & \text{if } i = 1, \\ p_N \mu_D \mu_{i-1} & \text{if } i = 2, 3, \dots, \end{cases}$$

and

$$\sum_{i=1}^{\infty} \nu_{NN}^{(i-1)} / \lambda^i = \frac{p_N \mu_{\tilde{D}-1}}{\lambda} + p_N \mu_D \sum_{i=2}^{\infty} \frac{1}{\lambda^i} \mu_{i-1} = \nu_{NN}(\lambda). \quad (3.21)$$

Equations (3.19), (3.20) and (3.21) yield the elements of the matrix  $V(\lambda)$  in equation (3.15),

$$V(\lambda) = \begin{pmatrix} \nu_{GG}(\lambda) & \nu_{GN}(\lambda) \\ \nu_{NG}(\lambda) & \nu_{NN}(\lambda) \end{pmatrix},$$

which are also the values in equation (3.15). Now that we have  $V(\lambda)$ , we investigate its dominant eigenvalue.

We have defined  $R_0$  to be the value of  $\lambda \in \mathbb{R}^+$  such that the maximal eigenvalue of  $V(\lambda)$  is 1. Since all elements of  $V(\lambda)$  are positive for all  $\lambda > 0$ , the Perron-Frobenius Theorem guarantees a positive real eigenvalue of maximum modulus. Denote the eigenvalues of  $V(\lambda)$  by  $\alpha_{\pm}$  and consider its characteristic equation in variable  $\alpha$ , which we denote  $g(\alpha, \lambda)$ . So

$$g(\alpha, \lambda) = \alpha^2 - \alpha(\nu_{GG}(\lambda) + \nu_{NN}(\lambda)) + \nu_{GG}(\lambda)\nu_{NN}(\lambda) - \nu_{GN}(\lambda)\nu_{NG}(\lambda).$$

This is a quadratic equation in  $\alpha$  and has roots  $\alpha_{\pm}$  where  $\alpha_- \leq \alpha_+$ . Let  $g'(\alpha, \lambda)$  be the derivative of  $g(\alpha, \lambda)$  with respect to  $\alpha$ . The coefficient of  $\alpha^2$  is positive, so  $g'(\alpha_-, \lambda) \leq 0$  and  $g'(\alpha_+, \lambda) \geq 0$ , with equality precisely when  $\alpha_- = \alpha_+$ . Therefore 1 is the maximal eigenvalue of  $V(\lambda)$  if  $g'(1, \lambda) \geq 0$  and  $g(1, \lambda) = 0$ . The constraint  $g'(1, \lambda) \geq 0$  yields

$$2 - \nu_{GG}(\lambda) - \nu_{NN}(\lambda) \geq 0,$$

which is the constraint in equation (3.17). Given that the largest eigenvalue of  $V(\lambda)$  is 1, we can substitute 1 into the characteristic equation to see that  $R_0$  is the largest root of the equation

$$1 - \nu_{GG}(\lambda) - \nu_{NN}(\lambda) + \nu_{GG}(\lambda)\nu_{NN}(\lambda) - \nu_{GN}(\lambda)\nu_{NG}(\lambda) = 0,$$

which is equation (3.16), thus finishing the proof.  $\square$

**Remark.** *As we would intuitively expect,  $R_0 = R_*$  if all households are of size 1, the argument for which we briefly state. It follows easily from equation (3.15) that if all households are of size 1,  $\lambda V(\lambda) = M$ . At  $\lambda = R_0$ , the largest eigenvalue of  $V(\lambda)$  is 1. The largest eigenvalue of  $M$  is defined to be  $R_*$ , and is equal to the largest eigenvalue of  $R_0 V(R_0)$ , which is  $R_0$ . Hence  $R_0 = R_*$  if all households are of size 1.*

### 3.2.4 Ordering between $R_*$ and $R_0$

For this section we assume that there is household structure and that  $\lambda_H > 0$ , since otherwise  $R_0 = R_*$ . Before considering the ordering between  $R_0$  and  $R_*$ , we introduce a proposition which gives an ordering of the maximal eigenvalues of two matrices given information about the ordering of the elements of the two matrices.

**Proposition 3.1.** *Let  $A$  and  $B$  be non-negative matrices and denote their largest eigenvalues by  $\rho(A)$  and  $\rho(B)$  respectively. Then, by Wielandt's Theorem (see, for example, Meyer (2000)), if  $A < B$  then  $\rho(A) < \rho(B)$ .*

We now present a theorem which orders  $R_*$  and  $R_0$ .

**Theorem 3.4.** *(i) If  $R_0 > 1$  then  $R_* > R_0$ .*

*(ii) If  $R_0 < 1$  then  $R_* < R_0$ .*

*(iii)  $R_* = 1 \Leftrightarrow R_0 = 1$ .*

*Proof.* Recall that  $R_*$  is the largest eigenvalue of the next-generation matrix  $M$ . Then  $R_*$  is the value of  $\lambda$  such that the maximal eigenvalue of  $V_*(\lambda)$  is 1, where  $V_*(\lambda) = M/\lambda$ . Thus, substituting in equations (3.14) and (2.6) and then changing the order of summation, we see that

$$V_*(\lambda) = \begin{bmatrix} \nu_{GG}^*(\lambda) & \nu_{GN}^*(\lambda) \\ \nu_{NG}^*(\lambda) & \nu_{NN}^*(\lambda) \end{bmatrix},$$

where

$$\begin{aligned}\nu_{GG}^*(\lambda) &= \frac{\lambda_G \mu_I}{\lambda} \sum_{i=1}^{\infty} \mu_{i-1}, \\ \nu_{NG}^*(\lambda) &= \nu_{GG}^*(\lambda), \\ \nu_{GN}^*(\lambda) &= \frac{\mu_D p_N}{\lambda} \sum_{i=1}^{\infty} \mu_{i-1}, \\ \nu_{NN}^*(\lambda) &= \frac{\mu_{\tilde{D}-1} p_N}{\lambda} + \frac{\mu_D p_N}{\lambda} \sum_{i=2}^{\infty} \mu_{i-1}.\end{aligned}$$

First note that  $V(1) = V_*(1)$  so, as required for part (iii),

$$R_* = 1 \Leftrightarrow R_0 = 1.$$

We write  $\rho(\lambda)$  and  $\rho_*(\lambda)$  for the maximal eigenvalues of  $V(\lambda)$  and  $V_*(\lambda)$  respectively. Note that, for  $\lambda > 0$ , both  $V(\lambda)$  and  $V_*(\lambda)$  are non-negative matrices and that  $\rho(\lambda)$  and  $\rho_*(\lambda)$  are both decreasing functions of  $\lambda$ . Furthermore,

$$\begin{aligned}V_*(\lambda) &< V(\lambda) && \text{if } 0 < \lambda < 1, \\ V_*(\lambda) &> V(\lambda) && \text{if } 1 < \lambda < \infty,\end{aligned}$$

Hence, by Proposition 3.1,

$$\begin{aligned}\rho_*(\lambda) &< \rho(\lambda) && \text{if } 0 < \lambda < 1, \\ \rho_*(\lambda) &> \rho(\lambda) && \text{if } 1 < \lambda < \infty.\end{aligned}$$

Suppose that  $R_0 < 1$ . Then

$$\rho_*(R_0) < \rho(R_0) = 1.$$

A further application of Proposition 3.1 yields that  $\rho_*(\lambda)$  is decreasing on  $(0, \infty)$ . Therefore, since  $\rho_*(R_*) = 1$ , we see that  $R_* < R_0$ . Similarly, if  $R_0 > 1$ , then

$$\rho_*(R_0) > \rho(R_0) = 1 \Rightarrow R_* > R_0.$$

□

### 3.2.5 Probability of a major outbreak

We begin by recalling some notation introduced in Section 3.2.2. The number of (type-1, type-2) offspring of a single type-1 and type-2 individual are denoted by  $(C_{GG}, C_{GN})$  and  $(C_{NG}, C_{NN})$  respectively. Let  $\mathbf{s} = (s_1, s_2)$ ,  $\boldsymbol{\pi} = (\pi_1, \pi_2)$  and  $\mathbf{f}_C(\mathbf{s}) = (f_{C_1}(\mathbf{s}), f_{C_2}(\mathbf{s})) = (\mathbb{E}[s_1^{C_{GG}} s_2^{C_{GN}}], \mathbb{E}[s_1^{C_{NG}} s_2^{C_{NN}}])$ . By standard branching process theory (see Section 2.3), if  $R_* > 1$  then the probability that the forward Galton-Watson branching process does not become extinct is given by  $\rho_{\text{maj}} = 1 - \pi_1$ , where  $\boldsymbol{\pi}$  is the smallest solution of the set of simultaneous equations  $\boldsymbol{\pi} = \mathbf{f}_C(\boldsymbol{\pi})$ . Therefore  $\rho_{\text{maj}}$  is the probability of a major outbreak since, by definition, in the limit  $m \rightarrow \infty$  a major outbreak occurs if and only if the forward Galton-Watson branching process does not become extinct.

We now give explicit expressions for the probability generating functions  $f_{C_1}(\mathbf{s})$  and  $f_{C_2}(\mathbf{s})$ , assuming a constant infectious period of length 1 so  $\phi_I(t) = e^{-t}$ . Note that if the infectious periods are not constant then the infectious periods of individuals infected by a household epidemic are not independent of the final size of that household epidemic, which invalidates the decomposition we use in equation (3.23) to determine  $\mathbf{f}_C(\mathbf{s})$ . For example, a large number of global contacts emanating from a single individual suggests that it had a long infectious period, thus increasing the probability that more household neighbours become infected and also increasing the total number of network contacts made by the household. Similarly to Ball et al. (2010), it is possible to use the theory of final state random variables developed in Ball and O'Neill (1999), by considering the joint probability generating function of the number of global contacts and contacts made via the network of a household epidemic, to express  $\mathbf{f}_C(\mathbf{s})$  in terms of Gontcharoff polynomials. Unfortunately these calculations are very involved and we do not present them here. For convenience let  $G_N(s_2) = f_D(1 - p_N + p_N s_2)$  and  $\tilde{G}_N(s_2) = f_{\tilde{D}-1}(1 - p_N + p_N s_2)$ .

**Theorem 3.5.** *Assuming a unit infectious period, the joint probability generating functions for the offspring distributions of the forward Galton-Watson branching process are given by*

$$\begin{aligned} f_{C_1}(s_1, s_2) &= e^{-\lambda_G(1-s_1)} G_N(s_2) \sum_{n=1}^{\infty} \tilde{\rho}_n f_{T^{(n)}}(e^{-\lambda_G(1-s_1)} G_N(s_2)), \\ f_{C_2}(s_1, s_2) &= e^{-\lambda_G(1-s_1)} \tilde{G}_N(s_2) \sum_{n=1}^{\infty} \tilde{\rho}_n f_{T^{(n)}}(e^{-\lambda_G(1-s_1)} G_N(s_2)). \end{aligned}$$

*Proof.* This proof proceeds by conditioning on the household size of an initial infective and on the size of its household epidemic, and applying independence results to consider the number of type-1 and type-2 offspring from each member of the initial infective's household epidemic separately.

Conditioning the probability generating functions  $f_{C_1}(s_1, s_2)$  and  $f_{C_2}(s_1, s_2)$  on the size of the primary infective's household yields

$$\begin{aligned} f_{C_1}(s_1, s_2) &= \sum_{n=1}^{\infty} \tilde{\rho}_n \mathbb{E} \left[ s_1^{C_{GG}^{(n)}} s_2^{C_{GN}^{(n)}} \right], \\ f_{C_2}(s_1, s_2) &= \sum_{n=1}^{\infty} \tilde{\rho}_n \mathbb{E} \left[ s_1^{C_{NG}^{(n)}} s_2^{C_{NN}^{(n)}} \right], \end{aligned} \tag{3.22}$$

where  $C_{GG}^{(n)}$ ,  $C_{NG}^{(n)}$ ,  $C_{GN}^{(n)}$  and  $C_{NN}^{(n)}$  are the quantities  $C_{GG}$ ,  $C_{NG}$ ,  $C_{GN}$  and  $C_{NN}$  conditioned on the primary infective being in a household of size  $n$ .

As the calculations of  $f_{C_1}(s_1, s_2)$  and  $f_{C_2}(s_1, s_2)$  are similar, we write  $C_{AG}$  and  $C_{AN}$ , where  $A \in \{G, N\}$ . We next decompose  $C_{AG}^{(n)}$  and  $C_{AN}^{(n)}$  into the number of contacts made by each member of the primary infective's household epidemic. So

$$\begin{aligned} C_{AG}^{(n)} &= C_{AG}(0) + \sum_{j=1}^{T^{(n)}} C_{AG}(j) \quad \text{and} \\ C_{AN}^{(n)} &= C_{AN}(0) + \sum_{j=1}^{T^{(n)}} C_{AN}(j), \end{aligned} \tag{3.23}$$

where  $T^{(n)}$  is the size of the primary infective's household epidemic, we have labelled the members of the primary infective's household epidemic  $0, 1, \dots, T^{(n)}$ , with 0 corresponding to the primary individual and  $C_{AG}(j)$  ( $C_{AN}(j)$ ) is the

number of global (network) contacts made by individual  $j$ . Therefore

$$\begin{aligned} \mathbb{E} \left[ s_1^{C_{AG}^{(n)}} s_2^{C_{AN}^{(n)}} \right] &= \mathbb{E} \left[ s_1^{C_{AG}(0) + \sum_{j=1}^{T^{(n)}} C_{AG}(j)} s_2^{C_{AN}(0) + \sum_{j=1}^{T^{(n)}} C_{AN}(j)} \right], \\ &= \mathbb{E} \left[ s_1^{C_{AG}(0)} s_2^{C_{AN}(0)} s_1^{\sum_{j=1}^{T^{(n)}} C_{AG}(j)} s_2^{\sum_{j=1}^{T^{(n)}} C_{AN}(j)} \right]. \end{aligned}$$

Consider a primary infective  $i$  and a secondary infective  $k$  in a household of size  $n$ . In the limit  $m \rightarrow \infty$ , all infectious contacts made by  $i$  and  $k$ , both global and via the network, are made to individuals belonging to different households. Furthermore, since there is a constant infectious period, conditioned on  $T^{(n)}$  we know that: the number of global contacts made by individual  $i$  (or  $k$ ), the degree distribution of  $i$  (or  $k$ ) and the probability a given network neighbour is contacted by  $i$  (or  $k$ ) are all mutually independent. So, for  $j = 0, 1, \dots, T^{(n)}$ ,  $C_{AG}(j)$  and  $C_{AN}(j)$  are pairwise independent for each  $j$  and also independent of  $T^{(n)}$ . Furthermore, the number of contacts made by each individual in the household epidemic are independent, so  $(C_{AG}(0), C_{AN}(0))$ ,  $(C_{AG}(1), C_{AN}(1))$ ,  $\dots$ ,  $(C_{AG}(T^{(n)}), C_{AN}(T^{(n)}))$  are all independent, so

$$\mathbb{E} \left[ s_1^{C_{AG}^{(n)}} s_2^{C_{AN}^{(n)}} \right] = \mathbb{E} \left[ s_1^{C_{AG}(0)} \right] \mathbb{E} \left[ s_2^{C_{AN}(0)} \right] \mathbb{E} \left[ s_1^{\sum_{j=1}^{T^{(n)}} C_{AG}(j)} s_2^{\sum_{j=1}^{T^{(n)}} C_{AN}(j)} \right]. \quad (3.24)$$

We consider the three expectations in equation (3.24) separately. We begin by turning our attention to the last term,

$$\mathbb{E} \left[ s_1^{\sum_{j=1}^{T^{(n)}} C_{AG}(j)} s_2^{\sum_{j=1}^{T^{(n)}} C_{AN}(j)} \right] = \mathbb{E} \left[ \prod_{j=1}^{T^{(n)}} s_1^{C_{AG}(j)} \prod_{j=1}^{T^{(n)}} s_2^{C_{AN}(j)} \right].$$

Conditioning on  $T^{(n)}$  and then using the independence of  $C_{AG}(j)$  and  $C_{AN}(j)$ ,  $j = 1, 2, \dots, T^{(n)}$ , yields

$$\begin{aligned} \mathbb{E} \left[ \prod_{j=1}^{T^{(n)}} s_1^{C_{AG}(j)} \prod_{j=1}^{T^{(n)}} s_2^{C_{AN}(j)} \right] &= \mathbb{E} \left[ \mathbb{E} \left[ \prod_{j=1}^{T^{(n)}} s_1^{C_{AG}(j)} \prod_{j=1}^{T^{(n)}} s_2^{C_{AN}(j)} \middle| T^{(n)} \right] \right] \\ &= \mathbb{E} \left[ \mathbb{E} \left[ \prod_{j=1}^{T^{(n)}} s_1^{C_{AG}(j)} \middle| T^{(n)} \right] \mathbb{E} \left[ \prod_{j=1}^{T^{(n)}} s_2^{C_{AN}(j)} \middle| T^{(n)} \right] \right]. \end{aligned} \quad (3.25)$$

Infectious contacts made by an individual through the network in the forward branching process occur independently through a given network edge with probability  $p_N$ . Let  $K_j$ ,  $j = 0, 1, \dots, T^{(n)}$ , be the number of network neighbours



of  $j$  which are not already infected. Then  $(C_{AN}(j) | K_j) \sim \text{Bin}(K_j, p_N)$ , so  $\mathbb{E} [s_2^{C_{AN}(j)} | K_j] = (1 - p_N + p_N s_2)^{K_j}$ .

Conditioning on  $K = \{K_j, j = 1, 2, \dots, T^{(n)}\}$ , and using the independence of  $C_{AN}(j)$ ,  $j = 1, 2, \dots, T^{(n)}$  yields

$$\begin{aligned} \mathbb{E} \left[ \prod_{j=1}^{T^{(n)}} s_2^{C_{AN}(j)} \middle| T^{(n)} \right] &= \mathbb{E} \left[ \mathbb{E} \left[ \prod_{j=1}^{T^{(n)}} s_2^{C_{AN}(j)} \middle| K, T^{(n)} \right] \middle| T^{(n)} \right] \\ &= \mathbb{E} \left[ \prod_{j=1}^{T^{(n)}} \mathbb{E} [s_2^{C_{AN}(j)} | K, T^{(n)}] \middle| T^{(n)} \right] \\ &= \mathbb{E} \left[ \prod_{j=1}^{T^{(n)}} (1 - p_N + p_N s_2)^{K_j} \middle| T^{(n)} \right]. \end{aligned}$$

For  $j = 1, 2, \dots, T^{(n)}$ ,  $K_j$  are independent copies of  $D$ , so

$$\begin{aligned} \mathbb{E} \left[ \prod_{j=1}^{T^{(n)}} s_2^{C_{AN}(j)} \middle| T^{(n)} \right] &= \mathbb{E} \left[ \prod_{j=1}^{T^{(n)}} (1 - p_N + p_N s_2)^D \middle| T^{(n)} \right] \\ &= (f_D(1 - p_N + p_N s_2))^{T^{(n)}}. \end{aligned} \quad (3.26)$$

Substituting equation (3.26) into equation (3.25) and recalling that  $G_N(s_2) = f_D(1 - p_N + p_N s_2)$  yields

$$\mathbb{E} \left[ \prod_{j=1}^{T^{(n)}} s_1^{C_{AG}(j)} \prod_{j=1}^{T^{(n)}} s_2^{C_{AN}(j)} \right] = \mathbb{E} \left[ \mathbb{E} \left[ \prod_{j=1}^{T^{(n)}} s_1^{C_{AG}(j)} \middle| T^{(n)} \right] (G_N(s_2))^{T^{(n)}} \right]. \quad (3.27)$$

For  $j = 0, 1, \dots, T^{(n)}$ , individual  $j$  makes infectious contacts at the points of a Poisson process with rate  $\lambda_G$ , and each infectious contact is made with a different individual in a different household. Thus  $C_{AG}(j) \sim \text{Poi}(\lambda_G)$ . Substituting  $\mathbb{E} [s_1^{C_{AG}(j)}] = e^{-\lambda_G(1-s_1)}$  into equation (3.27) and then (3.24) yields

$$\begin{aligned} \mathbb{E} [s_1^{C_{GG}^{(n)}} s_2^{C_{GN}^{(n)}}] &= e^{-\lambda_G(1-s_1)} \mathbb{E} [s_2^{C_{GN_0}}] \mathbb{E} \left[ \left( e^{-\lambda_G(1-s_1)} \right)^{T^{(n)}} (G_N(s_2))^{T^{(n)}} \right] \\ &= e^{-\lambda_G(1-s_1)} \mathbb{E} [s_2^{C_{GN_0}}] \mathbb{E} \left[ \left( e^{-\lambda_G(1-s_1)} G_N(s_2) \right)^{T^{(n)}} \right] \\ &= e^{-\lambda_G(1-s_1)} \mathbb{E} [s_2^{C_{GN_0}}] f_{T^{(n)}} \left( e^{-\lambda_G(1-s_1)} G_N(s_2) \right), \end{aligned} \quad (3.28)$$

where an expression for  $f_{T^{(n)}}(s)$  is given by equation (2.4) in Section 2.2.

Finally consider  $\mathbb{E} [s_2^{C_{GN}^{(0)}}]$  and  $\mathbb{E} [s_2^{C_{NN}^{(0)}}]$ . An individual that has been

contacted globally has  $D$  network neighbours, all of whom are susceptible. Thus  $K_0 \stackrel{\mathcal{D}}{=} D$  for a type-1 household and

$$\mathbb{E} \left[ s_2^{C_{GN}(0)} \right] = \mathbb{E} \left[ \mathbb{E} \left[ s_2^{C_{GN}(0)} \middle| K_0 \right] \right] = \mathbb{E} \left[ (1 - p_N + p_N s_2)^D \right] = G_N(s_2).$$

A primary infective in a type-2 household has been infected by one of its network neighbours, so the probability that a primary individual in a type-2 household has degree  $k$  is proportional to  $k p_k$  and the network degree distribution of this primary infective is given by  $\tilde{D}$ . Thus this individual has  $\tilde{D} - 1$  susceptible network neighbours and  $K_0 \stackrel{\mathcal{D}}{=} \tilde{D} - 1$ . So  $\mathbb{E} \left[ s_2^{C_{NN}(0)} \right] = f_{\tilde{D}-1}(1 - p_N + p_N s_2) = \tilde{G}_N(s_2)$ .

Substituting  $\mathbb{E} \left[ s_2^{C_{GN}(0)} \right] = G_N(s_2)$ ,  $\mathbb{E} \left[ s_2^{C_{NN}(0)} \right] = \tilde{G}_N(s_2)$  and equation (3.28) into equation (3.22) yields the required expressions for  $f_{C_1}(s_1, s_2)$  and  $f_{C_2}(s_1, s_2)$  given in the statement of the theorem.  $\square$

### 3.3 Final size of a major outbreak

#### 3.3.1 Susceptibility sets and size of a household susceptibility set

Before our calculation of the expected relative final size of a major outbreak, we first introduce susceptibility sets. The idea behind susceptibility sets is that for each individual in the population we can find all individuals that it would infect, if the individual itself is infected. We can then construct a directed graph based on these lists, in which there is an edge from vertex  $i$  to  $j$  if and only if, given individual  $i$  was infected, individual  $i$  would infect individual  $j$ . The susceptibility set of an individual  $j$  is all individuals that have a path to  $j$  in the digraph and  $j$  itself. Therefore, the probability that an individual ultimately becomes infected in the epidemic is equal to the probability that a member of its susceptibility set becomes infected. Details on susceptibility sets can be found in several papers, for example Ball (2000) and Ball et al. (2009).

We now define an individual's household susceptibility set. Similarly to our construction of  $\mathcal{G}_f^{(n)}$  in Section 3.2.3, we construct the directed graph,  $\mathcal{G}_{BH}^{(n)}$ , with vertices labelled  $0, 1, \dots, n-1$ , in which for any ordered pair  $(i, j)$  of distinct individuals there is a directed arc from  $i$  to  $j$  if and only if  $i$ , if infected, would contact  $j$  through the household. For  $i, k = 1, 2, \dots, n-1$  we write  $i \overset{H}{\rightsquigarrow} k$  if and

only if there is a chain of directed arcs from  $i$  to  $k$  in  $\mathcal{G}_{BH}^{(n)}$ , with the convention that  $i \overset{H}{\rightsquigarrow} i$ . We define the size of individual  $i$ 's household susceptibility set by  $S_i^{(n)} = \left| \left\{ j \in \{0, 1, \dots, n-1\} : j \overset{H}{\rightsquigarrow} i \right\} \right|$ . Note that we can consider  $\mathcal{G}_f^{(n)}$  as a graph in which arcs are added as the epidemic spreads through the population, whereas in  $\mathcal{G}_{BH}^{(n)}$  we follow these arcs backward.

For an individual  $i^*$  in a household of size  $n$ , we define  $M^{(n)}$  to be the size of  $i^*$ 's household susceptibility set, not counting  $i^*$  itself. We use a result of Ball (2000), Lemma 3.1, where it is shown that the probability mass function of  $M^{(n)}$  is given by

$$\mathbb{P}(M^{(n)} = k) = \frac{(n-1)!}{(n-1-k)!} q_{k+1}^{n-1-k} G_k(1|\mathbf{V}) \quad k = 0, 1, \dots, n-1, \quad (3.29)$$

where  $q_k = \phi_I(k\lambda_H)$ ,  $\mathbf{V} = (q_{k+1}, k = 0, 1, \dots)$ . As usual, we denote  $\mathbb{E}[s^{M^{(n)}}] = f_{M^{(n)}}(s)$ .

Next we define an individual's local susceptibility set. We construct the directed graph,  $\mathcal{G}_{BL}^N$ , with vertices labelled  $1, 2, \dots, N$ . For any ordered pair  $(i, j)$  of distinct individuals there is a directed arc from  $i$  to  $j$  if and only if  $i$ , if infected, would contact  $j$  through the household or network. For  $i, j \in \{1, 2, \dots, N\}$  we write  $i \rightsquigarrow j$  if and only if there is a chain of directed arcs from  $i$  to  $j$  in  $\mathcal{G}_{BL}^N$ , with the convention that  $i \rightsquigarrow i$ . We define the size of individual  $i$ 's local susceptibility set by  $S_L^{(N)}(i) = \{j \in \{1, 2, \dots, N\} : j \rightsquigarrow i\}$ . Then we conjecture that, assuming  $\sigma_H^2 < \infty$  and  $\sigma_D^2 < \infty$ , as  $m \rightarrow \infty$ ,  $S_L^{(N)}(i)$  converges in distribution to a limiting random variable,  $S_L$  say. We argue the proof that  $S_L^{(N)}(i) \xrightarrow{\mathcal{D}} S_L$  as  $m \rightarrow \infty$ , follows a similar construction and coupling argument to the proof of Theorem 3.1 given in Section 3.5 (see also Ball and Neal (2008) Section 4.2).

### 3.3.2 Heuristic calculation of the expected relative final size of a major outbreak

Given that an epidemic becomes a major outbreak it is useful to investigate the proportion of the population which becomes infected. We now make a heuristic argument for our calculations of the expected relative final size of a major outbreak, i.e. the proportion of susceptible individuals that become removed at the end of the epidemic process, hereafter referred to as the final size of a major outbreak. There are two main arguments for calculating the final

size of a major outbreak. We use an argument similar to that by Ball and Neal (2002) for the household model. However, Ball et al. (2009) use an alternative argument to give an expression for the final size of a major outbreak in a model with household and network structure by relating an individual's asymptotic susceptibility set to its fate in the event of a major outbreak. We assume that  $\lambda_G > 0$ , since otherwise we revert to the aforementioned model by Ball et al. (2009).

We follow the arguments given by Ball and Neal (2002), until we introduce a two-type branching process. Let  $z$  be the expected relative final size of a major outbreak, by which we mean  $z$  is the proportion of individuals that are ultimately infected by the epidemic in a major outbreak in the limit as  $m \rightarrow \infty$  when the initial infective was chosen uniformly at random from the population. Then  $1 - z$  is the probability that a typical initial susceptible avoids infection by the epidemic. The probability that a typical initial susceptible avoids infection by the epidemic is equal to the probability that all of the individuals in its local susceptibility set avoid infection from global contacts during the course of the epidemic. There are  $N$  individuals in the population and a proportion  $z$  of them are ultimately infected by the epidemic. Each of these individuals has an expected infectious period of  $\mu_I$ . Recalling that the individual to individual global infection rate is  $\lambda_G/N$  and that each individual has an expected infectious period of  $\mu_I$ , we see that the probability a given individual avoids global infection is approximately given by the probability that a Poisson process with rate  $\lambda_G/N$  has no events in a time interval with length  $Nz\mu_I$ . Thus the probability a given individual avoids global infection is approximately  $\pi = e^{-\lambda_G z \mu_I}$ .

In the limit as  $m \rightarrow \infty$  this approximation becomes exact and distinct individuals avoid global infection independently of each other. Thus the probability that a typical individual's local susceptibility avoids infection from global contacts is given by  $E[\pi^{|S_L|}]$ , and  $z$  satisfies

$$1 - z = E[\pi^{|S_L|}] = f_{|S_L|}(e^{-\lambda_G z \mu_I}). \quad (3.30)$$

We can investigate the roots of this equation by considering a single-type Galton-Watson branching process in which the number of offspring of a typical individual,  $\tilde{R}$  say, follows a Poisson distribution with random mean  $\lambda_G \mu_I |S^L|$ .

Then  $\tilde{R}$  has probability generating function given by

$$f_{\tilde{R}}(s) = \mathbb{E} [s^{\tilde{R}}] = \mathbb{E} [\mathbb{E} [s^{\tilde{R}} | S_1^L]] = \mathbb{E} [e^{-\lambda_G \mu_I |S_1^L|(1-s)}] = f_{|S_1^L|} (e^{-\lambda_G \mu_I (1-s)}).$$

Then  $s = 1 - z$  satisfies the equation  $s = f_{\tilde{R}}(s)$ , the equation governing the extinction probability of the above single-type Galton-Watson branching process with one initial ancestor chosen uniformly at random from the population. Therefore we conjecture that  $1 - z$  is equal to the probability that the above single-type Galton-Watson branching process with one initial ancestor becomes extinct.

Recall that in the forward Galton-Watson branching process (a two-type process), used to approximate the early stages of the epidemic, each individual in the branching process corresponds to a single-household epidemic and their type is determined by whether the primary infective is infected via a global or network contact. In contrast, the above single-type Galton-Watson branching process can be viewed as approximating the spread of an individuals susceptibility set through the population, with each individual in the branching process corresponding to a single local susceptibility set. We now consider a two-type Galton-Watson branching process, the backward Galton-Watson branching process, in which each individual in the branching process corresponds to a single household susceptibility set and their type is determined by whether the primary individual joins the susceptibility set via a global or network contact. Furthermore, we argue that the backward Galton-Watson branching process has the same probability of extinction as the above single-type Galton-Watson branching process, and thus can be applied to calculate  $z$ . We consider the backward Galton-Watson branching process over the single-type Galton-Watson branching process because we find the two-type branching process easier to analytically investigate.

We construct the two-type backward Galton-Watson branching process in a similar manner to Ball et al. (2010). So we start with an initial individual,  $j$ , who has been chosen uniformly at random from the population. The type-1 offspring in the first generation of the backward Galton-Watson branching process consists of the households containing an individual that makes global infectious contact with a member of individual  $j$ 's household susceptibility set (we call these individuals the type-1 primary individual in their household). Similarly, the type-2 offspring in the first generation of the backward Galton-Watson branching

process consists of the households containing an individual that makes infectious contact via the network with a member of individual  $j$ 's household susceptibility set (we call these individuals the type-2 primary individual in their household). Subsequent generations then consist of those households with individuals who make infectious contact globally (type-1), or via the network (type-2), with the household susceptibility set of any of the primary individuals in the previous generation of the backward branching process.

Then the single-type Galton-Watson branching process can be viewed as an 'embedded' version of the two-type backward Galton-Watson branching process, as we now explain. We remove the type-2 individuals in the two-type backward Galton-Watson branching process by attributing all offspring of a type-2 individual to its parent. Thus the total progeny of the new branching process is equal to the number of type-1 individuals in the two-type backward Galton-Watson branching process, and the number of offspring of an individual in the new branching process is equal to the total number of type-1 individuals in the two-type backward Galton-Watson branching process connected to the corresponding individual via a chain of type-2 individuals. Note that each individual in the new branching process then corresponds to a single local susceptibility set, and indeed the new branching process is the single-type Galton-Watson branching process. It is clear from the construction of the single-type Galton-Watson branching process from the backwards Galton-Watson branching process that, assuming  $\lambda_G > 0$ , the two-type backward Galton-Watson branching process has finite total progeny if and only if the single-type Galton-Watson branching process has finite total progeny. Therefore, both the backward Galton-Watson branching process and the single-type Galton-Watson branching process have the same probability of extinction, as required to calculate  $z$ .

### 3.3.3 The backward Galton-Watson branching process

Before considering the backward Galton-Watson branching process, we give the following proposition about the convergence in distribution of the Binomial distribution to the Poisson distribution, whose proof is given in, for example, Roussas (1997) Section 3.4, Theorem 1.

**Proposition 3.2.** *Let  $X^{(N)} \sim \text{Bin}(N, p(N))$  and  $Y \sim \text{Poi}(\lambda)$ . If  $p(N) \rightarrow 0$  and  $Np(N) \rightarrow \lambda > 0$  as  $N \rightarrow \infty$  then  $X^{(N)} \xrightarrow{\mathcal{D}} Y$ .*

Let  $B_{GG}$  be the number of global contacts made to the members of the household susceptibility set of a type-1 primary individual and let  $B_{NG}$  be the number of global contacts made to the members of the household susceptibility set of a type-2 primary individual. Similarly, let  $B_{GN}$  be the number of contacts made via the network to the members of the household susceptibility set of a type-1 primary individual and let  $B_{NN}$  be the number of contacts made via the network to the members of the household susceptibility set of a type-2 primary individual.

We let  $b_1(s_1, s_2)$ ,  $(s_1, s_2) \in [0, 1]^2$ , be the probability generating function for the offspring distribution of a type-1 household in the backward branching process, so  $b_1(s_1, s_2) = E[s_1^{B_{GG}} s_2^{B_{GN}}]$ . Similarly, let  $b_2(s_1, s_2)$  be the probability generating function for the offspring distribution of a single type-2 household in the backward branching process, so  $b_2(s_1, s_2) = E[s_1^{B_{NG}} s_2^{B_{NN}}]$ .

The expected relative final size of a major outbreak is  $z = 1 - \pi_1$ , where  $\pi = (\pi_1, \pi_2)$  is the smallest solution to the set of simultaneous equations  $\pi = \mathbf{b}(\pi)$ , where  $\mathbf{b}(s_1, s_2) = (b_1(s_1, s_2), b_2(s_1, s_2))$ .

**Theorem 3.6.** *The joint probability generating functions for the offspring distributions of the backward Galton-Watson branching process are given by*

$$\begin{aligned} b_1(s_1, s_2) &= e^{-\lambda_G \mu_I (1-s_1)} G_N(s_2) \sum_{n=1}^{\infty} \tilde{\rho}_n f_{M(n)} \left( e^{-\lambda_G \mu_I (1-s_1)} G_N(s_2) \right), \\ b_2(s_1, s_2) &= e^{-\lambda_G \mu_I (1-s_1)} \tilde{G}_N(s_2) \sum_{n=1}^{\infty} \tilde{\rho}_n f_{M(n)} \left( e^{-\lambda_G \mu_I (1-s_1)} G_N(s_2) \right). \end{aligned}$$

*Proof.* This proof follows by conditioning on the household size of a primary individual and the size of its household susceptibility set, and applying independence arguments to consider the number of global contacts and contacts made via the network to each member of its household susceptibility set separately.

Consider an individual  $i$  in the population. The probability that another individual  $j$  makes global infectious contact with  $i$  depends on  $j$ 's infectious period but not on  $i$ 's, thus  $i$ 's network degree is independent of the number of global infectious contacts  $i$  receives and, since the only difference between a type-1 household and a type-2 household is the network degree of the primary individual, we know that  $B_{GG} \stackrel{\mathcal{D}}{=} B_{NG}$ .

We first condition the probability generating functions  $b_1(s_1, s_2)$  and  $b_2(s_1, s_2)$  on the size of the household that the primary individual is in. So

$$\begin{aligned} b_1(s_1, s_2) &= \sum_{n=1}^{\infty} \tilde{\rho}_n \mathbb{E} \left[ s_1^{B_{GG}^{(n)}} s_2^{B_{GN}^{(n)}} \right], \\ b_2(s_1, s_2) &= \sum_{n=1}^{\infty} \tilde{\rho}_n \mathbb{E} \left[ s_1^{B_{NG}^{(n)}} s_2^{B_{NN}^{(n)}} \right], \end{aligned} \quad (3.31)$$

where  $B_{GG}^{(n)}$ ,  $B_{NG}^{(n)}$ ,  $B_{GN}^{(n)}$  and  $B_{NN}^{(n)}$  are the quantities  $B_{GG}$ ,  $B_{NG}$ ,  $B_{GN}$  and  $B_{NN}$  conditioned on the primary individual being in a household of size  $n$ .

As the calculations of  $b_1(s_1, s_2)$  and  $b_2(s_1, s_2)$  are similar, we write  $B_{AG}$  and  $B_{AN}$ , where  $A \in \{G, N\}$ . We next decompose  $B_{AG}^{(n)}$  and  $B_{AN}^{(n)}$  into the number of contacts made to each member of the primary individual's household susceptibility set. Thus

$$\begin{aligned} B_{AG}^{(n)} &= B_{AG}(0) + \sum_{j=1}^{M^{(n)}} B_{AG}(j), \\ B_{AN}^{(n)} &= B_{AN}(0) + \sum_{j=1}^{M^{(n)}} B_{AN}(j), \end{aligned}$$

where we have labelled the members of the primary individual's household susceptibility set  $0, 1, \dots, M^{(n)}$ , with 0 corresponding to the primary individual,  $B_{AG}(j)$  ( $B_{AN}(j)$ ) is the number of global (network) contacts made to individual  $j$  and  $M^{(n)}$  is the size of the primary individual's household susceptibility set.

Applying this decomposition we can break down  $\mathbb{E} \left[ s_1^{B_{AG}^{(n)}} s_2^{B_{AN}^{(n)}} \right]$ , so

$$\begin{aligned} \mathbb{E} \left[ s_1^{B_{AG}^{(n)}} s_2^{B_{AN}^{(n)}} \right] &= \mathbb{E} \left[ s_1^{B_{AG}(0) + \sum_{j=1}^{M^{(n)}} B_{AG}(j)} s_2^{B_{AN}(0) + \sum_{j=1}^{M^{(n)}} B_{AN}(j)} \right], \\ &= \mathbb{E} \left[ s_1^{B_{AG}(0)} s_2^{B_{AN}(0)} s_1^{\sum_{j=1}^{M^{(n)}} B_{AG}(j)} s_2^{\sum_{j=1}^{M^{(n)}} B_{AN}(j)} \right]. \end{aligned}$$

Consider a primary individual  $i$  and a secondary individual  $k$  in a household of size  $n$ . As  $m \rightarrow \infty$  and the susceptibility set process contains only a small proportion of the total population, all infectious contacts made to  $i$ 's household susceptibility set, both global and via the network, are made by individuals belonging to different households. Therefore all infectious contacts made to the



members of  $i$ 's household susceptibility set are made by individuals with independent and identically distributed infectious periods,  $I$ . We also know that the number of global contacts made is independent of an individual's network degree, so the number of global contacts made to  $i$  (or  $k$ ) is independent of the number of contacts made to  $i$  (or  $k$ ) via the network, so, for  $j = 0, 1, \dots, M^{(n)}$ ,  $B_{AG}(j)$  and  $B_{AN}(j)$  are pairwise independent for each  $j$  and also independent of  $M^{(n)}$ . Furthermore, the number of contacts made to each member of  $i$ 's household susceptibility set are independent, so  $(B_{AG}(0), B_{AN}(0))$ ,  $(B_{AG}(1), B_{AN}(1))$ ,  $\dots$ ,  $(B_{AG}(M^{(n)}), B_{AN}(M^{(n)}))$  are all independent and

$$\mathbb{E} \left[ s_1^{B_{AG}^{(n)}} s_2^{B_{AN}^{(n)}} \right] = \mathbb{E} \left[ s_1^{B_{AG}(0)} \right] \mathbb{E} \left[ s_2^{B_{AN}(0)} \right] \mathbb{E} \left[ s_1^{\sum_{j=1}^{M^{(n)}} B_{AG}(j)} s_2^{\sum_{j=1}^{M^{(n)}} B_{AN}(j)} \right]. \quad (3.32)$$

We now consider the last term in equation (3.32),

$$\mathbb{E} \left[ s_1^{\sum_{j=1}^{M^{(n)}} B_{AG}(j)} s_2^{\sum_{j=1}^{M^{(n)}} B_{AN}(j)} \right] = \mathbb{E} \left[ \prod_{j=1}^{M^{(n)}} s_1^{B_{AG}(j)} \prod_{j=1}^{M^{(n)}} s_2^{B_{AN}(j)} \right].$$

Conditioning on  $M^{(n)}$  and then using the independence of  $B_{AG}(j)$  and  $B_{AN}(j)$ ,  $j = 1, 2, \dots, M^{(n)}$  yields

$$\begin{aligned} \mathbb{E} \left[ \prod_{j=1}^{M^{(n)}} s_1^{B_{AG}(j)} \prod_{j=1}^{M^{(n)}} s_2^{B_{AN}(j)} \right] &= \mathbb{E} \left[ \mathbb{E} \left[ \prod_{j=1}^{M^{(n)}} s_1^{B_{AG}(j)} \prod_{j=1}^{M^{(n)}} s_2^{B_{AN}(j)} \middle| M^{(n)} \right] \right] \\ &= \mathbb{E} \left[ \mathbb{E} \left[ \prod_{j=1}^{M^{(n)}} s_1^{B_{AG}(j)} \middle| M^{(n)} \right] \mathbb{E} \left[ \prod_{j=1}^{M^{(n)}} s_2^{B_{AN}(j)} \middle| M^{(n)} \right] \right]. \end{aligned} \quad (3.33)$$

Infectious contacts made to an individual through the network in the backward branching process occur independently through a given network edge with probability  $p_N$ . If we let  $K_j$ ,  $j = 0, 1, \dots, M^{(n)}$ , be the number of network neighbours of  $j$  which are not already in a household susceptibility set we see that  $(B_{AN}(j) | K_j) \sim \text{Bin}(K_j, p_N)$  so  $\mathbb{E} [s_2^{B_{AN}(j)} | K_j] = (1 - p_N + p_N s_2)^{K_j}$ .

Conditioning on  $K = \{K_j, j = 1, 2, \dots, M^{(n)}\}$ , and applying the indepen-

dence of  $B_{AN}(j)$ ,  $j = 1, 2, \dots, M^{(n)}$  yields

$$\begin{aligned} \mathbb{E} \left[ \prod_{j=1}^{M^{(n)}} s_2^{B_{AN}(j)} \middle| M^{(n)} \right] &= \mathbb{E} \left[ \mathbb{E} \left[ \prod_{j=1}^{M^{(n)}} s_2^{B_{AN}(j)} \middle| K, M^{(n)} \right] \middle| M^{(n)} \right] \\ &= \mathbb{E} \left[ \prod_{j=1}^{M^{(n)}} \mathbb{E} \left[ s_2^{B_{AN}(j)} \middle| K, M^{(n)} \right] \middle| M^{(n)} \right] \\ &= \mathbb{E} \left[ \prod_{j=1}^{M^{(n)}} (1 - p_N + p_N s_2)^{K_j} \middle| M^{(n)} \right]. \end{aligned}$$

For  $j = 1, 2, \dots, M^{(n)}$ ,  $K_j$  are independent copies of  $D$  so

$$\begin{aligned} \mathbb{E} \left[ \prod_{j=1}^{M^{(n)}} s_2^{B_{AN}(j)} \middle| M^{(n)} \right] &= \mathbb{E} \left[ \prod_{j=1}^{M^{(n)}} (1 - p_N + p_N s_2)^D \middle| M^{(n)} \right] \\ &= (f_D(1 - p_N + p_N s_2))^{M^{(n)}}. \end{aligned} \quad (3.34)$$

Substituting equation (3.34) into equation (3.33), and recalling that  $G_N(s_2) = f_D(1 - p_N + p_N s_2)$  yields

$$\mathbb{E} \left[ \prod_{j=1}^{M^{(n)}} s_1^{B_{AG}(j)} \prod_{j=1}^{M^{(n)}} s_2^{B_{AN}(j)} \right] = \mathbb{E} \left[ \mathbb{E} \left[ \prod_{j=1}^{M^{(n)}} s_1^{B_{AG}(j)} \middle| M^{(n)} \right] (G_N(s_2))^{M^{(n)}} \right]. \quad (3.35)$$

Next we consider the distribution of  $B_{NG}(j)$ ,  $j = 0, 1, \dots, M^{(n)}$ . Recall that an individual has infectious period  $I$  and consider a population containing  $N$  individuals. Then, for specified individuals,  $l$  and  $k$  say,

$$\begin{aligned} \mathbb{P}(l \text{ globally contacts } k) &= 1 - \mathbb{E} \left[ e^{-\lambda_G I / N} \right] \\ &= 1 - \phi_I(\lambda_G / N). \end{aligned}$$

Denote by  $W_j^{(N)}$  the number of individuals that contact an individual  $j$  globally in a population of size  $N$ . Then  $W_j^{(N)} \sim \text{Bin}(N, 1 - \phi_I(\lambda_G / N))$ . Clearly  $\lim_{N \rightarrow \infty} W_j^{(N)} \stackrel{\mathcal{D}}{=} B_{GG}(j)$ . If, as  $N \rightarrow \infty$ ,

- (i)  $1 - \phi_I(\lambda_G / N) \rightarrow 0$ ,
- (ii)  $N(1 - \phi_I(\lambda_G / N)) \rightarrow \lambda_G \mu_I > 0$ ,

then applying Proposition 3.2 yields  $B_{GG_j} \sim \text{Poi}(\lambda_G \mu_I)$ . Therefore we need only check the conditions required for Proposition 3.2 hold.

Clearly  $\phi_I(\lambda_G/N) \rightarrow \phi_I(0) = 1$  as  $N \rightarrow \infty$ , so  $1 - \phi_I(\lambda_G/N) \rightarrow 0$ . Therefore we need only show that  $N(1 - \phi_I(\lambda_G/N)) \rightarrow \lambda_G \mu_I > 0$ . Substituting  $h = \lambda_G/N$  yields

$$N(1 - \phi_I(\lambda_G/N)) = \lambda_G \frac{(\phi_I(0) - \phi_I(\lambda_G/N))}{\lambda_G/N} = \lambda_G \frac{(\phi_I(0) - \phi_I(h))}{h}.$$

Since  $\phi_I(h) < \phi_I(0)$  and  $h \rightarrow 0$  as  $N \rightarrow \infty$ , by the definition of the derivative

$$\lim_{h \rightarrow 0} \lambda_G \frac{(\phi_I(0) - \phi_I(h))}{h} = \lambda_G \phi_I'(0) = \lambda_G \mu_I.$$

If  $\lambda_G = 0$  then no global contacts are made and  $B_{GG}(j) = 0$ . Otherwise, since  $I$  is a non-negative random variable and so  $\mu_I > 0$ ,  $\lambda_G \mu_I > 0$ . Thus, for  $j = 1, 2, \dots, M^{(n)}$ ,  $B_{GG}(j) \sim \text{Poi}(\lambda_G \mu_I)$  and thus  $\mathbb{E}[s_1^{B_{AG}(j)}] = e^{-\lambda_G \mu_I(1-s_1)}$ .

Substituting equation (3.35) into equation (3.32) yields

$$\begin{aligned} \mathbb{E}[s_1^{B_{GG}^{(n)}} s_2^{B_{GN}^{(n)}}] &= e^{-\lambda_G \mu_I(1-s_1)} \mathbb{E}[s_2^{B_{GN_0}}] \mathbb{E}\left[\left(e^{-\lambda_G \mu_I(1-s_1)}\right)^{M^{(n)}} (G_N(s_2))^{M^{(n)}}\right] \\ &= e^{-\lambda_G \mu_I(1-s_1)} \mathbb{E}[s_2^{B_{GN_0}}] \mathbb{E}\left[\left(e^{-\lambda_G \mu_I(1-s_1)} G_N(s_2)\right)^{M^{(n)}}\right] \\ &= e^{-\lambda_G \mu_I(1-s_1)} \mathbb{E}[s_2^{B_{GN_0}}] f_{M^{(n)}}\left(e^{-\lambda_G \mu_I(1-s_1)} G_N(s_2)\right), \end{aligned} \quad (3.36)$$

where an expression for  $f_{M^{(n)}}(s)$  is given in equation (3.29).

Finally we consider  $\mathbb{E}[s_2^{B_{GN}^{(0)}}]$  and  $\mathbb{E}[s_2^{B_{NN}^{(0)}}]$ . An individual chosen uniformly at random from the population has  $D$  network neighbours, all of whom are able to infect the primary individual through the network if they were to become infected. Thus for a type-1 household  $K_0 \stackrel{\mathcal{D}}{=} D$  and

$$\mathbb{E}[s_2^{B_{GN}^{(0)}}] = \mathbb{E}[\mathbb{E}[s_2^{B_{GN}^{(0)}} | K_0]] = \mathbb{E}[(1 - p_N + p_N s_2)^D] = G_N(s_2).$$

Next consider the primary individual in a type-2 household. We know that this primary individual has  $\tilde{D}$  network neighbours, and  $\tilde{D} - 1$  network neighbours which are not already in a susceptibility set, so  $K_0 \stackrel{\mathcal{D}}{=} \tilde{D} - 1$ . Thus  $\mathbb{E}[s_2^{B_{NN}^{(0)}}] = f_{\tilde{D}-1}(1 - p_N + p_N s_2) = \tilde{G}_N(s_2)$ .

Substituting the results  $\mathbb{E}[s_2^{B_{GN}^{(0)}}] = G_N(s_2)$ ,  $\mathbb{E}[s_2^{B_{NN}^{(0)}}] = \tilde{G}_N(s_2)$  and equation (3.36) into equation (3.31) yields the expressions for  $b_1(s_1, s_2)$  and  $b_2(s_1, s_2)$  given in the statement of the theorem as required.  $\square$

**Remark.** *In the case of a constant infectious period, it is clear that the probability generating functions for the final size of a household epidemic and the size of an individual's household susceptibility set, i.e.  $f_{T(n)}(s)$  and  $f_{M(n)}(s)$  given in equations (2.4) and (3.29) respectively, are equal. Therefore the probability generating functions for the offspring of the forward branching processes and backward branching processes are equal and the probability of a major outbreak is equal to the expected relative final size of a major outbreak.*

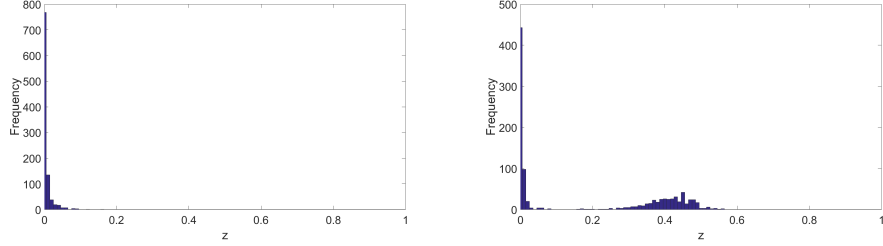
## 3.4 Numerical studies

### 3.4.1 Accuracy of asymptotic results for finite $m$

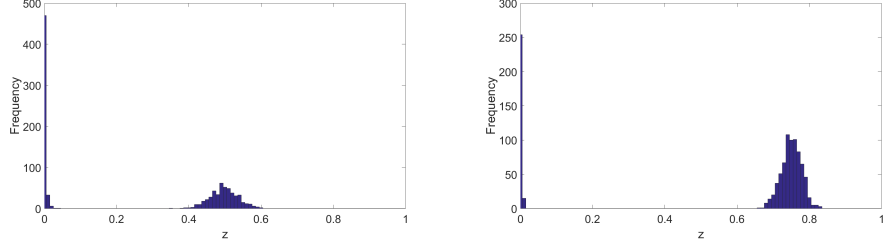
In this section we investigate whether the asymptotic results for the probability of a major outbreak and the final size of a major outbreak, given in Sections 3.2.5 and 3.3.3 respectively, give a good approximation for the final size of a major outbreak in finite populations. To do this we run 1000 simulations of the epidemic on finite populations and then estimate the quantities of interest empirically, comparing these results with the asymptotic calculations. In Appendix A.1 we give an algorithm in pseudo-code for a single simulation, however we now give an overview of the method.

For each simulation we generate a random population with the desired network and household structure and then run a single epidemic with the initial infective chosen uniformly at random. Note that in each simulation we consider a new population and construct a new network and household structure. We then determine a cut-off for whether a particular final size constitutes a major outbreak by inspecting histograms of the relative final size for our simulations, for which we find that a cut-off of 0.15 of the population size is appropriate for the population sizes and parameters we use (see Figures 3.2).

We also include error bounds for the probability and final size of a major outbreak in finite populations which are  $\pm 2$  standard errors ( $SE$ s) of the estimator, similarly to Ball et al. (2010). Note that for the probability of a major outbreak, estimated as  $\hat{p}_{maj}$ ,  $SE = \sqrt{\hat{p}_{maj}(1 - \hat{p}_{maj})/n}$ . For the mean relative final size  $SE = \hat{\sigma}_{RFS}/\sqrt{n_{maj}}$  where  $\hat{\sigma}_{RFS}$  is the sample standard deviation of the relative final sizes of the  $n_{maj}$  major outbreaks. The difference in size of the standard error between the probability of a major outbreak and the final size of a major



(a)  $\lambda_N = 0.1$ ,  $\lambda_H = 0.1$  and  $\lambda_G = 0.05$ , so  $R_0 = 0.89$ . (b)  $\lambda_N = 0.15$ ,  $\lambda_H = 0.3$ ,  $\lambda_G = 0.2$ , so  $R_0 = 1.5$ .



(c)  $\lambda_N = 0.25$ ,  $\lambda_H = 0.1$  and  $\lambda_G = 0.25$ , so  $R_0 = 2.0$ . (d)  $\lambda_N = 0.35$ ,  $\lambda_H = 0.4$  and  $\lambda_G = 0.25$ , so  $R_0 = 2.8$ .

Figure 3.2: Histograms showing the final size of the epidemic in finite populations. In all figures  $H \sim \text{Const}(3)$ ,  $D \sim \text{Geo}(1/5)$  and  $m = 200$ . Using the cutoff  $z = 0.15$ , we determine that no major outbreak occurs in Figure 3.2a, whereas major outbreaks do occur in Figures 3.2b, 3.2c and 3.2d.

outbreak is because each simulation tells us one piece of information about whether a major outbreak occurred or not, however for each major outbreak that occurs we receive information from each initial susceptible in the population. Although the information about the final size of a major outbreak is highly correlated, each simulation contains more information about the final size of a major outbreak than available from a single simulation about the probability of a major outbreak and thus leads to much smaller error bars.

As with the similar investigations by Ball and Neal (2008) and Ball et al. (2010), we find that the asymptotic probability and final size of a major outbreak are quite good approximations for a small number of individuals, as illustrated in Figures 3.3. Changing the household size distribution, without heavy-tails, seems to have little impact on the convergence (see Figures 3.3a and 3.3g and Figures 3.3b and 3.3h). Furthermore, changing the network distribution, without heavy-tails, also has little impact on the convergence (see Figures 3.3c and 3.3e and Figures 3.3d and 3.3f). However, we conjecture that investigating a degree distribution with heavy-tails would result in a slower convergence, similarly to Ball et al. (2010). Furthermore, we conjecture that investigating a household

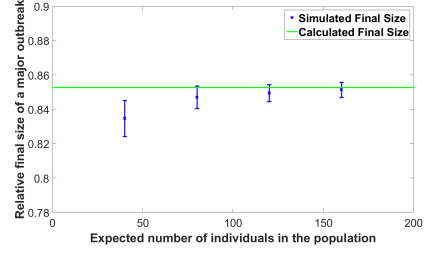
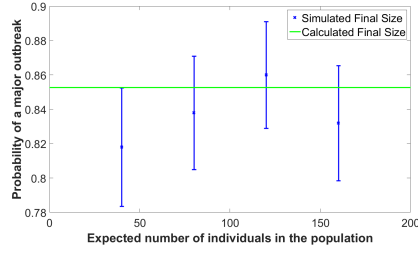
size distribution with heavy-tails would also result in a slower convergence.

### 3.4.2 Exploration of the model

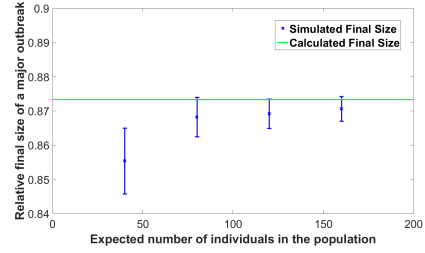
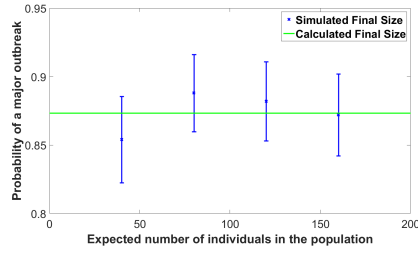
The epidemic model we introduce in this chapter allows for individuals to transmit the epidemic between households via global contacts and via a configuration model network structure. Therefore an important question to therefore consider is whether the complexity from the addition of both routes of transmission is warranted. For example, if we incorrectly assume that the population is homogeneously mixing outside of the household, when there is actually a network structure, and estimate the final size of a major outbreak, how ‘bad’ is our approximation? Furthermore, how should we tune these models so that they are comparable? In this section we choose to compare two epidemic models by keeping the threshold parameter  $R_0$  constant. Indeed, this is the method used by Clancy and Pearce (2013) to compare several properties of multi-type epidemic models including the final size of a major outbreak. Therefore whenever we reference reducing one or more infection rates at the expense of others, we are implicitly doing this in such a way that  $R_0$  remains fixed. We note that this is not the only way to compare different epidemic models, for example models can be matched by final size epidemic data or the real-time growth rate. This section provides motivation for Chapter 4, which explores analytical results on the simpler models of Ball et al. (1997) and Ball et al. (2009).

Firstly, if both  $\sigma_H^2$  and  $\sigma_D^2$  are small then fixing  $R_0$  and increasing either of their corresponding infection rates ( $\lambda_N$  or  $\lambda_H$  respectively) at the expense of the casual contact infection rate ( $\lambda_G$ ) will increase the final size of a major outbreak, as illustrated in Figure 3.4a. In contrast, if  $\sigma_D^2$  is ‘large’ then fixing  $R_0$  and increasing  $\lambda_N$  at the expensive of  $\lambda_G$  will instead decrease the final size of a major outbreak, as illustrated in Figure 3.4b. However, fixing  $R_0$  and increasing  $\lambda_N$  at the expensive of  $\lambda_G$  will not always result in a monotonic change in the final size of a major outbreak, as illustrated in Figure 3.4c. However, we note that the changes in Figure 3.4c are very small. Indeed, fixing  $R_0$  and increasing  $\lambda_H$  at the expense of  $\lambda_G$  also does not always result in a monotonic effect on the final size of a major outbreak, as illustrated in Figure 3.4d.

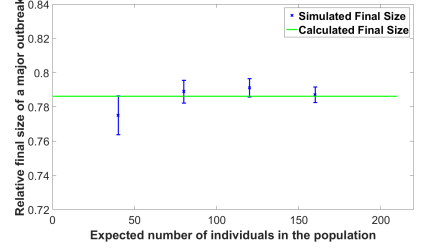
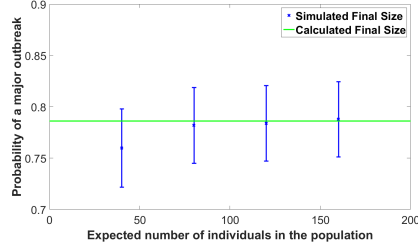
This implies that, assuming we correctly estimate  $R_0$ , the final size of a major outbreak is sensitive to our choice of parameters  $H$ ,  $D$ ,  $\lambda_H$ ,  $\lambda_N$  and  $\lambda_G$ , suggesting that casual contacts and network contacts can affect the final size



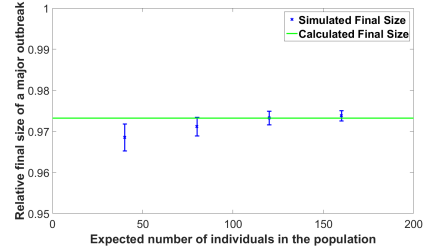
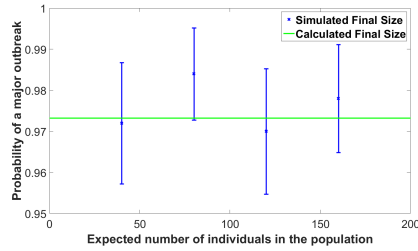
(a)  $\lambda_N = 0.45$ ,  $\lambda_H = 0.25$ ,  $\lambda_G = 0.1$ ,  $H \sim \text{Const}(4)$ ,  $D \sim \text{Poi}(4)$ . (b)  $\lambda_N = 0.45$ ,  $\lambda_H = 0.25$ ,  $\lambda_G = 0.1$ ,  $H \sim \text{Const}(4)$ ,  $D \sim \text{Poi}(4)$ .



(c)  $\lambda_N = 0.45$ ,  $\lambda_H = 0.25$ ,  $\lambda_G = 0.1$ ,  $H \sim \text{Poi}(3) + 1$ ,  $D \sim \text{Poi}(4)$ . (d)  $\lambda_N = 0.45$ ,  $\lambda_H = 0.25$ ,  $\lambda_G = 0.1$ ,  $H \sim \text{Poi}(3) + 1$ ,  $D \sim \text{Poi}(4)$ .

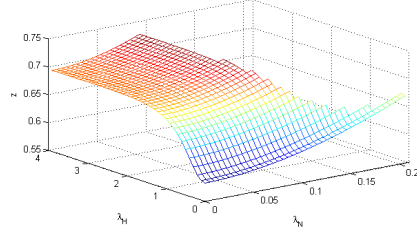


(e)  $\lambda_N = 0.45$ ,  $\lambda_H = 0.25$ ,  $\lambda_G = 0.1$ ,  $H \sim \text{Poi}(3) + 1$ ,  $D \sim \text{Geo}(1/5)$ . (f)  $\lambda_N = 0.45$ ,  $\lambda_H = 0.25$ ,  $\lambda_G = 0.1$ ,  $H \sim \text{Poi}(3) + 1$ ,  $D \sim \text{Geo}(1/5)$ .

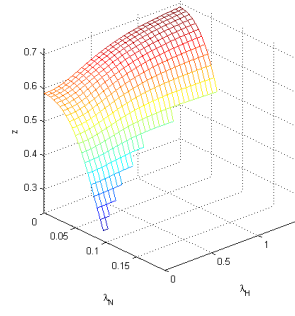


(g)  $\lambda_N = 0.45$ ,  $\lambda_H = 0.25$ ,  $\lambda_G = 0.1$ ,  $H \sim \text{Poi}(9) + 1$ ,  $D \sim \text{Poi}(4)$ . (h)  $\lambda_N = 0.45$ ,  $\lambda_H = 0.25$ ,  $\lambda_G = 0.1$ ,  $H \sim \text{Poi}(9) + 1$ ,  $D \sim \text{Poi}(4)$ .

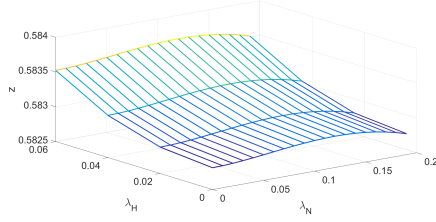
Figure 3.3: Numerical exploration of convergence of empirical estimates of  $p_{maj}$  and  $z$  in finite populations to the asymptotic final size.



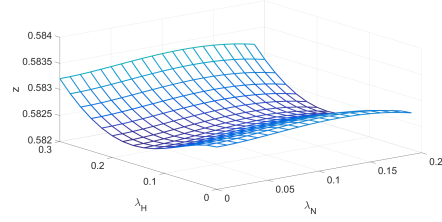
(a)  $D \sim \text{Const}(8)$  and  $\rho = [0.29, 0.35, 0.16, 0.15, 0.05]$ .



(b)  $D \sim \text{Geo}^+(1/9)$  and  $\rho = [0.29, 0.35, 0.16, 0.15, 0.05]$ .



(c)  $D \sim \text{Log}(0.62)$  and  $\rho = [0.29, 0.34, 0.16, 0.14, 0.05, 0.02]$ .



(d)  $D \sim \text{Log}(0.62)$  and  $\rho = [0.74, 0.13, 0.1, 0.03]$ .

Figure 3.4: Final size of a major outbreak,  $z$ , with  $R_0 = 1.5$  and varying infection rates  $\lambda_H$ ,  $\lambda_N$  and  $\lambda_G$ . Note the different axis (used for visibility). We discuss our choice of household size distributions in Section 4.2.

of a major outbreak in different ways. Unfortunately the interactions between these variables are complicated and difficult to investigate analytically, which is why in Chapter 4 we consider simpler models.

## 3.5 Proof of Theorem 3.1

### 3.5.1 Overview and Notation

In this section we provide a rigorous argument for the use of a two-type Galton-Watson branching process approximation to the epidemic process described in Section 3.1, and a proof of Theorem 3.1. We begin by giving a brief overview of this subsection. Firstly we define a realisation of the epidemic process on  $m$  households,  $\mathbf{E}^{(m)}$  say, viewed on a generational basis, and a realisation of an approximating branching process,  $\mathbf{Z}^{(m)}$  say. Let  $\hat{\mathbf{E}}^{(m)}$  be the total number of (type-1, type-2) infected households infected, including the initial infective, in the epidemic process  $\mathbf{E}^{(m)}$ , and let  $\hat{\mathbf{Z}}^{(m)}$  be the total progeny of the approximating branching process  $\mathbf{Z}^{(m)}$ . As  $m \rightarrow \infty$ ,  $\mathbf{Z}^{(m)}$  converges to the limiting



two-type Galton-Watson forward branching process,  $\mathbf{Z}$  say, with total progeny  $\hat{\mathbf{Z}}$ .

We show that as  $m \rightarrow \infty$ ,  $\hat{\mathbf{Z}}^{(m)}$  converges in distribution to  $\hat{\mathbf{Z}}$ . We then consider the number of previously contacted households in the population when a previously contacted household is contacted for a second time in the epidemic process, before finally giving the proof of Theorem 3.1.

The following notation holds only for Section 3.5. A household category  $\alpha = (n; d_1, d_2, \dots, d_n)$ , where  $n$  is the household size and  $d_1, d_2, \dots, d_n$  are the degrees of the  $n$  individuals in the household. Recall that the degree of an individual  $i$  is the number of half-edges emanating from  $i$ . We assume that if the household size is  $n$  then the individuals in it are labelled  $1, 2, \dots, n$  and  $d_i$  is the degree of individual  $i$ . This means that, for example, the household categories  $(2; 1, 2)$  and  $(2; 2, 1)$  are not the same. Let  $\mathcal{A}$  be the set of all possible household categories and  $\mathcal{A}_n$  be the set of all household categories with household size  $n$ . Note that, for each  $n \in \mathbb{Z}^+$ ,  $\mathcal{A}_n$  is a union of finitely many countable sets and is therefore countable. Furthermore,  $\cup_{n \in \mathbb{Z}^+} \mathcal{A}_n$  is countable and thus  $\mathcal{A}$  is countable.

### 3.5.2 Construction of the epidemic process and the approximating branching process on $m$ households

Let  $(\Omega_1, \mathcal{F}_1, \mathbf{P}_1)$  be a probability space, on which are defined the following independent quantities:

- (i) a sequence  $\mathbf{D} = (D_1, D_2, \dots)$  of independent random variables, each distributed according to the degree distribution  $D$ ;
- (ii) a sequence  $\mathbf{H} = (H_1, H_2, \dots)$  of independent random variables, each distributed according to the household size distribution  $H$ .

Let  $N^{(0)} = 0$  and for  $m = 1, 2, \dots$ , let  $N^{(m)} = \sum_{i=1}^m H_i$  be the number of individuals in the first  $m$  households. Then we construct the sequence  $\alpha = (\alpha_1, \alpha_2, \dots)$ , where  $\alpha_k \in \mathcal{A}$  for  $k = 1, 2, \dots$ , from the sequences  $\mathbf{D}$  and  $\mathbf{H}$ , with  $\alpha_k = (H_k; D_{N^{(k-1)}+1}, D_{N^{(k-1)}+2}, \dots, D_{N^{(k)}})$  for  $k = 1, 2, \dots$ .

Let  $(\Omega_2, \mathcal{F}_2, P_2)$  be a probability space, on which are defined the following mutually independent random quantities:

- (i) for every  $(\alpha, j) \in \mathcal{A} \times \mathbb{Z}^+$ , a sequence of random variables  $(\Psi_G^{(\alpha,j)}(1), \Phi_G^{(\alpha,j)}(1)), (\Psi_G^{(\alpha,j)}(2), \Phi_G^{(\alpha,j)}(2)), \dots$ , which are independent copies of the random variable  $(\Psi_G^{(\alpha,j)}, \Phi_G^{(\alpha,j)})$  defined below;
- (ii) for every  $(\alpha, j) \in \mathcal{A} \times \mathbb{Z}^+$  a sequence of random variables  $(\Psi_N^{(\alpha,j)}(1), \Phi_N^{(\alpha,j)}(1)), (\Psi_N^{(\alpha,j)}(2), \Phi_N^{(\alpha,j)}(2)), \dots$ , which are independent copies of the random variable  $(\Psi_N^{(\alpha,j)}, \Phi_N^{(\alpha,j)})$  defined below.

We also require uniformly distributed random variables defined on  $(\Omega_2, \mathcal{F}_2, P_2)$ , which we describe only informally because the detail is not important for our proofs.

The random variable  $(\Psi_G^{(\alpha,j)}, \Phi_G^{(\alpha,j)})$  describes the number of global (type-1) and network (type-2) contacts respectively made by the members of a household epidemic within a household with category  $\alpha$  in which individual  $j$  was contacted via a global contact. Similarly, the random variable  $(\Psi_N^{(\alpha,j)}, \Phi_N^{(\alpha,j)})$  describes the number of global and network contacts respectively made by the members of a household with category  $\alpha$  in which individual  $j$  was contacted via a network contact. The definitions of  $(\Psi_G^{(\alpha,j)}, \Phi_G^{(\alpha,j)})$  and  $(\Psi_N^{(\alpha,j)}, \Phi_N^{(\alpha,j)})$  are similar, so we let  $A \in \{G, N\}$  and define the random variables as follows.

Let  $G_\alpha$ , with  $\alpha = (n; d_1, d_2, \dots, d_n)$  say, be the random directed graph on the vertices  $V_\alpha = \{1, 2, \dots, n\}$  obtained as follows. For each vertex  $i \in V_\alpha$ , take independent realisations,  $I_i$  say, of the infectious period distribution  $I$ . Conditional upon  $I_1, I_2, \dots, I_n$ , for each  $i \in V_\alpha$ , we put an arc from vertex  $i$  to each other vertex in  $V_\alpha$  independently with probability  $1 - e^{-\lambda_H I_i}$ .

Fix the initial infective  $j$ . Then, given  $G_\alpha$ , with  $\alpha = (n; d_1, d_2, \dots, d_n)$ ,  $I_1, I_2, \dots, I_n$ , and vertices  $V_\alpha$ , let  $C_{AN}^{(n)}(1), C_{AN}^{(n)}(2), \dots, C_{AN}^{(n)}(n)$  be random variables, which are independent given  $I_1, I_2, \dots, I_n$ , with

$$C_{AN}^{(n)}(i) | I_1, I_2, \dots, I_n \sim \text{Bin} \left( d'_i, 1 - e^{-\lambda_N I_i} \right), \quad i = 1, 2, \dots, n,$$

where if  $A = G$ , then  $d'_i = d_i$  else if  $A = N$  then  $d'_i = d_i$  for  $i \neq j$  and  $d_j = d_j - 1$ . Let  $C_{AG}^{(n)}(1), C_{AG}^{(n)}(2), \dots, C_{AG}^{(n)}(n)$  be random variables, which are independent

given  $I_1, I_2, \dots, I_n$ , with

$$C_{AG}^{(n)}(i) | I_1, I_2, \dots, I_n \sim \text{Poi}(\lambda_G I_i), \quad i = 1, 2, \dots, n.$$

Then

$$\begin{aligned} \Psi_A^{(\alpha, j)} &= \sum_{i=1}^n \mathbb{1}_{\{j \rightsquigarrow i\}} C_{AN}^{(n)}(i), \\ \Phi_A^{(\alpha, j)} &= \sum_{i=1}^n \mathbb{1}_{\{j \rightsquigarrow i\}} C_{AG}^{(n)}(i), \end{aligned}$$

where  $j \rightsquigarrow i$  denotes the event that there is a directed path from vertex  $j$  to  $i$  in  $G_\alpha$ .

We now introduce some further notation. For  $\alpha = (n; d_1, d_2, \dots, d_n) \in \mathcal{A}$ , let  $d_\alpha = \sum_{i=1}^n d_i$  be the total degree of the household. The  $k$ th household,  $k = 1, 2, \dots$ , has household category  $\alpha_k$ , and consists of  $H_k$  individuals with total degree  $d_{\alpha_k}$ . Let  $N_D^{(m)} = \sum_{k=1}^m d_{\alpha_k}$ , be the total number of half-edges in the first  $m$  households.

The epidemic and approximating branching processes are defined on the probability space  $(\Omega, \mathcal{F}, \mathbb{P}) = (\Omega_1, \mathcal{F}_1, \mathbb{P}_1) \times (\Omega_2, \mathcal{F}_2, \mathbb{P}_2)$ . Conditional on the household category sequence  $\alpha$  and for every  $m = 1, 2, \dots$ , we now describe the construction of a branching process,  $\mathbf{Z}^{(m)} = (Z_G^{(m)}, Z_N^{(m)})$ , which approximates the early stages of the spread of the epidemic amongst households  $1, 2, \dots, m$ , and the epidemic process among  $m$  households,  $\mathbf{E}^{(m)} = (E_G^{(m)}, E_N^{(m)})$ .

First we describe the branching process  $\mathbf{Z}^{(m)}$ . Set

$$\mathbf{Z}^{(m)}(0) = (Z_G^{(m)}(0), Z_N^{(m)}(0)) = (1, 0).$$

To find the offspring of the initial individual, choose an individual uniformly at random from  $1, 2, \dots, N^{(m)}$ . Suppose that it is individual  $\varsigma$  from household  $\Delta_0 \in \{1, 2, \dots, m\}$ . Then

$$\mathbf{Z}^{(m)}(1) = (Z_G^{(m)}(1), Z_N^{(m)}(1)) = \left( \Psi_G^{(\alpha_{\Delta_0, \varsigma})}(1), \Phi_G^{(\alpha_{\Delta_0, \varsigma})}(1) \right).$$

For subsequent generations  $k \geq 2$  we continue the construction as follows. For each  $i = 1, 2, \dots, Z_N^{(m)}(k-1)$ , sample a half-edge uniformly at random from

the  $N_D^{(m)}$  half-edges in the population. Suppose this half-edge emanates from individual  $\varsigma_1$  in household  $\Delta_1$ . Set

$$\left( Z_{NG}^{(m)}(k, i), Z_{NN}^{(m)}(k, i) \right) = \left( \Psi_N^{(\alpha_{\Delta_1, \varsigma_1})}(\nu_N(\Delta_1, \varsigma_1) + 1), \Phi_N^{(\alpha_{\Delta_1, \varsigma_1})}(\nu_N(\Delta_1, \varsigma_1) + 1) \right),$$

where  $\nu_N(\Delta_1, \varsigma_1)$  is the number of times we have sampled previously from the sequence

$$\left( \Psi_N^{(\alpha_{\Delta_1, \varsigma_1})}(1), \Phi_N^{(\alpha_{\Delta_1, \varsigma_1})}(1) \right), \left( \Psi_N^{(\alpha_{\Delta_1, \varsigma_1})}(2), \Phi_N^{(\alpha_{\Delta_1, \varsigma_1})}(2) \right), \dots$$

Similarly, for each  $j = 1, 2, \dots, Z_G^{(m)}(k-1)$ , sample uniformly at random from the  $N^{(m)}$  individuals in the population. Suppose this is individual  $\varsigma_2$  in household  $\Delta_2$ . Set

$$\left( Z_{GG}^{(m)}(k, j), Z_{GN}^{(m)}(k, j) \right) = \left( \Psi_G^{(\alpha_{\Delta_2, \varsigma_2})}(\nu_G(\Delta_2, \varsigma_2) + 1), \Phi_G^{(\alpha_{\Delta_2, \varsigma_2})}(\nu_G(\Delta_2, \varsigma_2) + 1) \right),$$

where  $\nu_G(\Delta_2, \varsigma_2)$  is the number of times we have sampled previously from the sequence

$$\left( \Psi_G^{(\alpha_{\Delta_2, \varsigma_2})}(1), \Phi_G^{(\alpha_{\Delta_2, \varsigma_2})}(1) \right), \left( \Psi_G^{(\alpha_{\Delta_2, \varsigma_2})}(2), \Phi_G^{(\alpha_{\Delta_2, \varsigma_2})}(2) \right), \dots$$

Then

$$\begin{aligned} Z_G^{(m)}(k) &= \sum_{j=1}^{Z_G^{(m)}(k-1)} Z_{GG}^{(m)}(k, j) + \sum_{i=1}^{Z_N^{(m)}(k-1)} Z_{NG}^{(m)}(k, i), \\ Z_N^{(m)}(k) &= \sum_{j=1}^{Z_G^{(m)}(k-1)} Z_{GN}^{(m)}(k, j) + \sum_{i=1}^{Z_N^{(m)}(k-1)} Z_{NN}^{(m)}(k, i) \end{aligned}$$

and  $\mathbf{Z}^{(m)}(k) = (Z_G^{(m)}(k), Z_N^{(m)}(k))$ .

We construct the epidemic process  $\mathbf{E}^{(m)}$  in the same way as  $\mathbf{Z}^{(m)}$  until we sample an individual or half-edge contained within a previously chosen household. At this point the construction of the epidemic process  $\mathbf{E}^{(m)}$  can be continued, however the detail is not important for our purposes.

The branching process  $\mathbf{Z}^{(m)}$  and the epidemic process  $\mathbf{E}^{(m)}$  can be coupled by using the same  $\alpha$ ,  $(\Psi_G^{(\alpha, j)}, \Phi_G^{(\alpha, j)})$ 's,  $(\Psi_N^{(\alpha, j)}, \Phi_N^{(\alpha, j)})$ 's and uniformly random samples. The coupling breaks down when a half-edge is sampled that emanates

from a household that has been previously used in the epidemic or an individual is sampled from a household that has been previously used in the epidemic. As  $m \rightarrow \infty$ ,  $\mathbf{Z}^{(m)}$  converges to the limiting two-type Galton-Watson forward branching process, denoted by  $\mathbf{Z}$ , which uses the asymptotic distributions in its construction as opposed to the construction of  $\mathbf{Z}^{(m)}$ , which uses the empirical household size and degree distributions.

### 3.5.3 Analysis of the forward process

For  $m = 1, 2, \dots$ , let  $\hat{\mathbf{E}}^{(m)} = (\hat{E}_G^{(m)}, \hat{E}_N^{(m)})$  be the total number of (type-1, type-2) households infected in the epidemic  $\mathbf{E}^{(m)}$ , including the initial household. Similarly, let  $\hat{\mathbf{Z}}^{(m)}$  and  $\hat{\mathbf{Z}}$  be the total progeny, including the initial individual, of the branching processes  $\mathbf{Z}^{(m)}$  and  $\mathbf{Z}$  respectively.

**Lemma 3.1.**

- (i)  $\lim_{m \rightarrow \infty} \mathbb{P}(\hat{\mathbf{Z}}^{(m)} = (k, l)) = \mathbb{P}(\hat{\mathbf{Z}} = (k, l)), k, l = 0, 1, 2, \dots,$
- (ii)  $\mathbb{P}(\hat{\mathbf{Z}} = \infty) = 1 - \mathbb{P}(\hat{\mathbf{Z}} < \infty),$
- (iii)  $\lim_{m \rightarrow \infty} \mathbb{P}(\hat{\mathbf{Z}}^{(m)} = \infty) = \mathbb{P}(\hat{\mathbf{Z}} = \infty).$

*Proof.* Part (i) is proven by summing the probabilities of the finite number of sample paths of  $\hat{\mathbf{Z}}^{(m)}$  with  $\hat{\mathbf{Z}}^{(m)} = (k, l)$ ,  $k, l = 1, 2, \dots$ , and then noting that by the strong law of large numbers the empirical household size and degree distributions will almost surely converge to the asymptotic distributions and thus  $\lim_{m \rightarrow \infty} \mathbb{P}(\hat{\mathbf{Z}}^{(m)} = (k, l)) = \mathbb{P}(\hat{\mathbf{Z}} = (k, l))$  as required.

Since by our assumptions  $\mathbf{Z}$  is an irreducible, positively regular and nonsingular multi-type Galton-Watson branching process, part (ii) holds by standard branching process theory. See, for example, Mode (1971) Theorem 5.1.

Finally we consider part (iii). We assume that  $\lambda_G > 0$ , so we introduce the following embedded single-type branching processes. Let  $Y$  be the single-type (type-1) Galton-Watson process embedded in  $\mathbf{Z}$ , in which, for each type-1 individual in  $\mathbf{Z}$ , its parent in  $Y$  is given by its most recent type-1 ancestor when looking backwards in the family tree. Similarly, for  $m = 1, 2, \dots$ , let  $Y^{(m)}$  be the single-type (type-1) Galton-Watson process embedded in  $\mathbf{Z}^{(m)}$ , in which, for each type-1 individual in  $\mathbf{Z}^{(m)}$ , its parent in  $Y^{(m)}$  is given by its most recent type-1 ancestor when looking backwards in the family tree. Note that

the offspring distributions of  $Y$  and  $Y^{(m)}$  may have a mass at  $\infty$ .

Let  $\pi_Y$  and  $\pi_Y^{(m)}$  be the extinction probability of  $Y$  and  $Y^{(m)}$  respectively. Clearly  $Y$  becomes extinct if  $\mathbf{Z}$  becomes extinct. The only way  $\mathbf{Z}$  can survive given  $Y$  becomes extinct is the event  $\{\hat{Z}_G < \infty, \hat{Z}_N = \infty\}$ . However, part (ii) shows that  $P(\hat{Z}_G < \infty, \hat{Z}_N = \infty) = 0$ , so  $\mathbf{Z}$  becomes extinct if  $Y$  becomes extinct almost surely. Thus  $\pi_Y = \pi_1$ , where  $\pi_1$  is the extinction probability of  $\mathbf{Z}$  when starting with a single type-1 individual, and a similar argument yields  $\pi_Y^{(m)} = \pi_1^{(m)}$ , where  $\pi_1^{(m)}$  is the extinction probability of  $\mathbf{Z}^{(m)}$  when starting with a single type-1 individual.

We denote by the total progeny of  $Y$  and  $Y^{(m)}$  respectively. Then, as the probability that the total progeny of a branching process, with extinction probability  $\pi$ , is infinite is equal to  $1 - \pi$ , clearly, for  $m = 1, 2, \dots$ ,  $P(\hat{\mathbf{Z}}^{(m)} = \infty) = P(\hat{Y}^{(m)} = \infty)$  and  $P(\hat{\mathbf{Z}} = \infty) = P(\hat{Y} = \infty)$ . Thus we need only show that  $\lim_{m \rightarrow \infty} P(\hat{Y}^{(m)} = \infty) = P(\hat{Y} = \infty)$  to prove part (iii).

Let  $\tilde{Y}^{(m)}$  and  $\tilde{Y}$  be the offspring distribution of a single individual in the branching processes  $Y^{(m)}$  and  $Y$  respectively. Then  $\tilde{Y}^{(m)} \xrightarrow{\mathcal{D}} \tilde{Y}$  so, by Lefèvre and Utev (1999) Lemma 3.6,  $\hat{Y} \xrightarrow{\mathcal{D}} \hat{Y}^{(m)}$ . Thus, as required,

$$\lim_{m \rightarrow \infty} P(\hat{Y}^{(m)} = \infty) = P(\hat{Y} = \infty).$$

□

In order to prove the convergence of the total progeny of the epidemic process to the total progeny of the limiting branching process, we first investigate the number of previously contacted households in the population when a previously contacted household is contacted again in the epidemic process. Let  $\boldsymbol{\tau}_G^{(m)} = (\tau_{GG}^{(m)}, \tau_{GN}^{(m)})$  be the total number of (type-1, type-2) previously contacted households in the epidemic process when a member of a previously contacted household is contacted via a global infection event for the first time. Similarly, let  $\boldsymbol{\tau}_N^{(m)} = (\tau_{NG}^{(m)}, \tau_{NN}^{(m)})$  be the total number of (type-1, type-2) contacted households in the epidemic process when a member of a previously contacted household is contacted by a network infection event for the first time. Let  $\chi_G^{(m)}(i)$  be the household containing the individual chosen at the  $i$ th global infection event which contains  $\hat{\chi}_G^{(m)}(i)$  individuals,  $\bar{\chi}_G^{(m)}(i)$  half-edges and occurs in generation  $\xi_G^{(m)}(i)$ . Similarly, let  $\chi_N^{(m)}(i)$  be the household containing the half-edge chosen

at the  $i$ th network infection event which contains  $\hat{\chi}_N^{(m)}(i)$  individuals,  $\bar{\chi}_N^{(m)}(i)$  half-edges and occurs in generation  $\xi_N^{(m)}(i)$ . We now give bounds on the expected value of the quantities  $\hat{\chi}_G^{(m)}(i)$ ,  $\bar{\chi}_G^{(m)}(i)$  and  $\hat{\chi}_N^{(m)}(i)$ . The random variable  $\hat{\chi}_N^{(m)}(i)$  is considered separately, in the proof of Lemma 3.3.

**Proposition 3.3.** *For  $i = 1, 2, \dots, m$ ,*

$$\mathbb{E} \left[ \hat{\chi}_G^{(m)}(i) \right] \leq \mathbb{E} \left[ H^2 \right].$$

*Proof.* The probability that an individual chosen uniformly at random from the population is in a household of size  $n$ , conditioned on the household category sequence  $\alpha$ , is given by  $\sum_{i=1}^m H_i \mathbf{1}_{\{H_i=n\}} / \sum_{i=1}^m H_i$ . Recalling that  $H_i$ ,  $i = 1, 2, \dots, m$ , are independent and identically distributed copies of the random variable  $H$  and that households contain at least 1 individual, so  $N^{(m)} = \sum_{i=1}^m H_i \geq m$ , yields

$$\begin{aligned} \mathbb{E} \left[ \hat{\chi}_G^{(m)}(i) \right] &= \mathbb{E} \left[ \mathbb{E} \left[ \hat{\chi}_G^{(m)}(i) \mid \alpha \right] \right] \\ &= \mathbb{E} \left[ \sum_{n=1}^{\infty} n \frac{\sum_{i=1}^m H_i \mathbf{1}_{\{H_i=n\}}}{\sum_{i=1}^m H_i} \right] \\ &\leq \mathbb{E} \left[ \frac{\sum_{i=1}^m H_i \sum_{n=1}^{\infty} n \mathbf{1}_{\{H_i=n\}}}{m} \right] \\ &= \mathbb{E} \left[ \frac{\sum_{i=1}^m H_i^2}{m} \right] \\ &= \mathbb{E} \left[ H^2 \right]. \end{aligned}$$

□

**Proposition 3.4.** *For  $i = 1, 2, \dots, m$ ,*

$$\mathbb{E} \left[ \hat{\chi}_N^{(m)}(i) \right] \leq \mathbb{E} \left[ H^2 \right].$$

*Proof.* Each individual in the population has been assigned half-edges independently of the individual's household size, and these half-edges are then paired uniformly at random. Therefore the probability that an individual contacted by a network infection event is in a household of size  $n$  is equal to the probability that an individual chosen uniformly at random from the population is in a household of size  $n$ , so, for  $i = 1, 2, \dots, m$ ,

$$\mathbb{E} \left[ \hat{\chi}_N^{(m)}(i) \right] = \mathbb{E} \left[ \hat{\chi}_G^{(m)}(i) \right] \leq \mathbb{E} \left[ H^2 \right].$$

□

**Proposition 3.5.** For  $i = 1, 2, \dots, m$ ,

$$\mathbb{E} [\bar{\chi}_G^{(m)}(i)] \leq \mathbb{E} [H^2] \mu_D.$$

*Proof.* Recall that, for  $i = 1, 2, \dots, m$ , a household contacted by a global infection event contains  $\hat{\chi}_G^{(m)}(i)$  individuals, each of whom is independently given  $D_j$  half-edges,  $j = 1, 2, \dots, \hat{\chi}_G^{(m)}(i)$ , where  $D_j \stackrel{\mathcal{D}}{=} D$ . Therefore we find that  $\mathbb{E} [\bar{\chi}_G^{(m)}(i)] = \mathbb{E} [\sum_{j=1}^{\hat{\chi}_G^{(m)}(i)} D_j]$ , and applying Proposition 3.3 yields

$$\begin{aligned} \mathbb{E} [\bar{\chi}_G^{(m)}(i)] &= \mathbb{E} \left[ \sum_{j=1}^{\hat{\chi}_G^{(m)}(i)} D_j \right] \\ &= \mathbb{E} \left[ \mathbb{E} \left[ \sum_{j=1}^{\hat{\chi}_G^{(m)}(i)} D_j \middle| \hat{\chi}_G^{(m)}(i) \right] \right] \\ &= \mathbb{E} [\hat{\chi}_G^{(m)}(i)] \mu_D \\ &\leq \mathbb{E} [H^2] \mu_D. \end{aligned}$$

□

Lemma 3.2 states that if  $\lim_{m \rightarrow \infty} |\mathbf{g}(m)|^2/m = 0$  then  $\mathbb{P} (\tau_{\mathbf{G}}^{(m)} \leq \mathbf{g}(m)) \rightarrow 0$  as  $m \rightarrow \infty$ , where  $\tau_{\mathbf{G}}^{(m)}$  is the total number of previously contacted households in the epidemic process when a member of a previously contacted household is contacted via a global infection event for the first time.

**Lemma 3.2.** Let  $\mathbf{g}(m) = (g_1(m), g_2(m))$  be an integer-valued function that satisfies

$$\lim_{m \rightarrow \infty} |\mathbf{g}(m)|^2/m = 0. \quad (3.37)$$

Then

$$\mathbb{P} (\tau_{\mathbf{G}}^{(m)} \leq \mathbf{g}(m)) \rightarrow 0 \text{ as } m \rightarrow \infty.$$

*Proof.* Before proceeding, we recall that by our notation,

$$\mathbb{P} (\tau_{\mathbf{G}}^{(m)} \leq \mathbf{g}(m)) = \mathbb{P} (\{\tau_{GG}^{(m)} \leq g_1(m)\} \cap \{\tau_{GN}^{(m)} \leq g_2(m)\}).$$

For the event  $\{\tau_{\mathbf{G}}^{(m)} \leq \mathbf{g}(m)\}$  to occur, there must be at least one global infection event within a household that has already had a global or network infection event within the first  $g_1(m)$  global infection events and  $g_2(m)$  network infection events.



Note that this is similar to the birthday problem. We say that a match occurs between two infection events if they both occur in the same household. Let  $M_{GG}^{(m)}(\mathbf{g}(m))$  be the number of matches among the first  $g_1(m)$  global infection events and let  $M_{GN}^{(m)}(\mathbf{g}(m))$  be the number of matches between the first  $g_1(m)$  global infection events and the first  $g_2(m)$  network infection events when the network infection occurred before the global infection event. Then

$$M_{GG}^{(m)}(\mathbf{g}(m)) = \sum_{i=1}^{g_1(m)-1} \sum_{j=i+1}^{g_1(m)} \mathbb{1}_{\{\chi_G^{(m)}(i) = \chi_G^{(m)}(j)\}}, \quad (3.38)$$

$$M_{GN}^{(m)}(\mathbf{g}(m)) = \sum_{i=1}^{g_1(m)} \sum_{j=1}^{g_2(m)} \mathbb{1}_{\{\chi_G^{(m)}(i) = \chi_N^{(m)}(j)\}} \mathbb{1}_{\{\xi_G^{(m)}(i) > \xi_N^{(m)}(j)\}}. \quad (3.39)$$

Note that  $M_{GG}^{(m)}(\mathbf{g}(m))$  is the number of times a global infection event occurs in a household that has already had a global infection event, and  $M_{GN}^{(m)}(\mathbf{g}(m))$  is the number of times a global infection event occurs in a household that has already had a network infection event, within the first  $g_1(m)$  global infection events and the first  $g_2(m)$  network infection events. Therefore

$$\{\tau_G^{(m)} \leq \mathbf{g}(m)\} = \{M_{GG}^{(m)}(\mathbf{g}(m)) + M_{GN}^{(m)}(\mathbf{g}(m)) \geq 1\},$$

and, applying Markov's inequality,

$$\begin{aligned} \mathbb{P}(\tau_G^{(m)} \leq \mathbf{g}(m)) &= \mathbb{P}(M_{GG}^{(m)}(\mathbf{g}(m)) + M_{GN}^{(m)}(\mathbf{g}(m)) \geq 1) \\ &\leq \mathbb{P}(M_{GG}^{(m)}(\mathbf{g}(m)) \geq 1) + \mathbb{P}(M_{GN}^{(m)}(\mathbf{g}(m)) \geq 1) \\ &\leq \mathbb{E}[M_{GG}^{(m)}(\mathbf{g}(m))] + \mathbb{E}[M_{GN}^{(m)}(\mathbf{g}(m))]. \end{aligned} \quad (3.40)$$

Recall that the recipient of a global infection event is chosen uniformly at random from all  $N^{(m)} \geq m$  individuals in the population. Then, applying

Proposition 3.3 to the expectation of equation (3.38) yields

$$\begin{aligned}
\mathbb{E} \left[ M_{GG}^{(m)}(\mathbf{g}(m)) \right] &= \sum_{i=1}^{g_1(m)-1} \sum_{j=i+1}^{g_1(m)} \mathbb{P} \left( \chi_G^{(m)}(i) = \chi_G^{(m)}(j) \right) \\
&= \sum_{i=1}^{g_1(m)-1} \sum_{j=i+1}^{g_1(m)} \mathbb{E} \left[ \mathbb{P} \left( \chi_G^{(m)}(i) = \chi_G^{(m)}(j) \middle| \hat{\chi}_G^{(m)}(i), N^{(m)} \right) \right] \\
&= \sum_{i=1}^{g_1(m)-1} \sum_{j=i+1}^{g_1(m)} \mathbb{E} \left[ \frac{\hat{\chi}_G^{(m)}(i)}{N^{(m)}} \right] \\
&\leq \sum_{i=1}^{g_1(m)-1} \sum_{j=i+1}^{g_1(m)} \frac{\mathbb{E} [H^2]}{m} \\
&= \binom{g_1(m)}{2} \frac{\mathbb{E} [H^2]}{m} \\
&\leq (g_1(m))^2 \frac{\mathbb{E} [H^2]}{m}.
\end{aligned} \tag{3.41}$$

Similarly, applying Proposition 3.4 to the expectation of equation (3.39) and recalling that  $\xi_G^{(m)}(i)$  and  $\xi_N^{(m)}(i)$  are the generations in which the  $i$ th global or network infection event occurs yields

$$\begin{aligned}
\mathbb{E} \left[ M_{GN}^{(m)}(\mathbf{g}(m)) \right] &= \sum_{i=1}^{g_1(m)} \sum_{j=1}^{g_2(m)} \mathbb{P} \left( \chi_G^{(m)}(i) = \chi_N^{(m)}(j), \xi_G^{(m)}(i) > \xi_N^{(m)}(j) \right) \\
&\leq \sum_{i=1}^{g_1(m)} \sum_{j=1}^{g_2(m)} \mathbb{P} \left( \chi_G^{(m)}(i) = \chi_N^{(m)}(j) \middle| \xi_G^{(m)}(i) > \xi_N^{(m)}(j) \right) \\
&= \sum_{i=1}^{g_1(m)} \sum_{j=1}^{g_2(m)} \mathbb{E} \left[ \mathbb{P} \left( \chi_G^{(m)}(i) = \chi_N^{(m)}(j) \middle| \hat{\chi}_N^{(m)}(j), N^{(m)}, \xi_G^{(m)}(i) > \xi_N^{(m)}(j) \right) \right] \\
&= \sum_{i=1}^{g_1(m)} \sum_{j=1}^{g_2(m)} \mathbb{E} \left[ \frac{\hat{\chi}_N^{(m)}(j)}{N^{(m)}} \right] \\
&\leq \sum_{i=1}^{g_1(m)} \sum_{j=1}^{g_2(m)} \frac{\mathbb{E} [H^2]}{m} \\
&\leq g_1(m)g_2(m) \frac{\mathbb{E} [H^2]}{m}.
\end{aligned} \tag{3.42}$$

Substituting inequalities (3.41) and (3.42) into inequality (3.40) yields

$$\begin{aligned} \mathbb{P} \left( \tau_{\mathbf{G}}^{(m)} \leq \mathbf{g}(m) \right) &\leq (g_1(m))^2 \frac{\mathbb{E}[H^2]}{m} + g_1(m)g_2(m) \frac{\mathbb{E}[H^2]}{m} \\ &= g_1(m) [g_1(m) + g_2(m)] \frac{\mathbb{E}[H^2]}{m}. \end{aligned}$$

Applying the assumption given in equation (3.37) and the moment condition  $\sigma_H^2 < \infty$  yields  $\lim_{m \rightarrow \infty} \mathbb{P} \left( \tau_{\mathbf{G}}^{(m)} \leq \mathbf{g}(m) \right) = 0$  as required.  $\square$

Before giving a similar result for  $\tau_{\mathbf{N}}^{(m)}$ , we briefly state Chebyshev's inequality as a proposition. See, for example, Tucker (1967) Section 2.4 for a proof.

**Proposition 3.6** (Chebyshev's inequality). *Let  $X$  be a random variable with  $\sigma_X^2 < \infty$ . Then, for every  $\epsilon > 0$ ,*

$$\mathbb{P} (|X - \mathbb{E}[X]| > \epsilon) \leq \frac{\sigma_X^2}{\epsilon^2}. \quad (3.43)$$

The proof of Lemma 3.3 proceeds similarly to Lemma 3.2, although considers the number of half-edges in the population rather than the number of individuals.

**Lemma 3.3.** *Let  $\mathbf{g}(m) = (g_1(m), g_2(m))$  be an integer-valued function that satisfies*

$$\lim_{m \rightarrow \infty} |\mathbf{g}(m)|^2 / m = 0. \quad (3.44)$$

*Then*

$$\mathbb{P} \left( \tau_{\mathbf{N}}^{(m)} \leq \mathbf{g}(m) \right) \rightarrow 0 \text{ as } m \rightarrow \infty.$$

*Proof.* For the event  $\left\{ \tau_{\mathbf{N}}^{(m)} \leq \mathbf{g}(m) \right\}$  to occur, there must be at least one network contact infection event occurring in a household that has already had a global or network infection event within the first  $g_1(m)$  global infection events and  $g_2(m)$  network infection events. Let  $M_{NN}^{(m)}(\mathbf{g}(m))$  be the number of matches among the first  $g_2(m)$  network infection events and let  $M_{NG}^{(m)}(\mathbf{g}(m))$  be the number of matches between the first  $g_2(m)$  network infection events and the first  $g_1(m)$  global infection events when the global infections occurred before the network infection event. Then

$$M_{NN}^{(m)}(\mathbf{g}(m)) = \sum_{i=1}^{g_2(m)-1} \sum_{j=i+1}^{g_2(m)} \mathbb{1}_{\left\{ \chi_N^{(m)}(i) = \chi_N^{(m)}(j) \right\}}, \quad (3.45)$$

$$M_{NG}^{(m)}(\mathbf{g}(m)) = \sum_{i=1}^{g_2(m)} \sum_{j=1}^{g_1(m)} \mathbb{1}_{\left\{ \chi_N^{(m)}(i) = \chi_G^{(m)}(j) \right\}} \mathbb{1}_{\left\{ \xi_N^{(m)}(i) \geq \xi_G^{(m)}(j) \right\}}. \quad (3.46)$$

Note that  $M_{NN}^{(m)}(\mathbf{g}(m))$  is the number of times a network infection event occurs in a household that has already had a network infection event and  $M_{NG}^{(m)}(\mathbf{g}(m))$  is the number of times a network infection event occurs in a household that has already had a global infection event, within the first  $g_1(m)$  global infection events and the first  $g_2(m)$  network infection events. Therefore

$$\{\tau_N^{(m)} \leq \mathbf{g}(m)\} = \{M_{NN}^{(m)}(\mathbf{g}(m)) + M_{NG}^{(m)}(\mathbf{g}(m)) \geq 1\},$$

and, applying Markov's inequality,

$$\begin{aligned} \mathbb{P}(\tau_N^{(m)} \leq \mathbf{g}(m)) &= \mathbb{P}(M_{NN}^{(m)}(\mathbf{g}(m)) + M_{NG}^{(m)}(\mathbf{g}(m)) \geq 1) \\ &\leq \mathbb{P}(M_{NN}^{(m)}(\mathbf{g}(m)) \geq 1) + \mathbb{P}(M_{NG}^{(m)}(\mathbf{g}(m)) \geq 1) \\ &\leq \mathbb{E}[M_{NN}^{(m)}(\mathbf{g}(m))] + \mathbb{E}[M_{NG}^{(m)}(\mathbf{g}(m))]. \end{aligned} \quad (3.47)$$

Recall that  $N_D^{(m)} = \sum_{i=1}^m d_{\alpha_i}$  is the number of half-edges in the population of  $m$  households. For  $i = 1, 2, \dots, m$ ,  $d_{\alpha_i}$  are independent and identically distributed copies of some random variable,  $T_D$  say, so  $\mathbb{E}[N_D^{(m)}] = m\mathbb{E}[T_D]$  and  $\text{Var}[N_D^{(m)}] = m\text{Var}[T_D]$ . Note that  $T_D \stackrel{\mathcal{D}}{=} \sum_{j=1}^H D_j$ , where  $D_j$  are independent copies of the degree distribution  $D$ . So  $\mathbb{E}[T_D] = \mu_H \mu_D$  and, applying the conditional variance formula,

$$\begin{aligned} \text{Var}[T_D] &= \mathbb{E}[\text{Var}[T_D|H]] + \text{Var}[\mathbb{E}[T_D|H]] \\ &= \mathbb{E}\left[\text{Var}\left[\sum_{j=1}^H D_j \middle| H\right]\right] + \text{Var}\left[\mathbb{E}\left[\sum_{j=1}^H D_j \middle| H\right]\right] \\ &= \mathbb{E}[H\text{Var}[D]] + \text{Var}[H\mu_D] \\ &= \mu_H \sigma_D^2 + \sigma_H^2 \mu_D^2. \end{aligned} \quad (3.48)$$

So  $\text{Var}[N_D^{(m)}] = m(\mu_H \sigma_D^2 + \mu_D^2 \sigma_H^2) < \infty$  since we assume  $\sigma_H^2 < \infty$  and  $\sigma_D^2 < \infty$ . So, by Chebyshev's inequality,

$$\begin{aligned} \mathbb{P}\left(N_D^{(m)} < \frac{m\mu_D\mu_H}{2}\right) &\leq \mathbb{P}\left(|N_D^{(m)} - m\mu_D\mu_H| > \frac{m\mu_D\mu_H}{2}\right) \\ &\leq \frac{4\text{Var}[N_D^{(m)}]}{(m\mu_D\mu_H)^2} \\ &= \frac{4m(\mu_H \sigma_D^2 + \mu_D^2 \sigma_H^2)}{m^2 \mu_H^2 \mu_D^2} \\ &= \frac{4(\mu_H \sigma_D^2 + \mu_D^2 \sigma_H^2)}{m \mu_H^2 \mu_D^2}. \end{aligned} \quad (3.49)$$

The probability that a half-edge chosen uniformly at random from the population is in a household with total household degree  $k$ , conditioned on the household category sequence  $\alpha$ , is given by  $\sum_{i=1}^m d_{\alpha_i} \mathbf{1}_{\{d_{\alpha_i}=k\}} / N_D^{(m)}$ . Recalling that  $d_{\alpha_i}$ ,  $i = 1, 2, \dots, m$ , are independent and identically distributed copies of the random variable  $T_D$  and conditioning on  $L_m = \left\{ N_D^{(m)} \geq \frac{m\mu_D\mu_H}{2} \right\}$  yields

$$\begin{aligned}
\mathbb{E} \left[ \bar{\chi}_i^{N(m)} \middle| L_m \right] &= \mathbb{E} \left[ \mathbb{E} \left[ \bar{\chi}_i^{N(m)} \middle| L_m, \alpha \right] \middle| L_m \right] \\
&= \mathbb{E} \left[ \sum_{k=1}^{\infty} k \frac{\sum_{i=1}^m d_{\alpha_i} \mathbf{1}_{\{d_{\alpha_i}=k\}}}{N_D^{(m)}} \middle| L_m \right] \\
&\leq \mathbb{E} \left[ \sum_{k=1}^{\infty} k \frac{\sum_{i=1}^m d_{\alpha_i} \mathbf{1}_{\{d_{\alpha_i}=k\}}}{\frac{m\mu_D\mu_H}{2}} \middle| L_m \right] \\
&= \mathbb{E} \left[ \frac{\sum_{i=1}^m d_{\alpha_i} \sum_{k=1}^{\infty} k \mathbf{1}_{\{d_{\alpha_i}=k\}}}{\frac{m\mu_D\mu_H}{2}} \middle| L_m \right] \\
&= \frac{2}{m\mu_D\mu_H} \mathbb{E} \left[ \sum_{i=1}^m d_{\alpha_i}^2 \middle| L_m \right] \\
&= \frac{2m}{m\mu_D\mu_H} \mathbb{E} \left[ T_D^2 \middle| L_m \right] \\
&= \frac{2}{\mu_D\mu_H} \mathbb{E} \left[ T_D^2 \middle| L_m \right] \tag{3.50}
\end{aligned}$$

It follows from equation (3.45), by conditioning on whether  $L_m$  occurs, that

$$\begin{aligned}
&\mathbb{E} \left[ M_{NN}^{(m)}(\mathbf{g}(m)) \right] \\
&= \sum_{i=1}^{g_2(m)-1} \sum_{j=i+1}^{g_2(m)} \mathbb{P} \left( \chi_N^{(m)}(i) = \chi_N^{(m)}(j) \right) \\
&= \sum_{i=1}^{g_2(m)-1} \sum_{j=i+1}^{g_2(m)} \mathbb{P} \left( \chi_N^{(m)}(i) = \chi_N^{(m)}(j), L_m^c \right) + \mathbb{P} \left( \chi_N^{(m)}(i) = \chi_N^{(m)}(j), L_m \right) \\
&\leq \sum_{i=1}^{g_2(m)-1} \sum_{j=i+1}^{g_2(m)} \mathbb{P} \left( L_m^c \right) + \mathbb{P} \left( \chi_N^{(m)}(i) = \chi_N^{(m)}(j), L_m \right) \\
&\leq \binom{g_2(m)}{2} \mathbb{P} \left( L_m^c \right) + \sum_{i=1}^{g_2(m)-1} \sum_{j=i+1}^{g_2(m)} \mathbb{P} \left( \chi_N^{(m)}(i) = \chi_N^{(m)}(j) \middle| L_m \right) \mathbb{P} \left( L_m \right). \tag{3.51}
\end{aligned}$$

For  $i = 1, 2, \dots, m$ , the recipient of a network infection event is chosen uniformly at random from all  $N_D^{(m)}$  half-edges in the population, of which there are  $\bar{\chi}_N^{(m)}(i)$  in the  $i$ th household contacted by a network infection event.

Therefore

$$\mathbb{E} \left[ \mathbb{P} \left( \chi_N^{(m)}(i) = \chi_N^{(m)}(j) \middle| \chi_N^{(m)}(i), N_D^{(m)} \right) \right] = \mathbb{E} \left[ \frac{\chi_N^{(m)}(i)}{N_D^{(m)}} \right],$$

and so, also conditioning on  $L_m$ ,

$$\begin{aligned} \mathbb{P} \left( \chi_N^{(m)}(i) = \chi_N^{(m)}(j) \middle| L_m \right) &= \mathbb{E} \left[ \mathbb{P} \left( \chi_N^{(m)}(i) = \chi_N^{(m)}(j) \middle| L_m, \chi_N^{(m)}(i), N_D^{(m)} \right) \right] \\ &= \mathbb{E} \left[ \frac{\chi_N^{(m)}(i)}{N_D^{(m)}} \middle| L_m \right] \\ &\leq \mathbb{E} \left[ \frac{\chi_N^{(m)}(i)}{\frac{m\mu_D\mu_H}{2}} \middle| L_m \right] \\ &= \frac{2}{m\mu_D\mu_H} \mathbb{E} \left[ \chi_N^{(m)}(i) \middle| L_m \right]. \end{aligned} \quad (3.52)$$

Substituting equation (3.52) and inequalities (3.49) and (3.50) into inequality (3.51) yields

$$\begin{aligned} &\mathbb{E} \left[ M_{NN}^{(m)}(\mathbf{g}(m)) \right] \\ &\leq (g_2(m))^2 \mathbb{P}(L_m^c) + \sum_{i=1}^{g_2(m)-1} \sum_{j=i+1}^{g_2(m)} \frac{2}{m\mu_H\mu_D} \mathbb{E} \left[ \bar{\chi}_N^{(m)}(i) \middle| L_m \right] \mathbb{P}(L_m) \\ &= (g_2(m))^2 \mathbb{P}(L_m^c) + \sum_{i=1}^{g_2(m)-1} \sum_{j=i+1}^{g_2(m)} \frac{4}{m\mu_H^2\mu_D^2} \mathbb{E} \left[ T_D^2 \middle| L_m \right] \mathbb{P}(L_m) \\ &\leq (g_2(m))^2 \mathbb{P}(L_m^c) + \sum_{i=1}^{g_2(m)-1} \sum_{j=i+1}^{g_2(m)} \frac{4}{m\mu_H^2\mu_D^2} \mathbb{E} \left[ T_D^2 \right] \\ &= (g_2(m))^2 \mathbb{P}(L_m^c) + \binom{g_2(m)}{2} \frac{4}{m\mu_H^2\mu_D^2} \mathbb{E} \left[ T_D^2 \right] \\ &\leq (g_2(m))^2 \frac{4(\mu_H\sigma_D^2 + \mu_D^2\sigma_H^2)}{m\mu_H^2\mu_D^2} + (g_2(m))^2 \frac{4}{m\mu_H^2\mu_D^2} \mathbb{E} \left[ T_D^2 \right] \\ &= (g_2(m))^2 \frac{4}{m\mu_H^2\mu_D^2} \left[ \mu_H\sigma_D^2 + \mu_D^2\sigma_H^2 + \mathbb{E} \left[ T_D^2 \right] \right] \\ &= (g_2(m))^2 \frac{4}{m\mu_H^2\mu_D^2} \left[ 2\mu_H\sigma_D^2 + \mu_D^2\sigma_H^2 + \mathbb{E} \left[ H^2 \right] \mu_D^2 \right]. \end{aligned} \quad (3.53)$$

Similarly, it follows from equation (3.46), by applying definition of conditional

probability and the conditioning on whether  $L_m$  occurs,

$$\begin{aligned}
& \mathbb{E} \left[ M_{GN}^{(m)}(\mathbf{g}(m)) \right] \\
&= \sum_{i=1}^{g_1(m)} \sum_{j=1}^{g_2(m)} \mathbb{P} \left( \chi_N^{(m)}(i) = \chi_G^{(m)}(j), \xi_N^{(m)}(i) \geq \xi_G^{(m)}(j) \right) \\
&\leq \sum_{i=1}^{g_1(m)} \sum_{j=1}^{g_2(m)} \mathbb{P} \left( \chi_N^{(m)}(i) = \chi_G^{(m)}(j) \mid \xi_N^{(m)}(i) \geq \xi_G^{(m)}(j) \right) \\
&= \sum_{i=1}^{g_1(m)} \sum_{j=1}^{g_2(m)} \mathbb{P} \left( \chi_N^{(m)}(i) = \chi_G^{(m)}(j), L_m \mid \xi_N^{(m)}(i) \geq \xi_G^{(m)}(j) \right) \\
&\quad + \mathbb{P} \left( \chi_N^{(m)}(i) = \chi_G^{(m)}(j), L_m^c \mid \xi_N^{(m)}(i) \geq \xi_G^{(m)}(j) \right) \\
&\leq \sum_{i=1}^{g_1(m)} \sum_{j=1}^{g_2(m)} \mathbb{P} \left( \chi_N^{(m)}(i) = \chi_G^{(m)}(j), L_m \mid \xi_N^{(m)}(i) \geq \xi_G^{(m)}(j) \right) + \mathbb{P}(L_m^c) \\
&\leq g_1(m)g_2(m)\mathbb{P}(L_m^c) \\
&\quad + \sum_{i=1}^{g_1(m)} \sum_{j=1}^{g_2(m)} \mathbb{P} \left( \chi_N^{(m)}(i) = \chi_G^{(m)}(j) \mid L_m, \xi_N^{(m)}(i) \geq \xi_G^{(m)}(j) \right) \mathbb{P}(L_m). \quad (3.54)
\end{aligned}$$

For  $j = 1, 2, \dots, m$ , the recipient of a network infection event is chosen uniformly at random from all  $N_D^{(m)}$  half-edges in the population, of which there are  $\bar{\chi}_G^{(m)}(j)$  in the  $j$ th household contacted by a network infection event. Therefore,

$$\mathbb{P} \left( \chi_N^{(m)}(i) = \chi_G^{(m)}(j) \mid \xi_N^{(m)}(i) \geq \xi_G^{(m)}(j) \right) = \mathbb{E} \left[ \frac{\bar{\chi}_G^{(m)}(j)}{N_D^{(m)}} \right],$$

and so, also conditioning on  $L_m$ ,

$$\begin{aligned}
& \mathbb{P} \left( \chi_N^{(m)}(i) = \chi_G^{(m)}(j) \mid L_m, \xi_N^{(m)}(i) \geq \xi_G^{(m)}(j) \right) \\
&= \mathbb{E} \left[ \mathbb{P} \left( \chi_N^{(m)}(i) = \chi_G^{(m)}(j) \mid L_m, \bar{\chi}_N^{(m)}(i), N_D^{(m)}, \xi_N^{(m)}(i) \geq \xi_G^{(m)}(j) \right) \right] \\
&= \mathbb{E} \left[ \frac{\bar{\chi}_G^{(m)}(j)}{N_D^{(m)}} \mid L_m \right] \\
&\leq \mathbb{E} \left[ \frac{\bar{\chi}_G^{(m)}(j)}{\frac{m\mu_D\mu_H}{2}} \mid L_m \right] \\
&= \frac{2}{m\mu_D\mu_H} \mathbb{E} \left[ \bar{\chi}_G^{(m)}(j) \mid L_m \right]. \quad (3.55)
\end{aligned}$$

Substituting Proposition (3.5) and inequalities (3.49) and (3.55) into inequality

(3.54) yields

$$\begin{aligned}
& \mathbb{E} \left[ M_{GN}^{(m)}(\mathbf{g}(m)) \right] \\
& \leq g_1(m)g_2(m)\mathbb{P}(L_m^c) + \sum_{i=1}^{g_1(m)} \sum_{j=1}^{g_2(m)} \frac{2}{m\mu_D\mu_H} \mathbb{E} \left[ \bar{\chi}_G^{(m)}(j) | L_m \right] \mathbb{P}(L_m) \\
& \leq g_1(m)g_2(m)\mathbb{P}(L_m^c) + \sum_{i=1}^{g_1(m)} \sum_{j=1}^{g_2(m)} \frac{2\mathbb{E}[H^2]\mu_D}{m\mu_H\mu_D} \\
& \leq g_1(m)g_2(m) \frac{4(\mu_H\sigma_D^2 + \mu_D^2\sigma_H^2)}{m\mu_H^2\mu_D^2} + g_1(m)g_2(m) \frac{2\mathbb{E}[H^2]\mu_D}{m\mu_H\mu_D} \\
& = g_1(m)g_2(m) \frac{2}{m\mu_H\mu_D} \left[ \frac{2(\mu_H\sigma_D^2 + \mu_D^2\sigma_H^2)}{\mu_H\mu_D} + \mathbb{E}[H^2]\mu_D \right]. \tag{3.56}
\end{aligned}$$

Substituting inequalities (3.53) and (3.56) into inequality (3.47) yields

$$\begin{aligned}
& \mathbb{P} \left( \tau_N^{(m)} \leq \mathbf{g}(m) \right) \\
& \leq (g_2(m))^2 \frac{4}{m\mu_H^2\mu_D^2} \left[ 2\mu_H\sigma_D^2 + \mu_D^2\sigma_H^2 + \mathbb{E}[H^2]\mu_D^2 \right] \\
& \quad + g_1(m)g_2(m) \frac{2}{m\mu_H\mu_D} \left[ \frac{2(\mu_H\sigma_D^2 + \mu_D^2\sigma_H^2)}{\mu_H\mu_D} + \mathbb{E}[H^2]\mu_D \right].
\end{aligned}$$

Thus applying the assumption given in equation (3.44) and the moment restriction  $\sigma_H^2 < \infty$  yields  $\lim_{m \rightarrow \infty} \mathbb{P} \left( \tau_N^{(m)} \leq \mathbf{g}(m) \right) = 0$  as required.  $\square$

The following Lemma is used in the proof of Theorem 3.1, as it relates the total progeny of  $\hat{\mathbf{Z}}^{(m)}$  to the total progeny of  $\mathbf{Z}$ . For the rest of this section we say  $\mathbf{m}^\gamma = (\lfloor m^\gamma \rfloor, \lfloor m^\gamma \rfloor)$ .

**Lemma 3.4.** *For fixed  $\gamma \in (0, 1/2)$ ,*

$$\lim_{m \rightarrow \infty} \mathbb{P} \left( \hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma \right) = \mathbb{P} \left( \hat{\mathbf{Z}} < \infty \right)$$

*Proof.* For any  $k \in \mathbb{N}$

$$\liminf_{m \rightarrow \infty} \mathbb{P} \left( \hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma \right) \geq \liminf_{m \rightarrow \infty} \mathbb{P} \left( \hat{\mathbf{Z}}^{(m)} < (k, k) \right) = \mathbb{P} \left( \hat{\mathbf{Z}} < (k, k) \right). \tag{3.57}$$

Clearly  $\mathbb{P} \left( \hat{\mathbf{Z}} < (k, k) \right) \rightarrow \mathbb{P} \left( \hat{\mathbf{Z}} < \infty \right)$  as  $k \rightarrow \infty$ , so letting  $k \rightarrow \infty$  in equation (3.57) yields

$$\liminf_{m \rightarrow \infty} \mathbb{P} \left( \hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma \right) \geq \mathbb{P} \left( \hat{\mathbf{Z}} < \infty \right). \tag{3.58}$$



Also, applying Lemma 3.1(ii),

$$\limsup_{m \rightarrow \infty} \mathbb{P} \left( \hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma \right) \leq \limsup_{m \rightarrow \infty} \mathbb{P} \left( \hat{\mathbf{Z}}^{(m)} < \infty \right) = \mathbb{P} \left( \hat{\mathbf{Z}} < \infty \right).$$

Thus

$$\limsup_{m \rightarrow \infty} \mathbb{P} \left( \hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma \right) \leq \mathbb{P} \left( \hat{\mathbf{Z}} < \infty \right),$$

which, with equation (3.58), yields the required result.  $\square$

We are now ready to give the proof of Theorem 3.1, reiterated below.

**Theorem.**

(i) For  $k, l = 0, 1, \dots$ ,

$$\lim_{m \rightarrow \infty} \left| \mathbb{P} \left( \hat{\mathbf{E}}^{(m)} = (k, l) \right) - \mathbb{P} \left( \hat{\mathbf{Z}} = (k, l) \right) \right| = 0. \quad (3.59)$$

(ii) For  $\gamma \in (0, 1/2)$ ,

$$\lim_{m \rightarrow \infty} \left| \mathbb{P} \left( \hat{\mathbf{E}}^{(m)} < \mathbf{m}^\gamma \right) - \mathbb{P} \left( \hat{\mathbf{Z}} < \infty \right) \right| = 0. \quad (3.60)$$

*Proof.* We begin by considering part (i). Note that, by the triangle inequality,

$$\begin{aligned} \left| \mathbb{P} \left( \hat{\mathbf{E}}^{(m)} = (k, l) \right) - \mathbb{P} \left( \hat{\mathbf{Z}} = (k, l) \right) \right| &\leq \left| \mathbb{P} \left( \hat{\mathbf{E}}^{(m)} = (k, l) \right) - \mathbb{P} \left( \hat{\mathbf{Z}}^{(m)} = (k, l) \right) \right| \\ &\quad + \left| \mathbb{P} \left( \hat{\mathbf{Z}}^{(m)} = (k, l) \right) - \mathbb{P} \left( \hat{\mathbf{Z}} = (k, l) \right) \right|. \end{aligned}$$

The second term on the right-hand side of the above equation tends to 0 as  $m \rightarrow \infty$  by Lemma 3.1 part (i) and (iii), so we need only show that

$$\lim_{m \rightarrow \infty} \left| \mathbb{P} \left( \hat{\mathbf{E}}^{(m)} = (k, l) \right) - \mathbb{P} \left( \hat{\mathbf{Z}}^{(m)} = (k, l) \right) \right| = 0. \quad (3.61)$$

Recall that by our notation,

$$\mathbb{P} \left( \hat{\mathbf{E}}^{(m)} = (k, l) \right) = \mathbb{P} \left( \left\{ \hat{E}_G^{(m)} = k \right\} \cap \left\{ \hat{E}_N^{(m)} = l \right\} \right).$$

Let  $\boldsymbol{\tau}^{(m)} = (\tau_G^{(m)}, \tau_N^{(m)})$  be the total number of (type-1, type-2) infected households in the epidemic process when a member of an already infected household is contacted for the first time. Since  $\mathbf{Z}^{(m)}$  and  $\mathbf{E}^{(m)}$  are coupled until  $\boldsymbol{\tau}^{(m)}$ ,

$$\mathbb{P} \left( \hat{\mathbf{E}}^{(m)} = (k, l), \boldsymbol{\tau}^{(m)} > (k, l) \right) = \mathbb{P} \left( \hat{\mathbf{Z}}^{(m)} = (k, l), \boldsymbol{\tau}^{(m)} > (k, l) \right). \quad (3.62)$$

Note that  $P(\hat{\mathbf{E}}^{(m)} = (k, l), \tau_G^{(m)} > k, \tau_N^{(m)} \leq l) = 0$ , since this is the probability of two mutually exclusive events. Similarly,

- $P(\hat{\mathbf{E}}^{(m)} = (k, l), \tau_G^{(m)} \leq k, \tau_N^{(m)} > l) = 0$ ,
- $P(\hat{\mathbf{Z}}^{(m)} = (k, l), \tau_G^{(m)} > k, \tau_N^{(m)} \leq l) = 0$ ,
- $P(\hat{\mathbf{Z}}^{(m)} = (k, l), \tau_G^{(m)} \leq k, \tau_N^{(m)} > l) = 0$ .

Therefore,

$$\begin{aligned} P(\hat{\mathbf{E}}^{(m)} = (k, l)) &= P(\hat{\mathbf{E}}^{(m)} = (k, l), \boldsymbol{\tau}^{(m)} \leq (k, l)) \\ &\quad + P(\hat{\mathbf{E}}^{(m)} = (k, l), \boldsymbol{\tau}^{(m)} > (k, l)) \end{aligned} \quad (3.63)$$

and

$$\begin{aligned} P(\hat{\mathbf{Z}}^{(m)} = (k, l)) &= P(\hat{\mathbf{Z}}^{(m)} = (k, l), \boldsymbol{\tau}^{(m)} \leq (k, l)) \\ &\quad + P(\hat{\mathbf{Z}}^{(m)} = (k, l), \boldsymbol{\tau}^{(m)} > (k, l)). \end{aligned} \quad (3.64)$$

So, applying the triangle inequality and substituting equations (3.62), (3.63) and (3.64),

$$\begin{aligned} &|P(\hat{\mathbf{E}}^{(m)} = (k, l)) - P(\hat{\mathbf{Z}}^{(m)} = (k, l))| \\ &= |P(\hat{\mathbf{E}}^{(m)} = (k, l), \boldsymbol{\tau}^{(m)} \leq (k, l)) + P(\hat{\mathbf{E}}^{(m)} = (k, l), \boldsymbol{\tau}^{(m)} > (k, l)) \\ &\quad - P(\hat{\mathbf{Z}}^{(m)} = (k, l), \boldsymbol{\tau}^{(m)} \leq (k, l)) - P(\hat{\mathbf{Z}}^{(m)} = (k, l), \boldsymbol{\tau}^{(m)} > (k, l))| \\ &= |P(\hat{\mathbf{E}}^{(m)} = (k, l), \boldsymbol{\tau}^{(m)} \leq (k, l)) - P(\hat{\mathbf{Z}}^{(m)} = (k, l), \boldsymbol{\tau}^{(m)} \leq (k, l))| \\ &\leq 2P(\boldsymbol{\tau}^{(m)} \leq (k, l)). \end{aligned} \quad (3.65)$$

As defined above Lemma 3.2, let  $\boldsymbol{\tau}_G^{(m)} = (\tau_{GG}^{(m)}, \tau_{GN}^{(m)})$  be the total number of (type-1, type-2) infected households in the epidemic process when a member of an already infected household is contacted via a global contact for the first time. Similarly, let  $\boldsymbol{\tau}_N^{(m)} = (\tau_{NG}^{(m)}, \tau_{NN}^{(m)})$  be the total number of (type-1, type-2) infected households in the epidemic process when a member of an already infected household is contacted via a network contact for the first time. Note that either  $\tau_G^{(m)} \leq \tau_N^{(m)}$  or  $\tau_G^{(m)} \geq \tau_N^{(m)}$ , so  $\boldsymbol{\tau}^{(m)} = \min\{\boldsymbol{\tau}_G^{(m)}, \boldsymbol{\tau}_N^{(m)}\}$ . Thus

$$P(\boldsymbol{\tau}^{(m)} \leq (k, l)) \leq P(\boldsymbol{\tau}_G^{(m)} \leq (k, l)) + P(\boldsymbol{\tau}_N^{(m)} \leq (k, l)). \quad (3.66)$$

Note that, since  $\lim_{m \rightarrow \infty} |(k, l)|^2/m = 0$ , the two quantities on the right-hand

side of equations (3.66) tend to 0 as  $m \rightarrow \infty$  by Lemmas 3.2 and 3.3 respectively, so substituting equation (3.66) into equation (3.65) and considering the limit  $m \rightarrow \infty$  yields equation (3.61) as required.

Finally we consider part (ii), the proof of which proceeds analogously to the proof of part (i). Note that, by the triangle inequality,

$$\begin{aligned} \left| \mathbb{P}(\hat{\mathbf{E}}^{(m)} < \mathbf{m}^\gamma) - \mathbb{P}(\hat{\mathbf{Z}} < \infty) \right| &\leq \left| \mathbb{P}(\hat{\mathbf{E}}^{(m)} < \mathbf{m}^\gamma) - \mathbb{P}(\hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma) \right| \\ &\quad + \left| \mathbb{P}(\hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma) - \mathbb{P}(\hat{\mathbf{Z}} < \infty) \right|. \end{aligned}$$

The second term on the right-hand side of the above equation tends to 0 as  $m \rightarrow \infty$  by Lemma 3.4, so we need only show that

$$\lim_{m \rightarrow \infty} \left| \mathbb{P}(\hat{\mathbf{E}}^{(m)} < \mathbf{m}^\gamma) - \mathbb{P}(\hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma) \right| = 0. \quad (3.67)$$

Recall that  $\boldsymbol{\tau}^{(m)} = (\tau_G^{(m)}, \tau_N^{(m)})$  is the total number of (type-1, type-2) infected households in the epidemic process when a member of an already infected household is contacted for the first time. Since  $\mathbf{Z}^{(m)}$  and  $\mathbf{E}^{(m)}$  are coupled until  $\boldsymbol{\tau}^{(m)}$ ,

$$\mathbb{P}(\hat{\mathbf{E}}^{(m)} < \mathbf{m}^\gamma, \boldsymbol{\tau}^{(m)} > \mathbf{m}^\gamma) = \mathbb{P}(\hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma, \boldsymbol{\tau}^{(m)} > \mathbf{m}^\gamma). \quad (3.68)$$

Note that  $\mathbb{P}(\hat{\mathbf{E}}^{(m)} < \mathbf{m}^\gamma, \tau_G^{(m)} > m^\gamma, \tau_N^{(m)} \leq m^\gamma) = 0$ , since this is the probability of two mutually exclusive events. Similarly,

- $\mathbb{P}(\hat{\mathbf{E}}^{(m)} < \mathbf{m}^\gamma, \tau_G^{(m)} \leq m^\gamma, \tau_N^{(m)} > m^\gamma) = 0$ ,
- $\mathbb{P}(\hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma, \tau_G^{(m)} > m^\gamma, \tau_N^{(m)} \leq m^\gamma) = 0$ ,
- $\mathbb{P}(\hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma, \tau_G^{(m)} \leq m^\gamma, \tau_N^{(m)} > m^\gamma) = 0$ .

Therefore,

$$\mathbb{P}(\hat{\mathbf{E}}^{(m)} < \mathbf{m}^\gamma) = \mathbb{P}(\hat{\mathbf{E}}^{(m)} < \mathbf{m}^\gamma, \boldsymbol{\tau}^{(m)} \leq \mathbf{m}^\gamma) \quad (3.69)$$

$$+ \mathbb{P}(\hat{\mathbf{E}}^{(m)} < \mathbf{m}^\gamma, \boldsymbol{\tau}^{(m)} > \mathbf{m}^\gamma), \quad (3.70)$$

and

$$\begin{aligned} \mathbb{P}(\hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma) &= \mathbb{P}(\hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma, \boldsymbol{\tau}^{(m)} \leq \mathbf{m}^\gamma) \\ &\quad + \mathbb{P}(\hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma, \boldsymbol{\tau}^{(m)} > \mathbf{m}^\gamma). \end{aligned} \quad (3.71)$$

So, applying the triangle inequality and substituting equations (3.68), (3.70) and (3.71),

$$\begin{aligned}
& \left| \mathbb{P} \left( \hat{\mathbf{E}}^{(m)} < \mathbf{m}^\gamma \right) - \mathbb{P} \left( \hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma \right) \right| \\
&= \left| \mathbb{P} \left( \hat{\mathbf{E}}^{(m)} < \mathbf{m}^\gamma, \boldsymbol{\tau}^{(m)} \leq \mathbf{m}^\gamma \right) + \mathbb{P} \left( \hat{\mathbf{E}}^{(m)} < \mathbf{m}^\gamma, \boldsymbol{\tau}^{(m)} > \mathbf{m}^\gamma \right) \right. \\
&\quad \left. - \mathbb{P} \left( \hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma, \boldsymbol{\tau}^{(m)} \leq \mathbf{m}^\gamma \right) - \mathbb{P} \left( \hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma, \boldsymbol{\tau}^{(m)} > \mathbf{m}^\gamma \right) \right| \\
&= \left| \mathbb{P} \left( \hat{\mathbf{E}}^{(m)} < \mathbf{m}^\gamma, \boldsymbol{\tau}^{(m)} \leq \mathbf{m}^\gamma \right) - \mathbb{P} \left( \hat{\mathbf{Z}}^{(m)} < \mathbf{m}^\gamma, \boldsymbol{\tau}^{(m)} \leq \mathbf{m}^\gamma \right) \right| \\
&\leq 2\mathbb{P} \left( \boldsymbol{\tau}^{(m)} \leq \mathbf{m}^\gamma \right). \tag{3.72}
\end{aligned}$$

Recall that  $\boldsymbol{\tau}_G^{(m)} = (\tau_{GG}^{(m)}, \tau_{GN}^{(m)})$  is the total number of (type-1, type-2) infected households in the epidemic process when a member of an already infected household is contacted via a global contact for the first time. Similarly,  $\boldsymbol{\tau}_N^{(m)} = (\tau_{NG}^{(m)}, \tau_{NN}^{(m)})$  is the total number of (type-1, type-2) infected households in the epidemic process when a member of an already infected household is contacted via a network contact for the first time. Note that either  $\boldsymbol{\tau}_G^{(m)} \leq \boldsymbol{\tau}_N^{(m)}$  or  $\boldsymbol{\tau}_G^{(m)} \geq \boldsymbol{\tau}_N^{(m)}$ , so  $\boldsymbol{\tau}^{(m)} = \min \{ \boldsymbol{\tau}_G^{(m)}, \boldsymbol{\tau}_N^{(m)} \}$ . Thus

$$\mathbb{P} \left( \boldsymbol{\tau}^{(m)} \leq \mathbf{m}^\gamma \right) \leq \mathbb{P} \left( \boldsymbol{\tau}_G^{(m)} \leq \mathbf{m}^\gamma \right) + \mathbb{P} \left( \boldsymbol{\tau}_N^{(m)} \leq \mathbf{m}^\gamma \right). \tag{3.73}$$

For fixed  $\gamma \in (0, 1/2)$ ,  $\lim_{m \rightarrow \infty} |\mathbf{m}^\gamma|^2/m = 0$ , so the two quantities of the right-hand side of equation (3.73) tend to 0 as  $m \rightarrow \infty$  by Lemmas 3.2 and 3.3 respectively. Therefore, substituting equation (3.73) into equation (3.72) and considering the limit  $m \rightarrow \infty$  yields equation (3.67) as required.  $\square$

### 3.6 Concluding remarks

In this chapter, we introduce the epidemic model and a branching process approximation for the early stages of the epidemic along with limit theorems showing that as the number of households in the population tends to infinity the total number of individuals infected in the epidemic process converges in distribution to the total progeny of a branching process. This allows us to find asymptotic results including threshold parameters that give explicit criteria for whether a major outbreak can occur or not, along with the probability of a major outbreak assuming a constant infectious period. We then give a heuristic argument that the final size of a major outbreak is equal to the probability that a two-type branching process survives, and give the probability generating functions for the offspring distribution of said branching process. We then

discuss the convergence of the probability of a major outbreak and the final size of a major outbreak in finite populations to our asymptotic results. Finally, we briefly discuss one way to compare epidemic models, by keeping  $R_0$  constant, and use this to investigate how the addition of heterogeneity to the homogeneously mixing model affected the final size of a major outbreak. These numerical results are explored further in the next chapter along with analytic results.

### 3.7 Table of common notation introduced in Chapter 3

Symbol	Meaning	Page
$N$	Number of individuals in the population.	25
$m$	Number of households in population.	25
$D$	Network degree distribution.	25
$p_k$	$P(D = k)$ .	25
$I$	Infectious period distribution.	26
$\phi_I(\theta)$	Laplace-Stieltjes transform of $I$ .	26
$\lambda_H$	Household infection rate.	26
$p_H$	$1 - \phi_I(\lambda_H)$ .	26
$\lambda_N$	Network infection rate.	26
$p_N$	$1 - \phi_I(\lambda_N)$ .	26
$\lambda_G$	Global infection rate.	26
$H$	Household size distribution.	27
$\rho_n$	$P(H = n)$ .	27
$\mathbf{E}^{(m)}$	Epidemic process on $m$ households.	29
$\hat{\mathbf{E}}^{(m)}$	Total number of households infected in the epidemic process on $m$ households.	29
$\mathbf{Z}$	Forward branching process.	29
$\hat{\mathbf{Z}}$	Total progeny of the forward branching process.	29
$R_*$	Threshold parameter.	30
$M$	Mean next-generation matrix of the forward branching process.	30
$(C_{GG}, C_{GN})$	Total number of (type-1, type-2) offspring of a typical type-1 individual in the forward branching process.	31
$f_{C_1}(\mathbf{s})$	$E[s_1^{C_{GG}} s_2^{C_{GN}}]$ .	46
$(C_{NG}, C_{NN})$	Total number of (type-1, type-2) offspring of a typical type-2 individual in the forward branching process.	31
$f_{C_2}(\mathbf{s})$	$E[s_1^{C_{NG}} s_2^{C_{NN}}]$ .	46
$\mathbf{f}_C(\mathbf{s})$	$(f_{C_1}(\mathbf{s}), f_{C_2}(\mathbf{s}))$ .	46
$G_k(x U)$	$k$ th Gontcharoff polynomial.	19

$T^{(n)}$	Final size of a household epidemic.	20
$\mu_T$	Mean final size of a household epidemic in which the household size is sampled according to $\tilde{H}$ .	20
$R_0$	Basic reproduction number.	35
$\mu_i^{(n)}$	Mean number of infectives in generation $i$ of a single household epidemic with a household size of $n$ .	37
$\rho_{\text{maj}}$	Probability of a major outbreak.	46
$G_N(s_2)$	$f_D(1 - p_N + p_N s_2)$ .	46
$\tilde{G}_N(s_2)$	$f_{\tilde{D}-1}(1 - p_N + p_N s_2)$ .	46
$M^{(n)}$	Size of an individuals household susceptibility set when the individual is in a household of size $n$ .	51
$z$	Relative final size of a major outbreak.	52
$b_i(\mathbf{s})$	Joint generating function for the offspring of a typical type- $i$ individual in the backwards branching process.	55
$\mathbf{b}(\mathbf{s})$	Vector of joint generating functions for the offspring of each type of individual in the backwards branching process.	55
$\mathbf{Z}^{(m)}$	Forward branching process on $m$ households.	64
$\hat{\mathbf{Z}}^{(m)}$	Total progeny of the forward branching process on $m$ households.	64

### 3.7.1 Table of common notation introduced in Section 3.5

Symbol	Meaning	Page
$\alpha$	Household category.	65
$\mathcal{A}$	Set of all possible household categories.	65
$N^{(m)}$	Total number of individuals in the first $m$ households.	65
$d_\alpha$	Total degree of a household with household category $\alpha$ .	67

$\tau_{GG}^{(m)}$	Total number of type-1 previously contacted households in the epidemic process when a member of a previously contacted household is contacted via a global infection event for the first time.	70
$\tau_{GN}^{(m)}$	Total number of type-2 previously contacted households in the epidemic process when a member of a previously contacted household is contacted via a global infection event for the first time.	70
$\boldsymbol{\tau}_G^{(m)}$	$(\tau_{GG}^{(m)}, \tau_{GN}^{(m)})$ .	70
$\tau_{NG}^{(m)}$	Total number of type-1 previously contacted households in the epidemic process when a member of a previously contacted household is contacted via a network infection event for the first time.	70
$\tau_{NN}^{(m)}$	Total number of type-2 previously contacted households in the epidemic process when a member of a previously contacted household is contacted via a network infection event for the first time.	70
$\boldsymbol{\tau}_N^{(m)}$	$(\tau_{NG}^{(m)}, \tau_{NN}^{(m)})$ .	70
$\chi_G^{(m)}(i)$	Household containing the individual chosen at the $i$ th global infection event.	70
$\hat{\chi}_G^{(m)}(i)$	Number of individuals in household $\chi_G^{(m)}(i)$ .	70
$\bar{\chi}_G^{(m)}(i)$	Number of half-edges in household $\chi_G^{(m)}(i)$ .	70
$\xi_G^{(m)}(i)$	Generation in which the $i$ th global infection event occurs.	70
$\chi_N^{(m)}(i)$	Household containing the half-edge chosen at the $i$ th network infection event.	70
$\hat{\chi}_N^{(m)}(i)$	Number of individuals in household $\chi_N^{(m)}(i)$ .	70
$\bar{\chi}_N^{(m)}(i)$	Number of half-edges in household $\chi_N^{(m)}(i)$ .	70
$\xi_N^{(m)}(i)$	Generation in which the $i$ th network infection event occurs.	70



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## 4. Effect of heterogeneity on the final size of a major outbreak

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With the introduction of epidemic models to the literature which avoid some of the homogeneity assumptions exhibited by the early epidemic models, e.g. Kermack and McKendrick (1927), it has become increasingly clear that heterogeneity in the population structure or the types of individual in the population has a varied effect on the spread of an epidemic. Indeed, quantifying the effect of heterogeneity on the epidemic outcome, including the final size of a major outbreak, has been identified as one of the eight challenges for network epidemic modelling, Pellis et al. (2015). In this chapter we explore the effect of two types of heterogeneity, specifically heterogeneity introduced by the construction of a network and heterogeneity introduced by the inclusion of a household structure.

However, we first discuss how we compare two epidemic models with different heterogeneity. Kiss et al. (2006) investigate the effect of heterogeneity in a deterministic epidemic model with a network structure and global contacts on the relative final size of a major outbreak while keeping the overall transmission potential constant. In the same model, although studying a stochastic model, Ball and Neal (2008) investigate the effect of heterogeneity on  $R_0$  and the expected relative final size of a major outbreak while the mean of the degree distribution is fixed. Similarly, Britton and Trapman (2012) consider maximising the size of the giant component in the thinned configuration model, i.e. the relative final size of a major outbreak in the standard network model, while fixing the mean of the degree distribution, and prove that the giant component is maximised when the degrees of the individuals in the population are homogenised. We choose to compare models by fixing  $R_0$  because it is well studied, i.e. estimating  $R_0$  from data about an emerging epidemic, or the initial growth rate of the

epidemic, is a large topic of interest (e.g. Heesterbeek and Dietz (1996), Fraser (2007)) and it has a clear biological implication in many epidemic models.

Therefore we consider two epidemic models with differing amounts of heterogeneity, fix  $R_0$  and then compare the asymptotic results for the expected relative final size of a major outbreak (which we again refer to as the final size of a major outbreak). This is similar to Clancy and Pearce (2013), who fix  $R_0$  and investigate the effect of heterogeneity in a multi-type epidemic model on various epidemic outcomes, including the final size of a major outbreak. Clancy and Pearce (2013) give several conditions upon the parameters of the multi-type epidemic model for which fixing  $R_0$  and increasing the heterogeneity will increase or decrease the final size of a major outbreak.

We now give a brief comment on notation. We name models with multiple levels of mixing after the ways in which the infection can spread through the population, and assume that all unmentioned infection rates are 0 and unmentioned network or household structures are not included. So we call the model of Ball and Neal (2008) the network and global model, which is recovered from our model in Chapter 3 by setting  $\lambda_H = 0$  and  $\rho_1 = 1$ . For consistency, we call the households model of Ball et al. (1997) the household and global model, which we recover by setting  $\lambda_N = 0$  and  $p_0 = 1$ . We use the standard network model, see Newman (2002a), for the model which has no global contacts and no household structure, which is recovered from our model by setting  $\lambda_G = 0$  and  $\rho_1 = 1$ . Finally, for the model without network or household structure we use the standard name in the literature of the homogeneously mixing model, see Kermack and McKendrick (1927), which we recover by setting either  $p_0 = 1$  or  $\lambda_N = 1$  and either  $\rho_1 = 1$  or  $\lambda_H = 0$ .

We split Chapter 4 into two segments. In Section 4.1 we restrict our attention to the network and global model, and in Section 4.2 we consider the household and global model. In Section 4.1.1 we consider the effect of introducing a small amount of network heterogeneity to the homogeneously mixing model. In Section 4.1.2 we give examples of degree distributions for which when  $R_0$  is fixed the final size of a major outbreak is independent of some parameters of the degree distribution, which is then applied in Section 4.1.3 to give an ordering for the final size of a major outbreak on the standard network model. In Sections 4.1.4 and 4.1.5 we consider constant and logarithmic degree distributions respectively,

and use the Logarithmic degree distribution to prove that introducing network heterogeneity to the homogeneously mixing model does not always have a monotonic effect on the final size of a major outbreak. In Section 4.2.1 we investigate the effect of introducing a small amount of household heterogeneity to the homogeneously mixing model, and in Section 4.2.2 we discuss an ordering for the final size of a major outbreak on the households and global model with a saturated household infection rate. Finally, we give concluding remarks in Section 4.3 and a table of common notation introduced in this chapter in Section 4.4.

## 4.1 Network and global model

In this section we investigate how the choice of degree distribution affects the final size of a major outbreak in the household and global model while  $R_0 > 1$  is fixed. To do this we investigate how the final size of a major outbreak varies as we change the network infection rate,  $\lambda_N$ , while adjusting the global infection rate,  $\lambda_G$ , to keep  $R_0$  fixed for a range of degree distributions. Note that this means we fix all other parameters, including the distribution of the infectious period  $I$ .

Figure 4.1 plots the final size of a major outbreak in the network and global model against the global infection rate,  $\lambda_G$ , as the network infection rate,  $\lambda_N$ , is reduced to keep  $R_0 = 2$  constant, for a range of degree distributions, and  $\phi_I(t) = e^{-t}$ . Note that this means that each of the 7 distributions have a different value of  $\lambda_N$  when  $\lambda_G = 0$ , i.e. the left-hand side of Figure 4.1. For each of the 7 degree distributions considered in Figure 4.1, we see a monotonic change in the final size of a major outbreak as  $\lambda_G$  is increased, although whether it is increasing or decreasing depends on the degree distribution. For example, if  $D \sim \text{Const}(4)$ , then the final size of a major outbreak is monotonically decreasing in  $\lambda_G$ , whereas if  $D \sim \text{Geo}(1/9)$ , then the final size of a major outbreak is monotonically increasing in  $\lambda_G$ . In Section 4.1.5 we prove that fixing  $R_0$  and increasing  $\lambda_G$  while decreasing  $\lambda_N$  to keep  $R_0$  constant does not always have a monotonic effect on the final size of a major outbreak by considering a logarithmic degree distribution.

Figure 4.1 shows that the choice of degree distribution and  $\lambda_G$  can have a large impact on the final size of a major outbreak. For example, on the standard

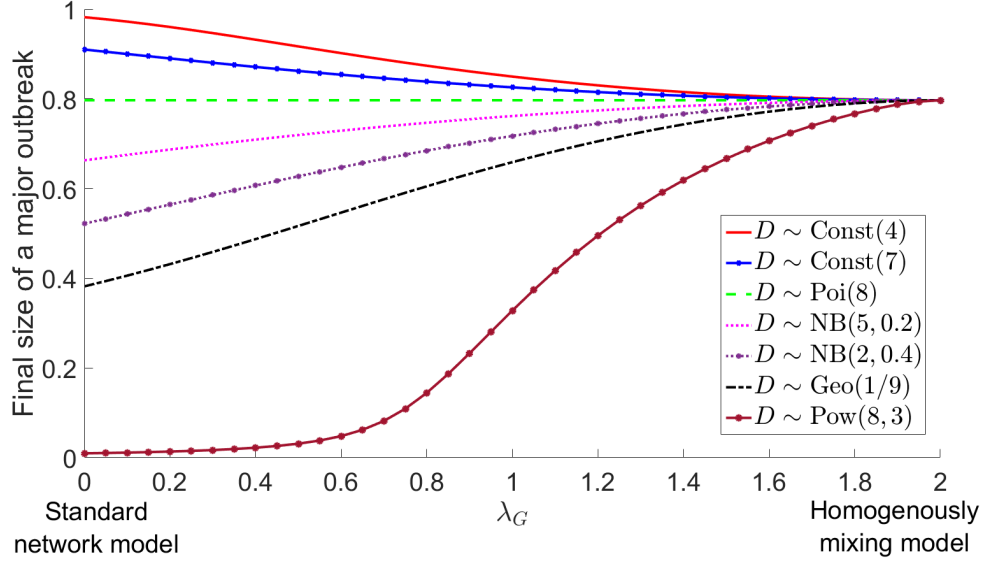


Figure 4.1: Effect of the degree distribution on the final size of a major outbreak in the network and global model for a range of degree distributions. Other parameters are  $R_0 = 2$  and  $I \sim \text{Const}(1)$ .

network model with  $R_0 = 2$  (i.e.  $\lambda_G = 0$  in Figure 4.1) if  $D \sim \text{Const}(4)$  then the final size of a major outbreak is close to 1, whereas if  $D \sim \text{Pow}(8, 3)$  then the final size of a major outbreak is close to 0.

We now recall results required to give the final size of a major outbreak and the formula for  $R_0$  in the network and global model. Recall that, by substituting  $\lambda_H = 0$  and  $\rho_1 = 1$  into  $b_1(s_1, s_2)$  and  $b_2(s_1, s_2)$  given in Theorem 3.6, the final size of a major outbreak in the network and global model  $z = 1 - \pi_1$ , where  $\pi = (\pi_1, \pi_2)$  is the smallest solution to the set of simultaneous equations  $\pi = \mathbf{b}(\pi)$ , with

$$b_1(s_1, s_2) = e^{-\lambda_G \mu_I (1-s_1)} f_D(1 - p_N + p_N s_2), \quad (4.1a)$$

$$b_2(s_1, s_2) = e^{-\lambda_G \mu_I (1-s_1)} f_{\tilde{D}-1}(1 - p_N + p_N s_2). \quad (4.1b)$$

**Remark.** *Ball and Neal (2008) consider a single-type Galton-Watson branching process, similar to that mentioned in Section 3.3.2, which is an embedded version of the two-type Galton-Watson branching process we consider. The single-type branching process is recovered from the two-type branching process we consider by removing the type-2 individuals in the two-type backward Galton-Watson branching process by attributing all offspring of a type-2 individual to its parent (analogously to the method discussed in Section 3.3.2). Both the single-type*

and two-type branching processes have the same extinction probability, but we consider the two-type branching process for consistency to the work in Chapter 3 and ease of analytical tractability. Note that Ball and Neal define their (network clump-based) threshold parameter  $R_*$  to be the expected offspring of the single-type branching process, and is therefore different to our (household-based) definition.

Let  $z_i = 1 - \pi_i$ ,  $i = 1, 2$ , so, substituting  $\mathbf{z} = (z_1, z_2)$  into equations (4.1), the final size of a major outbreak in the network and global model is equal to  $z_1$ , where  $\mathbf{z}$  is the largest solution in  $(0, 1]^2$  of the set of simultaneous equations

$$1 - z_1 = e^{-\lambda_G \mu_I z_1} f_D(1 - p_N z_2), \quad (4.2a)$$

$$1 - z_2 = e^{-\lambda_G \mu_I z_1} f_{\tilde{D}-1}(1 - p_N z_2). \quad (4.2b)$$

Finally, recall that in the network and global model  $R_0 = R_*$  (see Section 3.2.3) so  $R_0$  is the largest root of the equation

$$R_0^2 - R_0 (\lambda_G \mu_I + p_N \mu_{\tilde{D}-1}) + \lambda_G \mu_I p_N \mu_{\tilde{D}-1} - \lambda_G \mu_I p_N \mu_D = 0. \quad (4.3)$$

**Remark.** *The argument we use in Section 3.3 to calculate the final size of a major outbreak does not hold if  $\lambda_G = 0$ . Instead, the argument for calculating the final size of a major outbreak in the standard network model holds by an alternative argument (see, for example, Newman (2002a)). However, the final size of a major outbreak in the standard network with degree distribution  $D$  and network infection rate  $\lambda_N$  is given by  $z = 1 - f_D(1 - p_N z_2)$ , where  $z_2$  is the largest solution  $(0, 1]$  of the equation  $1 - z_2 = f_{\tilde{D}-1}(1 - p_N z_2)$ . Thus substituting  $\lambda_G = 0$  in equations (4.2) recovers the equations for the final size of a major outbreak in the standard network model. Similarly, in the standard network model  $R_0 = p_N \mu_{\tilde{D}-1}$ , which can be recovered by substituting  $\lambda_G = 0$  into equation (4.3).*

Let  $z_H(R_0)$  be the final size of a major outbreak in the homogeneously mixing model with basic reproduction number  $R_0$ , (so  $\lambda_G = R_0/\mu_I$ ). Then (see, for example, Bailey (1975))  $z_H(R_0)$  is the unique solution of the equation

$$1 - z_H(R_0) = e^{-R_0 z_H(R_0)}. \quad (4.4)$$

Thus for fixed  $R_0$ ,  $z_H(R_0)$  is independent of our choice of  $\lambda_G$ . We now consider an inequality required for two proofs discussed later in this chapter.

**Lemma 4.1.** For  $R_0 > 1$ ,

$$1 - R_0(1 - z_H(R_0)) > 0. \quad (4.5)$$

*Proof.* Let  $g(s) = 1 - s - e^{-R_0 s}$ , so  $z_H(R_0)$  is the unique root of  $g(s)$  in  $(0, 1]$ . Then  $g(z_H(R_0)) = 0$ ,  $g'(s) = -1 + R_0 e^{-R_0 s}$  and, substituting equation (4.4) into  $g'(z_H(R_0))$ ,  $g'(z_H(R_0)) = -1 + R_0(1 - z_H(R_0))$ . Note that  $g'(0) = R_0 - 1 > 0$ ,  $g(0) = 0$  and  $z_H(R_0) \in (0, 1]$  is the unique root of  $g(s)$ , so  $g'(z_H(R_0)) < 0$ . Therefore  $-1 + R_0(1 - z_H(R_0)) < 0$  and Lemma 4.1 follows.  $\square$

#### 4.1.1 Adding a small amount of network structure to the homogeneously mixing model

Firstly, we note that  $R_0$  and the final size of a major outbreak in the network and global model depend on  $\lambda_G$ ,  $\lambda_N$  (through  $p_N$ ),  $D$  and  $I$  (see equations (4.2) and (4.3)). Since we choose to compare epidemic models by fixing  $R_0$ , we can consider  $\lambda_G$  to be a value determined by  $p_N(\lambda_N)$ ,  $R_0$ ,  $D$  and  $I$ , so we write  $\lambda_G(p_N(\lambda_N); R_0, D, I)$ . Furthermore the final size of a major outbreak is now determined by  $p_N(\lambda_N)$ ,  $R_0$ ,  $D$  and  $\mu_I$ , so we write

$$\mathbf{z}(p_N(\lambda_N); R_0, D, I) = (z_1(p_N(\lambda_N); R_0, D, I), z_2(p_N(\lambda_N); R_0, D, I)),$$

where  $z_1(p_N(\lambda_N); R_0, D, I)$  is the final size of a major outbreak.

For notational simplicity we write  $p_N = p_N(\lambda_N)$ , i.e. we drop the explicit reference to  $\lambda_N$ , and note that  $p_N(0) = 0$ . So, for example, we write  $\lambda_G(p_N; R_0, D, I) = \lambda_G(p_N(\lambda_N); R_0, D, I)$ . Substituting this notation into equations (4.2) yields that  $\mathbf{z}(p_N; R_0, D, I)$  satisfies the equations

$$1 - z_1(p_N; R_0, D, I) = e^{-\lambda_G(p_N; R_0, D, I)\mu_I z_1(p_N; R_0, D, I)} f_D(1 - p_N z_2(p_N; R_0, D, I)), \quad (4.6a)$$

$$1 - z_2(p_N; R_0, D, I) = e^{-\lambda_G(p_N; R_0, D, I)\mu_I z_1(p_N; R_0, D, I)} f_{\tilde{D}-1}(1 - p_N z_2(p_N; R_0, D, I)), \quad (4.6b)$$

and, rearranging equation (4.3),

$$\lambda_G(p_N; R_0, D, I) = \frac{R_0(R_0 - p_N \mu_{\tilde{D}-1})}{\mu_I(R_0 + p_N(\mu_D - \mu_{\tilde{D}-1}))}. \quad (4.7)$$

To investigate the effect of fixing  $R_0$  and introducing a small amount of heterogeneity to the homogeneously mixing model on the final size of a major outbreak we require knowledge of the derivatives of equation (4.6) with respect to  $p_N$  evaluated at  $p_N = 0$ . Therefore we require expressions for  $\lambda_G(p_N; R_0, D, I)$  and its derivatives at the origin, which we give in the following lemma.

**Lemma 4.2.** *Consider the network and global model with fixed  $R_0$ . Then*

(i)

$$\lambda_G(0; R_0, D, I) = \frac{R_0}{\mu_I},$$

(ii)

$$\frac{d\lambda_G}{dp_N}(0; R_0, D, I) = \frac{-\mu_D}{\mu_I},$$

(iii)

$$\frac{d^2\lambda_G}{dp_N^2}(0; R_0, D, I) = \frac{2\mu_D \left(1 - \frac{\sigma_D^2}{\mu_D}\right)}{R_0\mu_I}.$$

*Proof.* Recall from Section 2.1 that

$$\mu_D - \mu_{\tilde{D}-1} = 1 - \sigma_D^2/\mu_D. \quad (4.8)$$

Substituting equation (4.8) into equation (4.7) yields

$$\lambda_G(p_N; R_0, D, I) = \frac{R_0 (R_0 - p_N \mu_{\tilde{D}-1})}{\mu_I (R_0 + p_N (1 - \sigma_D^2/\mu_D))}, \quad (4.9)$$

which, evaluated at  $p_N = 0$ , yields part (i).

Taking the derivative of equation (4.9) with respect to  $p_N$  and rearranging yields

$$\frac{d\lambda_G}{dp_N}(p_N; R_0, D, I) = \frac{-R_0^2 \mu_D}{\mu_I \left(R_0 + p_N \left(1 - \frac{\sigma_D^2}{\mu_D}\right)\right)^2}, \quad (4.10)$$

which, evaluated at  $p_N = 0$ , yields part (ii).

To prove part (iii) we consider the derivatives of equation (4.3). Taking the

derivative of equation (4.3) with respect to  $p_N$  yields

$$0 = -R_0 \left( \mu_I \frac{d\lambda_G}{dp_N} (p_N; R_0, D, I) + \mu_{\tilde{D}-1} \right) + \lambda_G \mu_I (\mu_{\tilde{D}-1} - \mu_D) \\ + \frac{d\lambda_G}{dp_N} (p_N; R_0, D, I) \mu_I p_N (\mu_{\tilde{D}-1} - \mu_D), \quad (4.11)$$

Taking the derivative of equation (4.11) with respect to  $p_N$  yields

$$0 = -R_0 \mu_I \frac{d^2 \lambda_G}{dp_N^2} (p_N; R_0, D, I) + 2 \frac{d\lambda_G}{dp_N} (p_N; R_0, D, I) \mu_I (\mu_{\tilde{D}-1} - \mu_D) \\ + \frac{d^2 \lambda_G}{dp_N^2} (p_N; R_0, D, I) \mu_I p_N (\mu_{\tilde{D}-1} - \mu_D). \quad (4.12)$$

Substituting equations (4.8) and (4.10) into equation (4.12) and then evaluating the result at  $p_N = 0$  yields part (iii) as required, i.e.

$$R_0 \mu_I \frac{d^2 \lambda_G}{dp_N^2} (0; R_0, D, I) = 2 \frac{d\lambda_G}{dp_N} (0; R_0, D, I) \mu_I (\mu_{\tilde{D}-1} - \mu_D) = 2\mu_D \left( 1 - \frac{\sigma_D^2}{\mu_D} \right).$$

□

The following theorem is a key result of this section, and states the effect of introducing a small amount of network heterogeneity to the homogeneously mixing model if  $R_0$  is fixed. The result of this theorem corresponds to considering the right-hand side of Figure 4.1, i.e.  $\lambda_G = 2$ .

**Theorem 4.1.** *Let  $z_1(p_N; R_0, D, I)$  be the final size of a major outbreak in the network and global model. Assume that  $z_1(0; R_0, D, I) < 1$  and  $p_0 < 1$  (i.e.  $P(D = 0) < 1$ ). Then*

(i)

$$\mathbf{z}(0; R_0, D, I) = (z_H(R_0), z_H(R_0)),$$

(ii)

$$\frac{dz_1}{d\lambda_N}(0; R_0, D, I) = 0,$$

(iii)

$$\frac{d^2 z_1}{d\lambda_N^2}(0; R_0, D, I) (1 - R_0 (1 - z_H(R_0))) = \left( 1 - \frac{\sigma_D^2}{\mu_D} \right) \left[ \frac{2}{R_0} + 3z_H(R_0) - 2 \right] \\ \times \mu_D z_H(R_0) (1 - z_H(R_0)) \mu_I^2,$$



(iv)

$$\operatorname{sgn} \left( \frac{d^2 z_1}{d\lambda_N^2} (0; R_0, D, I) \right) = \operatorname{sgn} \left( 1 - \frac{\sigma_D^2}{\mu_D} \right).$$

*Proof.* Recall that  $z(0; R_0, D, I)$  is the largest solution in  $(0, 1]^2$  of equations (4.6). Evaluating equations (4.6) at  $p_N = 0$  and substituting Lemma 4.2(i) yields

$$1 - z_1(0; R_0, D, I) = e^{-R_0 z_1(0; R_0, D, I)}, \quad (4.13a)$$

$$1 - z_2(0; R_0, D, I) = e^{-R_0 z_1(0; R_0, D, I)}. \quad (4.13b)$$

Therefore  $z_1(0; R_0, D, I)$ ,  $z_2(0; R_0, D, I)$  and  $z_H(R_0)$  are all the largest solution in  $(0, 1]$  of equation (4.4), so  $z_1(0; R_0, D, I) = z_2(0; R_0, D, I) = z_H(R_0)$ , as required for the proof of part (i).

We now consider parts (ii)-(iv). Differentiating equation (4.6a) with respect to  $p_N$  and multiplying both sides by  $-1$  yields

$$\begin{aligned} & \frac{dz_1}{dp_N} (p_N; R_0, D, I) \\ &= \left[ z_1(p_N; R_0, D, I) \frac{d\lambda_G}{dp_N} (p_N; R_0, D, I) \right. \\ & \quad \left. + \lambda_G(p_N; R_0, D, I) \frac{dz_1}{dp_N} (p_N; R_0, D, I) \right] \\ & \quad \times \mu_I f_D(1 - p_N z_2(p_N; R_0, D, I)) \\ & \quad + \left[ z_2(p_N; R_0, D, I) + p_N \frac{dz_2}{dp_N} (p_N; R_0, D, I) \right] \\ & \quad \times f'_D(1 - p_N z_2(p_N; R_0, D, I)) e^{-\lambda_G(p_N; R_0, D, I) \mu_I z_1(p_N; R_0, D, I)}. \end{aligned} \quad (4.14)$$

Substituting  $f_{\bar{D}-1}(s) = f'_D(s)/\mu_D$  and equations (4.6) into equation (4.14) yields

$$\begin{aligned} \frac{dz_1}{dp_N} (p_N; R_0, D, I) &= \left[ z_1(p_N; R_0, D, I) \frac{d\lambda_G}{dp_N} (p_N; R_0, D, I) \right. \\ & \quad \left. + \lambda_G(p_N; R_0, D, I) \frac{dz_1}{dp_N} (p_N; R_0, D, I) \right] \\ & \quad \times \mu_I (1 - z_1(p_N; R_0, D, I)) \\ & \quad + \left[ z_2(p_N; R_0, D, I) + p_N \frac{dz_2}{dp_N} (p_N; R_0, D, I) \right] \\ & \quad \times \mu_D (1 - z_2(p_N; R_0, D, I)). \end{aligned} \quad (4.15)$$

Evaluating equation (4.15) at  $p_N = 0$  and applying part (i) and Lemma 4.2(i) yields

$$\begin{aligned}
& \frac{dz_1}{dp_N} (0; R_0, D, I) [1 - \mu_I \lambda_G (0; R_0, D, I) (1 - z_1 (0; R_0, D, I))] \\
&= \mu_I z_1 (0; R_0, D, I) (1 - z_1 (0; R_0, D, I)) \frac{d\lambda_G}{dp_N} (0; R_0, D, I) \\
&\quad + \mu_D (1 - z_2 (0; R_0, D, I)) z_2 (0; R_0, D, I) \\
&= z_H(R_0)(1 - z_H(R_0)) \left[ \mu_I \frac{d\lambda_G}{dp_N} (0; R_0, D, I) + \mu_D \right]. \tag{4.16}
\end{aligned}$$

Clearly  $z_H(R_0)(1 - z_H(R_0)) \geq 0$ , and  $1 - R_0(1 - z_H(R_0)) > 0$  by Lemma 4.1. Therefore applying Lemma 4.2(ii) to equation (4.16) yields

$$\frac{dz_1}{dp_N} (0; R_0, D, I) = 0. \tag{4.17}$$

Substituting equation (4.17) and  $p'_N (0; R_0, D, I) = -\mu_I$  into

$$\frac{dz_1}{d\lambda_N} (0; R_0, D, I) = p'_N (0; R_0, D, I) \frac{dz_1}{dp_N} (0; R_0, D, I),$$

yields part (ii).

We now consider the calculation of  $\frac{d^2 z_1}{dp_N^2} (0; R_0, D, I)$ . Differentiating equation

(4.15) with respect to  $p_N$  yields

$$\begin{aligned}
& \frac{d^2 z_1}{dp_N^2} (p_N; R_0, D, I) \\
&= - \left[ z_1 (p_N; R_0, D, I) \frac{d\lambda_G}{dp_N} (p_N; R_0, D, I) \right. \\
&\quad \left. + \lambda_G (p_N; R_0, D, I) \frac{dz_1}{dp_N} (p_N; R_0, D, I) \right] \mu_I \frac{dz_1}{dp_N} (p_N; R_0, D, I) \\
&\quad + \mu_I (1 - z_1 (p_N; R_0, D, I)) \left\{ z_1 (p_N; R_0, D, I) \frac{d^2 \lambda_G}{dp_N^2} (p_N; R_0, D, I) \right. \\
&\quad \quad \left. + 2 \frac{d\lambda_G}{dp_N} (p_N; R_0, D, I) \frac{dz_1}{dp_N} (p_N; R_0, D, I) \right. \\
&\quad \quad \left. + \lambda_G (p_N; R_0, D, I) \frac{d^2 z_1}{dp_N^2} (p_N; R_0, D, I) \right\} \\
&\quad - \mu_D \frac{dz_2}{dp_N} (p_N; R_0, D, I) \left[ z_2 (p_N; R_0, D, I) + p_N \frac{dz_2}{dp_N} (p_N; R_0, D, I) \right] \\
&\quad + \mu_D (1 - z_2 (p_N; R_0, D, I)) \left[ 2 \frac{dz_2}{dp_N} (p_N; R_0, D, I) \right. \\
&\quad \quad \left. + p_N \frac{d^2 z_2}{dp_N^2} (p_N; R_0, D, I) \right]. \tag{4.18}
\end{aligned}$$

Evaluating equation (4.18) at  $p_N = 0$  and substituting equation (4.17) yields

$$\begin{aligned}
\frac{d^2 z_1}{dp_N^2} (0; R_0, D, I) &= \mu_I (1 - z_1 (0; R_0, D, I)) z_1 (0; R_0, D, I) \frac{d^2 \lambda_G}{dp_N^2} (0; R_0, D, I) \\
&\quad + \mu_I \lambda_G (0; R_0, D, I) (1 - z_1 (0; R_0, D, I)) \frac{d^2 z_1}{dp_N^2} (0; R_0, D, I) \\
&\quad - \mu_D \frac{dz_2}{dp_N} (0; R_0, D, I) z_1 (0; R_0, D, I) \\
&\quad + 2\mu_D (1 - z_2 (0; R_0, D, I)) \frac{dz_2}{dp_n} (0; R_0, D, I). \tag{4.19}
\end{aligned}$$

Rearranging equation (4.19) and applying part (i) and Lemma 4.2 yields

$$\begin{aligned}
\frac{d^2 z_1}{dp_N^2} (0; R_0, D, I) [1 - R_0 (1 - z_H(R_0))] &= 2\mu_D \left( 1 - \frac{\sigma_D^2}{\mu_D} \right) \frac{z_H(R_0) (1 - z_H(R_0))}{R_0} \\
&\quad - \mu_D (3z_H(R_0) - 2) \frac{dz_2}{dp_n} (0; R_0, D, I). \tag{4.20}
\end{aligned}$$

To complete the proof of part (iii) we now only require an expression for  $\frac{dz_2}{dp_n} (0; R_0, D, I)$ , which we now formulate. Dividing equation (4.6a) by (4.6b)

yields

$$\frac{1 - z_1(p_N; R_0, D, I)}{1 - z_2(p_N; R_0, D, I)} = \mu_D \frac{G_D(1 - p_N z_2(p_N; R_0, D, I))}{G'_D(1 - p_N z_2(p_N; R_0, D, I))},$$

so

$$0 = (1 - z_1(p_N; R_0, D, I)) f'_D(1 - p_N z_2(p_N; R_0, D, I)) - \mu_D (1 - z_2(p_N; R_0, D, I)) f_D(1 - p_N z_2(p_N; R_0, D, I)). \quad (4.21)$$

Taking the derivative of equation (4.21) with respect to  $p_N$  yields

$$\begin{aligned} 0 = & - \left[ z_2(p_N; R_0, D, I) + p_N \frac{dz_2}{dp_N}(p_N; R_0, D, I) \right] \\ & \times (1 - z_1(p_N; R_0, D, I)) f''_D(1 - p_N z_2(p_N; R_0, D, I)) \\ & - \frac{dz_1}{dp_N}(p_N; R_0, D, I) f'_D(1 - p_N z_2(p_N; R_0, D, I)) \\ & + \mu_D \frac{dz_2}{dp_N}(p_N; R_0, D, I) f_D(1 - p_N z_2(p_N; R_0, D, I)) \\ & + \left[ z_2(p_N; R_0, D, I) + p_N \frac{dz_2}{dp_N}(p_N; R_0, D, I) \right] \\ & \times \mu_D (1 - z_2(p_N; R_0, D, I)) f'_D(1 - p_N z_2(p_N; R_0, D, I)). \end{aligned} \quad (4.22)$$

Evaluating equation (4.22) at  $p_N = 0$ , applying part (i) and substituting equation (4.17) yields

$$z_H(R_0) (1 - z_H(R_0)) G''_D(1) = \mu_D \frac{dz_2}{dp_N}(0; R_0, D, I) + \mu_D^2 z_H(R_0) (1 - z_H(R_0)), \quad (4.23)$$

and, substituting  $\sigma_D^2 = f''_D(1) + \mu_D - \mu_D^2$ ,

$$\begin{aligned} \mu_D \frac{dz_2}{dp_N}(0; R_0, D, I) &= z_H(R_0) (1 - z_H(R_0)) (f''_D(1) - \mu_D^2) \\ &= -z_H(R_0) (1 - z_H(R_0)) \mu_D \left( 1 - \frac{\sigma_D^2}{\mu_D} \right). \end{aligned} \quad (4.24)$$

Substituting equation (4.24) into equation (4.20) yields

$$\begin{aligned} & \frac{d^2 z_1}{dp_N^2}(0; R_0, D, I) (1 - R_0 (1 - z_H(R_0))) \\ &= \mu_D z_H(R_0) (1 - z_H(R_0)) \left( 1 - \frac{\sigma_D^2}{\mu_D} \right) \left[ \frac{2}{R_0} + 3z_H(R_0) - 2 \right]. \end{aligned} \quad (4.25)$$

Applying the chain rule to  $\frac{d^2 z_1}{d\lambda_N^2}$  and substituting equation (4.17) yields

$$\left. \frac{d^2 z_1}{d\lambda_N^2} \right|_{\lambda_G=0} = \left[ \frac{d^2 z_1}{dp_N^2} \left( \frac{dp_N}{d\lambda_N} \right)^2 + \frac{dz_1}{dp_N} \frac{d^2 p_N}{d\lambda_N^2} \right] \Big|_{\lambda_G=0} = \mu_I^2 \frac{d^2 z_1}{dp_N^2} (0; R_0, D, I),$$

and part (iii) immediately follows by substituting equation (4.25).

Finally, applying Lemma 4.1 yields part (iv) if  $2/R_0 + 3z_H(R_0) - 2 > 0$ . Rearranging equation (4.5) in Lemma 4.1 yields the inequality  $z_H(R_0) > 1 - 1/R_0 > 0$ , so

$$\frac{2}{R_0} + 3z_H(R_0) - 2 > \frac{2}{R_0} + 3 \left( 1 - \frac{1}{R_0} \right) - 2 = 1 - \frac{1}{R_0} > 0, \quad (4.26)$$

as required.  $\square$

The key result from theorem 4.1 is that fixing  $R_0$  and introducing a small amount of network heterogeneity to the homogeneously mixing model increases the final size of a major outbreak if  $\mu_D > \sigma_D^2$  and decreases the final size of a major outbreak if  $\mu_D < \sigma_D^2$ , and is illustrated in the right-hand side of Figure 4.1.

Indeed, examining the model closely shows that a bifurcation occurring at  $\sigma_D^2/\mu_D - 1$  can be predicted. Recall that the expected number of uninfected network neighbours of a type-1 and type-2 individual is given by  $\mu_D$  and  $\mu_{\bar{D}-1} = \mu_D + \sigma_D^2/\mu_D - 1$  respectively. Therefore if  $\sigma_D^2/\mu_D - 1 < 0$  then a type-1 individual is expected to have more uninfected network neighbours than a type-2 individual, whereas if  $\sigma_D^2/\mu_D - 1 > 0$  then a type-2 individual is expected to have more uninfected network neighbours than a type-1 individual. Thus increasing  $\lambda_N$  at the expense of  $\lambda_G$  will logically cause a different effect on the final size of a major outbreak depending on the sign of  $\sigma_D^2/\mu_D - 1$ .

Theorem 4.1 also shows that changing  $R_0 > 1$  or  $I$  will not effect whether the final size of a major outbreak will increase or decrease when  $R_0$  is fixed and we introduce a small amount of network heterogeneity to the homogeneously mixing model.

### 4.1.2 Independence of some degree distribution parameters and the final size of a major outbreak

In contrast to the previous section in which we consider  $\lambda_G$  to be determined by our choice of  $p_N$ ,  $R_0$ ,  $D$  and  $I$ , we now consider  $\lambda_N$  to be a value determined by  $\lambda_G$ ,  $R_0$ ,  $D$  and  $I$ . We now consider the following motivating proposition. Let  $z_{\text{Poi}(\alpha)}(R_0, \lambda_G; I)$ ,  $R_0 > 1$ ,  $\alpha > 0$ , be the final size of a major outbreak in the network and global model with degree distribution  $D \sim \text{Poi}(\alpha)$ , basic reproduction number  $R_0$ ,  $\lambda_G \in [0, R_0/\mu_I]$  and infectious period distribution  $I$ .

**Proposition 4.1.** *For  $R_0 > 1$ ,  $\alpha, \beta \geq R_0$ ,  $\lambda_G \in [0, R_0/\mu_I]$ ,  $\lambda'_G \in [0, R_0/\mu_I]$ ,  $z_{\text{Poi}(\alpha)}(R_0, \lambda_G; I)$  is the largest solution  $(0, 1]$  of the equation*

$$1 - z = e^{-R_0 z}, \quad (4.27)$$

and

$$z_{\text{Poi}(\alpha)}(R_0, \lambda_G; I) = z_{\text{Poi}(\beta)}(R_0, \lambda'_G; I'). \quad (4.28)$$

*Proof.* Fix  $\lambda_G \in [0, R_0/\mu_I]$ . Recall from Table 2.2 on page 18 that if  $D \sim \text{Poi}(\alpha)$  then  $f_D(s) = e^{-\alpha(1-s)}$  and  $f_{\tilde{D}-1}(s) = e^{-\alpha(1-s)}$ . Therefore, substituting  $f_D(s)$  and  $f_{\tilde{D}-1}(s)$  into equations (4.2), by definition  $(z_{\text{Poi}(\alpha)}(R_0, \lambda_G; I), z_2)$  is the largest solution in  $(0, 1]^2$  of the set of simultaneous equations

$$1 - z = e^{-(\lambda_G \mu_I z + \alpha p_N z_2)}, \quad (4.29a)$$

$$1 - z_2 = e^{-(\lambda_G \mu_I z + \alpha p_N z_2)}. \quad (4.29b)$$

Therefore  $z_{\text{Poi}(\alpha)}(R_0, \lambda_G; I) = z_2$  and  $z_{\text{Poi}(\alpha)}(R_0, \lambda_G; I)$  is the largest solution in  $(0, 1]$  of the equation

$$1 - z = e^{-z(\lambda_G \mu_I + \alpha p_N)}. \quad (4.30)$$

Substituting  $\mu_D = \mu_{\tilde{D}-1} = \alpha$  into equation (4.3) yields

$$R_0^2 - R_0(\lambda_G \mu_I + p_N \alpha) = 0.$$

We require  $R_0 > 1$ , so  $R_0 = \lambda_G \mu_I + p_N \alpha$  and substituting  $R_0 = \lambda_G \mu_I + p_N \alpha$  into equation (4.30) yields equation (4.27). Then, as required,  $z_{\text{Poi}(\alpha)}(R_0, \lambda_G; I)$  is the largest solution in  $(0, 1]$  of the equation

$$1 - z = e^{-R_0 z}.$$

Finally, note that equation (4.27) is independent of our choice of  $\alpha$ ,  $\lambda_G$

and  $I$ , so  $z_{\text{Poi}(\alpha)}(R_0, \lambda_G; I) = z_{\text{Poi}(\beta)}(R_0, \lambda'_G; I')$  for all  $\lambda_G \in [0, R_0/\mu_I]$  and  $\lambda'_G \in [0, R_0/\mu_{I'}]$  as required.  $\square$

Proposition 4.1 proves that the final size of a major outbreak on the network and global model with a Poisson degree distribution with parameter  $\alpha$  depends only on  $R_0$ , and not on the balance between  $\lambda_G$ ,  $\lambda_N$ ,  $\alpha$  and  $\mu_I$ .

We now show that several other degree distributions (including the Binomial and Negative binomial degree distributions) have a similar property, by which we mean that the final size of a major outbreak on the network and global model with fixed  $R_0$  is independent of some parameters of the degree distribution. To classify the distributions which we conjecture share this property we must first define thinned distributions. For a random variable  $X$  with support in  $\mathbb{Z}^+ \cup \{0\}$ , we denote its thinned distribution by  $X^T(p)$ ,  $p \in (0, 1]$ , where  $X^T(p)|X \sim \text{Bin}(X, p)$ . We conjecture that the final size of a major outbreak on the network and global model with fixed  $R_0$  is independent of some parameters of the degree distribution if the degree distribution is chosen such that all its thinned distributions belong to the same distribution, although potentially with different parameters. For example, in Proposition 4.1 we use implicitly use the result that if  $D \sim \text{Poi}(\alpha)$ , then  $D^T(p) \sim \text{Poi}(p\alpha)$ ,  $p \in (0, 1]$ . However, a rigorous proof of this statement has proven to be elusive. We now present two propositions considering the special cases of a Binomial and Negative Binomial degree distribution respectively.

**Remark.** *In the standard network model with degree distribution  $D$ , the basic reproduction number  $R_0 = p_N \mu_{\bar{D}-1}$ ,  $p_N \in [0, 1]$ . Therefore fixing  $R_0$  and the degree distribution in the network and global model, imposes constraints on the range of possible global infection rates, e.g. if  $R_0 > \mu_{\bar{D}-1}$  then there is a minimum value of  $\lambda_G$  required to achieve the desired  $R_0$ .*

Let  $z_{\text{Bin}(n,p)}(R_0, \lambda_G; I)$  be the final size of a major outbreak in the network and global model with degree distribution  $D \sim \text{Bin}(n, p)$  ( $n \in \mathbb{Z}^+$ ,  $p \in (0, 1]$ ), global infection rate  $\lambda_G$  and basic reproduction number  $R_0$ . To compare the homogeneously mixing model with the standard network model with this degree distribution we require that  $n$  and  $R_0$  are chosen such that there is a positive probability of a major outbreak for some  $p \in (0, 1]$ , which is satisfied if the inequality  $(n - 1) \geq R_0 > 1$  holds. Finally, fixing the degree distribution and

$R_0$  imposes the following restriction upon  $\lambda_G$ ,

$$\lambda_G \in [\max \{0, R_0 (R_0 - p(n-1)) / (p + R_0) \mu_I\}, R_0 / \mu_I]. \quad (4.31)$$

We now show that the final size of a major outbreak on the network and global model with  $D \sim \text{Bin}(n, p)$  and fixed  $R_0$  and  $\lambda_G$  is independent of our choice of  $p$ .

**Proposition 4.2.** *For  $n \in \mathbb{Z}^+$ ,  $p, p_1 \in (0, 1]$ ,  $(n-1) \geq R_0 > 1$  and  $\lambda_G$  satisfying equation (4.31),  $(z_{\text{Bin}(n,p)}(R_0, \lambda_G; I), z_2)$  is the largest solution in  $(0, 1]^2$  of the set of simultaneous equations*

$$1 - z_1 = e^{-\lambda_G \mu_I z_1} \left( 1 - \frac{z_2 R_0 (R_0 - \lambda_G \mu_I)}{\lambda_G \mu_I + R_0 (n-1)} \right)^n, \quad (4.32a)$$

$$1 - z_2 = e^{-\lambda_G \mu_I z_1} \left( 1 - \frac{z_2 R_0 (R_0 - \lambda_G \mu_I)}{\lambda_G \mu_I + R_0 (n-1)} \right)^{n-1}. \quad (4.32b)$$

Furthermore,

$$z_{\text{Bin}(n,p)}(R_0, \lambda_G; I) = z_{\text{Bin}(n,p_1)}(R_0, \lambda_G; I).$$

*Proof.* The proof of Proposition 4.2 proceeds similarly to that of Proposition 4.1. Recall from Table 2.2 on page 18 that if  $D \sim \text{Bin}(n, p)$  then  $f_D(s) = (1 - p + ps)^n$  and  $f_{\tilde{D}-1}(s) = (1 - p + ps)^{n-1}$ . Therefore, substituting  $f_D(s)$  and  $f_{\tilde{D}-1}(s)$  into equations (4.2),  $(z_{\text{Bin}(n,p_1)}(R_0, \lambda_G; I), z_2)$  is the largest solution in  $(0, 1]^2$  of the set of simultaneous equations

$$1 - z_{\text{Bin}(n,p)}(R_0, \lambda_G; I) = e^{-\lambda_G \mu_I z_{\text{Bin}(n,p)}(R_0, \lambda_G; I)} (1 - pp_N z_2)^n, \quad (4.33a)$$

$$1 - z_2 = e^{-\lambda_G \mu_I z_{\text{Bin}(n,p)}(R_0, \lambda_G; I)} (1 - pp_N z_2)^{n-1}. \quad (4.33b)$$

Substituting  $\mu_D = np$  and  $\mu_{\tilde{D}-1} = (n-1)p$  into equation (4.3) yields

$$R_0^2 - R_0 (\lambda_G \mu_I + (n-1)pp_N) - \lambda_G \mu_I pp_N = 0. \quad (4.34)$$

Thus, rearranging equation (4.34) for  $pp_N$ ,

$$pp_N = \frac{R_0 (R_0 - \lambda_G \mu_I)}{\lambda_G \mu_I + R_0 (n-1)}. \quad (4.35)$$

Substituting equation (4.35) into equations (4.33) yields equations (4.32). Thus, as required,  $(z_{\text{Bin}(n,p)}(R_0, \lambda_G; I), z_2)$  is the largest solution in  $(0, 1]^2$  of the set of



simultaneous equations

$$\begin{aligned} 1 - z_1 &= e^{-\lambda_G \mu_I z_1} \left( 1 - \frac{z_2 R_0 (R_0 - \lambda_G \mu_I)}{\lambda_G \mu_I + R_0 (n - 1)} \right)^n, \\ 1 - z_2 &= e^{-\lambda_G \mu_I z_1} \left( 1 - \frac{z_2 R_0 (R_0 - \lambda_G \mu_I)}{\lambda_G \mu_I + R_0 (n - 1)} \right)^{n-1}. \end{aligned}$$

Finally, equations (4.32) are independent of our choice of  $p$ , so the equality  $z_{\text{Bin}(n,p)}(R_0, \lambda_G; I) = z_{\text{Bin}(n,p_1)}(R_0, \lambda_G; I)$  holds as required.  $\square$

Let  $z_{\text{NB}(r,p)}(R_0, \lambda_G; I)$  be the final size of a major outbreak in the network and global model with degree distribution  $D \sim \text{NB}(r, p)$  ( $r \in \mathbb{Z}^+, p \in (0, 1)$ ), global infection rate  $\lambda_G$  and basic reproduction number  $R_0 > 1$ . Fixing the degree distribution and  $R_0$  imposes the following restriction on  $\lambda_G$ ,

$$\lambda_G \in [\max \{0, R_0 (R_0 (1 - p) - p(r + 1)) / (R_0 (1 - p) - p) \mu_I\}, R_0 / \mu_I]. \quad (4.36)$$

We now show that the final size of a major outbreak on the network and global model with  $D \sim \text{NB}(r, p)$  and fixed  $R_0$  and  $\lambda_G$  is independent of our choice of  $p$ .

**Proposition 4.3.** *For  $r \in \mathbb{Z}^+, p, p_1 \in (0, 1), R_0 > 1$  and  $\lambda_G$  given in equation (4.31),  $(z_{\text{NB}(r,p)}(R_0, \lambda_G; I), z_2)$  is the largest solution in  $(0, 1]^2$  of the set of simultaneous equations*

$$1 - z_1 = e^{-\lambda_G \mu_I z_1} \left( 1 + z_2 \frac{R_0 (R_0 - \lambda_G \mu_I)}{R_0(r + 1) - \lambda_G \mu_I} \right)^{-r}, \quad (4.37a)$$

$$1 - z_2 = e^{-\lambda_G \mu_I z_1} \left( 1 + z_2 \frac{R_0 (R_0 - \lambda_G \mu_I)}{R_0(r + 1) - \lambda_G \mu_I} \right)^{-(r+1)}. \quad (4.37b)$$

and

$$z_{\text{NB}(r,p)}(R_0, \lambda_G; I) = z_{\text{NB}(r,p_1)}(R_0, \lambda_G; I). \quad (4.38)$$

*Proof.* The proof of Proposition 4.3 proceeds similarly to that of Propositions 4.1 and 4.2. Recall from Table 2.2 on page 18 that if  $D \sim \text{NB}(r, p)$  then  $f_D(s) = ((1 - p)/(1 - ps))^r$  and  $f_{\tilde{D}-1}(s) = ((1 - p)/(1 - ps))^{r+1}$ . Therefore, substituting  $f_D(s)$  and  $f_{\tilde{D}-1}(s)$  into equations (4.2),  $(z_{\text{NB}(r,p_1)}(R_0, \lambda_G; I), z_2)$  is the largest solution in  $(0, 1]^2$  of the set of simultaneous equations

$$1 - z_1 = e^{-\lambda_G \mu_I z_1} \left( \frac{1 - p}{1 - p + pp_N z_2} \right)^r, \quad (4.39a)$$

$$1 - z_2 = e^{-\lambda_G \mu_I z_1} \left( \frac{1 - p}{1 - p + pp_N z_2} \right)^{r+1}. \quad (4.39b)$$

Substituting  $\mu_D = rp/(1-p)$  and  $\mu_{\bar{D}-1} = (r+1)p/(1-p)$  into equation (4.3) yields

$$R_0^2 - R_0 \left( \lambda_G \mu_I + \frac{(r+1)pp_N}{(1-p)} \right) - \frac{\lambda_G \mu_I pp_N}{(1-p)} = 0. \quad (4.40)$$

Thus, rearranging equation (4.40) for  $pp_N/(1-p)$ ,

$$\frac{pp_N}{1-p} = \frac{R_0 (R_0 - \lambda_G \mu_I)}{R_0(r+1) - \lambda_G \mu_I}. \quad (4.41)$$

Substituting equation (4.41) into equations (4.39) yields equations (4.37) as required. Finally, equations (4.37) are independent of our choice of  $p$ , so  $z_{\text{NB}(r,p)}(R_0, \lambda_G) = z_{\text{NB}(r,p_1)}(R_0, \lambda_G)$  as required.  $\square$

### 4.1.3 Ordering the final size of a major outbreak in the standard network model by degree distribution

We now use Propositions 4.1, 4.2 and 4.3 to discuss an ordering of the final size of a major outbreak on the standard network model with various degree distributions, corresponding to the left-hand side of Figure 4.1, i.e.  $\lambda_G = 0$ .

Recall that in the standard network model with degree distribution  $D$ ,  $R_0 = p_N \mu_{\bar{D}-1}$ . Therefore, for fixed  $D$  we require  $R_0 \in (1, \mu_{\bar{D}-1}]$ . Furthermore, if  $D$  and  $R_0$  are fixed then  $p_N$  is immediately determined by the equation  $p_N = R_0 / \mu_{\bar{D}-1}$ .

We reuse the final size notation introduced in Section 4.1.2 with a slight modification. Since we only consider the case  $\lambda_G = 0$  we drop the second parameter,  $\lambda_G$ . For example,  $z_{\text{Bin}(n,p)}(R_0, 0; I) = z_{\text{Bin}(n,p)}(R_0; I)$  denotes the final size of a major outbreak in the standard network model with  $D \sim \text{Bin}(n, p)$ , basic reproduction number  $R_0$  and infectious period distribution  $I$ .

To order the final size of a major outbreak on the standard network model with different degree distributions we introduce the following lemma. Let  $D_1$  and  $D_2$  be two non-negative random variables with  $\min(\mu_{\bar{D}_1-1}, \mu_{\bar{D}_2-1}) > 1$  and fix  $R_0 \in (1, \min(\mu_{\bar{D}_1-1}, \mu_{\bar{D}_2-1})]$ . Let  $z_{D_1}(R_0; I) < 1$  and  $z_{D_2}(R_0; I) < 1$  be the final size of a major outbreak in the standard network model with basic reproduction number  $R_0$ , infectious period distribution  $I$  and degree distributions  $D_1$  and  $D_2$  respectively.

**Lemma 4.3.** *If, for all  $s \in (0, 1)$ ,*

$$f_{D_1} \left( 1 - \frac{R_0 s}{\mu_{\tilde{D}_1-1}} \right) < f_{D_2} \left( 1 - \frac{R_0 s}{\mu_{\tilde{D}_2-1}} \right), \quad (4.42a)$$

$$f_{\tilde{D}_1-1} \left( 1 - \frac{R_0 s}{\mu_{\tilde{D}_1-1}} \right) < f_{\tilde{D}_2-1} \left( 1 - \frac{R_0 s}{\mu_{\tilde{D}_2-1}} \right), \quad (4.42b)$$

then

$$z_{D_1}(R_0; I) > z_{D_2}(R_0; I).$$

*Proof.* For  $i = 1, 2$ , the final size of a major outbreak in the standard network model with degree distribution  $D_i$  is given by  $z_{D_i}(R_0; I) = 1 - f_{D_i} \left( 1 - \frac{R_0 \bar{z}_{D_i}}{\mu_{\tilde{D}_i-1}} \right)$ , where  $\bar{z}_{D_i;I} \in (0, 1]$  is the largest solution in  $(0, 1]$  of the equation

$$1 - \bar{z}_{D_i} = f_{\tilde{D}_i-1} \left( 1 - \frac{R_0 \bar{z}_{D_i}}{\mu_{\tilde{D}_i-1}} \right).$$

Since  $\bar{z}_{D_1}, \bar{z}_{D_2} \in (0, 1]$ , applying inequality (4.42b) yields

$$1 - \bar{z}_{D_1} = f_{\tilde{D}_1-1} \left( 1 - \frac{R_0 \bar{z}_{D_1}}{\mu_{\tilde{D}_1-1}} \right) < f_{\tilde{D}_2-1} \left( 1 - \frac{R_0 \bar{z}_{D_2}}{\mu_{\tilde{D}_2-1}} \right) = 1 - \bar{z}_{D_2}.$$

Therefore  $\bar{z}_{D_1} > \bar{z}_{D_2}$ .

For  $s \in (0, 1)$ ,  $f_{D_1} \left( 1 - \frac{R_0 s}{\mu_{\tilde{D}_1-1}} \right)$  and  $f_{D_2} \left( 1 - \frac{R_0 s}{\mu_{\tilde{D}_2-1}} \right)$  are both decreasing functions in  $s$ . So applying inequality (4.42a) and then the inequality  $\bar{z}_{D_1} > \bar{z}_{D_2}$  yields

$$\begin{aligned} 1 - z_{D_1}(R_0; I) &= f_{D_1} \left( 1 - \frac{R_0 \bar{z}_{D_1}}{\mu_{\tilde{D}_1-1}} \right) \\ &< f_{D_2} \left( 1 - \frac{R_0 \bar{z}_{D_1}}{\mu_{\tilde{D}_2-1}} \right) \\ &< f_{D_2} \left( 1 - \frac{R_0 \bar{z}_{D_2}}{\mu_{\tilde{D}_2-1}} \right) \\ &= 1 - z_{D_2}(R_0; I). \end{aligned}$$

Thus  $z_{D_1}(R_0; I) > z_{D_2}(R_0; I)$  as required.  $\square$

We now use Lemma 4.3 to prove the following theorem which gives an ordering for the final size of a major outbreak in the standard network model with Binomial, Poisson and Negative Binomial degree distributions. The inequalities in Theorem 4.2 hold for an appropriate choice of  $R_0$ , by which we mean that in

an inequality involving the final size of a major outbreak in two standard network models with degree distributions  $D_1$  and  $D_2$ ,  $R_0 \in \left(1, \min(\mu_{\tilde{D}_1-1}, \mu_{\tilde{D}_2-1})\right]$ .

**Theorem 4.2.** *For  $n, i, r, j \in \mathbb{Z}^+$ ,  $n \geq 2$ ,  $\alpha > 0$ ,  $p_1, p_2, p_3, p_4 \in (0, 1]$  and appropriate choice of  $R_0 > 1$  the following inequalities hold.*

$$\begin{aligned} z_{\text{Bin}(n, p_1)}(R_0; I) &> z_{\text{Bin}(n+i, p_2)}(R_0; I) > z_{\text{Poi}(\alpha)}(R_0; I), \\ z_{\text{Poi}(\alpha)}(R_0; I) &> z_{\text{NB}(r+j, p_3)}(R_0; I) > z_{\text{NB}(r, p_4)}(R_0; I). \end{aligned}$$

*Proof.* The proof of each inequality follows a similar layout. We use Propositions 4.1, 4.2 and 4.3 to remove the dependence of the final size of a major outbreak on our choice of  $p_1, p_2, p_3, p_4$  and then apply Lemma 4.3. However, checking the constraints required to apply Lemma 4.3 requires slightly different arguments for each inequality.

Substituting the appropriate probability generating functions of the degree distributions into equations (4.42) and applying Lemma 4.3 yields that the inequality  $z_{\text{Bin}(n, p_1)}(R_0; I) > z_{\text{Bin}(n+i, p_2)}(R_0; I)$  holds if, for all  $s \in (0, 1)$ ,

$$\left(1 - \frac{sR_0}{n-1}\right)^n < \left(1 - \frac{sR_0}{n+i-1}\right)^{n+i} \quad (4.43a)$$

and

$$\left(1 - \frac{sR_0}{n-1}\right)^{n-1} < \left(1 - \frac{sR_0}{n+i-1}\right)^{n+i-1} \quad (4.43b)$$

Taking the logarithm of both sides of inequality (4.43b) and Taylor expanding yields

$$\begin{aligned} &\left(1 - \frac{sR_0}{n-1}\right)^{n-1} < \left(1 - \frac{sR_0}{n+i-1}\right)^{n+i-1} \\ \iff &(n-1) \log \left(1 - \frac{sR_0}{n-1}\right) < (n+i-1) \log \left(1 - \frac{sR_0}{n+i-1}\right) \\ \iff &(n-1) \sum_{j=1}^{\infty} \frac{-(sR_0)^j}{j!(n-1)^j} < (n+i-1) \sum_{j=1}^{\infty} \frac{-(sR_0)^j}{j!(n+i-1)^j} \\ \iff &sR_0 + \frac{s^2 R_0^2}{2(n-1)} + \dots > sR_0 + \frac{s^2 R_0^2}{2(n+i-1)} + \dots \end{aligned} \quad (4.44)$$

Inequality (4.44) clearly holds for  $i > 0$ , so inequality (4.43b) holds. Similarly, taking the logarithm of both sides of inequality (4.43a) and Taylor expanding

yields

$$\begin{aligned}
& \left(1 - \frac{sR_0}{n-1}\right)^n < \left(1 - \frac{sR_0}{n+i-1}\right)^{n+i} \\
\iff & n \sum_{j=1}^{\infty} \frac{-(sR_0)^j}{j!(n-1)^j} < (n+i) \sum_{j=1}^{\infty} \frac{-(sR_0)^j}{j!(n+i-1)^j} \\
\iff & \frac{n}{n-1} > \frac{(n+i)}{n+i-1} \text{ and } \frac{1}{n-1} > \frac{1}{n+i-1}. \tag{4.45}
\end{aligned}$$

Inequalities (4.45) clearly hold for  $i > 0$ , so inequality (4.43a) holds and  $z_{\text{Bin}(n,p_1)}(R_0; I) > z_{\text{Bin}(n+i,p_2)}(R_0; I)$ .

We next consider the inequality  $z_{\text{Bin}(n+i,p_2)}(R_0; I) > z_{\text{Poi}(\alpha)}(R_0; I)$ . Substituting the appropriate probability generating functions of the degree distributions into equations (4.42), the inequality  $z_{\text{Bin}(n+i,p_2)}(R_0; I) > z_{\text{Poi}(\alpha)}(R_0; I)$  follows directly from Lemma 4.3 if, for all  $s \in (0, 1)$ ,

$$\left(1 - \frac{sR_0}{n+i-1}\right)^{n+i} < e^{-sR_0} \tag{4.46a}$$

and

$$\left(1 - \frac{sR_0}{n+i-1}\right)^{n+i-1} < e^{-sR_0}. \tag{4.46b}$$

Since  $1 - \frac{sR_0}{n+i-1} \in (0, 1)$ , it is clear that inequality (4.46a) holds if inequality (4.46b) holds. Taking the logarithm of both sides of inequality (4.46b) and Taylor expanding yields

$$\begin{aligned}
& \left(1 - \frac{sR_0}{n+i-1}\right)^{n+i-1} < e^{-sR_0} \\
\iff & (n-1) \log \left(1 - \frac{sR_0}{n-1}\right) < -sR_0 \\
\iff & (n-1) \sum_{j=1}^{\infty} \frac{-(sR_0)^j}{j!(n-1)^j} < -sR_0 \\
\iff & sR_0 + \frac{s^2 R_0^2}{2(n-1)} + \dots > sR_0,
\end{aligned}$$

which clearly holds. Therefore inequality (4.46b) holds and so, as required,  $z_{\text{Bin}(n+i,p_2)}(R_0; I) > z_{\text{Poi}(\alpha)}(R_0; I)$ .

We now turn our attention to the inequality  $z_{\text{Poi}(\alpha)}(R_0; I) > z_{\text{NB}(r+j,p_3)}(R_0; I)$ . Substituting the appropriate probability generating functions of the degree dis-

tributions into equations (4.42), the inequality  $z_{\text{Poi}(\alpha)}(R_0; I) > z_{\text{NB}(r+j, p_3)}(R_0; I)$  follows directly from Lemma 4.3 if, for all  $s \in (0, 1)$ ,

$$e^{-sR_0} < \left(1 + \frac{sR_0}{r+j+1}\right)^{-r-j} \quad (4.47a)$$

and

$$e^{-sR_0} < \left(1 + \frac{sR_0}{r+j+1}\right)^{-r-j-1}. \quad (4.47b)$$

Note that, since  $1 + \frac{sR_0}{r+j+1} > 1$ , if inequality (4.47b) holds then inequality (4.47a) also holds. We therefore need only consider inequality (4.47b) where, taking logarithms,

$$\begin{aligned} e^{-sR_0} &< \left(1 + \frac{sR_0}{r+j+1}\right)^{-r-j-1} \\ \iff -sR_0 &< (-r-j-1) \log \left(1 + \frac{sR_0}{r+j+1}\right) \\ \iff \frac{sR_0}{r+j+1} &> \log \left(1 + \frac{sR_0}{r+j+1}\right). \end{aligned} \quad (4.48)$$

Clearly, for  $x > 0$ ,  $\log(1+x) < x$  and, since  $sR_0/(r+j+1) > 0$ , inequality (4.48) holds. Thus  $z_{\text{Poi}(\alpha)}(R_0; I) > z_{\text{NB}(r+j, p_3)}(R_0; I)$ .

Finally, consider the inequality  $z_{\text{NB}(r+j, p_3)}(R_0; I) > z_{\text{NB}(r, p_4)}(R_0; I)$ . Substituting the appropriate probability generating functions of the degree distributions into equations (4.42), the inequality  $z_{\text{NB}(r+j, p_3)}(R_0; I) > z_{\text{NB}(r, p_4)}(R_0; I)$  follows directly from Lemma 4.3 if, for all  $s \in (0, 1)$ ,

$$\left(\frac{1}{1 + \frac{sR_0}{r+j+1}}\right)^{r+j} < \left(\frac{1}{1 + \frac{sR_0}{r+1}}\right)^r \quad (4.49a)$$

and

$$\left(\frac{1}{1 + \frac{sR_0}{r+j+1}}\right)^{r+j+1} < \left(\frac{1}{1 + \frac{sR_0}{r+1}}\right)^{r+1}. \quad (4.49b)$$

Rearranging inequality (4.49a), noting that  $|1/(1 + sR_0/(r+j+1))| < 1$

and  $|1/(1 + sR_0/(r + 1))| < 1$ , taking logarithms and then Taylor expanding,

$$\begin{aligned}
& \left( \frac{1}{1 + \frac{sR_0}{r+j+1}} \right)^{r+j} < \left( \frac{1}{1 + \frac{sR_0}{r+1}} \right)^r \\
& \iff \left( \frac{r+j+1}{r+j+1+sR_0} \right)^{r+j} < \left( \frac{sR_0}{r+1+sR_0} \right)^r \\
& \iff \left( 1 - \frac{sR_0}{r+j+1+sR_0} \right)^{r+j} < \left( 1 - \frac{sR_0}{r+1+sR_0} \right)^r \\
& \iff (r+j) \log \left( 1 - \frac{sR_0}{r+j+1+sR_0} \right) < r \log \left( 1 - \frac{sR_0}{r+1+sR_0} \right) \\
& \iff -(r+j) \sum_{n=1}^{\infty} \frac{1}{n} \left( \frac{sR_0}{r+j+1+sR_0} \right)^n < -r \sum_{n=1}^{\infty} \frac{1}{n} \left( \frac{sR_0}{r+1+sR_0} \right)^n \quad (4.50)
\end{aligned}$$

Rearranging inequality (4.50), inequality (4.49a) holds if

$$\sum_{n=1}^{\infty} \frac{1}{n} \left( \frac{sR_0}{(r+j+1+sR_0)(r+1+sR_0)} \right)^n [j(1+sR_0)] > 0,$$

which clearly holds. An analogous argument for inequality (4.49b) yields

$$\begin{aligned}
& \left( \frac{1}{1 + \frac{sR_0}{r+j+1}} \right)^{r+j+1} - \left( \frac{1}{1 + \frac{sR_0}{r+1}} \right)^{r+1} > 0 \\
& \iff \sum_{n=1}^{\infty} \frac{(sR_0)^n}{n} \left( \frac{r+1+sR_0}{r+j+1+sR_0} \right)^n [jsR_0] > 0,
\end{aligned}$$

which again clearly holds, so  $z_{\text{NB}(r+j,p_3)}(R_0; I) > z_{\text{NB}(r,p_4)}(R_0; I)$ .  $\square$

Note that the orderings given in Theorem 4.2 are illustrated in the left-hand side of Figure 4.1, i.e.  $\lambda_G = 0$ . Furthermore, Theorem 4.2 suggests that, for fixed  $R_0$ , increasing  $\sigma_D^2$  will decrease the final size of a major outbreak on the standard network model.

Recall from Theorem 4.1 that in the network and global model with degree distribution  $D$ , if  $\sigma_D^2 < \mu_D$  then adding a small amount of network heterogeneity to the homogeneously mixing model will increase the final size of a major outbreak and if  $\sigma_D^2 > \mu_D$  then adding a small amount of network heterogeneity to the homogeneously mixing model will decrease the final size of a major outbreak. For each of the degree distributions considered in Theorem 4.2, if  $\sigma_D^2 < \mu_D$  then the final size of a major outbreak is larger than  $z_H(R_0)$ , and if  $\sigma_D^2 > \mu_D$  then the final size of a major outbreak is smaller than  $z_H(R_0)$ .

This supports the conjecture that, for the distributions discussed in Theorem 4.2, for fixed  $R_0$  there is a monotonic increase or decrease in the final size of a major outbreak as  $\lambda_G$  is increased and  $\lambda_N$  is decreased (as illustrated in Figure 4.1). Furthermore, whether the final size of a major outbreak is increasing or decreasing is determined by  $\text{sgn}\left(1 - \frac{\sigma_D^2}{\mu_D}\right)$ .

#### 4.1.4 Analysis of the network and global model with a constant degree distribution

Investigating the effect of fixing  $R_0$  and substantially increasing the network heterogeneity in the network and global model on the final size of a major outbreak is mathematically difficult, which is why in Sections 4.1.1 and 4.1.3 we consider the special cases  $\lambda_G = R_0/\mu_I$  and  $\lambda_G = 0$  respectively. However, we now consider the special case of the network and global model with a constant degree distribution and give analytical results for a larger range of  $\lambda_G \in (0, R_0/\mu_I)$ .

In this section we prove that, for  $p_N$  and  $z$  large enough, fixing  $R_0$  and decreasing  $\lambda_G$  while increasing  $\lambda_N$  will increase the final size of a major outbreak in the network and global model. This result supports the conjecture that fixing  $R_0$  and moving from the homogeneously mixing model to the standard network model with a constant degree distribution will monotonically increase the final size of a major outbreak.

Applying the notational style of Section 4.1.1, the final size of a major outbreak on the network and global model  $z_1(p_N; R_0, D, I) = 1 - \pi_1(p_N; R_0, D, I)$ , where  $\boldsymbol{\pi}(p_N; R_0, D, I) = (\pi_1(p_N; R_0, D, I), \pi_2(p_N; R_0, D, I))$  is the smallest solution to the set of simultaneous equations

$$\boldsymbol{\pi}(p_N; R_0, D, I) = \mathbf{b}(\boldsymbol{\pi}(p_N; R_0, D, I), p_N; R_0, D, I),$$

where

$$b_1(\mathbf{s}, p_N; R_0, D, I) = e^{-\lambda_G(p_N; R_0, D, I)\mu_I(1-s_1)} f_D(1 - p_N + p_N s_2), \quad (4.51a)$$

$$b_2(\mathbf{s}, p_N; R_0, D, I) = e^{-\lambda_G(p_N; R_0, D, I)\mu_I(1-s_1)} f_{\bar{D}-1}(1 - p_N + p_N s_2). \quad (4.51b)$$

Note that since  $p_N = 1 - \phi_I(\lambda_N)$  is monotonically increasing in  $\lambda_N$ , if



$z(p_N; R_0, D, I) < z(p_N + \epsilon; R_0, D, I)$  then, for  $\hat{\epsilon} > 0$ ,

$$z(p_N(\lambda_N); R_0, D, I) < z(p_N(\lambda_N + \hat{\epsilon}); R_0, D, I).$$

In contrast to the previous sections in this Chapter, where we directly consider the final size of a major outbreak, in this section we consider an ordering for the extinction probabilities of the corresponding branching processes, from which an ordering on the final size of a major outbreak immediately follows.

Lemma 4.4 makes rigorous the intuitive idea that increasing both of the probability generating functions of the offspring distributions of a two-type branching process will decrease the probability of extinction of the branching process. Note that if the two-type branching process approximates the spread of an individuals susceptibility set then reducing the extinction probability of the branching process will increase final size of a major outbreak.

**Lemma 4.4.** *If*

$$\frac{\partial b_1}{\partial p_N}(\pi(p_N; R_0, D, I), p_N; R_0, D, I) < 0, \quad (4.52a)$$

and

$$\frac{\partial b_2}{\partial p_N}(\pi(p_N; R_0, D, I), p_N; R_0, D, I) < 0, \quad (4.52b)$$

then, for  $\epsilon > 0$ ,  $\pi(p_N + \epsilon; R_0, D, I) < \pi(p_N; R_0, D, I)$ .

*Proof.* We begin by recalling the following notation and result from Section 2.3. For  $i = 1, 2$ , let

$$b_i^{(0)}(\mathbf{s}, p_N; R_0, D, I) = b_i(\mathbf{0}, p_N; R_0, D, I),$$

and, for  $n = 1, 2, \dots$ ,

$$\begin{aligned} b_i^{(n)}(\mathbf{s}, p_N; R_0, D, I) &= b_i(b_1^{(n-1)}(\mathbf{s}, p_N; R_0, D, I), b_2^{(n-1)}(\mathbf{s}, p_N; R_0, D, I), p_N; R_0, D, I) \\ &= b_i^{n-1}(b_1(\mathbf{s}, p_N; R_0, D, I), b_2(\mathbf{s}, p_N; R_0, D, I), p_N; R_0, D, I). \end{aligned}$$

Furthermore, for  $i = 1, 2$  and any  $\mathbf{s} \in [0, 1]^2$ ,  $\mathbf{s} \neq (1, 1)$ ,

$$\lim_{n \rightarrow \infty} b_i^{(n)}(\mathbf{s}, p_N; R_0, D, I) = \pi_i(p_N; R_0, D, I).$$

We now give a sufficient condition for  $\boldsymbol{\pi}(p_N + \epsilon; R_0, D, I) < \boldsymbol{\pi}(p_N; R_0, D, I)$  to hold, and then prove that the sufficient condition holds given equations (4.52) hold. For  $n = 1, 2, \dots, i = 1, 2$ , if

$$b_i^{(n)}(\boldsymbol{\pi}(p_N; R_0, D, I), p_N + \epsilon; R_0, D, I) < \pi_i(p_N; R_0, D, I) \quad (4.53)$$

then

$$\begin{aligned} \pi_i(p_N + \epsilon; R_0, D, I) &= \lim_{n \rightarrow \infty} b_i^{(n)}(\boldsymbol{\pi}(p_N; R_0, D, I), p_N + \epsilon; R_0, D, I) \\ &< \lim_{n \rightarrow \infty} \pi_i(p_N; R_0, D, I) \\ &= \pi_i(p_N; R_0, D, I). \end{aligned}$$

Therefore to prove Lemma 4.4 it is sufficient to prove that equation (4.53) holds. We first consider the base case, i.e. for  $i = 1, 2$ ,

$$b_i(\boldsymbol{\pi}(p_N; R_0, D, I), p_N + \epsilon; R_0, D, I) < \pi_i(p_N; R_0, D, I)$$

Applying the limit definition of the partial derivative to equations (4.52) with respect to  $p_N$  yields, for  $i = 1, 2$ ,

$$b_i(\boldsymbol{\pi}(p_N; R_0, D, I), p_N + \epsilon; R_0, D, I) < b_i(\boldsymbol{\pi}(p_N; R_0, D, I), p_N; R_0, D, I), \quad (4.54)$$

and the base case follows noting that

$$b_i(\boldsymbol{\pi}(p_N; R_0, D, I), p_N; R_0, D, I) = \pi_i(p_N; R_0, D, I).$$

For the induction hypothesis assume that, for  $i = 1, 2, n = 1, 2, \dots$ ,

$$b_i^{(n)}(\boldsymbol{\pi}(p_N; R_0, D, I), p_N + \epsilon; R_0, D, I) < \pi_i(p_N; R_0, D, I),$$

and consider  $b_i^{(n+1)}(\boldsymbol{\pi}(p_N; R_0, D, I), p_N + \epsilon; R_0, D, I)$ . Applying equation (2.8) from Section 2.3, noting that  $b_i(\mathbf{s}, p_N; R_0, D, I)$  is an increasing function of  $\mathbf{s}$ ,

applying the induction hypothesis and then applying inequality (4.54) yields

$$\begin{aligned}
& b_i^{(n+1)}(\boldsymbol{\pi}(p_N; R_0, D, I), p_N + \epsilon; R_0, D, I) \\
&= b_i(\mathbf{b}^{(n)}(\boldsymbol{\pi}(p_N; R_0, D, I), p_N + \epsilon; R_0, D, I), p_N + \epsilon; R_0, D, I) \\
&< b_i(\pi_1(p_N; R_0, D, I), \pi_2(p_N; R_0, D, I), p_N + \epsilon; R_0, D, I) \\
&< b_i(\pi_1(p_N; R_0, D, I), \pi_2(p_N; R_0, D, I), p_N; R_0, D, I) \\
&= \pi_i(p_N; R_0, D, I),
\end{aligned}$$

thus completing the proof of Lemma 4.4.  $\square$

The following theorem shows that fixing  $R_0$  and increasing  $p_N$  while decreasing  $\lambda_G$  in the network and global model with a constant degree distribution will monotonically increase the final size of a major outbreak if  $p_N$  and the final size of a major outbreak are large enough.

**Theorem 4.3.** *Let  $D \sim \text{Const}(d)$ ,  $d \geq 2$ . Then, for  $R_0 > 1$  and  $\epsilon > 0$ , if*

$$0 < \boldsymbol{\pi}(p_N; R_0, D, I) = \mathbf{b}(\boldsymbol{\pi}(p_N; R_0, D, I), p_N; R_0, D, I)$$

and

$$\begin{aligned}
0 &< p_N \left[ 1 + \pi_2(p_N; R_0, D, I) (1 - \pi_2(p_N; R_0, D, I)) R_0^2 \right] \\
&\quad + R_0 [2 + R_0 (1 - 2\pi_2(p_N; R_0, D, I))], \tag{4.55a}
\end{aligned}$$

$$\begin{aligned}
0 &< p_N^2 \left[ (d-1) + d\pi_2(p_N; R_0, D, I) (1 - \pi_2(p_N; R_0, D, I)) R_0^2 \right] \\
&\quad + p_N R_0 [2(d-1) + dR_0 (1 - 2\pi_2(p_N; R_0, D, I))] - R_0^2, \tag{4.55b}
\end{aligned}$$

then

$$z(p_N; R_0, \text{Const}(d), I) < z(p_N + \epsilon; R_0, \text{Const}(d), I).$$

*Proof.* Recall that  $z(p_N; R_0, D, I) = 1 - \pi_1(p_N; R_0, D, I)$ . So if

$$\boldsymbol{\pi}(p_N + \epsilon; R_0, D, I) < \boldsymbol{\pi}(p_N; R_0, D, I),$$

then  $z(p_N; R_0, D, I) < z(p_N + \epsilon; R_0, D, I)$ . Therefore, by Lemma 4.4, to prove Theorem 4.3 it is sufficient to show that, given that the assumptions given in equation (4.55) hold,

$$\frac{\partial b_1}{\partial p_N}(\boldsymbol{\pi}(p_N; R_0, D, I), p_N; R_0, D, I) < 0, \tag{4.56a}$$

and

$$\frac{\partial b_2}{\partial p_N} (\boldsymbol{\pi} (p_N; R_0, D, I), p_N; R_0, D, I) < 0. \quad (4.56b)$$

Substituting  $f_D(s) = s^d$  and  $f_{\tilde{D}-1}(s) = s^{d-1}$  into equations (4.51) yields

$$b_1(\mathbf{s}, p_N; R_0, D, I) = e^{-\lambda_G(p_N; R_0, D, I) \mu_I(1-s_1)} (1 - p_N + p_N s_2)^d, \quad (4.57a)$$

$$b_2(\mathbf{s}, p_N; R_0, D, I) = e^{-\lambda_G(p_N; R_0, D, I) \mu_I(1-s_1)} (1 - p_N + p_N s_2)^{d-1}. \quad (4.57b)$$

Thus, considering equations (4.57),

$$b_1(\mathbf{s}, p_N; R_0, D, I) = b_2(\mathbf{s}, p_N; R_0, D, I) (1 - p_N + p_N s_2). \quad (4.57c)$$

Furthermore, substituting  $\mu_D = d$  and  $\mu_{\tilde{D}-1} = d - 1$  into equation (4.3) yields

$$\lambda_G(p_N; R_0, D, I) = \frac{R_0(R_0 - p_N(d-1))}{\mu_I(R_0 + p_N)}. \quad (4.58)$$

Taking the derivative of equations (4.57a), (4.57b) and (4.58), with respect to  $p_N$  yields

$$\begin{aligned} \frac{db_1}{dp_N}(\mathbf{s}, p_N; R_0, D, I) &= \left[ -(1-s_1)\mu_I \lambda'_G(0; R_0, D, I) (1 - p_N + p_N s_2)^d \right. \\ &\quad \left. - d(1-s_2)(1 - p_N + p_N s_2)^{d-1} \right] e^{-\lambda_G(p_N; R_0, D, I) \mu_I(1-s_1)}, \end{aligned} \quad (4.59a)$$

$$\begin{aligned} \frac{db_2}{dp_N}(\mathbf{s}, p_N; R_0, D, I) &= \left[ -(1-s_1)\mu_I \lambda'_G(0; R_0, D, I) (1 - p_N + p_N s_2)^{d-1} \right. \\ &\quad \left. - (d-1)(1-s_2)(1 - p_N + p_N s_2)^{d-2} \right] \\ &\quad \times e^{-\lambda_G(p_N; R_0, D, I) \mu_I(1-s_1)}, \end{aligned} \quad (4.59b)$$

$$\lambda'_G(p_N; R_0, D, I) = \frac{-dR_0^2}{(R_0 + p_N)^2}. \quad (4.59c)$$

Substituting equation (4.59c) into equations (4.59a) and (4.59b) yields

$$\begin{aligned} \frac{db_1}{dp_N}(\mathbf{s}, p_N; R_0, D, I) = & - \left[ (1 - s_2) - (1 - s_1)(1 - p_N + p_N s_2) \frac{R_0^2}{(R_0 + p_N)^2} \right] \\ & \times d e^{-\mu_I \lambda_G(p_N; R_0, D, I)(1-s_1)} (1 - p_N + p_N s_2)^{d-1}, \end{aligned} \quad (4.60a)$$

$$\begin{aligned} \frac{db_2}{dp_N}(\mathbf{s}, p_N; R_0, D, I) = & \\ & - \left[ (1 - s_2)(d - 1) - (1 - s_1)(1 - p_N + p_N s_2) \frac{dR_0^2}{(R_0 + p_N)^2} \right] \\ & \times e^{-\mu_I \lambda_G(p_N; R_0, D, I)(1-s_1)} (1 - p_N + p_N s_2)^{d-2}. \end{aligned} \quad (4.60b)$$

Evaluating equation (4.60a) at  $\boldsymbol{\pi}(p_N; R_0, D, I)$  yields the condition  $\frac{db_1}{dp_N}(\boldsymbol{\pi}(p_N; R_0, D, I), p_N; R_0, D, I) < 0$  if and only if

$$0 < 1 - \pi_2(p_N; R_0, D, I) - [1 - \pi_1(p_N; R_0, D, I)] [1 - p_N + p_N \pi_2(p_N; R_0, D, I)] \frac{R_0^2}{(R_0 + p_N)^2}. \quad (4.61)$$

Evaluating equation (4.57c) at  $\boldsymbol{\pi}(p_N; R_0, D, I)$  yields

$$\pi_1(p_N; R_0, D, I) = \pi_2(p_N; R_0, D, I) [1 + p_N \pi_2(p_N; R_0, D, I)], \quad (4.62)$$

and, substituting equation (4.62) into inequality (4.61),

$$\begin{aligned} 0 < 1 - \pi_2(p_N; R_0, D, I) & \\ & - [1 - \pi_1(p_N; R_0, D, I)] [1 - p_N + p_N \pi_2(p_N; R_0, D, I)] \frac{R_0^2}{(R_0 + p_N)^2} \\ \iff 0 < \left\{ 1 - [1 + p_N \pi_2(p_N; R_0, D, I)] [1 - p_N + p_N \pi_2(p_N; R_0, D, I)] \right. & \\ & \quad \left. \times \frac{R_0^2}{(R_0 + p_N)^2} \right\} [1 - \pi_2(p_N; R_0, D, I)] \\ \iff 0 < \left\{ -R_0^2 [1 + p_N \pi_2(p_N; R_0, D, I)] [1 - p_N + p_N \pi_2(p_N; R_0, D, I)] \right. & \\ & \quad \left. + (R_0 + p_N)^2 \right\} \frac{1 - \pi_2(p_N; R_0, D, I)}{(R_0 + p_N)^2} \\ \iff 0 < \left\{ p_N \left( 1 + \pi_2(p_N; R_0, D, I) [1 - \pi_2(p_N; R_0, D, I)] R_0^2 \right) \right. & \\ & \quad \left. + R_0 [2 + R_0 (1 - 2\pi_2(p_N; R_0, D, I))] \right\} p_N \frac{1 - \pi_2(p_N; R_0, D, I)}{(R_0 + p_N)^2}. \end{aligned} \quad (4.63)$$

Therefore if the assumption given in equation (4.55a) holds then equation (4.63) holds and  $\frac{db_1}{dp_N}(\boldsymbol{\pi}, p_N; R_0, D, I) < 0$ .

Finally, we need only show that  $\frac{db_2}{dp_N}(\boldsymbol{\pi}, p_N; R_0, D, I) < 0$  if equation (4.55b) holds. Evaluating inequality (4.60b) at  $\boldsymbol{\pi}(p_N; R_0, D, I)$  and substituting equation (4.62) yields

$$\begin{aligned}
0 &> \frac{db_2}{dp_N}(\boldsymbol{\pi}(p_N; R_0, D, I), p_N) \\
\iff 0 &< (d-1)[1 - \pi_2(p_N; R_0, D, I)] \\
&\quad - [1 - \pi_1(p_N; R_0, D, I)][1 - p_N + p_N\pi_2(p_N; R_0, D, I)] \frac{dR_0^2}{(R_0 + p_N)^2} \\
\iff 0 &< \left\{ -dR_0^2[1 + p_N\pi_2(p_N; R_0, D, I)][1 - p_N(1 - \pi_2(p_N; R_0, D, I))] \right. \\
&\quad \left. + (d-1)(R_0 + p_N)^2 \right\} \frac{1 - \pi_2(p_N; R_0, D, I)}{(R_0 + p_N)^2} \\
\iff 0 &< \left\{ p_N^2 \left[ (d-1) + d\pi_2(p_N; R_0, D, I)(1 - \pi_2(p_N; R_0, D, I)) R_0^2 \right] \right. \\
&\quad \left. + p_N R_0 [2(d-1) + dR_0(1 - 2\pi_2(p_N; R_0, D, I))] - R_0^2 \right\} \\
&\quad \times \frac{1 - \pi_2(p_N; R_0, D, I)}{(R_0 + p_N)^2},
\end{aligned}$$

which holds by the assumption given in equation (4.55b), as required for the proof of Theorem 4.3.  $\square$

The assumptions required to apply Theorem 4.3 are difficult to visualise. We now present a Corollary to Theorem 4.3 that gives sufficient conditions for the assumptions given in Theorem 4.3 to hold, and has a simpler interpretation for illustrative purposes.

**Corollary 4.1.** *Let  $D \sim \text{Const}(d)$ ,  $d \geq 2$ . Then, for  $R_0 > 1$  and  $\epsilon \in (0, 1 - p_N)$ , if*

$$1 > z(p_N; R_0, D, I) > \frac{1}{2} \left( 1 + \frac{p_N}{2} \right), \quad (4.64a)$$

and

$$p_N \geq \frac{R_0}{2(d-1)}, \quad (4.64b)$$

then

$$z(p_N; R_0, \text{Const}(d), I) < z(p_N + \epsilon; R_0, \text{Const}(d), I).$$

*Proof.* This proof proceeds by showing that equations (4.64) are sufficient conditions to apply Theorem 4.3. We begin by showing that inequality (4.64a)

implies inequality (4.55a). Note that inequality (4.55a) immediately holds if  $\pi_2(p_N; R_0, D, I) < 1/2$ , and recall that  $z(p_N; R_0, D, I) = 1 - \pi_1(p_N; R_0, D, I)$ . Then, substituting equation (4.62),

$$\begin{aligned}
z(p_N; R_0, D, I) &> \frac{1}{2} \left(1 + \frac{p_N}{2}\right) \\
\implies \pi_1(p_N; R_0, D, I) &< \frac{1}{2} \left(1 - \frac{p_N}{2}\right) \\
\implies \pi_2(p_N; R_0, D, I) [1 - p_N + p_N \pi_2(p_N; R_0, D, I)] &< \frac{1}{2} \left(1 - \frac{p_N}{2}\right) \\
\implies \pi_2(p_N; R_0, D, I) &\in \left(-\frac{2 - p_N}{2p_N}, \frac{1}{2}\right).
\end{aligned}$$

Since  $\pi_2(p_N; R_0, D, I) \in [0, 1)$ , if  $z(p_N; R_0, D, I) > 1/2 + p_N/4$  then the inequality  $\pi_2(p_N; R_0, D, I) < 1/2$  holds and inequality (4.64a) implies inequality (4.55a). Therefore to complete this proof we need only show that inequality (4.64b) implies inequality (4.55b). Note that, since  $\pi_2(p_N; R_0, D, I) < 1/2$ , inequality (4.55b) clearly holds if  $p_N R_0 2(d - 1) - R_0^2 \geq 0$ . Furthermore the inequality  $p_N R_0 2(d - 1) - R_0^2 \geq 0$  is a rearranged form of inequality (4.55b). Thus inequality (4.64b) implies inequality (4.55b) as required.  $\square$

Previously we conjectured that in the network and global model with a binomial degree distribution, fixing  $R_0$  and increasing  $p_N$  while decreasing  $\lambda_G$  causes a monotonic increase the final size of a major outbreak for all  $p_N \in (0, 1)$  for which  $z(p_N; R_0, D, I) < 1$ , as illustrated in Figure 4.1 on page 92. Theorem 4.3 is an analytic proof of this conjecture for the special case of a constant degree distribution and a subsection of  $p_N$  and  $z(p_N; R_0, D, I)$ . The difficulty in extending Theorem 4.3 to encompass all values of  $p_N$  and  $z(p_N; R_0, D, I)$  arises from the limitations of Lemma 4.4, i.e. we require both partial derivatives of the probability generating functions evaluated at the stationary points to have the same sign. In contrast, the difficulty in extending Theorem 4.3 to consider general degree distributions arises from the reliance of the proof on equation (4.57c), i.e.  $f_1(\mathbf{s}, p_N) = f_2(\mathbf{s}, p_N)(1 - p_N + p_N s_2)$ , which is a property of few distributions (although we note that Theorem 4.3 can therefore be extended to consider a binomial degree distribution). However, we note that Lemma 4.4 can be applied to general degree distributions, if its requirements are met.

### 4.1.5 Analysis of the network and global model with a logarithmic degree distribution

In this section we investigate the final size of a major outbreak on the network and global model with a logarithmic degree distribution. We consider a logarithmic degree distribution with parameter  $p$  because the final size of a major outbreak on the network and global model can be written explicitly as a simple function of  $R_0$  and  $p$ , which allows analytical analysis. Furthermore, we give a proof of the key result that fixing  $R_0$  and increasing the network heterogeneity does not always have a monotonic effect on the final size of a major outbreak. Thus the effect of introducing a small amount of network heterogeneity to the homogeneously mixing model is not necessarily the same as the effect of introducing more network heterogeneity to an already heterogeneous model.

Let  $z_{\text{Log}(p)}(R_0, \lambda_G; I)$  be the final size of a major outbreak in the network and global model with degree distribution  $D \sim \text{Log}(p)$  ( $p \in (0, 1)$ ), global infection rate  $\lambda_G \in [0, R_0/\mu_I]$ , basic reproduction number  $R_0$  and infectious period distribution  $I$ . We begin by noting some properties of the logarithmic degree distribution. Let  $D \sim \text{Log}(p)$ , then

$$\mu_D = \frac{-p}{(1-p)\log(1-p)}, \quad (4.65a)$$

$$\sigma_D^2 = \frac{-p(p + \log(1-p))}{(1-p)^2(\log(1-p))^2}, \quad (4.65b)$$

$$1 - \frac{\sigma_D^2}{\mu_D} = \frac{-p(1 + \log(1-p))}{(1-p)\log(1-p)}, \quad (4.65c)$$

$$\mu_{\tilde{D}-1} = \frac{p}{1-p}. \quad (4.65d)$$

In Proposition 4.4 we consider the following. In part (i) we consider the effect of introducing a small amount of network heterogeneity (with a logarithmic degree distribution) to the homogeneously mixing model, in part (ii) we consider the final size of a major outbreak on the standard network model with a logarithmic degree distribution and in part (iii) we prove that the final size of a major outbreak on the standard network model with a logarithmic degree distribution is always smaller than the final size of a major outbreak in the homogeneously mixing model. Recall that in the standard network model we require  $R_0 \in (1, \mu_{\tilde{D}-1}]$ , so if  $D \sim \text{Log}(p)$  then we require  $R_0 \in (1, p/(1-p)]$ , and that if  $\lambda_G = R_0/\mu_I$  then  $\lambda_N = 0$ .



**Proposition 4.4.** *Consider the network and global model with  $D \sim \text{Log}(p)$  and  $R_0 > 1$ .*

(i)

$$\text{sgn} \left( \frac{d^2 z_{\text{Log}(p)}(R_0, R_0/\mu_I; I)}{d\lambda_N^2} \right) \geq 0 \iff 1 - e^{-1} \geq p.$$

(ii) For  $R_0 \leq p/(1-p)$ ,

$$z_{\text{Log}(p)}(R_0, 0; I) = -\frac{\log(R_0)}{\log(1-p)}. \quad (4.66)$$

(iii) For  $R_0 \leq p/(1-p)$ ,

$$z_{\text{Log}(p)}(R_0, 0; I) < z_H(R_0).$$

*Proof.* Part (i) immediately follows by applying Theorem 4.1(iv) and substituting equation (4.65c).

To prove part (ii) we consider the standard network model with a logarithmic degree distribution. Recall from Table 2.2 that  $f_D(s) = \log(1-ps)/\log(1-p)$  and  $f_{\bar{D}-1}(s) = (1-p)/(1-ps)$ . Therefore, substituting  $f_D(s)$ ,  $f_{\bar{D}-1}(s)$  and  $p_N = R_0/\mu_{\bar{D}-1}$  (with  $\mu_{\bar{D}-1}$  given in equation (4.65d)) into equations (4.2),  $z_{\text{Log}(p)}(R_0, 0; I)$  satisfies the equation

$$1 - z_{\text{Log}(p)}(R_0, 0; I) = \frac{\log(1-p + z_2 R_0(1-p))}{\log(1-p)}, \quad (4.67a)$$

where  $z_2$  is the unique solution in  $(0, 1]$  of

$$1 - z_2 = \frac{1-p}{1-p + z_2 R_0(1-p)}. \quad (4.67b)$$

Rearranging equation (4.67b) yields

$$\begin{aligned} 1 - z_2 &= \frac{1-p}{1-p + z_2 R_0(1-p)} \\ \iff 1 - z_2 &= \frac{1}{1 + z_2 R_0} \\ \iff 0 &= z_2(z_2 R_0 - R_0 + 1). \end{aligned} \quad (4.68)$$

Thus, since  $z_2$  is the unique solution in  $(0, 1]$  of equation (4.68),  $z_2 R_0 = R_0 - 1$ . Rearranging equation (4.67a) and substituting  $z_2 R_0 = R_0 - 1$  yields part (ii),

i.e.

$$\begin{aligned} z_{\text{Log}(p)}(R_0, 0; I) &= 1 - \frac{\log((1-p)(1+z_2 R_0))}{\log(1-p)} = -\frac{\log(1+z_2 R_0)}{\log(1-p)} \\ &= -\frac{\log(R_0)}{\log(1-p)}. \end{aligned}$$

Finally we consider the proof of part (iii). Let

$$g(z_H(R_0)) = \left(1 - \frac{\log(1 - z_H(R_0))}{z_H(R_0)}\right)^{z_H(R_0)} + \frac{\log(1 - z_H(R_0))}{z_H(R_0)}. \quad (4.69)$$

We first show that if  $g(z_H(R_0)) \geq 0$  then  $z_{\text{Log}(p)}(R_0, 0; I) \leq z_H(R_0)$ . Finally we prove that  $g(z_H(R_0)) \geq 0$ , which completes the proof of part (iii).

Recall that in the standard network model with  $D \sim \text{Log}(p)$ ,  $R_0 \leq p/(1-p)$ , and

$$R_0 \leq p/(1-p) \iff \frac{1}{\log(1+R_0)} \geq -\frac{1}{\log(1-p)}. \quad (4.70)$$

Substituting inequality (4.70) into part (ii) yields

$$z_{\text{Log}(p)}(R_0, 0; I) = -\frac{\log(R_0)}{\log(1-p)} \leq \frac{\log(R_0)}{\log(1+R_0)}.$$

Therefore to prove  $z_{\text{Log}(p)}(R_0) \leq z_H(R_0)$  it is sufficient to show that

$$\frac{\log(R_0)}{\log(1+R_0)} \leq z_H(R_0). \quad (4.71)$$

Furthermore,

$$\begin{aligned} \frac{\log(R_0)}{\log(1+R_0)} &\leq z_H(R_0) \\ \iff \log(R_0) &\leq z_H(R_0) \log(1+R_0) \\ \iff R_0 &\leq (1+R_0)^{z_H(R_0)} \end{aligned} \quad (4.72)$$

Recall that  $z_H(R_0)$  is the unique solution in  $(0, 1]$  of  $1 - z = e^{-R_0 z}$ , so  $R_0$  and  $z_H(R_0)$  satisfy

$$R_0 = \frac{-\log(1 - z_H(R_0))}{z_H(R_0)}. \quad (4.73)$$

Substituting equation (4.73) into inequality (4.72), it is clear that to prove part (iii) it is sufficient to show that  $g(z_H(R_0)) \geq 0$ , where  $g(z_H(R_0))$  is defined in

equation (4.69). Substituting the Taylor series  $\log(1-x)/x = -\sum_{n=0}^{\infty} x^n/(n+1)!$  into equation (4.69) yields  $g(0) = 0$  and  $g(1) = 1$ . Furthermore,

$$\begin{aligned} g'(z_H(R_0)) &= \left[ \frac{-1}{z_H(R_0)(1-z_H(R_0))} + \frac{\log(1-z_H(R_0))}{z_H(R_0)^2} \right] \\ &\quad \times z_H(R_0) \left( 1 - \frac{\log(1-z_H(R_0))}{z_H(R_0)} \right)^{z_H(R_0)-1} \\ &\quad + \frac{1}{z_H(R_0)(1-z_H(R_0))} - \frac{\log(1-z_H(R_0))}{z_H(R_0)^2} \\ &= \frac{1}{z_H(R_0)(1-z_H(R_0))} \left[ 1 - z_H(R_0) \left( 1 - \frac{\log(1-z_H(R_0))}{z_H(R_0)} \right)^{z_H(R_0)-1} \right] \\ &\quad - \frac{\log(1-z_H(R_0))}{z_H(R_0)^2} \left[ 1 - z_H(R_0) \left( 1 - \frac{\log(1-z_H(R_0))}{z_H(R_0)} \right)^{z_H(R_0)-1} \right]. \end{aligned}$$

Thus for  $z_H(R_0) \in (0, 1)$ ,  $-\log(1-z_H(R_0)) > 0$  and so  $g'(z_H(R_0)) \geq 0$ . Therefore  $g(s)$  is an increasing function in  $(0, 1)$  and, since  $g(0) = 0$  and  $g(1) = 1$ ,  $g(z_H(R_0)) \geq 0$  as required for the proof of part (iii).  $\square$

Corollary 4.2 proves two results. Firstly, we show that, for the given parameters and fixed  $R_0$ , the final size of a major outbreak on the standard network model with a logarithmic degree distribution is smaller than the final size of a major outbreak on the homogeneously mixing model. We then prove (under the same parameters and fixed  $R_0$ ) that increasing  $\lambda_N$  at the expense of  $\lambda_G$  in the homogeneously mixing model will increase the final size of a major outbreak. Therefore Corollary 4.2 proves that fixing  $R_0$  and adding network heterogeneity to the homogeneously mixing model does not always have a monotonic effect on the final size of a major outbreak, and therefore the important result that the effect of introducing a small amount of network heterogeneity to the homogeneously mixing model is not necessarily the same as the effect of introducing more network heterogeneity to an already heterogeneous model.

**Corollary 4.2.** *For  $R_0 \in (1, e-1)$  and  $p \in [R_0/(R_0+1), 1-e^{-1}]$ ,*

$$z_{\text{Log}(p)}(R_0, 0; I) < z_H(R_0), \quad (4.74a)$$

and

$$\text{sgn} \left( \frac{d^2 z_{\text{Log}(p)}(R_0, R_0/\mu_I)}{d\lambda_N^2} \bigg|_{\lambda_N=0} \right) \leq 0. \quad (4.74b)$$

*Proof.* If  $p \leq 1-e^{-1}$  then equation (4.74b) holds by Proposition 4.4. Also by Proposition 4.4, equation (4.74a) holds if  $R_0 \leq p/(1-p)$ , which holds if and

only if  $p \geq R_0/(R_0 + 1)$ . Finally, the constraint  $R_0 \in (1, e - 1)$  is required for  $[R_0/R_0 + 1, 1 - e^{-1}]$  to be a positive interval.  $\square$

**Remark.** We can also consider the case  $D \sim \text{Geo}^+(p)$  to prove that fixing  $R_0$  and adding network heterogeneity to the homogeneously mixing model does not always have a monotonic effect on the final size of a major outbreak. The argument follows analogous arguments to those in the proof of Proposition 4.4 and Corollary 4.2.

## 4.2 Household and global model

In this section we investigate the effect of our choice of infection rates ( $\lambda_G$  and  $\lambda_H$ ) on the final size of a major outbreak for fixed  $R_0$  and household size distribution. Although our analytical results for the final size of a major outbreak and  $R_0$  allow for general household size distributions, requiring only  $\sigma_H^2 < \infty$ , for the numerical work in this section we focus on household size distributions with a finite maximal household size, since in practical applications households generally contain few individuals. We now introduce three household size distributions we consider. Let  $H_{2001}$  be a distribution with

$$(\rho_1, \rho_2, \rho_3, \rho_4, \rho_5, \rho_6) = (0.13, 0.3, 0.23, 0.18, 0.09, 0.03),$$

which is used as a household size distribution by Fraser (2007) and is based on UK census data from 2001. Let  $H_{1961}$  be a distribution with

$$(\rho_1, \rho_2, \rho_3, \rho_4, \rho_5, \rho_6) = (0.29, 0.34, 0.16, 0.14, 0.05, 0.02),$$

which is used as a household size distribution by Ball and Shaw (2015) and is based on UK census data from 1961. Finally, let  $H_M$  be a distribution with  $(\rho_1, \rho_2, \rho_3, \rho_4) = (0.74, 0.13, 0.1, 0.03)$ , which is chosen for its moment properties discussed in Remark 4.1.

Figure 4.2 plots the final size of a major outbreak in the household and global model against the household infection rate,  $\lambda_H$ , as the global infection rate,  $\lambda_G$ , is reduced to keep  $R_0 = 2$  fixed, for a range of household size distributions. So for a given value of  $\lambda_H$  each household size distribution has a different global infection rate,  $\lambda_G$ . To avoid numerical issues (see Section 7.6.1), in Figure 4.2 we condition the household size distribution  $H \sim \text{Poi}^+(3)$  on the maximal

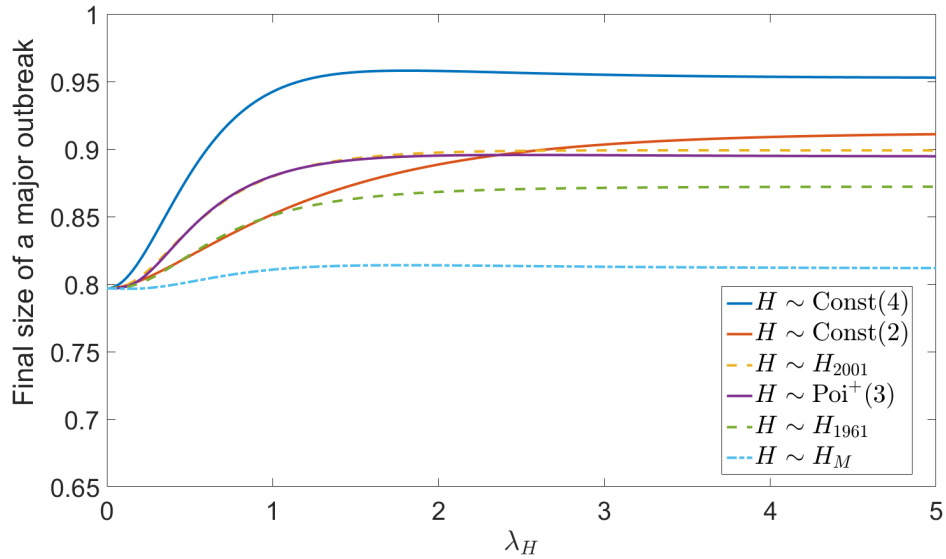


Figure 4.2: Effect of the household size distribution on the final size of a major outbreak in the household and global model for a range of household size distributions. Other parameters are  $R_0 = 2$  and  $I \sim \text{Const}(1)$ .

household size being at most 15.

We now discuss the numerical results from our investigations. Firstly, the effect of fixing  $R_0$  and introducing a small amount of household heterogeneity to the homogeneously mixing model is not necessarily the same as the effect of introducing more household heterogeneity to an already heterogeneous model, as illustrated in Figure 4.2 by the bottom line corresponding to the household size distribution  $H_M$ . Indeed, even ‘simple’ household size distributions such as  $H \sim \text{Const}(4)$  do not have a monotonic response in the final size of a major outbreak as  $\lambda_H$  is increased, as illustrated in the top line in Figure 4.2 which has a stationary point near  $\lambda_H = 1.56$ . We conjecture that, for each household size distribution, as  $\lambda_H \rightarrow \infty$  the final size of a major outbreak converges to an asymptotic limit. Finally, we note that even relatively simple household size distributions, such as those in Figure 4.2, do not admit a simple ordering in the final size of a major outbreak which holds for all  $\lambda_H$ .

In the following section we use analogous arguments to those in Section 4.1.1 to investigate the effect on the final size of a major outbreak of fixing  $R_0$  and introducing a small amount of household heterogeneity to the homogeneously mixing model. We then consider the final size in the household and global model with a saturated household infection rate, i.e.  $\lambda_H = \infty$ . However, we

now introduce the notation for the household and global model required in the later sections. By Ball et al. (1997), the probability generating function for the offspring distribution of the backward Galton-Watson branching process in the household and global model,  $b(s)$ , is given by

$$b(s) = e^{-\lambda_G \mu_I (1-s)} f_{M^{(n)}} \left( e^{-\lambda_G \mu_I (1-s)} \right).$$

Let  $z$  be the final size of a major outbreak in the household and global model. Then  $z = 1 - \pi_1$ , where  $\pi_1$  is the smallest solution to the equation  $b(s) = s$ .

Similarly to Section 4.1.1, we adjust our notation to reflect the dependencies in the model. So for the rest of this chapter we write  $z(\lambda_H; R_0, H, I)$ ,  $\lambda_G(\lambda_H; R_0, H, I)$ ,  $f_{M^{(n)}}(s, \lambda_H; I)$  and  $\mu_{i-1}^{(n)}(\lambda_H; I)$  for the quantities  $z$ ,  $\lambda_G$ ,  $f_{M^{(n)}}(s)$  and  $\mu_{i-1}^{(n)}$  respectively. Therefore,  $z(\lambda_H; R_0, H, I)$  is the largest solution in  $(0, 1]$  of the equation

$$\begin{aligned} 1 - z(\lambda_H; R_0, H, I) &= e^{-\lambda_G(\lambda_H; R_0, H, I) \mu_I z(\lambda_H; R_0, H, I)} \\ &\times \sum_{n=1}^{\infty} \tilde{\rho}_n f_{M^{(n)}} \left( e^{-\lambda_G(\lambda_H; R_0, H, I) \mu_I z(\lambda_H; R_0, H, I)}, \lambda_H; I \right). \end{aligned} \quad (4.75)$$

Furthermore, by Pellis et al. (2012),  $\lambda_G(\lambda_H; R_0, H, I)$  satisfies

$$\lambda_G(\lambda_H; R_0, H, I) \mu_I \sum_{i=1}^{\infty} \frac{1}{R_0^i} \sum_{n=i}^{\infty} \tilde{\rho}_n \mu_{i-1}^{(n)}(\lambda_H; I) = 1. \quad (4.76)$$

### 4.2.1 Adding a small amount of household structure to the homogeneously mixing model

We now consider the effect of fixing  $R_0$  and adding a small amount of household heterogeneity to the homogeneously mixing model, and give a similar result to Theorem 4.1. The results in Theorem 4.4 correspond to the gradients of the lines on the left-hand side of Figure 4.2, i.e.  $\lambda_H = 0$ .

**Theorem 4.4.** *Let  $z$  be the final size of a major outbreak on the household and global model with household size distribution  $H$ . Assume that  $z(0; R_0, H, I) < 1$ ,  $R_0 > 1$  and  $\rho_1 < 1$ . Then*

(i)

$$z(0; R_0, H, I) = z_H(R_0),$$

(ii)

$$\frac{dz}{d\lambda_H}(0; R_0, H, I) = 0,$$

(iii)

$$\begin{aligned} & \frac{d^2 z}{d\lambda_H^2}(0; R_0, H, I) (1 - R_0 (1 - z_H(R_0))) \\ &= \mu_I^2 z_H(R_0) (1 - z_H(R_0)) \left( \frac{2}{R_0} + 3z_H(R_0) - 2 \right) \\ & \quad \times (E[\tilde{H}]^2 + E[\tilde{H}] - E[\tilde{H}^2] - 1), \end{aligned}$$

(iv)

$$\text{sgn} \left( \frac{d^2 z}{d\lambda_H^2}(0; R_0, H, I) \right) = \text{sgn} \left( E[H^2] \left( 1 + \frac{E[H^2]}{E[H]} \right) - E[H^3] - E[H] \right).$$

The proof of Theorem 4.4 follows a similar argument to the proof of Theorem 4.1. However, the proof of Theorem 4.4 involves calculating the derivatives of the probability generating functions of the final size of household susceptibility set's and Gontcharoff polynomials with respect to  $\lambda_H$  and evaluated at  $\lambda_H = 0$ . Therefore the computations are far more involved while not substantially changing the flow of the proof so we postpone the proof of Theorem 4.4 to Section B.3.

Theorem 4.4 is the key analytic result in our investigation of the household and network model, and proves that the effect of fixing  $R_0$  and introducing a small amount of household heterogeneity to the homogeneously mixing model on the final size of a major outbreak is determined by a function of the first three moments of the household size distribution. In contrast, Theorem 4.1 shows that (for fixed  $R_0$ ) the effect of introducing a small amount of network heterogeneity to the homogeneously mixing model on the final size depends on the first two moments of the degree distribution. We conjecture that the difference in moments required to understand the effect of introducing a small amount of heterogeneity arises from the difference in moments required to calculate the household size distribution and the degree distribution of a globally contacted individual, which we now explore.

Firstly consider the network and global model. A globally contacted individual (i.e. type-1) in the backward branching process has degree distribution  $D$

and the second derivative of the final size of a major outbreak with respect to  $\lambda_N$ , evaluated at  $\lambda_N = 0$ , is a function of the first two moments of  $D$ . However, an individual contacted via the network (i.e. type-2) in the backward branching process has degree distribution  $\tilde{D}$ , where  $f_{\tilde{D}} = sf'_D(s)/\mu_D$ , and the second derivative of the final size of a major outbreak (starting with a single type-2 individual) with respect to  $\lambda_N$ , evaluated at  $\lambda_N = 0$  (which can be calculated by differentiating equation (4.22) in the proof of Theorem 4.1), is a function of the first three moments of  $D$ .

Next consider the household and global model. An individual in the backward branching process has household size distribution  $\tilde{H}$  and the second derivative of the final size of a major outbreak with respect to  $\lambda_H$ , evaluated at  $\lambda_H = 0$ , is a function of the first three moments of  $H$ . Clearly each derivative of the final size of a major outbreak with respect to  $\lambda_H$ , evaluated at  $\lambda_H = 0$ , requires knowledge of an additional moment.

In general, Theorem 4.4 shows that if the household size distribution has a small variance then introducing a small amount of household heterogeneity to the homogeneously mixing model is likely to increase the final size of a major outbreak. Similarly, by Theorem 4.1 if the degree distribution of the network and global model has a small variance then introducing a small amount of network heterogeneity to the homogeneously mixing model increases the final size of a major outbreak.

**Remark 4.1.** *An example of a household size distribution for which the final size will decrease with the addition of a small amount of household heterogeneity to the homogeneously mixing model is given by  $H_M$ , as illustrated in Figure 4.2. Examples of household size distributions for which the final size of a major outbreak will increase are given by  $H \sim \text{Const}(n)$ , for  $n = 2, 3, \dots$*

### 4.2.2 Considering a saturated household infection rate

We now simplify the analysis of the household and global model by assuming that the household infection rate is saturated, i.e.  $\lambda_H = \infty$ , similarly to Becker and Utev (1998). Therefore we consider an ordering of the lines on the right-hand side of Figure 4.2. For the rest of this section we assume that the household infection rate is saturated.



We now give a sufficient condition for an ordering of the final sizes of major outbreaks on household and global models with a saturated household infection rate. Let  $H_1$  and  $H_2$  be two distributions with support in the non-negative integers and size-biased distributions  $\tilde{H}_1$  and  $\tilde{H}_2$  respectively. Furthermore, let  $z(\lambda_H, H_1; R_0, I)$  and  $z(\lambda_H, H_2; R_0, I)$  be the final size of a major outbreak in the household and global model with basic reproduction number  $R_0$ , household infection rate  $\lambda_H$ , infectious period distribution  $I$  and household size distribution  $H_1$  and  $H_2$  respectively.

**Proposition 4.5.** *Assume that  $R_0 > 1$ .*

(i) *If, for all  $s \in (0, 1]$ ,*

$$f_{\tilde{H}_1} \left( \exp \left( - \frac{R_0^2 s}{R_0 + \mathbb{E} [\tilde{H}_1] - 1} \right) \right) < f_{\tilde{H}_2} \left( \exp \left( - \frac{R_0^2 s}{R_0 + \mathbb{E} [\tilde{H}_2] - 1} \right) \right) \quad (4.77)$$

*then  $z(\infty, H_2; R_0, I) < z(\infty, H_1; R_0, I)$ .*

(ii) *If, for all  $s \in (0, 1]$ ,*

$$f_{\tilde{H}_1} \left( \exp \left( - \frac{R_0^2 s}{R_0 + \mathbb{E} [\tilde{H}_1] - 1} \right) \right) < e^{-R_0 s} \quad (4.78)$$

*then  $z_H(R_0) < z(\infty, H_1; R_0, I)$ .*

*Proof.* Recall the mean number of cases in each generation of a household epidemic,  $\mu_{i-1}^{(n)}(\lambda_H, I)$ ,  $n = 1, 2, \dots$ ,  $i = 1, 2, \dots, n$ , discussed in Section 3.2.3. Clearly, in the limit  $\lambda_H \rightarrow \infty$ , a single infected individual will infect all household neighbours with probability 1 during its infectious period. Therefore, for  $n = 1, 2, \dots$ ,  $\mu_0^{(n)}(\infty, I) = 1$ ,  $\mu_1^{(n)}(\infty, I) = n - 1$  and  $\mu_{i-1}^{(n)}(\infty, I) = 0$  for  $i = 3, 4, \dots, n$ . Therefore, for a household size distribution  $H$ , considering equation (4.76) in the limit  $\lambda_H \rightarrow \infty$  yields

$$\begin{aligned} 1 &= \lambda_G(\infty; R_0, H, I) \mu_I \left( R_0^{-1} \sum_{n=1}^{\infty} \tilde{\rho}_n + R_0^{-2} \sum_{n=2}^{\infty} \tilde{\rho}_n (n - 1) \right) \\ &= \lambda_G(\infty; R_0, H, I) \mu_I \frac{1}{R_0^2} \left( R_0 + \mathbb{E} [\tilde{H}] - 1 \right), \end{aligned} \quad (4.79)$$

so

$$\lambda_G(\infty; R_0, H, I) \mu_I = \frac{R_0^2}{R_0 + \mathbb{E} [\tilde{H}] - 1}. \quad (4.80)$$

Taking the limit  $\lambda_H \rightarrow \infty$  of equation (4.75) and substituting equation (4.80) implies that  $z(\infty, H_1; R_0, I)$  and  $z(\infty, H_2; R_0, I)$  satisfy

$$\begin{aligned} 1 - z(\infty, H_1; R_0, I) &= \sum_{n=1}^{\infty} \tilde{\rho}_n e^{-n\lambda_G(\infty; R_0, H, I)\mu_I z(\infty, H_1; R_0, I)} \\ &= f_{\tilde{H}_1} \left( \exp \left( -\frac{R_0^2 z(\infty, H_1; R_0, I)}{R_0 + E[\tilde{H}_1] - 1} \right) \right) \end{aligned} \quad (4.81a)$$

and

$$\begin{aligned} 1 - z(\infty, H_2; R_0, I) &= \sum_{n=1}^{\infty} \tilde{\rho}_n e^{-n\lambda_G(\infty; R_0, H, I)\mu_I z(\infty, H_2; R_0, I)} \\ &= f_{\tilde{H}_2} \left( \exp \left( -\frac{R_0^2 z(\infty, H_2; R_0, I)}{R_0 + E[\tilde{H}_2] - 1} \right) \right) \end{aligned} \quad (4.81b)$$

respectively. For  $z(\infty, H; R_0, I) \in (0, 1]$ , the right-hand side of both equations (4.81) are decreasing functions. Therefore if there exists  $s \in (0, 1]$  such that  $1 - s < f_{\tilde{H}_2} \left( \exp \left( -\frac{R_0^2 s}{R_0 + E[\tilde{H}_2] - 1} \right) \right)$ , then  $z(\infty, H_2; R_0, I) < s$ . Therefore, to prove part (i) we need only show that

$$\begin{aligned} 1 - z(\infty, H_1; R_0, I) &= f_{\tilde{H}_1} \left( \exp \left( -\frac{R_0^2 z(\infty, H_1; R_0, I)}{R_0 + E[\tilde{H}_1] - 1} \right) \right) \\ &< f_{\tilde{H}_2} \left( \exp \left( -\frac{R_0^2 z(\infty, H_1; R_0, I)}{R_0 + E[\tilde{H}_2] - 1} \right) \right), \end{aligned}$$

which holds by the assumption given in equation (4.77).

Now consider part (ii). Recall that  $z_H(R_0)$  satisfies  $1 - z_H(R_0) = e^{-R_0 z_H(R_0)}$  and, following an analogous argument to part (i), we need only show that

$$\begin{aligned} 1 - z(\infty, H_1; R_0, I) &= f_{\tilde{H}_1} \left( \exp \left( -\frac{R_0^2 z(\infty, H_1; R_0, I)}{R_0 + E[\tilde{H}_1] - 1} \right) \right) \\ &< e^{-R_0 z(\infty, H_1; R_0, I)} \end{aligned}$$

to prove part (ii), which holds by the assumption given in equation (4.78).  $\square$

A simple application of Proposition 4.5 immediately yields an ordering of  $z_H(R_0)$  and the final size of a major outbreak in the household and global model with a constant household size distribution.

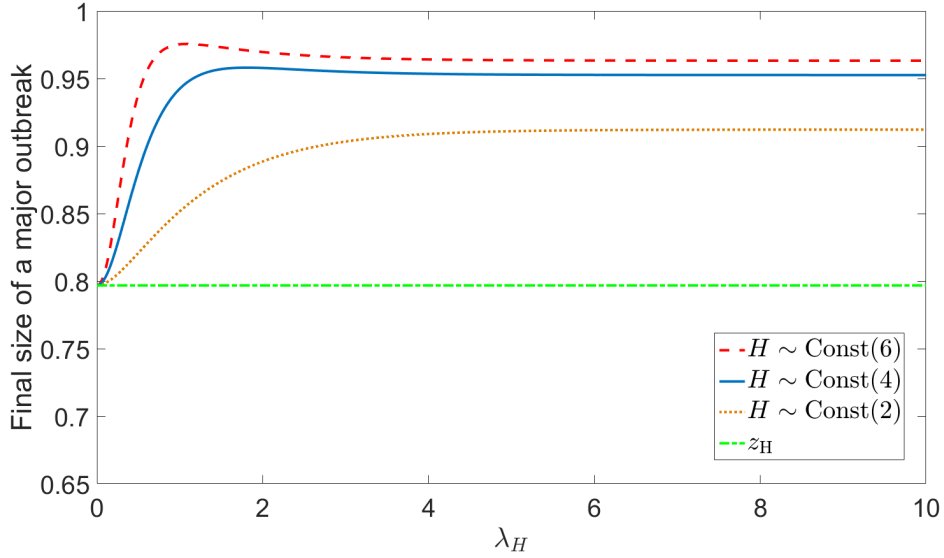


Figure 4.3: The effect of the household size distribution on the final size of a major outbreak in the household and global model for a range of constant household size distributions, along with the line  $z = z_H(R_0)$  for comparison. Other parameters are  $R_0 = 2$  and  $I \sim \text{Const}(1)$ .

**Corollary 4.3.** *If  $H_1 \sim \text{Const}(n_1)$  and  $H_2 \sim \text{Const}(n_2)$ ,  $1 < n_1 < n_2$  and  $z(\infty, H_2; R_0, I) < 1$ , then, for  $R_0 > 1$ ,*

$$z_H(R_0) < z(\infty, H_1; R_0, I) < z(\infty, H_2; R_0, I).$$

Corollary 4.3 is illustrated in the right-hand side of Figure 4.3, in which the final size of a major outbreak appears to converge to a limiting value as  $\lambda_H \rightarrow \infty$ , which is ordered with larger households having larger major outbreaks in the limit  $\lambda_H \rightarrow \infty$ .

### 4.3 Concluding remarks

In this chapter we consider the effect of network and household heterogeneity on the final size of a major outbreak while  $R_0$  is kept fixed, with the majority of our analytical focus on the extreme values of  $\lambda_G$ , i.e. considering the effect of fixing  $R_0$  and introducing a small amount of network or household heterogeneity to the homogeneously mixing model, or considering an ordering of the final size of a major outbreak on the standard network model with  $R_0$  kept fixed.

We show that fixing  $R_0$  and introducing a small amount of network heterogeneity (with degree distribution  $D$ ) to the homogeneously mixing model, by increasing the network infection rate while decreasing the global infection rate, will increase the final size of a major outbreak if  $1 - \sigma_D^2/\mu_D > 0$ , and decrease the final size of a major outbreak if  $1 - \sigma_D^2/\mu_D < 0$ . However, fixing  $R_0$  and introducing a small amount of household heterogeneity (with household size distribution  $H$ ) to the homogeneously mixing model, by increasing the household infection rate while decreasing the global infection rate, will increase the final size of a major outbreak if  $E[H^2] \left(1 + \frac{E[H^2]}{E[H]}\right) - E[H^3] - E[H] > 0$ , and decrease the final size of a major outbreak if  $E[H^2] \left(1 + \frac{E[H^2]}{E[H]}\right) - E[H^3] - E[H] < 0$ . Furthermore, we give an ordering for the final size of a major outbreak in the standard network model with a range of degree distributions, and in the household and global model with a saturated infection rate and some constant household size distributions.

Importantly, we prove that fixing  $R_0$  and introducing network or household heterogeneity to the homogeneously mixing model does not always have a monotonic effect on the final size of a major outbreak. Therefore the effect of fixing  $R_0$  and introducing a small amount of heterogeneity to the homogeneously mixing model is not necessarily the same as the effect of introducing more heterogeneity to an already heterogeneous model.

A major difficulty in evaluating the difference final size of a major outbreak in network and global models is that we need to compare the extinction probabilities of multiple two-type branching processes. This is a complicated problem, although we feel that further progress could be made by generalising Lemma 4.4 in Section 4.1.4 to allow for two partial derivatives to have opposite signs. Although analysis of the household and global model only requires knowledge of single-type branching processes, we find ourselves limited in our investigations by the complicated expressions determining the size of a household susceptibility set and the mean number of cases in each generation of a single household epidemic.

The results in Chapter 4 show that transmitting the disease through global contacts or a network structure have different effects on the final outcome of the epidemic.

## 4.4 Table of common notation introduced in Chapter 4

Symbol	Meaning	Page
$z_H(R_0)$	The final size of a major outbreak in the homogeneously mixing model with basic reproduction number $R_0$ .	93
$z_1(p_N(\lambda_N); R_0, D, I)$	The final size of a major outbreak in the network and global model with degree distribution $D$ , basic reproduction number $R_0$ , network infection rate $\lambda_N$ and infectious period distribution $I$ .	94
$z_{\text{Poi}(\alpha)}(R_0, \lambda_G; I)$	The final size of a major outbreak in the network and global model with degree distribution $D \sim \text{Poi}(\alpha)$ , basic reproduction number $R_0$ , global infection rate $\lambda_G$ and infectious period $I$ .	102
$z_{\text{Bin}(n,p)}(R_0, \lambda_G; I)$	The final size of a major outbreak in the network and global model with degree distribution $D \sim \text{Bin}(n, p)$ , basic reproduction number $R_0$ , global infection rate $\lambda_G$ and infectious period $I$ .	104
$z_{\text{NB}(r,p)}(R_0, \lambda_G; I)$	The final size of a major outbreak in the network and global model with degree distribution $D \sim \text{NB}(r, p)$ , basic reproduction number $R_0$ , global infection rate $\lambda_G$ and infectious period $I$ .	105
$z_{\text{Log}(p)}(R_0, \lambda_G; I)$	The final size of a major outbreak in the network and global model with degree distribution $D \sim \text{Log}(p)$ , basic reproduction number $R_0$ , global infection rate $\lambda_G$ and infectious period $I$ .	120
$z(\lambda_H, H; R_0, I)$	The final size of a major outbreak in the household and global model with basic reproduction number $R_0$ , household infection rate $\lambda_H$ , infectious period distribution $I$ and household size distribution $H$ .	129

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## 5. Effect of global contacts on the acquaintance vaccination strategy

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In this chapter we introduce three vaccination strategies on the network and global model: the acquaintance vaccination strategy, the uniform vaccination strategy and the optimal vaccination strategy. Under all three vaccination strategies we vaccinate individuals with a perfect vaccine after the network has been constructed but before the first infection occurs. We assume that individuals vaccinated with a perfect vaccine always become immune, with this immunity never waning, and immune individuals do not play any part in the epidemic, i.e. they cannot become infected, susceptible or removed.

First we consider the acquaintance vaccination strategy introduced by Ball and Sirl (2013) and Ball and Sirl (2017), in which neighbours of individuals sampled uniformly at random from the population are chosen for vaccination. Acquaintance vaccination is based on the acquaintance vaccination strategy introduced by Cohen et al. (2003) and further developed by Britton et al. (2007), which we refer to as the ‘single-neighbour’ acquaintance vaccination strategy. Under the single-neighbour vaccination strategy we sample individuals uniformly at random from the population and vaccinate a single neighbour chosen uniformly at random from the set of all possible neighbours. Both the acquaintance vaccination and single-neighbour acquaintance vaccination strategies tend to vaccinate individuals with larger degrees using only local knowledge of the network. This makes them a powerful tool in preventing the spread of epidemics since it is well-known that vaccination strategies that target individuals with large degrees often increase the effectiveness of vaccination (see, for example, Dezső and Barabási (2002)). However, we do not investigate the single-neighbour acquaintance vaccination strategy, since even the vaccination coverage does

not admit a simple closed form expression. Furthermore, the extension of the single-neighbour acquaintance vaccination strategy to the generalised vaccine reaction of Becker and Starczak (1998), assuming a non-perfect vaccine, requires a branching process with countably many types (see Ball and Sirl (2013)).

Next we contrast the acquaintance vaccination strategy with the uniform vaccination strategy, in which individuals are chosen uniformly at random for vaccination, and optimal vaccination, in which we use global knowledge of the network to order individuals by their degree and vaccinate individuals in descending order of degree to the required vaccination coverage. We give conditions under which, for a fixed vaccination coverage, the acquaintance vaccination strategy performs worse than the uniform vaccination strategy. For each vaccination strategy we numerically investigate the convergence of the final size of a major outbreak in finite populations to the asymptotic calculations. Finally, we discuss how the addition of global contacts affects the final size of a major outbreak and the critical vaccination coverage of the three vaccination strategies.

This chapter is laid out in the following way. In Sections 5.1 - 5.3 we analyse the acquaintance, uniform and optimal vaccination strategy respectively. In each case we determine a post-vaccination threshold parameter and expected relative final size of a major outbreak. In the context of acquaintance vaccination we also address the issue of choosing the parameters of the vaccination selection process. In Section 5.4 we compare the acquaintance and uniform vaccination strategies, giving conditions under which acquaintance vaccination strategy performs worse than the uniform vaccination strategy. In Section 5.5 we present numerical results, discussing the convergence of the final size of a major outbreak in finite populations to their asymptotic limits for the optimal, acquaintance and uniform vaccination strategies in Section 5.5.1, and the effect of global contacts on the critical vaccination coverage for the three vaccination strategies in Section 5.5.2. Finally we give our concluding remarks in Section 5.6 and a table of common notation introduced in this chapter in Section 5.7.

## 5.1 Acquaintance vaccination on the network and global model

### 5.1.1 Description of acquaintance vaccination

Before proceeding with the details of the acquaintance vaccination strategy we define the vaccination coverage and critical vaccination coverage. The vaccination coverage,  $c$ , of a vaccination strategy is the proportion of individuals in the population vaccinated, and the critical vaccination coverage,  $c^*$ , is the proportion of the population we need to vaccinate to reduce the basic reproduction number of the model under vaccination to 1. We now give a description of acquaintance vaccination and useful definitions and distributions required for further calculations, which are also given in Ball and Sirl (2013).

Recall that the network and global model is discussed in Section 4.1. Under the acquaintance vaccination strategy each individual in the population is sampled independently with probability  $p_S$  and each network neighbour of a sampled individual is independently chosen for vaccination with probability  $p_C$ . Finally, any individual which has been chosen for vaccination at least once is vaccinated with the perfect vaccine. Note that for this vaccination strategy to be applied we require an underlying network structure, i.e. to apply acquaintance vaccination we require  $p_0 < 1$ , which we assume for the rest of this Chapter. Note that if  $p_0 > 0$  then the maximum vaccination coverage that can be achieved will be less than 1.

Under the acquaintance vaccination strategy, for an individual  $i$  to be chosen for vaccination by a given network neighbour  $j$ ,  $j$  must be sampled, occurring with probability  $p_S$ , and choose  $i$  for vaccination, occurring with conditional probability  $p_C$ . Therefore the probability that an individual is not chosen for vaccination by a given network neighbour is  $1 - p_S p_C$ . Thus, since an individual  $i$  chosen uniformly at random from the population has  $D$  network neighbours, each of whom does not choose  $i$  for vaccination independently with probability  $1 - p_S p_C$ , the probability that an individual chosen uniformly at random from the population is vaccinated is

$$p_V = 1 - \sum_{k=0}^{\infty} p_k (1 - p_S p_C)^k = 1 - f_D(1 - p_S p_C). \quad (5.1)$$



Note that, by definition,  $p_V$  is also the vaccination coverage of the vaccination strategy.

By restricting our attention to a perfect vaccine we need only consider a two-type forward and backward branching process as in the network and global model. In the forward branching process, used to calculate the threshold parameter  $R_0$ , individuals are typed by whether they were infected by a global (type-1) or network (type-2) contact. In the backward branching process, used to calculate the final size of a major outbreak, individuals are typed by whether they join an individuals susceptibility set by a global (type-1) or network (type-2) contact. Before considering a threshold parameter and final size of a major outbreak for this model, we calculate the degree distributions of an unvaccinated individual chosen uniformly at random from the population, i.e. a type-1 individual, and an unvaccinated individual contacted via the network, i.e. a type-2 individual.

Consider an unvaccinated individual,  $i$  say, that has been globally contacted, and denote their degree distribution by  $D_U$ . Let  $U$  be the event that an individual is unvaccinated. Then a priori  $i$ 's degree is distributed according to  $D$  and  $i$  is vaccinated with probability  $p_V$ , given in equation (5.1). Thus  $D_U$  is given by

$$P(D_U = d) = \frac{P(D = d)P(U|D = d)}{P(U)} = \frac{p_d(1 - psp_C)^d}{1 - p_V}, \quad d = 0, 1, \dots \quad (5.2)$$

Next consider an unvaccinated individual contacted via the network,  $i$  say, and denote their degree distribution by  $\tilde{D}_U$ . Then  $i$  has unconditional degree distribution  $\tilde{D}$ , and we know that  $i$  avoids vaccination by all of its neighbours. Note that we do not count  $i$ 's parent in the branching process, which must not vaccinate  $i$  by definition (leading to the  $d - 1$  term in equation (5.3)). Therefore

$$P(\tilde{D}_U = d) = \frac{P(\tilde{D} = d, U)}{P(U)} = \frac{\tilde{p}_d(1 - psp_C)^{d-1}}{1 - \tilde{p}_V}, \quad d = 1, 2, \dots \quad (5.3)$$

where

$$\tilde{p}_V = \sum_{i=2}^{\infty} \tilde{p}_i(1 - (1 - psp_C)^{i-1}) = 1 - f_{\tilde{D}-1}(1 - psp_C) \quad (5.4)$$

is the a priori probability that  $i$  is vaccinated.

Finally, we denote by  $I_S$  the event that an individual is sampled, and note

that we know that an individual contacted via the network does not choose its parent in the branching process for vaccination. Therefore the probability that an individual contacted via the network is sampled, given that they did not choose their parent in the branching process for vaccination, is given by

$$\tilde{p}_{SU} = P(I_S | \text{does not choose parent}) = \frac{p_S(1 - p_C)}{p_S(1 - p_C) + 1 - p_S} = \frac{p_S(1 - p_C)}{1 - p_S p_C}. \quad (5.5)$$

### 5.1.2 Threshold parameter

We now calculate the threshold parameter,  $R_0^A$ , which determines whether or not the forward Galton-Watson branching process can have infinite progeny and therefore, by definition, whether or not a major outbreak can occur (see Section 3.2.1). Since the network and global model does not contain any household structure,  $R_0^A$  is the largest eigenvalue of the mean next-generation matrix  $M^A$  given in Theorem 5.1, so

$$R_0^A = \frac{m_{11}^A + m_{22}^A + \sqrt{(m_{11}^A - m_{22}^A)^2 + 4m_{12}^A m_{21}^A}}{2}. \quad (5.6)$$

**Theorem 5.1.** *The next-generation matrix  $M^A$  is given by*

$$M^A = \begin{bmatrix} \lambda_G \mu_I (1 - p_V) & p_N (1 - \tilde{p}_V) (1 - p_S p_C) \mathbb{E}[D_U] \\ \lambda_G \mu_I (1 - p_V) & p_N (1 - \tilde{p}_V) (1 - \tilde{p}_{SU} p_C) \mathbb{E}[\tilde{D}_U - 1] \end{bmatrix}. \quad (5.7)$$

*Proof.* This proof uses similar arguments to the work in Ball and Neal (2008) and Ball and Sirl (2013), and we follow similar notation to that introduced in Section 3.2.2.

First consider the expected number of infectious global contacts made by type-1 and type-2 individuals. Note that the number of global contacts made by an individual is independent of its network degree, so the distribution of the number of global contacts made by an infected individual does not depend on its type, thus  $m_{11}^A = m_{21}^A$ .

Let  $C_{GG}$  be the number of infectious global contacts made by a single infected individual who was contacted globally. An infectious individual makes global contacts at rate  $\lambda_G$  throughout an infectious period  $I$  with mean  $\mu_I$ , and these global contacts are made with distinct individuals chosen uniformly

at random from the population. An individual chosen uniformly at random from the population is unvaccinated with probability  $1 - p_V$ . Therefore the total number of global contacts an infectious individual makes has a Poisson distribution with parameter  $\lambda_G \mu_I$  and, conditional on the number of global contacts an infectious individual makes,  $C_{GG}^P$ , the number of infectious global contacts follows a Binomial distribution with  $C_{GG}^P$  trials and success probability  $(1 - p_V)$ . Thus

$$m_{11}^A = \mathbb{E}[C_{GG}] = \lambda_G \mu_I (1 - p_V) = m_{21}^A. \quad (5.8)$$

Next we consider the number of infectious network contacts. Let  $C_{GN}$  and  $C_{NN}$  be the total number of infectious network contacts made by a typical type-1 and type-2 individual, respectively. The expectations of both  $C_{GN}$  and  $C_{NN}$  can be determined by conditioning on the individual's infectious period,  $I$ , and the number of uninfected neighbours it has in the network, which is  $D_U$  for a type-1 individual and  $\tilde{D}_U - 1$  otherwise. All infectious periods have the same distribution,  $I$ , and note that a type-1 or type-2 individual is sampled with probability  $p_S$  or  $\tilde{p}_{SU}$  respectively, and otherwise unsampled.

Consider a type-1 individual  $i$ . Conditional on  $i$ 's infectious period,  $i$  makes infectious contact with a given network neighbour,  $j$ , if all of the following hold:  $j$  is not chosen for vaccination by  $i$ , occurring with probability  $1 - p_S p_C$ ;  $j$  is not already vaccinated by another network neighbour, occurring with probability  $1 - \tilde{p}_V$ ; and  $j$  is contacted, occurring with probability  $(1 - e^{-\lambda_N I_i})$ . Thus

$$C_{GN}|I, D_U \sim \text{Bin}\left(D_U, (1 - e^{-\lambda_N I}) (1 - \tilde{p}_V) (1 - p_S + p_S(1 - p_C))\right)$$

and, recalling that an individual's infectious period is independent of its degree distribution,

$$m_{12}^A = p_N (1 - \tilde{p}_V) (1 - p_S p_C) \mathbb{E}[D_U]. \quad (5.9)$$

The same calculations hold for  $m_{22}^A$  if  $D_U$  and  $p_S$  are replaced by  $\tilde{D}_U - 1$  and  $\tilde{p}_{SU}$  respectively. Therefore

$$m_{22}^A = p_N (1 - \tilde{p}_V) (1 - \tilde{p}_{SU} p_C) \mathbb{E}[\tilde{D}_U - 1]. \quad (5.10)$$

Equations (5.8), (5.9) and (5.10) yield the entries of the next-generation matrix  $M^A$  as required.  $\square$

### 5.1.3 Final size of a major outbreak

We define the expected relative final size of a major outbreak in a population that contains vaccinated individuals to be the fraction of initially susceptible individuals that are ultimately infected by the epidemic in a major outbreak when there was a single initial infective chosen uniformly at random from the population. Therefore to recover the proportion of ultimately infected individuals among the entire population, including the vaccinated individuals, we must multiply the final size of a major outbreak by  $1 - c$ . In this chapter we refer to the expected relative final size of a major outbreak as the final size of a major outbreak.

To calculate the final size of a major outbreak in the network and global model under the acquaintance vaccination strategy we consider a backwards Galton-Watson branching process that approximates the spread of an individual's susceptibility set, similarly to Section 3.3. Let  $B_{GG}$  and  $B_{NG}$  be the number of individuals that join the susceptibility set of a typical type-1 and type-2 individual respectively through a direct global contact. Similarly, let  $B_{GN}$  and  $B_{NN}$  be the number of individuals that join the susceptibility set of a typical type-1 and type-2 individual respectively via a network contact.

Let the probability generating functions for the offspring distribution of a type-1 and type-2 individual in the backward branching process be given by  $b_1(s_1, s_2)$  and  $b_2(s_1, s_2)$ ,  $(s_1, s_2) \in [0, 1]^2$ , respectively, so  $b_1(s_1, s_2) = \mathbb{E} [s_1^{B_{GG}} s_2^{B_{GN}}]$  and  $b_2(s_1, s_2) = \mathbb{E} [s_1^{B_{NG}} s_2^{B_{NN}}]$ . Let  $\mathbf{b}(s_1, s_2) = (b_1(s_1, s_2), b_2(s_1, s_2))$ .

By similar arguments to those given in Section 3.3, the final size of a major outbreak is  $z = 1 - \pi_1$ , where  $\boldsymbol{\pi} = (\pi_1, \pi_2)$  is the smallest solution to the set of simultaneous equations  $\boldsymbol{\pi} = \mathbf{b}(\boldsymbol{\pi})$ .

**Theorem 5.2.** *The joint probability generating functions for the offspring distributions of the backward Galton-Watson branching process are given by*

$$\begin{aligned} b_1(s_1, s_2) &= e^{-\lambda_G \mu_I (1-p_V)(1-s_1)} f_{D_U} [1 - p_N(1 - \tilde{p}_V)(1 - p_{SPC})(1 - s_2)], \\ b_2(s_1, s_2) &= e^{-\lambda_G \mu_I (1-p_V)(1-s_1)} f_{\tilde{D}_{U-1}} [1 - p_N(1 - \tilde{p}_V)(1 - \tilde{p}_{SUPC})(1 - s_2)]. \end{aligned}$$

*Proof.* The proof of Theorem 5.2 proceeds similarly to the proof of Theorem 3.6. Since the calculations of  $b_1(s_1, s_2)$  and  $b_2(s_1, s_2)$  are similar, we write  $B_{AG}$  and  $B_{AN}$ , where  $A \in \{G, N\}$ .

Consider an individual  $i$  in the population. All infectious contacts made to  $i$ , both global and via the network, are made by different individuals. Therefore all infectious contacts made to  $i$  are made by individuals with independent and identically distributed infectious periods,  $I$ . Furthermore, we know that the number of global contacts made to individual  $i$ , the degree distribution of  $i$ , the probability that a given unvaccinated network neighbour contacts  $i$  and whether  $i$  is sampled are all mutually independent. So  $B_{AG}$  and  $B_{AN}$  are independent and, since the only difference between a type-1 individual and a type-2 individual is the network degree of the individual, we know that  $B_{GG} \stackrel{\mathcal{D}}{=} B_{NG}$ .

A given network neighbour,  $j$ , of  $i$  can only make infectious contact with  $i$  if  $j$ : is not chosen for vaccination by  $i$ , is not already vaccinated by another neighbour and contacts  $i$ . Thus if  $i$  is a type-1 individual then  $j$  contacts  $i$  with probability  $(1 - p_{spC})(1 - \tilde{p}_V)p_N$ , and if  $i$  is a type-2 individual then  $j$  contacts  $i$  with probability  $(1 - \tilde{p}_{supC})(1 - \tilde{p}_V)p_N$ . Conditioning on the number of network neighbours of  $i$ , which is distributed as  $D_U$  or  $\tilde{D}_U$  for a type-1 or type-2 individual respectively, yields

$$B_{GN}|D_U \sim \text{Bin}(D_U, p_N(1 - \tilde{p}_V)(1 - p_{spC})) \quad (5.11a)$$

and

$$B_{NN}|\tilde{D}_U \sim \text{Bin}(\tilde{D}_U - 1, p_N(1 - \tilde{p}_V)(1 - \tilde{p}_{supC})). \quad (5.11b)$$

So, applying the independence of  $B_{AG}$  and  $B_{AN}$  to  $b_1(s_1, s_2)$  and  $b_2(s_1, s_2)$  and using equations (5.11),

$$\begin{aligned} \mathbb{E}[s_1^{B_{GG}} s_2^{B_{GN}}] &= \mathbb{E}[s_1^{B_{GG}}] \mathbb{E}[s_2^{B_{GN}}], \\ &= \mathbb{E}[s_1^{B_{GG}}] \mathbb{E}[\mathbb{E}[s_2^{B_{GN}}|D_U]] \\ &= \mathbb{E}[s_1^{B_{GG}}] f_{D_U}[1 - p_N(1 - \tilde{p}_V)(1 - p_{spC})(1 - s_2)], \end{aligned} \quad (5.12a)$$

and

$$\mathbb{E}[s_1^{B_{NG}} s_2^{B_{NN}}] = \mathbb{E}[s_1^{B_{NG}}] f_{\tilde{D}_U-1}[1 - p_N(1 - \tilde{p}_V)(1 - \tilde{p}_{supC})(1 - s_2)]. \quad (5.12b)$$

Finally, since  $B_{GG} \stackrel{\mathcal{D}}{=} B_{NG}$  we need only calculate  $\mathbb{E}[s_1^{B_{GG}}]$ . Consider a population containing  $N$  individuals and, for a specified individual,  $i$  say, let  $N'_i$  be the number of individuals in the population excluding those belonging to  $i$ 's

local susceptibility set. Then, for any of the  $N'_i$  individuals,  $j$  say,

$$\begin{aligned} \text{P}(j \text{ is unvaccinated and } j \text{ globally contacts } i) &= \left(1 - \text{E} \left[ e^{-\lambda_G I/N} \right] \right) (1 - p_V) \\ &= (1 - \phi_I(\lambda_G/N)) (1 - p_V). \end{aligned}$$

Recall that an individual  $i$ 's local susceptibility set is the set of individuals that, were they to become infected, would contact  $i$  via a chain of contacts made via the network (see Section 3.3.1). Denote by  $W_i^{(N)}$  the number of individuals that contact  $i$  globally in a population of size  $N$ , excluding  $i$ 's local susceptibility set which contains  $N - N'_i$  individuals, and clearly  $W_i^{(N)} \sim \text{Bin}[N'_i, (1 - \phi_I(\lambda_G/N))(1 - p_V)]$ . Since  $\sigma_D^2 < \infty$ , in the limit  $N \rightarrow \infty$  the probability that a member of  $i$ 's local susceptibility set will globally contact  $i$  is 0, so  $W_i^{(N)} \xrightarrow{\mathcal{D}} B_{GG}$  as  $N \rightarrow \infty$ . Thus, following analogous arguments to those in the proof of Theorem 3.6 (i.e. considering the Poisson approximation of the Binomial distribution),  $B_{GG} \sim \text{Poi}(\lambda_G \mu_I (1 - p_V))$ .

Substituting  $\text{E} \left[ s_1^{B_{GG}} \right] = e^{-\lambda_G \mu_I (1 - p_V)(1 - s_1)}$  and  $\text{E} \left[ s_1^{B_{NG}} \right] = e^{-\lambda_G \mu_I (1 - p_V)(1 - s_1)}$  into equations (5.12) yields the joint probability generating functions given in Theorem 5.2.  $\square$

#### 5.1.4 Balance between $p_S$ and $p_C$ for a fixed vaccination coverage

The acquaintance vaccination strategy has two parameters, the probability of sampling an individual ( $p_S$ ) and the probability a given network neighbour is chosen for vaccination ( $p_C$ ), which control the allocation of the vaccine. Furthermore, the vaccination coverage depends only on the product  $p_S p_C$ . Therefore, in this section we use the same methodology as Ball and Sirl (2013) to investigate the trade-off between  $p_S$  and  $p_C$  for a fixed (expected) vaccination coverage.

We begin by rearranging the next-generation matrix  $M$  into a form amenable to investigation. Consider the quantities  $\text{E}[D_U]$  and  $\text{E}[\tilde{D}_U - 1]$ . Substituting equation (5.2) into the definition of  $\text{E}[D_U]$  and then applying the formula

$f_{\tilde{D}-1}(s) = f'_D(s)/\mu_D$  yields

$$\begin{aligned} \mathbb{E}[D_U] &= \sum_{d=0}^{\infty} dP(D_U = d) \\ &= \frac{1}{1-p_V} \sum_{d=0}^{\infty} dp_d(1-p_S p_C)^d \\ &= \frac{1-p_S p_C}{1-p_V} f'_D(1-p_S p_C). \end{aligned} \quad (5.13)$$

Similarly, substituting equation (5.3) into the definition of  $\mathbb{E}[\tilde{D}_U - 1]$  yields

$$\begin{aligned} \mathbb{E}[\tilde{D}_U - 1] &= \sum_{d=0}^{\infty} dP(\tilde{D}_U - 1 = d) \\ &= \frac{1}{1-\tilde{p}_V} \sum_{d=0}^{\infty} d\tilde{p}_{d+1}(1-p_S p_C)^d \\ &= \frac{1-p_S p_C}{1-\tilde{p}_V} f'_{\tilde{D}-1}(1-p_S p_C). \end{aligned} \quad (5.14)$$

Recall that the vaccination coverage satisfies  $c = 1 - f_D(1 - p_S p_C)$ , so, for a fixed degree distribution, the vaccination coverage only depends on the product  $p_S p_C$  and so we write  $p' = p_S p_C$ . Substituting equations (5.1), (5.4), (5.13) and (5.14) into the next-generation matrix  $M^A$  given in Theorem 5.1 yields

$$M^A = \begin{bmatrix} \lambda_G \mu_I f_D(1-p') & p_N \frac{((1-p')f'_D(1-p'))^2}{\mu_D f_D(1-p')} \\ \lambda_G \mu_I f_D(1-p') & p_N(1-2p' + p'p_C)f'_{\tilde{D}-1}(1-p') \end{bmatrix}.$$

Note that, for a fixed vaccination coverage,  $m_{11}^A$ ,  $m_{12}^A$  and  $m_{21}^A$  are constant as  $p_C$  is varied and  $m_{22}^A$  is strictly increasing in  $p_C$ . Recall from Proposition 3.1 on page 44 that increasing or decreasing a single element of a matrix will increase or decrease its maximal eigenvalue respectively. Therefore, since  $R_0^A$  is the maximal eigenvalue of the next-generation matrix  $M$ , the following proposition holds by applying Proposition 3.1.

**Proposition 5.1.** *If  $P(D > 1) > 0$  and  $\lambda_N > 0$ , then, for a fixed vaccination coverage,  $R_0^A$  is strictly increasing in  $p_C$ . Otherwise, for a fixed vaccination coverage,  $R_0^A$  does not change with  $p_C$ .*

Firstly, Proposition 5.1 shows that if  $\lambda_N = 0$  or  $P(D > 1) = 0$ , i.e. if type-2 individuals cannot have type-2 offspring, then the exact balance between  $p_S$  and  $p_C$  is not important. This is what we would expect for the case  $\lambda_N = 0$ , as if there are no network infections occurring then we are considering the homogeneously

mixing model with vaccination, and the vaccination allocation strategy is not important, only the vaccination coverage. Similarly, in the presence of network contacts we would expect  $R_0^A$  to be increasing in  $p_C$ , for a fixed vaccination coverage, since intuitively the effect of vaccination under this strategy would be greater if everyone in the population chose a few individuals to be vaccinated, rather than a few individuals choosing all their neighbours for vaccination.

We note that in the case  $P(D > 1) > 0$  and  $\lambda_N > 0$  our results coincide with the results on the standard network model discussed by Ball and Sirl (2013), i.e. that  $R_0^A$  is minimised when  $(p_S, p_C) = (1, p')$  and maximised when  $(p_S, p_C) = (p', 1)$ , and that the greatest difference occurs when  $p'$ , and therefore also the vaccination coverage, is large. However, the size of the difference in  $R_0^A$  between maximising  $p_S$  vs  $p_C$  will be diluted compared to the standard network model, owing to the inclusion of global contacts.

So far in this section we have only considered the effect of increasing  $p_C$  on  $R_0^A$ , for a fixed vaccination coverage. We now turn our attention to the effect of the balance between  $p_S$  and  $p_C$ , for a fixed vaccination coverage, on the final size of a major outbreak. A rearrangement of the probability generating functions for the offspring of the backwards branching process, given in Theorem 5.2, similar to the rearrangement of  $M_A$ , yields

$$b_1(s_1, s_2) = e^{-\lambda_G \mu_I f_D(1-p')(1-s_1)} \frac{f_D\left((1-p')\left[1 - p_N(1-p')(1-s_2)f_{\bar{D}-1}(1-p')\right]\right)}{f_D(1-p')},$$

$$b_2(s_1, s_2) = e^{-\lambda_G \mu_I f_D(1-p')(1-s_1)} \frac{f_{\bar{D}-1}\left(1 - p_N(1 - 2p' + p'p_C)(1-s_2)f_{\bar{D}-1}(1-p')\right)}{f_{\bar{D}-1}(1-p')}.$$

Thus, for a fixed vaccination coverage,  $b_1(s_1, s_2)$  is not affected by the balance between  $p_S$  and  $p_C$  and  $b_2(s_1, s_2)$  is strictly decreasing in  $p_C$ . To highlight the dependence of  $b_1(s_1, s_2)$ ,  $b_2(s_1, s_2)$  and  $\mathbf{b}(s_1, s_2)$  on  $p_C$  we write  $b_1(s_1, s_2, p_C)$ ,  $b_2(s_1, s_2, p_C)$  and  $\mathbf{b}(s_1, s_2, p_C)$  respectively. Let  $z^\pm = 1 - \pi_1^\pm$ , where  $\pi^\pm = (\pi_1^\pm, \pi_2^\pm)$  is the smallest solution to the set of simultaneous equations  $\pi^\pm = \mathbf{b}(\pi^\pm, p_C^\pm)$ , with  $0 \leq p_C^- < p_C^+ \leq 1$ .

**Theorem 5.3.** *If  $P(D > 1) > 0$ ,  $\lambda_N > 0$  and  $0 < p_C^- < p_C^+ \leq 1$  then  $z^- < z^+$ .*

*Proof.* To show that  $z^- < z^+$  it is sufficient to show that  $\pi^- > \pi^+$ , which we do by following a similar argument to the proof of Lemma 4.4. Therefore, recall



the following notation and result from Section 2.3. For  $i = 1, 2$ , let

$$b_i^{(0)}(\mathbf{s}, p_C) = b_i(\mathbf{0}, p_C),$$

and, for  $n = 1, 2, \dots$ ,

$$b_i^{(n)}(\mathbf{s}, p_C) = b_i(b_1^{(n-1)}(\mathbf{s}, p_C), b_2^{(n-1)}(\mathbf{s}, p_C), p_C) = b_i^{n-1}(b_1(\mathbf{s}, p_C), b_2(\mathbf{s}, p_C), p_C).$$

Furthermore, for  $i = 1, 2$  and any  $\mathbf{s} \in [0, 1]^2$ ,  $\mathbf{s} \neq (1, 1)$ ,

$$\lim_{n \rightarrow \infty} b_i^{(n)}(\mathbf{s}, p_C^\pm) = \pi_i^\pm.$$

We now give a sufficient condition for the result  $\boldsymbol{\pi}^+ < \boldsymbol{\pi}^-$  which we then prove by induction. For  $n = 2, 3, \dots$ ,  $i = 1, 2$ , if

$$b_i^{(n)}(\boldsymbol{\pi}^-, p_C^+) < \pi_i^- \quad (5.15)$$

then

$$\pi_i^+ = \lim_{n \rightarrow \infty} b_i^{(n)}(\boldsymbol{\pi}^-, p_C^+) < \lim_{n \rightarrow \infty} \pi_i^- = \pi_i^-.$$

Therefore to prove Theorem 5.3 it is sufficient to prove that equation (5.15) holds. We first show the base case,  $b_i^{(2)}(\boldsymbol{\pi}^-, p_C^+) < \pi_i^-$ ,  $i = 1, 2$ . Recall that, for  $\mathbf{s} \in [0, 1]^2$ ,  $\mathbf{s} \neq (1, 1)$ ,  $b_1(\mathbf{s}, p_C^-) = b_1(\mathbf{s}, p_C^+)$  and  $b_2(\mathbf{s}, p_C^-) > b_2(\mathbf{s}, p_C^+)$ . Thus, for  $i = 1, 2$ , applying equation (2.8) from Section 2.3 and noting that  $b_i(\mathbf{s}, p_C)$  is an increasing function of  $\mathbf{s}$ ,

$$\begin{aligned} b_i^{(2)}(\boldsymbol{\pi}^-, p_C^+) &= b_i(b_1(\boldsymbol{\pi}^-, p_C^+), b_2(\boldsymbol{\pi}^-, p_C^+), p_C^+) \\ &< b_i(b_1(\boldsymbol{\pi}^-, p_C^-), b_2(\boldsymbol{\pi}^-, p_C^-), p_C^+) \\ &= b_i(\pi_1^-, \pi_2^-, p_C^+) \\ &\leq b_i(\pi_1^-, \pi_2^-, p_C^-), \end{aligned}$$

and the base case follows by noting that  $b_i(\pi_1^-, \pi_2^-, p_C^-) = \pi_i^-$ .

For the induction hypothesis, assume that, for  $i = 1, 2$ ,  $n = 1, 2, \dots$ ,

$$b_i^{(n)}(\boldsymbol{\pi}^-, p_C^+) < \pi_i^-,$$

and consider  $b_i^{(n+1)}(\boldsymbol{\pi}^-, p_C^+)$ . For  $i = 1, 2$ , applying equation (2.8) from Section 2.3 and noting that  $b_i(\mathbf{s}, p_C)$  is an increasing function of  $\mathbf{s}$ , applying the induction

hypothesis yields

$$\begin{aligned}
b_i^{(n+1)}(\pi^-, p_C^+) &= b_i(b_1^{(n)}(\pi^-, p_C^+), b_2^{(n)}(\pi^-, p_C^+), p_C^+) \\
&< b_i(\pi_1^-, \pi_2^-, p_C^+) \\
&\leq b_i(\pi_1^-, \pi_2^-, p_C^-) \\
&= \pi_i^-,
\end{aligned}$$

as required.  $\square$

Theorem 5.3 proves that, for a fixed vaccination coverage, the final size of a major outbreak is maximised when  $p_C = 1$  and minimised when  $p_S = 1$ .

## 5.2 Uniform Vaccination on the network and global model

### 5.2.1 Description of uniform vaccination

Under the uniform vaccination strategy, each individual in the population is vaccinated with the perfect vaccine independently with probability  $p_V$ . Clearly this means that the vaccination coverage  $c = p_V$ . Similarly to the calculations for the acquaintance vaccination strategy discussed in Section 5.1, we consider two-type forward and backward branching processes as in the network and global model. In the forward branching process, used to calculate the threshold parameter  $R_0$ , individuals are typed by whether they were infected by a global (type-1) or network (type-2) contact. In the backward branching process, used to calculate the final size of a major outbreak, individuals are typed by whether they join an individual's susceptibility set by a global (type-1) or network (type-2) contact.

### 5.2.2 Threshold parameter

We now give the calculations for a threshold parameter  $R_0^U$ . Similarly to Section 5.1.2, the threshold parameter  $R_0^U$  is the largest eigenvalue of the mean next-generation matrix  $M^U$ , which is given in the Theorem below.

**Theorem 5.4.** *The next-generation matrix  $M^U$  is given by*

$$M^U = \begin{bmatrix} \lambda_G \mu_I (1 - p_V) & p_N (1 - p_V) \mu_D \\ \lambda_G \mu_I (1 - p_V) & p_N (1 - p_V) \mu_{\tilde{D}-1} \end{bmatrix}. \quad (5.16)$$

*Proof.* This proof proceeds similarly to the proof of Theorem 5.1. The difference between the proof of Theorems 5.1 and 5.4 is in the probability that a contact is made with an unvaccinated individual.

First consider the expected number of type-1 offspring of a typical type-1 or type-2 individual. The probability an individual chosen uniformly at random from the population, i.e. an individual contacted globally, is unvaccinated is  $1 - p_V$ , so, applying analogous arguments to those in the proof of Theorem 5.1,

$$m_{11}^U = m_{21}^U = \lambda_G \mu_I (1 - p_V). \quad (5.17)$$

We now consider the number of type-2 offspring of a single type-1 or type-2 individual, which we define by  $C_{GN}$  and  $C_{NN}$  respectively. The probability that each neighbour of a type-1 or type-2 individual is unvaccinated is  $1 - p_V$ , independently of the event that any other neighbour is unvaccinated. Therefore analogous arguments to those in the proof of Theorem 5.1 yield

$$m_{12}^U = p_N (1 - p_V) \mu_D, \quad (5.18a)$$

$$m_{22}^U = p_N (1 - p_V) \mu_{\tilde{D}-1}. \quad (5.18b)$$

Equations (5.17) and (5.18) yield the entries of the next-generation matrix  $M^U$  as required. □

We now comment on the critical vaccination coverage of two network and global models under the uniform vaccination strategy matched by  $R_0$ . Consider the network and global model under no vaccination, i.e.  $p_V = 0$  in Theorem 5.4. This model has basic reproduction number  $R_0$  and the approximating forward branching process has mean next-generation matrix  $M$ . Then consider a network and global model with the same parameters except that we apply the uniform vaccination strategy with each individual in the population vaccinated with probability  $p_V$ , which has basic reproduction number  $R_0^U$  and mean next-generation matrix  $M^U$ . Note that  $M^U = (1 - p_V)M$ , so  $R_0^U = (1 - p_V)R_0$  and thus the critical vaccination coverage under the uniform vaccination strategy

for the network and global model depends on  $R_0$ , and not the specific choice of  $D$ ,  $\lambda_G$  or  $\lambda_N$ , yielding the following remark.

**Remark 5.1.** *Two network and global models matched by  $R_0$  have the same critical vaccination coverage,  $c^* = 1 - 1/R_0$ , under the uniform vaccination strategy.*

Remark 5.1 is intuitive, since the critical vaccination coverage under both the standard network model and the homogeneously mixing model with basic reproduction number  $R_0$  is  $c^* = 1 - 1/R_0$  and the network and global model is an amalgamation of these two models.

### 5.2.3 Final size of a major outbreak

To calculate the final size of a major outbreak in this model, we consider a backwards Galton-Watson branching process that approximates the spread of an individual's susceptibility set, similarly to Section 3.3. Let  $B_{GG}$  and  $B_{NG}$  be the number of type-1 offspring of a type-1 and type-2 individual respectively in the backwards branching process, and similarly let  $B_{GN}$  and  $B_{NN}$  be the number of type-2 offspring of a type-1 and type-2 individual respectively.

Let the probability generating functions for the offspring distribution of a type-1 and type-2 individual in the backward branching process be given by  $b_1(s_1, s_2)$  and  $b_2(s_1, s_2)$ ,  $(s_1, s_2) \in [0, 1]^2$ , respectively, so  $b_1(s_1, s_2) = E[s_1^{B_{GG}} s_2^{B_{GN}}]$  and  $b_2(s_1, s_2) = E[s_1^{B_{NG}} s_2^{B_{NN}}]$ . Furthermore let  $\mathbf{b}(s_1, s_2) = (b_1(s_1, s_2), b_2(s_1, s_2))$ .

By similar arguments to those given in Section 3.3, the relative final size of a major outbreak is  $z = 1 - \pi_1$ , where  $\boldsymbol{\pi} = (\pi_1, \pi_2)$  is the smallest solution to the set of simultaneous equations  $\boldsymbol{\pi} = \mathbf{b}(\boldsymbol{\pi})$ .

**Theorem 5.5.** *The joint probability generating functions for the offspring distributions of the backward Galton-Watson branching process are given by*

$$\begin{aligned} b_1(s_1, s_2) &= e^{-\lambda_G \mu_I (1-p_V)(1-s_1)} f_D(1 - p_N(1 - p_V) + p_N(1 - p_V)s_2), \\ b_2(s_1, s_2) &= e^{-\lambda_G \mu_I (1-p_V)(1-s_1)} f_{\bar{D}-1}(1 - p_N(1 - p_V) + p_N(1 - p_V)s_2). \end{aligned}$$

*Proof.* The proof of Theorem 5.5 proceeds similarly to the proof of Theorem 5.2. Similarly to the proof of Theorem 5.2, we write  $B_{AG}$  and  $B_{AN}$ , where  $A \in \{G, N\}$ .

A given network neighbour,  $j$ , of  $i$  can only make infectious contact with  $i$  if  $j$  is not already vaccinated and  $j$  contacts  $i$ . Thus, regardless of whether  $i$  is a type-1 or type-2 individual,  $j$  contacts  $i$  with probability  $p_N(1 - p_V)$ . Therefore, conditional on the number of uninfected neighbours of  $i$ , which is  $D$  or  $\tilde{D} - 1$  for a type-1 or type-2 individual respectively,

$$B_{GN}|D \sim \text{Bin}(D, p_N(1 - p_V))$$

and

$$B_{NN}|\tilde{D} \sim \text{Bin}(\tilde{D} - 1, p_N(1 - p_V)).$$

Applying similar arguments to those in the proof of Theorem 5.2, we know that  $B_{AG}$  and  $B_{AN}$  are independent and  $B_{GG} \stackrel{\mathcal{D}}{=} B_{NG}$ . Therefore

$$\begin{aligned} \mathbb{E}[s_1^{B_{GG}} s_2^{B_{GN}}] &= \mathbb{E}[s_1^{B_{GG}}] \mathbb{E}[s_2^{B_{GN}}], \\ &= \mathbb{E}[s_1^{B_{GG}}] \mathbb{E}[\mathbb{E}[s_2^{B_{GN}}|D]] \\ &= \mathbb{E}[s_1^{B_{GG}}] f_D(1 - p_N(1 - p_V) + p_N(1 - p_V)s_2), \end{aligned} \quad (5.20a)$$

and

$$\mathbb{E}[s_1^{B_{NG}} s_2^{B_{NN}}] = \mathbb{E}[s_1^{B_{NG}}] f_{\tilde{D}-1}(1 - p_N(1 - p_V) + p_N(1 - p_V)s_2). \quad (5.20b)$$

Finally, since  $B_{GG} \stackrel{\mathcal{D}}{=} B_{NG}$  we need only calculate  $\mathbb{E}[s_1^{B_{GG}}]$ . Consider a population containing  $N$  individuals. Then, for specified individuals,  $i$  and  $j$  say,

$$\begin{aligned} \mathbb{P}(j \text{ is unvaccinated and } j \text{ globally contacts } i) &= (1 - \mathbb{E}[e^{-\lambda_G I/N}]) (1 - p_V) \\ &= (1 - \phi_I(\lambda_G/N)) (1 - p_V). \end{aligned}$$

Denote by  $W_i^{(N)}$  the number of people that contact individual  $i$  globally in a population of size  $N$ . Then  $W_i^{(N)} \sim \text{Bin}[N, (1 - \phi_I(\lambda_G/N))(1 - p_V)]$ . Clearly  $W_i^{(N)} \xrightarrow{\mathcal{D}} B_{GG}$  as  $N \rightarrow \infty$ . Thus, following analogous arguments to those in the proof of Theorem 3.6, i.e. considering the Poisson approximation of the Binomial distribution,  $B_{GG} \sim \text{Poi}(\lambda_G \mu_I(1 - p_V))$ .

Substituting  $\mathbb{E}[s_1^{B_{GG}}] = e^{-\lambda_G \mu_I(1 - p_V)(1 - s_1)}$  and  $\mathbb{E}[s_1^{B_{NG}}] = e^{-\lambda_G \mu_I(1 - p_V)(1 - s_1)}$  into equations (5.20) yields the joint probability generating functions given in Theorem 5.5.  $\square$

## 5.3 Optimal Vaccination on the network and global model

### 5.3.1 Description of optimal vaccination

We now extend the work in Ball and Sirl (2013), Appendix B, for the optimal vaccination strategy in the standard network model to the network and global model, although limiting our attention to a perfect vaccine. We conjecture that this vaccination strategy is optimal in the network and global model since the addition of global contacts, which are equally likely to infect any individual in the population, are unlikely to change the choice of vertices for optimal vaccination. Therefore, as in Ball and Sirl (2013), the optimal vaccination strategy when  $\lambda_N > 0$  is to vaccinate individuals with a degree larger than a cut-off value, the cut-off being determined by the desired vaccination coverage.

Given the desired vaccination coverage,  $c$ , let  $d_c$  be the smallest degree of an individual which we vaccinate, so  $d_c = \max \{n \in \mathbb{Z}^+ : \sum_{k=0}^{n-1} p_k < 1 - c\}$ . We vaccinate no individuals of degree  $d_c - 1$  or lower, all individuals of degree  $d_c + 1$  or higher, and some proportion  $\delta \in (0, 1]$  of individuals of degree  $d_c$ . Clearly we require  $c = \sum_{k=d_c+1}^{\infty} p_k + \delta p_{d_c}$ , so  $\delta = (c - \sum_{k=d_c+1}^{\infty} p_k) / p_{d_c}$ .

Similarly to the calculations for the acquaintance vaccination strategy discussed in Section 5.1, we consider two-type branching processes as in the network and global model. In the forward branching process, used to calculate the threshold parameter  $R_0$ , individuals are typed by whether they were infected by a global (type-1) or network (type-2) contact. In the backward branching process, used to calculate the final size of a major outbreak, individuals are typed by whether they join an individuals susceptibility set by a global (type-1) or network (type-2) contact.

Let  $D_U^O$  be the degree distribution of an unvaccinated individual chosen uniformly at random from the population. An individual chosen uniformly at random from the population has unconditional degree  $D$ , so, for  $k = 0, 1, \dots, d_c - 1$ ,  $P(D_U^O = k) = p_k(1 - c)^{-1}$  and  $P(D_U^O = d_c) = (1 - \delta)p_{d_c}(1 - c)^{-1}$ .

Similarly, let  $\tilde{D}_U^O$  be the degree distribution of an unvaccinated individual contacted via the network. An unvaccinated individual contacted via the net-

work will have unconditional degree distribution  $\tilde{D}$ , so, for  $k = 0, 1, \dots, d_c - 1$ ,  $P(\tilde{D}_U^O = k) = \tilde{p}_k(1 - \tilde{p}_V)^{-1}$  and  $P(\tilde{D}_U^O = d_c) = (1 - \delta)\tilde{p}_{d_c}(1 - \tilde{p}_V)^{-1}$ , where  $\tilde{p}_V = \delta\tilde{p}_{d_c} + \sum_{k=d_c+1}^{\infty} \tilde{p}_k$  is the probability that an individual contacted via the network is vaccinated.

### 5.3.2 Threshold parameter

We now give the calculations for a threshold parameter  $R_0^O$ . Similarly to Section 5.1.2, the threshold parameter  $R_0^O$  is the largest eigenvalue of the mean next-generation matrix  $M^O$ , which is given in the Theorem below.

**Theorem 5.6.** *The next-generation matrix  $M^O$  is given by*

$$M^O = \begin{bmatrix} \lambda_G \mu_I (1 - p_V) & p_N (1 - \tilde{p}_V) E[D_U^O] \\ \lambda_G \mu_I (1 - p_V) & p_N (1 - \tilde{p}_V) E[\tilde{D}_U^O - 1] \end{bmatrix}. \quad (5.21)$$

*Proof.* As with the proof of Theorem 5.4, this proof proceeds similarly to the proof of Theorem 5.1, differentiated by the degree distributions of type-1 and type-2 individuals and the probability that individuals are unvaccinated.

The probability an individual chosen uniformly at random from the population, i.e. an individual contacted globally, is unvaccinated is  $1 - p_V$ , so, applying analogous arguments to those in the proof of Theorem 5.1,

$$m_{11}^O = m_{21} = \lambda_G \mu_I (1 - p_V). \quad (5.22)$$

The degree distribution of a type-1 individual and type-2 individual is given by  $D_U^O$  and  $\tilde{D}_U^O$  respectively and, since the vaccination is given to individuals based on their degree and half-edges are paired uniformly at random, each neighbour of a type-1 or type-2 individual is independently unvaccinated with probability  $1 - \tilde{p}_V$  and otherwise vaccinated. Therefore, by similar arguments to the proof of Theorem 5.1,

$$m_{12}^O = p_N (1 - \tilde{p}_V) E[D_U^O], \quad (5.23a)$$

$$m_{22}^O = p_N (1 - \tilde{p}_V) E[\tilde{D}_U^O - 1]. \quad (5.23b)$$

Equations (5.22) and (5.23) yield the entries of the next-generation matrix  $M^O$  as required.  $\square$

### 5.3.3 Final size of a major outbreak

To calculate the final size of a major outbreak in this model we consider a backwards Galton-Watson branching process that approximates the spread of an individual's susceptibility set, similarly to Section 3.3. Let  $B_{GG}$  and  $B_{NG}$  be the number of type-1 offspring of a type-1 and type-2 individual respectively in the backwards branching process, and similarly let  $B_{GN}$  and  $B_{NN}$  be the number of type-2 offspring of a type-1 and type-2 individual respectively.

Let the probability generating functions for the offspring distribution of a type-1 and type-2 individual in the backward branching process be given by  $b_1(s_1, s_2)$  and  $b_2(s_1, s_2)$ ,  $(s_1, s_2) \in [0, 1]^2$ , respectively, so  $b_1(s_1, s_2) = \mathbb{E} [s_1^{B_{GG}} s_2^{B_{GN}}]$  and  $b_2(s_1, s_2) = \mathbb{E} [s_1^{B_{NG}} s_2^{B_{NN}}]$ . Furthermore let  $\mathbf{b}(s_1, s_2) = (b_1(s_1, s_2), b_2(s_1, s_2))$ .

By similar arguments to those given in Section 3.3, the relative final size of a major outbreak is  $z = 1 - \pi_1$ , where  $\boldsymbol{\pi} = (\pi_1, \pi_2)$  is the smallest solution to the set of simultaneous equations  $\boldsymbol{\pi} = \mathbf{b}(\boldsymbol{\pi})$ .

**Theorem 5.7.** *The joint probability generating functions for the offspring distributions of the backward Galton-Watson branching process are given by*

$$\begin{aligned} b_1(s_1, s_2) &= e^{-\lambda_G \mu_I (1-p_V)(1-s_1)} f_{D_U^O}(1 - p_N(1 - \tilde{p}_V) + p_N(1 - \tilde{p}_V)s_2), \\ b_2(s_1, s_2) &= e^{-\lambda_G \mu_I (1-p_V)(1-s_1)} f_{\tilde{D}_U^O-1}(1 - p_N(1 - \tilde{p}_V) + p_N(1 - \tilde{p}_V)s_2). \end{aligned}$$

*Proof.* The proof of Theorem 5.7 proceeds similarly to the proofs of Theorem 5.2 and 5.5, differentiated by the degree distributions of type-1 and type-2 individuals and the probability that individuals are unvaccinated. Similarly to the proof of Theorem 5.2, we write  $B_{AG}$  and  $B_{AN}$ , where  $A \in \{G, N\}$ .

Consider an individual  $i$ . A given network neighbour,  $j$  say, of  $i$  can only make infectious contact with  $i$  if  $j$  is not already vaccinated and  $j$  contacts  $i$ . Thus, regardless of whether  $i$  is a type-1 or type-2 individual,  $j$  contacts  $i$  with probability  $p_N(1 - \tilde{p}_V)$ . Therefore, conditioning on the number of uninfected neighbours of  $i$ , which is  $D_U^O$  or  $\tilde{D}_U^O - 1$  for a type-1 or type-2 individual respectively,

$$B_{GN} | D_U^O \sim \text{Bin}(D_U^O, p_N(1 - \tilde{p}_V)) \quad (5.24a)$$

and

$$B_{NN} | \tilde{D}_U^O \sim \text{Bin}(\tilde{D}_U^O - 1, p_N(1 - \tilde{p}_V)). \quad (5.24b)$$



By the same arguments as Theorem 5.5,  $B_{AG}$  and  $B_{AN}$  are independent. Thus, substituting equations (5.24),

$$\begin{aligned} \mathbb{E} [s_1^{B_{GG}} s_2^{B_{GN}}] &= \mathbb{E} [s_1^{B_{GG}}] \mathbb{E} [s_2^{B_{GN}}], \\ &= \mathbb{E} [s_1^{B_{GG}}] \mathbb{E} [\mathbb{E} [s_2^{B_{GN}} | D_U^O]] \\ &= \mathbb{E} [s_1^{B_{GG}}] f_{D_U^O} (1 - p_N(1 - \tilde{p}_V) + p_N(1 - \tilde{p}_V)s_2), \end{aligned} \quad (5.25a)$$

and

$$\mathbb{E} [s_1^{B_{NG}} s_2^{B_{NN}}] = \mathbb{E} [s_1^{B_{NG}}] f_{\tilde{D}_U^O - 1} (1 - p_N(1 - \tilde{p}_V) + p_N(1 - \tilde{p}_V)s_2). \quad (5.25b)$$

Finally, for analogous arguments to those given in the proof of Theorem 5.5, we know that  $B_{GG} \stackrel{\mathcal{D}}{=} B_{NG}$  and  $B_{GG_j} \sim \text{Poi}(\lambda_G \mu_I(1 - p_V))$ . Thus substituting  $\mathbb{E} [s_1^{B_{GG}}] = e^{-\lambda_G \mu_I(1 - p_V)(1 - s_1)}$  and  $\mathbb{E} [s_1^{B_{NG}}] = e^{-\lambda_G \mu_I(1 - p_V)(1 - s_1)}$  into equations (5.25) yields the joint probability generating functions given in Theorem 5.7.  $\square$

## 5.4 An analytical comparison of the acquaintance and uniform vaccination strategies

We might intuitively expect that for a fixed vaccination coverage and a constant degree distribution the performance of the uniform and acquaintance vaccination strategies would be similar. However, in this section we show that if the degree distribution has a small variance and  $p_S < 1$  then applying the uniform vaccination strategy can result in a smaller basic reproduction number than applying the acquaintance vaccination strategy with the same vaccination coverage, and consequently the uniform vaccination strategy has a smaller critical vaccination coverage than the acquaintance vaccination strategy. As a motivating example we begin by comparing the two vaccination strategies on the standard network model with a constant degree distribution.

Consider the standard network model with degree distribution  $D \sim \text{Const}(d)$ ,  $d = 2, 3, \dots$  (i.e. the network and global model with  $\lambda_G = 0$ ). Recalling that  $p' = p_{spc}$ , the vaccination coverage of the acquaintance vaccination strategy is equal to  $p_V = 1 - f_D(1 - p')$ . Therefore we compare the acquaintance vaccination strategy to the uniform vaccination strategy in which we vaccinate individuals uniformly at random with probability  $p_V$ . Then, substituting  $p_V$  and  $f_D(s) = s^d$

into the basic reproduction numbers given in Sections 5.1.4 and 5.2.2,

$$\begin{aligned} R_0^A &= p_N(1 - 2p' + p'p_C)(d - 1)(1 - p')^{d-2}, \\ R_0^U &= p_N(d - 1)(1 - p')^d. \end{aligned}$$

So  $R_0^U(1 - 2p' + p'p_C) = R_0^A(1 - p')^2$ . Since  $p' = p_S p_C$ ,  $(1 - 2p' + p'p_C) > (1 - p')^2$  and thus  $R_0^U \leq R_0^A$ , with equality if and only if  $p_S = 1$ . Furthermore, since our choice of  $p_V \in (0, 1)$  is arbitrary, the critical vaccination coverage of the uniform vaccination strategy is less than or equal to the critical vaccination coverage of the acquaintance vaccination strategy, with equality if and only if  $p_S = 1$ . We conjecture that the difference in critical vaccination coverages is caused by the acquaintance vaccination strategy with  $p_S < 1$  creating clusters of vaccinated individuals and thus, due to the similarity of an individual's degree, reducing the probability that a neighbour of an unvaccinated individual is also unvaccinated. In other words, conditioned on an individual being unvaccinated under the acquaintance vaccination strategy with  $p_S < 1$  this individual is unlikely to be sampled and thus less likely to have vaccinated neighbours compared to an individual in a population which has been vaccinated under the uniform vaccination strategy.

The underperformance of the acquaintance vaccination strategy with  $p_S < 1$  compared to the uniform vaccination strategy is not limited to the case of a constant degree distribution on the standard network. Figure 5.1 illustrates an example case for this underperformance on the standard network model with a degree distribution which has non-zero variance. Furthermore, Figure 5.1 also shows that partial vaccination can result in the final size of a major outbreak on the standard network model under the acquaintance vaccination strategy being larger than the final size of a major outbreak on the standard network model under the uniform vaccination strategy with the same vaccination coverage. We now give sufficient conditions under which the acquaintance vaccination strategy performs better and worse than the uniform vaccination strategy on the network and global model.

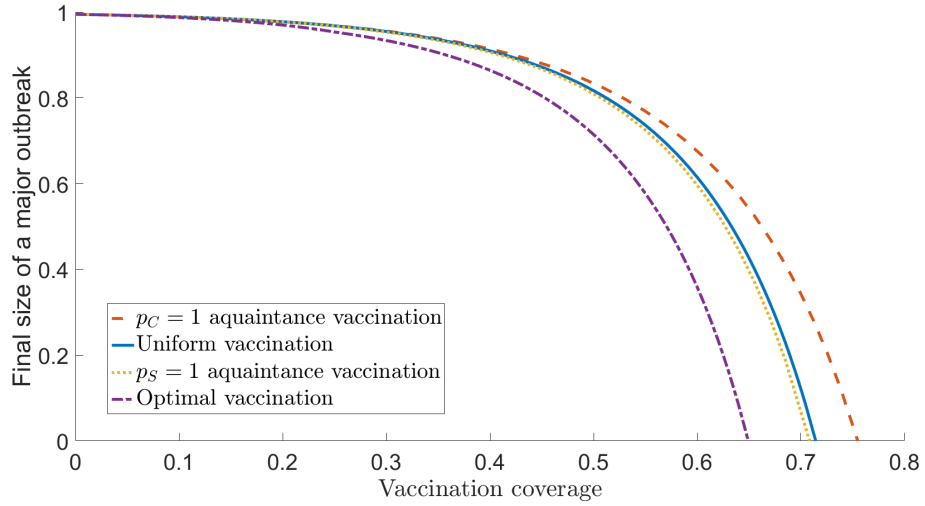


Figure 5.1: An example case in which the acquaintance vaccination strategy with  $p_C = 1$  has a larger critical vaccination coverage than the uniform vaccination strategy. The parameters of the model are  $\lambda_G = 0$ ,  $p_N = 0.5$ ,  $I \sim \text{Const}(1)$ ,  $P(D = 7) = 1/4$ ,  $P(D = 8) = 1/2$  and  $P(D = 9) = 1/4$ . Therefore  $R_0 = 3.5$ .

**Proposition 5.2.** *Consider the network and global model with degree distribution  $D$  under the acquaintance or uniform vaccination strategy with vaccination coverage  $p_V = 1 - f_D(1 - p')$ .*

(i) *If*

$$(1 - p')f'_D(1 - p') \leq \mu_D f_D(1 - p'),$$

*and*

$$(1 - 2p' + p'p_C)f''_D(1 - p') \leq f''_D(1)f_D(1 - p'),$$

*then  $R_0^A \leq R_0^U$ .*

(ii) *If*

$$(1 - p')f'_D(1 - p') \geq \mu_D f_D(1 - p'),$$

*and*

$$(1 - 2p' + p'p_C)f''_D(1 - p') \geq f''_D(1)f_D(1 - p'),$$

*then  $R_0^A \geq R_0^U$ .*

*Proof.* Consider the next-generation matrices  $M^A$  and  $M^U$  from Sections 5.1.4 and 5.2.2 respectively. Fixing the vaccination coverage  $p_V = 1 - f_D(1 - p')$  and

recalling that  $f_{\bar{D}-1}(s) = f'(s)/\mu_D$ , so  $f'_{\bar{D}-1}(1-p') = f''_D(1-p')/\mu_D$ , yields

$$M^A = \begin{bmatrix} \lambda_G \mu_I f_D(1-p') & p_N(1-p')^2 \frac{(f'_D(1-p'))^2}{\mu_D f_D(1-p')} \\ \lambda_G \mu_I f_D(1-p') & p_N(1-2p' + p'p_C) \frac{f''_D(1-p')}{\mu_D} \end{bmatrix}, \quad (5.26a)$$

and

$$M^U = \begin{bmatrix} \lambda_G \mu_I f_D(1-p') & p_N \mu_D f_D(1-p') \\ \lambda_G \mu_I f_D(1-p') & p_N \frac{f''_D(1)}{\mu_D} f_D(1-p') \end{bmatrix}. \quad (5.26b)$$

Clearly  $m_{11}^A = m_{21}^A = m_{11}^U = m_{21}^U$ . Furthermore, recall from Proposition 3.1 on page 44 that increasing or decreasing a single element of a matrix will increase or decrease its maximal eigenvalue respectively. Therefore if  $M^A \leq M^U$  then  $R_0^A \leq R_0^U$  and if  $M^A \geq M^U$  then  $R_0^A \geq R_0^U$ . So to prove Proposition 5.2 we need only compare  $M^A$  and  $M^U$  element-wise. Firstly note that if  $m_{12}^A = m_{12}^U$  and  $m_{22}^A = m_{22}^U$  then  $R_0^A = R_0^U$ , and then consider the following three cases:

- 1)  $m_{12}^A \leq m_{12}^U$  and  $m_{22}^A \leq m_{22}^U$ ,
- 2)  $m_{12}^A \geq m_{12}^U$  and  $m_{22}^A \geq m_{22}^U$ ,
- 3) Either  $m_{12}^A \leq m_{12}^U$  and  $m_{22}^A \geq m_{22}^U$  or  $m_{12}^A \geq m_{12}^U$  and  $m_{22}^A \leq m_{22}^U$ .

Firstly note that the requirements  $m_{12}^A \leq m_{12}^U$  and  $m_{22}^A \leq m_{22}^U$  (Case 1) correspond to the conditions for part (i) and so, by Proposition 3.1,  $R_0^A \leq R_0^U$  as required for part (i). Secondly note that the requirements  $m_{12}^A \geq m_{12}^U$  and  $m_{22}^A \geq m_{22}^U$  (Case 2) correspond to the conditions for part (ii) and so, by Proposition 3.1,  $R_0^A \geq R_0^U$ .

□

Note that Proposition 5.2 does not involve Case 3, i.e. if either  $m_{12}^A \leq m_{12}^U$  and  $m_{22}^A \geq m_{22}^U$  or  $m_{12}^A \geq m_{12}^U$  and  $m_{22}^A \leq m_{22}^U$ . Therefore Proposition 5.2 only gives sufficient conditions determining whether the ordering between  $R_0^A$  and  $R_0^U$ , and not necessary conditions. However, the consideration of Case 3 requires a direct investigation of  $R_0^A$  and  $R_0^U$ , which we do not consider in this thesis.

Although Proposition 5.2 gives an ordering between  $R_0^A$  and  $R_0^U$  for a given vaccination coverage, we can extend the argument to compare the critical vaccination coverage of the two vaccination strategies. Let  $c_A^*$  and  $c_U^*$  be the critical vaccination coverage under the acquaintance and uniform vaccination strategy respectively. If the degree distribution satisfies Proposition 5.2 part (i) or (ii) for all  $p' \in [0, 1]$  then the critical vaccination coverages of the acquaintance

and uniform vaccination strategies are ordered in the same way as the basic reproduction numbers, i.e. if  $R_0^A \leq R_0^U$  for all  $p' \in [0, 1]$  then  $c_A^* \leq c_U^*$  and if  $R_0^A \geq R_0^U$  for all  $p' \in [0, 1]$  then  $c_A^* \geq c_U^*$ .

Consider the network and global model with  $D \sim \text{Poi}(\alpha)$ ,  $\alpha > 0$ . Substituting  $f_D(s) = e^{-\alpha(1-s)}$  and its derivatives into the conditions for Proposition 5.2 part (i) yields that  $R_0^A \leq R_0^U$  if

$$(1 - p')\alpha e^{-\alpha p'} \leq \alpha e^{-\alpha p'}, \quad (5.27a)$$

$$(1 - 2p' + p'p_C)\alpha^2 e^{-\alpha p'} \leq \alpha^2 e^{-\alpha p'}. \quad (5.27b)$$

Inequalities (5.27) clearly hold for all  $p' \in (0, 1)$ , so in the network and global model with  $D \sim \text{Poi}(\alpha)$  for a fixed vaccination coverage  $R_0^A \leq R_0^U$  and  $c_A^* \leq c_U^*$ . Considering the network and global model with  $D \sim \text{Geo}(p)$ ,  $p \in (0, 1]$  and numerically investigating the conditions in Proposition 5.2 suggests that  $R_0^A \leq R_0^U$  and  $c_A^* < c_U^*$ .

Numerical investigations suggest that the situation  $c_A^* \geq c_U^*$  only occurs if  $\sigma_D^2$  is small. The acquaintance vaccination strategy was primarily introduced to target vaccination upon individuals with large degrees in the network using only local knowledge of the network. Therefore most applications of the acquaintance vaccination strategy involve models in which the degree distribution has sizeable variance, which suggests that we satisfy Proposition 5.2(ii). Thus for the remainder of this chapter we assume that the degree distribution satisfies the conditions of Proposition 5.2(ii), so  $c_A^* \leq c_U^*$  and the acquaintance vaccination strategy is no worse than the uniform vaccination strategy for a fixed vaccination coverage.

We note that our numerical and analytical investigations suggest that for a fixed vaccination coverage the acquaintance vaccination strategy can only underperform compared to the uniform vaccination strategy if  $p_S < 1$ . Indeed, we conjecture that the critical vaccination coverage of the acquaintance vaccination strategy with  $p_S = 1$  is always less than or equal to the critical vaccination coverage of the uniform vaccination strategy.

Recall that the acquaintance vaccination strategy is based on the single-neighbour acquaintance vaccination strategy introduced by Cohen et al. (2003). Applying an analogous argument to the proof of Proposition 5.2, i.e. com-

paring the reproduction numbers of the standard network model under the single-neighbour acquaintance and uniform vaccination strategies with a fixed vaccination coverage, (with the necessary equations given in, for example, Ball and Sirl (2013)) yields the following remark.

**Remark 5.2.** *In the standard network model with a constant degree distribution the single-neighbour acquaintance vaccination strategy has the same critical vaccination coverage as the uniform vaccination strategy.*

Furthermore, numerical investigations suggests that, similarly to the acquaintance vaccination strategy with  $p_S = 1$ , the critical vaccination coverage of the single-neighbour acquaintance vaccination strategy is always less than or equal to the critical vaccination coverage of the uniform vaccination strategy. As noted in Britton et al. (2007), an equivalent version of the single-neighbour acquaintance vaccination strategy involves sampling each individual in the population a random number of times which has a Poisson distribution. Therefore both the acquaintance vaccination strategy with  $p_S = 1$  and the single-neighbour acquaintance vaccination strategy involves sampling every individual in the population. We conjecture that the acquaintance vaccination strategy with  $p_S < 1$  not sampling every individual in the population causes the underperformance of the vaccination strategy compared to the uniform vaccination strategy.

## 5.5 Numerical investigations

### 5.5.1 Convergence of final size of a major outbreak in finite populations to asymptotic results

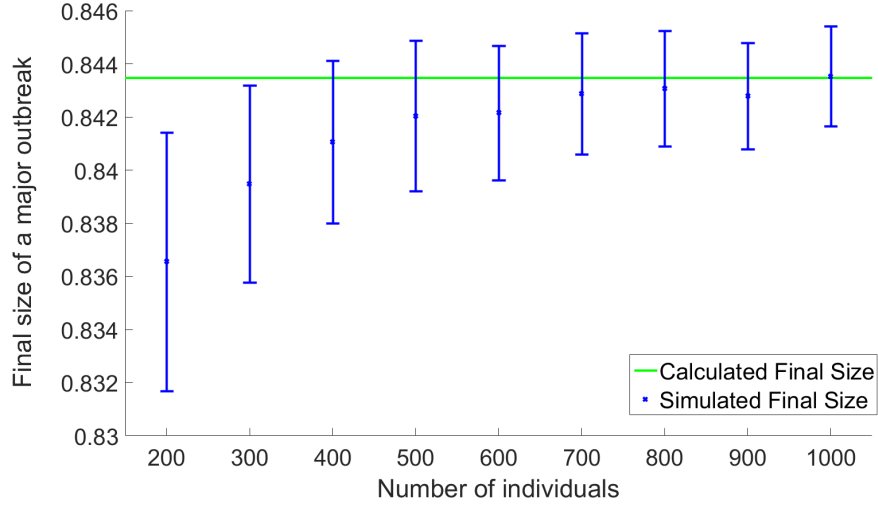
In this section we investigate whether the asymptotic results for the final size of a major outbreak and vaccination coverage under the acquaintance, uniform and optimal vaccination strategies give a good approximation for the final size of a major outbreak and vaccination coverage in finite populations, similarly to Section 3.4.1. To do this we run 1000 simulations of the epidemic in finite populations and then estimate the final size of a major outbreak empirically, comparing these results with the asymptotic calculations. The algorithm we follow for a single simulation is given in pseudo-code in Appendix A.2. Note that the vaccination coverage of the optimal vaccination strategy on a fixed population size is not random (and thus remains constant for each simulation), whereas the vaccination coverage of the acquaintance and uniform vaccination

strategies is random (and thus varies between each simulation).

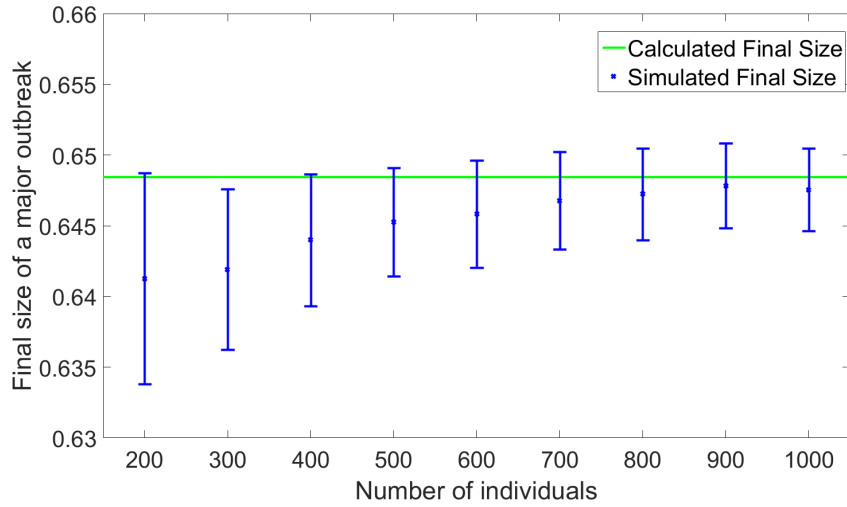
Under the uniform and optimal vaccination strategies the asymptotic final size of a major outbreak is a quite good approximation for a small number of individuals, as illustrated in Figures 5.2a and 5.2b respectively, although the accuracy of the approximation is linked to the expected number of susceptibles in the population after vaccination, rather than the number of individuals in the population. As for the model with three levels of mixing discussed in Section 3.4.1, we conjecture that if we investigated a degree distribution with heavy-tails we would find a slower convergence.

Similarly, under the acquaintance vaccination strategy the asymptotic final size of a major outbreak is a quite good approximation for a small number of individuals, with the accuracy of the approximation linked to the expected number of susceptibles in the population after vaccination, as illustrated in Figure 5.3, and we conjecture that if we investigated a degree distribution with heavy-tails we would find a slower convergence. However, an unexpected consequence of the acquaintance vaccination strategy is that the asymptotic calculation for the final size of a major outbreak no longer acts as an upper bound for the empirical final size of a major outbreak in finite populations. Instead, the asymptotic calculation for the final size of a major outbreak underestimates the final size of a major outbreak in finite populations, suggesting that the asymptotic critical vaccination coverage actually leads to undervaccination in finite populations. We conjecture that the underestimation of the final size of a major outbreak in finite populations is caused by undervaccination under the acquaintance vaccination strategy causing more frequent and larger major outbreaks. As evidence for this conjecture we now consider the difference between the effect of variable vaccination coverage caused by the uniform and acquaintance vaccination strategies on the final size of a major outbreak.

Firstly, we note that the empirical vaccination coverage of the uniform and acquaintance vaccination strategies in finite populations follows a symmetric distribution with mean close to  $c$ , independent of the population size, as illustrated in Figures 5.4. We suggest that the slight undervaccination of the acquaintance vaccination strategy is caused by network imperfections, such as loops and multiple edges. However, a difference arises between the vaccination coverage of the uniform and acquaintance vaccination strategies in finite popu-



(a) Comparing the final size of a major outbreak under the uniform vaccination strategy in finite populations to asymptotic results. The parameters are  $D \sim \text{Geo}(1/16)$ ,  $c = 0.7$ ,  $\lambda_N = 1.5$ ,  $\lambda_G = 1.5$  and  $I \sim \text{Const}(1)$ .



(b) Comparing the final size of a major outbreak under the optimal vaccination strategy in finite populations to asymptotic calculations. The parameters are  $D \sim \text{Geo}(1/9)$ ,  $c = 0.25$ ,  $\lambda_N = 1$ ,  $\lambda_G = 0.85$ .

Figure 5.2: Comparing empirical calculations for the final size of a major outbreak with the asymptotic calculations.



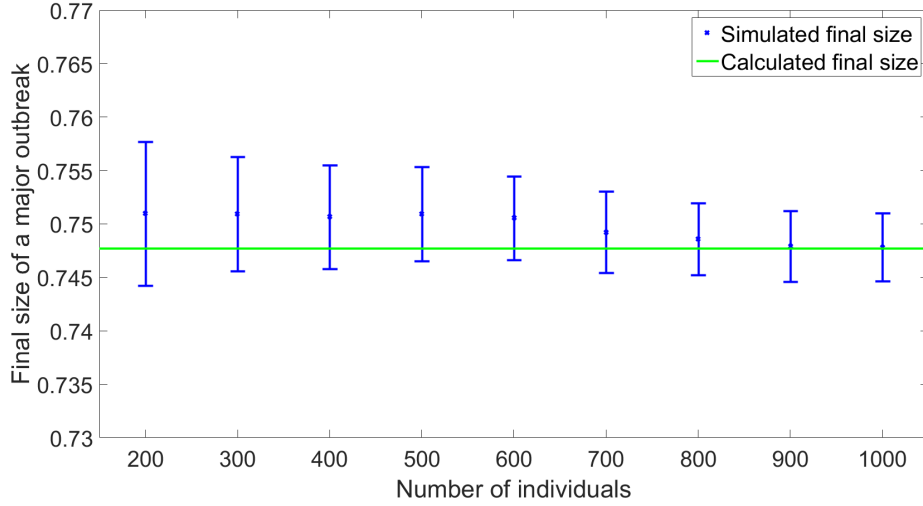
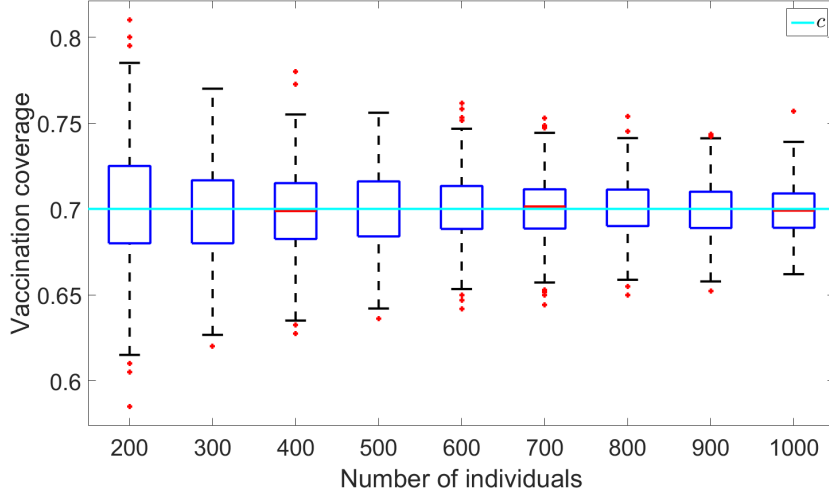


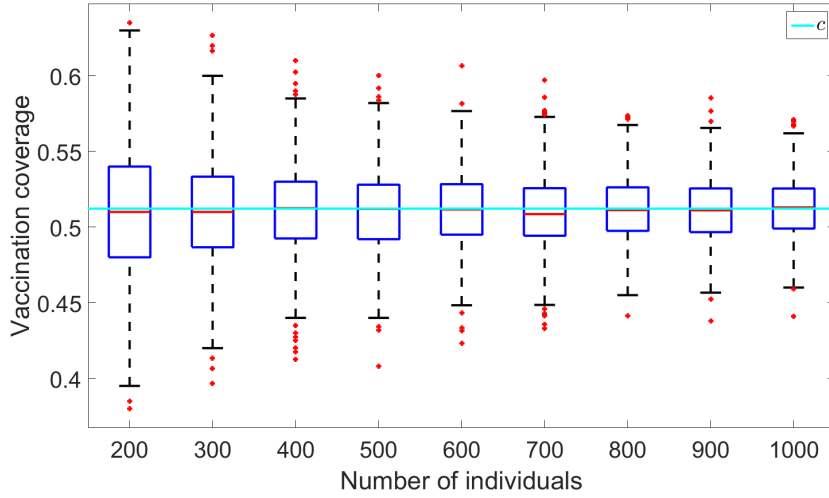
Figure 5.3: Comparing the final size of a major outbreak under the acquaintance vaccination strategy in finite populations to asymptotic results. The simulations are run with the parameters  $D \sim \text{Geo}(1/15)$ ,  $p_S = 0.5$ ,  $p_C = 0.15$ ,  $\lambda_N = 2$ ,  $\lambda_G = 1.8$  and  $I \sim \text{Const}(1)$ . Therefore  $c = 0.51$ .

lations when we only consider the simulations in which major outbreaks occur. When only considering the subset of simulations in which major outbreaks occur under the uniform vaccination strategy in finite populations the quartiles of the vaccination coverage have a negligible change compared to the original data (see Figures 5.4a and 5.5a). However, considering the subset of simulations in which major outbreak occurs under the acquaintance vaccination strategy in finite populations the quartiles of the vaccination coverage do substantially decrease (see Figures 5.4b and 5.5b). Therefore we now consider the relationship between the vaccination coverage and the probability and final size of a major outbreak in finite populations. To aid this analysis, let  $r_{c,maj}^U$  and  $r_{c,z}^U$  be the sample Pearson's correlation coefficient between the vaccination coverage under the uniform vaccination strategy and respectively the indicator function for whether a major outbreak occurs and final size of a major outbreak in a population with  $m$  households. Define  $r_{c,maj}^A$  and  $r_{c,z}^A$  similarly for the case when we apply the acquaintance vaccination strategy.

To graphically investigate the correlation between the vaccination coverage and the indicator function for whether a major outbreak occurs we apply the following method. For each population size, we order the simulations by vaccination coverage and then group the data into quartiles. So Quartile 1 consists of the  $1000/4 = 250$  simulations with the smallest empirical vaccination coverages,

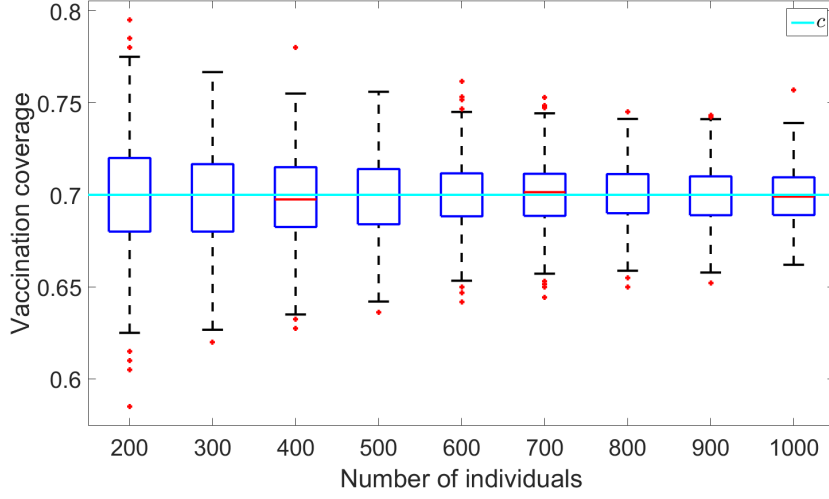


(a) The uniform vaccination strategy with  $D \sim \text{Geo}(1/16)$ ,  $c = 0.7$ ,  $\lambda_N = 1.5$ ,  $\lambda_G = 1.5$  and  $I \sim \text{Const}(1)$ .

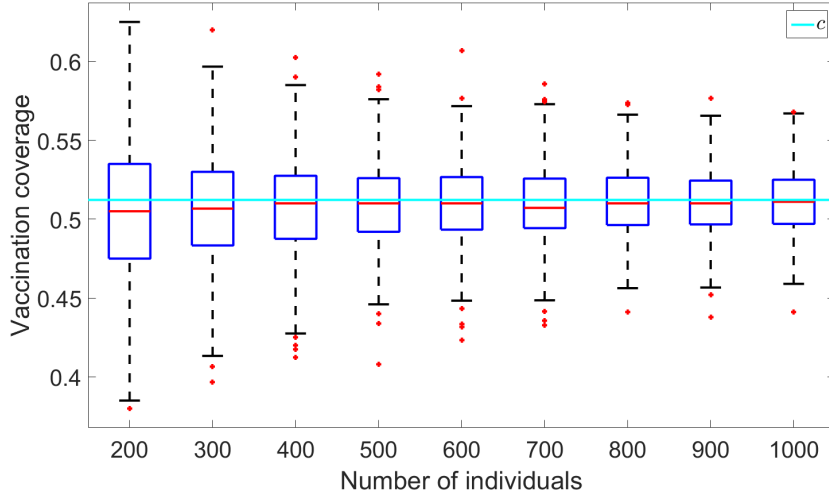


(b) The acquaintance vaccination strategy with parameters  $D \sim \text{Geo}(1/15)$ ,  $p_S = 0.5$ ,  $p_C = 0.15$ ,  $\lambda_N = 2$ ,  $\lambda_G = 1.8$  and  $I \sim \text{Const}(1)$ .

Figure 5.4: Comparing the empirical vaccination coverage under the uniform and acquaintance vaccination strategies in finite populations with the asymptotic vaccination coverage.



(a) The uniform vaccination strategy on the network and global model with  $D \sim \text{Geo}(1/16)$ ,  $c = 0.7$ ,  $\lambda_N = 1.5$ ,  $\lambda_G = 1.5$  and  $I \sim \text{Const}(1)$ .



(b) The acquaintance vaccination strategy on the network and global model with parameters  $D \sim \text{Geo}(1/15)$ ,  $p_S = 0.5$ ,  $p_C = 0.15$ ,  $\lambda_N = 2$ ,  $\lambda_G = 1.8$  and  $I \sim \text{Const}(1)$ .

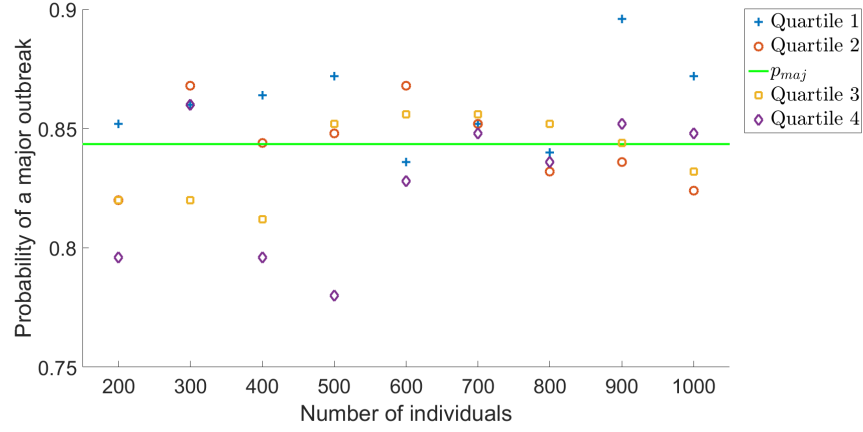
Figure 5.5: Comparing the empirical vaccination coverage under the uniform and acquaintance vaccination strategies in finite populations in which major outbreaks occur with the asymptotic vaccination coverage.

$m$	$r_{c,maj}^U$	$r_{c,maj}^A$	$r_{c,z}^U$	$r_{c,z}^A$
200	-0.11	-0.26	-0.32	-0.56
300	-0.08	-0.23	-0.39	-0.57
400	-0.10	-0.26	-0.36	-0.61
500	-0.13	-0.19	-0.31	-0.58
600	-0.05	-0.19	-0.35	-0.63
700	-0.05	-0.09	-0.37	-0.60
800	-0.02	-0.14	-0.35	-0.63
900	-0.06	-0.17	-0.31	-0.59
1000	-0.07	-0.16	-0.36	-0.61

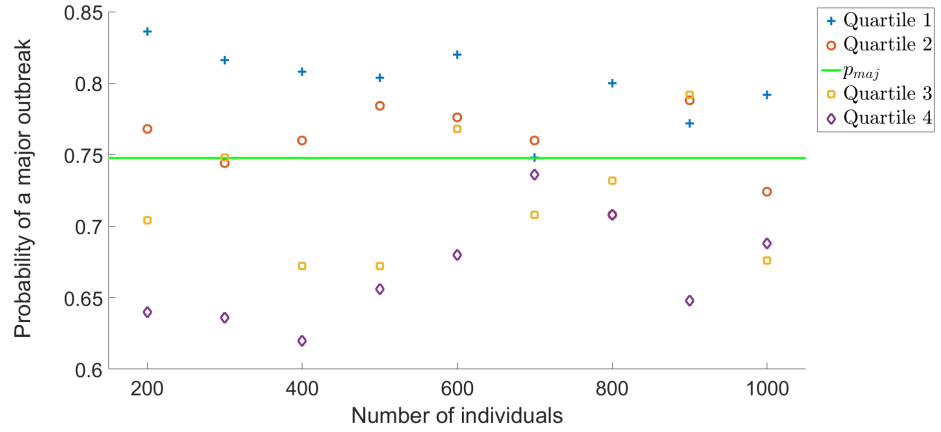
Table 5.1: Correlation coefficients between the vaccination coverage under the uniform and acquaintance vaccination strategies and the indicator function for whether a major outbreak occurs and the final size of a major outbreak for a range of population sizes.

Quartile 2 consists of the 250 simulations with the next lowest empirical vaccination coverages and so on. Then for each quartile we can calculate an empirical probability of a major outbreak (which can be compared to the asymptotic probability of a major outbreak by recalling that  $p_{maj} = z$  if the infectious period is constant). This breakdown shows that there is a very weak negative correlation between the vaccination coverage under the uniform vaccination strategy and the indicator function for whether a major outbreak occurs ( $r_{c,maj}^U \approx -0.07$ , see Table 5.1), i.e. undervaccination has a negligible effect on the probability that a major outbreak will occur, as illustrated in Figure 5.6a. In contrast, there is a weak negative correlation between the vaccination coverage under the acquaintance vaccination strategy and the indicator function for whether a major outbreak occurs ( $r_{c,maj}^A \approx -0.19$ , see Table 5.1), as illustrated in Figure 5.6b. Similarly, the final size of a major outbreak and the vaccination coverage under the uniform vaccination strategy in finite populations has a weak negative correlation ( $r_{c,z}^U \approx -0.35$ , see Table 5.1), whereas the final size of a major outbreak and the vaccination coverage under the acquaintance vaccination strategy in finite populations has a strong negative correlation ( $r_{c,z}^A \approx -0.6$ , see Table 5.1), as illustrated in Figures 5.7a and 5.7b respectively.

The correlation between the vaccination coverage under the acquaintance vaccination strategy and the indicator function for whether a major outbreak occurs and final size of a major outbreak suggest that undervaccination will lead to more major outbreaks, and furthermore that the major outbreaks which

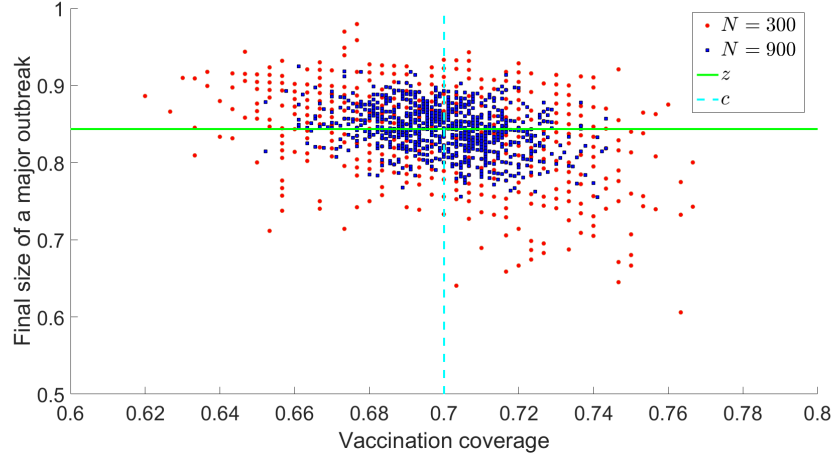


(a) The uniform vaccination strategy with  $D \sim \text{Geo}(1/16)$ ,  $c = 0.7$ ,  $\lambda_N = 1.5$ ,  $\lambda_G = 1.5$  and  $I \sim \text{Const}(1)$ .

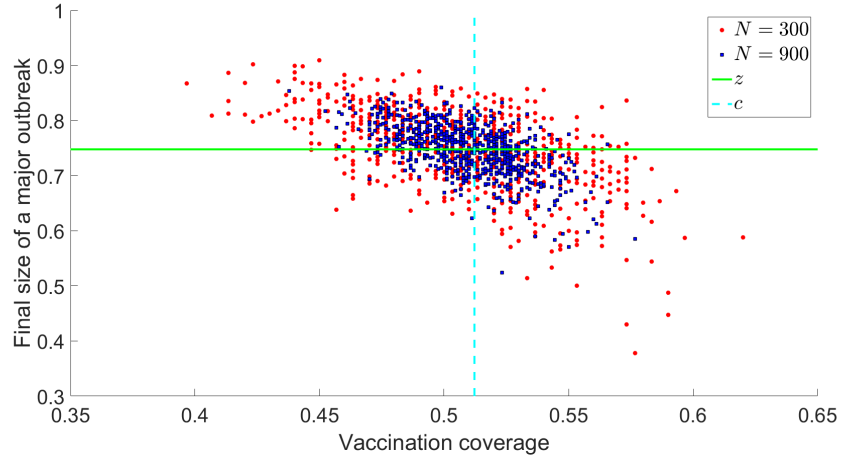


(b) The acquaintance vaccination strategy on the network and global model with parameters  $D \sim \text{Geo}(1/15)$ ,  $p_S = 0.5$ ,  $p_C = 0.15$ ,  $\lambda_N = 2$ ,  $\lambda_G = 1.8$  and  $I \sim \text{Const}(1)$ .

Figure 5.6: Investigating the dependence of the probability of a major outbreak on the vaccination coverage under the uniform and acquaintance vaccination strategies in finite populations. For each population size we order the simulations by vaccination coverage and then group the data into quartiles.



(a) The uniform vaccination strategy with  $D \sim \text{Geo}(1/16)$ ,  $c = 0.7$ ,  $\lambda_N = 1.5$ ,  $\lambda_G = 1.5$  and  $I \sim \text{Const}(1)$ .



(b) The acquaintance vaccination strategy on the network and global model with parameters  $D \sim \text{Geo}(1/15)$ ,  $p_S = 0.5$ ,  $p_C = 0.15$ ,  $\lambda_N = 2$ ,  $\lambda_G = 1.8$  and  $I \sim \text{Const}(1)$ .

Figure 5.7: Investigating the dependence of the final size of a major outbreak on the vaccination coverage under the uniform and acquaintance vaccination strategies in finite populations.

do occur are more likely to be large. Therefore the correlation between the vaccination coverage under the acquaintance vaccination strategy and the indicator function for whether a major outbreak occurs and final size of a major outbreak can explain why the asymptotic final size of a major outbreak in the network and global model under the acquaintance vaccination strategy is an underestimate for the final size of a major outbreak in finite populations. In contrast, since there is negligible correlation between the vaccination coverage under the uniform vaccination strategy and the indicator function for whether a major outbreak occurs, the correlation between the vaccination coverage under the uniform vaccination strategy and the final size of a major outbreak is cancelled out by the symmetric vaccination coverage distribution, leading to the usual result that the asymptotic final size of a major outbreak in the network and global model under the uniform vaccination strategy is an overestimate for the final size of a major outbreak in finite populations.

An important consideration for further work is understanding why there is weak negative correlation between the vaccination coverage under the acquaintance vaccination strategy and the indicator function for whether a major outbreak occurs, whereas there is negligible correlation between the vaccination coverage under the uniform vaccination strategy and the indicator function for whether a major outbreak occurs. Recall that in Section 5.4 we show that the acquaintance vaccination strategy with  $p_S < 1$  can underperform compared to the uniform vaccination strategy, and we conjecture that this is due to the acquaintance vaccination strategy clustering the vaccination in a closely connected group of individuals. It is possible that when undervaccination occurs under the acquaintance vaccination strategy the vaccine is also clustered into groups of individuals, leading to a strong underperformance of the vaccine. However, further work is required to understand this phenomenon. Indeed, it would be interesting to investigate whether maximising  $p_S$  or  $p_C$  (with a fixed vaccination coverage) will affect the correlation between the vaccination coverage under the acquaintance vaccination strategy and the indicator function for whether a major outbreak occurs and final size of a major outbreak in finite populations.

### 5.5.2 Effect of global contacts on the critical vaccination coverage of vaccination strategies

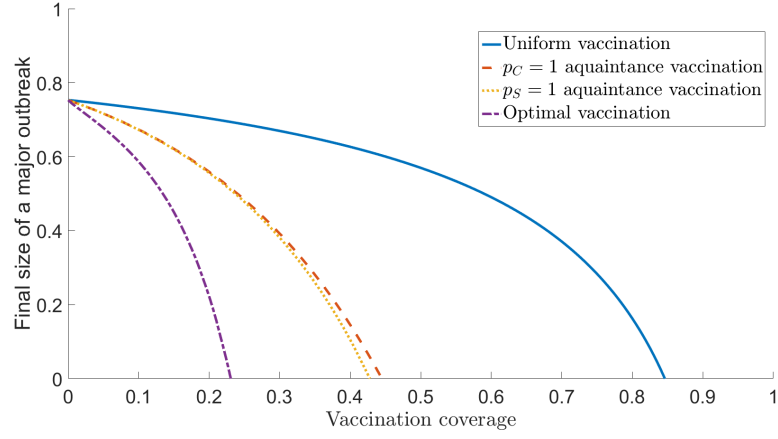
In this section we investigate the effect of global contacts on the uniform, acquaintance and optimal vaccination strategies. To do this we fix the degree distribution and match the network and global models without any vaccination (i.e.  $c = 0$ ) by either  $R_0$  (as in Chapter 4) or by the final size of a major outbreak. We can then compare the critical vaccination coverages of the three vaccination strategies in the different the models.

We consider the extreme cases of acquaintance vaccination with either  $p_S = 1$  or  $p_C = 1$  since, as we discuss in Section 5.1.4, for a fixed vaccination coverage  $R_0^A$  is strictly increasing in  $p_C$ . However, the difference in critical vaccination coverage between these two acquaintance vaccination strategies is small, as illustrated in Figures 5.8, 5.9 and 5.10.

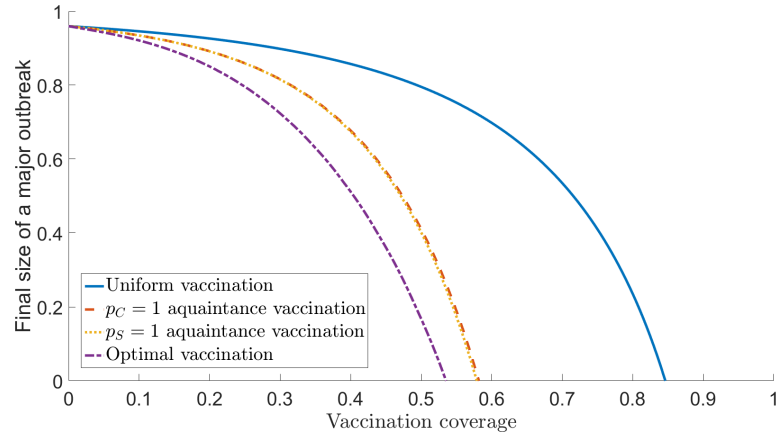
The addition of global contacts (while fixing either  $R_0$  or the final size of a major outbreak) decreases the difference in critical vaccination coverage of the optimal, acquaintance and uniform vaccination strategies, as illustrated in Figures 5.8, 5.9 and 5.10. This is as we would intuitively expect; the effect of vaccination strategies targeting individuals with large degrees will be diluted when the epidemic depends less upon the network structure. Furthermore, a larger decrease in the difference between critical vaccination coverages is observed when the network and global model has a heavy-tailed degree distribution. We also note that with the addition of global contacts the critical vaccination coverage of the acquaintance vaccination strategy often becomes closer to the critical vaccination coverage of the optimal vaccination strategy rather than the critical vaccination coverage of the uniform vaccination strategy, especially when considering heavy-tailed degree distributions, as illustrated in Figure 5.11.

Consider a network and global model with a given fixed degree distribution, infectious period and  $R_0$  and assume that  $\lambda_G = 0$ . With the knowledge that the critical vaccination coverage in the network and global model depends only on  $R_0$  we expect that, as  $\lambda_N$  is decreased and  $\lambda_G$  is increased to fix  $R_0$ , the critical vaccination coverage under the optimal and acquaintance vaccination will converge from below to  $1 - 1/R_0$ . This effect can be observed in Figures 5.8 and 5.9, and occurs regardless of the final size of a major outbreak under no vac-

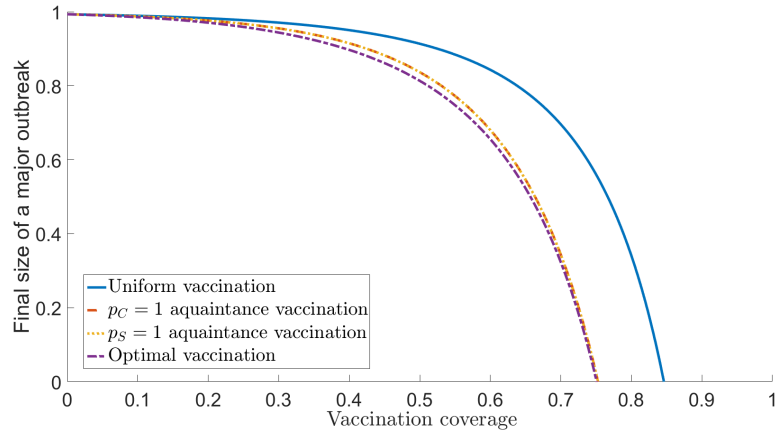




(a)  $\lambda_G = 0$  and  $p_N = 0.41$ .

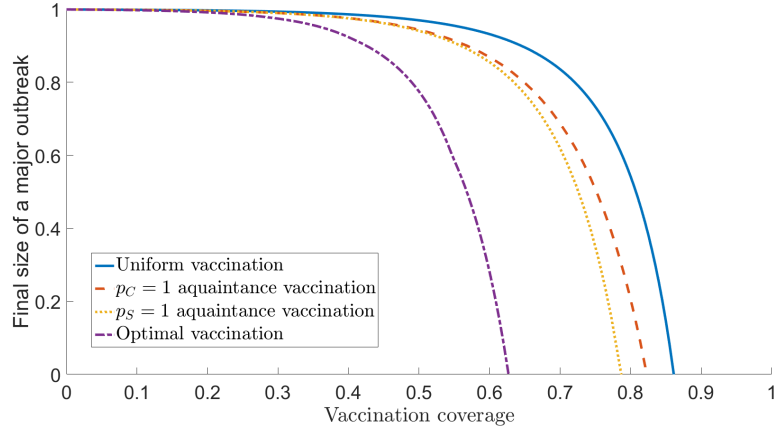


(b)  $\lambda_G = 2$  and  $p_N = 0.33$ .

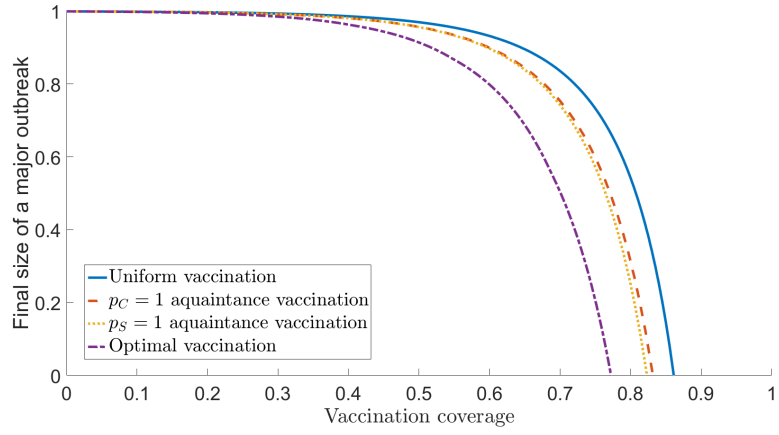


(c)  $\lambda_G = 4$  and  $p_N = 0.23$ .

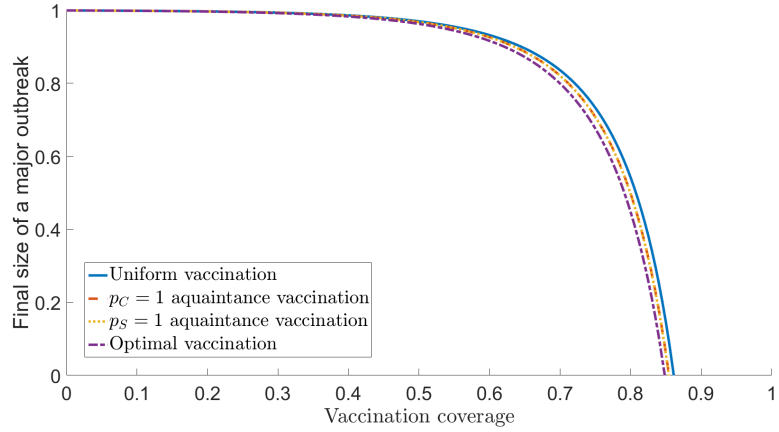
Figure 5.8: The effect of global contacts on vaccination strategies on network and global models matched with  $R_0 = 6.5$ . The other parameters are  $I \sim \text{Const}(1)$  and  $D \sim \text{Geo}(1/9)$ .



(a)  $\lambda_G = 0$  and  $p_N = 0.9$ .

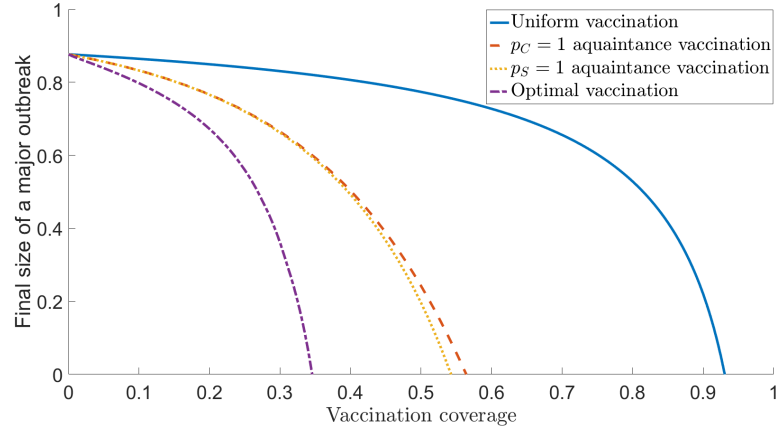


(b)  $\lambda_G = 3.2$  and  $p_N = 0.5$ .

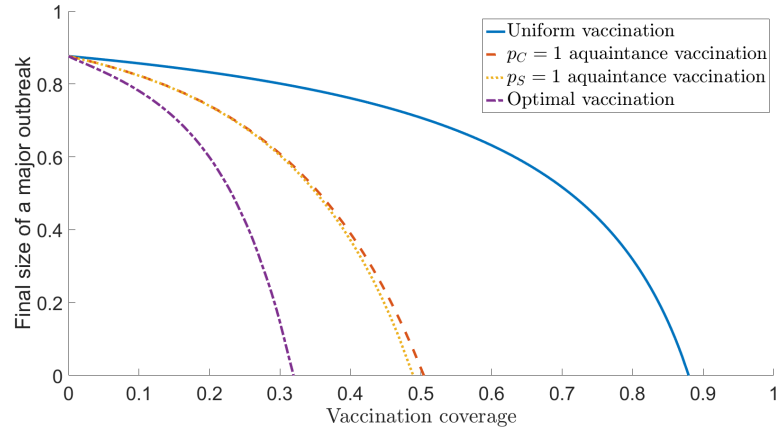


(c)  $\lambda_G = 6.4$  and  $p_N = 0.1$ .

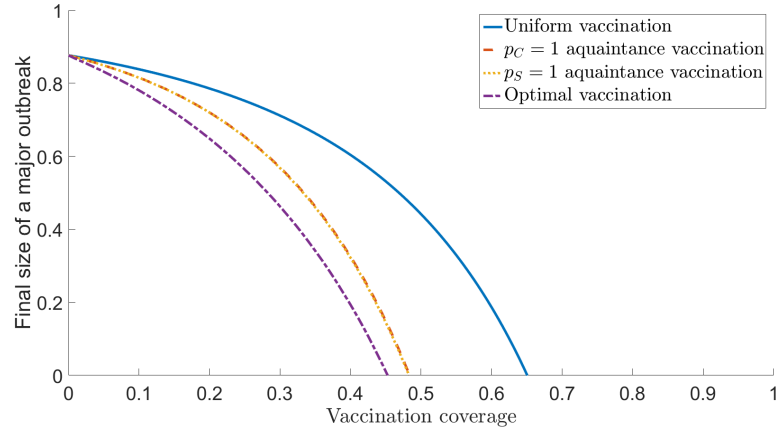
Figure 5.9: The effect of global contacts on vaccination strategies on network and global models matched with  $R_0 = 7.2$ . The other parameters are  $I \sim \text{Const}(1)$  and  $D \sim \text{Poi}(8)$ .



(a)  $\lambda_G = 0$  and  $p_N = 0.9$ , so  $R_0 = 14$ .

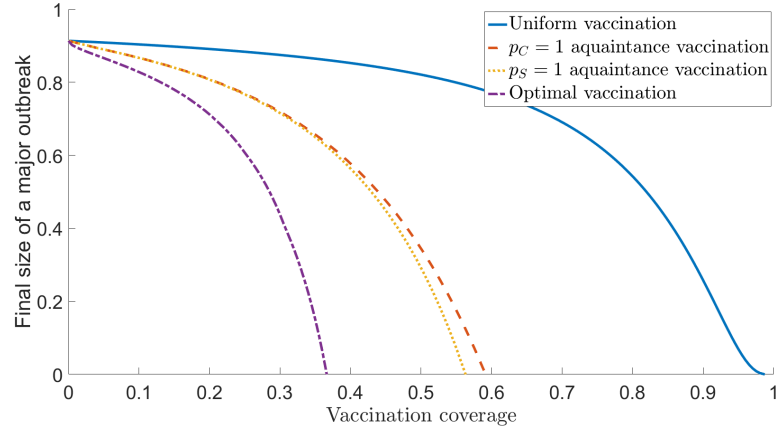


(b)  $\lambda_G = 0.57$  and  $p_N = 0.5$ , so  $R_0 = 8.3$ .

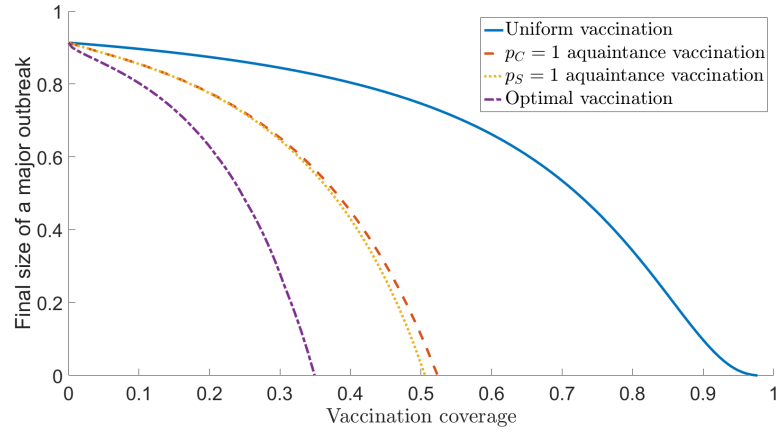


(c)  $\lambda_G = 1.75$  and  $p_N = 0.1$ , so  $R_0 = 2.9$ .

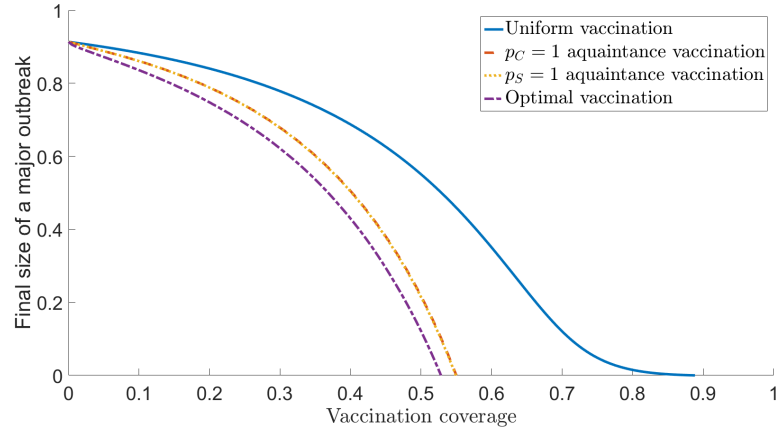
Figure 5.10: The effect of global contacts on vaccination strategies on network and global models matched by final size, with parameters  $I \sim \text{Const}(1)$  and  $D \sim \text{Geo}(1/9)$ .



(a)  $\lambda_G = 0$  and  $p_N = 0.9$ , so  $R_0 = 78$ .



(b)  $\lambda_G = 0.6525$  and  $p_N = 0.5$ , so  $R_0 = 44$ .



(c)  $\lambda_G = 1.75$  and  $p_N = 0.1$ , so  $R_0 = 9$ .

Figure 5.11: The effect of global contacts on vaccination strategies on network and global models matched by final size, with parameters  $I \sim \text{Const}(1)$  and  $D \sim \text{Pow}(8, 3)$ .

cination. Thus (assuming there is a sizeable variance in the degree distribution so that the acquaintance vaccination strategy has a smaller critical vaccination coverage than the uniform vaccination strategy) implementing an acquaintance vaccination strategy will lead to an underestimation of the required critical vaccination coverage if we have accurately estimated  $R_0$  but incorrectly assumed the epidemic spreads primarily via the network, and instead the epidemic spreads via global contacts.

In contrast, if models are matched by the final size of a major outbreak the effect of global contacts on the critical vaccination coverage is less clear, and we discuss some possible interactions in the following paragraphs. Similarly to Chapter 4, in which we consider the difference between the final size of a major outbreak in two network and global models with the same degree distribution matched by  $R_0$ , when we match two network and global models with the same degree distribution by the final size of a major outbreak they are likely to have different basic reproduction numbers, unless  $\sigma_D^2 = \mu_D$ .

Since the critical vaccination coverage of the uniform vaccination strategy depends only on  $R_0$ , the effect of global contacts on the critical vaccination coverage under the uniform vaccination strategy when the models are matched by the final size of a major outbreak is clear: if  $R_0$  increases then the critical vaccination coverage of the uniform vaccination strategy will increase, and if  $R_0$  decreases then the critical vaccination coverage will decrease, as illustrated in Figure 5.10.

Consideration of the optimal and acquaintance vaccination strategies is more complex. We expect both these strategies to be most effective when there is sizeable variance in the degree distribution. However, if there is a sizeable variance in the degree distribution then  $R_0$  is generally increasing as the  $\lambda_G$  is decreased while  $p_N$  is increased to maintain the final size of a major outbreak. This creates a tradeoff, in which the model may require a larger critical vaccination coverage under the optimal and acquaintance vaccination strategies due to the increased  $R_0$ , or a smaller critical vaccination coverage owing to the optimal and acquaintance vaccination strategies being more effective in the network due to the large variability in the degree distribution.

When network and global model with a fixed degree distribution are matched

by the final size of a major outbreak, the critical vaccination coverage under the optimal or acquaintance vaccination strategy in the model with small  $\lambda_G$  can be an overestimate or an underestimate of the critical vaccination coverage of the vaccination strategy when  $\lambda_G$  is increased, as illustrated in Figures 5.10 and 5.9. Furthermore, it is possible that the critical vaccination coverage of the optimal or acquaintance vaccination strategy on the standard network model is an overestimate of the critical vaccination coverage of the strategy when a small number of global contacts are introduced (compare Figures 5.11a and 5.11b for the optimal vaccination strategy) but also an underestimate of the critical vaccination coverage of the vaccination strategy when a large number of global contacts are introduced (compare Figures 5.11b and 5.11c for the optimal vaccination strategy).

Finally, recall that the final size of a major outbreak in the network and global model with a Poisson degree distribution depends on  $R_0$  but not the specific value of  $\lambda_G$  and  $p_N$  (see Proposition 4.1). However the specific value of  $p_N$  and  $\lambda_G$  does have an impact on the critical vaccination coverages of the acquaintance and optimal vaccination strategies, as illustrated in Figure 5.9.

## 5.6 Concluding remarks

In this chapter we consider three vaccination strategies on the network and global model, specifically the acquaintance, uniform and optimal vaccination strategies. For each vaccination strategy, we show how to find a threshold parameter determining whether a major outbreak can occur and the final size of a major outbreak.

Under the acquaintance vaccination strategy we prove that, for a fixed vaccination coverage, maximising  $p_C$  will maximise  $R_0^A$  and the final size of a major outbreak. Under the uniform vaccination strategy, we show that the critical vaccination coverage of the network and global model is equal to  $1 - 1/R_0$ , where  $R_0$  is the basic reproduction number of the network and global model under no vaccination. We prove that the critical vaccination coverage of the acquaintance vaccination strategy is not always smaller than the critical vaccination coverage of the uniform vaccination strategy, and we give conditions under which the acquaintance vaccination strategy has a larger critical vaccination coverage than the uniform vaccination strategy.

We compare our asymptotic calculations to simulations of the epidemic in finite populations. Our results are as expected under the optimal and uniform vaccination strategies, with the asymptotic calculation of the final size of a major outbreak being an overestimation of the simulated final size of a major outbreak in finite populations. However, this relationship is reversed under the acquaintance vaccination strategy, with the asymptotic calculation of the final size of a major outbreak being an underestimation of the simulated final size of a major outbreak in finite populations. Further analysis suggests that the underestimation is caused by the correlation between the vaccination coverage under the acquaintance vaccination strategy and both the indicator function for whether a major outbreak occurs and the final size of a major outbreak.

Finally we investigate the effect of global contacts on the critical vaccination coverage of the optimal, acquaintance and uniform vaccination strategies by comparing models with either  $R_0$  or the final size of a major outbreak kept fixed. We show that the addition of global contacts will lead to a decreased difference in critical vaccination coverage between the vaccination strategies, thus diluting the benefit of the acquaintance vaccination strategy. Furthermore, we show that the critical vaccination coverage in the standard network model under the optimal and acquaintance vaccination strategies matched by  $R_0$  or the final size of a major outbreak can be either an underestimate or overestimate of the critical vaccination coverage when global contacts are added.

Throughout this chapter we assume that we vaccinate individuals with a perfect vaccine. However, this is an unrealistic assumption in many practical applications. Some vaccines never result in full immunity, only reducing the probability of infection, and sometimes vaccinated individuals will not become immune at all. However, the perfect vaccine assumption allows us to analyse the network and global model under the three vaccination strategies with two-type branching processes whereas under the generalised vaccine reaction model we would require the use of 12-type branching processes, which are more difficult to analyse. An extension of this model to the generalised vaccine action model would be an interesting topic for further research.

Although we do not consider the single-neighbour acquaintance vaccination strategy, we conjecture that global contacts would cause a similar dilution to the

effects found for the acquaintance vaccination strategy, and thus the acquaintance vaccination strategy and the single-neighbour acquaintance vaccination strategy would provide similar results, as in Ball and Sirl (2013).

Finally, we acknowledge that the acquaintance vaccination strategy presented here would be very difficult, and indeed potentially morally questionable, to implement in practice in human populations. However, we note that the acquaintance vaccination strategy could be very effective in static computer networks, in which moral questions do not arise. Nevertheless, the study of acquaintance vaccination is still very useful to understand the impact of vaccination strategies that target high degree individuals. Furthermore, understanding exactly why the acquaintance vaccination strategy can underperform compared to the uniform vaccination strategy and why the asymptotic final size of a major outbreak in the network and global model under the acquaintance vaccination strategy is an underestimate for finite populations may result in the construction of better vaccination strategies that can be applied to human populations.



## 5.7 Table of common notation introduced in Chapter 5

Symbol	Meaning	Page
$c$	Vaccination coverage.	136
$c^*$	Critical vaccination coverage.	136
$p_S$	Probability that an individual chosen uniformly at random from the population is sampled under the acquaintance vaccination strategy.	136
$p_C$	Probability that a given network neighbour of a sampled individual is vaccinated under the acquaintance vaccination strategy.	136
$p_V$	Probability that an individual selected uniformly at random from the population is vaccinated.	136
$U$	Event that an individual is unvaccinated.	137
$D_U$	Degree distribution of an unvaccinated (by the acquaintance vaccination strategy) individual chosen uniformly at random from the population.	137
$\tilde{D}_U$	Degree distribution of an unvaccinated (by the acquaintance vaccination strategy) individual contacted via the network.	137
$\tilde{p}_V$	A priori probability that an individual contacted via the network is vaccinated unvaccinated (by the acquaintance vaccination strategy).	137
$I_S$	Event that an individual is sampled under the acquaintance vaccination strategy.	138
$\tilde{p}_{SU}$	Probability that an individual contacted via the network is sampled, given that they did not choose their parent in the branching process for vaccination (under the acquaintance vaccination strategy).	138
$R_0^A$	Basic reproduction number for the network and global model under the acquaintance vaccination strategy.	138

$M^A$	Next-generation matrix for the forward branching process in the network and global model under the acquaintance vaccination strategy.	138
$R_0^U$	Basic reproduction number for the network and global model under the uniform vaccination strategy.	146
$M^U$	Next-generation matrix for the forward branching process in the network and global model under the uniform vaccination strategy.	146
$d_c$	Smallest total degree of an individual which we vaccinate under the optimal vaccination strategy.	150
$\delta$	Proportion of individuals with total degree $d_c$ which are chosen uniformly at random for vaccination under the optimal vaccination strategy.	150
$D_U^O$	Degree distribution of an unvaccinated (by the optimal vaccination strategy) individual chosen uniformly at random from the population.	150
$\tilde{D}_U^O$	Degree distribution of an unvaccinated (by the optimal vaccination strategy) individual contacted via the network.	150
$\tilde{p}_V$	Probability that an individual contacted via the network is vaccinated (under the optimal vaccination strategy).	150
$R_0^O$	Basic reproduction number for the network and global model under the optimal vaccination strategy.	151
$c_U^*$	Critical vaccination coverage under the uniform vaccination strategy.	156
$c_A^*$	Critical vaccination coverage under the acquaintance vaccination strategy.	156

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## 6. The effect of edge-disjoint triangle clustering on vaccination strategies

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In this chapter we introduce a network model with tunable clustering, the ‘rewired edge-triangle’ model. The rewired edge-triangle model consists of the network model with clustering introduced by Newman (2009) extended with rewiring, introduced by Miller (2009). We extend the model further by allowing for partial rewiring, similarly to Ball et al. (2013). Note that Newman (2009) introduces clustering to the standard network model via the addition of edge-disjoint triangles. The rewiring process used in the rewired edge-triangle model allows for a comparison of models differing only in the number of unbroken triangles, so the effect of clustering upon properties of interest, such as the critical vaccination coverage of vaccination strategies, can be isolated.

As the mathematical tools available to model the spread of epidemics on networks evolve there is increasing interest in quantifying the effects of properties of networks, such as the clustering coefficient, upon the spread of an epidemic (see, for example, Miller (2009), Gleeson (2009), Ball et al. (2013) and Coupechoux and Lelarge (2014)). Meanwhile there has also been interest in vaccination strategies that target high degree individuals in the network using local knowledge of the network, such as the acquaintance vaccination strategy discussed in Chapter 5. In networks with zero clustering the acquaintance vaccination strategies have been shown to be very effective (see Ball and Sirl (2013)). However, there has been little investigation into the effect of clustering on the performance of these vaccination strategies. Therefore we introduce three vaccination strategies with a perfect vaccine on the rewired edge-triangle model: the acquaintance vaccination strategy, the uniform vaccination strategy and what we call the ‘optimal’ vaccination strategy. We consider the acquaintance

vaccination strategy discussed in Chapter 5, first introduced by Ball and Sirl (2013), in which neighbours of individuals sampled uniformly at random from the population are chosen for vaccination. Furthermore, we compare the effect of edge-disjoint triangle clustering (i.e. clustering introduced via edge-disjoint triangles) on the performance of the acquaintance vaccination strategy and the uniform vaccination strategy.

This chapter is laid out in the following way. In Section 6.1 we introduce the rewired edge-triangle model, along with the notation required for further analysis of the model, and calculate the clustering coefficient. In Sections 6.2, 6.3 and 6.4 we analyse respectively the uniform, acquaintance and optimal vaccination strategies on the rewired edge-triangle model. In each case we determine a post-vaccination threshold parameter and the expected relative final size of a major outbreak. In Section 6.5 we calculate  $R_0$  for the rewired edge-triangle model under no vaccination and in Section 6.6 we numerically compare the optimal, acquaintance and uniform vaccination strategies and investigate the effect of edge-disjoint triangle clustering on the performance of the vaccination strategies. Finally, we give our concluding remarks in Section 6.7 and a table of common notation introduced in this chapter in Section 6.8.

## 6.1 The rewired edge-triangle model

The rewired edge-triangle model consists of a finite, closed population of  $N$  individuals. To construct the clustered network we assign each individual in the population a number of stub half-edges and corners of triangles according to independent samples from an arbitrary but specified joint probability distribution  $(S, T)$  with  $P(S = s, T = t) = p_{st}$ ,  $s, t = 0, 1, 2, \dots$ . Conditional on the total number of stub half-edges and corners of triangles being a multiple of 2 and 3 respectively, the stub half-edges are then paired uniformly at random to construct complete edges and we choose trios of corners of triangles uniformly at random and join them to form complete triangles. Let  $p_{RW}$ , the probability of rewiring, be a real number satisfying  $0 \leq p_{RW} \leq 1$ . Then, independently for each triangle created by the joining of corners, with probability  $p_{RW}$  each of the three edges in the triangle is broken into two triangle half-edges. We then pair these triangle half-edges uniformly at random which, along with the edges constructed by the pairing of stub half-edges and the unbroken triangles, constructs the network. We say that an individual can have three types of

network neighbours: stub neighbours, created by the pairing of stub half-edges; triangle neighbours, created by the construction of triangles; and rewired triangle neighbours, created by the pairing of broken triangle half-edges. We say that an individual assigned  $s$  stub half-edges and  $t$  corners of triangles before the rewiring process has joint stub and triangle degree  $(s, t)$ .

Similarly to Section 3.1, the construction of the network may yield imperfections in the network, i.e. self-loops and multiple edges. However, if both  $S$  and  $T$  have finite variance then, following arguments analogous to those in Section 3.1, the fraction of such imperfections is negligible as  $N \rightarrow \infty$  (i.e. the total number of these imperfections per individual converges in probability to 0 as  $N \rightarrow \infty$ ) and removing them has no effect on the asymptotic properties we study in this thesis. Furthermore, if both  $S$  and  $T$  have finite variance then (applying analogous arguments to those in Ball et al. (2013)) the fraction of imperfections created by the rewiring process becomes negligible as  $N \rightarrow \infty$ .

To relate the rewired edge-triangle model to the household and network model we can view the triangles in the network as households of size 3. Therefore, in the limit  $N \rightarrow \infty$ , an individual in the population with  $t$  triangles can be said to be part of  $t$  households, each of size 3. Furthermore, we can call the 2-regular graph created by a rewired triangle a rewired household, so in the limit  $N \rightarrow \infty$  an individual in the population with  $r$  rewired triangles can be said to be part of  $r$  rewired households.

We now consider the evolution of the epidemic. Similarly to Chapter 3 we consider an SIR epidemic which starts with a single infectious individual chosen uniformly at random from the population which is otherwise susceptible. Throughout an individual's infectious period it can make infectious contact with any given network neighbour (i.e. each stub, triangle and rewired triangle neighbour) at the points of a Poisson process with rate  $\lambda_N$ . All infectious periods, Poisson processes and samplings from  $(S, T)$  are assumed to be mutually independent. In contrast to the previous chapters we assume that each individual has a constant infectious period of length 1 to simplify the calculations of the spread of the epidemic through triangles and rewired triangles. Finally, we denote the marginal probability that an infected individual infects a given susceptible neighbour by  $p_N = 1 - e^{-\lambda_N}$ .

Our analysis is of the  $N \rightarrow \infty$  limiting epidemic process so, similarly to the model with three levels of mixing discussed in Section 3.2.1, we approximate the early stages of the epidemic with a forward branching process. We conjecture that the branching process approximation can be made rigorous similarly to the proof of Theorem 3.1 given in Section 3.5. Namely that we can construct the branching process and, for each  $N = 1, 2, \dots$ , a realisation of the epidemic process on a common probability space and use a coupling argument to show that, as  $N \rightarrow \infty$ , the total number of infected individuals in the epidemic process converges in distribution to the total progeny of the forward branching process. Thus whether or not a major outbreak occurs with non-zero probability is determined by whether or not the forward branching process is supercritical. Furthermore, we conjecture that if a major outbreak does occur then, as  $N \rightarrow \infty$ , its expected relative final size converges in distribution to the survival probability of the backwards branching process which represents the spread of an individual's susceptibility set (see Section 3.3.1). We hypothesize that this argument can be made rigorous by applying similar arguments to those used for the network and household model in Ball et al. (2009), namely that we can use the backwards branching process to calculate the probability that a given individual is infected in the event of a major outbreak and, by an exchangeability argument, show that this probability is equal to the asymptotic mean proportion of the population that are ultimately infected by a major outbreak, i.e. the expected relative final size of a major outbreak.

### 6.1.1 Notation

The probability generating functions introduced in this section were given by Newman (2009) and are used in calculating the properties of the rewired edge-triangle model. Let  $f_{S,T}(x, y) = \mathbb{E} [x^S y^T]$  be the joint probability generating function for the stub and triangle distribution. Therefore the probability  $p_k$ ,  $k = 0, 1, \dots$ , that an individual has  $k$  network neighbours in total (both via stubs, through triangles and rewired triangles) is

$$p_k = \sum_{s=0}^{\infty} \sum_{t=0}^{\infty} p_{st} \delta_{k, s+2t}, \quad (6.1)$$

since each stub contributes 1 to the total degree and each corner of a triangle contributes 2. Let  $D_T$  be the total degree distribution, so  $\mathbb{P}(D_T = k) = p_k$ ,

$k = 0, 1, \dots$ . Then, substituting equation (6.1),

$$f_{D_T}(x) = \sum_{k=0}^{\infty} p_k x^k = \sum_{k=0}^{\infty} \sum_{s=0}^{\infty} \sum_{t=0}^{\infty} p_{st} \delta_{k,s+2t} x^k = f_{S,T}(x, x^2). \quad (6.2)$$

To investigate the rewired edge-triangle model we also require two size-biased distributions: the joint distribution of the number of stubs and corners of triangles attached to a vertex reached by traversing a stub (including the traversed stub), which is denoted by  $q_{st}$ , and the corresponding distribution for a vertex reached by traversing a triangle or rewired triangle, which is denoted by  $r_{st}$ . So, for  $s, t = 0, 1, \dots$ ,

$$q_{st} = \frac{sp_{st}}{\mathbb{E}[S]} \quad \text{and} \quad r_{st} = \frac{tp_{st}}{\mathbb{E}[T]}.$$

Then the probability generating functions for the stub and triangle size-biased distributions for an individual reached via a stub or triangle (or rewired triangle) are given respectively by

$$f_{\tilde{S}-1, T^S}(x, y) = \sum_{s=0}^{\infty} \sum_{t=0}^{\infty} q_{s+1,t} x^s y^t = \frac{1}{\mathbb{E}[S]} \frac{\partial f_{S,T}}{\partial x}(x, y),$$

and

$$f_{S^T, \tilde{T}-1}(x, y) = \sum_{s=0}^{\infty} \sum_{t=0}^{\infty} r_{s,t+1} x^s y^t = \frac{1}{\mathbb{E}[T]} \frac{\partial f_{S,T}}{\partial y}(x, y).$$

To construct the rewired edge-triangle model with a given total degree distribution we consider the following method. Given the target total degree distribution,  $D_T$ , we choose a parameter  $p \in [0, 1]$  which controls the proportion of edges allocated to triangles. So, given that an individual has total degree  $d$ , the number of triangles this individual belongs to is binomially distributed with parameters  $\lfloor d/2 \rfloor$  and  $p$ . Therefore we say that a model has total degree distribution  $D_T$  and triangle allocation distribution  $\text{Bin}(D_T, p)$  if the stub and triangle distribution  $(S, T)$  is given by, for  $s, t = 0, 1, \dots$ ,

$$\mathbb{P}(S = s, T = t) = \binom{\lfloor (s+2t)/2 \rfloor}{t} p^t (1-p)^{\lfloor (s+2t)/2 \rfloor - t} p_{s+2t}. \quad (6.4)$$

### 6.1.2 Clustering coefficient and degree correlation

There are several measures of clustering in the literature. For example, Watts and Strogatz (1998) introduce a local clustering coefficient, measuring how close a vertex is to being part of a complete graph, and then average the local

clustering coefficient over all vertices within the network to calculate an overall clustering measure. However, we use the ‘probabilistic’ measure of clustering (cf. Trapman (2007)), for which we need the following definitions. We say that a triplet consists of three connected vertices and that a triplet is closed if the first and last vertex is the same, i.e. a triangle. Then for a network consisting of  $N$  vertices we define the clustering coefficient of the network,  $\mathcal{C}^{(N)}$ , to be the number of closed triplets in the network (i.e. three times the number of triangles) divided by the total number of triplets. Note that  $\mathcal{C}^{(N)}$  is then the probability that an ordered triplet of nodes  $(i, j, k)$  chosen uniformly at random from all triplets in the network has the property that  $i$  and  $k$  are connected (i.e. that  $(i, j, k)$  form a triangle). Finally, we say that the clustering coefficient of the asymptotic network is given by  $\lim_{N \rightarrow \infty} \mathcal{C}^{(N)}$ .

We now consider the clustering coefficient of the rewired edge-triangle model, previously given for the case  $p_{RW} = 1$  in Newman (2009). Let  $\mathcal{C}^\Delta$  be the clustering coefficient in the rewired edge-triangle model and, in a population of size  $N$ , let  $N_\Delta^{(N)}$  and  $N_3^{(N)}$  be the total number of triangles and triplets respectively, so  $\mathcal{C}^{(N)} = N_\Delta^{(N)} / N_3^{(N)}$  and  $\mathcal{C}^\Delta = \lim_{N \rightarrow \infty} \mathcal{C}^{(N)}$ .

Before rewiring, for a large population size the strong law of large numbers implies that the number of triangles in the network per individual is well approximated by  $E[T]$ . So, since triangles are rewired independently and uniformly at random with probability  $p_{RW}$ ,  $N^{-1}N_\Delta^{(N)}$  is well approximated by  $(1 - p_{RW})E[T] = (1 - p_{RW})\frac{\partial f_{S,T}}{\partial y}(1, 1)$ . An individual with total degree  $k$  contributes to  $\binom{k}{2}$  triplets so, in a large population, the law of large numbers implies that  $N^{-1}N_3$  is well approximated by  $\sum_{k=0}^{\infty} \binom{k}{2} p_k = \frac{1}{2} \frac{d^2 f_{D,T}}{dx^2}(1)$ . Therefore

$$\mathcal{C}^{(N)} \approx \frac{(1 - p_{RW})\frac{\partial f_{S,T}}{\partial y}(1, 1)}{\frac{1}{2} \frac{d^2 f_{D,T}}{dx^2}(1)}.$$



This formula becomes exact as  $N \rightarrow \infty$  so, using equation (6.2),

$$\begin{aligned}
\mathcal{C}^\Delta &= \frac{(1 - p_{RW})}{2} \frac{\frac{\partial f_{S,T}}{\partial y}(1, 1)}{\frac{d^2 f_{D_T}}{dx^2}(1)} \\
&= \frac{(1 - p_{RW})}{2} \left( \frac{\frac{\partial f_{S,T}}{\partial y}(1, 1)}{\frac{\partial^2 f_{S,T}}{\partial x^2}(1, 1) + 4 \frac{\partial^2 f_{S,T}}{\partial x \partial y}(1, 1) + 2 \frac{\partial^2 f_{S,T}}{\partial y^2}(1, 1) + 4 \frac{\partial^2 f_{S,T}}{\partial y^2}(1, 1)} \right) \\
&= \frac{(1 - p_{RW})}{2} \left( \frac{\mathbb{E}[T]}{\mathbb{E}[S(S-1)] + 4\mathbb{E}[ST] + 2\mathbb{E}[T^2] + 2\mathbb{E}[T(T-1)]} \right).
\end{aligned}$$

Note that in the limit  $N \rightarrow \infty$  the network will have non-zero clustering if and only if  $p_{RW} < 1$  and  $\mathbb{P}(T = 0) < 1$ . However, although the rewired edge-triangle model can produce clustered networks, the model cannot produce networks with a large mean total degree and clustering coefficient (see Wang et al. (2014)). For example, it is clear that  $\lim_{\mathbb{E}[D_T] \rightarrow \infty} \mathcal{C}^\Delta = 0$ , and this limitation can be problematic when parameterising the rewired edge-triangle model using real-world data. The limitation of the mean total degree and clustering coefficient has been mentioned previously in the literature (see, for example, Gleeson et al. (2010) or Heath and Parikh (2011)) and is caused by the rewired edge-triangle model over-using edges to create triangles (recall that all triangles in the rewired edge-triangle model are edge-disjoint). To see this, assume that  $p_{RW} = 0$  (so that the clustering coefficient is maximised) and consider an individual with  $s$  stub neighbours who is part of  $t$  triangles. Increasing the total degree of this individual by the addition of another triangle will increase  $N_\Delta$  by 1, but the number of triplets will increase by  $2s + 2t + 1$ , leading to an overall decrease in the clustering coefficient.

For example, consider the rewired edge-triangle model with total degree distribution  $D_T \sim \text{Geo}^+(1/2)$  and triangle allocation distribution  $\text{Bin}(D_T, 1)$ . Then  $\mathbb{E}[D_T] = 2$  and if  $p_{RW} = 0$  then  $\mathcal{C}^\Delta = 1/3$ . However, if  $D_T \sim \text{Geo}^+(1/4)$  and the triangle allocation distribution is given by  $\text{Bin}(D_T, 1)$  then  $\mathbb{E}[D_T] = 4$  and if  $p_{RW} = 0$  then  $\mathcal{C}^\Delta = 1/7$ . Thus increasing the mean total degree by 2 has resulted in a greater than 50% reduction in the clustering coefficient.

There are several possible adaptations to the rewired edge-triangle model to allow for increased mean total degree and clustering coefficient combinations. For example, Karrer and Newman (2010) propose replacing the addition of triangles with arbitrary subgraphs, e.g. 4-regular squares (an entirely connected

graph with 4 vertices), which allow for an increased mean total degree and clustering coefficient combinations. An alternative suggestion by Gleeson (2009) is to allow individuals to belong to a single clique of a fixed size, a special case of the clustered network model we discuss in Chapter 7.

We now briefly remark on another important property of social networks, the degree correlation. Following Newman (2002*b*), the degree correlation is the correlation between the total degrees of the vertices connected to an edge chosen uniformly at random from all edges in the network. Therefore the degree correlation lies between  $-1$  and  $1$ , with the degree correlation being near  $1$  implying that vertices have similar degrees to their neighbours. Note that the rewiring process does not change the total degree of any individuals in the rewired edge-triangle model, and therefore does not change the degree correlation. Finally, note that if  $P(T = 0) < 1$  and  $p_{RW} = 1$  then the rewired edge-triangle model with total degree distribution  $D_T$  will not have the same properties, such as the threshold parameter and final size of a major outbreak, as the standard network model with degree distribution  $D_T$  due to the differences in degree correlation of the two models.

## 6.2 Uniform vaccination on the rewired edge-triangle model

Under the uniform vaccination strategy each individual in the population is vaccinated with the perfect vaccine independently with probability  $p_V$ . Clearly this means that the vaccination coverage  $c = p_V$ . In the following sections we consider two-type forward and backward branching processes to calculate the threshold parameter and final size of a major outbreak respectively.

### 6.2.1 Threshold parameter

We begin by informally describing the forward branching process used to calculate the threshold parameter  $R_T^U$ . Unlike the household models previously discussed in this thesis an individual in the forward branching process corresponds to a single individual in the epidemic process. Similarly to Section 3.2.1 we are interested in the final outcome of the epidemic and not its precise evolution, so we can think of the process evolving in the following way. First consider the number of stub network contacts made by the initial infective,

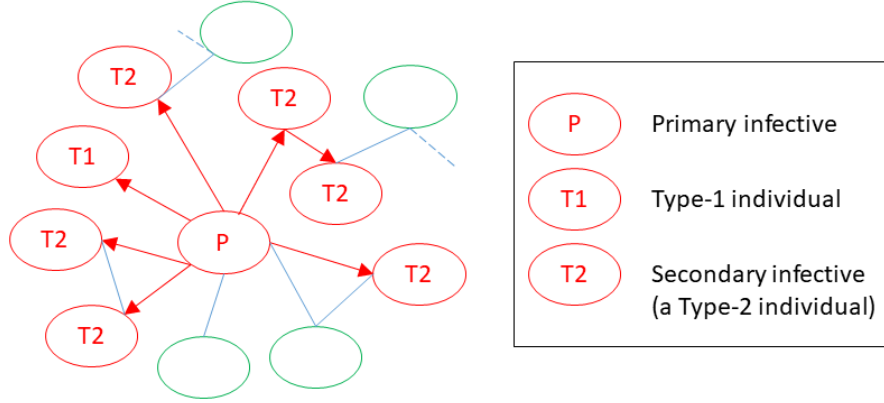


Figure 6.1: Figure illustrating the branching process terminology.

which we call type-1 individuals in the branching process. Then consider the epidemic spreading only through the triangles and rewired triangles containing the initial infective, in which each infected individual corresponds to a type-2 individual in the branching process. We then let each newly infected individual proceed in the same manner. We call the initial infective the primary infective, and any subsequent infected individuals in the triangles and rewired triangles containing the primary infective secondary infectives (as illustrated in Figure 6.1). In the early stages of the epidemic it is likely that each contact will be with uninfected individuals that, except for the triangles and rewired households connected to the primary infective, are in edge-disjoint triangles and rewired households. Note that the offspring distribution of the initial individual (chosen uniformly at random from the population) will be different to the offspring distribution of subsequent generations. The backwards branching process is constructed in an analogous way. Thus for a primary individual,  $i^*$ , type-1 individuals are those that would infect  $i^*$  via a stub, were they to become infected, and type-2 individuals are those that would infect  $i^*$  via a triangle or rewired triangle containing  $i^*$ , were they to become infected.

The spread of the epidemic through the household and network model, see Ball et al. (2009), is approximated by a single type branching process whereas we approximate the spread of the epidemic in the rewired edge-triangle model with a 2-type branching process. The difference in typing is owing to allowing a single individual to be part of multiple triangles and rewired triangles. In the single type branching process approximating the early stages of the epidemic in the household and network model we first consider the spread of the epidemic throughout the household, and the offspring in the forward branching process

are individuals that are contacted via the network by all individuals contacted in the household epidemic. An analogous argument for the rewired edge-triangle model would be to define a household epidemic to consist of all individuals contacted via a chain of contacts made solely via the triangle and rewired triangle edges to the primary infective.

Before calculating the threshold parameter,  $R_T^U$ , which determines whether or not the forward branching process can have infinite progeny and therefore, by definition, whether or not a major outbreak can occur, we briefly comment upon the properties of  $R_T^U$ . Firstly note that  $R_T^U$  is an individual-based reproduction number, unlike the household-based reproduction number  $R_*$ . However, the spread of the forward branching process through the triangles and rewired triangles means that a generation in the forward branching process differs from the global generations (see Section 3.2.3) used to calculate the basic reproduction number  $R_0$ , thus  $R_T^U \neq R_0$ .

The reproduction number  $R_T^U$  is the largest eigenvalue of the mean next-generation matrix  $M^U$  given below in Theorem 6.1. For notational simplicity let  $p_{NV} = p_N(1 - p_V)$  and  $\Delta_E = p_{NV}(3 - 2p_N) + (1 - p_V)(1 - p_N)^2 + p_V$  (the interpretation of these quantities will become clear in the proof).

**Theorem 6.1.** *If  $p_{NV} < 1$  then the mean next-generation matrix  $M^U$  is given by*

$$M^U = p_{NV} \begin{bmatrix} \mathbb{E}[\tilde{S}^S - 1] & 2\mathbb{E}[T^S] \left[ (1 - p_{RW})\Delta_E + p_{RW}\frac{1}{1-p_{NV}} \right] \\ \mathbb{E}[S^T] & 2\mathbb{E}[\tilde{T}^T - 1] \left[ (1 - p_{RW})\Delta_E + p_{RW}\frac{1}{1-p_{NV}} \right] \end{bmatrix}. \quad (6.5)$$

*Proof.* We first consider the expected number of type-1 offspring of a type-1 individual, i.e. the expected number of infectious contacts made via a stub by a type-1 individual. Let  $C_{11}$  be the total number of infectious contacts made via a stub by a type-1 individual. A type-1 individual,  $i^*$  say, has joint stub and triangle distribution  $(\tilde{S}^S, T^S)$  and  $i^*$  makes infectious contact with a given stub neighbour,  $k$  say, if:  $k$  is not vaccinated, occurring with probability  $1 - p_V$ , and  $i^*$  contacts  $k$ , occurring with probability  $p_N$ . So  $i^*$  contacts  $k$  with probability  $(1 - p_V)p_N = p_{NV}$  and

$$C_{11}|\tilde{S}^S, T^S \sim \text{Bin}(\tilde{S}^S - 1, p_{NV}). \quad (6.6)$$

Thus, applying equation (6.6),

$$\mathbb{E}[C_{11}] = \mathbb{E}\left[\mathbb{E}\left[C_{11}|\tilde{S}^S, T^S\right]\right] = p_{NV}\mathbb{E}\left[\tilde{S}^S - 1\right]. \quad (6.7)$$

Similarly, let  $C_{21}$  be the total number of infectious contacts made via a stub by a type-2 individual. A type-2 individual has joint stub and triangle distribution  $(S^T, \tilde{T}^T)$  and analogous arguments to those leading to equation (6.7) yield

$$\mathbb{E}[C_{21}] = \mathbb{E}\left[\mathbb{E}\left[C_{21}|S^T, \tilde{T}^T\right]\right] = p_{NV}\mathbb{E}\left[S^T\right]. \quad (6.8)$$

Next we consider the expected number of type-2 offspring of a type-1 individual, i.e. the expected number of individuals infected within the triangles and rewired triangles containing a type-1 individual. Let  $C_{12}$  be the total number of infectious contacts made via a triangle or rewired triangle by a type-1 individual. We calculate  $C_{12}$  by conditioning on the joint stub and triangle degree of a type-1 individual,  $(\tilde{S}^S, T^S)$  and then decomposing the total number of infected individuals into the number of infected individuals within each intact triangle and rewired triangle. So consider a type-1 individual  $i^*$  with stub and triangle degree  $(\tilde{S}^S, T^S)$  and condition on  $i^*$  being part of  $R \in [0; T^S]$  rewired triangles. Then

$$C_{12} | \tilde{S}^S, T^S, R = \sum_{r=1}^{T^S-R} C_{12}^T(r) + \sum_{r=T^S-R+1}^{T^S} C_{12}^R(r), \quad (6.9)$$

where: we have labelled the  $T^S - R$  triangles containing  $i^*$   $1, 2, \dots, T^S - R$ , we have labelled the  $R$  rewired triangles containing  $i^*$   $T^S - R + 1, T^S - R + 2, \dots, T^S$ ,  $C_{12}^T(r)$  is the number of individuals infected within triangle  $r$  and  $C_{12}^R(r)$  is the number of individuals infected in rewired triangle  $r$ . Recall that we assume a constant infectious period and that all triangles and rewired triangles containing the primary infective  $i^*$  are edge-disjoint. Thus the summands in equation (6.9) are all mutually independent, and also independent of  $\tilde{S}^S, T^S$  and  $R$ .

Before completing our calculation of  $\mathbb{E}[C_{12}]$  we calculate the expected number of individuals infected within a triangle and rewired triangle epidemic. First consider the final size of a single triangle epidemic. For both triangle neighbours of  $i^*$  to become infected we require that both individuals are unvaccinated (occurring with probability  $(1-p_V)^2$ ) and that either the primary infective infects both neighbours (occurring with probability  $p_N^2$ ), or that the primary infective infects a single individual who then infects the final triangle member (occurring with probability  $2p_N^2(1-p_V)$ ). Thus both triangle neighbours of  $i^*$  are infected

with probability  $p_N^2(1-p_V)^2 + 2p_N^2(1-p_N)(1-p_V)^2 = p_N^2(1-p_V)^2(3-2p_N)$ . For only a single triangle neighbour of  $i^*$  to become infected we require that the primary infective infects a single neighbour (occurring with probability  $2p_N(1-p_V)$ ) and that the final triangle neighbour is either vaccinated (occurring with probability  $p_V$ ) or unvaccinated and uninfected (occurring with probability  $(1-p_V)(1-p_N)^2$ ). So with probability  $2p_N(1-p_V)[p_V + (1-p_N)^2(1-p_V)]$  a single triangle neighbour of  $i^*$  becomes infected. Therefore, for  $1 \leq r \leq T^S - R$ ,

$$\begin{aligned} \mathbb{E} [C_{12}^T(r)] &= 2p_N^2(1-p_V)^2(3-2p_N) + 2p_N(1-p_V) [(1-p_N)^2 + p_V] \\ &= 2p_{NV}\Delta_E. \end{aligned} \quad (6.10)$$

Next consider the spread of the epidemic through a rewired triangle. A rewired triangle is constructed by pairing rewired triangle half-edges uniformly at random. Furthermore, as in Ball et al. (2013), in the limit  $N \rightarrow \infty$  the rewired triangle will be locally tree-like. Since we assume that the rewired household is locally tree-like in a graph in which every node has degree 2, the final size of a rewired household epidemic is equal in distribution to the sum of two independent geometric distributions with support from 0 and success parameter  $p_{NV}$ . Therefore if  $p_{NV} < 1$  then, for  $T^S - R < r \leq T^S$ ,

$$\mathbb{E} [C_{21}^R(r)] = \frac{2p_{NV}}{1-p_{NV}}. \quad (6.11)$$

We now return to the calculation of  $\mathbb{E} [C_{12}]$ . Note that each triangle in the network is independently rewired with probability  $p_{RW}$ , so  $R|\tilde{S}^S, T^S$  is binomially distributed with parameters  $T^S$  and  $p_{RW}$ . Therefore substituting equations (6.10) and (6.11) into equation (6.9) and substituting  $\mathbb{E} [R|\tilde{S}^S, T^S] = T^S p_{RW}$  yields

$$\mathbb{E} [C_{12}] = 2p_{NV}\mathbb{E} [T^S] \left[ (1-p_{RW})\Delta_E + p_{RW}\frac{1}{1-p_{NV}} \right]. \quad (6.12)$$

Finally, let  $C_{22}$  be the total number of individuals infected within the triangles and rewired triangles containing a type-2 individual. Then analogous arguments to those given in the calculation of  $\mathbb{E} [C_{12}]$  yield

$$\mathbb{E} [C_{22}] = 2p_{NV}\mathbb{E} [\tilde{T}^T - 1] \left[ (1-p_{RW})\Delta_E + p_{RW}\frac{1}{1-p_{NV}} \right]. \quad (6.13)$$

Note that  $m_{i,j}^U = \mathbb{E} [C_{ij}]$ ,  $i, j = 1, 2$ , so equations (6.7), (6.8), (6.12) and (6.13) yield the required entries of  $M^U$  given in equation (6.5) as required.  $\square$

We say that a vaccination strategy can control the epidemic in a model if the vaccination strategy can prevent a major outbreak from occurring without vaccinating every individual in the population. Since we assume that both the stub and triangle distributions have finite variance, each entry in  $M^U$  is finite and there exists a constant  $K \in (0, 1)$  such that  $R_T^U < (1 - p_V)K$ . Thus we can always choose a value of  $p_V < 1$  such that  $R_T^U < 1$  and a major outbreak cannot occur, yielding the following remark.

**Remark 6.1.** *The uniform vaccination strategy can always control the epidemic in the rewired edge-triangle model.*

### 6.2.2 Final size of a major outbreak

As in Chapter 5, we define the expected relative final size of a major outbreak in a population that contains vaccinated individuals to be the fraction of initially susceptible individuals that are ultimately infected by the epidemic in a major outbreak. To recover the proportion of ultimately infected individuals among the entire population, including the  $c$  vaccinated individuals, we must multiply the expected relative final size of a major outbreak by  $1 - c$ . We refer to the expected relative final size of a major outbreak as the final size of a major outbreak.

In Section 6.1 we hypothesize that if a major outbreak does occur then its relative final size converges in distribution to the survival probability of the backwards branching process given informally in Section 6.2.1. For  $i = 1, 2$ , let  $\tilde{\mathbf{B}}_i = (\tilde{B}_{i1}, \tilde{B}_{i2})$  be the offspring random vector of a typical type- $i$  individual in a non-initial generation of the backwards branching process. Similarly, let  $\mathbf{B} = (B_1, B_2)$  be the offspring random vector in the initial generation of the backwards branching process. Let  $b_{\mathbf{B}}(\mathbf{s})$ ,  $\mathbf{s} = (s_1, s_2)$  be the joint probability generating function of  $\mathbf{B}$  and let  $\mathbf{b}_{\tilde{\mathbf{B}}}(\mathbf{s}) = (b_{\tilde{\mathbf{B}}_1}(\mathbf{s}), b_{\tilde{\mathbf{B}}_2}(\mathbf{s}))$  where, for  $i = 1, 2$ ,  $b_{\tilde{\mathbf{B}}_i}(\mathbf{s})$  is the joint probability generating function of  $\tilde{\mathbf{B}}_i$ .

Then the final size of a major outbreak is  $z = 1 - b_{\mathbf{B}}(\boldsymbol{\pi})$ , where  $\boldsymbol{\pi} = (\pi_1, \pi_2)$  is the smallest solution to the set of simultaneous equations  $\boldsymbol{\pi} = \mathbf{b}_{\tilde{\mathbf{B}}}(\boldsymbol{\pi})$ . Recall that  $p_{NV} = p_N(1 - p_V)$ .

**Theorem 6.2.** *If  $p_{NV} < 1$  then the joint probability generating functions for the offspring distributions of the backward Galton-Watson branching process are given by*

$$b_{\tilde{\mathbf{B}}_1}(\mathbf{s}) = f_{\tilde{S}^S, T^S} \left( 1 - p_{NV}(1 - s_1), (1 - p_{RW})b_{\Delta}(s_2) + p_{RW} \left( \frac{1 - p_{NV}}{1 - s_2 p_{NV}} \right)^2 \right),$$

$$b_{\tilde{\mathbf{B}}_2}(\mathbf{s}) = f_{S^T, \tilde{T}^T-1} \left( 1 - p_{NV}(1 - s_1), (1 - p_{RW})b_{\Delta}(s_2) + p_{RW} \left( \frac{1 - p_{NV}}{1 - s_2 p_{NV}} \right)^2 \right),$$

and

$$b_{\mathbf{B}}(\mathbf{s}) = f_{S, T} \left( 1 - p_{NV}(1 - s_1), (1 - p_{RW})b_{\Delta}(s_2) + p_{RW} \left( \frac{1 - p_{NV}}{1 - s_2 p_{NV}} \right)^2 \right),$$

where

$$b_{\Delta}(s_2) = (1 - p_V)^2 p_N^2 (3 - 2p_N) s_2^2 + 2(1 - p_V) p_N \left[ (1 - p_V)(1 - p_N)^2 + p_V \right] s_2 + (p_V + (1 - p_V)(1 - p_N))^2. \quad (6.15)$$

*Proof.* We begin by calculating  $b_{\mathbf{B}}(\mathbf{s})$  and proceed by considering an individual chosen uniformly at random from the population,  $i^*$  say, and condition on  $i^*$ 's joint stub and triangle distribution,  $(S, T)$ . Recall that we assume a constant infectious period of length 1 and note that, in the limit  $N \rightarrow \infty$ , all infectious contacts made to  $i^*$  via a stub originate from individuals in edge-disjoint triangles and rewired triangles which do not contain  $i^*$ . Thus, conditioned on  $(S, T)$ , the number of contacts made to  $i^*$  via stubs is independent of the number of contacts made to  $i^*$  via triangles or rewired triangles so

$$\mathbb{E}[\mathbf{s}^{\mathbf{B}}] = \mathbb{E}[\mathbb{E}[\mathbf{s}^{\mathbf{B}} | S, T]] = \mathbb{E}[\mathbb{E}[s_1^{B_1} | S, T] \mathbb{E}[s_2^{B_2} | S, T]]. \quad (6.16)$$

We now turn our attention to the calculation of  $\mathbb{E}[s_1^{B_1} | S, T]$ . A given stub neighbour of  $i^*$ ,  $k$  say, can only make infectious contact with  $i^*$  if  $k$  is not vaccinated and contacts  $i^*$ . Thus  $B_1 | S, T \sim \text{Bin}(S, p_{NV})$ , and

$$\mathbb{E}[s_1^{B_1} | S, T] = (1 - p_{NV}(1 - s_1))^S. \quad (6.17)$$

To calculate  $\mathbb{E}[s_2^{B_2} | S, T]$  we condition on  $i^*$  being part of  $R \in [0; T]$  rewired triangles and decompose  $B_2 | S, T, R$  into the number of contacts made to  $i^*$



through each triangle or rewired triangle containing  $i^*$ . Hence

$$B_2 \mid S, T, R = \sum_{r=1}^{T-R} B_2^T(r) + \sum_{r=T-R+1}^T B_2^R(r), \quad (6.18)$$

where: we have labelled the  $T - R$  triangles containing  $i^*$   $1, 2, \dots, T - R$ , we have labelled the  $R$  rewired triangles containing  $i^*$   $T - R + 1, T - R + 2, \dots, T$ ,  $B_2^T(r)$  is the number of individuals contacting  $i^*$  through triangle  $r$  and  $B_2^R(r)$  is the number of individuals contacting  $i^*$  through rewired triangle  $r$ . Recall that we assume a constant infectious period and that all triangles and rewired triangles containing the primary individual  $i^*$  are edge-disjoint. Thus the summands in equation (6.18) are all mutually independent, and also independent of  $S$ ,  $T$  and  $R$ .

To complete the calculation of  $E[s_2^{B_2} \mid S, T]$  we consider the size of an individual's triangle and rewired triangle susceptibility set. Let  $\Delta$  be the size of a single triangle susceptibility set. Note that, applying similar arguments to those leading to equation (6.10),

$$\begin{aligned} P(\Delta = 2) &= (1 - p_V)^2 p_N^2 (3 - 2p_N), \\ P(\Delta = 1) &= 2(1 - p_V) p_N \left[ (1 - p_V)(1 - p_N)^2 + p_V \right], \\ P(\Delta = 0) &= (p_V + (1 - p_V)(1 - p_N))^2. \end{aligned}$$

Therefore an expression for  $b_\Delta(s)$  is given in equation (6.15). We next consider the spread of the epidemic through a rewired triangle. Let  $\angle$  be the size of a rewired triangle susceptibility set. A rewired triangle is constructed by pairing rewired triangle half-edges uniformly at random. Furthermore, as in Ball et al. (2013), in the limit  $N \rightarrow \infty$  the rewired triangle will be locally tree-like in a graph in which every node has degree 2. Therefore  $\angle$  is equal in distribution to the sum of two independent geometric distributions with success parameter  $p_{NV}$ , and  $b_\angle(s) = ((1 - p_{NV})/(1 - s_2 p_{NV}))^2$ .

Applying the decomposition of equation (6.18) to  $E[s_2^{B_2} \mid S, T, R]$  and then applying the independence of  $B_2^T(r_1)$ ,  $B_2^T(r_2)$ ,  $B_2^R(r_3)$  and  $B_2^R(r_4)$  and substituting

$b_{\Delta}(s)$  and  $b_{\angle}(s)$  yields

$$\begin{aligned} \mathbb{E} \left[ s_2^{B_2} \middle| S, T, R \right] &= \mathbb{E} \left[ s_2^{\sum_{r=1}^{T-R} B_2^T(r) + \sum_{r=T-R+1}^T B_2^R(r)} \middle| S, T, R \right] \\ &= \mathbb{E} \left[ (b_{\Delta}(s_2))^{T-R} (b_{\angle}(s_2))^R \middle| S, T, R \right]. \end{aligned} \quad (6.19)$$

Each triangle in the network is independently rewired with probability  $p_{RW}$ , so  $R|S, T \sim \text{Bin}(T, p_{RW})$  and substituting equation (6.19) into  $\mathbb{E} \left[ s_2^{B_2} \middle| S, T \right]$  yields

$$\begin{aligned} \mathbb{E} \left[ s_2^{B_2} \middle| S, T \right] &= \mathbb{E} \left[ \mathbb{E} \left[ s_2^{B_2} \middle| S, T, R \right] \right] \\ &= ((1 - p_{RW})b_{\Delta}(s_2) + p_{RW}b_{\angle}(s_2))^T. \end{aligned} \quad (6.20)$$

Substituting equations (6.17) and (6.20) into equation (6.16) yields  $b_{\mathbf{B}}(\mathbf{s})$  given in the statement of the theorem.

Finally, the calculations of  $b_{\mathbf{B}}(\mathbf{s})$ ,  $b_{\tilde{\mathbf{B}}_1}(\mathbf{s})$  and  $b_{\tilde{\mathbf{B}}_2}(\mathbf{s})$  differ only in the joint stub and triangle distributions considered. An individual contacted via a stub has joint stub and triangle distribution  $(\tilde{S}^S, T^S)$  and an individual contacted via a triangle or rewired triangle has joint stub and triangle distribution  $(S^T, \tilde{T}^T)$ . Therefore analogous arguments to the calculation of  $b_{\mathbf{B}}(\mathbf{s})$  yields both  $b_{\tilde{\mathbf{B}}_1}(\mathbf{s})$  and  $b_{\tilde{\mathbf{B}}_2}(\mathbf{s})$  as required.  $\square$

## 6.3 Acquaintance vaccination on the rewired edge-triangle model

### 6.3.1 Description of acquaintance vaccination

In this section we consider an extension of the acquaintance vaccination strategy with a perfect vaccine, introduced in Ball and Sirl (2013), to the rewired edge-triangle model. Under the acquaintance vaccination strategy each individual in the population is sampled independently with probability  $p_S$  and each network neighbour (i.e. each stub, triangle and rewired triangle neighbour) of a sampled individual is independently chosen for vaccination with probability  $p_C$ . Finally, any individual which has been chosen for vaccination at least once is vaccinated with a perfect vaccine.

Under the acquaintance vaccination strategy, for an individual  $i$  to be chosen

for vaccination by a given network neighbour  $j$ ,  $j$  must be sampled, occurring with probability  $p_S$ , and choose  $i$  for vaccination, occurring with conditional probability  $p_C$ . Therefore the probability that an individual is not chosen for vaccination by a given network neighbour is  $1 - p_S p_C$ . Thus, since an individual  $i$  chosen uniformly at random from the population has  $S + 2T$  network neighbours, each of whom does not choose  $i$  for vaccination independently with probability  $1 - p_S p_C$ , the probability that an individual chosen uniformly at random from the population is vaccinated is

$$p_V = 1 - \sum_{s=0}^{\infty} \sum_{t=0}^{\infty} p_{st} (1 - p_S p_C)^{s+2t} = 1 - f_{S,T} \left( 1 - p_S p_C, (1 - p_S p_C)^2 \right). \quad (6.21)$$

By definition,  $p_V$  is also the vaccination coverage of the acquaintance vaccination strategy.

In contrast to the branching process used to calculate the post-vaccination threshold parameter for the uniform vaccination strategy, discussed in Section 6.2.1, we now use a 4-type Galton-Watson branching process to approximate the early stages of the epidemic. This is due to the acquaintance vaccination strategy causing a difference in degree distribution between individuals contacted via a triangle and a rewired triangle, and the final size of a triangle epidemic being dependent upon the number of sampled and unsampled individuals within the triangle and the different offspring distributions of sampled and unsampled individuals. We now give an informal description of the discrete time 4-type Galton-Watson branching process, i.e. the forward branching process, used to approximate the early stages of the epidemic. Similarly to Section 6.2.1, an individual in the forward branching process corresponds to a single individual in the epidemic process. Furthermore we are interested in the final outcome of the epidemic and not its precise evolution so, similarly to Section 3.2.1, we can think of the process evolving in the following way. First consider the number of infectious stub network contacts made by the initial infective, which we call type-1 individuals in the branching process. Then consider the epidemic spreading only through the triangles containing the initial infective, in which each infected sampled or unsampled individual results in a type-2 or type-3 individual respectively in the branching process. Finally, consider the epidemic spreading only through the rewired households containing the initial infective, in which each infected individual results in a type-4 individual in the branching process. We then consider each newly infected individual in the same manner. In the

early stages of the epidemic it is likely that each contact will be with uninfected individuals that, except for the triangles and rewired households containing the initial infective, are in edge-disjoint triangles and rewired households. Note that the offspring distribution of the initial individual (chosen uniformly at random from the population) will be different to the offspring distribution of subsequent generations.

Type of individual in branching process	Corresponding individual in epidemic process
Type-1	Individual contacted via a stub.
Type-2	Sampled individual contacted via a triangle.
Type-3	Unsampled individual contacted via a triangle.
Type-4	Individual contacted via a rewired household.

Before considering a threshold parameter and the final size of a major outbreak for this model, we calculate the joint stub and triangle distributions of the following. An unvaccinated individual chosen uniformly at random from the population, an unvaccinated individual contacted via a stub, an unvaccinated individual contacted via a triangle and an unvaccinated individual contacted via a rewired triangle. We note that the calculations in the remainder of this section are very similar to the work of Ball and Sirl (2013), (2016).

Consider an unvaccinated individual,  $i$  say, that has been chosen uniformly at random from the population. Denote  $i$ 's joint stub and triangle distribution by  $(S_U, T_U)$ . Then a priori  $i$ 's joint stub and triangle distribution is distributed according to  $(S, T)$  and  $i$  is unvaccinated with probability  $1 - p_V$ , given in equation (6.21). Let  $U$  be the event that an individual is unvaccinated. Then the probability mass function of  $(S_U, T_U)$  is given by, for  $s, t = 0, 1, \dots$ ,

$$\begin{aligned}
P(S_U = s, T_U = t) &= \frac{P(S = s, T = t) P(U | (S = s, T = t))}{P(U)} \\
&= \frac{p_{st}(1 - p_S p_C)^{s+2t}}{1 - p_V}.
\end{aligned} \tag{6.22}$$

Next consider an unvaccinated individual contacted via a stub,  $i$  say, and denote  $i$ 's joint stub and triangle distribution by  $(\tilde{S}_U^S, T_U^S)$ . Then  $i$  has unconditional joint stub and triangle distribution  $(\tilde{S}^S, T^S)$  and we know that  $i$  avoids

vaccination by all of its neighbours. Note that we do not count  $i$ 's parent in the branching process, which must not vaccinate  $i$  by definition (leading to the  $s - 1$  term in equation (6.23)). Therefore, for  $s = 1, 2, \dots, t = 0, 1, \dots$ ,

$$\mathrm{P}(\tilde{S}_U^S = s, T_U^S = t) = \frac{\mathrm{P}(\tilde{S}^S = s, T^S = t, U)}{\mathrm{P}(U)} = \frac{q_{st}(1 - p_{SPC})^{s-1+2t}}{1 - \tilde{q}_V}, \quad (6.23)$$

where

$$\begin{aligned} \tilde{q}_V &= \sum_{s=0}^{\infty} \sum_{t=0}^{\infty} q_{st}(1 - (1 - p_{SPC})^{s-1+2t}) \\ &= 1 - f_{\tilde{S}^{S-1}, T^S}(1 - p_{SPC}, (1 - p_{SPC})^2) \end{aligned} \quad (6.24)$$

is the a priori probability that  $i$  is vaccinated.

Next consider an unvaccinated individual contacted via a triangle,  $i$  say, and denote  $i$ 's joint stub and triangle distribution by  $(S_U^T, \tilde{T}_U^T)$ . Then  $i$  has unconditional joint stub and triangle distribution  $(S^T, \tilde{T}^T)$  and we know that  $i$  avoids vaccination by all of its neighbours. Note that we do not count the triangle containing  $i$ 's parent in the branching process, which must not vaccinate  $i$  by definition (leading to the  $t - 2$  term in equation (6.25)). Therefore, for  $s = 0, 1, \dots, t = 1, 2, \dots$ ,

$$\mathrm{P}(S_U = s, \tilde{T}_U^T = t) = \frac{\mathrm{P}(S^T = s, \tilde{T}^T = t, U)}{\mathrm{P}(U)} = \frac{r_{st}(1 - p_{SPC})^{s+2t-2}}{1 - \tilde{r}_V}, \quad (6.25)$$

where

$$\begin{aligned} \tilde{r}_V &= \sum_{s=0}^{\infty} \sum_{t=0}^{\infty} r_{st}(1 - (1 - p_{SPC})^{s+2t-2}) \\ &= 1 - f_{S^T, \tilde{T}^{T-1}}(1 - p_{SPC}, (1 - p_{SPC})^2) \end{aligned} \quad (6.26)$$

is the a priori probability that  $i$  is vaccinated.

Finally consider an unvaccinated individual contacted via a rewired triangle,  $i$  say, and denote  $i$ 's joint stub and triangle distribution by  $(S_U^R, \tilde{T}_U^R)$ . Then  $i$  has unconditional joint stub and triangle distribution  $(S^T, \tilde{T}^T)$  and we know that  $i$  avoids vaccination by all of its neighbours. Note that we do not count the segment of the rewired triangle containing  $i$ 's parent in the branching process, which must not vaccinate  $i$  by definition (leading to the  $t - 1$  term in equation

(6.27)). Therefore, for  $s = 0, 1, \dots, t = 1, 2, \dots$ ,

$$\mathbb{P}(S_U^R = s, \tilde{T}_U^R = t) = \frac{\mathbb{P}(S^T = s, \tilde{T}^T = t, U)}{\mathbb{P}(U)} = \frac{r_{st}(1 - p_S p_C)^{s+2t-1}}{1 - \tilde{r}_V^R}, \quad (6.27)$$

where

$$\begin{aligned} \tilde{r}_V^R &= \sum_{s=0}^{\infty} \sum_{t=0}^{\infty} r_{st}(1 - (1 - p_S p_C)^{s+2t-1}) \\ &= 1 - (1 - p_S p_C) f_{S^T, \tilde{T}^T-1}(1 - p_S p_C, (1 - p_S p_C)^2) \end{aligned} \quad (6.28)$$

is the a priori probability that  $i$  is vaccinated.

We denote by  $I_S$  or  $I_S^C$  the events that an individual is sampled or unsampled respectively, and note that we know that an individual contacted via a stub or rewired triangle does not choose its parent in the branching process for vaccination. Therefore the probability that an individual contacted via the network is sampled, given that they did not choose their parent in the branching process for vaccination, is given by

$$\tilde{p}_{SU} = P(I_S | \text{does not choose parent}) = \frac{p_S(1 - p_C)}{1 - p_S p_C}.$$

To simplify the calculations of a threshold parameter and final size of a major outbreak for the rewired edge-triangle model under the acquaintance vaccination strategy, in the next section we consider the spread of the epidemic through triangles and through rewired triangles.

### 6.3.2 The spread of the epidemic through a triangle and rewired triangle

We begin by considering the spread of the epidemic within a triangle. Let  $(\Delta_{SS}^F, \Delta_{SN}^F)$  be respectively the number of sampled and unsampled individuals infected within a single triangle epidemic when the primary infective is sampled. Consider a single triangle containing the primary infective. Then each triangle neighbour of the primary infective (hereafter referred to as a non-primary individual) is independently sampled with probability  $\tilde{p}_{SU}$  and otherwise unsampled. Furthermore, conditioned on the number of sampled individuals in the triangle, each non-primary individual is independently vaccinated or unvaccinated. Consider a triangle in which the primary infective is sampled and there are  $j$ ,

$j = 0, 1, 2$ , sampled non-primary individuals. To be unvaccinated, each sampled non-primary individual must avoid vaccination from the sampled primary infective (occurring with probability  $(1 - p_C)$ ), the  $j - 1$  other non-primary individuals (occurring with probability  $(1 - p_C)^{j-1}$ ) and its other stub and triangle neighbours (occurring with probability  $1 - \tilde{r}_V$ ). Therefore, conditioned upon the triangle containing  $j$  non-primary sampled individuals,  $j = 0, 1, 2$ , the number of secondary unvaccinated sampled individuals has a Binomial distribution with parameters  $(j, (1 - \tilde{r}_V)(1 - p_C)^j)$ . Analogous arguments yield that, conditioned upon the triangle containing  $j$  non-primary sampled individuals,  $j = 0, 1, 2$ , the number of secondary unvaccinated unsampled individuals has a Binomial distribution with parameters  $(2 - j, (1 - \tilde{r}_V)(1 - p_C)^{j+1})$ . Finally, since we assume a constant infectious period and all Poisson processes are independent, an infected individual independently contacts each given triangle neighbour with probability  $p_N$ . For notational simplicity let  $\tilde{r}'_{VC} = (1 - \tilde{r}_V)(1 - p_C)$ . Then the joint probability mass function for  $(\Delta_{SS}^F, \Delta_{SN}^F)$  is given by

$$P((\Delta_{SS}^F, \Delta_{SN}^F) = (2, 0)) = \tilde{p}_{SU}^2 (1 - p_C)^2 \tilde{r}'_{VC}{}^2 (3 - 2p_N) p_N^2, \quad (6.29a)$$

$$P((\Delta_{SS}^F, \Delta_{SN}^F) = (0, 2)) = (1 - \tilde{p}_{SU})^2 \tilde{r}'_{VC}{}^2 (3 - 2p_N) p_N^2, \quad (6.29b)$$

$$P((\Delta_{SS}^F, \Delta_{SN}^F) = (1, 1)) = 2\tilde{p}_{SU}(1 - \tilde{p}_{SU})(1 - p_C) \tilde{r}'_{VC}{}^2 (3 - 2p_N) p_N^2, \quad (6.29c)$$

$$P((\Delta_{SS}^F, \Delta_{SN}^F) = (1, 0)) = 2\tilde{p}_{SU}(1 - p_C)(1 - \tilde{r}_V)(1 - \tilde{p}_{SU}p_C) \\ \times [1 - (1 - p_C)\tilde{r}'_{VC}(2 - p_N)p_N] p_N \quad (6.29d)$$

$$P((\Delta_{SS}^F, \Delta_{SN}^F) = (0, 1)) = 2(1 - \tilde{p}_{SU})\tilde{r}'_{VC}(1 - \tilde{p}_{SU}p_C) \\ \times [1 - \tilde{r}'_{VC}(2 - p_N)p_N] p_N, \quad (6.29e)$$

$$P((\Delta_{SS}^F, \Delta_{SN}^F) = (0, 0)) = 1 - \sum_{i+j>0} P((\Delta_{SS}^F, \Delta_{SN}^F) = (i, j)). \quad (6.29f)$$

Similarly, let  $(\Delta_{NS}^F, \Delta_{NN}^F)$  be respectively the number of sampled and unsampled individuals infected within a triangle epidemic when the primary infective is unsampled. Then analogous arguments to those leading to equations (6.29)

yield

$$\begin{aligned}
P\left((\Delta_{NS}^F, \Delta_{NN}^F) = (2, 0)\right) &= \tilde{p}_{SU}^2 \tilde{r}_{VC}'^2 (3 - 2p_N) p_N^2 \\
P\left((\Delta_{NS}^F, \Delta_{NN}^F) = (0, 2)\right) &= (1 - \tilde{p}_{SU})^2 (1 - \tilde{r}_V)^2 (3 - 2p_N) p_N^2 \\
P\left((\Delta_{NS}^F, \Delta_{NN}^F) = (1, 1)\right) &= 2\tilde{p}_{SU}(1 - \tilde{p}_{SU})\tilde{r}_{VC}'(1 - \tilde{r}_V)(3 - 2p_N) p_N^2 \\
P\left((\Delta_{NS}^F, \Delta_{NN}^F) = (1, 0)\right) &= 2\tilde{p}_{SU}(1 - \tilde{r}_V)(1 - \tilde{p}_{SU}p_C) \\
&\quad \times [1 - \tilde{r}_{VC}'(2 - p_N)p_N] p_N \\
P\left((\Delta_{NS}^F, \Delta_{NN}^F) = (0, 1)\right) &= 2(1 - \tilde{p}_{SU})(1 - \tilde{r}_V)(1 - \tilde{p}_{SU}p_C) \\
&\quad \times [1 - (1 - \tilde{r}_V)(2 - p_N)p_N] p_N \\
P\left((\Delta_{NS}^F, \Delta_{NN}^F) = (0, 0)\right) &= 1 - \sum_{i+j>0} P\left((\Delta_{NS}^F, \Delta_{NN}^F) = (i, j)\right).
\end{aligned}$$

Let  $f_{\Delta S}(s_1, s_2)$  and  $f_{\Delta N}(s_1, s_2)$  be the joint probability generating functions for  $(\Delta_{SS}^F, \Delta_{SN}^F)$  and  $(\Delta_{NS}^F, \Delta_{NN}^F)$  respectively.

We now consider the spread of the epidemic through a rewired triangle. Since a rewired triangle is locally tree-like (see the arguments leading to equation (6.11)) we can approximate the spread of the epidemic through a rewired triangle with a single-type branching process. Let  $\angle_S^F$  be the final size of a rewired triangle epidemic belonging to a sampled primary infective and let  $\angle_N^F$  be the final size of a rewired triangle epidemic belonging to an unsampled primary infective. Let  $f_{\angle S}(s)$  and  $f_{\angle N}(s)$  be the probability generating functions for  $\angle_S^F$  and  $\angle_N^F$  respectively.

Consider an infected individual  $i$  in a rewired triangle. Since we assume that the rewired household is locally tree-like, if  $i$  is the primary infective then  $i$  has 2 possible neighbours to infect and if  $i$  is a secondary infective then  $i$  has 1 possible neighbour to infect. A given rewired household neighbour of  $i$ ,  $j$  say, is infected by  $i$  if  $j$ : is not chosen for vaccination by  $i$ , is not already vaccinated by another neighbour and is contacted by  $i$ . So if  $i$  is a sampled primary infective then  $i$  contacts  $j$  with probability  $(1 - p_C)(1 - \tilde{r}_V^R)p_N$ , and if  $i$  is an unsampled primary infective then  $i$  contacts  $j$  with probability  $(1 - \tilde{r}_V^R)p_N$ . A secondary individual in a rewired household epidemic is sampled with probability  $\tilde{p}_{SU}$  and otherwise unsampled, so if  $i$  is a secondary individual then  $i$  contacts  $j$  with probability  $(1 - \tilde{p}_{SU}p_C)(1 - \tilde{r}_V^R)p_N$ . Thus, by standard branching process theory



(Section 2.3),

$$f_{\angle S}(s) = \left[1 - p_N(1 - p_C)(1 - \tilde{r}_V^R) \left(1 - \hat{f}^{(3)}(s)\right)\right]^2, \quad (6.31a)$$

$$f_{\angle N}(s) = \left[1 - p_N(1 - \tilde{r}_V^R) \left(1 - \hat{f}^{(3)}(s)\right)\right]^2, \quad (6.31b)$$

where  $\hat{f}^{(3)}(s)$  is the unique solution in  $[0, 1]$  of the equation

$$\hat{f}^{(3)}(s) = s \left[1 - p_N(1 - \tilde{r}_V^R)(1 - \tilde{p}_{SU}p_C) \left(1 - \hat{f}^{(3)}(s)\right)\right].$$

Finally, we consider the size of a triangle susceptibility set belonging to a sampled individual and an unsampled individual, and the size of a rewired triangle susceptibility set belonging to a sampled individual and an unsampled individual. Let  $(\Delta_{SS}^B, \Delta_{SN}^B)$  be respectively the number of sampled and unsampled individuals contained within a triangle susceptibility set belonging to a sampled individual and let  $(\Delta_{NS}^B, \Delta_{NN}^B)$  be the number of sampled and unsampled individuals contained within a triangle susceptibility set belonging to an unsampled individual. Denote by  $b_{\Delta S}(s_1, s_2)$  and  $b_{\Delta N}(s_1, s_2)$  the joint probability generating functions of  $(\Delta_{SS}^B, \Delta_{SN}^B)$  and  $(\Delta_{NS}^B, \Delta_{NN}^B)$  respectively. Similarly, let  $b_{\angle S}(s)$  and  $b_{\angle N}(s)$  be the probability generating function for the size of a rewired triangle susceptibility set with a sampled and unsampled primary individual respectively. Note that calculating the size of an individual  $i$ 's triangle or rewired triangle susceptibility set corresponds to calculating the number of individuals that would infect  $i$ , were they themselves to become infected, within the triangle or rewired triangle respectively. Since we assume a constant infectious period the calculation of the size of a triangle or rewired triangle susceptibility set is analogous to the calculation of the final size of a triangle or rewired triangle epidemic, so

$$b_{\Delta S}(s_1, s_2) = f_{\Delta S}(s_1, s_2), \quad (6.32a)$$

$$b_{\Delta N}(s_1, s_2) = f_{\Delta N}(s_1, s_2), \quad (6.32b)$$

$$b_{\angle S}(s) = f_{\angle S}(s), \quad (6.32c)$$

$$b_{\angle N}(s) = f_{\angle N}(s). \quad (6.32d)$$

### 6.3.3 Threshold parameter

The reproduction number  $R_T^A$  is the largest eigenvalue of the mean next-generation matrix  $M^A$  given below in Theorem 6.3.

**Theorem 6.3.** *The mean next-generation matrix  $M^A = [m_{ij}^A]$  is given by*

$$\begin{aligned}
\begin{bmatrix} m_{11}^A \\ m_{21}^A \\ m_{31}^A \\ m_{41}^A \end{bmatrix} &= p_N(1 - \tilde{q}_V) \begin{bmatrix} (1 - \tilde{p}_{SUPC}) \mathbb{E} [\tilde{S}_U^S - 1] \\ (1 - p_C) \mathbb{E} [S_U^T] \\ \mathbb{E} [S_U^T] \\ (1 - \tilde{p}_{SUPC}) \mathbb{E} [S_U^R] \end{bmatrix}, \\
\begin{bmatrix} m_{12}^A \\ m_{22}^A \\ m_{32}^A \\ m_{42}^A \end{bmatrix} &= (1 - p_{RW}) \begin{bmatrix} (\tilde{p}_{SU} \mathbb{E} [\Delta_{SS}^F] + (1 - \tilde{p}_{SU}) \mathbb{E} [\Delta_{NS}^F]) \mathbb{E} [T_U^S] \\ \mathbb{E} [\Delta_{SS}^F] \mathbb{E} [\tilde{T}_U^T - 1] \\ \mathbb{E} [\Delta_{NS}^F] \mathbb{E} [\tilde{T}_U^T - 1] \\ (\tilde{p}_{SU} \mathbb{E} [\Delta_{SS}^F] + (1 - \tilde{p}_{SU}) \mathbb{E} [\Delta_{NS}^F]) \mathbb{E} [\tilde{T}_U^R - 1] \end{bmatrix}, \\
\begin{bmatrix} m_{13}^A \\ m_{23}^A \\ m_{33}^A \\ m_{43}^A \end{bmatrix} &= (1 - p_{RW}) \begin{bmatrix} (\tilde{p}_{SU} \mathbb{E} [\Delta_{SN}^F] + (1 - \tilde{p}_{SU}) \mathbb{E} [\Delta_{NN}^F]) \mathbb{E} [T_U^S] \\ \mathbb{E} [\Delta_{SN}^F] \mathbb{E} [\tilde{T}_U^T - 1] \\ \mathbb{E} [\Delta_{NN}^F] \mathbb{E} [\tilde{T}_U^T - 1] \\ (\tilde{p}_{SU} \mathbb{E} [\Delta_{SN}^F] + (1 - \tilde{p}_{SU}) \mathbb{E} [\Delta_{NN}^F]) \mathbb{E} [\tilde{T}_U^R - 1] \end{bmatrix}, \\
\begin{bmatrix} m_{14}^A \\ m_{24}^A \\ m_{34}^A \\ m_{44}^A \end{bmatrix} &= p_{RW} \begin{bmatrix} (\tilde{p}_{SU} \mathbb{E} [\angle_S^F] + (1 - \tilde{p}_{SU}) \mathbb{E} [\angle_N^F]) \mathbb{E} [T_U^S] \\ \mathbb{E} [\angle_S] \mathbb{E} [\tilde{T}_U^T - 1] \\ \mathbb{E} [\angle_N] \mathbb{E} [\tilde{T}_U^T - 1] \\ (\tilde{p}_{SU} \mathbb{E} [\angle_S^F] + (1 - \tilde{p}_{SU}) \mathbb{E} [\angle_N^F]) \mathbb{E} [\tilde{T}_U^R - 1] \end{bmatrix},
\end{aligned}$$

where the joint probability distributions for  $(\Delta_{SS}^F, \Delta_{SN}^F)$  and  $(\Delta_{NS}^F, \Delta_{NN}^F)$  are given in equations (6.29) and (6.30) respectively and the probability generating functions for  $\angle_S^F$  and  $\angle_N^F$  are given in equations (6.31).

*Proof.* This proof follows similar arguments to the proof of Theorem 6.1 for the calculation of  $M^U$ . We begin by considering the expected number of offspring of a type-1 individual, i.e. an individual contacted via a stub. Let  $C_{1k}$ ,  $k = 1, 2, 3, 4$ , be the total number of type- $k$  offspring of a typical type-1 individual, so  $m_{1k} = \mathbb{E} [C_{1k}]$ . We first consider the calculation of  $C_{11}$ . Note that a type-1 individual,  $i^*$  say, has joint stub and triangle distribution  $(\tilde{S}_U^S, T_U^S)$  and is sampled with probability  $\tilde{p}_{SU}$  and otherwise unsampled. So  $i^*$  makes infectious contact with a given stub neighbour,  $j$  say, if:  $j$  is not vaccinated by  $i^*$ , occurring with probability  $1 - \tilde{p}_{SUPC}$ ;  $j$  is not vaccinated by another stub or triangle neighbour, occurring with probability  $1 - \tilde{q}_V$ ; and  $i^*$  contacts  $j$ , occurring with probability  $p_N$ . Therefore  $i^*$  contacts  $j$  with probability  $(1 - \tilde{p}_{SUPC})(1 - \tilde{q}_V)p_N$  and, conditioned on  $i^*$ 's stub and triangle distribution  $\tilde{S}_U^S, T_U^S$ ,

$$C_{11} | \tilde{S}_U^S, T_U^S \sim \text{Bin}(\tilde{S}_U^S - 1, (1 - \tilde{p}_{SUPC})(1 - \tilde{q}_V)p_N). \quad (6.34)$$

Considering  $E[C_{11}]$  and applying equation (6.34) yields

$$E[C_{11}] = E\left[E\left[C_{11} \mid \tilde{S}_U^S, T_U^S\right]\right] = (1 - \tilde{p}_{SUPC})(1 - \tilde{q}_V)p_N E[\tilde{S}^S - 1]. \quad (6.35)$$

Next we consider the calculation of  $E[C_{12}]$ , i.e. the expected number of sampled individuals infected within the triangles containing a typical type-1 individual. We calculate  $E[C_{12}]$  by conditioning on the joint stub and triangle degree of a type-1 individual,  $i^*$  say, the number of rewired triangles containing  $i^*$  and whether  $i^*$  is sampled or unsampled. We then decompose the total number of sampled individuals infectiousy contacted by  $i^*$  via triangles and rewired triangles into the number of sampled individuals infectiousy contacted within each triangle and rewired triangle. So consider a type-1 individual  $i^*$  with joint stub and triangle degree  $(\tilde{S}_U^S, T_U^S)$ , and condition on  $i^*$  being sampled ( $I_S$ ) and part of  $R \in [0; T_U^S]$  rewired triangles. Then

$$C_{12} \mid \tilde{S}_U^S, T_U^S, I_S, R = \sum_{r=1}^{T_U^S - R} C_{12}^{ST}(r), \quad (6.36)$$

where we have labelled the  $T_U^S - R$  triangles containing  $i^*$   $1, 2, \dots, T_U^S - R$  and  $C_{12}^{ST}(r)$  is the number of sampled individuals infected within triangle  $r$  given that  $i^*$  is sampled. Recall that we assume a constant infectious period and that all triangles containing the primary infective  $i^*$  are edge-disjoint. Thus the summands in equation (6.36) are all mutually independent, and also independent of  $\tilde{S}_U^S, T_U^S$  and  $R$ . Furthermore, the number of sampled individuals infected by a sampled individual through a triangle is distributed according to  $\Delta_{SS}^F$  (see Section 6.3.2, equations (6.29)), so  $C_{21}^{ST}(r) \stackrel{\mathcal{D}}{=} \Delta_{SS}^F, r = 1, 2, \dots, T_U^S - R$ . Thus taking the expectation of equation (6.36) yields

$$E[C_{12} \mid \tilde{S}_U^S, T_U^S, I_S, R] = (T_U^S - R) E[\Delta_{SS}^F]. \quad (6.37)$$

Similarly, conditioning on  $i^*$  being unsampled, recalling that the number of sampled individuals infected by an unsampled individual through a triangle is distributed according to  $\Delta_{NS}$  (given in equations (6.30)) and following analogous arguments to those leading to equation (6.37) yields

$$E[C_{12} \mid \tilde{S}_U^S, T_U^S, I_S^C, R] = (T_U^S - R) E[\Delta_{NS}^F]. \quad (6.38)$$

Recall that  $i^*$  is sampled with probability  $\tilde{p}_{SU}$  and otherwise unsampled and that each triangle in the network is independently rewired with probability

$p_{RW}$ , so  $R|\tilde{S}_U^S, T_U^S \sim \text{Bin}(T_U^S, p_{RW})$ . Thus considering the expectation of  $C_{12}$  and substituting equations (6.37) and (6.38) yields

$$\begin{aligned}
\mathbb{E}[C_{12}] &= \mathbb{E}[\mathbb{E}[C_{12}|\tilde{S}_U^S, T_U^S, R]] \\
&= \mathbb{E}[\mathbb{E}[(T_U^S - R)\{\tilde{p}_{SU}\mathbb{E}[\Delta_{SS}^F] + (1 - \tilde{p}_{SU})\mathbb{E}[\Delta_{NS}^F]\}|\tilde{S}_U^S, T_U^S, R]] \\
&= \mathbb{E}[\mathbb{E}[T_U^S(1 - p_{RW})\{\tilde{p}_{SU}\mathbb{E}[\Delta_{SS}^F] + (1 - \tilde{p}_{SU})\mathbb{E}[\Delta_{NS}^F]\}|\tilde{S}_U^S, T_U^S]] \\
&= \mathbb{E}[T_U^S](1 - p_{RW})\{\tilde{p}_{SU}\mathbb{E}[\Delta_{SS}^F] + (1 - \tilde{p}_{SU})\mathbb{E}[\Delta_{NS}^F]\}. \tag{6.39}
\end{aligned}$$

We now consider the calculation of  $\mathbb{E}[C_{13}]$ , i.e. the expected number of unsampled individuals infected within the triangles containing a typical type-1 individual. The calculation of  $\mathbb{E}[C_{13}]$  follows analogous arguments to those for the calculation of  $\mathbb{E}[C_{12}]$ , differing only in counting the number of infected unsampled individuals instead of the number of infected sampled individuals. Recall that the number of sampled individuals infected by a sampled or unsampled individual through a triangle is distributed according to  $\Delta_{SN}$  or  $\Delta_{NN}$  respectively (see Section 6.3.2, equations (6.29) or (6.30)). Thus

$$\mathbb{E}[C_{13}] = \mathbb{E}[T_U^S](1 - p_{RW})\{\tilde{p}_{SU}\mathbb{E}[\Delta_{SN}^F] + (1 - \tilde{p}_{SU})\mathbb{E}[\Delta_{NN}^F]\}. \tag{6.40}$$

Next we consider the calculation of  $\mathbb{E}[C_{14}]$ , i.e. the expected number of individuals infected within the rewired triangles containing a typical type-1 individual. The calculation of  $\mathbb{E}[C_{14}]$  follows similar arguments to the calculation of  $\mathbb{E}[C_{12}]$ , although considering the total number of individuals contacted through rewired triangle epidemics rather than intact triangle epidemics. So consider a type-1 individual  $i^*$  with joint stub and triangle degree  $(\tilde{S}_U^S, T_U^S)$  and condition on  $i^*$  being sampled and part of  $R \in [0; T_U^S]$  rewired triangles. Then

$$C_{14} | \tilde{S}_U^S, T_U^S, I_S, R = \sum_{r=1}^R C_{12}^{SR}(r), \tag{6.41}$$

where we have labelled the  $R$  rewired triangles containing  $i^*$   $1, 2, \dots, R$  and  $C_{12}^{SR}(r)$  is the number of individuals infected within rewired triangle  $r$  given that  $i^*$  is sampled. Recall that we assume a constant infectious period and that all rewired triangles containing the primary infective  $i^*$  are edge-disjoint. Thus the summands in equation (6.41) are all mutually independent, and also independent of  $\tilde{S}_U^S, T_U^S$  and  $R$ . Furthermore,  $C_{14}^{SR}(r) \stackrel{\mathcal{D}}{=} \angle_S^F, r = 1, 2, \dots, R$ , with  $\angle_S^F$  given in equation (6.31a). Thus taking the expectation of equation (6.41)

yields

$$\mathbb{E} [C_{14} | \tilde{S}_U^S, T_U^S, I_S, R] = RE [\angle_S^F]. \quad (6.42)$$

Similarly, conditioning on  $i^*$  being unsampled, recalling that the number of individuals infected by an unsampled individual through a rewired triangle is distributed according to  $\angle_N^F$ , given in equation (6.31b), and following analogous arguments to those leading to equation (6.42) yields

$$\mathbb{E} [C_{14} | \tilde{S}_U^S, T_U^S, I_S^C, R] = RE [\angle_N^F]. \quad (6.43)$$

Recall that  $i^*$  is sampled with probability  $\tilde{p}_{SU}$  and otherwise unsampled and that each triangle in the network is independently rewired with probability  $p_{RW}$ , so  $R | \tilde{S}_U^S, T_U^S \sim \text{Bin}(T_U^S, p_{RW})$ . Therefore considering the expectation of  $C_{14}$  and substituting equations (6.42) and (6.43) yields

$$\begin{aligned} \mathbb{E} [C_{14}] &= \mathbb{E} [\mathbb{E} [C_{14} | \tilde{S}_U^S, T_U^S, R]] \\ &= \mathbb{E} [\mathbb{E} [R \{ \tilde{p}_{SU} \mathbb{E} [\angle_S^F] + (1 - \tilde{p}_{SU}) \mathbb{E} [\angle_N^F] \} | \tilde{S}_U^S, T_U^S, R]] \\ &= \mathbb{E} [\mathbb{E} [T_U^S p_{RW} \{ \tilde{p}_{SU} \mathbb{E} [\angle_S^F] + (1 - \tilde{p}_{SU}) \mathbb{E} [\angle_N^F] \} | \tilde{S}_U^S, T_U^S]] \\ &= \mathbb{E} [T_U^S] p_{RW} \{ \tilde{p}_{SU} \mathbb{E} [\angle_S^F] + (1 - \tilde{p}_{SU}) \mathbb{E} [\angle_N^F] \}. \end{aligned} \quad (6.44)$$

Since  $m_{1j} = \mathbb{E} [C_{1j}]$ ,  $j = 1, 2, 3, 4$ ,  $m_{11}$ ,  $m_{12}$ ,  $m_{13}$  and  $m_{14}$  are given in equations (6.35), (6.39), (6.40) and (6.44) respectively.

The calculations of  $m_{ij}$ ,  $i = 2, 3, 4$ ,  $j = 1, 2, 3, 4$ , follow analogous arguments to the calculations of  $m_{1j}$ . The calculation of  $m_{2j}$  differs to the calculation of  $m_{1j}$  in that a type-2 individual is always sampled and has joint stub and triangle distribution  $(S_U^T, \tilde{T}_U^T)$ . The calculation of  $m_{3j}$  differs to the calculation of  $m_{1j}$  in that a type-3 individual is always unsampled and has joint stub and triangle distribution  $(S_U^T, \tilde{T}_U^T)$ . Finally, the calculation of  $m_{4j}$  differs to the calculation of  $m_{1j}$  in that a type-4 individual has joint stub and triangle distribution  $(S_U^R, \tilde{T}_U^R)$ .  $\square$

### 6.3.4 Final size of a major outbreak

The backwards branching process approximating the spread of an individual's susceptibility set is constructed using similar typing to the forward branching process described in Section 6.3.1. First consider the number of stub neighbours that would infect an individual,  $i^*$  say, should they become infected, which

correspond to the type-1 individuals in the first generation of the backwards branching process. Then consider the number of sampled or unsampled individuals that would infect  $i^*$ , were they to become infected, spreading only through the triangles containing  $i^*$ , and each such sampled or unsampled individual corresponds respectively to a type-2 or type-3 individual in the first generation of the backwards branching process. Finally, consider the number of individuals that would infect  $i^*$ , were they to become infected, spreading only through the rewired households containing  $i^*$ , in which each such individual corresponds to a type-4 individual in the first generation of the backwards branching process. We then repeat the process to obtain the second generation of the branching process and so on. Note that the offspring of the initial individual (chosen uniformly at random from the population) will be different to the offspring of subsequent generations. We call  $i^*$  the primary individual and any individuals in the triangles or rewired triangles containing  $i^*$  are called secondary individuals.

Recall from Section 6.1 that we hypothesize that if a major outbreak does occur then its relative final size converges in distribution to the survival probability of the above backwards branching process. Let  $\tilde{\mathbf{B}}_i = (\tilde{B}_{i1}, \tilde{B}_{i2}, \tilde{B}_{i3}, \tilde{B}_{i4})$ ,  $i = 1, 2, 3, 4$ , be the offspring random vector of a typical type- $i$  individual in a non-initial generation of the backwards branching process. Similarly, let  $\mathbf{B} = (B_1, B_2, B_3, B_4)$  be the offspring random vector in the initial generation of the backwards branching process. Let  $b_{\mathbf{B}}(\mathbf{s})$ ,  $\mathbf{s} = (s_1, s_2, s_3, s_4)$  be the joint probability generating function of  $\mathbf{B}$  and let  $\mathbf{b}_{\tilde{\mathbf{B}}}(\mathbf{s}) = (b_{\tilde{\mathbf{B}}_1}(\mathbf{s}), b_{\tilde{\mathbf{B}}_2}(\mathbf{s}), b_{\tilde{\mathbf{B}}_3}(\mathbf{s}), b_{\tilde{\mathbf{B}}_4}(\mathbf{s}))$ .

Then the final size of a major outbreak is  $z = 1 - b_{\mathbf{B}}(\boldsymbol{\pi})$ , where  $\boldsymbol{\pi} = (\pi_1, \pi_2, \pi_3, \pi_4)$  is the smallest solution to the set of simultaneous equations  $\boldsymbol{\pi} = \mathbf{b}_{\tilde{\mathbf{B}}}(\boldsymbol{\pi})$ .

**Theorem 6.4.** *The joint probability generating functions for the offspring distributions of the backward Galton-Watson branching process are given by*

$$\begin{aligned} b_{\mathbf{B}}(\mathbf{s}) = & f_{S_U, T_U}(1 - p_N(1 - p_S p_C)(1 - \tilde{q}_V)(1 - s_1), \\ & p_S [(1 - p_{RW})f_{\Delta S}(s_2, s_3) + p_{RW}b_{\angle S}(s_4)] \\ & + (1 - p_S) [(1 - p_{RW})b_{\Delta N}(s_2, s_3) + p_{RW}b_{\angle N}(s_4)]), \end{aligned}$$

and

$$\begin{aligned}
b_{\tilde{\mathbf{B}}_1}(\mathbf{s}) &= f_{\tilde{S}_U^S-1, T_U^S} (1 - p_N(1 - \tilde{p}_{SU}p_C)(1 - \tilde{q}_V)(1 - s_1), \\
&\quad \tilde{p}_{SU} [(1 - p_{RW})b_{\Delta S}(s_2, s_3) + p_{RW}b_{\angle S}(s_4)] \\
&\quad + (1 - \tilde{p}_{SU}) [(1 - p_{RW})b_{\Delta N}(s_2, s_3) + p_{RW}b_{\angle N}(s_4)]), \\
b_{\tilde{\mathbf{B}}_2}(\mathbf{s}) &= f_{S_U^T, \tilde{T}_U^T-1} (1 - p_N(1 - p_C)(1 - \tilde{q}_V)(1 - s_1), \\
&\quad (1 - p_{RW})b_{\Delta S}(s_2, s_3) + p_{RW}b_{\angle S}(s_4)), \\
b_{\tilde{\mathbf{B}}_3}(\mathbf{s}) &= f_{S_U^T, \tilde{T}_U^T-1} (1 - p_N(1 - \tilde{q}_V)(1 - s_1), \\
&\quad (1 - p_{RW})b_{\Delta N}(s_2, s_3) + p_{RW}b_{\angle N}(s_4)), \\
b_{\tilde{\mathbf{B}}_4}(\mathbf{s}) &= f_{S_U^R, \tilde{T}_U^R-1} (1 - p_N(1 - \tilde{p}_{SU}p_C)(1 - \tilde{q}_V)(1 - s_1), \\
&\quad \tilde{p}_{SU} [(1 - p_{RW})b_{\Delta S}(s_2, s_3) + p_{RW}b_{\angle S}(s_4)] , \\
&\quad (1 - \tilde{p}_{SU}) [(1 - p_{RW})b_{\Delta N}(s_2, s_3) + p_{RW}b_{\angle N}(s_4)]),
\end{aligned}$$

where the probability generating functions  $b_{\Delta S}(s_2, s_3)$ ,  $b_{\Delta N}(s_2, s_3)$ ,  $b_{\angle S}(s_4)$  and  $b_{\angle N}(s_4)$  are given in equations (6.32).

*Proof.* We first consider the calculation of  $b_{\mathbf{B}}(\mathbf{s})$ . Note that an individual chosen uniformly at random from the population has joint stub and triangle distribution  $(S_U, T_U)$  and is sampled with probability  $p_S$  and otherwise unsampled. Recall that we assume a constant infectious period and that, in the limit  $N \rightarrow \infty$ , all infectious contacts made to  $i^*$  via a stub originate from individuals in edge-disjoint triangles and rewired triangles which do not contain  $i^*$ . Thus the number of contacts made to  $i^*$  via stubs is independent of the number of contacts made to  $i^*$  via triangles or rewired triangles so  $B_1|S_U, T_U$  is independent of  $B_k|S_U, T_U$ ,  $k = 2, 3, 4$ . Thus, conditioning on whether the primary individual is sampled or unsampled,

$$\begin{aligned}
\mathbb{E}[\mathbf{s}^{\mathbf{B}}] &= \mathbb{E} \left[ p_S \mathbb{E}[\mathbf{s}^{B_i} | S_U, T_U, I_S] + (1 - p_S) \mathbb{E}[\mathbf{s}^{B_i} | S_U, T_U, I_S^C] \right] \\
&= \mathbb{E} \left[ p_S \mathbb{E}[s_1^{B_1} | S_U, T_U, I_S] \mathbb{E} \left[ \prod_{k=2}^4 s_k^{B_k} | S_U, T_U, I_S \right] \right. \\
&\quad \left. + (1 - p_S) \mathbb{E}[s_1^{B_1} | S_U, T_U, I_S^C] \mathbb{E} \left[ \prod_{k=2}^4 s_k^{B_k} | S_U, T_U, I_S^C \right] \right]. \quad (6.46)
\end{aligned}$$

We now consider the calculation of  $\mathbb{E}[s_1^{B_1} | S_U, T_U, I_S]$  and  $\mathbb{E}[s_1^{B_1} | S_U, T_U, I_S^C]$ . A given network neighbour of  $i^*$ ,  $j$  say, can only make infectious contact with  $i^*$  if  $j$ : is not chosen for vaccination by  $i^*$ , is not already vaccinated by another neighbour and contacts  $i^*$ . So if  $i^*$  is sampled or unsampled then  $j$  contacts  $i^*$

with probability  $(1 - p_C)(1 - \tilde{q}_V)p_N$  or  $(1 - \tilde{q}_V)p_N$  respectively. Therefore

$$B_1|S_U, T_U, I_S \sim \text{Bin}(S_U, p_N(1 - p_C)(1 - \tilde{q}_V)). \quad (6.47a)$$

$$B_1|S_U, T_U, I_S^C \sim \text{Bin}(S_U, p_N(1 - \tilde{q}_V)). \quad (6.47b)$$

Next we consider the calculation of  $E[\prod_{k=2}^4 s_k^{B_k} | S_U, T_U, I_S]$  by conditioning on  $i^*$  being part of  $R \in [0; T_U]$  rewired triangles and decomposing the vector  $(B_2, B_3, B_4) | S_U, T_U, I_S, R$  into the number of contacts made to  $i^*$  through each triangle or rewired triangle separately. So

$$\begin{aligned} (B_2, B_3, B_4) | S_U, T_U, I_S, R &= \sum_{r=1}^{T_U-R} (B_2^T(r), B_3^T(r), B_4^T(r)) \\ &+ \sum_{r=T_U-R+1}^{T_U} (B_2^R(r), B_3^R(r), B_4^R(r)), \end{aligned} \quad (6.48)$$

where: we have labelled the  $T_U - R$  triangles containing  $i^*$   $1, 2, \dots, T_U - R$ , we have labelled the  $R$  rewired triangles containing  $i^*$   $T_U - R + 1, T_U - R + 2, \dots, T_U$ ,  $(B_2^T(r), B_3^T(r), B_4^T(r))$  is the number of (type-2, type-3, type-4) offspring in triangle  $r$  and  $(B_2^R(r), B_3^R(r), B_4^R(r))$  is the number of (type-2, type-3, type-4) offspring in rewired triangle  $r$ . Recall that we assume that all triangles and rewired triangles containing  $i^*$  are edge-disjoint so all the summands in equation (6.48) are mutually independent and also independent of  $S_U, T_U$  and  $R$ . Furthermore, the spread of  $i^*$ 's susceptibility set through a triangle cannot result in type-4 offspring, so  $B_4^T(r_1) = 0$  for  $r_1 \in [1; T_U - R]$ , and the spread of  $i^*$ 's susceptibility set through a rewired triangle cannot result in type-2 or type-3 offspring, so  $B_2^R(r_3) = B_3^R(r_3) = 0$  for  $r_3 \in [T_U - R + 1; T_U]$ .

Recall that the size of a triangle and rewired triangle susceptibility set is calculated in Section 6.3.2 so, for  $r_1 \in [1; T_U - R]$ ,

$$(B_2^T(r_1), B_3^T(r_1), B_4^T(r_1)) \stackrel{\mathcal{D}}{=} (\Delta_{SS}^B, \Delta_{SN}^B, 0), \quad (6.49a)$$

and, for  $r_3 \in [T_U - R + 1; T_U]$ ,

$$(B_2^R(r_3), B_3^R(r_3), B_4^R(r_3)) \stackrel{\mathcal{D}}{=} (0, 0, \angle_S^B), \quad (6.49b)$$

where the probability generating functions for  $(\Delta_{SS}^B, \Delta_{SN}^B)$  and  $\angle_S^B$  are given in equations (6.32).



Applying the decomposition of equation (6.48) to  $E \left[ \prod_{k=2}^4 s_k^{B_k} \middle| S_U, T_U, I_S, R \right]$  and using equations (6.49) yields

$$\begin{aligned} E \left[ \prod_{k=2}^4 s_k^{B_k} \middle| S_U, T_U, I_S, R \right] &= E \left[ \prod_{k=2}^4 s_k^{\sum_{r=1}^{T_U-R} B_k^T(r) + \sum_{r=T_U-R+1}^{T_U} B_k^R(r)} \middle| S_U, T_U, I_S, R \right] \\ &= E \left[ (f_{\Delta S}(s_2, s_3))^{T_U-R} (f_{\angle S}(s_4))^R \middle| S_U, T_U, I_S, R \right]. \end{aligned} \quad (6.50)$$

Each triangle in the network is independently rewired with probability  $p_{RW}$  so  $R|S_U, T_U \sim \text{Bin}(T_U, p_{RW})$  which, applied to equation (6.50), yields

$$\begin{aligned} E \left[ \prod_{k=2}^4 s_k^{B_k} \middle| S_U, T_U, I_S \right] &= E \left[ E \left[ \prod_{k=2}^4 s_k^{B_k} \middle| S_U, T_U, I_S, R \right] \right] \\ &= ((1 - p_{RW})f_{\Delta S}(s_2, s_3) + p_{RW}f_{\angle S}(s_4))^{T_U}. \end{aligned} \quad (6.51)$$

Considering  $E \left[ \prod_{k=2}^4 s_k^{B_k} \middle| S_U, T_U, I_S^C \right]$  and applying analogous arguments to those leading to equations (6.51) yields

$$E \left[ \prod_{k=2}^4 s_k^{B_k} \middle| S_U, T_U, I_S^C \right] = ((1 - p_{RW})f_{\Delta N}(s_2, s_3) + p_{RW}f_{\angle N}(s_4))^{T_U}. \quad (6.52)$$

Substituting equations (6.51) and (6.52) into equation (6.46) and applying equations (6.47) yields  $b_{\mathbf{B}}(\mathbf{s})$  in the statement of the theorem.

Finally, we consider the calculation of  $b_{\tilde{\mathbf{B}}_k}(\mathbf{s})$ ,  $k = 1, 2, 3, 4$ . The calculation of  $b_{\tilde{\mathbf{B}}_k}(\mathbf{s})$  follow analogous arguments to the calculation of  $b_{\mathbf{B}}(\mathbf{s})$  and requires only a substitution of the appropriate joint stub and triangle distribution (with an adjustment to account for the individuals parent) and the probability that the primary infective is sampled. To calculate  $b_{\tilde{\mathbf{B}}_1}(\mathbf{s})$  or  $b_{\tilde{\mathbf{B}}_4}(\mathbf{s})$  we note that the primary individual has joint stub and triangle distribution  $(\tilde{S}_U^S, T_U^S)$  or  $(S_U^R, \tilde{T}_U^R)$  respectively, and in both cases is sampled with probability  $\tilde{p}_{SU}$ . To calculate  $b_{\tilde{\mathbf{B}}_2}(\mathbf{s})$  or  $b_{\tilde{\mathbf{B}}_3}(\mathbf{s})$  we note that the primary individual is sampled with probability 1 or 0 respectively, and has joint stub and triangle distribution  $(S_U^T, \tilde{T}_U^T)$ .  $\square$

## 6.4 Optimal vaccination on the rewired edge-triangle model

### 6.4.1 Description of optimal vaccination

We now consider the ‘optimal’ vaccination strategy in the rewired edge-triangle model. Under the optimal vaccination strategy we vaccinate individuals with a larger total degree than a cut-off value, the cut-off being determined by the desired vaccination coverage.

Recall that the total degree distribution is given by  $D_T$  and  $P(D_T = k) = p_k$ ,  $k = 0, 1, \dots$ . Then, given the desired vaccination coverage,  $c$ , let  $d_c$  be the smallest total degree of an individual which we vaccinate. Therefore  $d_c = \max \{n \in \mathbb{Z}^+ : \sum_{k=0}^{n-1} p_k < 1 - c\}$ . We vaccinate no individuals of total degree  $d_c - 1$  or lower and all individuals of total degree  $d_c + 1$  or higher. We then vaccinate some proportion  $\delta \in (0, 1]$  of individuals of total degree  $d_c$ , which are chosen uniformly at random. Clearly we require  $c = \sum_{k=d_c+1}^{\infty} p_k + \delta p_{d_c}$ , so  $\delta = (c - \sum_{k=d_c+1}^{\infty} p_k) / p_{d_c}$ . Note that by choosing individuals with total degree  $d_c$  uniformly at random for vaccination we make no distinction between individuals with a larger stub or triangle degrees. Therefore the optimal vaccination strategy we consider is unlikely to be the true optimal vaccination strategy, which should incorporate the difference in stub and triangle degrees (and the difference in their effect on the spread of the epidemic), however it is likely to be a very good vaccination strategy since it targets individuals with large total degree using global knowledge of the network.

Before considering a threshold parameter and the final size of a major outbreak we calculate the joint stub and triangle distributions of an unvaccinated individual chosen uniformly at random from the population, an unvaccinated individual contacted via a stub and an unvaccinated individual contacted via a triangle or rewired triangle.

Let  $(S_O, T_O)$  be the joint stub and triangle distribution of an unvaccinated individual chosen uniformly at random from the population. An individual chosen uniformly at random from the population has unconditional joint stub and triangle distribution  $(S, T)$ , however we also know that this individual is unvaccinated. So, for  $s, t = 0, 1, \dots$  such that  $s + 2t < d_c - 1$ ,  $P(S_O = s, T_O = t) = p_{st}(1 - c)^{-1}$

and, for  $s, t$  such that  $s + 2t = d_c$ ,  $P(S_O = s, T_O = t) = (1 - \delta)p_{st}(1 - c)^{-1}$ .

Let  $(\tilde{S}_O^S, T_O^S)$  be the joint stub and triangle distribution of an unvaccinated individual contacted via a stub. Recalling that an individual contacted via a stub will have unconditional joint stub and triangle distribution  $(\tilde{S}^S, T^S)$ , for  $s = 1, 2, \dots, t = 0, 1, \dots$  such that  $s + 2t < d_c - 1$

$$P(\tilde{S}_O^S = s, T_O^S = t) = q_{st}(1 - \tilde{p}_V^S)^{-1},$$

and, for  $s, t$  such that  $s + 2t = d_c$

$$P(\tilde{S}_O^S = s, T_O^S = t) = (1 - \delta)q_{st}(1 - \tilde{p}_V^S)^{-1},$$

where  $\tilde{p}_V^S = \delta \sum_{s+2t=d_c} q_{st} + \sum_{s+2t>d_c} q_{st}$  is the a priori probability that an individual contacted via a stub is vaccinated.

Similarly let  $(S_O^T, \tilde{T}_O^T)$  be the joint stub and triangle distribution of an unvaccinated individual contacted via a triangle or rewired triangle. Recalling that an individual contacted via a triangle will have unconditional joint stub and triangle distribution  $(S^T, \tilde{T}^T)$ , for  $s = 0, 1, \dots, t = 1, 2, \dots$ , such that  $s + 2t < d_c - 1$ ,

$$P(S_O^T = s, \tilde{T}_O^T = t) = r_{st}(1 - \tilde{p}_V^T)^{-1},$$

and, for  $s, t$  such that  $s + 2t = d_c$ ,

$$P(S_O^T = s, \tilde{T}_O^T = t) = (1 - \delta)r_{st}(1 - \tilde{p}_V^T)^{-1},$$

where  $\tilde{p}_V^T = \delta \sum_{s+2t=d_c} r_{st} + \sum_{s+2t>d_c} r_{st}$  is the a priori probability that an individual contacted via a triangle or rewired triangle is vaccinated.

### 6.4.2 Threshold parameter

Similarly to the calculations for the uniform vaccination strategy discussed in Section 6.2.1, we consider a two-type branching process to calculate a threshold parameter. In the forward branching process, used to calculate the threshold parameter  $R_T^O$ , individuals are typed by whether they were infected via a stub (type-1) or via the spread of the epidemic through the triangles and rewired triangles containing the primary infective (type-2). The reproduction number  $R_T^O$  is the largest eigenvalue of the mean next-generation matrix  $M^O$  given below

in Theorem 6.5.

**Theorem 6.5.** *If  $p_N(1 - \tilde{p}_V^S) < 1$  then the mean next-generation matrix  $M^O$  is given by*

$$M^O = \begin{bmatrix} p_N(1 - \tilde{p}_V^S)E[\tilde{S}_O^S - 1] & 2E[T^S] \left[ (1 - p_{RW})\Delta_E^O + p_{RW} \frac{p_N(1 - \tilde{p}_V^T)}{1 - p_N(1 - \tilde{p}_V^T)} \right] \\ p_N(1 - \tilde{p}_V^S)E[S_O^T] & 2E[\tilde{T}^T] \left[ (1 - p_{RW})\Delta_E^O + p_{RW} \frac{p_N(1 - \tilde{p}_V^T)}{1 - p_N(1 - \tilde{p}_V^T)} \right] \end{bmatrix},$$

where

$$\Delta_E^O = p_N(1 - \tilde{p}_V^T) \left[ p_N(1 - \tilde{p}_V^T)(3 - 2p_N) + (1 - \tilde{p}_V^T)(1 - p_N)^2 + \tilde{p}_V^T \right].$$

We omit the proof of Theorem 6.5 since it proceeds analogously to the proofs of Theorems 6.1 and 6.3.

### 6.4.3 Final size of a major outbreak

Similarly to the calculations for the uniform vaccination strategy discussed in Section 6.2.2, we consider a two-type branching process to calculate the final size of a major outbreak. In the backwards branching process individuals are typed by whether they join an individual's susceptibility set via a stub (type-1) or via the spread of the epidemic through the triangles and rewired triangles containing the primary individual (type-2).

The final size of a major outbreak is  $z = 1 - f_B(\boldsymbol{\pi})$ , where  $\boldsymbol{\pi} = (\pi_1, \pi_2)$  is the smallest solution to the set of simultaneous equations  $\boldsymbol{\pi} = \mathbf{b}_{\tilde{B}}(\boldsymbol{\pi})$ .

**Theorem 6.6.** *The joint probability generating functions for the offspring distributions of the backward Galton-Watson branching process are given by*

$$\begin{aligned} b_{\tilde{B}_1}(\mathbf{s}) &= f_{\tilde{S}_O^S - 1, T_O^S} \left( 1 - p_N(1 - \tilde{p}_V^S)(1 - s_1), \right. \\ &\quad \left. (1 - p_{RW})b_{\Delta^O}(s_2) + p_{RW} \left( \frac{1 - p_N(1 - \tilde{p}_V^T)}{1 - s_2 p_N(1 - \tilde{p}_V^T)} \right)^2 \right), \\ b_{\tilde{B}_2}(\mathbf{s}) &= f_{S_O^T, \tilde{T}_O^T - 1} \left( 1 - p_N(1 - \tilde{p}_V^S)(1 - s_1), \right. \\ &\quad \left. (1 - p_{RW})b_{\Delta^O}(s_2) + p_{RW} \left( \frac{1 - p_N(1 - \tilde{p}_V^T)}{1 - s_2 p_N(1 - \tilde{p}_V^T)} \right)^2 \right), \end{aligned}$$

and

$$b_B(\mathbf{s}) = f_{S_O T_O} \left( 1 - p_N(1 - \tilde{p}_V^S)(1 - s_1), \right. \\ \left. (1 - p_{RW})b_{\Delta^O}(s_2) + p_{RW} \left( \frac{1 - p_N(1 - \tilde{p}_V^T)}{1 - s_2 p_N(1 - \tilde{p}_V^T)} \right)^2 \right),$$

where

$$b_{\Delta^O}(s_2) = (1 - \tilde{p}_V^T)^2 p_N^2 (3 - 2p_N) s_2^2 + 2(1 - \tilde{p}_V^T) p_N \left[ (1 - \tilde{p}_V^T)(1 - p_N)^2 + \tilde{p}_V^T \right] s_2 \\ + \left( \tilde{p}_V^T + (1 - \tilde{p}_V^T)(1 - p_N) \right)^2.$$

We omit the proof of Theorem 6.6 since it proceeds analogously to the proofs of Theorems 6.2 and 6.4.

## 6.5 Calculation of $R_0$ in the rewired edge-triangle model

Before calculating  $R_0$ , we define rank generations (see Section 3.2.3) of a single rewired triangle epidemic and then calculate the mean number of cases in each rank generation of a single triangle and rewired triangle epidemic.

### 6.5.1 Mean number of cases in each rank generation of a single triangle and rewired triangle epidemic

We define the rank generations of a single rewired triangle epidemic analogously to the definition of a single household epidemic given in Section 3.2.3. Therefore consider the spread of the epidemic within a single rewired triangle containing  $n$  individuals. We label the initial infective 0 and label the remaining susceptibles in the rewired triangle  $1, 2, \dots$ . Then construct a directed graph,  $\mathcal{G}_{RT}$ , with vertices labelled  $0, 1, \dots$ , in which for any ordered pair of distinct vertices  $(i, j)$ , there is a directed edge from  $i$  to  $j$  if and only if individual  $i$  would infect  $j$ , if  $i$  became infected. We say that individual 0 has a rank generation 0. Those individuals that are in 0's list are then said to have rank generation 1. Individuals not in individual 0's list, or individual 0, but who are in a rank generation 1 infective's list have rank generation 2 and so on. The set of people ultimately infected within a rewired triangle by the epidemic comprises those individuals

in  $\mathcal{G}_R$  that have a chain of directed edges leading to them from individual 0 and the rank generation number of such an infected individual,  $i$  say, is the length of the shortest chain joining 0 to  $i$ , where the length of a chain is the number of edges in it. The rank generation of a single triangle is identical to the rank generation of a single household of size 3, given in Section 3.2.3.

Denote by  $\mu_i^\Delta$ ,  $i = 1, 2, \dots$ , the mean number of infectives in rank generation  $i$  of a single triangle epidemic. Similarly, denote by  $\mu_i^\angle$ ,  $i = 1, 2, \dots$ , the mean number of infectives in rank generation  $i$  of a single rewired triangle epidemic. Clearly  $\mu_i^\Delta = 0$  for  $i > 2$  and

$$\mu_1^\Delta = 2p_N^2 + 2p_N(1 - p_N) = 2p_N, \quad (6.54a)$$

$$\mu_2^\Delta = 2p_N^2(1 - p_N). \quad (6.54b)$$

Since the graph created by a rewired triangle is locally tree-like when  $N$  is large (See the arguments leading to equation (6.11)) we can approximate the spread of the epidemic through a rewired triangle by the spread of the epidemic through a tree. The initial infective has 2 susceptible neighbours and any subsequent infective in the rewired triangle has 1 susceptible neighbour. Therefore, since an infected individual infects any given susceptible neighbour with probability  $p_N$ , for  $i = 1, 2, \dots$ , rank generation  $i$  of a single rewired triangle epidemic consists of 2 individuals with probability  $p_N^{2i}$  and a single individual with probability  $2p_N^i(1 - p_N^i)$ . Thus, for  $i = 1, 2, \dots$ ,

$$\mu_i^\angle = 2p_N^i. \quad (6.55)$$

### 6.5.2 Calculation of $R_0$

We now consider the calculation of  $R_0$  in the rewired edge-triangle model under no vaccination. Recall that we use the definition of  $R_0$  introduced by Pellis et al. (2012), which we discuss in Section 3.2.3 for the model with three levels of mixing. To calculate  $R_0$  we introduce a discrete-time two-type branching process, which we call the forward  $R_0$  branching process, which proceeds in the following way. First consider the number of stub network contacts made by the initial infective, which we call type-1 individuals in the branching process. Then consider the epidemic spreading in multiple generations through the triangles and rewired triangles containing the initial infective, in which each infected individual results in a type-2 offspring in the branching process. In the forward

$R_0$  branching process a time period corresponds to a new generation being infected and an individual in the forward  $R_0$  branching process corresponds to a single individual becoming infected in the epidemic process, although an individual will have type-2 offspring at multiple time points as the epidemic spreads through the triangles and rewired triangles. Similarly to the forward branching process discussed in Section 6.1, we conjecture that as  $N \rightarrow \infty$  the total number of infected individuals in the epidemic process converges in distribution to the total progeny of the forward  $R_0$  branching process.

Recall from the beginning of Section 3.2.3 that the global generation of an infective is its generation in the epidemic at large. So the initial infective in the epidemic,  $i^*$ , has global generation 0. Individuals that  $i^*$  would contact, through either stubs, triangles or rewired triangles, are then global generation 1. Similarly, individuals which are not members of a previous global generation that members of generation 1 would infect, through either stubs, triangles or rewired triangles are then generation 2 and so on. In the forward  $R_0$  branching process we say that an individual's time of birth is given by the global generation of the corresponding individual in the epidemic process. An individual in the forward  $R_0$  branching process may reproduce at ages  $1, 2, \dots$ . For a type- $i$ ,  $i = 1, 2$ , individual we denote the mean number of type- $j$ ,  $j = 1, 2$ , offspring at age  $i + 1$  by  $\nu_{ij}^{(i)}$ .

Then, similarly to Section 3.2.3,  $R_0$  is given by the asymptotic (Malthusian) geometric growth rate of the forward  $R_0$  branching process, which is the value of  $\lambda \in \mathbb{R}^+$  such that the maximal eigenvalue of  $V(\lambda)$  is 1, where  $V(\lambda)$  is given by

$$V(\lambda) = \sum_{i=1}^{\infty} \begin{bmatrix} \nu_{11}^{(i-1)}/\lambda^i & \nu_{12}^{(i-1)}/\lambda^i \\ \nu_{21}^{(i-1)}/\lambda^i & \nu_{22}^{(i-1)}/\lambda^i \end{bmatrix}.$$

**Theorem 6.7.** *If  $p_N < 1$  then, for  $\lambda > p_N$ ,*

$$V(\lambda) = \begin{bmatrix} v_{11}(\lambda) & v_{12}(\lambda) \\ v_{21}(\lambda) & v_{22}(\lambda) \end{bmatrix},$$

where

$$\begin{aligned} v_{11}(\lambda) &= \mathbb{E} [\tilde{S}^S - 1] \frac{p_N}{\lambda}, \\ v_{12}(\lambda) &= 2p_N \mathbb{E} [T^S] \left\{ (1 - p_{RW}) \left[ \frac{1}{\lambda} + \frac{p_N(1 - p_N)}{\lambda^2} \right] + p_{RW} \frac{1}{\lambda - p_N} \right\}, \\ v_{21}(\lambda) &= \mathbb{E} [S^T] \frac{p_N}{\lambda}, \\ v_{22}(\lambda) &= 2p_N \mathbb{E} [\tilde{T}^T - 1] \left\{ (1 - p_{RW}) \left[ \frac{1}{\lambda} + \frac{p_N(1 - p_N)}{\lambda^2} \right] + p_{RW} \frac{1}{\lambda - p_N} \right\}. \end{aligned}$$

*Proof.* We begin by calculating  $v_{11}(\lambda)$  and  $v_{21}(\lambda)$ . For  $j = 1, 2$ ,  $i = 1, 2, \dots$ , let  $C_{j1}(i)$  be the total number of contacts made via a stub of a single type- $j$  individual in rank generation  $i$ . Consider an individual,  $l$  say, and let  $\bar{S}$  be the number of uninfected stub neighbours of  $l$ . Conditioned on  $\bar{S}$ , individual  $l$  makes contact with each of its  $\bar{S}$  susceptible neighbours independently with probability  $p_N$ . Thus, since all contacts made via a stub occur when an individual is at age 1,  $C_{j1}(1)|\bar{S} \sim \text{Bin}(\bar{S}, p_N)$ ,  $C_{j1}(i) = 0$  for  $i = 2, 3, \dots$ , and

$$\mathbb{E} [C_{j1}(1)] = \mathbb{E} [\mathbb{E} [C_{j1}(1)|\bar{S}]] = \mathbb{E} [\bar{S}] p_N.$$

A type-1 or type-2 individual has  $\tilde{S}^S - 1$  or  $S^T$  uninfected stub neighbours respectively. So  $\nu_{j1}^{(1)} = \mathbb{E} [C_{j1}(1)]$  and

$$\begin{aligned} \nu_{11}^{(i)} &= \begin{cases} \mathbb{E} [\tilde{S}^S - 1] p_N & \text{if } i = 1, \\ 0 & \text{otherwise,} \end{cases} \\ \nu_{21}^{(i)} &= \begin{cases} \mathbb{E} [S^T] p_N & \text{if } i = 1, \\ 0 & \text{otherwise.} \end{cases} \end{aligned}$$

Furthermore, since  $v_{j1}(\lambda) = \sum_{i=1}^{\infty} \nu_{j1}^{(i-1)} / \lambda^i$ ,

$$v_{11}(\lambda) = \mathbb{E} [\tilde{S}^S - 1] \frac{p_N}{\lambda}, \tag{6.57a}$$

$$v_{21}(\lambda) = \mathbb{E} [S^T] \frac{p_N}{\lambda}. \tag{6.57b}$$



Let  $C_{j2}(i)$ ,  $j = 1, 2$ ,  $i = 1, 2, \dots$ , be the number of individuals infected via a triangle or rewired triangle of a single type- $j$  individual in rank generation  $i$ . Consider an individual,  $i^*$  say, and let  $\bar{T}$  be the total number of triangles and rewired triangles containing  $i^*$  and no other infected individuals. Similarly, let  $R$  be the number of rewired triangles containing  $i^*$  and no other infected individuals. Note that, since each triangle is rewired independently and uniformly at random with probability  $p_{RW}$ ,  $E[R|\bar{T}] = p_{RW}\bar{T}$ . Furthermore, note that  $E[C_{j2}(i)|\bar{T}, R] = (\bar{T} - R)\mu_i^\Delta + R\mu_i^\angle$  and, since all triangles and rewired triangles containing  $i^*$  are edge-disjoint,

$$\begin{aligned} E[C_{j2}(i)] &= E[E[C_{j2}(i)|\bar{T}, R]] \\ &= E[E[(\bar{T} - R)\mu_i^\Delta + R\mu_i^\angle|\bar{T}]] \\ &= E[\bar{T}] (1 - p_{RW})\mu_i^\Delta + E[\bar{T}] p_{RW}\mu_i^\angle. \end{aligned} \quad (6.58)$$

Note that  $\nu_{j2}^{(i-1)} = E[C_{j2}(i)]$  so, since  $p_N < 1$ , substituting equations (6.54), (6.55) and (6.58) into  $v_{j2}(\lambda) = \sum_{i=1}^{\infty} \nu_{j2}^{(i-1)} / \lambda^i$ ,  $j = 1, 2$  yields

$$\begin{aligned} v_{j2}(\lambda) &= \sum_{i=1}^{\infty} \frac{E[\bar{T}] ((1 - p_{RW})\mu_i^\Delta + p_{RW}\mu_i^\angle)}{\lambda^i} \\ &= E[\bar{T}] \left( (1 - p_{RW}) \sum_{i=1}^{\infty} \frac{\mu_i^\Delta}{\lambda^i} + p_{RW} \sum_{i=1}^{\infty} \frac{\mu_i^\angle}{\lambda^i} \right) \\ &= E[\bar{T}] \left( (1 - p_{RW}) \left( \frac{2p_N}{\lambda} + \frac{2p_N^2(1 - p_N)}{\lambda^2} \right) + p_{RW} \sum_{i=1}^{\infty} \frac{2p_N^i}{\lambda^i} \right) \\ &= E[\bar{T}] \left( (1 - p_{RW}) \left( \frac{2p_N}{\lambda} + \frac{2p_N^2(1 - p_N)}{\lambda^2} \right) + p_{RW} \frac{2p_N}{\lambda - p_N} \right). \end{aligned}$$

Recall that for a type-1 individual  $\bar{T} \stackrel{\mathcal{D}}{=} T^S$  and for a type-2 individual  $\bar{T} \stackrel{\mathcal{D}}{=} \tilde{T}^T - 1$ , so

$$v_{12}(\lambda) = E[T^S] \left( (1 - p_{RW}) \left( \frac{2p_N}{\lambda} + \frac{2p_N^2(1 - p_N)}{\lambda^2} \right) + p_{RW} \frac{2p_N}{\lambda - p_N} \right), \quad (6.59a)$$

$$v_{22}(\lambda) = E[\tilde{T}^T - 1] \left( (1 - p_{RW}) \left( \frac{2p_N}{\lambda} + \frac{2p_N^2(1 - p_N)}{\lambda^2} \right) + p_{RW} \frac{2p_N}{\lambda - p_N} \right). \quad (6.59b)$$

Equations (6.57) and (6.59) yield the entries of  $V(\lambda)$  given in the theorem as required.  $\square$

Note that if  $p_N < 1$  then every entry in  $V(\lambda)$ , given in Theorem 6.7, is finite

for  $\lambda > 0$  which yields the following remark.

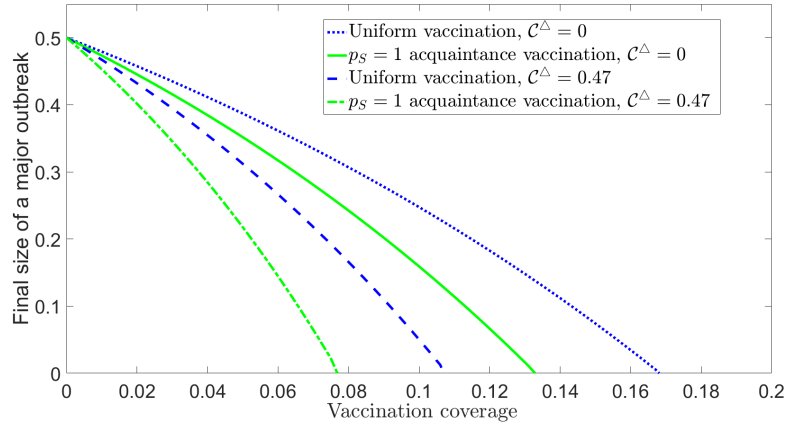
**Remark 6.2.** *If  $p_N < 1$  then  $R_0 < \infty$  in the rewired edge-triangle model.*

## 6.6 Numerical investigation of the rewired edge-triangle model

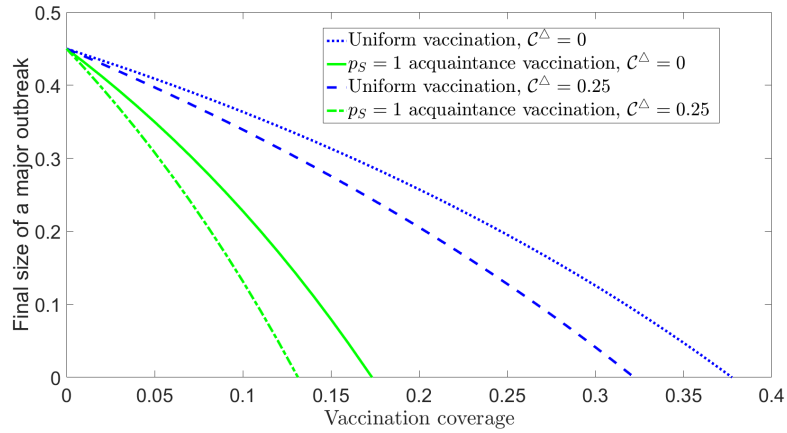
In this section we consider the rewired edge-triangle model as a clustered network with a fixed total degree distribution and, since changing the probability of rewiring does not affect the degree correlation, use the probability of rewiring to investigate the effect of clustering introduced via edge-disjoint triangles on the performance of vaccination strategies.

Note that two rewired edge-triangle models with the same stub and triangle distributions and infection rates but varying in  $p_{RW}$  will differ in both the final size of a major outbreak ( $z$ ) and basic reproduction number ( $R_0$ ). Therefore in the investigations discussed in this Section we compare models with the same stub and triangle distribution (and therefore total degree distribution) and differing in  $p_{RW}$  by choosing an infection rate  $\lambda_N$  to fix either  $z$  or  $R_0$  in the rewired edge-triangle model without any vaccination. We choose to fix  $z$  because in applications epidemic models are often parameterised by final size data (see Becker and Utev (1998)), and we find an investigation of models parameterised by  $R_0$  illuminating.

Similarly to previous investigations of the acquaintance vaccination strategies (see, for example, Section 5.1.4 or Ball and Sirl (2013)), our numerical work suggests that for a fixed vaccination coverage the threshold parameter and final size of a major outbreak are monotonically increasing in  $p_C$ . In other words, for a fixed vaccination coverage the effect of vaccination under the acquaintance vaccination strategy is greater if everyone in the population chooses a few individuals to be vaccinated rather than a few individuals choosing all their neighbours for vaccination. Therefore in our numerical investigations of the acquaintance vaccination strategy we focus on the extreme situations  $p_S = 1$  or  $p_C = 1$ , which we call the  $p_S = 1$  or  $p_C = 1$  acquaintance vaccination strategies respectively. Let  $c_U^*$ ,  $c_{p_S}^*$ ,  $c_{p_C}^*$  and  $c_O^*$  be the critical vaccination coverages under the uniform,  $p_S = 1$  acquaintance,  $p_C = 1$  acquaintance and optimal vaccination strategies respectively.



(a) Models matched with  $z = 0.5$  and  $f_{S,T} = 0.6t + 0.4t^2$ , so  $E[D_T] = 2.8$ .



(b) Models matched with  $z = 0.45$ , total degree distribution  $\text{Geo}^+(1/2)$  and triangle allocation distribution  $\text{Bin}(D_T, 0.75)$ , so  $E[D_T] = 2$ .

Figure 6.2: Figures illustrating that the  $p_S = 1$  acquaintance vaccination strategy outperforms the uniform vaccination strategy.

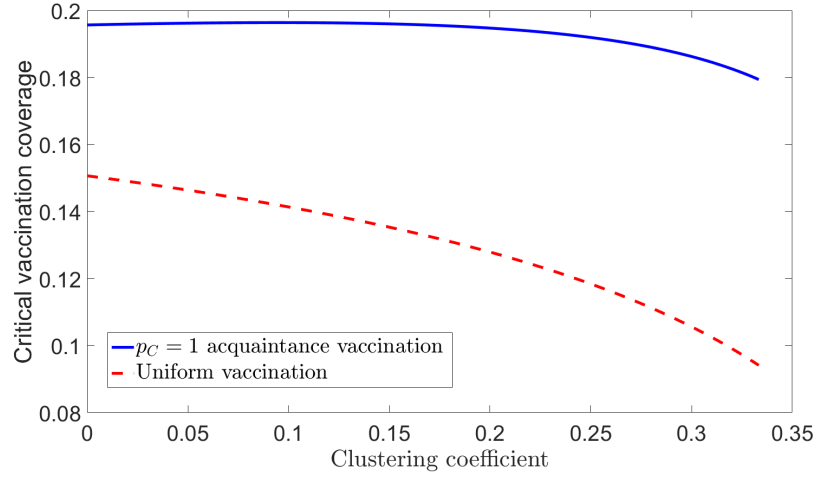
### 6.6.1 Comparing the acquaintance and uniform vaccination strategies

As we would intuitively expect, for a fixed stub and triangle distribution,  $p_{RW}$ , and vaccination coverage, the  $p_S = 1$  acquaintance vaccination strategy performs at least as well as the uniform vaccination strategy, with equality in performance occurring when the total degree distribution has zero variance (i.e.  $\text{Var}[D_T] = 0$ ) and an increasing difference between the two strategies as  $\text{Var}[D_T]$  increases, as illustrated in Figure 6.2. Furthermore, the difference between  $c_U^*$  and  $c_{p_S}^*$  remains mostly constant as  $p_{RW}$  varies.

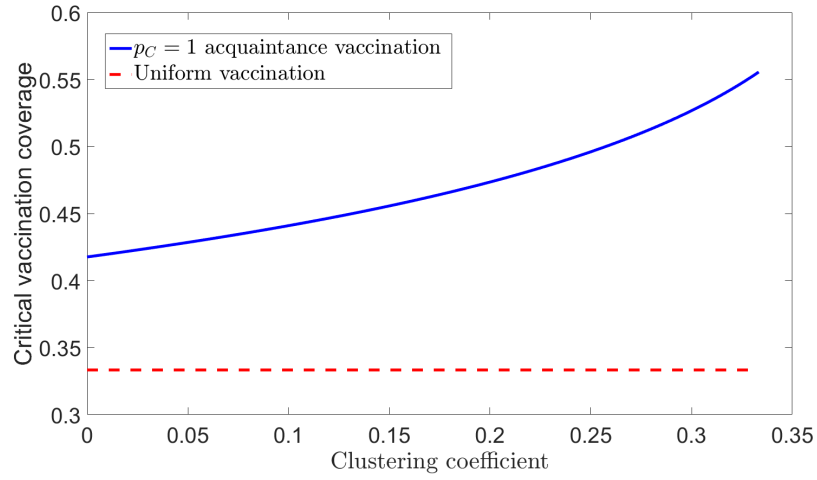
In contrast, if  $\text{Var}[D_T]$  is small then  $c_{p_C}^* > c_U^*$ , so the  $p_C = 1$  acquaintance

vaccination strategy can perform worse than the uniform vaccination strategy. Recall that in Section 5.4 we prove that in the network and global model the critical vaccination coverage under the  $p_C = 1$  acquaintance vaccination strategy is larger than the critical vaccination coverage under the uniform vaccination strategy if the network degree distribution has a small variance. Since setting  $P(T > 0) = 0$  in the rewired edge-triangle model recovers the standard network model (a submodel of the network and global model), the result  $c_{p_C}^* > c_U^*$  if  $\text{Var}[D_T]$  is small is unsurprising. However, an important result is that the addition of edge-disjoint triangle clustering when  $\text{Var}[D_T]$  is small and either  $z$  or  $R_0$  is fixed increases the difference between  $c_{p_C}^*$  and  $c_U^*$ , as illustrated in Figures 6.3a and 6.3b. Similarly to Section 5.4, we conjecture that the  $p_C = 1$  acquaintance vaccination strategy is clustering the vaccination among groups of individuals and thus, owing to the similarity of an individual's total degree, reducing the probability that the neighbour of an unvaccinated individual is also unvaccinated compared to the uniform vaccination. Furthermore, as the clustering coefficient is increased (by decreasing  $p_{RW}$ ), for a fixed vaccination coverage the  $p_C = 1$  acquaintance vaccination strategy will prevent infectious contacts from occurring along fewer edges in the network than the uniform vaccination strategy, leading to the increasing difference between  $c_{p_C}^*$  and  $c_U^*$  with the addition of edge-disjoint triangle clustering. To see this note that under the  $p_C = 1$  acquaintance vaccination strategy a sampled individual chooses on average  $E[S] + 2E[T]$  individuals for vaccination. If  $p_{RW} = 1$  (so the rewired edge-triangle model is unclustered) then these  $E[S] + 2E[T]$  individuals are edge-disjoint and vaccinating these individuals is expected to prevent infectious contacts from occurring along  $E[S] (E[\tilde{S}^S] + 2E[T^S]) + 2E[T] (E[S^T] + 2E[\tilde{T}^T])$  edges. However, if  $p_{RW} = 0$  then these  $E[S] + 2E[T]$  individuals are not edge-disjoint and vaccinating the same number of individuals will only prevent infectious contacts from occurring along  $E[S] (E[\tilde{S}^S] + 2E[T^S]) + 2E[T] (E[S^T] + 2E[\tilde{T}^T] - 1)$  edges.

Indeed, it is possible to find stub and triangle distributions with small  $\text{Var}[D_T]$  for which (fixing either  $z$  or  $R_0$ ) if  $p_{RW} = 0$  then  $c_{p_C}^* > c_U^*$ , whereas if  $p_{RW} = 1$  then  $c_{p_C}^* < c_U^*$ , as illustrated in Figures 6.4a and 6.4b. However, for larger  $\text{Var}[D_T]$  the  $p_C = 1$  acquaintance vaccination strategy outperforms the uniform vaccination strategy (fixing either  $z$  or  $R_0$ ), as illustrated in Figures 6.5a and 6.5b.

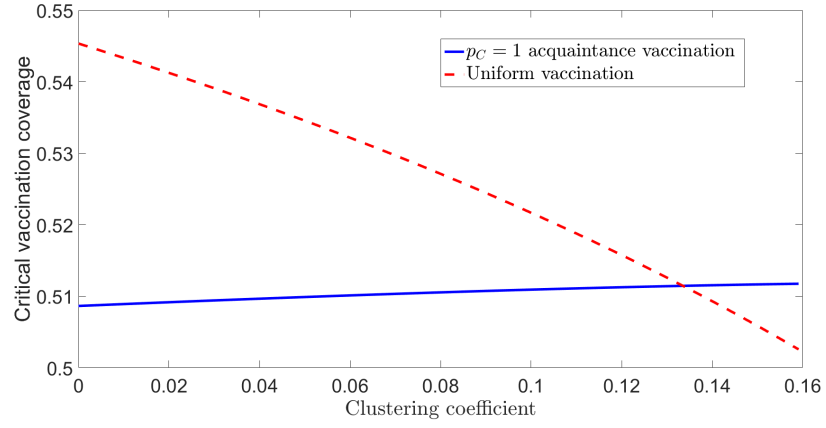


(a) Models matched with  $z = 0.5$ .

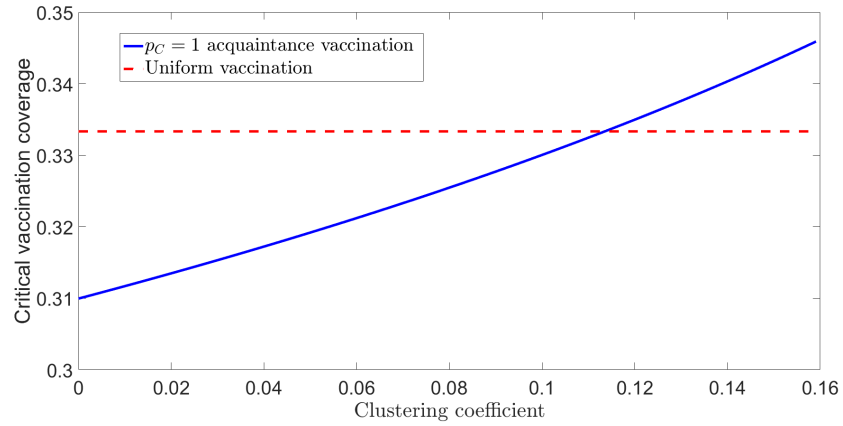


(b) Models matched with  $R_0 = 1.5$ .

Figure 6.3: The effect of clustering on the difference between the critical vaccination coverage of the  $p_C = 1$  acquaintance and uniform vaccination strategies with  $f_{S,T}(s, t) = t^2$ , so  $E[D_T] = 4$ .

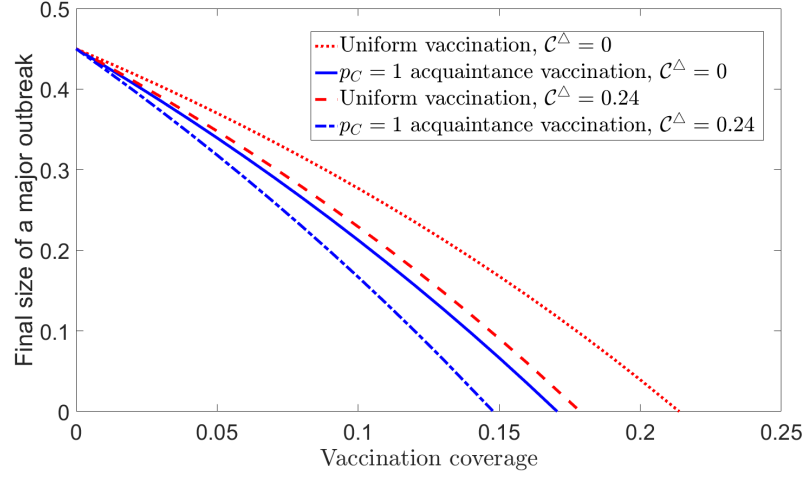


(a) Models matched with  $z = 0.9$ .

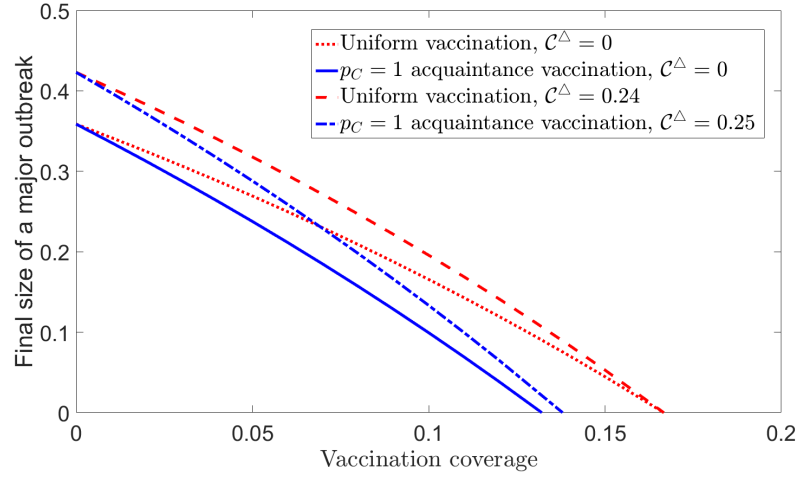


(b) Models matched with  $R_0 = 1.5$ .

Figure 6.4: The effect of clustering on the difference between the critical vaccination coverage of the  $p_C = 1$  acquaintance and uniform vaccination strategies with  $f_{S,T}(s, t) = \frac{1}{6}(s^2 + t + 2s^2t + s^4t + t^3)$ , as used by Miller (2009), so  $E[D_T] = 4$  and  $\text{Var}[D_T] = 2\frac{2}{3}$ .



(a) Models matched with  $z = 0.45$ .



(b) Models matched with  $R_0 = 1.2$ .

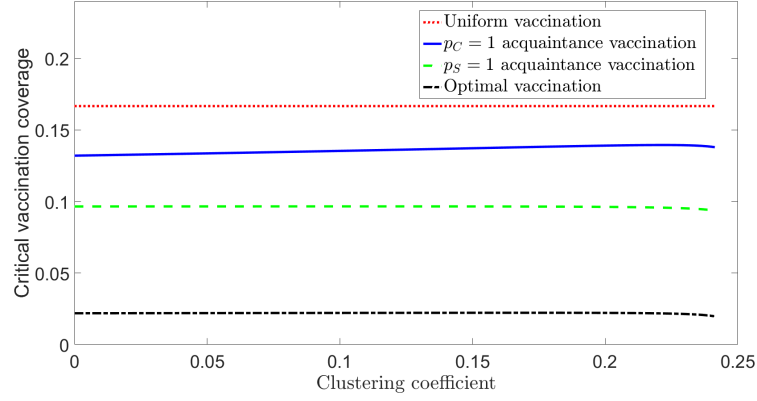
Figure 6.5: The effect of clustering on the difference between the critical vaccination coverage of the  $p_C = 1$  acquaintance and uniform vaccination strategies with  $D_T \sim \text{Poi}^+(1.59)$  and triangle allocation distribution  $\text{Bin}(D_T, 0.55)$ , so  $E[D_T] = 2$ .

### 6.6.2 The effect of clustering on the optimal, acquaintance and uniform vaccination strategies

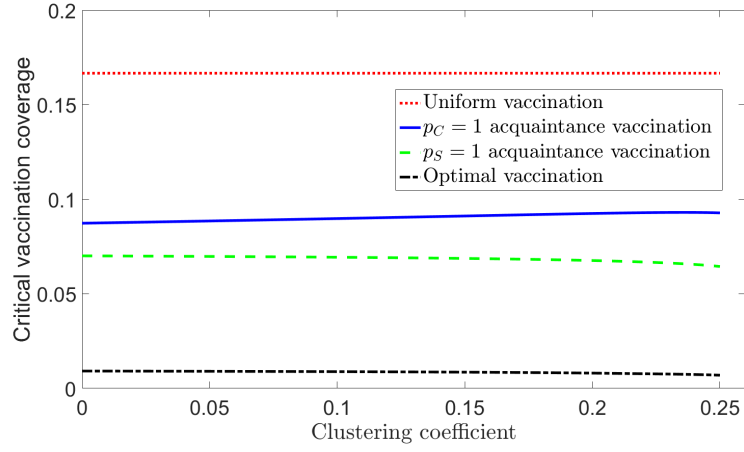
We find that fixing  $R_0$  and increasing the clustering coefficient (by increasing  $p_{RW}$  and adjusting  $\lambda_N$ ) has a negligible effect on  $c_U^*$ ,  $c_{ps}^*$  and  $c_O^*$ , as illustrated in Figures 6.6 and 6.7. Therefore we conjecture that (for a fixed joint stub and triangle distribution)  $c_U^*$ ,  $c_{ps}^*$  and  $c_O^*$  depend on  $R_0$  and not on the clustering coefficient. Recall that in Section 5.4 we prove that in the network and global model under the uniform vaccination strategy the critical vaccination coverage depends upon  $R_0$ , and indeed the critical vaccination coverage is equal to  $1 - 1/R_0$ . In general, the link between the critical vaccination coverage of the uniform vaccination strategy and  $R_0$  breaks down in networks incorporating household structure with households larger than 3, see Ball et al. (2016). However, in the rewired edge-triangle model we do not introduce households larger than 3 (although we do allow for an individual to be part of multiple households) so the result that  $c_U^* = 1 - 1/R_0$ , independent of the clustering coefficient, is an expected result. However, less expected is that for a fixed joint stub and triangle distribution both  $c_{ps}^*$  and  $c_O^*$  are determined by  $R_0$ , and also independent of the clustering coefficient.

Similarly, we find that fixing  $z$  and increasing the clustering coefficient (by increasing  $p_{RW}$  and adjusting  $\lambda_N$ ) has a negligible effect on  $c_U^*$ ,  $c_{ps}^*$  and  $c_O^*$  unless  $p_{RW}$  is close to 1 and  $E[D_T]$  is small, as illustrated in Figures 6.8a, 6.9a, 6.10a and 6.11. If  $E[D_T]$  is small, then as  $p_{RW}$  approaches 1 there can be a large decrease in  $c_U^*$ ,  $c_{ps}^*$  and  $c_O^*$ , as illustrated in Figures 6.8a and 6.9a, which we conjecture is explained by considering the corresponding change in  $R_0$ . Recall that Miller (2009) shows that fixing  $p_N$  and introducing edge-disjoint triangle clustering into the network will decrease the final size of a major outbreak. Therefore, to fix  $z$  as  $p_{RW}$  is increased we must also decrease  $p_N$ , and thus  $R_0$  is likely to be changed. Furthermore, if  $E[D_T]$  is small then as  $p_{RW}$  approaches 1 the basic reproduction number  $R_0$  is decreasing, as illustrated in Figures 6.8b and 6.9b, resulting in a lower critical vaccination coverage. We note that as  $E[D_T]$  is increased the change in  $R_0$  is increasingly decreasing, thus resulting in a smaller change in critical vaccination coverages, as illustrated in Figure 6.10b. Therefore we conjecture that the decrease in  $c_U^*$ ,  $c_{ps}^*$  and  $c_O^*$  when  $z$  is fixed,  $E[D_T]$  is small and  $p_{RW}$  approaches 1 is explained by the change in  $R_0$ .

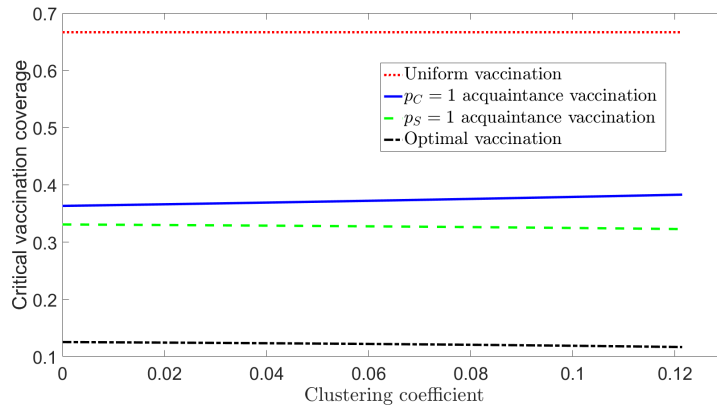




(a) Models matched with  $R_0 = 1.2$ ,  $D_T \sim \text{Poi}^+(1.59)$  and triangle allocation distribution  $\text{Bin}(D_T, 0.55)$ , so  $E[D_T] = 2$ .

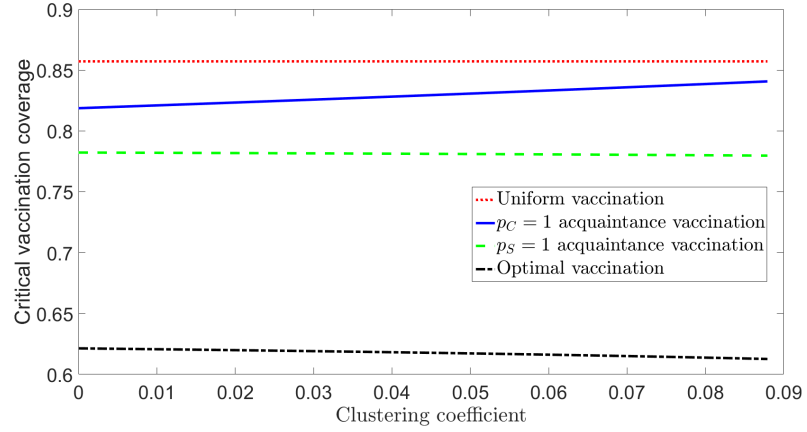


(b) Models matched with  $R_0 = 1.2$ ,  $D_T \sim \text{Geo}^+(1/2)$  and triangle allocation distribution  $\text{Bin}(D_T, 0.75)$ , so  $E[D_T] = 2$ .

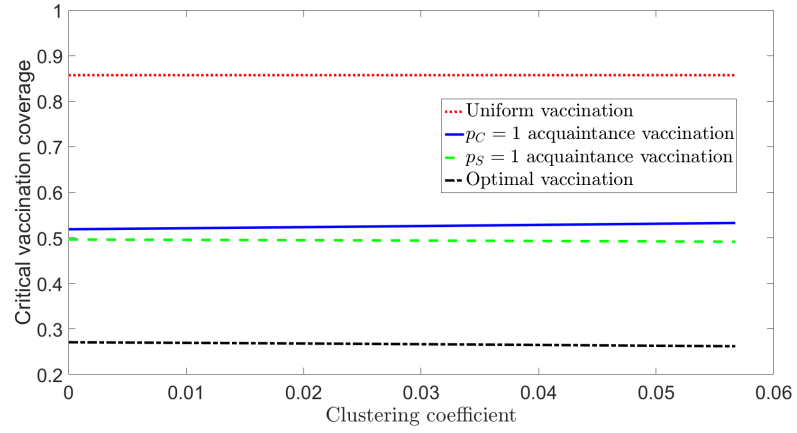


(c) Models matched with  $R_0 = 3$ ,  $D_T \sim \text{Geo}^+(1/4)$  and triangle allocation distribution  $\text{Bin}(D_T, 0.85)$ , so  $E[D_T] = 4$ .

Figure 6.6: The effect of clustering on the critical vaccination coverage under various joint stub and triangle distributions and vaccination strategies.

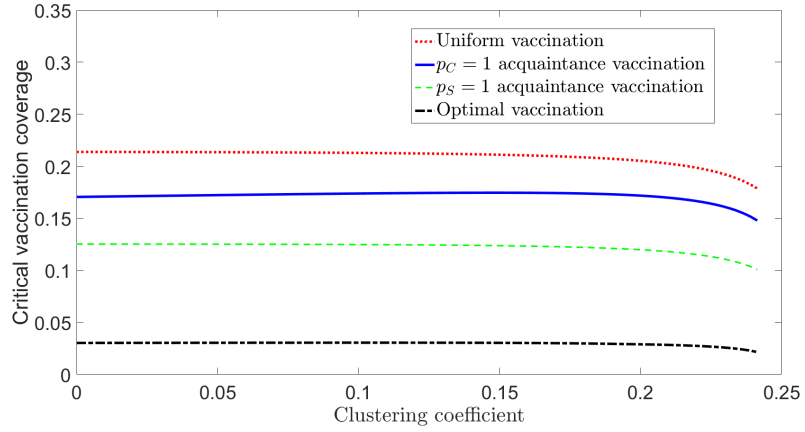


(a)  $D_T \sim \text{Poi}^+(8)$  and triangle allocation distribution  $\text{Bin}(D_T, 0.75)$ , so  $E[D_T] = 8$ .

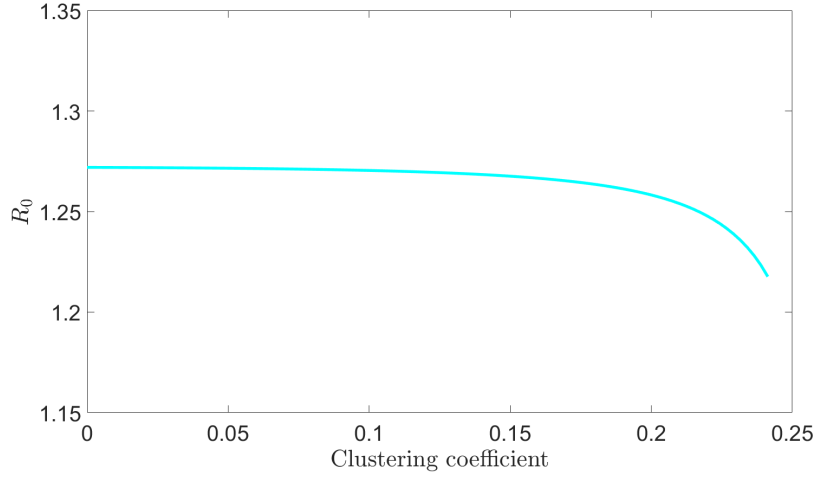


(b)  $D_T \sim \text{Geo}^+(1/8)$  and triangle allocation distribution  $\text{Bin}(D_T, 0.85)$ , so  $E[D_T] = 8$ .

Figure 6.7: The effect of clustering on the critical vaccination coverage under various vaccination strategies. Models matched with  $R_0 = 7$ .



(a) The effect of clustering on the critical vaccination coverage under various vaccination strategies.



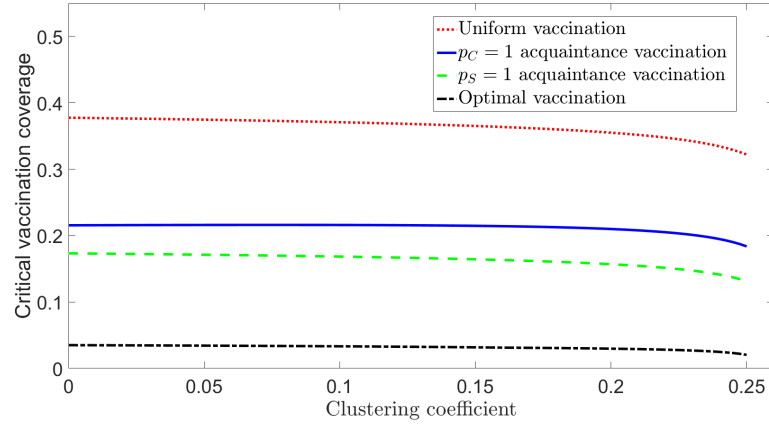
(b) The basic reproduction number  $R_0$  in the model without vaccination.

Figure 6.8: Models matched with  $z = 0.45$ ,  $D_T \sim \text{Poi}^+(1.59)$  and triangle allocation distribution  $\text{Bin}(D_T, 0.55)$ , so  $\mathbb{E}[D_T] = 2$ .

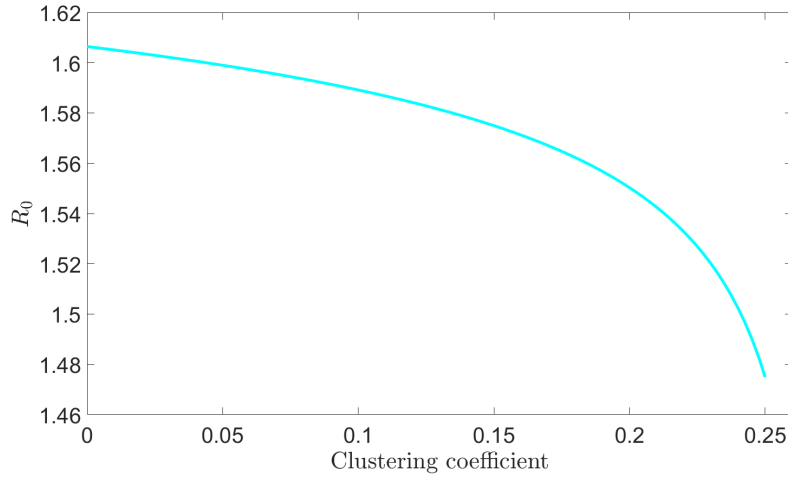
Finally, for a fixed joint stub and triangle distribution and either  $R_0$  or  $z$ , we note that  $c_{p_C}^*$  can either increase (as discussed in Section 6.6.1) or decrease, depending on our choice of  $(S, T)$ .

## 6.7 Concluding remarks

In this chapter we consider three vaccination strategies on the rewired edge-triangle model, specifically the optimal, acquaintance and uniform vaccination strategies. For each vaccination strategy we show how to find a threshold parameter, which determines whether a major outbreak can occur, and the expected

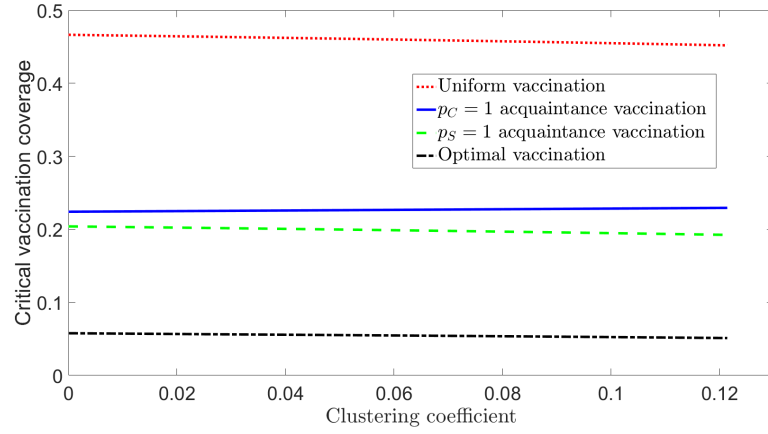


(a) The effect of clustering on the critical vaccination coverage under various vaccination strategies.

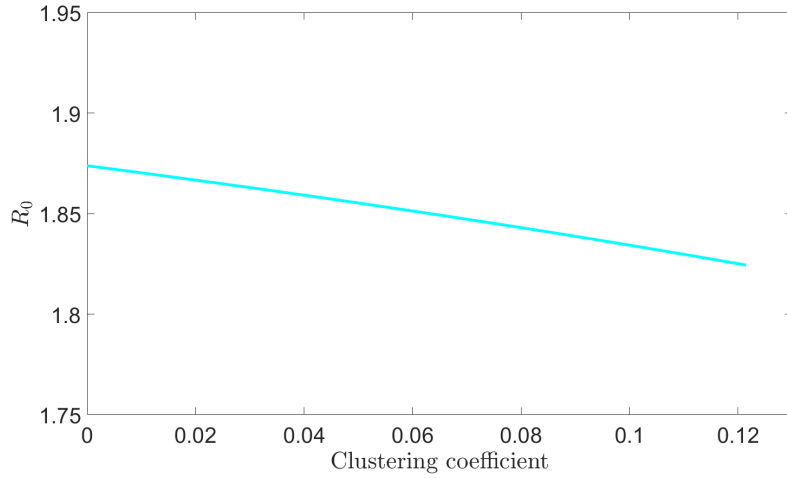


(b) The basic reproduction number  $R_0$  in the model without vaccination.

Figure 6.9: Models matched with  $z = 0.45$ ,  $D_T \sim \text{Geo}^+(1/2)$  and triangle allocation distribution  $\text{Bin}(D_T, 0.75)$ , so  $\text{E}[D_T] = 2$ .

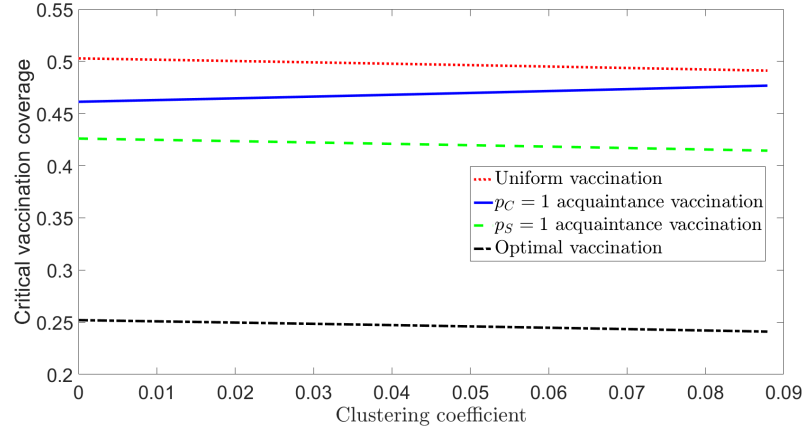


(a) The effect of clustering on the critical vaccination coverage under various vaccination strategies.

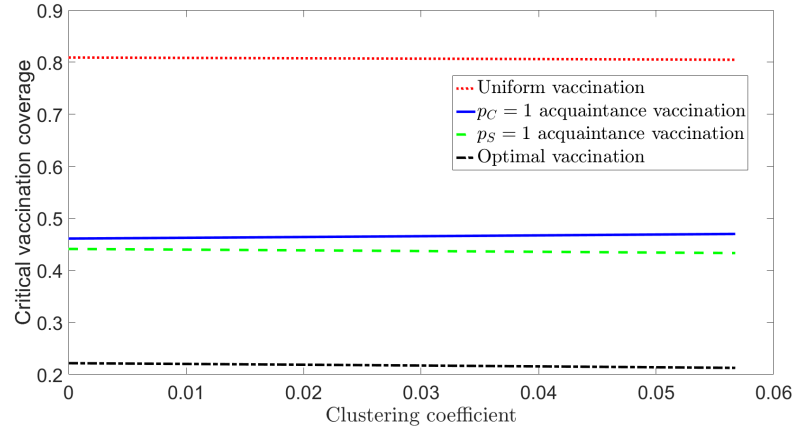


(b) The basic reproduction number  $R_0$  in the model without vaccination.

Figure 6.10: Models matched with  $z = 0.45$ ,  $D_T \sim \text{Geo}^+(1/4)$  and triangle allocation distribution  $\text{Bin}(D_T, 0.85)$ , so  $\text{E}[D_T] = 4$ .



(a)  $D_T \sim \text{Poi}^+(8)$  and triangle allocation distribution  $\text{Bin}(D_T, 0.75)$ , so  $E[D_T] = 8$ .



(b)  $D_T \sim \text{Geo}^+(1/8)$  and triangle allocation distribution  $\text{Bin}(D_T, 0.85)$ , so  $E[D_T] = 8$ .

Figure 6.11: The effect of clustering on the critical vaccination coverage under various vaccination strategies. Models matched with  $z = 0.8$ .

relative final size of a major outbreak. We find that the rewired edge-triangle model cannot construct clustered networks with a large mean total degree, owing to the model only containing edge-disjoint triangles.

Similarly to Section 5.1.4, for a fixed vaccination coverage we find that maximising  $p_C$  maximises the post-vaccination threshold parameter, the final size of a major outbreak and the critical vaccination coverage under the acquaintance vaccination strategy in the rewired edge-triangle model. Furthermore, similarly to Section 5.4 we find that if the variance of the total degree distribution is small then the critical vaccination coverage under the  $p_C = 1$  acquaintance vaccination strategy is larger than the critical vaccination coverage under the uniform vaccination strategy. However, as the variance of the total degree distribution increases then, for a fixed clustering coefficient, the critical vaccination coverage under the uniform vaccination coverage is larger than the critical vaccination coverage under the  $p_C = 1$  acquaintance vaccination strategy, which is larger than the critical vaccination coverage under the  $p_S = 1$  acquaintance vaccination strategy, which is then larger than the critical vaccination coverage under the optimal vaccination strategy, as we expect.

Using the rewiring process to investigate the effect of clustering on the critical vaccination coverage of the vaccination strategies (fixing either  $z$  or  $R_0$ ), we find that if the total degree distribution has a small variance then the difference in critical vaccination coverage between the uniform and  $p_C = 1$  acquaintance vaccination strategies increases as the clustering coefficient increases. We conjecture that the decreased performance of the  $p_C = 1$  acquaintance vaccination strategy in clustered networks with a small total degree distribution is caused by the increased vaccination coverage required to form the same number of ‘vaccinated edges’ (by which we mean edges connected to at least one vaccinated vertex). Indeed, it is possible that further analysis of vaccinated edges may yield analytical insight as to the difference in critical vaccination coverages in clustered and unclustered networks under various vaccination strategies, since vaccination strategies on networks that target high degree vertices (and thus result in an increased number of vaccinated edges) perform best.

We find that fixing  $R_0$  in the unvaccinated model and increasing the clustering coefficient (by increasing  $p_{RW}$  and adjusting  $\lambda_N$ ) has a negligible effect the critical vaccination coverage of the uniform,  $p_S = 1$  acquaintance and optimal

vaccination strategies. This suggests that for a fixed joint stub and triangle distribution the critical vaccination coverages of these three vaccination strategies are determined by  $R_0$ , and independent of the clustering coefficient (similarly to the results of House and Keeling (2011)). However, we find that fixing  $z$  in the unvaccinated model and increasing the clustering coefficient does have an effect on the critical vaccination coverage of the uniform,  $p_S = 1$  acquaintance and optimal vaccination strategies if the mean total degree is small and  $p_{RW}$  is close to 1. We conjecture that fixing  $z$  while changing  $p_N$  and  $p_{RW}$  results in changing the value of the basic reproduction number  $R_0$ , and thus changes the critical vaccination coverages of these three vaccination strategies.

These results suggest that if all the triangles in the underlying network are edge-disjoint, then to calculate the critical vaccination coverage of the uniform,  $p_S = 1$  acquaintance or optimal vaccination strategy we need only know  $R_0$  and the network stub and triangle distribution, and not the exact clustering coefficient.



## 6.8 Table of common notation introduced in Chapter 6

Symbol	Meaning	Page
$(S, T)$	Joint stub and triangle distribution.	180
$p_{st}$	$P(S = s, T = t)$ .	180
$p_{RW}$	Probability of rewiring.	180
$p_N$	Marginal probability that an infected individual infects a given susceptible neighbour.	181
$D_T$	Total degree distribution.	182
$p_k$	$P(D_T = k)$ .	182
$q_{st}$	Probability that an individual reached by traversing a stub has joint stub and triangle degree $(s, t)$ .	183
$r_{st}$	Probability that an individual reached by traversing a triangle or rewired triangle has joint stub and triangle degree $(s, t)$ .	183
$(\tilde{S}^S - 1, T^S)$	Joint stub and triangle size-biased distribution of an individual reached via a stub.	183
$(S^T, \tilde{T}^T - 1)$	Joint stub and triangle size-biased distribution of an individual reached via a triangle or rewired triangle.	183
$\mathcal{C}^\Delta$	Clustering coefficient of the rewired edge-triangle model.	184
$R_T^U$	Threshold parameter for the rewired edge-triangle model under the uniform vaccination strategy.	186
$p_{NV}$	Marginal probability that an infected individual infects a given stub neighbour under the uniform vaccination strategy.	188
$\triangle_E$	Expected final size of a triangle epidemic under the uniform vaccination strategy.	188
$\tilde{\mathbf{B}}_i$	Offspring random vector of a typical type- $i$ individual in a non-initial generation of the backwards branching process.	188

$\mathbf{B}$	Offspring random vector in the initial generation of the backwards branching process.	188
$\triangle$	Size of a single triangle susceptibility set.	193
$\angle$	Size of a rewired triangle susceptibility set.	193
$p_S$	Probability that an individual chosen uniformly at random from the population is sampled under the acquaintance vaccination strategy.	194
$p_C$	Probability that a given neighbour of a sampled individual is vaccinated under the acquaintance vaccination strategy.	194
$(S_U, T_U)$	Joint stub and triangle distribution of an unvaccinated (by the acquaintance vaccination strategy) individual chosen uniformly at random from the population.	196
$(\tilde{S}_U^S, T_U^S)$	Joint stub and triangle distribution of an unvaccinated (by the acquaintance vaccination strategy) individual contacted via a stub.	196
$\tilde{q}_V$	A priori probability that an individual contacted via a stub avoids vaccination (by the acquaintance vaccination strategy).	197
$(S_U^T, \tilde{T}_U^T)$	Joint stub and triangle distribution of an unvaccinated (by the acquaintance vaccination strategy) individual contacted via an intact triangle.	197
$\tilde{r}_V$	A priori probability that an individual contacted via a triangle avoids vaccination (by the acquaintance vaccination strategy).	197
$(S_U^R, \tilde{T}_U^R)$	Joint stub and triangle distribution of an unvaccinated (by the acquaintance vaccination strategy) individual contacted via a rewired triangle.	197
$\tilde{r}_V^R$	A priori probability that an individual contacted via a rewired triangle avoids vaccination (by the acquaintance vaccination strategy).	197
$I_S$	Event that an individual is sampled under the acquaintance vaccination strategy.	198
$I_S^C$	Event that an individual is unsampled under the acquaintance vaccination strategy.	198

$(\Delta_{SS}^F, \Delta_{SN}^F)$	Number of sampled and unsampled individuals respectively infected within a triangle epidemic when the primary infective is sampled.	198
$(\Delta_{NS}^F, \Delta_{NN}^F)$	Number of sampled and unsampled individuals respectively infected within a triangle epidemic when the primary infective is unsampled.	199
$\angle_S^F$	Final size of a rewired triangle epidemic belonging to a sampled primary infective.	200
$\angle_N^F$	Final size of a rewired triangle epidemic belonging to an unsampled primary infective.	200
$(\Delta_{SS}^B, \Delta_{SN}^B)$	Number of sampled and unsampled individuals respectively contained within a triangle susceptibility set belonging to a sampled individual.	201
$b_{\Delta S}(s_1, s_2)$	Joint probability generating function of $(\Delta_{SS}^B, \Delta_{SN}^B)$ .	201
$(\Delta_{NS}^B, \Delta_{NN}^B)$	Number of sampled and unsampled individuals respectively contained within a triangle susceptibility set belonging to an unsampled individual.	201
$b_{\Delta N}(s_1, s_2)$	Joint probability generating function of $(\Delta_{NS}^B, \Delta_{NN}^B)$ .	201
$b_{\angle S}(s)$	Generating function for the final size of a rewired triangle epidemic belonging to a sampled primary individual.	201
$b_{\angle N}(s)$	Generating function for the final size of a rewired triangle epidemic belonging to an unsampled primary individual.	201
$R_T^A$	Threshold parameter for the rewired edge-triangle model under the acquaintance vaccination strategy.	201
$d_c$	Smallest total degree of an individual which we vaccinate under the optimal vaccination strategy.	210
$\delta$	Proportion of individuals with total degree $D_c$ which are chosen uniformly at random for vaccination under the optimal vaccination strategy.	210

$(S_O, T_O)$	Joint stub and triangle distribution of an unvaccinated (by the optimal vaccination strategy) individual chosen uniformly at random from the population.	210
$(\tilde{S}_O^S, T_O^S)$	Joint stub and triangle distribution of an unvaccinated (by the optimal vaccination strategy) individual contacted via a stub.	211
$\tilde{p}_V^S$	A priori probability that an individual contacted via a stub is vaccinated (under the optimal vaccination strategy).	211
$(S_O^T, \tilde{T}_O^T)$	Joint stub and triangle distribution of an unvaccinated (by the optimal vaccination strategy) individual contacted via a triangle or rewired triangle.	211
$\tilde{p}_V^T$	A priori probability that an individual contacted via a triangle or rewired triangle is vaccinated (under the optimal vaccination strategy).	211
$R_T^O$	Threshold parameter for the rewired edge-triangle model under the optimal vaccination strategy.	211
$\mu_i^\Delta$	Mean number of infectives in rank generation $i$ of a single triangle epidemic.	214
$\mu_i^\angle$	Mean number of infectives in rank generation $i$ of a single rewired triangle epidemic.	214
$c_U^*$	Critical vaccination coverage under the uniform vaccination strategy.	218
$c_{p_S}^*$	Critical vaccination coverage under the $p_S = 1$ acquaintance vaccination strategy.	218
$c_{p_C}^*$	Critical vaccination coverage under the $p_C = 1$ acquaintance vaccination strategy.	218
$c_O^*$	Critical vaccination coverage under the optimal vaccination strategy.	218

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## 7. The effect of household clustering on vaccination strategies

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In this chapter we introduce an alternative network model with tunable clustering, which we call the ‘clustered network’ model. The clustered network model is very similar to a special case of the model by Ball et al. (2013), i.e. the household and network model incorporating rewiring. In contrast to the rewired edge-triangle model discussed in Chapter 6, the clustered network model introduces clustering to the standard network model via the addition of households and so the triangles in the model need not be edge-disjoint.

The rewiring process used in the clustered network model allows for a comparison of models differing only in the number of unbroken households, so the effect of clustering upon properties of interest, such as the critical vaccination coverage of vaccination strategies, can be isolated. However, we show that the rewiring process can introduce undesirable properties into the clustered network model, such as an infinite basic reproduction number  $R_0$ .

We are interested in investigating the effect of clustering on the performance of vaccination strategies, similarly to Chapter 6. We introduce two vaccination strategies with a perfect vaccine on the clustered network model, the acquaintance vaccination strategy and the uniform vaccination strategy. We consider the acquaintance vaccination strategy discussed in Chapter 5, in which neighbours of individuals sampled uniformly at random from the population are chosen for vaccination. We allow a sampled individual to choose any neighbour for vaccination, i.e. both household and network neighbours of a sampled individual can be chosen for vaccination. An acquaintance vaccination strategy on the household and network model has been previously discussed by Ball and Sirl

(2017), however Ball and Sirl (2017) do not allow a sampled individual to choose household neighbours for vaccination. Whereas Ball and Sirl (2017) compare the performance of their acquaintance vaccination strategy to the performance of household-based vaccination strategies, we are interested in the effect of household clustering (i.e. clustering introduced by households) on the performance of vaccination strategies and therefore the household structure is considered to be part of the network, which is why we allow household neighbours to be chosen for vaccination.

This chapter is laid out in the following way. In Section 7.1 we introduce the clustered network model and calculate the clustering coefficient. In Sections 7.2 and 7.3 we analyse respectively the acquaintance and uniform vaccination strategies on the clustered network model. In each case we determine a post-vaccination threshold parameter, whether or not the vaccination strategy can control the epidemic and the expected relative final size of a major outbreak. We calculate the basic reproduction number  $R_0$  in Section 7.4 and give conditions under which the rewiring process causes  $R_0$  to be infinite. In Section 7.5 we introduce three approaches to the rewiring process and give an analytical comparison of the final size of a major outbreak under the approaches for a fixed clustering coefficient. In Section 7.6 we numerically compare the acquaintance and uniform vaccination strategies and investigate the effect of household clustering on the performance of the vaccination strategies. Finally, we give our concluding remarks in Section 7.7 and a table of common notation introduced in this chapter in Section 7.8.

## 7.1 Overview of the clustered network model

The clustered network model is essentially a special case of the model by Ball et al. (2013), i.e. the household and network model incorporating tuneable degree correlation and rewiring, without the tuneable degree correlation and extended to allow for the probability of rewiring to depend upon household size. The model is constructed with an analogous method to the construction of the model with three levels of mixing discussed in Section 3.1, with  $\lambda_G = 0$  and with the addition of a rewiring process (described in the following paragraph) after the network is constructed via the configuration model. Recall that the network structure is constructed via the configuration model with network degree distribution  $D$ , and that we set  $H$  to be the asymptotic household

size distribution. Furthermore, we denote the marginal probability that an infected individual infects a given susceptible network neighbour and household (or rewired household) neighbour by  $p_N = 1 - \phi_I(\lambda_N)$  and  $p_H = 1 - \phi_I(\lambda_H)$  respectively.

The rewiring process proceeds in the following way. Label each edge within a household according to the household size. For  $n = 1, 2, \dots$ , let  $p_{RW}(n)$  be a real number satisfying  $0 \leq p_{RW}(n) \leq 1$ . Then, for  $n = 1, 2, \dots$  and independently for each household, with probability  $p_{RW}(n)$ , the household edges in each household of size  $n$  are each broken into two rewired household stubs which retain their household size labels. For each  $n = 2, 3, \dots$ , the rewired household stubs with label  $n$  are paired uniformly at random which, together with the network and unbroken households, creates a new network.

Our analysis is asymptotic as the number of households  $m \rightarrow \infty$  and, as in Section 3.1, assuming  $\sigma_H^2 < \infty$  and  $\sigma_D^2 < \infty$  also means that the fraction of imperfections (e.g. the total number of household self-loops and multiple edges per individual) created by the rewiring process becomes negligible as  $m \rightarrow \infty$  (See, for example, Ball et al. (2013)).

Recall that we denote the size-biased household size distribution by  $\tilde{H}$ . Then the total degree distribution of the clustered network model,  $D_T$ , is given by  $D_T \stackrel{\mathcal{D}}{=} D + \tilde{H} - 1$ , i.e. the summation of two independent random variables (see Ball et al. (2013)). Therefore, assuming  $P(H = 1) < 1$ , a given total degree distribution can be obtained if and only if the total degree distribution is a convolution of two distributions with support in the non-negative integers. Note that extending the clustered network model to allow for dependent household size and network degree distributions would mean that any total degree distribution could be achieved.

Recall from Section 6.1.2 that we define the degree correlation of a network to be the correlation between the total degrees of the nodes adjacent to an edge chosen uniformly at random from the population (see Newman (2002b)). Note that, as in Ball et al. (2013), the rewiring process does not change the distribution of the total degree of the neighbours of any given individual. Therefore the rewiring process does not change the degree correlation of the clustered network. An important consequence of this construction means that if the model is

fully rewired (i.e. if  $P(H = 1) < 1$  and  $p_{RW}(n) = 1$ ,  $n = 1, 2, \dots$ ) then the clustered network model will not have the same properties, such as the threshold parameter and final size of a major outbreak, as the standard network model with the same total degree distribution due to the difference in degree correlation of the two models.

### 7.1.1 Clustering coefficient

We now consider the clustering coefficient of the clustered network model,  $\mathcal{C}^H$ . Recall from Section 6.1.2 that we define the clustering coefficient to be the number of closed triplets divided by the total number of triplets in the asymptotic network (i.e. in the limit  $m \rightarrow \infty$ ). Ball et al. (2010) show that, given  $\sigma_D^2 < \infty$  and  $E[H^3] < \infty$ , the number of closed triplets and the number of triplets in the network without rewiring converges almost surely to  $E[H(H-1)(H-2)]$  and  $E[H(D+H-1)(D+H-2)]$  respectively as  $m \rightarrow \infty$ . Furthermore, analogously to Ball et al. (2013), the number of triangles plus self loops created in the rewiring process has a Poisson distribution whose mean depends only upon the first two moments of  $D$  and three moments of  $H$ . Therefore in the limit  $m \rightarrow \infty$  the number of triangles not within unbroken households, per individual, tends to 0 and

$$\mathcal{C}^H = \frac{E[(1 - p_{RW}(H))H(H-1)(H-2)]}{E[H(D+H-1)(D+H-2)]}. \quad (7.1)$$

Similarly to the rewired edge-triangle model, discussed in Section 6.1.2, the clustered network model will have non-zero clustering if  $P(H \geq 3) > 0$  and  $p_{RW}(n) < 1$ . However, unlike the rewired edge-triangle model, triangles within the clustered network model are not edge-disjoint, so the clustered network model can produce networks with both large mean degree and large clustering coefficient, an important property seen in many social networks (see, for example, Newman (2003)). We say that a probability distribution is infinitely divisible if it can be expressed as the sum of an arbitrary number of independent and identically distributed random variables. Then Ball et al. (2013) show that the clustered network model with any infinitely divisible total degree distribution  $D_T$  may be decomposed so that the clustering coefficient is any rational number in  $[0, 1)$ .



## 7.2 Acquaintance vaccination on the clustered network model

### 7.2.1 Description of acquaintance vaccination

Under the acquaintance vaccination strategy each individual in the population is sampled independently with probability  $p_S$ . Each network neighbour of a sampled individual is independently chosen for vaccination with probability  $p_{CG}$ , and each household or rewired household neighbour of a sampled individual is independently chosen for vaccination with probability  $p_{CH}$ . Finally, any individual which has been chosen for vaccination at least once is vaccinated with the perfect vaccine.

Note that setting  $P(H = 1) = 1$  recovers the standard network model under the acquaintance vaccination strategy with a perfect vaccine, discussed by Ball and Sirl (2013). Furthermore, setting  $p_{CH} = 0$  recovers the household and network model under an acquaintance vaccination strategy with a perfect vaccine discussed by Ball and Sirl (2017).

Under the acquaintance vaccination strategy, for an individual  $i$  to be chosen for vaccination by a given network neighbour  $j$ ,  $j$  must be sampled, occurring with probability  $p_S$ , and choose  $i$  for vaccination, occurring with conditional probability  $p_{CG}$ . Therefore the probability that an individual is not chosen for vaccination by a given network neighbour is  $1 - p_S p_{CG}$ . Similarly, the probability that an individual is not chosen for vaccination by a given household or rewired household neighbour is  $1 - p_S p_{CH}$ . Thus, since an individual  $i$  chosen uniformly at random from the population has  $D$  network neighbours and  $\tilde{H} - 1$  household or rewired household neighbours, the probability that an individual chosen uniformly at random from the population is vaccinated is

$$\begin{aligned} p_V &= 1 - \sum_{k=0}^{\infty} \sum_{n=1}^{\infty} p_k \tilde{p}_n (1 - p_S p_{CG})^k (1 - p_S p_{CH})^{n-1} \\ &= 1 - f_D(1 - p_S p_{CG}) f_{\tilde{H}-1}(1 - p_S p_{CH}). \end{aligned} \quad (7.2)$$

By restricting our attention to a perfect vaccine we need only consider a single-type forward and backward branching process. The forward branching process proceeds similarly to the branching process approximating the model

with three levels of mixing, discussed in Section 3.2.1, in which the individuals in the forward branching process correspond to infected households or rewired households in the epidemic process and the offspring of a given individual in the branching process are all households and rewired households that are contacted through the network by members of the household and rewired epidemic. Recall that we call the initial infective,  $i^*$  say, in a household the primary infective, and any subsequent infected individuals in the household or rewired household containing the primary infective secondary infectives. Furthermore, we call the household neighbours of  $i^*$  non-primary individuals in the household. For example, an infected household of size  $n$  contains 1 primary infective and  $n - 1$  non-primary individuals. Similarly the backwards branching process consists of considering the household and rewired household susceptibility set (see Section 3.3.1) of an individual,  $i^*$  say, and then considering all individuals that would contact  $i^*$ 's household susceptibility set through the network, were they themselves to become infected. Note that in both the forwards and backwards branching processes the initial generation has a different offspring distribution to the following generations since the epidemic starts with an individual chosen uniformly at random from the population.

Before considering a threshold parameter and the final size of a major outbreak for this model, we calculate the household size label (i.e. the household size or number of rewired household neighbours of an individual) and network degree distributions of an unvaccinated individual chosen uniformly at random from the population and an unvaccinated individual contacted via the network. These calculations are very similar to the work in Ball and Sirl (2013), (2016). We also consider the distribution of the number of sampled and unsampled secondary individuals within a household of size  $n$ ,  $n = 1, 2, \dots$ .

Consider an unvaccinated individual,  $i^*$  say, chosen uniformly at random from the population. Note that  $i^*$  has unconditional household size label  $\tilde{H}$  and degree distribution  $D$ , however we also know that  $i^*$  is unvaccinated. Therefore let  $\tilde{H}_U$  and  $D_U$  be  $i^*$ 's household size label and degree distribution respectively.

Then, applying Bayes' theorem, for  $n = 1, 2, \dots$ ,  $k = 0, 1, \dots$ ,

$$\begin{aligned}
P(\tilde{H}_U = n, D_U = k) &= \frac{P(\tilde{H} = n, D = k)P(U|\tilde{H} = n, D = k)}{P(U)} \\
&= \frac{\tilde{\rho}_n p_k (1 - p_{spCH})^{n-1} (1 - p_{spCG})^k}{\sum_{k=0}^{\infty} \sum_{n=1}^{\infty} p_k \tilde{\rho}_n (1 - p_{spCG})^k (1 - p_{spCH})^{n-1}} \\
&= \frac{\tilde{\rho}_n p_k (1 - p_{spCH})^{n-1} (1 - p_{spCG})^k}{f_{\tilde{H}-1}(1 - p_{spCH}) f_D(1 - p_{spCG})} \\
&= \frac{\tilde{\rho}_n (1 - p_{spCH})^{n-1}}{f_{\tilde{H}-1}(1 - p_{spCH})} \frac{p_k (1 - p_{spCG})^k}{f_D(1 - p_{spCG})}.
\end{aligned}$$

Thus  $\tilde{H}_U$  and  $D_U$  are independent and

$$\begin{aligned}
P(\tilde{H}_U = n) &= \tilde{\rho}_n^U = \frac{\tilde{\rho}_n (1 - p_{spCH})^{n-1}}{f_{\tilde{H}-1}(1 - p_{spCH})}, \quad n = 1, 2, \dots, \\
P(D_U = k) &= \frac{p_k (1 - p_{spCG})^k}{1 - p_{VG}}, \quad k = 0, 1, \dots,
\end{aligned}$$

where  $p_{VG} = 1 - f_D(1 - p_{spCG})$  is the a priori probability that an individual with degree  $D$  is vaccinated through the network.

Similarly, we now consider the distribution of the household size label and degree distribution of an individual,  $i$  say, contacted via the network, denoted by  $\tilde{H}_U^D$  and  $\tilde{D}_U$  respectively. An individual contacted via the network has unconditional household size label  $\tilde{H}$  and degree distribution  $D$ , however we also know that  $i$  avoids vaccination from all of its neighbours. Note that we do not count  $i$ 's parent in the branching process, which must not vaccinate  $i$  by definition (leading to the  $k - 1$  term in the equation below). So, for  $n = 1, 2, \dots$ ,  $k = 1, 2, \dots$ ,

$$\begin{aligned}
P(\tilde{H}_U^D = n, \tilde{D}_U = k) &= \frac{P(\tilde{H} = n, \tilde{D} = k)P(U|\tilde{H} = n, \tilde{D} = k)}{P(U)} \\
&= \frac{\tilde{\rho}_n \tilde{p}_k (1 - p_{spCH})^{n-1} (1 - p_{spCG})^{k-1}}{f_{\tilde{H}-1}(1 - p_{spCH}) f_{\tilde{D}-1}(1 - p_{spCG})} \\
&= \frac{\tilde{\rho}_n (1 - p_{spCH})^{n-1}}{f_{\tilde{H}-1}(1 - p_{spCH})} \frac{\tilde{p}_k (1 - p_{spCG})^{k-1}}{f_{\tilde{D}-1}(1 - p_{spCG})}.
\end{aligned}$$

Thus  $\tilde{H}_U^D$  and  $\tilde{D}_U$  are independent,  $\tilde{H}_U^D \stackrel{\mathcal{D}}{=} \tilde{H}_U$  and

$$P(\tilde{D}_U = k) = \frac{\tilde{p}_k (1 - p_{spCG})^{k-1}}{f_{\tilde{D}-1}(1 - p_{spCG})}, \quad k = 1, 2, \dots$$

Let  $\tilde{p}_V = 1 - f_{\bar{H}-1}(1 - p_S p_{CH}) f_{\bar{D}-1}(1 - p_S p_{CG})$  be the a priori probability that an individual contacted via the network is vaccinated.

Consider a primary infective in an intact household in the forward branching process,  $i^*$  say. Note that, since individuals are independently sampled, whether  $i^*$  is sampled or unsampled is independent of the number of sampled non-primary individuals within the household. However, the number of sampled non-primary individuals within the household is dependent on the knowledge that  $i^*$  is unvaccinated. So, similarly to Section 5.1.1, let  $\tilde{p}_{SU}^D = p_S(1 - p_{CG})/(1 - p_S p_{CG})$  and  $\tilde{p}_{SU}^H = p_S(1 - p_{CH})/(1 - p_S p_{CH})$  be the probabilities that an individual infected via the network and rewired household respectively is sampled given that it did not vaccinate its parent in the branching process. Furthermore, let  $\tilde{p}_{SU}^{(n,\alpha)}$ ,  $n = 1, 2, \dots$ ,  $\alpha = 0, 1, \dots, n-1$ , be the probability that a household of size  $n$  contains  $\alpha$  sampled non-primary individuals given that the primary infective in the household is unvaccinated. So

$$\tilde{p}_{SU}^{(n,\alpha)} = \frac{\binom{n-1}{\alpha} p_S^\alpha (1 - p_{CH})^\alpha (1 - p_S)^{n-1-\alpha}}{(1 - p_S p_{CH})^{n-1}}. \quad (7.3)$$

### 7.2.2 The spread of the epidemic through a household and rewired household

We now consider the spread of the epidemic within a single household or rewired household. We first consider the expected number of sampled and unsampled individuals within a household and rewired household epidemic in which the primary infective is sampled or unsampled. Then we consider the distribution of the number of sampled and unsampled members of an individual's household susceptibility set in which the primary individual is sampled or unsampled. Finally we consider the distribution of the total number of members of an individual's rewired household susceptibility set in which the primary individual is sampled or unsampled.

We begin by defining a distribution required for the results in this section. For a random variable  $X$ , we write  $X \sim \text{HGeo}(n, M, K)$ ,  $M = 0, 1, \dots$ ,  $n = 0, 1, \dots, M$ ,  $K = 0, 1, \dots, M$ , if  $X$  has a hypergeometric distribution where  $P(X = k)$  is the probability that there are  $k$  successes in  $n$  draws without replacement from a finite group of size  $M$  containing exactly  $K$  successes. Recall (from Sections 2.2 and 3.3.1 respectively) that the final size of a household

epidemic and the size of a household susceptibility set in a household of size  $n$  are given by  $T^{(n)}$  and  $M^{(n)}$  respectively.

Consider a household of size  $n$ ,  $n = 1, 2, \dots$ , containing  $\alpha$ ,  $\alpha = 0, 1, \dots, n-1$ , sampled non-primary individuals and assume that the primary infective is sampled. Let  $I_{SS}^{(n,\alpha)}$  and  $I_{SN}^{(n,\alpha)}$  be the number of sampled and unsampled non-primary individuals which are vaccinated respectively. Furthermore, let  $T_{SS}^{(n,\alpha)}$  and  $T_{SN}^{(n,\alpha)}$  be respectively the number of infected sampled and unsampled non-primary individuals within a household epidemic in which the primary infective is sampled. Define  $I_{NS}^{(n,\alpha)}$ ,  $I_{NN}^{(n,\alpha)}$ ,  $T_{NS}^{(n,\alpha)}$  and  $T_{NN}^{(n,\alpha)}$  similarly for the case when the primary infective is unsampled.

**Proposition 7.1.** For  $A \in \{S, N\}$ ,  $n = 1, 2, \dots$ ,  $\alpha = 0, 1, \dots, n-1$ ,

$$\mathbb{E} \left[ T_{AS}^{(n,\alpha)} \right] = \sum_{\tilde{\alpha}=0}^{\alpha} \sum_{\tilde{\beta}=0}^{n-1-\alpha} \mathbb{P} \left( I_{AS}^{(n,\alpha)} = \tilde{\alpha} \right) \mathbb{P} \left( I_{AN}^{(n,\alpha)} = \tilde{\beta} \right) \frac{\alpha - \tilde{\alpha}}{n-1-\tilde{\alpha}-\tilde{\beta}} \mathbb{E} \left[ T^{(n-\tilde{\alpha}-\tilde{\beta})} \right],$$

and

$$\mathbb{E} \left[ T_{AN}^{(n,\alpha)} \right] = \sum_{\tilde{\alpha}=0}^{\alpha} \sum_{\tilde{\beta}=0}^{n-1-\alpha} \mathbb{P} \left( I_{AS}^{(n,\alpha)} = \tilde{\alpha} \right) \mathbb{P} \left( I_{AN}^{(n,\alpha)} = \tilde{\beta} \right) \frac{n-1-\alpha-\tilde{\beta}}{n-1-\tilde{\alpha}-\tilde{\beta}} \mathbb{E} \left[ T^{(n-\tilde{\alpha}-\tilde{\beta})} \right],$$

where

$$I_{SS}^{(n,\alpha)} \sim \text{Bin} \left( \alpha, 1 - (1 - p_{VG})(1 - p_{CH})^\alpha \right), \quad (7.4a)$$

$$I_{SN}^{(n,\alpha)} \sim \text{Bin} \left( n-1-\alpha, 1 - (1 - p_{VG})(1 - p_{CH})^{\alpha+1} \right), \quad (7.4b)$$

$$I_{NS}^{(n,\alpha)} \sim \text{Bin} \left( \alpha, 1 - (1 - p_{VG})(1 - p_{CH})^{\alpha-1} \right), \quad (7.4c)$$

$$I_{NN}^{(n,\alpha)} \sim \text{Bin} \left( n-1-\alpha, 1 - (1 - p_{VG})(1 - p_{CH})^\alpha \right). \quad (7.4d)$$

*Proof.* Consider a household of size  $n$  containing  $\alpha$  sampled non-primary individuals and let  $\tilde{\alpha} \in [0; \alpha]$  and  $\tilde{\beta} \in [0; n-1-\alpha]$  be the number of vaccinated sampled and unsampled non-primary individuals respectively within the household. Then, conditioned upon the final size of the household epidemic,  $T^{(n)}$  (i.e. the total number of sampled and unsampled non-primary individuals infected), the probability that  $k \in [0; \alpha - \tilde{\alpha}]$  of the sampled non-primary individuals are infected is equal to the probability that there are  $k$  successes in  $T^{(n-\tilde{\alpha}-\tilde{\beta})}$  draws without replacement from a finite population of size  $n-1-\tilde{\alpha}-\tilde{\beta}$  containing exactly

$\alpha - \tilde{\alpha}$  successes. Therefore, for  $A \in \{S, N\}$ ,

$$T_{AS}^{(n,\alpha)} \mid I_{AS}^{(n,\alpha)} = \tilde{\alpha}, I_{AN}^{(n,\alpha)} = \tilde{\beta}, T^{(n-\tilde{\alpha}-\tilde{\beta})} \sim \text{HGeo}\left(T^{(n-\tilde{\alpha}-\tilde{\beta})}, n-1-\tilde{\alpha}-\tilde{\beta}, \alpha-\tilde{\alpha}\right),$$

and

$$T_{AN}^{(n,\alpha)} \mid \left(I_{AS}^{(n,\alpha)} = \tilde{\alpha}, I_{AN}^{(n,\alpha)} = \tilde{\beta}, T^{(n-\tilde{\alpha}-\tilde{\beta})}, T_{AS}^{(n,\alpha)}\right) = T^{(n-\tilde{\alpha}-\tilde{\beta})} - T_{AS}^{(n,\alpha)}.$$

Therefore, since if  $X \sim \text{HGeo}(n, M, K)$  then  $\mathbb{E}[X] = nK/M$ ,

$$\begin{aligned} \mathbb{E}\left[T_{AS}^{(n,\alpha)}\right] &= \mathbb{E}\left[\mathbb{E}\left[T_{AS}^{(n,\alpha)} \mid I_{AS}^{(n,\alpha)} = \tilde{\alpha}, I_{AN}^{(n,\alpha)} = \tilde{\beta}, T^{(n-\tilde{\alpha}-\tilde{\beta})}\right]\right] \\ &= \mathbb{E}\left[\sum_{\tilde{\alpha}=0}^{\alpha-k} \sum_{\tilde{\beta}=0}^{n-1-\alpha-j} \mathbb{P}\left(I_{AS}^{(n,\alpha)} = \tilde{\alpha}\right) \mathbb{P}\left(I_{AN}^{(n,\alpha)} = \tilde{\beta}\right) \frac{\alpha - \tilde{\alpha}}{n-1-\tilde{\alpha}-\tilde{\beta}} T^{(n-\tilde{\alpha}-\tilde{\beta})}\right] \\ &= \sum_{\tilde{\alpha}=0}^{\alpha-k} \sum_{\tilde{\beta}=0}^{n-1-\alpha-j} \mathbb{P}\left(I_{AS}^{(n,\alpha)} = \tilde{\alpha}\right) \mathbb{P}\left(I_{AN}^{(n,\alpha)} = \tilde{\beta}\right) \frac{\alpha - \tilde{\alpha}}{n-1-\tilde{\alpha}-\tilde{\beta}} \mathbb{E}\left[T^{(n-\tilde{\alpha}-\tilde{\beta})}\right] \end{aligned}$$

and

$$\mathbb{E}\left[T_{AN}^{(n,\alpha)}\right] = \sum_{\tilde{\alpha}=0}^{\alpha-k} \sum_{\tilde{\beta}=0}^{n-1-\alpha-j} \mathbb{P}\left(I_{AS}^{(n,\alpha)} = \tilde{\alpha}\right) \mathbb{P}\left(I_{AN}^{(n,\alpha)} = \tilde{\beta}\right) \frac{n-1-\alpha-\tilde{\beta}}{n-1-\tilde{\alpha}-\tilde{\beta}} \mathbb{E}\left[T^{(n-\tilde{\alpha}-\tilde{\beta})}\right].$$

Finally we consider the distributions of  $I_{SS}^{(n,\alpha)}$ ,  $I_{SN}^{(n,\alpha)}$ ,  $I_{NS}^{(n,\alpha)}$  and  $I_{NN}^{(n,\alpha)}$ . Consider a non-primary individual,  $j$  say, within the household of size  $n$ . Then to be unvaccinated  $j$  needs to avoid vaccination from all network neighbours of  $j$ , the sampled secondary individuals in  $j$ 's household and the primary infective. Recall that  $j$  avoids vaccination from all network neighbours with probability  $1 - p_{VG}$  and from  $\alpha$  sampled non-primary individuals with probability  $(1 - p_{CH})^\alpha$ . If the primary infective is sampled or unsampled then  $j$  avoids vaccination from the primary infective with probability  $1 - p_{CH}$  or 1 respectively. Therefore, since conditioned upon the number of sampled individuals within the household each non-primary individual is chosen for vaccination independently of any other,  $I_{SS}^{(n,\alpha)}$ ,  $I_{SN}^{(n,\alpha)}$ ,  $I_{NS}^{(n,\alpha)}$  and  $I_{NN}^{(n,\alpha)}$  are all binomially distributed with the parameters as given in equations (7.4).  $\square$

Let  $\hat{T}_S^{(n)}$  and  $\hat{T}_N^{(n)}$  be the final size of a rewired household epidemic, i.e. the total number of infected sampled and unsampled individuals, with household size label  $n$ ,  $n = 1, 2, \dots$ , in which the primary infective is sampled and unsampled respectively. For notational simplicity let  $\tilde{p}_H(n) = (1 - p_{VG})(1 - \tilde{p}_{SU}^H p_{CH})^{n-1} p_H$ , and note that the following proposition is an extension of Ball et al. (2013)

Equation (34) to allow for individuals within the rewired household to be vaccinated by the acquaintance vaccination strategy.

**Proposition 7.2.**

$$\begin{aligned} \mathbb{E} [\hat{T}_N^{(1)}] &= 0, \\ \mathbb{E} [\hat{T}_N^{(2)}] &= p_H(1 - p_{VG}), \end{aligned}$$

and, for  $n \geq 3$ ,

$$\mathbb{E} [\hat{T}_N^{(n)}] = \begin{cases} \frac{(n-1)(1-p_{VG})(1-\tilde{p}_{SU}^H p_{CH})^{n-2} p_H}{1-(n-2)\tilde{p}_H(n)} & \text{if } \tilde{p}_H(n) < \frac{1}{n-2}, \\ \infty & \text{if } \tilde{p}_H(n) \geq \frac{1}{n-2}. \end{cases}$$

Finally, for  $n = 1, 2, \dots$ ,

$$\mathbb{E} [\hat{T}_S^{(n)}] = (1 - p_{CH}) \mathbb{E} [\hat{T}_N^{(n)}].$$

*Proof.* A rewired household with household size label  $n$  is constructed by pairing rewired household stubs with the label  $n$  uniformly at random. Therefore the spread of the epidemic through a rewired household is a special case of the spread of the epidemic on the standard network model in which every individual in the population has degree  $n$ , and acquaintance vaccination on the standard network model has been previously studied (see, for example, Section 5.1 or Ball and Sirl (2013)). If  $N$  is large then the rewired household is locally tree-like, and we can approximate the spread of the epidemic through a rewired household with a branching process.

Note that  $\hat{T}_S^{(1)} = \hat{T}_N^{(1)} = 0$  since households of size 1 contain no non-primary individuals. Households of size 2 contain a single non-primary individual,  $j$  say, who is infected by the primary infective if:  $j$  is contacted by the primary individual,  $j$  is not vaccinated by the primary individual and  $j$  is not vaccinated by its network neighbours. So

$$\begin{aligned} \mathbb{E} [\hat{T}_S^{(2)}] &= p_H(1 - p_{CH})(1 - p_{VG}), \\ \mathbb{E} [\hat{T}_N^{(2)}] &= p_H(1 - p_{VG}). \end{aligned}$$

Finally, consider a primary individual  $i$  and a non-primary individual  $k$  in a rewired household of size  $n \geq 3$ . Since we assume that the rewired household is locally tree-like,  $i$  has  $n - 1$  uninfected rewired household neighbours and  $k$

has  $n - 2$  uninfected rewired household neighbours. The primary individual  $i$  will only infect a given rewired household neighbour,  $j$  say, if  $j$ : is not chosen for vaccination by  $i$ , is not already vaccinated by another network neighbour, is not already vaccinated by another of its  $n - 2$  rewired household neighbours and is contacted by  $i$ . Thus if  $i$  is sampled then  $i$  infects  $j$  with probability  $(1 - p_{CH})(1 - p_{VG})(1 - \tilde{p}_{SU}^H p_{CH})^{n-2} p_H$  and if  $i$  is unsampled then  $i$  contacts  $j$  with probability  $(1 - p_{VG})(1 - \tilde{p}_{SU}^H p_{CH})^{n-2} p_H$ . Applying analogous arguments and noting that  $k$  is sampled with probability  $\tilde{p}_{SU}^H$  and otherwise unsampled,  $k$  infects a given rewired household neighbour  $l$  with probability  $(1 - \tilde{p}_{SU}^H p_{CH})(1 - p_{VG})(1 - \tilde{p}_{SU}^H p_{CH})^{n-2} p_H$ . Thus in the (single-type) branching process which gives the size of successive generations of infectives in the rewired household epidemic with household size  $n$ , the initial ancestor has offspring mean  $(n-1)(1-p_{CH})(1-p_{VG})(1-\tilde{p}_{SU}^H p_{CH})^{n-2} p_H$  or  $(n-1)(1-p_{VG})(1-\tilde{p}_{SU}^H p_{CH})^{n-2} p_H$ , depending on whether the ancestor is sampled or not, and all subsequent individuals have  $(n-2)(1-\tilde{p}_{SU}^H p_{CH})(1-p_{VG})(1-\tilde{p}_{SU}^H p_{CH})^{n-2} p_H = (n-2)\tilde{p}_H(n)$  mean offspring. Thus, for  $n \geq 3$ ,

$$\mathbb{E} [\hat{T}_N^{(n)}] = \begin{cases} \frac{(n-1)(1-p_{VG})(1-\tilde{p}_{SU}^H p_{CH})^{n-2} p_H}{1-(n-2)\tilde{p}_H(n)} & \text{if } \tilde{p}_H(n) < \frac{1}{n-2}, \\ \infty & \text{if } \tilde{p}_H(n) \geq \frac{1}{n-2}, \end{cases}$$

and  $\mathbb{E} [\hat{T}_S^{(n)}] = \mathbb{E} [\hat{T}_N^{(n)}] (1 - p_{CH})$  as required.  $\square$

We now turn our attention to the distribution of the number of sampled and unsampled individuals within an individual's household susceptibility set in which the primary individual is sampled or unsampled. For  $n = 1, 2, \dots$  and  $\alpha = 0, 1, \dots, n - 1$  let  $M_{SS}^{(n,\alpha)}$  and  $M_{SN}^{(n,\alpha)}$  be respectively the number of sampled and unsampled non-primary individuals within a household susceptibility set in which the primary individual is sampled. Define  $M_{NS}^{(n,\alpha)}$  and  $M_{NN}^{(n,\alpha)}$  similarly for the case when the primary individual is unsampled. Note that Proposition 7.3 is an extension of Ball et al. (2013) Equations (35) and (36) to allow for individuals within the rewired household to be vaccinated by the acquaintance vaccination strategy.



**Proposition 7.3.** For  $A \in \{S, N\}$ ,  $n = 1, 2, \dots$ ,  $\alpha = 0, 1, \dots, n-1$ ,  $k = 0, 1, \dots, \alpha$ ,  $j = 0, 1, \dots, n-1-\alpha$ ,

$$\begin{aligned} & \mathbb{P} \left( M_{(AS)}^{(n,\alpha)} = k, M_{(AN)}^{(n,\alpha)} = j \right) \\ &= \sum_{\tilde{\alpha}=0}^{\alpha-k} \sum_{\tilde{\beta}=0}^{n-1-\alpha-j} \mathbb{P} \left( I_{AS}^{(n,\alpha)} = \tilde{\alpha} \right) \mathbb{P} \left( I_{AN}^{(n,\alpha)} = \tilde{\beta} \right) \mathbb{P} \left( M^{(n-\tilde{\alpha}-\tilde{\beta})} = k+j \right) \\ & \quad \times \frac{\binom{\alpha-\tilde{\alpha}}{k} \binom{n-1-\alpha-\tilde{\beta}}{j}}{\binom{n-1-\tilde{\alpha}-\tilde{\beta}}{k+j}}. \end{aligned}$$

We omit the proof of Proposition 7.3 since it follows analogous arguments to the proof of Proposition 7.1. The difference arises in considering the spread of an individual's household susceptibility set, rather than the spread of an individual's household epidemic. Therefore the proof of Proposition 7.3 follows by conditioning on the size of an individual's susceptibility set, which is denoted by  $M^{(n)}$  for an individual within a household of size  $n$ , instead of the final size of a household epidemic. For  $A \in \{S, N\}$ , let  $f_{M_A}^{(n,\alpha)}(s_1, s_2) = \mathbb{E} \begin{bmatrix} M_{(AS)}^{(n,\alpha)} & M_{(AN)}^{(n,\alpha)} \\ s_1 & s_2 \end{bmatrix}$ .

Finally, we consider the size of a rewired household susceptibility set. Let  $\hat{M}_S^{(n)}$  and  $\hat{M}_N^{(n)}$  be the final size of a rewired household susceptibility set with household size label  $n$  in which the primary individual is sampled and unsampled respectively.

**Proposition 7.4.** For  $n = 1, 2, \dots$ ,  $s \in [0, 1]$ ,

$$\begin{aligned} f_{\hat{M}_S^{(n)}}(s) &= \left( 1 - p_H(1 - p_{CH})(1 - \tilde{p}_{SU}^H p_{CH})^{n-2}(1 - p_{VG})(1 - \hat{f}^{(n)}(s)) \right)^{n-1}, \\ f_{\hat{M}_N^{(n)}}(s) &= \left( 1 - p_H(1 - \tilde{p}_{SU}^H p_{CH})^{n-2}(1 - p_{VG})(1 - \hat{f}^{(n)}(s)) \right)^{n-1}, \end{aligned}$$

where  $\hat{f}^{(n)}(s)$  is the unique solution in  $[0, 1]$  of the equation

$$\hat{f}^{(n)}(s) = s \left( 1 - p_H(1 - \tilde{p}_{SU}^H p_{CH})^{n-1}(1 - p_{VG})(1 - \hat{f}^{(n)}(s)) \right)^{n-2}.$$

*Proof.* Similarly to the proof of Proposition 7.2, we approximate the spread of an individual's rewired susceptibility set through a rewired household with a branching process.

Note that  $\hat{M}_S^{(1)} = 0$  and  $\hat{M}_N^{(1)} = 0$  since households of size 1 contain no non-primary individuals. Households of size 2 contain a single non-primary individual,  $j$  say, who joins the rewired household susceptibility set if  $j$ : contacts

the primary individual, is not vaccinated by the primary individual and is not vaccinated by it's network neighbours. So

$$\begin{aligned} f_{\hat{M}_S^{(2)}}(s) &= 1 - p_H(1 - p_{CH})(1 - p_{VG})(1 - s), \\ f_{\hat{M}_N^{(2)}}(s) &= 1 - p_H(1 - p_{VG})(1 - s). \end{aligned}$$

Finally, consider a primary individual  $i$  and a non-primary individual  $k$  in a rewired household of size  $n \geq 3$ . We assume that the rewired household is locally tree-like so all infectious contacts made to  $i$  via the rewired household are made by distinct individuals who are unconnected if  $i$  is removed from the network. Therefore all infectious contacts made to  $i$  via the rewired household are made by individuals with independent and identically distributed infectious periods. Similarly, all infectious contacts made to  $k$  via the rewired household are made by individuals with independent and identically distributed infectious periods. Note that  $i$  has  $n - 1$  rewired household neighbours and a given rewired household neighbour,  $j$ , of  $i$  can only make infectious contact with  $i$  if  $j$ : is not chosen for vaccination by  $i$ , is not already vaccinated by a network neighbour, is not already vaccinated by another of it's  $n - 2$  rewired household neighbours and contacts  $i$ . Thus if  $i$  is sampled then  $j$  contacts  $i$  with probability  $(1 - p_{CH})(1 - p_{VG})(1 - \tilde{p}_{SU}^H p_{CH})^{n-2} p_H$  and if  $i$  is unsampled then  $j$  infects  $i$  with probability  $(1 - p_{VG})(1 - \tilde{p}_{SU}^H p_{CH})^{n-2} p_H$ . Noting that  $k$  is sampled with probability  $\tilde{p}_{SU}^H$  and otherwise unsampled and applying analogous arguments yields that  $k$  is infected by a given rewired household neighbour  $l$  with probability  $(1 - \tilde{p}_{SU}^H p_{CH})(1 - p_{VG})(1 - \tilde{p}_{SU}^H p_{CH})^{n-2} p_H$ . Therefore standard branching process arguments (see Section 2.3) yield that, for  $n \geq 3$ ,  $s \in [0, 1]$ ,

$$\begin{aligned} f_{\hat{M}_S^{(n)}}(s) &= \left(1 - p_H(1 - p_{CH})(1 - \tilde{p}_{SU}^H p_{CH})^{n-2}(1 - p_{VG})(1 - \hat{f}^{(n)}(s))\right)^{n-1}, \\ f_{\hat{M}_N^{(n)}}(s) &= \left(1 - p_H(1 - \tilde{p}_{SU}^H p_{CH})^{n-2}(1 - p_{VG})(1 - \hat{f}^{(n)}(s))\right)^{n-1}, \end{aligned}$$

where  $\hat{f}^{(n)}(s)$  is the unique solution in  $[0, 1]$  of the equation

$$\hat{f}^{(n)}(s) = s \left(1 - p_H(1 - \tilde{p}_{SU}^H p_{CH})^{n-1}(1 - p_{VG})(1 - \hat{f}^{(n)}(s))\right)^{n-2}.$$

□

### 7.2.3 Threshold parameter

Before giving  $R_*^A$  in Theorem 7.1 we introduce the following notation.

$$\begin{aligned} \mu_{T^{(n,\alpha)}} = p_N(1 - \tilde{p}_V)E[D_U] & \left( (1 - \tilde{p}_{SU}^D) \left\{ (1 - p_{CG})E[T_{NS}^{(n,\alpha)}] + E[T_{NN}^{(n,\alpha)}] \right\} \right. \\ & \left. + \tilde{p}_{SU}^D \left\{ (1 - p_{CG})E[T_{SS}^{(n,\alpha)}] + E[T_{SN}^{(n,\alpha)}] \right\} \right), \end{aligned} \quad (7.11a)$$

and

$$\mu_{\hat{T}^{(n)}} = p_N(1 - \tilde{p}_V)E[D_U] (1 - \tilde{p}_{SU}^D p_{CG}) \left[ \tilde{p}_{SU}^D E[\hat{T}_S^{(n)}] + (1 - \tilde{p}_{SU}^D) E[\hat{T}_N^{(n)}] \right], \quad (7.11b)$$

where  $E[T_{SS}^{(n,\alpha)}]$ ,  $E[T_{NS}^{(n,\alpha)}]$ ,  $E[T_{SN}^{(n,\alpha)}]$  and  $E[T_{NN}^{(n,\alpha)}]$  are given in Proposition 7.1 and  $E[\hat{T}_S^{(n)}]$  and  $E[\hat{T}_N^{(n)}]$  are given in Proposition 7.3. Note that  $\mu_{T^{(n,\alpha)}}$  is the expected number of individuals infected by the non-primary members of a household epidemic with household size  $n$  and  $\alpha$  sampled non-primary individuals, and that  $\mu_{\hat{T}^{(n)}}$  is the expected number of individuals infected by the non-primary members of a rewired household epidemic with household size label  $n$ .

**Theorem 7.1.**

$$\begin{aligned} R_*^A = p_N(1 - \tilde{p}_V)E[\tilde{D}_U - 1] & \left( 1 - \tilde{p}_{SU}^D p_{CG} \right) \\ & + \sum_{n=1}^{\infty} \tilde{\rho}_n^U \left\{ p_{RW}(n) \mu_{\hat{T}^{(n)}} + (1 - p_{RW}(n)) \sum_{\alpha=0}^{n-1} \tilde{p}_{SU}^{(n,\alpha)} \mu_{T^{(n,\alpha)}} \right\}, \end{aligned}$$

where  $\mu_{T^{(n,\alpha)}}$  and  $\mu_{\hat{T}^{(n)}}$  are given in equations (7.11).

*Proof.* Consider an unvaccinated individual contacted via the network and let  $\tilde{C}$  be the total number of network infections made by the resulting household or rewired household epidemic, so  $R_*^A = E[\tilde{C}]$ . We proceed with this proof by conditioning  $E[\tilde{C}]$  on the household size label of the unvaccinated primary infective and whether the primary infective is in a household or rewired household. An unvaccinated individual contacted via the network is within a household of size  $n$  with probability  $\tilde{\rho}_n^U$  and households are rewired independently and uniformly at random with probability  $p_{RW}(n)$ , so

$$E[\tilde{C}] = \sum_{n=1}^{\infty} \tilde{\rho}_n^U \left[ (1 - p_{RW}(n)) E[\tilde{C}_H^{(n)}] + p_{RW}(n) E[\tilde{C}_R^{(n)}] \right], \quad (7.12)$$

where  $\tilde{C}_H^{(n)}$  and  $\tilde{C}_R^{(n)}$  are the random variable  $\tilde{C}$  conditioned on the primary infective being in a household of size  $n$  and a rewired household with household size label  $n$  respectively.

We first consider  $E[\tilde{C}_H^{(n)}]$ , i.e. the expected number of infections made via the network by the members of a household epidemic with household size  $n$ , when the initial infective was contacted via the network. Conditioning  $E[\tilde{C}_H^{(n)}]$  on the number of sampled secondary individuals in the household and whether the primary infective is sampled or not yields

$$E[\tilde{C}_H^{(n)}] = \sum_{\alpha=0}^{n-1} \tilde{p}_{SU}^{(n,\alpha)} [\tilde{p}_{SU}^D E[\tilde{C}_{HS}^{(n,\alpha)}] + (1 - \tilde{p}_{SU}^D) E[\tilde{C}_{HN}^{(n,\alpha)}]], \quad (7.13)$$

where  $\tilde{C}_{HS}^{(n,\alpha)}$  ( $\tilde{C}_{HN}^{(n,\alpha)}$ ) is the quantity  $\tilde{C}_H^{(n)}$  conditioned on the primary infective being sampled (unsampled) and the household containing  $\alpha$  sampled non-primary individuals.

Next we decompose  $\tilde{C}_{HS}^{(n,\alpha)}$  and  $\tilde{C}_{HN}^{(n,\alpha)}$  into the number of network infections made by each member of the primary infective's household epidemic. Therefore we label the primary infective in the household 0, the  $\alpha$  sampled members of the primary infective's household  $1, 2, \dots, \alpha$  and the  $n - 1 - \alpha$  unsampled members of the primary infective's household  $\alpha + 1, \alpha + 2, \dots, n - 1 - \alpha$ . Furthermore, let  $\chi_i^S$  and  $\chi_i^N$  be the indicator functions of the events that individual  $i$  is infected by the household epidemic when the primary infective is sampled and unsampled respectively, i.e.  $\chi_i^S = 1$  if  $i$  is infected when the primary infective is sampled and 0 otherwise. Thus

$$\tilde{C}_{HS}^{(n,\alpha)} = \tilde{C}_{HS}^{(n,\alpha)}(0) + \sum_{i=1}^{\alpha} \chi_i^S \tilde{C}_{HS}^{(n,\alpha,S)}(i) + \sum_{i=\alpha+1}^{n-1} \chi_i^S \tilde{C}_{HS}^{(n,\alpha,N)}(i), \quad (7.14a)$$

$$\tilde{C}_{HN}^{(n,\alpha)} = \tilde{C}_{HN}^{(n,\alpha)}(0) + \sum_{i=1}^{\alpha} \chi_i^N \tilde{C}_{HN}^{(n,\alpha,S)}(i) + \sum_{i=\alpha+1}^{n-1} \chi_i^N \tilde{C}_{HN}^{(n,\alpha,N)}(i), \quad (7.14b)$$

where:  $\tilde{C}_{HS}^{(n,\alpha)}(0)$  ( $\tilde{C}_{HN}^{(n,\alpha)}(0)$ ) is the number of contacts made by the sampled (unsampled) primary infective,  $\tilde{C}_{HS}^{(n,\alpha,S)}(i)$  ( $\tilde{C}_{HS}^{(n,\alpha,N)}(i)$ ) is the number of contacts made by sampled non-primary individual  $i$  when the primary infective is sampled (unsampled) conditioned on  $i$  becoming infected and  $\tilde{C}_{HS}^{(n,\alpha,N)}(i)$  ( $\tilde{C}_{HN}^{(n,\alpha,N)}(i)$ ) is the number of contacts made by the unsampled non-primary individual  $i$  when the primary infective is sampled (unsampled) conditioned on  $i$  becoming infected.

The event that an individual  $i$  is infected is independent of how many contacts  $i$  would make if  $i$  became infected, as whether  $i$  is infected in the household epidemic is independent of  $i$ 's infectious period. Therefore, for  $A \in \{S, N\}$ ,  $\chi_i^A$  and  $\tilde{C}_{HA}^{(n,\alpha,S)}(i)$  are independent for  $i = 1, 2, \dots, \alpha$  and  $\chi_i^A$  and  $\tilde{C}_{HA}^{(n,\alpha,N)}(i)$  are independent for  $i = \alpha + 1, \alpha + 2, \dots, n - 1 - \alpha$ . Furthermore,  $(\tilde{C}_{HA}^{(n,\alpha,S)}(i), \chi_i^A)$ ,  $i = 1, 2, \dots, \alpha$ , have the same distribution and, for  $i = \alpha + 1, \alpha + 2, \dots, n - 1 - \alpha$ ,  $(\tilde{C}_{HA}^{(n,\alpha,N)}(i), \chi_i^A)$  have the same distribution. Thus taking the expectation of equations (7.14) yields

$$\mathbb{E} [\tilde{C}_{HS}^{(n,\alpha)}] = \mathbb{E} [\tilde{C}_{HS}^{(n,\alpha)}(0)] + \mathbb{E} [T_{SS}^{(n,\alpha)}] \mathbb{E} [\tilde{C}_{HS}^{(n,\alpha,S)}(1)] + \mathbb{E} [T_{SN}^{(n,\alpha)}] \mathbb{E} [\tilde{C}_{HS}^{(n,\alpha,N)}(1)], \quad (7.15a)$$

$$\mathbb{E} [\tilde{C}_{HN}^{(n,\alpha)}] = \mathbb{E} [\tilde{C}_{HN}^{(n,\alpha)}(0)] + \mathbb{E} [T_{NS}^{(n,\alpha)}] \mathbb{E} [\tilde{C}_{HN}^{(n,\alpha,S)}(1)] + \mathbb{E} [T_{NN}^{(n,\alpha)}] \mathbb{E} [\tilde{C}_{HN}^{(n,\alpha,N)}(1)], \quad (7.15b)$$

where  $T_{SS}^{(n,\alpha)}$  and  $T_{SN}^{(n,\alpha)}$  are the number of sampled and unsampled in the primary infective's household epidemic when the primary infective is sampled and  $T_{NS}^{(n,\alpha)}$  and  $T_{NN}^{(n,\alpha)}$  are the number of sampled and unsampled in the primary infective's household epidemic when the primary infective is unsampled. Recall that  $\mathbb{E} [T_{SS}^{(n,\alpha)}]$ ,  $\mathbb{E} [T_{SN}^{(n,\alpha)}]$ ,  $\mathbb{E} [T_{NS}^{(n,\alpha)}]$  and  $\mathbb{E} [T_{NN}^{(n,\alpha)}]$  are given in Proposition 7.1.

To complete our calculation of  $\mathbb{E} [\tilde{C}_H^{(n)}]$  we need only consider the expected number of infectious network contacts made by a sampled and unsampled primary infective and the non-primary infectives in the household epidemic. For  $A \in \{S, N\}$ , the expectations of  $\tilde{C}_{HA}^{(n,\alpha)}(0)$ ,  $\tilde{C}_{HA}^{(n,\alpha,S)}(1)$  and  $\tilde{C}_{HA}^{(n,\alpha,N)}(1)$  can be determined by conditioning on the individual's infectious period,  $I$ , and the number of uninfected network neighbours it has, which is  $D_U$  for a secondary infective and  $\tilde{D}_U - 1$  for the primary infective. Consider an infected individual  $i^*$ . Conditional on  $i^*$ 's infectious period,  $i^*$  makes infectious contact with a given network neighbour,  $j$ , if all of the following hold:  $j$  is not chosen for vaccination by  $i^*$ ,  $j$  is not already vaccinated by another neighbour and  $j$  is contacted by  $i^*$ . Thus if  $i^*$  is sampled then  $i^*$  contacts  $j$  with probability  $(1 - p_{CG})(1 - \tilde{p}_V) (1 - e^{-\lambda_N I_i})$  and if  $i^*$  is unsampled then  $i^*$  contacts  $j$  with

probability  $(1 - \tilde{p}_V) (1 - e^{-\lambda_N I_i})$ . Therefore, for  $A \in \{S, N\}$ ,

$$\begin{aligned}\tilde{C}_{HA}^{(n,\alpha,S)}(1)|D_U, I_1 &\sim \text{Bin} \left( D_U, \left(1 - e^{-\lambda_N I_1}\right) (1 - p_{CG})(1 - \tilde{p}_V) \right), \\ \tilde{C}_{HA}^{(n,\alpha,N)}(1)|D_U, I_1 &\sim \text{Bin} \left( D_U, \left(1 - e^{-\lambda_N I_1}\right) (1 - \tilde{p}_V) \right), \\ \tilde{C}_{HS}^{(n,\alpha)}(0)|\tilde{D}_U, I_0 &\sim \text{Bin} \left( \tilde{D}_U - 1, \left(1 - e^{-\lambda_N I_0}\right) (1 - p_{CG})(1 - \tilde{p}_V) \right), \\ \tilde{C}_{HN}^{(n,\alpha)}(0)|\tilde{D}_U, I_0 &\sim \text{Bin} \left( \tilde{D}_U - 1, \left(1 - e^{-\lambda_N I_0}\right) (1 - \tilde{p}_V) \right).\end{aligned}$$

Furthermore, since an individual's infectious period is independent of its degree distribution,

$$\mathbb{E} \left[ \tilde{C}_{HA}^{(n,\alpha,S)}(1) \right] = \mathbb{E} [D_U] p_N (1 - p_{CG})(1 - \tilde{p}_V), \quad (7.17a)$$

$$\mathbb{E} \left[ \tilde{C}_{HA}^{(n,\alpha,N)}(1) \right] = \mathbb{E} [D_U] p_N (1 - \tilde{p}_V), \quad (7.17b)$$

$$\mathbb{E} \left[ \tilde{C}_{HS}^{(n,\alpha)}(0) \right] = \mathbb{E} [\tilde{D}_U - 1] p_N (1 - p_{CG})(1 - \tilde{p}_V), \quad (7.17c)$$

$$\mathbb{E} \left[ \tilde{C}_{HN}^{(n,\alpha)}(0) \right] = \mathbb{E} [\tilde{D}_U - 1] p_N (1 - \tilde{p}_V). \quad (7.17d)$$

Substituting equations (7.17) into equation (7.15) yields

$$\begin{aligned}\mathbb{E} \left[ \tilde{C}_{HS}^{(n,\alpha)} \right] &= p_N (1 - \tilde{p}_V) \left\{ (1 - p_{CG}) \left( \mathbb{E} [\tilde{D}_U - 1] + \mathbb{E} [T_{SS}^{(n,\alpha)}] \mathbb{E} [D_U] \right) \right. \\ &\quad \left. + \mathbb{E} [T_{SN}^{(n,\alpha)}] \mathbb{E} [D_U] \right\},\end{aligned} \quad (7.18a)$$

$$\begin{aligned}\mathbb{E} \left[ \tilde{C}_{HN}^{(n,\alpha)} \right] &= p_N (1 - \tilde{p}_V) \left\{ \mathbb{E} [\tilde{D}_U - 1] + (1 - p_{CG}) \mathbb{E} [T_{NS}^{(n,\alpha)}] \mathbb{E} [D_U] \right. \\ &\quad \left. + \mathbb{E} [T_{NN}^{(n,\alpha)}] \mathbb{E} [D_U] \right\},\end{aligned} \quad (7.18b)$$

where  $\mathbb{E} [T_{SS}^{(n,\alpha)}]$ ,  $\mathbb{E} [T_{SN}^{(n,\alpha)}]$ ,  $\mathbb{E} [T_{NS}^{(n,\alpha)}]$  and  $\mathbb{E} [T_{NN}^{(n,\alpha)}]$  are given in Proposition 7.1. Furthermore, substituting equations (7.18) into equation (7.13) yields

$$\mathbb{E} \left[ \tilde{C}_H^{(n)} \right] = p_N (1 - \tilde{p}_V) (1 - \tilde{p}_{SU}^D p_{CG}) \mathbb{E} [\tilde{D}_U - 1] + \mu_{T^{(n,\alpha)}}, \quad (7.19)$$

where  $\mu_{T^{(n,\alpha)}}$  is given in equation (7.11a).

To complete our calculation of  $R_*^A$  we need only calculate  $\mathbb{E} [\tilde{C}_R^{(n)}]$ , i.e. the expected number of network contacts made by the members of a rewired household epidemic of an individual with household size label  $n$ . We begin by conditioning  $\tilde{C}_R^{(n)}$  on whether the primary infective is sampled or not, so

$$\mathbb{E} [\tilde{C}_R^{(n)}] = \tilde{p}_{SU}^D \mathbb{E} [\tilde{C}_{RS}^{(n)}] + (1 - \tilde{p}_{SU}^D) \mathbb{E} [\tilde{C}_{RN}^{(n)}], \quad (7.20)$$

where  $\tilde{C}_{RS}^{(n)}$  and  $\tilde{C}_{RN}^{(n)}$  are the random variable  $\tilde{C}_R^{(n)}$  conditioned on the primary infective being sampled and unsampled respectively. We then decompose  $\tilde{C}_{RS}^{(n)}$  and  $\tilde{C}_{RN}^{(n)}$  into the number of contacts made by each member of the primary infective's rewired household epidemic, were they to become infected, along with an indicator random variable indicating whether each individual becomes infected. Furthermore, by analogous arguments to those leading to equation (7.15), the event that an individual  $i$  is infected is independent of how many contacts  $i$  would make if  $i$  became infected, and the offspring of each secondary infective in the rewired household epidemic are identically distributed. Thus

$$\mathbb{E} [\tilde{C}_{RS}^{(n)}] = \mathbb{E} [\tilde{C}_{RS}^{(n)}(0)] + \mathbb{E} [\hat{T}_S^{(n)}] \mathbb{E} [\tilde{C}_{RSU}(1)], \quad (7.21a)$$

$$\mathbb{E} [\tilde{C}_{RN}^{(n)}] = \mathbb{E} [\tilde{C}_{RN}^{(n)}(0)] + \mathbb{E} [\hat{T}_N^{(n)}] \mathbb{E} [\tilde{C}_{RSU}(1)], \quad (7.21b)$$

where:  $\mathbb{E} [\tilde{C}_{RS}^{(n)}(0)]$  and  $\mathbb{E} [\tilde{C}_{RN}^{(n)}(0)]$  are the expected number of infectious network contacts made by a primary infective that is sampled and unsampled respectively,  $\mathbb{E} [\tilde{C}_{RSU}(1)]$  is the expected number of infectious network contacts made by a secondary infective in the rewired household epidemic, and  $\mathbb{E} [\hat{T}_S^{(n)}]$  ( $\mathbb{E} [\hat{T}_N^{(n)}]$ ) is the expected final size of a rewired household epidemic with household size label  $n$  in which the primary infective is sampled (unsampled).

Furthermore, noting that an individual contacted via the network is sampled with probability  $\tilde{p}_{SU}^D$  and otherwise unsampled and applying analogous arguments to those leading to equation (7.17),

$$\mathbb{E} [\tilde{C}_{RSU}(1)] = \mathbb{E} [D_U] p_N (1 - \tilde{p}_{SU}^D p_{CG}) (1 - \tilde{p}_V), \quad (7.22a)$$

$$\mathbb{E} [\tilde{C}_{RS}^{(n)}(0)] = \mathbb{E} [\tilde{D}_U - 1] p_N (1 - p_{CG}) (1 - \tilde{p}_V), \quad (7.22b)$$

$$\mathbb{E} [\tilde{C}_{RN}^{(n)}(0)] = \mathbb{E} [\tilde{D}_U - 1] p_N (1 - \tilde{p}_V). \quad (7.22c)$$

Substituting equations (7.22) into equations (7.21) and the result into equation (7.20) yields

$$\begin{aligned} \mathbb{E} [\tilde{C}_R^{(n)}] &= \tilde{p}_{SU}^D \mathbb{E} [\tilde{C}_{RS}^{(n)}] + (1 - \tilde{p}_{SU}^D) \mathbb{E} [\tilde{C}_{RN}^{(n)}] \\ &= p_N (1 - \tilde{p}_V) (1 - \tilde{p}_{SU}^D p_{CG}) \mathbb{E} [\tilde{D}_U - 1] \\ &\quad + p_N (1 - \tilde{p}_V) (1 - \tilde{p}_{SU}^D p_{CG}) \mathbb{E} [D_U] \mu_{\hat{T}^{(n)}}, \end{aligned} \quad (7.23)$$

where  $\mu_{\hat{T}^{(n)}}$  is given in equation (7.11b).

Finally, substituting equations (7.19) and (7.23) into equation (7.12) yields  $R_*^A$  as given in the statement of the theorem.  $\square$

In Section 5.1.4 we analytically investigate the trade-off between  $p_S$  and  $p_C$  for a fixed vaccination coverage in the network and global model under the acquaintance vaccination strategy. We have not carried out a similar analytic investigation for the trade-off between  $p_S$  and  $p_C$  for a fixed vaccination coverage in the clustered network model, however we conjecture that  $R_*^A$  is strictly increasing in  $p_C$ .

## 7.2.4 Can the acquaintance vaccination strategy control the epidemic?

An important question to consider in evaluating vaccination strategies is whether the vaccination strategy can prevent a major outbreak occurring without vaccinating every individual in the population and therefore control the epidemic. In this section we show that  $R_*^A$  can be made arbitrarily small by choosing appropriate  $p_S \in (0, 1)$  and  $p_{CH} \in (0, 1)$ , so the acquaintance vaccination strategy can control the epidemic.

**Proposition 7.5.** *If  $p_{CH} > 0$ ,  $p_{CG} > 0$  and  $p_S > 0$  then the acquaintance vaccination strategy can control the epidemic in the clustered network model.*

*Proof.* Note that  $\sum_{\alpha=0}^{n-1} \tilde{p}_{SU}^{(n,\alpha)} \mu_{T(n,\alpha)}$ ,  $n = 1, 2, \dots$ , is a weighted sum of the number of sampled and unsampled members of a household epidemic within a household of size  $n$  and is therefore bounded from above by  $n$ . Thus applying the inequalities  $p_N < 1$ ,  $p_{CG} \leq 1$ ,  $\tilde{p}_{SU}^D < 1$ ,  $\tilde{p}_{SU}^H < 1$  and  $\sum_{\alpha=0}^{n-1} \tilde{p}_{SU}^{(n,\alpha)} \mu_{T(n,\alpha)} < n$  to  $R_*^U$  given in Theorem 7.1 yields the inequality

$$R_*^A \leq (1 - \tilde{p}_V) \left\{ \mathbb{E} [D_U] \sum_{n=1}^{\infty} \tilde{\rho}_n^U \left( \tilde{p}_{SU}^D \mathbb{E} [\hat{T}_S^{(n)}] + (1 - \tilde{p}_{SU}^D) \mathbb{E} [\hat{T}_N^{(n)}] + n \right) + \mathbb{E} [\tilde{D}_U - 1] \right\}. \quad (7.24)$$

Recall from Proposition 7.2 that  $\mathbb{E} [\hat{T}_N^{(1)}] = 0$ ,  $\mathbb{E} [\hat{T}_N^{(2)}] = p_H(1 - p_{VG}) < 1$ , for  $n \geq 3$ ,

$$\mathbb{E} [\hat{T}_N^{(n)}] = \begin{cases} \frac{(n-1)(1-p_{VG})(1-\tilde{p}_{SU}^H p_{CH})^{n-2} p_H}{1-(n-2)\tilde{p}_H(n)} & \text{if } \tilde{p}_H(n) < \frac{1}{n-2}, \\ \infty & \text{if } \tilde{p}_H(n) \geq \frac{1}{n-2}, \end{cases}$$



and, for  $n = 1, 2, \dots$ ,

$$\mathbb{E} [\hat{T}_S^{(n)}] = (1 - p_{CH})\mathbb{E} [\hat{T}_N^{(n)}] \leq \mathbb{E} [\hat{T}_N^{(n)}],$$

where  $\tilde{p}_H(n) = (1 - p_{VG})(1 - \tilde{p}_{SU}^H p_{CH})^{n-1} p_H$ .

Note that  $\tilde{p}_H(n) < (1 - \tilde{p}_{SU}^H p_{CH})^{n-1}$  and  $\lim_{n \rightarrow \infty} (n-2)(1 - \tilde{p}_{SU}^H p_{CH})^{n-1} = 0$ . Therefore, by ensuring the stationary point of  $(n-2)(1 - \tilde{p}_{SU}^H p_{CH})^{n-1}$  occurs at  $n \leq 3$ , we can choose a value of  $\tilde{p}_{SU}^H p_{CH}$ ,  $p^*$  say, such that for all  $n = 3, 4, \dots$ ,  $\tilde{p}_H(n) < (1 - \tilde{p}_{SU}^H p_{CH})^{n-1} < 1/(n-2)$  and  $\tilde{p}_{SU}^H p_{CH} > p^*$ . Furthermore, if  $\tilde{p}_H(n) < 1/(n-2)$  then for all  $n = 3, 4, \dots$  there exists a constant  $K > 0$  such that  $1 - (n-2)\tilde{p}_H(n) > K$ . Therefore, for  $\tilde{p}_{SU}^H p_{CH} > p^* > 1 - e^{-1}$  and  $n \geq 3$ ,

$$\begin{aligned} \mathbb{E} [\hat{T}_S^{(n)}] &\leq \mathbb{E} [\hat{T}_N^{(n)}] = \frac{(n-1)(1 - p_{VG})(1 - \tilde{p}_{SU}^H p_{CH})^{n-2} p_H}{1 - (n-2)\tilde{p}_H(n)} \\ &< \frac{(n-1)(1 - \tilde{p}_{SU}^H p_{CH})^{n-2}}{K}. \end{aligned} \quad (7.25)$$

Applying inequality (7.25) to inequality (7.24) yields

$$R_*^A \leq (1 - \tilde{p}_V) \left\{ \mathbb{E} [D_U] \sum_{n=3}^{\infty} \tilde{\rho}_n^U \left( \frac{2(n-1)(1 - \tilde{p}_{SU}^H p_{CH})^{n-2}}{K} + n \right) + \mathbb{E} [\tilde{D}_U - 1] \right\} \quad (7.26)$$

We assume  $\sigma_H^2 < \infty$  and  $\sigma_D^2 < \infty$ , so  $\sum_{n=3}^{\infty} \tilde{\rho}_n(n-1)(1 - \tilde{p}_{SU}^H p_{CH})^{n-2} < \infty$  by the ratio test and  $\sum_{n=3}^{\infty} \tilde{\rho}_n^U n \leq \mathbb{E} [\tilde{H}_U]$ . Therefore, for  $\tilde{p}_{SU}^H p_{CH} > p^*$ , there exists a constant  $K^*$  such that  $R_*^A \leq (1 - \tilde{p}_V)K^*$ . So  $R_*^A$  can be made arbitrarily small by choosing appropriate  $p_S$ ,  $p_{CH}$  and  $p_{CG}$  as required.  $\square$

The proof of Proposition 7.5 requires  $p_{CH} > 0$  to control the spread of the epidemic through large rewired households. Indeed, we note that the vaccination strategy investigated by Ball and Sirl (2017) (i.e.  $p_{CH} = 0$ ) cannot control the spread of the epidemic in the clustered network model if for all  $k \in \mathbb{Z}^+$  there exists an  $n \geq k$  such that  $\rho_n p_{RW}(n) > 0$ .

### 7.2.5 Final size of a major outbreak

Let  $B$  be the offspring random variable for the initial individual in the backwards branching process and let  $\tilde{B}$  be the offspring random variable for subsequent generations in the backwards branching process. Let  $b(s)$  and  $\tilde{b}(s)$  be the probability generating functions of  $B$  and  $\tilde{B}$  respectively. Then the final size

of a major outbreak is  $z = 1 - b(\pi)$ , where  $\pi$  is the smallest solution to the equation  $\pi = \tilde{b}(\pi)$ . Before giving the probability generating functions  $b(s)$  and  $\tilde{b}(s)$  we introduce the following notation. Let

$$G_{\tilde{D}_U}^S(s) = f_{\tilde{D}_U-1}(1 - p_N(1 - p_{CG})(1 - \tilde{p}_V)(1 - s)), \quad (7.27a)$$

$$G_{\tilde{D}_U}^N(s) = f_{\tilde{D}_U-1}(1 - p_N(1 - \tilde{p}_V)(1 - s)), \quad (7.27b)$$

$$G_{D_U}^S(s) = f_{D_U}(1 - p_N(1 - p_{CG})(1 - \tilde{p}_V)(1 - s)), \quad (7.27c)$$

$$G_{D_U}^N(s) = f_{D_U}(1 - p_N(1 - \tilde{p}_V)(1 - s)), \quad (7.27d)$$

$$G_{D_U}^{SU}(s) = f_{D_U}\left(1 - p_N(1 - \tilde{p}_{SU}^H p_{CG})(1 - \tilde{p}_V)(1 - s)\right), \quad (7.27e)$$

$$\mathbf{G}_{D_U}(s) = (G_{D_U}^S(s), G_{D_U}^N(s)). \quad (7.27f)$$

**Theorem 7.2.** *The probability generating functions for the initial and subsequent offspring distributions of the backward Galton-Watson branching process are given by*

$$\begin{aligned} b(s) = \sum_{n=1}^{\infty} \tilde{\rho}_n^U \Bigg\{ & (1 - p_{RW}(n)) \sum_{\alpha=0}^{n-1} \tilde{p}_{SU}^{(n,\alpha)} \left[ p_S G_{\tilde{D}_U}^S(s) f_{M_S}^{(n,\alpha)}(\mathbf{G}_{D_U}(s)) \right. \\ & \left. + (1 - p_S) G_{\tilde{D}_U}^N(s) f_{M_N}^{(n,\alpha)}(\mathbf{G}_{D_U}(s)) \right] \\ & + p_{RW}(n) \left[ p_S G_{\tilde{D}_U}^S(s) f_{\hat{M}_S^{(n)}}(G_{D_U}^{SU}(s)) \right. \\ & \left. + (1 - p_S) G_{\tilde{D}_U}^N(s) f_{\hat{M}_N^{(n)}}(G_{D_U}^{SU}(s)) \right] \Bigg\} \end{aligned}$$

and

$$\begin{aligned} \tilde{b}(s) = \sum_{n=1}^{\infty} \tilde{\rho}_n^U \Bigg\{ & (1 - p_{RW}(n)) \sum_{\alpha=0}^{n-1} \tilde{p}_{SU}^{(n,\alpha)} \left[ \tilde{p}_{SU}^D G_{\tilde{D}_U}^S(s) f_{M_S}^{(n,\alpha)}(\mathbf{G}_{D_U}(s)) \right. \\ & \left. + (1 - \tilde{p}_{SU}^D) G_{\tilde{D}_U}^N(s) f_{M_N}^{(n,\alpha)}(\mathbf{G}_{D_U}(s)) \right] \\ & + p_{RW}(n) \left[ \tilde{p}_{SU}^D G_{\tilde{D}_U}^S(s) f_{\hat{M}_S^{(n)}}(G_{D_U}^{SU}(s)) \right. \\ & \left. + (1 - \tilde{p}_{SU}^D) G_{\tilde{D}_U}^N(s) f_{\hat{M}_N^{(n)}}(G_{D_U}^{SU}(s)) \right] \Bigg\}, \end{aligned}$$

where  $f_{M_S}^{(n,\alpha)}(s_1, s_2)$  and  $f_{M_N}^{(n,\alpha)}(s_1, s_2)$  are defined below Proposition 7.3 and  $f_{\hat{M}_S^{(n)}}(s)$  and  $f_{\hat{M}_N^{(n)}}(s)$  are given in Proposition 7.4.

*Proof.* We first consider the calculation of  $\tilde{b}(s)$  and begin by conditioning on the household size label of the unvaccinated primary individual, distributed according to  $\tilde{H}_U$ , and whether the primary individual is in a household or rewired

household. Households of size  $n$  are rewired independently and uniformly at random with probability  $p_{RW}(n)$ , so

$$\mathbb{E} [s^{\tilde{B}}] = \sum_{n=1}^{\infty} \tilde{\rho}_n^U \left[ (1 - p_{RW}(n)) \mathbb{E} [s^{\tilde{B}_H^{(n)}}] + p_{RW}(n) \mathbb{E} [s^{\tilde{B}_R^{(n)}}] \right], \quad (7.29)$$

where  $\tilde{B}_H^{(n)}$  and  $\tilde{B}_R^{(n)}$  are the random variable  $\tilde{B}$  conditioned on the primary individual being in a household of size  $n$  and a rewired household with household size label  $n$  respectively.

We now consider  $\mathbb{E} [s^{\tilde{B}_H^{(n)}}]$  by conditioning on the number of sampled non-primary individuals in the household and whether the primary individual is sampled or not. The primary individual is sampled with probability  $\tilde{p}_{SU}^D$  and otherwise unsampled and with probability  $\tilde{p}_{SU}^{(n,\alpha)}$ ,  $\alpha = 0, 1, \dots, n-1$ , there are  $\alpha$  secondary sampled individuals within a household of size  $n$ . So

$$\mathbb{E} [s^{\tilde{B}_H^{(n)}}] = \sum_{\alpha=0}^{n-1} \tilde{p}_{SU}^{(n,\alpha)} \left[ \tilde{p}_{SU}^D \mathbb{E} [s^{\tilde{B}_{HS}^{(n,\alpha)}}] + (1 - \tilde{p}_{SU}^D) \mathbb{E} [s^{\tilde{B}_{HN}^{(n,\alpha)}}] \right], \quad (7.30)$$

where  $\tilde{B}_{HS}^{(n,\alpha)}$  ( $\tilde{B}_{HN}^{(n,\alpha)}$ ) is the quantity  $\tilde{B}_H^{(n)}$  conditioned on the primary individual being sampled (unsampled) and the household containing  $\alpha$  sampled non-primary individuals.

Next we decompose  $\tilde{B}_{HS}^{(n,\alpha)}$  and  $\tilde{B}_{HN}^{(n,\alpha)}$  into the number of contacts made to each member of the primary individual's household susceptibility set. Label the primary infective 0. Then if the primary individual is sampled we label the sampled and unsampled members of the primary individual's household susceptibility set  $1, 2, \dots, M_{SS}^{(n,\alpha)}$  and  $1, 2, \dots, M_{SN}^{(n,\alpha)}$  respectively. If the primary individual is unsampled we label the sampled and unsampled members of the primary individual's household susceptibility set  $1, 2, \dots, M_{NS}^{(n,\alpha)}$  and  $1, 2, \dots, M_{NN}^{(n,\alpha)}$  respectively. Thus

$$\tilde{B}_{HS}^{(n,\alpha)} = \tilde{B}_{HS}^{(n,\alpha)}(0) + \sum_{i=1}^{M_{SS}^{(n,\alpha)}} \tilde{B}_{HS}^{(n,\alpha,S)}(i) + \sum_{i=1}^{M_{SN}^{(n,\alpha)}} \tilde{B}_{HS}^{(n,\alpha,N)}(i), \quad (7.31a)$$

$$\tilde{B}_{HN}^{(n,\alpha)} = \tilde{B}_{HN}^{(n,\alpha)}(0) + \sum_{i=1}^{M_{NS}^{(n,\alpha)}} \tilde{B}_{HN}^{(n,\alpha,S)}(i) + \sum_{i=1}^{M_{NN}^{(n,\alpha)}} \tilde{B}_{HN}^{(n,\alpha,N)}(i), \quad (7.31b)$$

where:  $\tilde{B}_{HS}^{(n,\alpha)}(0)$  ( $\tilde{B}_{HN}^{(n,\alpha)}(0)$ ) is the number of contacts made to the primary in-

dividual,  $\tilde{B}_{HS}^{(n,\alpha,S)}(i)$  ( $\tilde{B}_{HN}^{(n,\alpha,S)}(i)$ ) is the number of contacts made to the sampled non-primary individual  $i$  and  $\tilde{B}_{HS}^{(n,\alpha,N)}(i)$  ( $\tilde{B}_{HN}^{(n,\alpha,N)}(i)$ ) is the number of contacts made to the unsampled secondary individual  $i$ .

Consider a primary individual  $i^*$  in a household of size  $n$ . By analogous arguments to those leading to equation (3.32), all infectious contacts made to the members of  $i^*$ 's household susceptibility set are made by individuals with independent and identically distributed infectious periods,  $I$ . Furthermore, the number of contacts made to each member of  $i^*$ 's household susceptibility set are independent so the summands in equation (7.31a) are all mutually independent, and also independent of  $M_{A_1 A_2}^{(n,\alpha)}$ ,  $A_1, A_2 \in \{S, N\}$ . Furthermore, applying analogous arguments to those leading to equation (5.11) yields

$$\begin{aligned}\tilde{B}_{HS}^{(n,\alpha)}(0)|\tilde{D}_U &\sim \text{Bin}(\tilde{D}_U - 1, p_N(1 - p_{CG})(1 - \tilde{p}_V)), \\ \tilde{B}_{HN}^{(n,\alpha)}(0)|\tilde{D}_U &\sim \text{Bin}(\tilde{D}_U - 1, p_N(1 - \tilde{p}_V)), \\ \tilde{B}_{HA}^{(n,\alpha,S)}(1)|D_U &\sim \text{Bin}(D_U, p_N(1 - p_{CG})(1 - \tilde{p}_V)), \\ \tilde{B}_{HA}^{(n,\alpha,N)}(1)|D_U &\sim \text{Bin}(D_U, p_N(1 - \tilde{p}_V)),\end{aligned}$$

and, applying the notation from equation (7.27),

$$\mathbb{E} \left[ s^{\tilde{B}_{HS}^{(n,\alpha)}(0)} \middle| \tilde{D}_U \right] = G_{\tilde{D}_U}^S(s), \quad (7.33a)$$

$$\mathbb{E} \left[ s^{\tilde{B}_{HN}^{(n,\alpha)}(0)} \middle| \tilde{D}_U \right] = G_{\tilde{D}_U}^N(s), \quad (7.33b)$$

$$\mathbb{E} \left[ s^{\tilde{B}_{HA}^{(n,\alpha,S)}(1)} \middle| D_U \right] = G_{D_U}^S(s), \quad (7.33c)$$

$$\mathbb{E} \left[ s^{\tilde{B}_{HA}^{(n,\alpha,N)}(1)} \middle| D_U \right] = G_{D_U}^N(s). \quad (7.33d)$$

Considering the probability generating function of  $\tilde{B}_{HS}^{(n,\alpha)}$ , given in equation (7.31a), applying the independence of  $\tilde{B}_{HS}^{(n,\alpha)}(0)$ ,  $\tilde{B}_{HS}^{(n,\alpha,N)}(i)$  and  $\tilde{B}_{HS}^{(n,\alpha,S)}(j)$ , conditioning on  $M_{SS}^{(n,\alpha)}$  and  $M_{SN}^{(n,\alpha)}$  and the appropriate degree distributions and

substituting equations (7.33) yields

$$\begin{aligned}
\mathbb{E} \left[ s^{\tilde{B}_{HS}^{(n,\alpha)}} \right] &= \mathbb{E} \left[ s^{\tilde{B}_{HS}^{(n,\alpha)}(0)} \prod_{i=1}^{M_{SS}^{(n,\alpha)}} s^{\tilde{B}_{HS}^{(n,\alpha,S)}(i)} \prod_{i=1}^{M_{SN}^{(n,\alpha)}} s^{\tilde{B}_{HS}^{(n,\alpha,N)}(i)} \right] \\
&= \mathbb{E} \left[ s^{\tilde{B}_{HS}^{(n,\alpha)}(0)} \right] \mathbb{E} \left[ \mathbb{E} \left[ \prod_{i=1}^{M_{SS}^{(n,\alpha)}} s^{\tilde{B}_{HS}^{(n,\alpha,S)}(1)} \prod_{i=1}^{M_{SN}^{(n,\alpha)}} s^{\tilde{B}_{HS}^{(n,\alpha,N)}(1)} \middle| M_{SS}^{(n,\alpha)}, M_{SN}^{(n,\alpha)} \right] \right] \\
&= \mathbb{E} \left[ s^{\tilde{B}_{HS}^{(n,\alpha)}(0)} \right] \mathbb{E} \left[ \mathbb{E} \left[ s^{\tilde{B}_{HS}^{(n,\alpha,S)}(1)} \right]^{M_{SS}^{(n,\alpha)}} \mathbb{E} \left[ s^{\tilde{B}_{HS}^{(n,\alpha,N)}(1)} \right]^{M_{SN}^{(n,\alpha)}} \right] \\
&= G_{\tilde{D}_U}^S(s) \mathbb{E} \left[ \left[ G_{\tilde{D}_U}^S(s) \right]^{M_{SS}^{(n,\alpha)}} \left[ G_{\tilde{D}_U}^N(s) \right]^{M_{SN}^{(n,\alpha)}} \right] \tag{7.34a}
\end{aligned}$$

Similarly, considering the probability generating function of  $\tilde{B}_{HN}^{(n,\alpha)}$ , given in equation (7.31b), applying the independence of  $\tilde{B}_{HN}^{(n,\alpha)}(0)$ ,  $\tilde{B}_{HN}^{(n,\alpha,N)}(i)$  and  $\tilde{B}_{HN}^{(n,\alpha,S)}(j)$ , conditioning on  $M_{NS}^{(n,\alpha)}$  and  $M_{NN}^{(n,\alpha)}$  and the appropriate degree distributions and substituting equations (7.33) yields

$$\mathbb{E} \left[ s^{\tilde{B}_{HN}^{(n,\alpha)}} \right] = G_{\tilde{D}_U}^N(s) \mathbb{E} \left[ \left[ G_{\tilde{D}_U}^S(s) \right]^{M_{NS}^{(n,\alpha)}} \left[ G_{\tilde{D}_U}^N(s) \right]^{M_{NN}^{(n,\alpha)}} \right]. \tag{7.34b}$$

Recall that, for  $A \in \{S, N\}$ , the joint distribution of  $(M_{(AS)}^{(n,\alpha)}, M_{(AN)}^{(n,\alpha)})$  is given in Proposition 7.3 and, for  $A \in \{S, N\}$ ,  $f_{M_A}^{(n,\alpha)}(s_1, s_2) = \mathbb{E} \left[ s_1^{M_{(AS)}^{(n,\alpha)}} s_2^{M_{(AN)}^{(n,\alpha)}} \right]$ . Substituting  $f_{M_A}^{(n,\alpha)}(s_1, s_2)$  into equations (7.34) and recalling from equations (7.27) that  $\mathbf{G}_{\tilde{D}_U}(s) = (G_{\tilde{D}_U}^S(s), G_{\tilde{D}_U}^N(s))$  yields

$$\mathbb{E} \left[ s^{\tilde{B}_{HS}^{(n,\alpha)}} \right] = G_{\tilde{D}_U}^S(s) f_{M_S}^{(n,\alpha)}(\mathbf{G}_{\tilde{D}_U}(s)), \tag{7.35a}$$

$$\mathbb{E} \left[ s^{\tilde{B}_{HN}^{(n,\alpha)}} \right] = G_{\tilde{D}_U}^N(s) f_{M_N}^{(n,\alpha)}(\mathbf{G}_{\tilde{D}_U}(s)). \tag{7.35b}$$

Substituting equations (7.35) into equation (7.30) yields

$$\begin{aligned}
\mathbb{E} \left[ s^{\tilde{B}_H^{(n)}} \right] &= \sum_{\alpha=0}^{n-1} \tilde{p}_{SU}^{(n,\alpha)} \left[ \tilde{p}_{SU}^D G_{\tilde{D}_U}^S(s) f_{M_S}^{(n,\alpha)}(\mathbf{G}_{\tilde{D}_U}(s)) \right. \\
&\quad \left. + (1 - \tilde{p}_{SU}^D) G_{\tilde{D}_U}^N(s) f_{M_N}^{(n,\alpha)}(\mathbf{G}_{\tilde{D}_U}(s)) \right]. \tag{7.36}
\end{aligned}$$

To complete our calculation of  $\tilde{b}(s)$  we need only calculate  $\mathbb{E} \left[ s^{\tilde{B}_R^{(n)}} \right]$ ,  $n = 1, 2, \dots$ , i.e. the probability generating function for the number of network

contacts made to the rewired household susceptibility set of an individual with household size label  $n$ . Applying a similar decomposition to that leading to equations (7.30) and (7.31) yields

$$\tilde{B}_R^{(n)} = \tilde{p}_{SU}^D \left( \tilde{B}_{RS}^{(n)}(0) + \sum_{i=1}^{\hat{M}_S^{(n)}} \tilde{B}_{RS}^{(n)}(i) \right) + (1 - \tilde{p}_{SU}^D) \left( \tilde{B}_{RN}^{(n)}(0) + \sum_{i=1}^{\hat{M}_N^{(n)}} \tilde{B}_{RN}^{(n)}(i) \right), \quad (7.37)$$

where  $\tilde{B}_{RS}^{(n)}(0)$  ( $\tilde{B}_{RN}^{(n)}(0)$ ) is the number of contacts made to the primary individual,  $\tilde{B}_{RS}^{(n)}(i)$  ( $\tilde{B}_{RN}^{(n)}(i)$ ) is the number of contacts made to non-primary individual  $i$  and  $\hat{M}_S^{(n)}$  and  $\hat{M}_N^{(n)}$  are respectively the total number of sampled and unsampled members of the primary individual's rewired household susceptibility set.

By analogous arguments to those leading to equation (7.33), the summands in equation (7.37) are all mutually independent and also independent of  $\hat{M}_S^{(n)}$  and  $\hat{M}_N^{(n)}$ . Furthermore, applying similar arguments to those leading to equation (7.33) and the notation from equation (7.27),

$$\mathbb{E} \left[ s^{\tilde{B}_{RS}^{(n)}(0)} \middle| \tilde{D}_U \right] = G_{\tilde{D}_U}^S(s), \quad (7.38a)$$

$$\mathbb{E} \left[ s^{\tilde{B}_{RN}^{(n)}(0)} \middle| \tilde{D}_U \right] = G_{\tilde{D}_U}^N(s), \quad (7.38b)$$

$$\mathbb{E} \left[ s^{\tilde{B}_{RA}^{(n)}(i)} \middle| D_U \right] = G_{D_U}^{SU}(s). \quad (7.38c)$$

Thus applying the independence of  $\tilde{B}_{RA}^{(n)}(0)$ ,  $\tilde{B}_{RA}^{(n)}(i)$  and  $\tilde{B}_{RA}^{(n)}(j)$ ,  $i \neq j$ , to the probability generating function of equation (7.37), conditioning on  $\hat{M}_S^{(n)}$  and  $\hat{M}_N^{(n)}$  and the appropriate degree distributions and substituting equations (7.38) yields

$$\begin{aligned} \mathbb{E} \left[ s^{\tilde{B}_R^{(n)}} \right] &= \tilde{p}_{SU}^D \mathbb{E} \left[ s^{\tilde{B}_{RS}^{(n)}(0)} \prod_{i=1}^{\hat{M}_S^{(n)}} s^{\tilde{B}_{RS}^{(n)}(i)} \right] + (1 - \tilde{p}_{SU}^D) \mathbb{E} \left[ s^{\tilde{B}_{RN}^{(n)}(0)} \prod_{i=1}^{\hat{M}_N^{(n)}} s^{\tilde{B}_{RN}^{(n)}(i)} \right] \\ &= \tilde{p}_{SU}^D \mathbb{E} \left[ s^{\tilde{B}_{RS}^{(n)}(0)} \right] \mathbb{E} \left[ \mathbb{E} \left[ \prod_{i=1}^{\hat{M}_S^{(n)}} s^{\tilde{B}_{RS}^{(n)}(i)} \middle| \hat{M}_S^{(n)} \right] \right] \\ &\quad + (1 - \tilde{p}_{SU}^D) \mathbb{E} \left[ s^{\tilde{B}_{RN}^{(n)}(0)} \right] \mathbb{E} \left[ \mathbb{E} \left[ \prod_{i=1}^{\hat{M}_N^{(n)}} s^{\tilde{B}_{RN}^{(n)}(i)} \middle| \hat{M}_N^{(n)} \right] \right] \\ &= \tilde{p}_{SU}^D G_{\tilde{D}_U}^S(s) \mathbb{E} \left[ \left( G_{D_U}^{SU}(s) \right)^{\hat{M}_S^{(n)}} \right] + (1 - \tilde{p}_{SU}^D) G_{\tilde{D}_U}^N(s) \mathbb{E} \left[ \left( G_{D_U}^{SU}(s) \right)^{\hat{M}_N^{(n)}} \right]. \end{aligned} \quad (7.39)$$

Recall that the probability generating functions for  $\hat{M}_S^{(n)}$  and  $\hat{M}_N^{(n)}$ , denoted by  $f_{\hat{M}_S^{(n)}}(s)$  and  $f_{\hat{M}_N^{(n)}}(s)$  respectively, are given in Proposition 7.4 which, substituted into equation (7.39), yields

$$\mathbb{E} \left[ s^{\tilde{B}_R^{(n)}} \right] = \tilde{p}_{SU}^D G_{\tilde{D}_U}^S(s) f_{\hat{M}_S^{(n)}}(G_{D_U}^{SU}(s)) + (1 - \tilde{p}_{SU}^D) G_{\tilde{D}_U}^N(s) f_{\hat{M}_N^{(n)}}(G_{D_U}^{SU}(s)). \quad (7.40)$$

Substituting equations (7.36) and (7.40) into equation (7.29) yields  $\tilde{b}(s)$  as given in the statement of the theorem.

The calculation of  $b(s)$  follows analogous arguments to the calculation of  $\tilde{b}(s)$ , noting that the primary individual is sampled with probability  $p_S$  instead of  $\tilde{p}_{SU}^D$  and has degree distribution  $D_U$  instead of  $\tilde{D}_U$ . Therefore making the appropriate substitutions into equations (7.30), (7.33), (7.37) and (7.38) yields  $b(s)$  as required.  $\square$

Recall that an acquaintance vaccination strategy on the household and network model is investigated by Ball and Sirl (2017), differing from our acquaintance vaccination strategy in that Ball and Sirl (2017) do now allow sampled individuals to choose household neighbours for vaccination. The key differences in extending the acquaintance vaccination strategy to allow for vaccinating household neighbours as well as network neighbours is the dependence of the following: the event that a primary individual is unvaccinated, the number of sampled secondary individuals within the household and the number of vaccinated sampled and unsampled individuals within the household. Unfortunately the resulting expression for the number of sampled and unsampled individuals within a household susceptibility set relies on the hypergeometric distribution, and consequently has no known simple closed form expression. Therefore we note that if  $p_{RW}(n) < 1$ ,  $n = 1, 2, \dots$ , then the acquaintance vaccination strategy with  $p_{CH} > 0$  is more computationally intensive than the acquaintance vaccination strategy with  $p_{CH} = 0$ .

## 7.3 Uniform vaccination on the clustered network model

### 7.3.1 Description of uniform vaccination

Under the uniform vaccination strategy each individual in the population is vaccinated with the perfect vaccine independently with probability  $p_V$ , so the vaccination coverage  $c = p_V$ . Similarly to the calculations for the acquaintance vaccination strategy discussed in Section 7.2 we consider household-based single-type forward and backward branching processes to calculate the threshold parameter and the final size of a major outbreak.

### 7.3.2 Threshold parameter

The threshold parameter  $R_*^U$  is given in Theorem 7.3 below.

**Theorem 7.3.**

$$R_*^U = p_N(1 - p_V)E[D] \sum_{n=1}^{\infty} \tilde{\rho}_n \left\{ (1 - p_{RW}(n)) \left[ \sum_{v=0}^n \binom{n}{v} p_V^v (1 - p_V)^{n-v} \mu_{T(n-v)} \right] + p_{RW}(n) \mu_{\hat{T}(n)} \right\} + p_N(1 - p_V)E[\tilde{D} - 1],$$

where  $\mu_{T(n)}$ , given in equation (2.5), is the expected final size of the household epidemic amongst the secondary individuals in a household of size  $n$  and

$$\begin{aligned} \mu_{\hat{T}(1)} &= 0, \\ \mu_{\hat{T}(2)} &= p_H(1 - p_V), \end{aligned}$$

and, for  $n = 3, 4, \dots$ ,

$$\mu_{\hat{T}(n)} = \begin{cases} \frac{(n-1)p_H(1-p_V)}{1-(n-2)p_H(1-p_V)} & \text{if } p_H(1 - p_V) < \frac{1}{n-2}, \\ \infty & \text{if } p_H(1 - p_V) \geq \frac{1}{n-2}, \end{cases}$$

is the expected final size of a rewired household epidemic conditioned on the primary infective having household size label  $n$ .

Note that if  $p_{RW}(n) = 0$ ,  $n = 1, 2, \dots$ , we recover the household and network model with individuals chosen uniformly at random for vaccination discussed in, for example, Ball and Sirl (2017). Furthermore, the spread of a rewired



household epidemic within a household of size  $n$  can be approximated by the spread of the epidemic on the standard network model in which every individual has degree  $n$  and is vaccinated independently with probability  $p_V$ , which is a special case of Section 5.2.3. Therefore, since the investigation of the clustered network model can be considered as an amalgamation of well-studied models, we omit the proof of Theorem 7.3.

### 7.3.3 Can the uniform vaccination strategy control the epidemic?

Similarly to Section 7.2.4 we now investigate whether the uniform vaccination strategy can control the epidemic. We show that the uniform vaccination strategy can prevent a major outbreak occurring with  $p_V < 1$  only if the household size distribution has finite support or there exists a  $k \in \mathbb{Z}^+$  such that  $\rho_n p_{RW}(n) = 0$  for all  $n \geq k$ .

**Proposition 7.6.** *If the household size distribution has finite support or if there exists a  $k \in \mathbb{Z}^+$  such that  $\rho_n p_{RW}(n) = 0$  for all  $n \geq k$  then the uniform vaccination strategy can control the epidemic in the clustered network model. Otherwise, the uniform vaccination strategy cannot control the epidemic in the clustered network model.*

*Proof.* Recall that  $\mu_{T(n)}$  is the expected final size of the household epidemic amongst the secondary individuals in a household of size  $n$  so  $\mu_{T(n-i)} < n$ ,  $i = 0, 1, \dots, n-1$ . Therefore

$$\sum_{v=0}^n \binom{n}{v} p_V^v (1-p_V)^{n-v} \mu_{T(n-v)} < \sum_{v=0}^n \binom{n}{v} p_V^v (1-p_V)^{n-v} n = n. \quad (7.42)$$

Substituting inequality (7.42) into  $R_*^U$ , given in Theorem 7.3, yields

$$R_*^U \leq (1-p_V) \left\{ \mathbb{E}[D] \sum_{n=1}^{\infty} \tilde{\rho}_n (n + \mu_{\hat{T}(n)}) + \mathbb{E}[\tilde{D} - 1] \right\}. \quad (7.43)$$

Now consider  $\mu_{\hat{T}(n)}$ . Recall from Theorem 7.3 that  $\mu_{\hat{T}(n)}$  is the expected final size of a rewired household epidemic conditioned on the primary infective having household size label  $n$  and, for  $n \geq 3$ ,

$$\mu_{\hat{T}(n)} = \begin{cases} (n-1)p_H(1-p_V) (1 - (n-2)p_H(1-p_V))^{-1} & \text{if } p_H(1-p_V) < \frac{1}{n-2}, \\ \infty & \text{if } p_H(1-p_V) \geq \frac{1}{n-2}. \end{cases}$$

If  $H$  has finite support or there exists a  $k \in \mathbb{Z}^+$  such that  $\rho_n p_{RW}(n) = 0$  for all  $n \geq k$  then  $\sum_{n=1}^{\infty} \tilde{\rho}_n (n + \mu_{\hat{T}(n)}) < \infty$  and, since  $E[H^2]E[D^2] < \infty$ , there exists a constant  $K$  such that  $R_*^U < K(1 - p_V)$ . Therefore we can choose a value of  $p_V < 1$  such that  $R_*^U < 1$  and the uniform vaccination strategy can control the epidemic on the clustered network model.

Now assume that for all  $k \in \mathbb{Z}^+$  there exists some  $n \geq k$  such that  $\rho_n p_{RW}(n) > 0$  and fix  $p_H$  and  $p_V < 1$ . Then we can always find an  $n^*$  such that  $p_H(1 - p_V) \geq 1/(n - 2)$  for  $n > n^*$ , so  $\lim_{n \rightarrow \infty} \mu_{\hat{T}(n)} = \infty$ . Thus  $R_*^U = \infty$  for  $p_V < 1$  and the uniform vaccination strategy cannot control the epidemic on the clustered network model.  $\square$

Recall that the uniform vaccination strategy can always control the epidemic in the rewired edge-triangle model (see Remark 6.1). However, Proposition 7.6 shows that rewiring large households drastically changes the performance of the uniform vaccination strategy.

### 7.3.4 Final size of a major outbreak

Before considering the final size of a major outbreak in the clustered network model under the uniform vaccination strategy we introduce the following notation. Let

$$\begin{aligned} G_D^V(s) &= f_{\bar{D}-1}(1 - p_N(1 - p_V)(1 - s)), \\ G_D^V(s) &= f_{D_U}(1 - p_N(1 - p_V)(1 - s)). \end{aligned}$$

The final size of a major outbreak  $z = 1 - b(\pi)$ , where  $\pi$  is the smallest solution to the equation  $\pi = \tilde{b}(\pi)$ , and  $b(s)$  and  $\tilde{b}(s)$  are given below in Theorem 7.4.

**Theorem 7.4.** *The probability generating functions for the initial and subsequent offspring distributions of the backward Galton-Watson branching process are*

given by

$$\begin{aligned}
b(s) &= G_D^V(s) \sum_{n=1}^{\infty} \tilde{\rho}_n \left\{ (1 - p_{RW}(n)) \sum_{v=0}^n \binom{n}{v} p_V^v (1 - p_V)^{n-v} f_{M(n-v)}(G_D^V(s)) \right. \\
&\quad \left. + p_{RW}(n) f_{\hat{M}(n)}(G_D^V(s)) \right\}, \\
\tilde{b}(s) &= G_D^V(s) \sum_{n=1}^{\infty} \tilde{\rho}_m \left\{ (1 - p_{RW}(n)) \sum_{v=0}^n \binom{n}{v} p_V^v (1 - p_V)^{n-v} f_{M(n-v)}(G_D^V(s)) \right. \\
&\quad \left. + p_{RW}(n) f_{\hat{M}(n)}(G_D^V(s)) \right\},
\end{aligned}$$

where  $f_{\hat{M}(1)}(s) = 1$  and, for  $n = 2, 3, \dots$ ,

$$f_{\hat{M}(n)}(s) = \left( 1 - p_H(1 - p_V) \left( 1 - \hat{f}_U^{(n)}(s) \right) \right)^{n-1},$$

and  $\hat{f}_U^{(n)}(s)$  is the unique solution in  $[0, 1]$  of the equation

$$\hat{f}_U^{(n)}(s) = s \left( 1 - p_H(1 - p_V) \left( 1 - \hat{f}_U^{(n)}(s) \right) \right)^{n-2}.$$

Since the investigation of the clustered network model under the uniform vaccination strategy can be considered as an amalgamation of well-studied models (see Section 7.3.2) we omit the proof of Theorem 7.4.

## 7.4 Analysis of $R_0$ in the clustered network

### 7.4.1 Calculation of $R_0$

Before calculating  $R_0$  in the clustered network model under no vaccination we consider the forward  $R_0$  branching process used to calculate  $R_0$  and the mean rank generation sizes of a single rewired household epidemic. Note that we define the rank generations of a single rewired household epidemic in an identical manner to the rank generations of a rewired triangle epidemic, given in Section 6.5.

The forward  $R_0$  branching process is constructed analogously to the forward individual-based branching process discussed in Section 3.2.3, used to calculate  $R_0$  in the model with three levels of mixing. Recall that the global generation of an infective is its generation in the epidemic at large (see Section 3.2.3). Each ‘individual’ in the forward  $R_0$  branching process consists of an infected household

or rewired household. We consider the infections in the household or rewired household epidemic occurring in multiple generations as the infection spreads throughout the household or rewired household. A time period corresponds to a new generation being infected, so in the forward individual-based branching process an individual's age is which generation of the household or rewired household epidemic it is in. Thus an individual in this branching process may have offspring at multiple time points, as the epidemic spreads through the household or rewired household. We hypothesise that, as  $m \rightarrow \infty$ , the total number of infected households infected in the epidemic process on  $m$  households will converge in distribution to the total progeny of the forward  $R_0$  branching process, similarly to the forward Galton-Watson branching process in Section 3.2.1.

In the forward  $R_0$  branching process we say that an individual's time of birth is given by the global generation of the corresponding household primary case in the epidemic process. An individual in this branching process may reproduce at ages  $1, 2, \dots$ . We denote the mean number of offspring at age  $i + 1$  by a household and a rewired household by  $\nu_H^{(i)}$  and  $\nu_R^{(i)}$  respectively. Note that if  $p_{RW}(n) = 0$ ,  $n = 1, 2, \dots$ , then  $\nu_H^{(i)} = \nu_{NN}^{(i)}$ , as calculated in Theorem 3.3. Furthermore, including the probability of rewiring,

$$\nu_H^{(i-1)} = \begin{cases} p_N \mu_{\tilde{D}-1} \sum_{n=1}^{\infty} \tilde{\rho}_n (1 - p_{RW}(n)) & \text{if } i = 1, \\ p_N \mu_D \sum_{n=i}^{\infty} \tilde{\rho}_n \mu_{i-1}^{(n)} (1 - p_{RW}(n)) & \text{if } i = 2, 3, \dots, \end{cases} \quad (7.46)$$

where  $\mu_{i-1}^{(n)}$  is the mean number of infectives in rank generation  $i$  of a single household epidemic in a household size of  $n$ , given in Section 3.2.3.

We assume that a rewired household has a locally tree-like structure, so to calculate  $\nu_R^{(i)}$  we consider a branching process which gives the size of successive generations of infectives in the rewired household epidemic with household size label  $n$ ,  $n = 2, 3, \dots$ . Note that, conditioned on the primary infective being in a rewired household with household size label  $n$ , the primary infective has  $n - 1$  rewired household neighbours and the expected number of individuals in the first rank generation of a rewired household of size  $n$  is  $p_H(n - 1)$ . Furthermore, a secondary infective in a rewired household has  $n - 2$  rewired household neighbours, each infected with probability  $p_H$ . Thus, by standard branching process theory, the expected number of individuals in the  $i$ th rank generation of a rewired

household with household size label  $n$  is given by  $p_H(n-1)(p_H(n-2))^{i-2}$ . Therefore, noting that a primary infective in a rewired household has household label  $n$  with probability  $\tilde{\rho}_n$ ,

$$\nu_R^{(i-1)} = \begin{cases} p_N \mu_{\tilde{D}-1} \sum_{n=1}^{\infty} \tilde{\rho}_n p_{RW}(n) & \text{if } i = 1, \\ p_N \mu_D p_H \sum_{n=2}^{\infty} \tilde{\rho}_n (n-1) (p_H(n-2))^{i-2} p_{RW}(n) & \text{if } i = 2, 3, \dots, \end{cases} \quad (7.47)$$

**Proposition 7.7.** *The basic reproduction number  $R_0$  is given by the asymptotic (Malthusian) geometric growth rate of the forward  $R_0$  branching process, which is the unique solution in  $(0, \infty)$  of  $\lambda$  such that  $V(\lambda) = 1$ , where*

$$V(\lambda) = \sum_{i=1}^{\infty} \frac{\nu_H^{(i-1)} + \nu_R^{(i-1)}}{\lambda^i},$$

with  $\nu_H^{(i-1)}$  and  $\nu_R^{(i-1)}$  given in equations (7.46) and (7.47) respectively.

#### 7.4.2 The effect of the rewiring process on $R_0$

**Proposition 7.8.** *If the household size distribution  $H$  has finite support or there exists a  $k \in \mathbb{Z}^+$  such that  $\rho_n p_{RW}(n) = 0$  for all  $n \geq k$ , then  $R_0 < \infty$ . Otherwise the forward  $R_0$  branching process has a faster than geometric growth rate and we say that  $R_0 = \infty$ .*

*Proof.* Recall that  $\mu_i^{(n)}$ ,  $i = 0, 1, \dots, n-1$ , are the mean number of infectives in rank generation  $i$  of a single household epidemic in a household size of  $n$  so  $\mu_i^{(n)}(1 - p_{RW}(n)) \leq n$  and consequently

$$\begin{aligned} \sum_{i=1}^{\infty} \frac{\nu_H^{(i-1)}}{\lambda^i} &= \frac{p_N \mu_{\tilde{D}-1} \sum_{n=1}^{\infty} \tilde{\rho}_n (1 - p_{RW}(n))}{\lambda} + \sum_{i=2}^{\infty} \frac{p_N \mu_D \sum_{n=i}^{\infty} \tilde{\rho}_n \mu_{i-1}^{(n)} (1 - p_{RW}(n))}{\lambda^i} \\ &\leq \frac{p_N \mu_{\tilde{D}-1}}{\lambda} + \sum_{i=2}^{\infty} \frac{p_N \mu_D \sum_{n=i}^{\infty} \tilde{\rho}_n n}{\lambda^i} \\ &\leq \frac{p_N \mu_{\tilde{D}-1}}{\lambda} + \sum_{i=2}^{\infty} \frac{p_N \mu_D \mathbb{E}[\tilde{H}]}{\lambda^i} \end{aligned} \quad (7.48)$$

We now consider  $\sum_{i=1}^{\infty} \frac{\nu_R^{(i-1)}}{\lambda^i}$ . Note that

$$\begin{aligned}
\sum_{i=1}^{\infty} \frac{\nu_R^{(i-1)}}{\lambda^i} &= \frac{p_N \mu_{\tilde{D}-1} \sum_{n=1}^{\infty} \tilde{\rho}_n p_{RW}(n)}{\lambda} \\
&\quad + \sum_{i=2}^{\infty} \frac{p_N \mu_{DPH} \sum_{n=2}^{\infty} \tilde{\rho}_n (n-1) (p_H(n-2))^{i-2} p_{RW}(n)}{\lambda^i} \\
&= \frac{p_N \mu_{\tilde{D}-1} \sum_{n=1}^{\infty} \tilde{\rho}_n p_{RW}(n)}{\lambda} \\
&\quad + p_N \mu_{DPH} \frac{\sum_{i=2}^{\infty} \sum_{n=2}^{\infty} \tilde{\rho}_n (n-1) (p_H(n-2))^{i-2} p_{RW}(n)}{\lambda^i} \\
&= \lambda^{-1} p_N \mu_{\tilde{D}-1} \sum_{n=1}^{\infty} \tilde{\rho}_n p_{RW}(n) \\
&\quad + p_N \mu_D \lambda^{-2} \sum_{n=2}^{\infty} \tilde{\rho}_n (n-1) p_{RW}(n) \sum_{i=2}^{\infty} (p_H \lambda^{-1} (n-2))^{i-2}. \quad (7.49)
\end{aligned}$$

Firstly, note that since  $E[H^2] E[D^2] < \infty$  the right-hand side of equation (7.48) can be made arbitrarily small by choosing sufficiently large  $\lambda$ . Furthermore, if  $H$  has finite support or there exists a  $k \in \mathbb{Z}^+$  such that  $\rho_n p_{RW}(n) = 0$  for all  $n \geq k$  then the right-hand side of equation (7.49) can also be made arbitrarily small. Therefore if  $H$  has finite support or there exists a  $k \in \mathbb{Z}^+$  such that  $\rho_n p_{RW}(n) = 0$  for all  $n \geq k$  then  $V(\lambda)$  can be made arbitrarily small by choosing sufficiently large  $\lambda$ . Thus the equation  $V(\lambda) = 1$  always has a solution  $\lambda^* < \infty$  and  $R_0 = \lambda^*$ .

Next consider the case that for all  $k \in \mathbb{Z}^+$  there exists some  $n \geq k$  such that  $\rho_n p_{RW}(n) > 0$ . Note that  $\sum_{i=2}^{\infty} (p_H \lambda^{-1} (n-2))^{i-2} < \infty$  if and only if  $p_H \lambda^{-1} (n-2) < 1$ , i.e.  $n < 2 + (\lambda/p_H)$ . However, since for all  $k \in \mathbb{Z}^+$  there exists some  $n \geq k$  such that  $\rho_n p_{RW}(n) > 0$ , for fixed  $p_H$  and  $\lambda < \infty$  there exists some  $n^* \geq k$  such that  $\rho_n p_{RW}(n^*) > 0$  and  $n^* > 2 + (\lambda/p_H)$ . Therefore  $V(\lambda) = \infty$  for all  $\lambda < \infty$  and thus  $R_0 = \infty$ .  $\square$

It is clear from the proof of Proposition 7.8 that it is very large rewired households that cause  $R_0$  to become infinite. Furthermore, large rewired households significantly affect the ability of the uniform vaccination strategy to prevent a major outbreak on the clustered network model (see Proposition 7.6). To make the clustered network model more applicable, and to allow for a comparison of the uniform and acquaintance vaccination strategies, we introduce a rewiring cap,  $\kappa$  say, which is the size of the largest household which has a positive probability of rewiring. Note that by Proposition 7.8 if  $\kappa = \max \{n : \rho_n p_{RW}(n) > 0\} < \infty$

then  $R_0 < \infty$ . Furthermore, it is clear that we can always choose  $\kappa$  large enough such that a given positive clustering coefficient can be achieved (see equation (7.1)). Finally, note that the rewiring cap means that the uniform vaccination strategy can always control the epidemic (see Proposition 7.6).

## 7.5 Rewiring approaches for the clustered network model

Consider the clustered network model with household size distribution  $H$  and network degree distribution  $D$ . Then  $\mathcal{C}_{max}^H$ , the maximum clustering coefficient possible for a given household size and network degree distribution, is achieved by setting  $p_{RW}(n) = 0$ ,  $n = 1, 2, \dots$  in equation (7.1). Note that for a given target clustering coefficient  $\mathcal{C}^H \in (0, \mathcal{C}_{max}^H)$  there are infinitely many choices of  $p_{RW}(n)$ ,  $n = 1, 2, \dots$ . In this section we investigate the effect of the choice of  $p_{RW}(n)$ ,  $n = 1, 2, \dots$ , on the final size of a major outbreak in the clustered network model with a fixed clustering coefficient. We focus on three approaches to selecting  $p_{RW}(n)$  for a given a target clustering coefficient: equal rewiring, in which  $p_{RW}(n) = p_{RW}$  for  $n = 1, 2, \dots$ ; rewiring from 1, in which we rewire the households in order from the smallest household size to the largest until we reach the desired clustering coefficient; and rewiring from  $\infty$ , in which we rewire the households in order from the largest size to the smallest until we reach the desired clustering coefficient. Note that equal rewiring corresponds to the rewiring process introduced by Ball et al. (2013). Before comparing the rewiring from 1 and rewiring from  $\infty$  approaches we introduce the following preliminary work.

Firstly note that under the rewiring from 1 approach

$$p_{RW}(n) = \begin{cases} 1 & \text{if } n < c_1, \\ p_{RW}^1 & \text{if } n = c_1, \\ 0 & \text{if } n > c_1, \end{cases} \quad (7.50)$$

where  $c_1 \in \mathbb{Z}^+$  and  $p_{RW}^1 \in [0, 1]$  are predetermined by the target clustering coefficient. The clustering coefficient under the rewiring from 1 approach,  $\mathcal{C}_1^H$ ,

is calculated by substituting equation (7.50) into equation (7.1), so

$$\mathcal{C}_1^H = \frac{\sum_{n=c_1+1}^{\infty} \rho_n n(n-1)(n-2) + (1-p_{RW}^1) \rho_{c_1} c_1(c_1-1)(c_1-2)}{\mathbb{E}[H(D+H-1)(D+H-2)]}. \quad (7.51)$$

Let  $b_1(s)$  and  $\tilde{b}_1(s)$  be the resulting probability generating functions for the initial and subsequent offspring distributions of the backwards Galton-Watson branching process. So, substituting  $p_V = 0$  and equation (7.50) into Theorem 7.4,

$$\begin{aligned} b_1(s) = f_D(1-p_N+p_N s) & \left[ \sum_{n=1}^{c_1-1} \tilde{\rho}_n f_{\hat{M}^{(n)}}(f_D(1-p_N+p_N s)) \right. \\ & + \sum_{n=c_1+1}^{\infty} \tilde{\rho}_n f_{M^{(n)}}(f_D(1-p_N+p_N s)) \\ & + p_{RW}^1 \tilde{\rho}_{c_1} f_{\hat{M}^{(c_1)}}(f_D(1-p_N+p_N s)) \\ & \left. + (1-p_{RW}^1) \tilde{\rho}_{c_1} f_{M^{(c_1)}}(f_D(1-p_N+p_N s)) \right], \end{aligned} \quad (7.52a)$$

$$\begin{aligned} \tilde{b}_1(s) = f_{\tilde{D}-1}(1-p_N+p_N s) & \left[ \sum_{n=1}^{c_1-1} \tilde{\rho}_n f_{\hat{M}^{(n)}}(f_D(1-p_N+p_N s)) \right. \\ & + \sum_{n=c_1+1}^{\infty} \tilde{\rho}_n f_{M^{(n)}}(f_D(1-p_N+p_N s)) \\ & + p_{RW}^1 \tilde{\rho}_{c_1} f_{\hat{M}^{(c_1)}}(f_D(1-p_N+p_N s)) \\ & \left. + (1-p_{RW}^1) \tilde{\rho}_{c_1} f_{M^{(c_1)}}(f_D(1-p_N+p_N s)) \right]. \end{aligned} \quad (7.52b)$$

Secondly note that under the rewiring from  $\infty$  approach

$$p_{RW}(n) = \begin{cases} 0 & \text{if } n < c_{\infty}, \\ p_{RW}^{\infty} & \text{if } n = c_{\infty}, \\ 1 & \text{if } n > c_{\infty}, \end{cases} \quad (7.53)$$

where  $c_{\infty} \in \mathbb{Z}^+$  and  $p_{RW}^{\infty} \in [0, 1]$  are predetermined by the target clustering coefficient. The clustering coefficient under the rewiring from  $\infty$  approach,  $\mathcal{C}_{\infty}^H$ , is calculated by substituting equation (7.53) into equation (7.1), so

$$\mathcal{C}_{\infty}^H = \frac{\sum_{n=0}^{c_{\infty}-1} \rho_n n(n-1)(n-2) + (1-p_{RW}^{\infty}) \rho_{c_{\infty}} c_{\infty}(c_{\infty}-1)(c_{\infty}-2)}{\mathbb{E}[H(D+H-1)(D+H-2)]}. \quad (7.54)$$



Let  $b_\infty(s)$  and  $\tilde{b}_\infty(s)$  be the resulting probability generating functions for the initial and subsequent offspring distributions of the backwards Galton-Watson branching process. So, substituting  $p_V = 0$  and equation (7.50) into Theorem 7.4,

$$b_\infty(s) = f_D(1 - p_N + p_N s) \left[ \sum_{n=1}^{c_\infty-1} \tilde{\rho}_n f_{M^{(n)}}(f_D(1 - p_N + p_N s)) \right. \\ + \sum_{n=c_\infty+1}^{\infty} \tilde{\rho}_n f_{\hat{M}^{(n)}}(f_D(1 - p_N + p_N s)) \\ + p_{RW}^\infty \tilde{\rho}_{c_\infty} f_{\hat{M}^{(c_\infty)}}(f_D(1 - p_N + p_N s)) \\ \left. + (1 - p_{RW}^\infty) \tilde{\rho}_{c_\infty} f_{M^{(c_\infty)}}(f_D(1 - p_N + p_N s)) \right], \quad (7.55a)$$

$$\tilde{b}_\infty(s) = f_{\tilde{D}-1}(1 - p_N + p_N s) \left[ \sum_{n=1}^{c_\infty-1} \tilde{\rho}_n f_{M^{(n)}}(f_D(1 - p_N + p_N s)) \right. \\ + \sum_{n=c_\infty+1}^{\infty} \tilde{\rho}_n f_{\hat{M}^{(n)}}(f_D(1 - p_N + p_N s)) \\ + p_{RW}^\infty \tilde{\rho}_{c_\infty} f_{\hat{M}^{(c_\infty)}}(f_D(1 - p_N + p_N s)) \\ \left. + (1 - p_{RW}^\infty) \tilde{\rho}_{c_\infty} f_{M^{(c_\infty)}}(f_D(1 - p_N + p_N s)) \right]. \quad (7.55b)$$

Let  $b_d(s) = (b_1(s) - b_\infty(s)) / f_D(1 - p_N + p_N s)$ , and note that  $\tilde{b}_1(s) - \tilde{b}_\infty(s) = b_d(s) f_{\tilde{D}-1}(1 - p_N + p_N s)$ . Therefore, comparing equations (7.52) and (7.55),  $\text{sgn}(b_d(s)) = \text{sgn}(b_1(s) - b_\infty(s)) = \text{sgn}(\tilde{b}_1(s) - \tilde{b}_\infty(s))$ .

Finally, let  $R_*^1$  and  $R_*^\infty$  be the threshold parameters for the clustered network model under no vaccination and applying the approach of rewiring from 1 and  $\infty$  respectively, where the clustered network model has household size distribution  $H$ , network degree  $D$ , network infection rate  $\lambda_N$ , household infection rate  $\lambda_H$  and clustering coefficient  $\mathcal{C}^H$ . Similarly, let  $z_1$  and  $z_\infty$  be the final size of a major outbreak on this clustered network model applying the approach of rewiring from 1 and  $\infty$  respectively.

### 7.5.1 Comparing the final size of a major outbreak under the rewiring from 1 and $\infty$ approaches

To compare the final size of a major outbreak on the clustered network model under the rewiring from 1 and  $\infty$  approaches we fix the clustering coefficient of the model. We first consider a trivial case which highlights the drastic effect that rewiring large households can have on the properties of the model.

**Proposition 7.9.** *If for all  $k \in \mathbb{Z}^+$  there exists some  $n \geq k$  such that  $\rho_n > 0$  and  $R_*^1 < 1$  then  $z_\infty > z_1 = 0$ .*

*Proof.* If  $R_*^1 < 1$  then by standard branching process theory the probability of extinction of the approximating branching process is 1, so  $z_1 = 0$ . However, since for all  $k \in \mathbb{Z}^+$  there exists some  $n \geq k$  such that  $\rho_n > 0$ , applying Proposition 7.8 yields  $R_*^\infty > 1$ , so, by standard branching process,  $z_\infty > 0$  and Proposition 7.9 immediately follows.  $\square$

We now show that if we only rewire a fraction of households of size 3 under the rewiring from 1 approach and  $p_H = 1$  then  $z_1 > z_\infty$ .

**Proposition 7.10.** *If  $R_*^1 > 1$ ,  $p_H = 1$ ,  $c_\infty > 3$ ,  $c_1 = 3$ ,  $p_{RW}^1 > 0$  and  $\tilde{\rho}_3 > 0$  then  $z_1 > z_\infty$ .*

*Proof.* We begin by showing that at  $p_H = 1$ ,  $b_d(s) < 0$ ,  $s \in (0, 1]$ . We then show that  $b_d(s) < 0$  is a sufficient condition for the result  $z_1 > z_\infty$ .

Since we require both models to have equal clustering coefficients, considering the equation  $\mathcal{C}_1^H = \mathcal{C}_\infty^H$ , substituting equations (7.51) and (7.54),  $c_1 = 3$  and  $\rho_n = n\tilde{\rho}_n/\mu_H$ ,  $n = 1, 2, \dots$ , yields

$$\begin{aligned} \mathcal{C}_1^H &= \mathcal{C}_\infty^H \\ \iff 6p_{RW}^1\rho_3 &= \sum_{n=c_\infty+1}^{\infty} \rho_n n(n-1)(n-2) + \rho_{c_\infty} p_{RW}^\infty c_\infty (c_\infty - 1)(c_\infty - 2) \\ \iff 2p_{RW}^1\tilde{\rho}_3 &= \sum_{n=c_\infty+1}^{\infty} \tilde{\rho}_n (n-1)(n-2) + \tilde{\rho}_{c_\infty} p_{RW}^\infty (c_\infty - 1)(c_\infty - 2). \end{aligned} \quad (7.56)$$

Substituting equations (7.52), (7.55) and  $c_1 = 3$  into  $b_d(s) = b_1(s) - b_\infty(s)/f_D(1 -$

$p_N + p_N s$ ) yields

$$\begin{aligned}
b_d(s) &= \tilde{\rho}_3 p_{RW}^1 [f_{\hat{M}^{(3)}}(f_D(1 - p_N + p_N s)) - f_{M^{(3)}}(f_D(1 - p_N + p_N s))] \\
&\quad + \sum_{n=c_\infty+1}^{\infty} \tilde{\rho}_n [f_{M^{(n)}}(f_D(1 - p_N + p_N s)) - f_{\hat{M}^{(n)}}(f_D(1 - p_N + p_N s))] \\
&\quad + \tilde{\rho}_{c_\infty} p_{RW}^\infty [f_{M^{(c_\infty)}}(f_D(1 - p_N + p_N s)) - f_{\hat{M}^{(c_\infty)}}(f_D(1 - p_N + p_N s))].
\end{aligned} \tag{7.57}$$

For  $n \geq 3$ , if  $p_H = 1$  then  $f_{M^{(n)}}(s) = s^{n-1}$  and  $f_{\hat{M}^{(n)}}(s) = 0$  (since a rewired household with household size label  $n \geq 3$  contains infinitely many individuals) which, substituted into equation (7.57), yields

$$\begin{aligned}
b_d(s) &= -\tilde{\rho}_3 p_{RW}^1 f_D(1 - p_N + p_N s)^2 \\
&\quad + \sum_{n=c_\infty+1}^{\infty} \tilde{\rho}_n f_D(1 - p_N + p_N s)^{n-1} \\
&\quad + \tilde{\rho}_{c_\infty} p_{RW}^\infty f_D(1 - p_N + p_N s)^{c_\infty-1}.
\end{aligned} \tag{7.58}$$

Substituting equation (7.56) into equation (7.58) and noting that  $f_D(s) \leq 1$  for  $s \in [0, 1]$  yields

$$\begin{aligned}
b_d(s) &= \tilde{\rho}_{c_\infty} p_{RW}^\infty \left[ f_D(1 - p_N + p_N s)^{c_\infty-1} - \frac{(c_\infty-1)(c_\infty-2)}{2} f_D(1 - p_N + p_N s)^2 \right] \\
&\quad + \sum_{n=c_\infty+1}^{\infty} \tilde{\rho}_n \left[ f_D(1 - p_N + p_N s)^{n-1} - \frac{(n-1)(n-2)}{2} f_D(1 - p_N + p_N s)^2 \right] \\
&= \tilde{\rho}_{c_\infty} p_{RW}^\infty f_D(1 - p_N + p_N s)^2 \left[ f_D(1 - p_N + p_N s)^{c_\infty-3} - \frac{(c_\infty-1)(c_\infty-2)}{2} \right] \\
&\quad + \sum_{n=c_\infty+1}^{\infty} \tilde{\rho}_n f_D(1 - p_N + p_N s)^2 \left[ f_D(1 - p_N + p_N s)^{n-3} - \frac{(n-1)(n-2)}{2} \right] \\
&\leq \tilde{\rho}_{c_\infty} p_{RW}^\infty f_D(1 - p_N + p_N s)^2 \left[ 1 - \frac{(c_\infty-1)(c_\infty-2)}{2} \right] \\
&\quad + \sum_{n=c_\infty+1}^{\infty} \tilde{\rho}_n f_D(1 - p_N + p_N s)^2 \left[ 1 - \frac{(n-1)(n-2)}{2} \right].
\end{aligned} \tag{7.59}$$

We assume that  $c_\infty > 3$ , so  $1 - (n-1)(n-2)/2 < 0$  for  $n \geq c_\infty$ . Furthermore, since  $E[D^2] < \infty$ ,  $P(D = \infty) < 1$  and so  $f_D(1 - p_N + p_N s) > 0$  unless  $p_N = 1$  and  $s = 0$ . Thus  $b_d(s) < 0$  for  $s \in (0, 1]$  which implies  $b_1(s) < b_\infty(s)$  and  $\tilde{b}_1(s) < \tilde{b}_\infty(s)$  for  $s \in (0, 1]$ .

By definition  $z_1 = 1 - b_1(\pi_1)$ , where  $\pi_1$  is the smallest solution to the

equation  $\pi_1 = \tilde{b}_1(\pi_1)$ , and  $z_\infty = 1 - b_\infty(\pi_\infty)$ , where  $\pi_\infty$  is the smallest solution to the equation  $\pi_\infty = \tilde{b}_\infty(\pi_\infty)$ . Therefore, since for  $s \in [0, 1]$ ,  $\tilde{b}_\infty(s)$  is a strictly increasing function, if  $s < \tilde{b}_\infty(s)$  then  $s < \pi_\infty$ . Thus, since  $\tilde{b}_1(s) < \tilde{b}_\infty(s)$  for  $s \in (0, 1]$  and we assume that  $z_1 < 1$  (i.e.  $\pi_1 > 0$ ),  $\pi_1 = \tilde{b}_1(\pi_1) < \tilde{b}_\infty(\pi_1)$  and so  $\pi_1 < \pi_\infty$ . Analogous arguments yield  $b_1(\pi_1) < b_\infty(\pi_\infty)$  and  $z_1 > z_\infty$  as required.  $\square$

We now show that if we rewire all households except for a positive proportion of households of size 3 under the rewiring from  $\infty$  approach and  $p_H = 1$  then  $z_1 > z_\infty$ .

**Proposition 7.11.** *If  $R_*^1 > 1$ ,  $p_H = 1$ ,  $c_1 > 3$ ,  $c_\infty = 3$ ,  $p_{RW}^\infty > 0$  and  $\tilde{\rho}_3 > 0$ , then  $z_1 > z_\infty$ .*

*Proof.* The proof of Proposition 7.11 follows analogous arguments to the proof of Proposition 7.9, so we only outline the key equations of the proof. Since we require both models to have identical clustering coefficients, considering the equation  $\mathcal{C}_1^H = \mathcal{C}_\infty^H$ , substituting equations (7.51) and (7.54),  $c_\infty = 3$  and  $\rho_n = n\tilde{\rho}_n/\mu_H$ ,  $n = 1, 2, \dots$ , yields

$$\begin{aligned} \mathcal{C}_1^H &= \mathcal{C}_\infty^H \\ \iff 2\tilde{\rho}_3(1 - p_{RW}^\infty) &= \sum_{n=c_1+1}^{\infty} \tilde{\rho}_n(n-1)(n-2) + \tilde{\rho}_{c_1}(1 - p_{RW}^1)(c_1-1)(c_1-2). \end{aligned} \quad (7.60)$$

Substituting equations (7.52), (7.55) and  $c_\infty = 3$  into  $b_d(s)$  yields

$$\begin{aligned} b_d(s) &= \tilde{\rho}_3(1 - p_{RW}^\infty) [f_{\hat{M}^{(3)}}(f_D(1 - p_N + p_N s)) - f_{M^{(3)}}(f_D(1 - p_N + p_N s))] \\ &\quad + \sum_{n=c_1+1}^{\infty} \tilde{\rho}_n [f_{M^{(n)}}(f_D(1 - p_N + p_N s)) - f_{\hat{M}^{(n)}}(f_D(1 - p_N + p_N s))] \\ &\quad + \tilde{\rho}_{c_1}(1 - p_{RW}^1) [f_{M^{(c_1)}}(f_D(1 - p_N + p_N s)) - f_{\hat{M}^{(c_1)}}(f_D(1 - p_N + p_N s))]. \end{aligned} \quad (7.61)$$

For  $n \geq 3$ , if  $p_H = 1$  then  $f_{M^{(n)}}(s) = s^{n-1}$  and  $f_{\hat{M}^{(n)}}(s) = 0$  which, substituted into equation (7.61), yields

$$\begin{aligned} b_d(s) &= -\tilde{\rho}_3(1 - p_{RW}^\infty) f_D(1 - p_N + p_N s)^2 \\ &\quad + \sum_{n=c_1+1}^{\infty} \tilde{\rho}_n f_D(1 - p_N + p_N s)^{n-1} \\ &\quad + \tilde{\rho}_{c_1}(1 - p_{RW}^1) f_D(1 - p_N + p_N s)^{c_1-1}. \end{aligned} \quad (7.62)$$

Substituting equation (7.60) into equation (7.62) and applying analogous arguments to those leading to equation (7.59) yields

$$b_d(s) \leq \tilde{\rho}_{c_1}(1 - p_{RW}^1)f_D(1 - p_N + p_N s)^2 \left[ 1 - \frac{(c_1 - 1)(c_1 - 2)}{2} \right] \\ + \sum_{n=c_1+1}^{\infty} \tilde{\rho}_n f_D(1 - p_N + p_N s)^2 \left[ 1 - \frac{(n - 1)(n - 2)}{2} \right].$$

Therefore, by analogous arguments to those below equation (7.59),  $b_d(s) < 0$  for  $s \in (0, 1]$  and thus  $z_1 > z_\infty$  as required.  $\square$

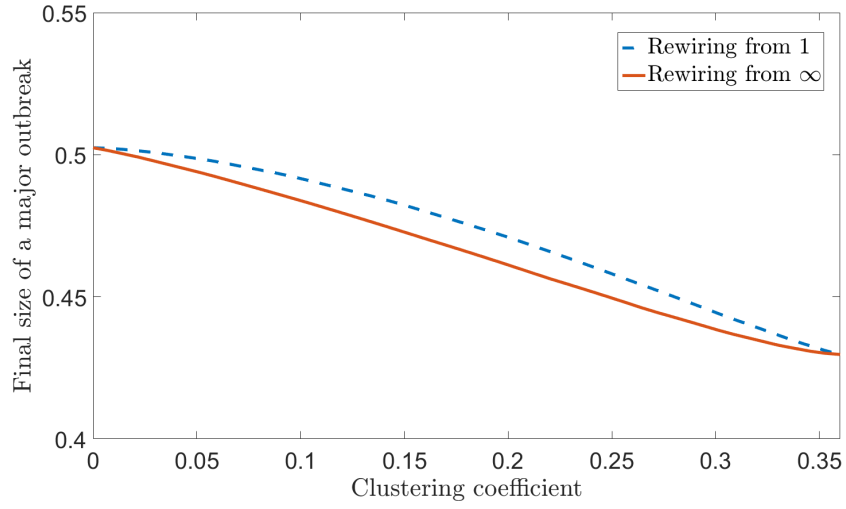
Although the restriction  $z_1 < 1$  may appear restrictive, we note that if  $P(D = 0) > 0$  and  $P(H = 1) > 0$  then  $z_1 < 1$ .

Our numerical investigations suggest that if the clustered network model has very small or near maximum clustering coefficient then, as Propositions 7.10 and 7.11 suggest,  $z_1 > z_\infty$ . Indeed, if  $p_H$  is large then  $z_1 > z_\infty$  regardless of the clustering coefficient, as illustrated in Figure 7.1a. However, as  $p_H$  decreases then the ordering flips and  $z_1 < z_\infty$ , as illustrated in Figure 7.1b.

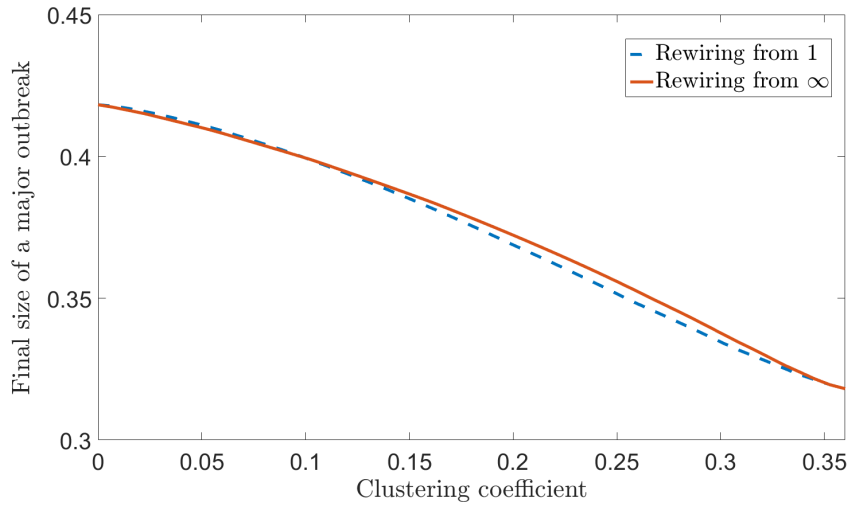
## 7.6 Numerical investigation of the clustered network model

### 7.6.1 Instability of Gontcharoff polynomials

In applications of the household-based epidemic models it is often assumed that households are relatively small (see, for example, Ball and Shaw (2015) or Fraser (2007)). However, in this chapter we are primarily interested in using the household structure as a method of creating a clustered network. A common assumption in modelling social networks is assuming that the total degree distribution has support in the non-negative integers, e.g. a power law distribution (see, for example, Barabási and Albert (1999)). Furthermore, if the total degree distribution has support in the non-negative integers then the clustering coefficient of the clustered network model is non-zero if and only if the household size distribution also has support in the non-negative integers. However, calculating the final size of a household epidemic or susceptibility set within large households becomes numerically difficult. See, for example, Demiris and O'Neill (2006) or House et al. (2012).



(a)  $p_H = p_N = 0.14$ .



(b)  $p_H = p_N = 0.13$ .

Figure 7.1: The effect of the rewiring from 1 and  $\infty$  approaches on the final size of a major outbreak. The parameters are  $H \sim \text{Poi}^+(6)$ ,  $D \sim \text{Poi}(4)$  and  $I \sim \text{Const}(1)$ .

To consider household size distributions which require the investigation of very large households we bound the final size of a household epidemic or the size of a susceptibility set from above and below in the following way. To calculate a lower bound for the final size of a household epidemic we ignore all within-household infections not emanating from the primary infective. To calculate an upper bound for the final size of a household epidemic we assume that the primary infective immediately infects all unvaccinated household neighbours with probability 1. For example, let  $E[T_L^{(n)}]$  and  $E[T_U^{(n)}]$  be the lower and upper bounds on  $E[T^{(n)}]$  respectively, where  $E[T^{(n)}]$  is the expected final size of a household epidemic in a household of size  $n$ . Then  $E[T_L^{(n)}] = (n-1)p_H$  and  $E[T_U^{(n)}] = (n-1)$ . Similarly, to calculate a lower bound for the size of a household susceptibility set we ignore all within-household infections not directly leading to the primary individual and to calculate an upper bound for the size of a household susceptibility set we assume that the primary individual is immediately contacted by all unvaccinated household neighbours with probability 1. However, we omit the upper and lower bounds in the figures given in the following section because the bounds are too close together to decipher.

### 7.6.2 The effect of clustering on the vaccination strategies

In this section we investigate the effect of household clustering on the performance of the acquaintance and uniform vaccination strategies, in contrast to Section 6.6 in which we investigate the effect of edge-disjoint triangle clustering on the performance of the acquaintance and uniform vaccination strategies by considering the rewired edge-triangle model. Since changing  $p_{RW}(n)$ ,  $n = 1, 2, \dots$ , does not affect the degree correlation we can change  $p_{RW}(n)$  to investigate the effect of household clustering on the performance of the vaccination strategies. Recall that we let  $\kappa$  be the size of the largest household to be rewired (so  $\rho_n p_{RW}(n) = 0$  for  $n > \kappa$ , see Section 7.4) which ensures that  $R_0 < \infty$  (see Proposition 7.8) and that the uniform vaccination strategy can control the epidemic (see Proposition 7.6). Since we are interested in the clustered network model as a method of including clustering into the model, we set  $\lambda_H = \lambda_N$  and  $p_{CH} = p_{CG}$ .

Note that two clustered network models with the same household size distribution, degree distribution, infectious period,  $\kappa$  and infection rates, thus varying

only in  $p_{RW}(n)$ ,  $n = 1, 2, \dots$ , will have different final sizes of major outbreaks. Therefore, since in applications epidemic models are often parameterised by final size data (see, for example, Becker and Utev (1998)) in this section we compare models with the same household size distribution, degree distribution, infectious period and infection rates and differing in  $p_{RW}(n)$  by choosing an infection rate  $\lambda_N$  to fix the final size of a major outbreak ( $z$ ) in the clustered network model without any vaccination.

Similarly to previous investigations of the acquaintance vaccination strategies (see, for example, Section 5.1.4 or Ball and Sirl (2013)), our numerical work suggests that for a fixed vaccination coverage the threshold parameter and final size of a major outbreak are monotonically increasing in  $p_C$ . Therefore, similarly to Section 6.6, in this section we focus on the extreme situations  $p_S = 1$  or  $p_{CH} = p_{CG} = 1$ , which we call the  $p_S = 1$  or  $p_C = 1$  acquaintance vaccination strategies respectively. Let  $c_U^*$ ,  $c_{p_S}^*$  and  $c_{p_C}^*$  be the critical vaccination coverages under the uniform,  $p_S = 1$  acquaintance and  $p_C = 1$  acquaintance vaccination strategies respectively.

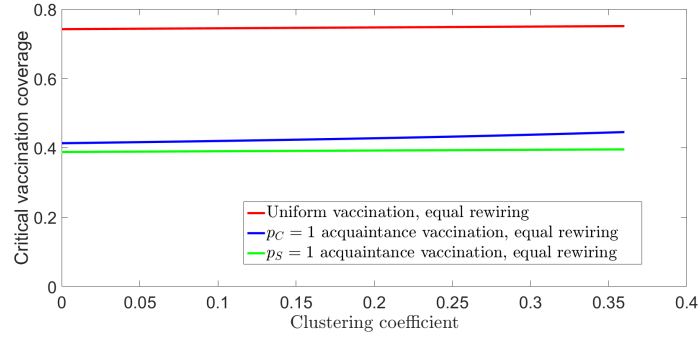
In the clustered network model with a fixed network degree distribution, household size distribution,  $p_{RW}(n)$  and vaccination coverage, the  $p_S = 1$  acquaintance vaccination strategy performs at least as well as the uniform vaccination strategy, with equality in performance when the household size and degree distributions have zero variance and an increasing difference between the two strategies as the variance of the household size and degree distributions increases. We find that fixing  $z$  and increasing the clustering coefficient decreases  $c_U^*$  and  $c_{p_S}^*$ , as illustrated in Figures 7.2a and 7.3a. However, as  $\text{Var}[D_T]$  increases the difference between  $c_U^*$  and  $c_{p_S}^*$  increases with the clustering coefficient, as illustrated in Figure 7.4. We conjecture that the effect of fixing  $z$  and increasing the clustering coefficient on  $c_U^*$  and  $c_{p_S}^*$  can be determined by considering the change in  $R_0$ , similarly to the rewired edge-triangle model in Section 6.6.2. However, due to the numerical difficulty in considering  $R_0$  in the clustered network model we have not performed a similar analysis here.

For each of the vaccination strategies, fixing the clustering coefficient yields that the critical vaccination coverage of the strategy is largest if we follow the rewiring from  $\infty$  approach and smallest if we follow the rewiring from 1 approach, as illustrated in Figures 7.2b and 7.2c. Since rewiring large households

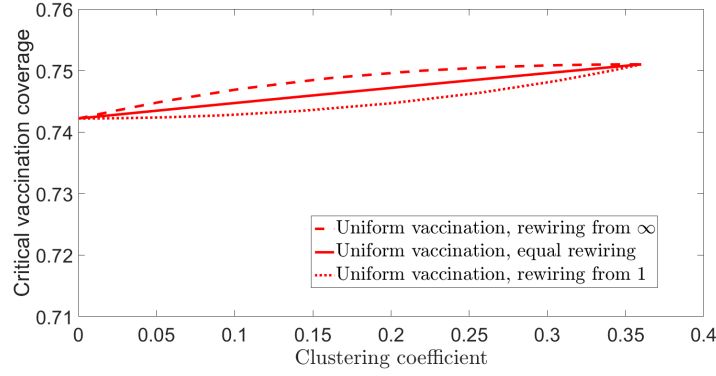


can significantly change the effect of vaccination strategies upon the network (e.g. preventing the uniform vaccination from controlling the epidemic, see Section 7.3.3), we conjecture that this ordering in critical vaccination coverages under the three approaches is caused by the rewiring of large households. Furthermore, this suggests that although the acquaintance vaccination strategy can always control the epidemic (see Section 7.2.4), the presence of large households does hinder the performance of the acquaintance vaccination strategy. For a fixed vaccination coverage and both the  $p_C = 1$  acquaintance and uniform vaccination strategies, increasing  $\text{Var}[D_T]$  has a negligible effect on the critical vaccination coverages between following the rewiring from 1 and  $\infty$  approaches (as illustrated in Figures 7.2b, 7.3b, 7.2c and 7.3c). However, for moderate rewiring, increasing  $\text{Var}[D_T]$  increases the difference between the critical vaccination coverage of the  $p_S = 1$  acquaintance vaccination strategy under the rewiring from 1 and  $\infty$  approaches, as illustrated in Figures 7.2c, 7.3c and 7.4.

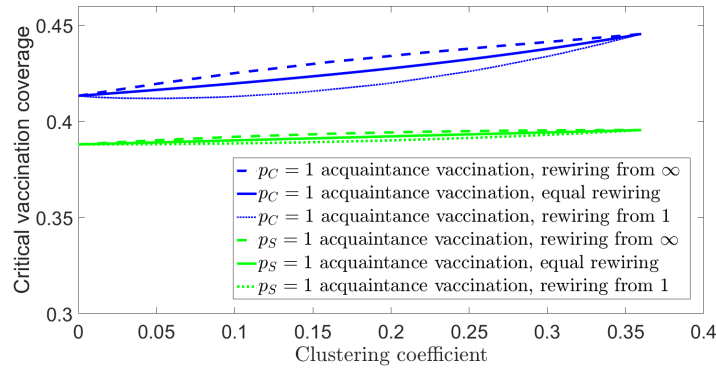
We find that if  $\text{Var}[D_T]$  is small then  $c_{p_C}^* > c_U^*$  and the final size of a major outbreak under the  $p_C = 1$  acquaintance vaccination strategy is larger than the final size of a major outbreak under the uniform vaccination strategy. Furthermore, there is an increasing difference between the two strategies as the clustering coefficient increases, as illustrated in Figures 7.5a and 7.5b. Similarly to Section 6.6, in which we find that the addition edge-disjoint triangle clustering increases the difference in critical vaccination coverage under the  $p_C = 1$  acquaintance and uniform vaccination strategies in the rewired edge-triangle model, we note that, for a fixed vaccination coverage, increasing the clustering coefficient will lead to the  $p_C = 1$  acquaintance vaccination strategy preventing infectious contacts from occurring along fewer edges in the network than the uniform vaccination strategy. This results in the  $p_C = 1$  acquaintance vaccination strategy underperforming compared to the uniform vaccination strategy when  $\text{Var}[D_T]$  is small. However, as  $\text{Var}[D_T]$  increases the  $p_C = 1$  acquaintance vaccination strategy outperforms the uniform vaccination strategy for a fixed vaccination coverage (as illustrated in Figures 7.2a and 7.4).



(a) Investigating the effect of clustering on the critical vaccination coverage of the uniform,  $p_C = 1$  and  $p_S = 1$  acquaintance vaccination strategies under the equal rewiring approach.

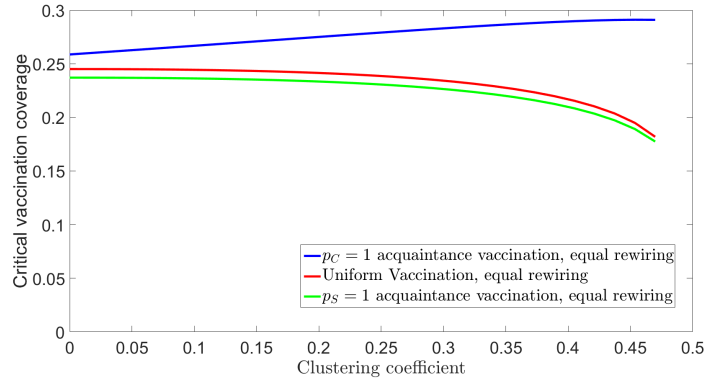


(b) Investigating the effect of clustering on the critical vaccination coverage of the uniform vaccination strategy under the rewiring from  $\infty$ , equal rewiring and rewiring from 1 approaches.

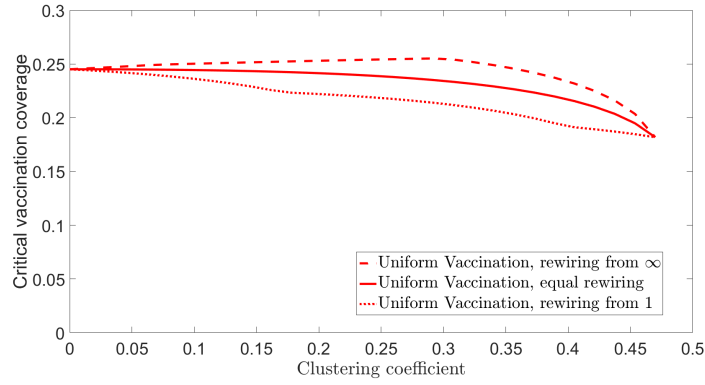


(c) Investigating the effect of clustering on the critical vaccination coverage of the  $p_C = 1$  and  $p_S = 1$  vaccination strategies under the rewiring from  $\infty$ , equal rewiring and rewiring from 1 approaches.

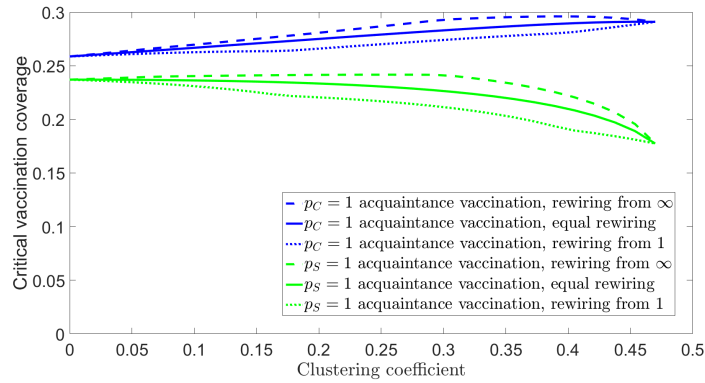
Figure 7.2: The models are matched with  $z = 0.7$  and the other parameters are  $H \sim \text{Poi}^+(6)$ ,  $D \sim \text{Poi}(4)$ ,  $I \sim \text{Const}(1)$  and  $\kappa = 25$  so  $E[D_T] = 10$ .



(a) Investigating the effect of clustering on the critical vaccination coverage of the uniform,  $p_C = 1$  and  $p_S = 1$  acquaintance vaccination strategies under the equal rewiring approach.



(b) Investigating the effect of clustering on the critical vaccination coverage of the uniform vaccination strategy under the rewiring from  $\infty$ , equal rewiring and rewiring from 1 approaches.



(c) Investigating the effect of clustering on the critical vaccination coverage of the  $p_C = 1$  and  $p_S = 1$  vaccination strategies under the rewiring from  $\infty$ , equal rewiring and rewiring from 1 approaches.

Figure 7.3: The models are matched with  $z = 0.5$  and the other parameters are  $f_H(s) = \frac{1}{4}(s^7 + 2s^8 + s^9)$ ,  $f_D(s) = \frac{1}{4}(s^2 + 2s^3 + s^4)$ ,  $I \sim \text{Const}(1)$  and  $\kappa = 10$ , so  $E[D_T] = 10$ .

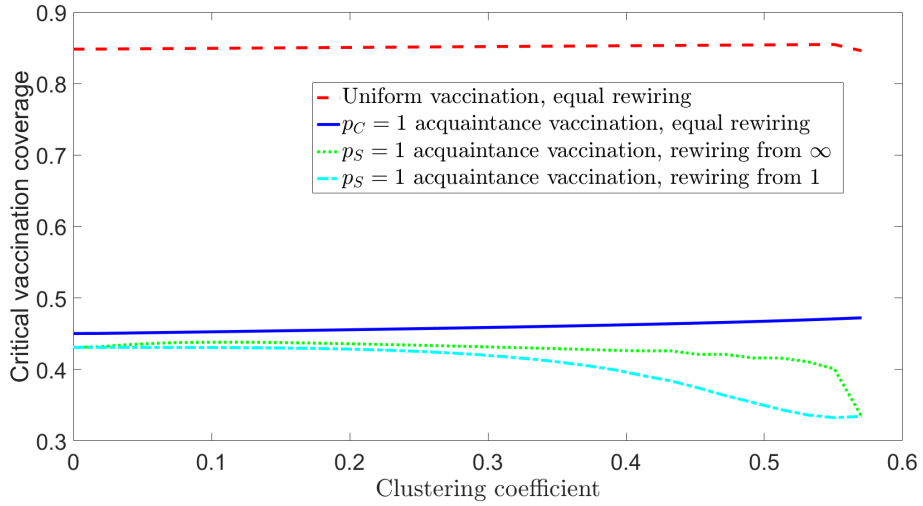
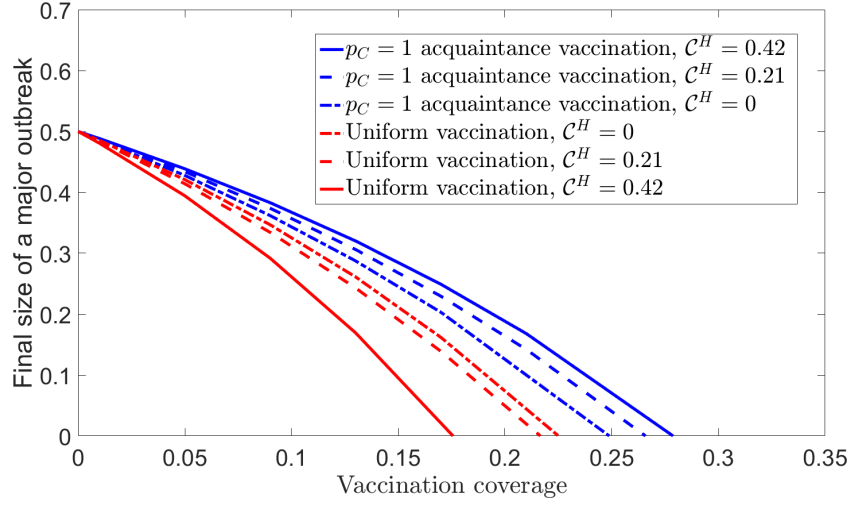


Figure 7.4: Investigating the effect of clustering on the critical vaccination coverage of the uniform,  $p_C = 1$  and  $p_S = 1$  acquaintance vaccination strategies. The models are matched with  $z = 0.7$ , and the other parameters are  $H \sim \text{Geo}^+(9/40)$ ,  $D \sim \text{Geo}^+(1/3)$ ,  $I \sim \text{Const}(1)$  and  $\kappa = 35$  so  $E[D_T] = 10$ .

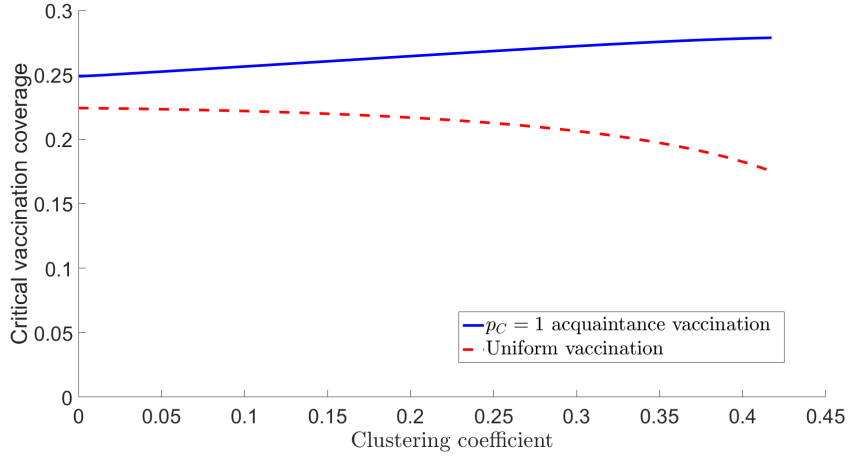
## 7.7 Concluding remarks

In this chapter we consider two vaccination strategies on the clustered network model, specifically the acquaintance and uniform vaccination strategies. For each vaccination strategy, we show how to find a threshold parameter, which determines whether a major outbreak can occur, and the expected relative final size of a major outbreak.

The rewiring process contained within the clustered network model is used to investigate the effect of clustering upon the variables of interest, such as the critical vaccination coverage of vaccination strategies. We find that rewiring large households can drastically affect the performance of the epidemic. For example, if the household size distribution has support in the non-negative integers and we rewire a positive fraction households of all sizes, then the basic reproduction number is infinite and the uniform vaccination strategy can only prevent a major outbreak from occurring by vaccinating every individual within the population. Note that the rewired edge-triangle model discussed in Chapter 6 does not contain large households so the rewired edge-triangle model always has a finite basic reproduction number and the uniform vaccination strategy can control the epidemic.



(a) Investigating the effect of clustering on the final size of a major outbreak of the  $p_C = 1$  acquaintance and uniform vaccination strategies.



(b) Investigating the effect of clustering on the critical vaccination coverage of the  $p_C = 1$  acquaintance and uniform vaccination strategies.

Figure 7.5: The models are matched with  $z = 0.5$  and the other parameters are  $H \sim \text{Const}(7)$ ,  $D \sim \text{Const}(3)$  and  $I \sim \text{Const}(1)$  so  $E[D_T] = 9$ .

We consider three approaches to select the rewiring probabilities, for a given clustering coefficient in the clustered network model: rewiring from  $\infty$ , equal rewiring and rewiring from 1. We find that if the household infection rate is large or the model is extremely rewired or not rewired then, for a given clustering coefficient, the final size of a major outbreak under the rewiring from 1 approach is larger than the final size of a major outbreak under the rewiring from  $\infty$  approach. However, if the household infection rate is small or there is moderate rewiring then the final size of a major outbreak under the rewiring from 1 approach is smaller than the final size of a major outbreak under the rewiring from  $\infty$  approach. If the final size of a major outbreak is fixed in the unvaccinated model then for each of the vaccination strategies fixing the clustering coefficient yields that the critical vaccination coverage of the strategy is largest if we follow the rewiring from  $\infty$  approach and smallest if we follow the rewiring from 1 approach.

Similarly to Sections 5.1.4 and 6.6.1, we find that, for a fixed vaccination coverage, maximising  $p_C = p_{CH} = p_{CG}$  maximises the post-vaccination threshold parameter, the final size of a major outbreak and the critical vaccination coverage under the acquaintance vaccination strategy in the clustered network model. Furthermore, similarly to Section 5.4 we find that if the variance of the total degree distribution is small then the critical vaccination coverage under the  $p_C = 1$  acquaintance vaccination strategy is larger than the critical vaccination coverage under the uniform vaccination strategy. Similarly to Section 6.6.1, we find that if the final size of a major outbreak is fixed and the total degree distribution has a small variance then the difference in critical vaccination coverage between the uniform and  $p_C = 1$  acquaintance vaccination strategies increases as the clustering coefficient increases. However, if the variance of the total degree distribution is large then, for a fixed clustering coefficient, the critical vaccination coverage under the uniform vaccination coverage is larger than the critical vaccination coverage under the  $p_C = 1$  acquaintance vaccination strategy, which is larger than the critical vaccination coverage under the  $p_S = 1$  acquaintance vaccination strategy.

We find that if the final size of a major outbreak is fixed and the clustering coefficient is increased then the critical vaccination coverage under the uniform and  $p_S = 1$  acquaintance vaccination strategies will decrease. Furthermore, if the total degree distribution has a large variance then the difference in critical

vaccination coverage between the two strategies will increase with the clustering coefficient, improving the performance of the  $p_S = 1$  acquaintance vaccination strategy compared to the uniform vaccination strategy in clustered networks. We suggest that investigating that the effect of fixing  $R_0$  and increasing the clustering coefficient on these critical vaccination coverages would be an interesting topic to explore further. Especially since in Section 6.6.2 we find that fixing  $R_0$  and changing the clustering coefficient has a negligible effect on the critical vaccination coverage of many of the vaccination strategies applied to the rewired edge-triangle model.

Recently Ball and Sirl (2017) also show that the clustered network model with  $p_{RW}(n) = 0$ ,  $n = 1, 2, \dots$ , under the acquaintance vaccination strategy with  $p_{CH} = 0$  can outperform household-based vaccination strategies if the network degree distribution is heavy-tailed. Therefore another area of further research is to investigate the difference between the acquaintance vaccination strategy with  $p_{CH} = 0$  and with  $p_{CH} > 0$ . Such research is especially highlighted owing to the difficulty in calculating the threshold parameter and final size of a major outbreak on the clustered network model under the acquaintance vaccination strategy with  $p_{CH} > 0$  due to the dependence between the number of sampled and unsampled individuals within a contacted household and the final size of a household epidemic.

## 7.8 Table of common notation introduced in Chapter 7

Symbol	Meaning	Page
$p_{RW}(n)$	Probability that a household of size $n$ is rewired.	239
$\mathcal{C}^H$	Clustering coefficient of the clustered network model.	240
$D_T$	Total degree distribution.	239
$p_S$	Probability that an individual chosen uniformly at random from the population is sampled under the acquaintance vaccination strategy.	241
$p_{CG}$	Probability that a given network neighbour of a sampled individual is vaccinated under the acquaintance vaccination strategy.	241
$p_{CH}$	Probability that a given household or rewired household neighbour of a sampled individual is vaccinated under the acquaintance vaccination strategy.	241
$\tilde{H}_U$	Household size label of an unvaccinated (by the acquaintance vaccination strategy) individual chosen uniformly at random from the population.	242
$\tilde{\rho}_n^U$	$P(\tilde{H}_U = n)$ .	242
$D_U$	Network degree distribution of an unvaccinated (by the acquaintance vaccination strategy) individual chosen uniformly at random from the population.	242
$p_{VG}$	A priori probability that an individual with network degree $D$ is vaccinated (by the acquaintance vaccination strategy) through the network.	243
$\tilde{D}_U$	Network degree distribution of an unvaccinated (by the acquaintance vaccination strategy) individual contacted via the network.	243



$\tilde{p}_V$	A priori probability that an individual contacted via the network is vaccinated (by the acquaintance vaccination strategy).	244
$\tilde{p}_{SU}^{(n,\alpha)}$	Probability that a household of size $n$ contains $\alpha$ sampled non-primary individuals given that the primary infective in the household is unvaccinated.	244
$I_{SS}^{(n,\alpha)}$	Number of sampled non-primary individuals which are vaccinated (by the acquaintance vaccination strategy) in a household of size $n$ with a sampled primary infective and $\alpha$ sampled non-primary individuals.	245
$I_{SN}^{(n,\alpha)}$	Number of unsampled non-primary individuals which are vaccinated (by the acquaintance vaccination strategy) in a household of size $n$ with a sampled primary infective and $\alpha$ sampled non-primary individuals.	245
$T_{SS}^{(n,\alpha)}$	Number of infected sampled non-primary individuals which are vaccinated (by the acquaintance vaccination strategy) in a household of size $n$ with a sampled primary infective and $\alpha$ sampled non-primary individuals.	245
$T_{SN}^{(n,\alpha)}$	Number of infected unsampled non-primary individuals which are vaccinated (by the acquaintance vaccination strategy) in a household of size $n$ with a sampled primary infective and $\alpha$ sampled non-primary individuals.	245
$I_{NS}^{(n,\alpha)}$	Number of sampled non-primary individuals which are vaccinated (by the acquaintance vaccination strategy) in a household of size $n$ with an unsampled primary infective and $\alpha$ sampled non-primary individuals.	245

$I_{NN}^{(n,\alpha)}$	Number of unsampled non-primary individuals which are vaccinated (by the acquaintance vaccination strategy) in a household of size $n$ with an unsampled primary infective and $\alpha$ sampled non-primary individuals.	245
$T_{NS}^{(n,\alpha)}$	Number of infected sampled non-primary individuals which are vaccinated (by the acquaintance vaccination strategy) in a household of size $n$ with an unsampled primary infective and $\alpha$ sampled non-primary individuals.	245
$T_{NN}^{(n,\alpha)}$	Number of infected unsampled non-primary individuals which are vaccinated (by the acquaintance vaccination strategy) in a household of size $n$ with an unsampled primary infective and $\alpha$ sampled non-primary individuals.	245
$\hat{T}_S^{(n)}$	Final size of a rewired household epidemic with household size label $n$ and in which the primary infective is sampled.	246
$\hat{T}_N^{(n)}$	Final size of a rewired household epidemic with household size label $n$ and in which the primary infective is unsampled.	246
$\tilde{p}_H(n)$	$(1 - p_{VG})(1 - \tilde{p}_{SUPCH})^{n-1}p_H$ .	246
$M_{SS}^{(n,\alpha)}$	Number of sampled non-primary individuals within a household susceptibility set when the primary individual is sampled and in a household in a household of size $n$ containing $\alpha$ sampled non-primary individuals.	248
$M_{SN}^{(n,\alpha)}$	Number of unsampled non-primary individuals within a household susceptibility set when the primary individual is sampled and in a household in a household of size $n$ containing $\alpha$ sampled non-primary individuals.	248

$M_{NS}^{(n,\alpha)}$	Number of sampled non-primary individuals within a household susceptibility set when the primary individual is unsampled and in a household in a household of size $n$ containing $\alpha$ sampled non-primary individuals	248
$M_{NN}^{(n,\alpha)}$	Number of unsampled non-primary individuals within a household susceptibility set when the primary individual is unsampled and in a household in a household of size $n$ containing $\alpha$ sampled non-primary individuals.	248
$f_{M_A}^{(n,\alpha)}(s_1, s_2)$	$E \left[ \begin{matrix} M_{(AS)}^{(n,\alpha)} & M_{(AN)}^{(n,\alpha)} \\ s_1 & s_2 \end{matrix} \right], A \in \{S, N\}.$	249
$\hat{M}_S^{(n)}$	Size of a rewired household susceptibility set with household size label $n$ in which the primary individual is sampled.	249
$\hat{M}_N^{(n)}$	Size of a rewired household susceptibility set with household size label $n$ in which the primary individual is unsampled.	249
$R_*^A$	Threshold parameter for the clustered network model under the acquaintance vaccination strategy.	251
$\mu_{T(n,\alpha)}$	Expected number of individuals infected by the non-primary members of a household epidemic with household size $n$ and $\alpha$ sampled non-primary individuals.	251
$\mu_{\hat{T}(n)}$	Expected number of individuals infected by the non-primary members of a rewired household epidemic with household size label $n$ .	251
$R_T^U$	Threshold parameter for the clustered network model under the uniform vaccination strategy.	264
$\mathcal{C}_{max}^H$	Maximum clustering coefficient possible for a given household size and network degree distribution.	271
$\mathcal{C}_1^H$	Clustering coefficient of the clustered network model under the rewiring from 1 approach.	272

$b_1(s)$	Generating function for the initial offspring distribution of the backwards branching process used to calculate the final size of a major outbreak of the clustered network model applying the rewiring from 1 approach.	272
$\tilde{b}_1(s)$	Generating function for the non-initial offspring distribution of the backwards branching process used to calculate the final size of a major outbreak of the clustered network model applying the rewiring from 1 approach.	272
$\mathcal{C}_\infty^H$	Clustering coefficient of the clustered network model under the rewiring from $\infty$ approach.	272
$b_\infty(s)$	Generating function for the initial offspring distribution of the backwards branching process used to calculate the final size of a major outbreak of the clustered network model applying the rewiring from $\infty$ approach.	273
$\tilde{b}_\infty(s)$	Generating function for the non-initial offspring distribution of the backwards branching process used to calculate the final size of a major outbreak of the clustered network model applying the rewiring from $\infty$ approach.	273
$b_d(s)$	$(b_1(s) - b_\infty(s)) / f_D(1 - p_N + p_N s)$ .	273
$R_*^1$	Threshold parameter for the clustered network model applying the approach of rewiring from 1.	273
$R_*^\infty$	Threshold parameter for the clustered network model applying the approach of rewiring from $\infty$ .	273
$z_1$	Final size of a major outbreak on the clustered network model applying the approach of rewiring from 1.	273
$z_\infty$	Final size of a major outbreak on the clustered network model applying the approach of rewiring from $\infty$ .	273

$c_U^*$	Critical vaccination coverage under the uniform vaccination strategy.	280
$c_{p_S}^*$	Critical vaccination coverage under the $p_S = 1$ acquaintance vaccination strategy.	280
$c_{p_C}^*$	Critical vaccination coverage under the $p_C = 1$ acquaintance vaccination strategy.	280

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## 8. Conclusion

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### 8.1 Summary of key results

In Chapter 3 we introduce an SIR epidemic model for the spread of an epidemic among a population of individuals, with a random network of social contacts, that is partitioned into households and in which individuals also make casual contacts. This epidemic model is an extension of previous models such as the households model of Ball et al. (1997), the standard household model, the network and global model of Ball and Neal (2008) and the household and network model of Ball et al. (2009). We introduce a branching process approximation for the early stages of the epidemic in Section 3.2, proving that as the number of households in the population tends to infinity the total number of individuals infected in the epidemic process converges in distribution to the total progeny of a branching process, allowing us to calculate a household-based reproduction number and (in the special case of a constant infectious period) the probability of a major outbreak. We give a heuristic argument that the final size of a major outbreak is equal to the probability that a two-type branching process avoids extinction and thus calculate the expected relative final size of a major outbreak.

In Chapter 4 we set out to investigate the difference in transmitting infections between households via global contacts and via a configuration model network structure. To do this we fix  $R_0$  and use the models of Ball et al. (2009) and Ball and Neal (2008) to investigate the effect network and household heterogeneity on the expected relative final size of a major outbreak. In Section 4.1.1 we show that fixing  $R_0$  and introducing a small amount of network heterogeneity to the homogeneously mixing model increases the final size of a major outbreak if the ratio of the variance to the mean of the degree distribution is less than 1, and decreases the final size of a major outbreak if the ratio of the variance to

the mean of the degree distribution is greater than 1. In contrast, in Section 4.2.1 we show that the effect of fixing  $R_0$  and introducing a small amount of household heterogeneity to the homogeneously mixing model depends on the first three moments of the household size distribution. Importantly, fixing  $R_0$  and introducing either network or household heterogeneity to the model is likely to decrease the final size of a major outbreak if the variance in the household size or degree distribution is large, e.g. if the corresponding distribution has heavy tails. However, in Section 4.1.5 we use the network and global model with a logarithmic degree distribution to prove that fixing  $R_0$  and introducing network heterogeneity to the homogeneously mixing model does not always have a monotonic effect on the final size of a major outbreak.

In Section 4.1.3 we give an ordering for the final size of a major outbreak for a range of degree distributions on the standard network model with fixed  $R_0$ . We conjecture that fixing  $R_0$  and homogenising the degree distribution maximises the expected relative final size of a major outbreak, and increasing the variance of the degree distributions will decrease the expected relative final size of a major outbreak.

Therefore the results in Chapter 4 show that transmitting the disease through global contacts or through a network structure have different effects on the final outcome of the epidemic. Furthermore, the effect of fixing  $R_0$  and introducing a small amount of heterogeneity to the homogeneously mixing model is not necessarily the same as the effect of fixing  $R_0$  and introducing more heterogeneity to an already heterogeneous model.

In Chapter 5 we set out to investigate the performance of the uniform, acquaintance and optimal vaccination strategies applied to the configuration model when the population is also homogeneously mixing, and how these vaccination strategies perform in finite populations. To do this we consider the three vaccination strategies applied to the network and global model. Recall that under the acquaintance vaccination strategy each individual in the population is sampled independently with probability  $p_S$ , and each network neighbour of a sampled individual is independently chosen for vaccination with probability  $p_C$ . Similarly to Ball and Sirl (2013), in Section 5.1.4 we prove that under the acquaintance vaccination strategy with a fixed vaccination coverage maximising  $p_C$  will maximise  $R_0$  and the final size of a major outbreak. Furthermore, in

Section 5.4 we prove conditions under which the critical vaccination coverage of the acquaintance vaccination strategy is less than the critical vaccination coverage of the uniform vaccination strategy, namely if the degree distribution has small variance and  $p_S < 1$ . We conjecture that if  $p_S < 1$  and the degree distribution has small variance then the acquaintance vaccination strategy clusters the vaccination among groups of individuals, and thus large groups of unvaccinated individuals form through which the epidemic can spread unimpeded, causing the vaccination strategy to underperform compared to the uniform vaccination strategy.

In Section 5.5.1 our numerical investigations show that under the uniform or optimal vaccination strategies the asymptotic expected relative final size of a major outbreak is an overestimate for the expected relative final size of a major outbreak in finite populations. However, under the acquaintance vaccination strategy the asymptotic expected relative final size of a major outbreak of the network and global model is an underestimate for the expected relative final size of a major outbreak in finite populations. This is a very undesirable property of the model, since if we vaccinate the asymptotic critical vaccination coverage a major outbreak can still occur in finite populations. Although the vaccination coverage of the uniform vaccination strategy is correlated to both the indicator function for whether a major outbreak occurs and the expected relative final size of a major outbreak, there is much stronger correlation between the vaccination coverage of the acquaintance vaccination strategy and both the indicator function for whether a major outbreak occurs and the expected relative final size of a major outbreak, which we conjecture results in the different behaviour in finite populations.

In Section 5.2.2 we prove that the critical vaccination coverage of the uniform vaccination strategy applied to the network and global model is equal to  $1 - 1/R_0$ , where  $R_0$  is the basic reproduction number of the underlying model under no vaccination strategy. However, in Section 5.5.2 we numerically show that the critical vaccination coverage of the optimal and acquaintance vaccination strategies on the standard network model (fixing either  $R_0$  or expected relative final size of a major outbreak) can either underestimate or overestimate the critical vaccination coverage of the strategy in the homogeneously mixing model, dependent on the variance of the degree distribution. Furthermore, we show that fixing  $R_0$  and increasing the global infection rate leads to a decreased



difference in critical vaccination coverage between the vaccination strategies, thus diluting the benefit of the acquaintance vaccination strategy compared to the uniform vaccination strategy.

In Chapters 6 and 7 we investigate the effect of clustering introduced via edge-disjoint triangles and households respectively on the performance of vaccination strategies. To do so, we introduce two network models with tunable clustering: the rewired edge-triangle model and the clustered network model. The rewired edge-triangle model is based on the network model with clustering of Newman (2009), extended to include a rewiring process to tune the clustering coefficient, and the clustered network model is based on the model of Ball et al. (2013), modified to include a general rewiring process. In Section 6.1.2 we show that the rewired edge-triangle model cannot construct networks with both large mean total degree and large clustering coefficient due to the network only containing edge-disjoint triangles. In contrast, the triangles within the clustered network model need not be edge-disjoint, since the clustered network model contains households (i.e. complete graphs) that can contain more than 3 individuals, so networks with both large mean degree and clustering coefficient can be constructed (see Section 7.1.1). We then consider three vaccination strategies on the rewired edge-triangle model, specifically the optimal, acquaintance and uniform vaccination strategies, and two vaccination strategies on the clustered network model, specifically the acquaintance and uniform vaccination strategies. In Section 7.5 we consider three approaches to calculate the rewiring probabilities ( $p_{RW}(n)$ ,  $n = 1, 2, \dots$ ) for a given clustering coefficient on the clustered network model, specifically rewiring from  $\infty$ , equal rewiring and rewiring from 1.

The rewiring process contained within both the rewired edge-triangle model and the clustered network model is used to investigate the effect of clustering upon the variables of interest, such as the critical vaccination coverage of vaccination strategies, without changing other properties of the model such as the degree correlation. However, we find that rewiring large households can drastically affect the performance of the epidemic on the network. For example, we prove that if the household size distribution has support in the non-negative integers and we rewire a positive fraction  $p_{RW}$  of households of all sizes then  $R_0 = \infty$  (see Section 7.4.2) and that the uniform vaccination strategy can only prevent a major outbreak from occurring by vaccinating every individual within the population (see Section 7.3.3). However, the rewired edge-triangle model

does not contain large households so the rewired edge-triangle model always has finite  $R_0$  (see Section 6.5.2) and the uniform vaccination strategy can always control the epidemic (see Section 6.2.1).

Recall that applying the acquaintance vaccination strategy to the clustered network model involves sampling each individual in the population independently with probability  $p_S$ , and then each global network or household neighbour of a sampled individual is independently chosen for vaccination with probability  $p_{CG}$  or  $p_{CH}$  respectively. In Sections 6.6 and 7.6.2 we numerically investigate the effect of clustering in the rewired edge-triangle and clustered network models on the performance of the vaccination strategies. We find that, in both models, maximising  $p_C = p_{CG} = p_{CH}$  maximises the final size of a major outbreak, the post-vaccination threshold parameter and the critical vaccination coverage under the acquaintance vaccination strategy.

As expected from the conclusions in Section 5.4, our numerical investigations find that in both models if the total degree distribution has small variance and  $p_S < 1$  then the critical vaccination coverage of the acquaintance vaccination strategy is less than the critical vaccination coverage of the uniform vaccination strategy. Interestingly, the difference in critical vaccination coverage between the two vaccination strategies increases as the clustering coefficient increases in both models. Increasing the clustering coefficient in the rewired edge-triangle or clustered network model increases the probability that an individual's neighbours are themselves connected. Therefore, recalling that a sampled individual chooses its neighbours independently with a given probability for vaccination, increasing the clustering coefficient increases the probability that two connected individuals are vaccinated, thus increasing the number of edges joining two vaccinated individuals and decreasing the performance of the acquaintance vaccination strategy.

Our numerical studies find that fixing  $R_0$  and changing the clustering coefficient in the rewired edge-triangle model via the rewiring process generally causes a negligible change in the critical vaccination coverage of the uniform, acquaintance (with  $p_S = 1$ ) and optimal vaccination strategies. Furthermore, fixing the expected relative final size of a major outbreak and increasing the clustering coefficient causes the critical vaccination coverage of these three vaccination strategies to change with  $R_0$  in the underlying model without vaccination. For example, if increasing the clustering coefficient increases  $R_0$ , then the critical

vaccination coverage of the uniform, acquaintance (with  $p_S = 1$ ) and optimal vaccination strategies will also increase. Therefore we conjecture that for a fixed joint stub and triangle distribution the critical vaccination coverages of the three vaccination strategies are determined by  $R_0$ , and are independent of the clustering coefficient. Indeed, we note that this conjecture agrees with the work of House and Keeling (2011). Similarly, we find that fixing the expected relative final size of a major outbreak and increasing the clustering coefficient in the clustered network model via the rewiring process will generally decrease the critical vaccination coverages of the uniform and acquaintance (with  $p_S = 1$ ) vaccination strategies. Therefore we conjecture that the addition of clustering to a network will decrease the critical vaccination coverage of the uniform, optimal and acquaintance (with  $p_S = 1$ ) vaccination strategies.

## 8.2 Future research

We have numerically shown that the asymptotic expected relative final size of a major outbreak of the network and global model under the acquaintance vaccination strategy is an underestimate for the final size of a major outbreak in finite populations. Investigating the causes behind the different correlations between the vaccination coverage and both the indicator function for whether a major outbreak occurs and expected relative final size of a major outbreak in the models under the acquaintance and uniform vaccination strategies is a key area of future research. Indeed, identifying why the acquaintance vaccination strategy increases the correlation between the vaccination coverage and both the indicator function for whether a major outbreak occurs and expected relative final size of a major outbreak may provide insight into ways to further improve the vaccination strategies. Furthermore, future research involves extending this analysis to the single-neighbour acquaintance vaccination strategy of Cohen et al. (2003), to determine whether the undervaccination of the acquaintance vaccination strategy (and potential underperformance compared to the uniform vaccination strategy if  $p_S < 1$ ) is owing to the utilisation of acquaintance-based vaccination schemes, or introduced by the modification of the acquaintance vaccination scheme to allow an individual to choose multiple neighbours for vaccination. It is possible that investigating ‘vaccinated edges’ (by which we mean edges connected to at least one vaccinated vertex) may yield analytical progress in this area.

Ball and Sirl (2017) show that the acquaintance vaccination strategy (with  $p_{CH} = 0$ ) on the clustered network model with no rewiring (i.e.  $p_{RW}(n) = 0$ ,  $n = 1, 2, \dots$ ) can outperform household-based vaccination strategies if the global network degree distribution is heavy-tailed. It would be interesting to investigate the difference between the acquaintance vaccination strategy with  $p_{CH} = 0$  and with  $p_{CH} > 0$ . Such analysis would be particularly interesting since the dependence between the number of sampled and unsampled individuals within a contacted household and the final size of a household epidemic complicates calculations in the acquaintance vaccination strategy we consider (greatly increasing the computational time required for calculations), and these dependencies do not occur if  $p_{CH} = 0$  (i.e. in the acquaintance vaccination strategy of Ball and Sirl (2017)).

Another area of research is to extend the work in Chapter 4 to investigate the effect of introducing heterogeneity to the homogeneously mixing model on the expected relative final size of a major outbreak while fixing parameters other than  $R_0$ . For example, we might consider fixing the early real-time exponential growth rate of the epidemic (See Section 1.3). Alternatively, we can consider the analogous problem of investigating the effect of introducing heterogeneity to the model on the critical vaccination coverage of various vaccination strategies (instead of the final size of a major outbreak), similarly to Becker and Utev (1998).

An assumption we make throughout this thesis is that all vaccination strategies utilize a perfect vaccine, an unrealistic assumption in many practical applications. Therefore an avenue for future work is to extend the vaccination strategies to allow for a generalised vaccine reaction, introduced by Becker and Starczak (1998). Indeed, Ball and Sirl (2017) show that minimising the threshold parameter under household-based vaccination strategies using an imperfect vaccine can result in a larger final size of a major outbreak than the household-based vaccination strategy that maximises the household-based threshold parameter. Therefore the extension of the vaccination strategies to allow for imperfect vaccines on the network and global or household and network models may yield interesting results.

Finally, in the rewired edge-triangle and clustered network models we emphasise that the rewiring process does not change the degree correlation of

the network, since changing the degree correlation can change the properties of the epidemic such as the final size of a major outbreak (see, for example, Miller (2009) or Ball et al. (2013)). Therefore investigating the effect of degree correlation on the performance of vaccination strategies, especially the acquaintance vaccination strategy, is another area of future work. Indeed, it is possible that changes in the degree correlation, rather than variance in the total degree distribution, are responsible for the  $p_C = 1$  acquaintance vaccination strategy underperforming compared to the uniform vaccination strategy.

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## A. Algorithms to simulate the spread of the epidemic

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In this appendix we present pseudo-code algorithms for simulating the spread of an epidemic in a finite population. The results of these simulations can be used to empirically calculate the probability and final size of a major outbreak. In Section A.1 we give the algorithm for the model with three levels of mixing discussed in Chapter 3. In Section A.2 we give modifications to the algorithm which allow for the acquaintance, uniform or optimal vaccination strategies to be applied to the network and global model (see respectively Sections 5.1, 5.2 or 5.3 for the details of these vaccination strategies).

### A.1 Model with three levels of mixing

We first give the pseudo-code algorithm for a single simulation. We begin with the following parameters. The number of households,  $m$ ; the network degree, distributed as  $D$ ; the household size, distributed as  $H$ ; the infectious period, distributed as  $I$ ; the global infection rate,  $\lambda_G$ ; the network infection rate,  $\lambda_N$ ; and the household infection rate,  $\lambda_H$ .

1. For each of the  $m$  households take independent samples from  $H$  to determine their household size. Note that this process fixes the number of individuals in the population,  $N$ , and determines the number of household neighbours of each individual.
2. For each of the  $N$  individuals take independent samples from  $D$ . These samples are the number of half-edges each individual has. Pair all half-edges uniformly at random, discarding a single half-edge chosen uniformly at random if there is an odd total number of half-edges.

3. Select an unvaccinated individual chosen uniformly at random from the population to be the initial infective, and list this individual as currently infected.
4. For each of the  $N$  individuals take independent samples from  $I$  to be that individual's infectious period. So individual  $i$ ,  $i = 1, 2, \dots, N$ , has infectious period  $I_i$ .
5. For each individual in the currently infected list,  $i$  say, add each susceptible household neighbour of  $i$  to the next infected list with probability  $1 - e^{-I_i \lambda_H}$ .
6. For each individual in the currently infected list,  $i$  say, and each network edge emanating from  $i$  which leads to a susceptible individual,  $j$  say, add  $j$  to the next infected list with probability  $1 - e^{-I_i \lambda_N}$ .
7. For each individual in the currently infected list,  $i$  say, take an independent sample from  $\text{Poi}(I_i \lambda_G)$ ,  $G_i$  say. Then choose  $G_i$  individuals independently and uniformly at random from the population with replacement. Each susceptible individual chosen in this way is added to the next infected list.
8. Add the currently infected list to the removed list and then clear the currently infected list.
9. If there is at least 1 individual in the next infected list, move the individuals in the next infected list to the currently infected list, clear the next infected list and go to Step 1. Otherwise continue to Step 10.
10. Note the final size of the epidemic, which is the size of the removed list. To calculate the relative final size of the epidemic divide the final size of the epidemic by the total number of individuals in the population,  $N$ .

After running the required number of simulations, we determine a cut-off for whether a particular final size constitutes a major outbreak by inspecting histograms of the relative final size for our simulations, as described in Section 3.4.1. We can then calculate the empirical probability and final size of a major outbreak as required.

### A.1.1 Comments on the algorithm

Each simulation begins by sampling from  $H$  to construct the households (see Step 1). Therefore each simulation may have a different total population size,  $N$ .

In Step 2 the half-edges are paired uniformly at random, and we do not prevent multiple edges, self-loops, household self-loops or multiple edges between households.

The initial infective chosen in Step 3 is in global generation 0 of the epidemic (see Section 3.2.3). The individuals added to the next infected list in iteration  $i$  of Steps 5 - 7,  $i = 1, 2, \dots$ , are in global generation  $i$  of the epidemic.

For a currently infected individual,  $i$  say, Step 5 considers the household infections made by  $i$ , Step 6 considers the network infections made by  $i$  and Step 7 considers the global contacts made by  $i$ . We do not prevent an individual from contacting another individual multiple times, either through multiple edges or a combination of global, network and household contacts.

## A.2 Network and global model

The majority of the simulation of the spread of the epidemic in the network and global model proceeds analogously to the model with three levels of mixing discussed in Section A.1. The key differences being that we do not construct the household (Step 1), instead starting with  $N = m$  individuals, and that no household infections occur (Step 5). The vaccination strategy is applied after the network is constructed and before the epidemic starts, i.e. between Steps 2 and 3, and involves constructing a vaccinated list. Individuals in the vaccinated list are not susceptible, and they are not involved in the calculation of the final size of the epidemic. Note that vaccination strategies require additional parameters. If we are applying the uniform vaccination strategy we require the probability that each individual is vaccinated,  $p_V$ . If we are applying the acquaintance vaccination strategy we require the probability of sampling,  $p_S$  and the probability of choosing,  $p_C$ . If we are applying the optimal vaccination strategy we require the desired vaccination coverage,  $c$ .

- Under the uniform vaccination strategy: for each of the  $N$  individuals in the population we sample independently from the Uniform distribution



and if the sample is smaller than  $p_V$  add them to the vaccinated list.

- Under the acquaintance vaccination strategy: for each of the  $N$  individuals in the population we sample independently from the Uniform distribution and if the sample is smaller than  $p_S$  add them to the sampled list. Then, for each individual in the sampled list ( $i$  say), add each network neighbour of  $i$  to the chosen list with probability  $p_C$  by sampling from the Uniform distribution. Finally, each individual in the chosen list at least once is added to the vaccinated list. Note that under the acquaintance vaccination strategy we allow for the possibility that an individual chooses itself (via self-loops) and also the possibility that an individual chooses the same individual multiple times if they have parallel edges.
- Under the optimal vaccination strategy: we use the empirical degree distributions to calculate the largest degree of an unvaccinated individual,  $\bar{d}_c$ , and the number of individuals of degree  $\bar{d}_c$  that must be vaccinated,  $\delta^N$ , that results in the desired vaccination coverage  $c$ . We then add all individuals with degree larger than  $\bar{d}_c + 1$  to the vaccinated list and add  $\delta^N$  individuals, chosen uniformly at random without replacement from the individuals with degree  $\bar{d}_c$  by sampling from the Uniform distribution, to the vaccinated list.

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## B. Proof of Theorem 4.4 and Lemmas

### B.1 and B.2

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To give the proof of Theorem 4.4 in Section B.3 we require knowledge of the derivatives of both  $\lambda_G(\lambda_H; R_0, H, I)$  and  $f_{M^{(n)}}(s, \lambda_H)$ . In Section B.1 we discuss the derivatives of  $\lambda_G(\lambda_H; R_0, H, I)$  with respect to  $\lambda_H$ , with preliminary results involving the mean number of cases in each generation of a single household epidemic and their derivatives in Section B.1.1. In Section B.2 we discuss the derivatives of the Gontcharoff Polynomials in Section B.2.1, which are required to calculate the derivatives of  $f_{M^{(n)}}(s, \lambda_H, I)$  given in Section B.2.2. Finally, we give the proof of Theorem 4.4 in Section B.2.2.

For formatting reasons we write  $z(\lambda_H)$ ,  $\lambda_G(\lambda_H)$ ,  $z_H(R_0)$ ,  $f_{M^{(n)}}(s, \lambda_H)$  and  $\mu_{i-1}^{(n)}(\lambda_H)$  for the quantities  $z(\lambda_H; R_0, H, I)$ ,  $\lambda_G(\lambda_H; R_0, H, I)$ ,  $z_H$ ,  $f_{M^{(n)}}(s, \lambda_H; I)$  and  $\mu_{i-1}^{(n)}(\lambda_H; I)$  respectively.

### B.1 Preliminary results involving the global infection rate and its derivatives

#### B.1.1 Calculations involving the mean number of cases in each generation of a single household epidemic and their derivatives

To calculate  $\lambda_G(\lambda_H)$  and its derivatives at the origin we must first consider the mean number of cases in each generation of a single household epidemic and their derivatives. Recall from Section 3.2.3, with notation adjusted to highlight

dependence on  $\lambda_H$ , that, for  $i = 1, 2, \dots, n$ ,

$$\mu_{i-1}^{(n)}(\lambda_H) = \mu_{1,n-1,i-1}(\lambda_H), \quad (\text{B.1})$$

where, for  $a = 1, 2, \dots, s = 1, 2, \dots$ , and  $k = 1, 2, \dots, s$ ,

$$\mu_{a,s,k}(\lambda_H) = \sum_{i=1}^{s-k+1} P_a(s-i, s, \lambda_H) \mu_{i,s-i,k-1}(\lambda_H), \quad (\text{B.2})$$

with boundary conditions

$$\mu_{a,s,0}(\lambda_H) = a, \quad (\text{B.3a})$$

$$\mu_{a,0,k}(\lambda_H) = 0. \quad (\text{B.3b})$$

and, for  $m = 0, 1, \dots, s$ ,

$$P_a(m, s, \lambda_H) = \binom{s}{m} \sum_{k=m}^s (-1)^{k-m} \binom{s-m}{k-m} \phi_I(k\lambda_H)^a. \quad (\text{B.4})$$

**Remark B.1.** We can consider  $\mu_{a,s,k}(\lambda_H)$  as a multivariate polynomial in  $\phi_I(i\lambda_H)$ ,  $i = 1, 2, \dots, s$ , with order less than  $a^k s^k$ . So, given  $\mu_I < \infty$  and  $\phi_I''(0) < \infty$ ,  $\frac{d\mu_{a,s,k}(0)}{d\lambda_H} < \infty$ .

Before considering equations (B.1), (B.2) and (B.4) and their derivatives with respect to  $\lambda_H$ , we introduce the following combinatoric identity, whose proof is given in, for example, Ruiz (1996) Corollary 2.

**Proposition B.1.** For  $n > 0$ ,  $x \in \mathbb{R}$  and  $i = 1, 2, \dots, n$ ,

$$\sum_{j=0}^n (-1)^j \binom{n}{j} (x-j)^{n-i} = 0.$$

We now consider  $P_a(m, s, \lambda_H)$ , given in equation (B.4), and its derivatives with respect to  $\lambda_H$ , evaluated at  $\lambda_H = 0$ .

**Proposition B.2.** For  $a = 1, 2, \dots$ ,  $s = 1, 2, \dots$ , and  $m = 0, 1, \dots, s$ ,

(i)

$$P_a(m, s, 0) = \begin{cases} 1, & \text{if } s = m, \\ 0, & \text{otherwise.} \end{cases} \quad (\text{B.5})$$

(ii)

$$\frac{\partial P_a(m, s, 0)}{\partial \lambda_H} = \begin{cases} -as\mu_I, & \text{if } s = m, \\ as\mu_I, & \text{if } s - m = 1, \\ 0, & \text{otherwise.} \end{cases}$$

(iii)

$$\frac{\partial^2 P_a(m, s, 0)}{\partial \lambda_H^2} = \begin{cases} a [\phi_I''(0) + (a-1)\mu_I^2] s^2, & \text{if } s = m, \\ -a [\phi_I''(0) + (a-1)\mu_I^2] s(2s-1), & \text{if } s - m = 1, \\ a [\phi_I''(0) + (a-1)\mu_I^2] s(s-1), & \text{if } s - m = 2, \\ 0, & \text{otherwise.} \end{cases} \quad (\text{B.6})$$

*Proof.* Evaluating equation (B.4) at  $\lambda_H = 0$  yields

$$P_a(m, s, 0) = \binom{s}{m} \sum_{k=m}^s (-1)^{k-m} \binom{s-m}{k-m}. \quad (\text{B.7})$$

Substituting  $m = s$ , into equation (B.7) immediately yields

$$P_a(s, s, 0) = 1. \quad (\text{B.8})$$

Assume that  $s \geq 2$  and  $m = 1, 2, \dots, s-1$ . Substituting  $j = k - m$  into equation (B.7) and applying Proposition B.1 with  $n = s - m$  and  $i = n$  yields

$$\begin{aligned} P_a(m, s, 0) &= \binom{s}{m} \sum_{k=m}^s (-1)^{k-m} \binom{s-m}{k-m} \\ &= \binom{s}{m} \sum_{j=0}^{s-m} (-1)^j \binom{s-m}{j} \\ &= 0, \end{aligned}$$

which, along with equation (B.8), yields part (i).

Taking the derivative of equation (B.4) with respect to  $\lambda_H$  yields

$$\frac{dP_a(m, s, \lambda_H)}{d\lambda_H} = a \binom{s}{m} \sum_{k=m}^s (-1)^{k-m} \binom{s-m}{k-m} k \phi_I(k\lambda_H)^{a-1} \phi_I'(k\lambda_H), \quad (\text{B.9})$$

and, evaluating equation (B.9) at  $\lambda_H = 0$ ,

$$\frac{dP_a(m, s, 0)}{d\lambda_H} = a \phi_I'(0) \binom{s}{m} \sum_{k=m}^s (-1)^{k-m} \binom{s-m}{k-m} k. \quad (\text{B.10})$$

Substituting  $m = s$  into equation (B.10) yields

$$\frac{dP_a(s, s, 0)}{d\lambda_H} = -as\mu_I. \quad (\text{B.11})$$

Substituting  $m = s - 1$  into equation (B.10) yields

$$\frac{dP_a(s-1, s, 0)}{d\lambda_H} = as\mu_I. \quad (\text{B.12})$$

Assume that  $s \geq 3$  and  $m = 1, 2, \dots, s-2$ . Substituting  $j = k - m$  into equation (B.10) and applying Proposition B.1 with  $n = s - m$ ,  $i = n - 1$  and  $x = -m$  yields

$$\begin{aligned} \frac{dP_a(m, s, 0)}{d\lambda_H} &= a \phi_I'(0) \binom{s}{m} \sum_{k=m}^s (-1)^{k-m} \binom{s-m}{k-m} k \\ &= a \phi_I'(0) \binom{s}{m} \sum_{j=0}^{s-m} (-1)^j \binom{s-m}{j} (j+m) \\ &= 0, \end{aligned}$$

which, along with equations (B.11) and (B.12), yields part (ii).

Taking the derivative of equation (B.9) with respect to  $\lambda_H$  yields

$$\begin{aligned} \frac{d^2P_a(m, s, \lambda_H)}{d\lambda_H^2} &= a \binom{s}{m} \sum_{k=m}^s (-1)^{k-m} \binom{s-m}{k-m} k^2 \left[ \phi_I(k\lambda_H)^{a-1} \phi_I''(k\lambda_H) \right. \\ &\quad \left. + (a-1) \phi_I(k\lambda_H)^{a-2} (\phi_I'(k\lambda_H))^2 \right], \end{aligned} \quad (\text{B.13})$$

and, evaluating equation (B.13) at  $\lambda_H = 0$ ,

$$\frac{d^2P_a(m, s, 0)}{d\lambda_H^2} = a \left[ \phi_I''(0) + (a-1)\mu_I^2 \right] \binom{s}{m} \sum_{k=m}^s (-1)^{k-m} \binom{s-m}{k-m} k^2. \quad (\text{B.14})$$

Substituting  $m = s$  into equation (B.14) yields

$$\frac{d^2 P_a(m, s, 0)}{d\lambda_H^2} = a [\phi_I''(0) + (a-1)\mu_I^2] s^2. \quad (\text{B.15})$$

Substituting  $m = s - 1$  into equation (B.14) yields

$$\frac{d^2 P_a(m, s, 0)}{d\lambda_H^2} = -a [\phi_I''(0) + (a-1)\mu_I^2] s(2s-1). \quad (\text{B.16})$$

Substituting  $m = s - 2$  into equation (B.14) yields

$$\frac{d^2 P_a(m, s, 0)}{d\lambda_H^2} = a [\phi_I''(0) + (a-1)\mu_I^2] s(s-1). \quad (\text{B.17})$$

Assume that  $s \geq 4$  and  $m = 1, 2, \dots, s-3$ . Substituting  $j = k - m$  into equation (B.14) and applying Proposition B.1 with  $n = s - m$ ,  $i = n - 2$  and  $x = -m$  yields

$$\begin{aligned} \frac{d^2 P_a(m, s, 0)}{d\lambda_H^2} &= a [\phi_I''(0) + (a-1)\mu_I^2] \binom{s}{m} \sum_{k=m}^s (-1)^{k-m} \binom{s-m}{k-m} k^2 \\ &= a [\phi_I''(0) + (a-1)\mu_I^2] \binom{s}{m} \sum_{j=0}^{s-m} (-1)^j \binom{s-m}{j} (j+m)^2 \\ &= 0, \end{aligned}$$

which, along with equations (B.15), (B.16) and (B.17) yields part (ii).  $\square$

We now consider the mean generation final sizes and their derivatives evaluated at  $\lambda_H = 0$ .

**Proposition B.3.** *For  $a = 1, 2, \dots$ ,  $s = 1, 2, \dots$ , and  $k = 0, 1, \dots, s$ ,*

(i)

$$\mu_{a,s,k}(0) = \begin{cases} a, & \text{if } k = 0, \\ 0, & \text{otherwise.} \end{cases}$$

(ii)

$$\frac{d\mu_{a,s,k}(0)}{d\lambda_H} = \begin{cases} as\mu_I, & \text{if } k = 1, \\ 0, & \text{otherwise.} \end{cases}$$

(iii)

$$\frac{d^2 \mu_{a,s,k}(0)}{d\lambda_H^2} = \begin{cases} -as [\phi_I''(0) + (a-1)\mu_I^2], & \text{if } k = 1, \\ 2a^2s(s-1)\mu_I^2, & \text{if } k = 2, \\ 0, & \text{otherwise.} \end{cases}$$

*Proof.* Consider part (i). Recall that, from equation (B.3a),  $\mu_{a,s,0}(\lambda_H) = a$ , so  $\mu_{a,s,0}(0) = a$ . Evaluating equation (B.2) at  $\lambda_H = 0$  yields

$$\mu_{a,s,k}(0) = \sum_{i=1}^{s-k+1} P_a(s-i, s, 0) \mu_{i,s-i,k-1}(0). \quad (\text{B.18})$$

So, applying Proposition B.2(i) to equation (B.18), for  $k = 1, 2, \dots, s$ ,

$$\mu_{a,s,k}(0) = 0,$$

which yields part (i).

Taking the derivative of equations (B.3) yields

$$\frac{d\mu_{a,s,0}(\lambda_H)}{d\lambda_H} = 0, \quad (\text{B.19a})$$

$$\frac{d\mu_{a,0,k}(\lambda_H)}{d\lambda_H} = 0. \quad (\text{B.19b})$$

Taking the derivative of equation (B.2) with respect to  $\lambda_H$  yields, for  $k = 1, 2, \dots, s$ ,

$$\begin{aligned} \frac{d\mu_{a,s,k}(\lambda_H)}{d\lambda_H} &= \sum_{i=1}^{s-k+1} \left[ \frac{dP_a(s-i, s, \lambda_H)}{d\lambda_H} \mu_{i,s-i,k-1}(\lambda_H) \right. \\ &\quad \left. + P_a(s-i, s, \lambda_H) \frac{d\mu_{i,s-i,k-1}(\lambda_H)}{d\lambda_H} \right], \end{aligned} \quad (\text{B.20})$$

and, evaluating equation (B.20) at  $\lambda_H = 0$ ,

$$\begin{aligned} \frac{d\mu_{a,s,k}(0)}{d\lambda_H} &= \sum_{i=1}^{s-k+1} \left[ \frac{dP_a(s-i, s, 0)}{d\lambda_H} \mu_{i,s-i,k-1}(0) \right. \\ &\quad \left. + P_a(s-i, s, 0) \frac{d\mu_{i,s-i,k-1}(0)}{d\lambda_H} \right]. \end{aligned} \quad (\text{B.21})$$

Applying Proposition B.2 and Remark B.1 yields

$$\frac{d\mu_{a,s,k}(0)}{d\lambda_H} = as\mu_I\mu_{1,s-1,k-1}(0),$$

and, applying part (i),  $\frac{d\mu_{a,s,k}(0)}{d\lambda_H} = 0$  unless  $k = 1$ , in which case  $\mu_{1,s-1,0}(0) = 1$  and  $\frac{d\mu_{a,s,1}(0)}{d\lambda_H} = as\mu_I$ , which, along with equations (B.19), yields part (ii).

Taking the derivative of equations (B.19) with respect to  $\lambda_H$  yields

$$\frac{d^2\mu_{a,s,0}(\lambda_H)}{d\lambda_H^2} = 0, \quad (\text{B.22a})$$

$$\frac{d^2\mu_{a,0,k}(\lambda_H)}{d\lambda_H^2} = 0. \quad (\text{B.22b})$$

Taking the derivative of equation (B.20) with respect to  $\lambda_H$  yields, for  $k = 1, 2, \dots, s$ ,

$$\begin{aligned} \frac{d^2\mu_{a,s,k}(\lambda_H)}{d\lambda_H^2} = \sum_{i=1}^{s-k+1} & \left[ \frac{d^2P_a(s-i, s, \lambda_H)}{d\lambda_H^2} \mu_{i,s-i,k-1}(\lambda_H) \right. \\ & + 2 \frac{dP_a(s-i, s, \lambda_H)}{d\lambda_H} \frac{d\mu_{i,s-i,k-1}(\lambda_H)}{d\lambda_H} \\ & \left. + P_a(s-i, s, \lambda_H) \frac{d^2\mu_{i,s-i,k-1}(\lambda_H)}{d\lambda_H^2} \right], \end{aligned} \quad (\text{B.23})$$

and, evaluating equation (B.23) at  $\lambda_H = 0$ ,

$$\begin{aligned} \frac{d^2\mu_{a,s,k}(0)}{d\lambda_H^2} = \sum_{i=1}^{s-k+1} & \left[ \frac{d^2P_a(s-i, s, 0)}{d\lambda_H^2} \mu_{i,s-i,k-1}(0) \right. \\ & + 2 \frac{dP_a(s-i, s, 0)}{d\lambda_H} \frac{d\mu_{i,s-i,k-1}(0)}{d\lambda_H} \\ & \left. + P_a(s-i, s, 0) \frac{d^2\mu_{i,s-i,k-1}(0)}{d\lambda_H^2} \right]. \end{aligned} \quad (\text{B.24})$$

Applying Proposition B.2 and Remark B.1 to equation (B.24) yields

$$\begin{aligned} \frac{d^2\mu_{a,s,k}(0)}{d\lambda_H^2} = & -a \left[ \phi_I''(0) + (a-1)\mu_I^2 \right] s(2s-1)\mu_{1,s-1,k-1}(0) \\ & + a \left[ \phi_I''(0) + (a-1)\mu_I^2 \right] s(s-1)\mu_{2,s-2,k-1}(0) \\ & + 2as\mu_I \frac{d\mu_{1,s-1,k-1}(0)}{d\lambda_H}. \end{aligned} \quad (\text{B.25})$$



Applying parts (i) and (ii) to equation (B.25) yields  $\frac{d^2\mu_{a,s,k}(0)}{d\lambda_H^2} = 0$  unless  $k = 1$  or  $k = 2$ .

If  $k = 1$ , then  $\mu_{1,s-1,k-1}(0) = 1$ ,  $\mu_{2,s-2,k-1}(0) = 2$  and  $\frac{d\mu_{1,s-1,k-1}(0)}{d\lambda_H} = 0$ , so

$$\frac{d^2\mu_{a,s,1}(0)}{d\lambda_H^2} = -as \left[ \phi_I''(0) + (a-1)\mu_I^2 \right]. \quad (\text{B.26})$$

If  $k = 2$ , then  $\mu_{1,s-1,k-1}(0) = 0$ ,  $\mu_{2,s-2,k-1}(0) = 0$  and  $\frac{d\mu_{1,s-1,k-1}(0)}{d\lambda_H} = a(s-1)\mu_I$ , so

$$\frac{d^2\mu_{a,s,2}(0)}{d\lambda_H^2} = 2a^2s(s-1)\mu_I^2. \quad (\text{B.27})$$

Equations (B.22), (B.26) and (B.27), along with the result  $\frac{d^2\mu_{a,s,k}(0)}{d\lambda_H^2} = 0$  for  $k \geq 2$ , yields part (iii).  $\square$

Finally, we consider the derivatives of the mean number of cases in each generation of a single household epidemic and their derivatives.

**Proposition B.4.** *For  $n = 1, 2, \dots$ ,  $i = 1, 2, \dots, n$ ,*

(i)

$$\mu_{i-1}^{(n)}(0) = \begin{cases} 1, & \text{if } i = 1, \\ 0, & \text{otherwise.} \end{cases}$$

(ii)

$$\frac{d\mu_{i-1}^{(n)}}{d\lambda_H}(0) = \begin{cases} (n-1)\mu_I, & \text{if } i = 1, \\ 0, & \text{otherwise.} \end{cases}$$

(iii)

$$\frac{d^2\mu_{i-1}^{(n)}}{d\lambda_H^2}(0) = \begin{cases} -(n-1)\phi_I''(0), & \text{if } i = 1, \\ 2(n-1)(n-2)\mu_I^2, & \text{if } i = 2, \\ 0, & \text{otherwise.} \end{cases}$$

*Proof.* Evaluating equation (B.1) at  $\lambda_H = 0$  and applying Proposition B.4(i) yields

$$\mu_{i-1}^{(n)}(0) = \mu_{1,n-1,i-1}(0) = \begin{cases} 1, & \text{if } i = 1, \\ 0, & \text{otherwise,} \end{cases}$$

as required for part (i).

Taking the derivative of equation (B.1) with respect to  $\lambda_H$  and applying Proposition B.4(ii) yields

$$\frac{d\mu_{i-1}^{(n)}(0)}{d\lambda_H} = \frac{d\mu_{1,n-1,i-1}(0)}{d\lambda_H} = \begin{cases} (n-1)\mu_I, & \text{if } i = 1, \\ 0, & \text{otherwise,} \end{cases} \quad (\text{B.28})$$

as required for part (ii).

Taking the derivative of equation (B.28) with respect to  $\lambda_H$  and applying Proposition B.4(iii) yields

$$\frac{d^2\mu_{i-1}^{(n)}(0)}{d\lambda_H^2} = \frac{d^2\mu_{1,n-1,i-1}(0)}{d\lambda_H^2} = \begin{cases} -(n-1)\phi_I''(0), & \text{if } i = 1, \\ 2(n-1)(n-2)\mu_I^2, & \text{if } i = 2, \\ 0, & \text{otherwise,} \end{cases} \quad (\text{B.29})$$

as required for part (iii).  $\square$

### B.1.2 Derivatives of the global infection rate

We now calculate the derivatives of  $\lambda_G(\lambda_H)$  at the origin.

**Lemma B.1.**

(i)

$$\lambda_G(0) = \frac{R_0}{\mu_I},$$

(ii)

$$\lambda'_G(0) = -\left(\mathbb{E}[\tilde{H}] - 1\right),$$

(iii)

$$\lambda''_G(0) = \frac{\phi_I''(0)}{\mu_I} \left(\mathbb{E}[\tilde{H}] - 1\right) + \frac{2\mu_I}{R_0} \left\{ \mathbb{E}[\tilde{H}]^2 - \mathbb{E}[\tilde{H}^2] + \mathbb{E}[\tilde{H}] - 1 \right\}.$$

*Proof.* Evaluating equation (4.76) at  $\lambda_H = 0$  and applying Proposition B.4(i) yields part (i),

$$\lambda_G(0) = \frac{R_0}{\mu_I}.$$

Taking the derivative of equation (4.76) with respect to  $\lambda_H$  yields

$$\lambda'_G(\lambda_H)\mu_I \sum_{i=1}^{\infty} R_0^{-i} \sum_{n=i}^{\infty} \tilde{\rho}_n \mu_{i-1}^{(n)}(\lambda_H) + \lambda_G(\lambda_H)\mu_I \sum_{i=1}^{\infty} R_0^{-i} \sum_{n=i}^{\infty} \tilde{\rho}_n \frac{d\mu_{i-1}^{(n)}}{d\lambda_H}(\lambda_H) = 0. \quad (\text{B.30})$$

Evaluating equation (B.30) at  $\lambda_H = 0$  and applying part (i) and Proposition B.4 yields

$$0 = \lambda'_G(0)\frac{\mu_I}{R_0} + \frac{\mu_I}{R_0} \sum_{n=2}^{\infty} \tilde{\rho}_n(n-1),$$

from which part (ii) follows with rearrangement.

Taking the derivative of equation (B.30) with respect to  $\lambda_H$  yields

$$\begin{aligned} 0 = & \lambda''_G(\lambda_H)\mu_I \sum_{i=1}^{\infty} R_0^{-i} \sum_{n=i}^{\infty} \tilde{\rho}_n \mu_{i-1}^{(n)}(\lambda_H) + 2\lambda'_G(\lambda_H)\mu_I \sum_{i=1}^{\infty} R_0^{-i} \sum_{n=i}^{\infty} \tilde{\rho}_n \frac{d\mu_{i-1}^{(n)}}{d\lambda_H}(\lambda_H) \\ & + \lambda_G(\lambda_H)\mu_I \sum_{i=1}^{\infty} R_0^{-i} \sum_{n=i}^{\infty} \tilde{\rho}_n \frac{d^2\mu_{i-1}^{(n)}}{d\lambda_H^2}(\lambda_H). \end{aligned} \quad (\text{B.31})$$

Evaluating equation (B.31) at  $\lambda_H = 0$  and applying parts (i) and (ii) and Proposition B.4 yields

$$\begin{aligned} 0 = & \lambda''_G(0)\mu_I R_0^{-1} - 2\mu_I^2 R_0^{-2} \left( \mathbb{E} [\tilde{H}] - 1 \right) \sum_{n=1}^{\infty} \tilde{\rho}_n(n-1) \\ & + R_0 \left( -R_0^{-2} \phi''_I(0) \sum_{n=2}^{\infty} \tilde{\rho}_n(n-1) + 2R_0^{-3} \mu_I^2 \sum_{n=3}^{\infty} \tilde{\rho}_n(n-1)(n-2) \right) \\ = & \mu_I R_0^{-1} \left[ \lambda''_G(0) - 2\mu_I R_0^{-1} (\mathbb{E} [\tilde{H}] - 1) \sum_{n=1}^{\infty} \tilde{\rho}_n(n-1) \right. \\ & \left. - \frac{1}{\mu_I} \phi''_I(0) \sum_{n=1}^{\infty} \tilde{\rho}_n(n-1) + 2\mu_I R_0^{-1} \sum_{n=1}^{\infty} \tilde{\rho}_n(n-1)(n-2) \right] \\ = & \mu_I R_0^{-1} \left[ \lambda''_G(0) - \frac{\phi''_I(0)}{\mu_I} (\mathbb{E} [\tilde{H}] - 1) \right. \\ & \left. + \frac{2\mu_I}{R_0} \left\{ \mathbb{E} [(\tilde{H}) - 1] (\tilde{H} - 2) - (\mathbb{E} [\tilde{H}] - 1)^2 \right\} \right] \\ = & \mu_I R_0^{-1} \left[ \lambda''_G(0) - \frac{\phi''_I(0)}{\mu_I} (\mathbb{E} [\tilde{H}] - 1) \right. \\ & \left. + \frac{2\mu_I}{R_0} \left\{ \mathbb{E} [\tilde{H}^2] - \mathbb{E} [\tilde{H}]^2 - \mathbb{E} [\tilde{H}] + 1 \right\} \right], \end{aligned}$$

from which part (iii) follows with rearrangement. □

## B.2 Preliminary results involving the size of a household susceptibility set and its derivatives

To calculate the derivative of  $f_{M(n)}(s, \lambda_H)$  with respect to  $\lambda_H$  we first need expressions for the partial derivatives of the Gontcharoff polynomials.

### B.2.1 The Gontcharoff Polynomials

We first introduced the Gontcharoff polynomials in Section 2.2. For Section B.2 we adjust our notation to highlight the dependence on  $\lambda_H$  in the equations we consider. Recall that  $G_0(x \mid \mathbf{V}(\lambda_H)) = 1$  and, for  $k = 1, 2, \dots$ ,

$$G_k(x \mid \mathbf{V}(\lambda_H)) = \frac{x^k}{k!} - \sum_{j=0}^{k-1} \frac{\phi_I((j+1)\lambda_H)^{k-j}}{(k-j)!} G_j(x \mid \mathbf{V}(\lambda_H)), \quad (\text{B.32})$$

where  $\mathbf{V}(\lambda_H) = (q_{i+1}(\lambda_H), i = 0, 1, \dots)$  and  $q_k(\lambda_H) = \phi_I(k\lambda_H)$ . Note that we need only consider the case  $x = 1$ , see equation (3.29). Furthermore, at  $\lambda_H = 0$ ,  $q_k(0) = 1$ ,  $k = 1, 2, \dots$ , and  $\mathbf{V}(0) = \mathbf{1} = (1, 1, 1, \dots)$ .

**Proposition B.5.**

(i)

$$G_k(1 \mid \mathbf{V}(0)) = \begin{cases} 1, & \text{if } k = 0, \\ 0, & \text{otherwise.} \end{cases} \quad (\text{B.33})$$

(ii)

$$\frac{\partial G_k(1 \mid \mathbf{V}(0))}{\partial \lambda_H} = \begin{cases} \mu_I, & \text{if } k = 1, \\ 0, & \text{otherwise.} \end{cases} \quad (\text{B.34})$$

(iii)

$$\frac{\partial^2 G_k(1 \mid \mathbf{V}(0))}{\partial \lambda_H^2} = \begin{cases} -\phi_I''(0), & \text{if } k = 1, \\ 3\mu_I^2, & \text{if } k = 2, \\ 0, & \text{otherwise.} \end{cases} \quad (\text{B.35})$$

*Proof.* We prove all three parts of Proposition B.5 by complete induction. Note

that the first three Gontcharoff polynomials are equal to:

$$G_0(x \mid \mathbf{V}(\lambda_H)) = 1, \quad (\text{B.36a})$$

$$G_1(x \mid \mathbf{V}(\lambda_H)) = x - \phi_I(\lambda_H), \quad (\text{B.36b})$$

$$G_2(x \mid \mathbf{V}(\lambda_H)) = \frac{x^2}{2} - \phi_I(2\lambda_H)x + \phi_I(\lambda_H)\phi_I(2\lambda_H) - \frac{\phi_I(\lambda_H)^2}{2}. \quad (\text{B.36c})$$

$$\begin{aligned} G_3(x \mid \mathbf{V}(\lambda_H)) &= \frac{x^3}{6} - \frac{\phi_I(3\lambda_H)x^2}{2} + \left( \phi_I(2\lambda_H)\phi_I(3\lambda_H) - \frac{\phi_I(2\lambda_H)^2}{2} \right) x \\ &\quad + \frac{\phi_I(\lambda_H)\phi_I(2\lambda_H)^2}{2} + \frac{\phi_I(\lambda_H)^2\phi_I(3\lambda_H)}{2} \\ &\quad - \phi_I(\lambda_H)\phi_I(2\lambda_H)\phi_I(3\lambda_H) - \frac{\phi_I(\lambda_H)^3}{6}. \end{aligned} \quad (\text{B.36d})$$

Consider part (i). For the base cases, substituting  $x = 1$  and  $\lambda_H = 0$  into equations (B.36a) and (B.36b) yields  $G_0(1 \mid \mathbf{V}(0)) = 1$  and  $G_1(1 \mid \mathbf{V}(0)) = 0$  respectively.

Fix  $k \geq 2$  and, for the induction step, assume the induction hypothesis that, for  $i = 1, 2, \dots, k-1$ ,  $G_i(1 \mid \mathbf{V}(0)) = 0$ . Substituting  $\lambda_H = 0$  into equation (B.32) yields, for  $k = 2, 3, \dots$ ,

$$G_k(x \mid \mathbf{V}(0)) = \frac{x^k}{k!} - \sum_{j=0}^{k-1} \frac{1}{(k-j)!} G_j(x \mid \mathbf{V}(0)). \quad (\text{B.37})$$

Evaluating equation (B.37) at  $x = 1$  and applying the induction hypothesis yields, for  $k = 2, 3, \dots$ ,

$$\begin{aligned} G_k(1 \mid \mathbf{V}(0)) &= \frac{1}{k!} - \sum_{j=0}^{k-1} \frac{1}{(k-j)!} G_j(1 \mid \mathbf{V}(0)) \\ &= \frac{1}{k!} - \frac{1}{k!} G_0(1 \mid \mathbf{V}(0)), \\ &= 0, \end{aligned}$$

so part (i) holds.

Next consider part (ii). Taking the derivative of equations (B.36) with

respect to  $\lambda_H$  yields

$$\frac{dG_0(x \mid \mathbf{V}(\lambda_H))}{d\lambda_H} = 0, \quad (\text{B.38a})$$

$$\frac{dG_1(x \mid \mathbf{V}(\lambda_H))}{d\lambda_H} = -\phi'_I(\lambda_H), \quad (\text{B.38b})$$

$$\begin{aligned} \frac{dG_2(x \mid \mathbf{V}(\lambda_H))}{d\lambda_H} &= -2\phi'_I(2\lambda_H)x + \phi'_I(\lambda_H)\phi_I(2\lambda_H) \\ &\quad + 2\phi_I(\lambda_H)\phi'_I(2\lambda_H) - \phi'_I(\lambda_H)\phi_I(\lambda_H). \end{aligned} \quad (\text{B.38c})$$

$$\begin{aligned} \frac{dG_3(x \mid \mathbf{V}(\lambda_H))}{d\lambda_H} &= -\frac{3\phi'_I(3\lambda_H)x^2}{2} + (2\phi'_I(2\lambda_H)\phi_I(3\lambda_H) + 3\phi_I(2\lambda_H)\phi'_I(3\lambda_H))x \\ &\quad - 2\phi'_I(2\lambda_H)\phi_I(2\lambda_H)x + \frac{\phi'_I(\lambda_H)\phi_I(2\lambda_H)^2}{2} \\ &\quad + 2\phi_I(\lambda_H)\phi'_I(2\lambda_H)\phi_I(2\lambda_H) \\ &\quad + \phi'_I(\lambda_H)\phi_I(\lambda_H)\phi_I(3\lambda_H) + \frac{3\phi_I(\lambda_H)^2\phi'_I(3\lambda_H)}{2} \\ &\quad - \phi'_I(\lambda_H)\phi_I(2\lambda_H)\phi_I(3\lambda_H) - 2\phi_I(\lambda_H)\phi'_I(2\lambda_H)\phi_I(3\lambda_H) \\ &\quad - 3\phi_I(\lambda_H)\phi_I(2\lambda_H)\phi'_I(3\lambda_H) - \frac{\phi_I(\lambda_H)^2\phi'_I(\lambda_H)}{2}. \end{aligned} \quad (\text{B.38d})$$

Therefore, for the base cases, recalling that  $\phi'_I(0) = -\mu_I$  and substituting  $x = 1$  and  $\lambda_H = 0$  into equations (B.38a), (B.38b) and (B.38c) yields

$$\begin{aligned} \frac{dG_0(x \mid \mathbf{V}(0))}{d\lambda_H} &= 0, \\ \frac{dG_1(x \mid \mathbf{V}(0))}{d\lambda_H} &= \mu_I, \\ \frac{dG_2(x \mid \mathbf{V}(0))}{d\lambda_H} &= 0. \end{aligned}$$

Fix  $k \geq 3$  and, for the induction step, assume the induction hypothesis that, for  $i = 2, 3, \dots, k-1$ ,  $\frac{dG_i(1 \mid \mathbf{V}(0))}{d\lambda_H} = 0$ . Taking the derivative of equation (B.32) with respect to  $\lambda_H$  yields

$$\begin{aligned} \frac{dG_k(x \mid \mathbf{V}(\lambda_H))}{d\lambda_H} &= \sum_{j=0}^{k-1} \left[ \frac{-(j+1)\phi_I((j+1)\lambda_H)^{k-j-1} \phi'_I((j+1)\lambda_H)}{(k-j-1)!} G_j(x \mid \mathbf{V}(\lambda_H)) \right. \\ &\quad \left. - \frac{\phi_I((j+1)\lambda_H)^{k-j}}{(k-j)!} \frac{dG_j(x \mid \mathbf{V}(\lambda_H))}{d\lambda_H} \right] \end{aligned} \quad (\text{B.39})$$

Substituting  $\lambda_H = 0$  into equation (B.39) yields, for  $k = 3, 4, \dots$ ,

$$\begin{aligned} \frac{dG_k(x \mid \mathbf{V}(0))}{d\lambda_H} &= \sum_{j=0}^{k-1} \left[ \frac{(j+1)\mu_I}{(k-j-1)!} G_j(x \mid \mathbf{V}(0)) \right. \\ &\quad \left. - \frac{1}{(k-j)!} \frac{dG_j(x \mid \mathbf{V}(0))}{d\lambda_H} \right] \end{aligned} \quad (\text{B.40})$$

Evaluating equation (B.40) at  $x = 1$ , applying part (i) and the induction hypothesis yields, for  $k = 3, 4, \dots$ ,

$$\begin{aligned} \frac{dG_k(1 \mid \mathbf{V}(0))}{d\lambda_H} &= \sum_{j=0}^{k-1} \left[ \frac{(j+1)\mu_I}{(k-j-1)!} G_j(1 \mid \mathbf{V}(0)) \right. \\ &\quad \left. - \frac{1}{(k-j)!} \frac{dG_j(1 \mid \mathbf{V}(0))}{d\lambda_H} \right] \\ &= \frac{\mu_I}{(k-1)!} G_0(1 \mid \mathbf{V}(0)) - \frac{1}{(k-1)!} \frac{dG_1(1 \mid \mathbf{V}(0))}{d\lambda_H} \\ &= 0, \end{aligned}$$

so part (ii) holds.

Finally, we consider part (iii). Taking the derivative of equations (B.38) with

respect to  $\lambda_H$  yields

$$\frac{d^2 G_0(x \mid \mathbf{V}(\lambda_H))}{d\lambda_H^2} = 0, \quad (\text{B.41a})$$

$$\frac{d^2 G_1(x \mid \mathbf{V}(\lambda_H))}{d\lambda_H^2} = -\phi_I''(\lambda_H), \quad (\text{B.41b})$$

$$\begin{aligned} \frac{d^2 G_2(x \mid \mathbf{V}(\lambda_H))}{d\lambda_H^2} = & -4\phi_I''(2\lambda_H)x + \phi_I''(\lambda_H)\phi_I(2\lambda_H) + 4\phi_I'(\lambda_H)\phi_I'(2\lambda_H) \\ & + 4\phi_I(\lambda_H)\phi_I''(2\lambda_H) - \phi_I''(\lambda_H)\phi_I(\lambda_H) - \phi_I'(\lambda_H)^2. \end{aligned} \quad (\text{B.41c})$$

$$\begin{aligned} \frac{d^2 G_3(x \mid \mathbf{V}(\lambda_H))}{d\lambda_H^2} = & -\frac{9\phi_I''(3\lambda_H)x^2}{2} + 4\phi_I''(2\lambda_H)\phi_I(3\lambda_H)x + 12\phi_I'(2\lambda_H)\phi_I'(3\lambda_H)x \\ & + 9\phi_I(2\lambda_H)\phi_I''(3\lambda_H)x - 4\phi_I''(2\lambda_H)\phi_I(2\lambda_H)x - 4\phi_I'(2\lambda_H)^2x \\ & + \frac{\phi_I''(\lambda_H)\phi_I(2\lambda_H)^2}{2} + 4\phi_I'(\lambda_H)\phi_I(2\lambda_H)\phi_I'(2\lambda_H) \\ & + 4\phi_I(\lambda_H)\phi_I''(2\lambda_H)\phi_I(2\lambda_H) + 4\phi_I(\lambda_H)\phi_I'(2\lambda_H)^2 \\ & + \phi_I''(\lambda_H)\phi_I(\lambda_H)\phi_I(3\lambda_H) + \phi_I'(\lambda_H)^2\phi_I(3\lambda_H) \\ & - \phi_I''(\lambda_H)\phi_I(2\lambda_H)\phi_I(3\lambda_H) + 6\phi_I'(\lambda_H)\phi_I(\lambda_H)\phi_I'(3\lambda_H) \\ & + \frac{9\phi_I(\lambda_H)^2\phi_I''(3\lambda_H)}{2} - 4\phi_I'(\lambda_H)\phi_I'(2\lambda_H)\phi_I(3\lambda_H) \\ & - 6\phi_I'(\lambda_H)\phi_I(2\lambda_H)\phi_I'(3\lambda_H) - 4\phi_I(\lambda_H)\phi_I''(2\lambda_H)\phi_I(3\lambda_H) \\ & - 12\phi_I(\lambda_H)\phi_I'(2\lambda_H)\phi_I'(3\lambda_H) - 9\phi_I(\lambda_H)\phi_I(2\lambda_H)\phi_I''(3\lambda_H) \\ & - \phi_I(\lambda_H)(\phi_I'(\lambda_H))^2 - \frac{\phi_I(\lambda_H)^2\phi_I''(\lambda_H)}{2}. \end{aligned} \quad (\text{B.41d})$$

Therefore, for the base cases, substituting  $x = 1$  and  $\lambda_H = 0$  into equations (B.41) yields

$$\begin{aligned} \frac{d^2 G_0(x \mid \mathbf{V}(\lambda_H))}{d\lambda_H^2} &= 0, \\ \frac{d^2 G_1(x \mid \mathbf{V}(\lambda_H))}{d\lambda_H^2} &= -\phi_I''(0), \\ \frac{d^2 G_2(x \mid \mathbf{V}(\lambda_H))}{d\lambda_H^2} &= 3\mu_I^2 \\ \frac{d^2 G_3(x \mid \mathbf{V}(\lambda_H))}{d\lambda_H^2} &= 0. \end{aligned}$$

Fix  $k \geq 4$  and, for the induction step, assume the induction hypothesis that, for  $i = 3, 4, \dots, k-1$ ,  $\frac{d^2 G_i(1 \mid \mathbf{V}(0))}{d\lambda_H^2} = 0$ . Taking the derivative of equation (B.39)



with respect to  $\lambda_H$  yields

$$\begin{aligned}
\frac{d^2 G_k(x | \mathbf{V}(\lambda_H))}{d\lambda_H^2} = & \sum_{j=0}^{k-2} \left[ \frac{-(j+1)^2 \phi_I((j+1)\lambda_H)^{k-j-2}}{(k-j-2)!} \left( (\phi'_I((j+1)\lambda_H))^2 \right. \right. \\
& \left. \left. + \frac{\phi_I((j+1)\lambda_H) \phi''_I((j+1)\lambda_H)}{k-j-1} \right) G_j(x | \mathbf{V}(\lambda_H)) \right. \\
& - \frac{2(j+1)\phi_I((j+1)\lambda_H)^{k-j-1} \phi'_I((j+1)\lambda_H)}{(k-j-1)!} \frac{dG_j(x | \mathbf{V}(\lambda_H))}{d\lambda_H} \\
& \left. - \frac{\phi_I((j+1)\lambda_H)^{k-j}}{(k-j)!} \frac{d^2 G_j(x | \mathbf{V}(\lambda_H))}{d\lambda_H^2} \right] \\
& - k^2 \phi''_I(k\lambda_H) G_{k-1}(x | \mathbf{V}(\lambda_H)) - 2k \phi'_I(k\lambda_H) \frac{dG_{k-1}(x | \mathbf{V}(\lambda_H))}{d\lambda_H} \\
& - \phi_I(k\lambda_H) \frac{d^2 G_{k-1}(x | \mathbf{V}(\lambda_H))}{d\lambda_H^2}. \tag{B.42}
\end{aligned}$$

Substituting  $\lambda_H = 0$  into equation (B.42) yields, for  $k = 4, 5, \dots$ ,

$$\begin{aligned}
\frac{d^2 G_k(x | \mathbf{V}(0))}{d\lambda_H^2} = & \sum_{j=0}^{k-2} \left[ \frac{2(j+1)\mu_I}{(k-j-1)!} \frac{dG_j(x | \mathbf{V}(0))}{d\lambda_H} - \frac{1}{(k-j)!} \frac{d^2 G_j(x | \mathbf{V}(0))}{d\lambda_H^2} \right. \\
& \left. - \frac{(j+1)^2}{(k-j-2)!} \left( \mu_I^2 + \frac{\phi''_I(0)}{k-j-1} \right) G_j(x | \mathbf{V}(0)) \right] \\
& - k^2 \phi''_I(0) G_{k-1}(x | \mathbf{V}(0)) + 2k\mu_I \frac{dG_{k-1}(x | \mathbf{V}(0))}{d\lambda_H} \\
& - \frac{d^2 G_{k-1}(x | \mathbf{V}(0))}{d\lambda_H^2}. \tag{B.43}
\end{aligned}$$

Evaluating equation (B.43) at  $x = 1$ , applying parts (i) and (ii) and the induction hypothesis yields, for  $k = 4, 5, \dots$ ,

$$\begin{aligned}
\frac{d^2 G_k(1 | \mathbf{V}(0))}{d\lambda_H^2} = & \frac{-1}{(k-2)!} \left( \mu_I^2 + \frac{\phi''_I(0)}{k-1} \right) G_0(1 | \mathbf{V}(0)) \\
& + \frac{4\mu_I}{(k-2)!} \frac{dG_1(1 | \mathbf{V}(0))}{d\lambda_H} - \frac{1}{(k-1)!} \frac{d^2 G_1(1 | \mathbf{V}(0))}{d\lambda_H^2} \\
& - \frac{1}{(k-2)!} \frac{d^2 G_2(1 | \mathbf{V}(0))}{d\lambda_H^2} \\
= & \frac{-1}{(k-2)!} \left( \mu_I^2 + \frac{\phi''_I(0)}{k-1} \right) + \frac{4\mu_I^2}{(k-2)!} + \frac{\phi''_I(0)}{(k-1)!} - \frac{3\mu_I^2}{(k-2)!} \\
= & 0,
\end{aligned}$$

so part (iii) holds. □

## B.2.2 Derivatives of the size of a household susceptibility set

Recall from equation (3.29),

$$f_{M^{(n)}}(s, \lambda_H) = \sum_{k=0}^{n-1} s^k \frac{(n-1)!}{(n-1-k)!} \phi_I((k+1)\lambda_H)^{n-1-k} G_k(1|\mathbf{V}(\lambda_H)). \quad (\text{B.44})$$

Considering the derivatives of equation B.44 evaluated at  $\lambda_H = 0$  yields the following Proposition.

**Lemma B.2.** *For  $n = 1, 2, \dots$ ,  $s \in [0, 1]$ ,*

(i)

$$f_{M^{(n)}}(s, 0) = 1,$$

(ii)

$$\frac{\partial f_{M^{(n)}}(s, 0)}{\partial s} = 0,$$

(iii)

$$\frac{\partial^2 f_{M^{(n)}}(s, 0)}{\partial s^2} = 0,$$

(iv)

$$\frac{\partial f_{M^{(n)}}(s, 0)}{\partial \lambda_H} = -(1-s)(n-1)\mu_I,$$

(v)

$$\frac{\partial^2 f_{M^{(n)}}(s, 0)}{\partial \lambda_H^2} = (n-1) \left[ \phi_I''(0) (1-s) + \mu_I^2 (n-2) (1-4s+3s^2) \right],$$

(vi)

$$\frac{\partial^2 f_{M^{(n)}}(s, 0)}{\partial s \partial \lambda_H} = (n-1)\mu_I.$$

*Proof.* Taking the first and second derivatives of equation (B.44) with respect to  $s$  yields

$$\frac{\partial f_{M^{(n)}}(s, \lambda_H)}{\partial s} = \sum_{k=1}^{n-1} k s^{k-1} \frac{(n-1)!}{(n-1-k)!} \phi_I((k+1)\lambda_H)^{n-1-k} G_k(1|\mathbf{V}(\lambda_H)) \quad (\text{B.45a})$$

and

$$\frac{\partial^2 f_{M^{(n)}}(s, \lambda_H)}{\partial s^2} = \sum_{k=2}^{n-1} k(k-1)s^{k-2} \frac{(n-1)!}{(n-1-k)!} \phi_I((k+1)\lambda_H)^{n-1-k} G_k(1|\mathbf{V}(\lambda_H)) \quad (\text{B.45b})$$

respectively.

Evaluating equations (B.45) at  $\lambda_H = 0$  and applying Proposition B.5(i) yields parts (ii) and (iii).

For  $n \geq 2$ , taking the derivative of equation (B.44) with respect to  $\lambda_H$  yields

$$\begin{aligned} \frac{\partial f_{M^{(n)}}(s, \lambda_H)}{\partial \lambda_H} &= \sum_{k=0}^{n-1} s^k \frac{(n-1)!}{(n-1-k)!} \phi_I((k+1)\lambda_H)^{n-1-k} \frac{dG_k(1|\mathbf{V}(\lambda_H))}{d\lambda_H} \\ &\quad + \sum_{k=0}^{n-2} s^k \frac{(n-1)!(k+1)}{(n-2-k)!} \phi_I((k+1)\lambda_H)^{n-2-k} \phi'_I((k+1)\lambda_H) G_k(1|\mathbf{V}(\lambda_H)). \end{aligned} \quad (\text{B.46})$$

For  $n = 1$ , taking the derivative of equation (B.44) with respect to  $\lambda_H$  immediately yields 0. So evaluating equation (B.46) at  $\lambda_H = 0$  and applying Proposition B.5 yields part (iv).

For  $n \geq 3$ , taking the derivative of equation (B.46) with respect to  $\lambda_H$  yields

$$\begin{aligned} \frac{\partial f_{M^{(n)}}(s, \lambda_H)}{\partial \lambda_H} &= \sum_{k=0}^{n-1} s^k \frac{(n-1)!}{(n-1-k)!} \phi_I((k+1)\lambda_H)^{n-1-k} \frac{d^2 G_k(1|\mathbf{V}(\lambda_H))}{d\lambda_H^2} \\ &\quad + \sum_{k=0}^{n-2} s^k \frac{2(n-1)!(k+1)}{(n-2-k)!} \phi_I((k+1)\lambda_H)^{n-2-k} \phi'_I((k+1)\lambda_H) \frac{dG_k(1|\mathbf{V}(\lambda_H))}{d\lambda_H} \\ &\quad + \sum_{k=0}^{n-3} s^k \frac{(n-1)!(k+1)^2}{(n-3-k)!} \phi_I((k+1)\lambda_H)^{n-3-k} (\phi'_I((k+1)\lambda_H))^2 G_k(1|\mathbf{V}(\lambda_H)) \\ &\quad + \sum_{k=0}^{n-2} s^k \frac{(n-1)!(k+1)^2}{(n-2-k)!} \phi_I((k+1)\lambda_H)^{n-2-k} \phi''_I((k+1)\lambda_H) G_k(1|\mathbf{V}(\lambda_H)). \end{aligned} \quad (\text{B.47})$$

For  $n = 2$ , taking the derivative of equation (B.46) with respect to  $\lambda_H$  yields

$$\begin{aligned} \frac{\partial f_{M(2)}(s, \lambda_H)}{\partial \lambda_H} &= \sum_{k=0}^1 s^k \phi_I((k+1)\lambda_H)^{1-k} \frac{d^2 G_k(1|\mathbf{V}(\lambda_H))}{d\lambda_H^2} \\ &\quad + 2\phi'_I(\lambda_H) \frac{dG_0(1|\mathbf{V}(\lambda_H))}{d\lambda_H} + \phi''_I(\lambda_H) G_0(1|\mathbf{V}(\lambda_H)). \end{aligned} \quad (\text{B.48})$$

For  $n = 1$ , taking the derivative of equation (B.46) with respect to  $\lambda_H$  immediately yields 0. So evaluating equations (B.47) and (B.48) at  $\lambda_H = 0$  and applying Proposition B.5 yields part (v).

Finally, for  $n \geq 2$ , taking the derivative of equation (B.46) with respect to  $s$  yields

$$\begin{aligned} &\frac{\partial^2 f_{M(n)}(s, \lambda_H)}{\partial s \partial \lambda_H} \\ &= \sum_{k=0}^{n-2} k s^{k-1} \frac{(n-1)!(k+1)}{(n-2-k)!} \phi_I((k+1)\lambda_H)^{n-2-k} \phi'_I((k+1)\lambda_H) G_k(1|\mathbf{V}(\lambda_H)) \\ &\quad + \sum_{k=0}^{n-1} k s^{k-1} \frac{(n-1)!}{(n-1-k)!} \phi_I((k+1)\lambda_H)^{n-1-k} \frac{dG_k(1|\mathbf{V}(\lambda_H))}{d\lambda_H}. \end{aligned} \quad (\text{B.49})$$

For  $n = 1$ , taking the derivative of equation (B.46) with respect to  $s$  immediately yields 0. So evaluating equation (B.49) at  $\lambda_H = 0$  and applying Proposition B.5 yields part (vi).  $\square$

### B.3 Proof of Theorem 4.4

Recall that for formatting reasons we write  $z(\lambda_H)$ ,  $\lambda_G(\lambda_H)$ ,  $z_H(R_0)$ ,  $f_{M(n)}(s, \lambda_H)$  and  $\mu_{i-1}^{(n)}(\lambda_H)$  for the quantities  $z(\lambda_H; R_0, H, I)$ ,  $\lambda_G(\lambda_H; R_0, H, I)$ ,  $z_H$ ,  $f_{M(n)}(s, \lambda_H; I)$  and  $\mu_{i-1}^{(n)}(\lambda_H; I)$  respectively.

*Proof of Theorem 4.4.* Part (i) follows from noting that  $z_H$  is the unique solution in  $(0, 1]$  of  $1 - z_H = e^{-R_0 z_H}$ . This equation is also satisfied by  $z$  ( see equation (4.75) evaluated at  $\lambda_H = 0$  and applying Lemma B.2(i) ) so  $z(0) = z_H$ .

We now consider parts (ii) and (iii). Taking the derivative of equation (4.75)

with respect to  $\lambda_H$  yields

$$\begin{aligned} -\frac{dz}{d\lambda_H}(\lambda_H) &= -\mu_I e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)} \left[ \lambda_G(\lambda_H) \frac{dz}{d\lambda_H}(\lambda_H) + z(\lambda_H) \lambda'_G(\lambda_H) \right] \\ &\quad \times \sum_{n=1}^{\infty} \tilde{\rho}_n f_{M^{(n)}} \left( e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)}, \lambda_H \right) \\ &\quad + e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)} \sum_{n=1}^{\infty} \tilde{\rho}_n \left[ \frac{df_{M^{(n)}}}{d\lambda_H} \left( e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)}, \lambda_H \right) \right]. \end{aligned} \quad (\text{B.50})$$

Rearranging equation (B.50) and substituting equation (4.75) yields

$$\begin{aligned} \frac{dz}{d\lambda_H}(\lambda_H) &= \mu_I (1 - z(\lambda_H)) \left[ z(\lambda_H) \lambda'_G(\lambda_H) + \lambda_G(\lambda_H) \frac{dz}{d\lambda_H}(\lambda_H) \right] \\ &\quad - \sum_{n=1}^{\infty} \tilde{\rho}_n \left\{ \frac{\partial f_{M^{(n)}}}{\partial \lambda_H} \left( e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)}, \lambda_H \right) \right. \\ &\quad \left. - \mu_I e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)} \left[ \lambda_G(\lambda_H) \frac{dz}{d\lambda_H}(\lambda_H) + z(\lambda_H) \lambda'_G(\lambda_H) \right] \right. \\ &\quad \left. \times \frac{\partial f_{M^{(n)}}}{\partial s} \left( e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)}, \lambda_H \right) \right\} e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)}. \end{aligned} \quad (\text{B.51})$$

Evaluating equation (B.51) at  $\lambda_H = 0$  and applying part (i), Lemmas B.2(ii), B.2(iv), B.1(i) and B.1(ii) yields

$$\begin{aligned} \frac{dz}{d\lambda_H}(0) &= \mu_I (1 - z_H) \left[ -z_H \left( \mathbb{E} [\tilde{H}] - 1 \right) + \frac{R_0}{\mu_I} \frac{dz}{d\lambda_H}(0) \right] \\ &\quad - e^{-R_0 z_H} \sum_{n=1}^{\infty} \tilde{\rho}_n \left\{ -\mu_I (n-1) \left( 1 - e^{-R_0 z_H} \right) \right\}. \end{aligned} \quad (\text{B.52})$$

Recall that  $1 - z_H = e^{-R_0 z_H}$ , so, rearranging equation (B.52),

$$\begin{aligned} \frac{dz}{d\lambda_H}(0) [1 - R_0 (1 - z_H)] &= -\mu_I z_H (1 - z_H) \left( \mathbb{E} [\tilde{H}] - 1 \right) \\ &\quad + \mu_I z_H (1 - z_H) \left( \mathbb{E} [\tilde{H}] - 1 \right) \\ &= 0, \end{aligned}$$

from which part (ii) follows by Lemma 4.1, i.e.  $[1 - R_0(1 - z_H)] > 0$ .

Taking the derivative of equation (B.51) with respect to  $\lambda_H$  yields

$$\begin{aligned}
\frac{d^2 z}{d\lambda_H^2}(\lambda_H) = & \mu_I (1 - z(\lambda_H)) \left[ z(\lambda_H) \lambda_G''(\lambda_H) + 2\lambda_G'(\lambda_H) \frac{dz}{d\lambda_H}(\lambda_H) \right. \\
& \left. + \lambda_G(\lambda_H) \frac{d^2 z}{d\lambda_H^2}(\lambda_H) \right] \\
& - \mu_I \frac{dz}{d\lambda_H}(\lambda_H) \left[ z(\lambda_H) \lambda_G'(\lambda_H) + \lambda_G(\lambda_H) \frac{dz}{d\lambda_H}(\lambda_H) \right] \\
& + e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)} \sum_{n=1}^{\infty} \tilde{\rho}_n \left\{ -\frac{\partial^2 f_{M^{(n)}}}{\partial \lambda_H^2} \left( e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)}, \lambda_H \right) \right. \\
& + \mu_I \left[ \lambda_G(\lambda_H) \frac{dz}{d\lambda_H}(\lambda_H) \right. \\
& \quad \left. + z(\lambda_H) \lambda_G'(\lambda_H) \right] \frac{\partial f_{M^{(n)}}}{\partial \lambda_H} \left( e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)}, \lambda_H \right) \\
& - 2\mu_I^2 e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)} \left[ \lambda_G(\lambda_H) \frac{dz}{d\lambda_H}(\lambda_H) \right. \\
& \quad \left. + z(\lambda_H) \lambda_G'(\lambda_H) \right]^2 \frac{\partial f_{M^{(n)}}}{\partial s} \left( e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)}, \lambda_H \right) \\
& + \mu_I e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)} \left[ z(\lambda_H) \lambda_G''(\lambda_H) + 2\lambda_G'(\lambda_H) \frac{dz}{d\lambda_H}(\lambda_H) \right. \\
& \quad \left. + \lambda_G(\lambda_H) \frac{d^2 z}{d\lambda_H^2}(\lambda_H) \right] \frac{\partial f_{M^{(n)}}}{\partial s} \left( e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)}, \lambda_H \right) \\
& + 2\mu_I e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)} \left[ \lambda_G(\lambda_H) \frac{dz}{d\lambda_H}(\lambda_H) \right. \\
& \quad \left. + z(\lambda_H) \lambda_G'(\lambda_H) \right] \frac{\partial^2 f_{M^{(n)}}}{\partial s \partial \lambda_H} \left( e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)}, \lambda_H \right) \\
& + \mu_I e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)} \left[ \lambda_G(\lambda_H) \frac{dz}{d\lambda_H}(\lambda_H) \right. \\
& \quad \left. + z(\lambda_H) \lambda_G'(\lambda_H) \right] \frac{\partial^2 f_{M^{(n)}}}{\partial s^2} \left( e^{-\mu_I \lambda_G(\lambda_H) z(\lambda_H)}, \lambda_H \right) \Big\}.
\end{aligned} \tag{B.53}$$

Next we evaluate equation (B.53) at  $\lambda_H = 0$ . We begin by recalling that  $1 - z_H = e^{-R_0 z_H}$ , applying parts (i) and (ii), and Lemmas B.2(ii) and B.2(iii). We then apply the remaining parts of Lemma B.2 and Lemma B.1 and rearrange,

yielding part (iii).

$$\begin{aligned}
& \frac{d^2 z}{d\lambda_H^2}(0) [1 - R_0 (1 - z_H)] \\
&= \mu_I z_H (1 - z_H) \lambda_G''(0) \\
&\quad + (1 - z_H) \sum_{n=1}^{\infty} \tilde{\rho}_n \left\{ \mu_I z_H \lambda_G'(0) \frac{\partial f_{M^{(n)}}}{\partial \lambda_H} (1 - z_H, 0) \right. \\
&\quad \quad - \frac{\partial^2 f_{M^{(n)}}}{\partial \lambda_H^2} (1 - z_H, 0) \\
&\quad \quad \left. + 2\mu_I z_H (1 - z_H) \lambda_G'(0) \frac{\partial^2 f_{M^{(n)}}}{\partial s \partial \lambda_H} (1 - z_H, 0) \right\} \\
&= \mu_I z_H (1 - z_H) \left( \frac{2\mu_I}{R_0} \left[ E[\tilde{H}]^2 - E[\tilde{H}^2] + E[\tilde{H}] - 1 \right] \right. \\
&\quad \quad \left. + \frac{\phi_I''(0)}{\mu_I} E[\tilde{H} - 1] \right) \\
&\quad + (1 - z_H) \sum_{n=1}^{\infty} \tilde{\rho}_n \left\{ \mu_I^2 z_H^2 E[\tilde{H} - 1] (n - 1) \right. \\
&\quad \quad - (n - 1) \left[ \mu_I^2 (n - 2) (1 - 4(1 - z_H) + 3(1 - z_H)^2) \right. \\
&\quad \quad \quad \left. + z_H \phi_I''(0) \right] \\
&\quad \quad \left. - 2\mu_I^2 z_H (1 - z_H) E[\tilde{H} - 1] (n - 1) \right\} \\
&= \mu_I^2 z_H (1 - z_H) \left\{ \frac{2}{R_0} \left[ E[\tilde{H}]^2 - E[\tilde{H}^2] + E[\tilde{H}] - 1 \right] \right. \\
&\quad \quad + z_H E[\tilde{H} - 1]^2 - (3z_H - 2) [E[\tilde{H}^2] - 3E[\tilde{H}] + 2] \\
&\quad \quad \left. - 2(1 - z_H) E[\tilde{H} - 1]^2 \right\} \\
&= \mu_I^2 z_H (1 - z_H) \left( \frac{2}{R_0} + 3z_H - 2 \right) (E[\tilde{H}]^2 - E[\tilde{H}^2] + E[\tilde{H}] - 1).
\end{aligned}$$

Finally we consider part (iv). Recall that  $\rho_n = n\rho_n/E[H]$ , so

$$E[\tilde{H}] = E[H^2]/E[H] \quad (\text{B.54a})$$

$$E[\tilde{H}^2] = E[H^3]/E[H] \quad (\text{B.54b})$$

$$E[H] > 0 \quad (\text{B.54c})$$

Applying the sgn function, Lemma 4.1 and equations (4.26) and (B.54) to

part (iii) yields

$$\begin{aligned}
\operatorname{sgn}\left(\frac{d^2 z}{d\lambda_H^2}(0)\right) &= \operatorname{sgn}\left(E[\tilde{H}]^2 - E[\tilde{H}^2] + E[\tilde{H}] - 1\right) \\
&= \operatorname{sgn}\left(\left(\frac{E[H^2]}{E[H]}\right)^2 - \frac{E[H^3]}{E[H]} + \frac{E[H^2]}{E[H]} - 1\right) \\
&= \operatorname{sgn}\left(E[H^2] \left(1 + \frac{E[H^2]}{E[H]}\right) - E[H^3] - E[H]\right),
\end{aligned}$$

as required for the proof of part (iv). □