University of Nottingham

School of Mathematical Sciences

Disease-induced herd immunity and household epidemic models

Liam Critcher

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Abstract

The rate at which individuals in a population mix with one another can have a large impact on how far a disease spreads among that population, as well as what fraction of the population needs to be immune from infection in order to protect the remaining susceptible population from a major outbreak. In this thesis we consider both deterministic and stochastic SEIR (susceptible $\rightarrow$ exposed$\rightarrow$ infectious $\rightarrow$ recovered) epidemic models. We impose a household structure on the population, so that individuals mix globally with the population at large and, at a higher rate, locally with members of their household. We also consider an extension of this model in which individuals are typed, making global contacts at different rates dependent on their type. We investigate herd immunity for these models, providing a more realistic insight than the standard epidemic model in which all individuals in the population mix at the same rate.

The disease-induced herd immunity level h_D is the fraction of the population that must be infected by an epidemic to ensure that a new epidemic among the remaining susceptible population is not supercritical. For a homogeneously mixing population h_D equals the classical herd immunity level h_C, which is the fraction of the population that must be vaccinated in advance of an epidemic so that the epidemic is not supercritical. A detailed comparison of h_D and h_C is given for the households model, where we also define an approximation $\tilde{h}_D$ of h_D which is more amenable to analysis. It is found that $h_D > h_C$ unless the household size variability is sufficiently large, in contrast to other models with heterogeneous mixing of individuals, in which $h_D < h_C$ typically occurs.

We obtain the asymptotic variance for h_D as the population size goes to infinity, using a Gaussian approximation. We then consider a model with individual types and household structure, deriving several reproduction numbers and a central limit theorem for the final outcome under the assumption of proportionate global mixing, which we show greatly simplifies these calculations and results. We provide comparison of $\tilde{h}_D$ and h_C when these individual types correspond to activity levels, showing that the ordering of these herd immunity levels is strongly dependent on the distri-

bution of the individuals of each activity level among the households.

Finally, we consider the impact of global restrictions on disease-induced herd immunity in a model with household structure and types of individuals. We extend the approximation $\tilde{h}_D$ to account for local infection being increased during times of global restrictions. We then consider a scenario in which two supercritical epidemics can occur, the first with constant control measures, and find an optimal control such that the number of individuals ever infected across the two epidemics is minimised.

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Contents

1 Introduction

In this chapter we provide contextualisation of this thesis by considering historical advances in mathematical models of epidemics. We describe several epidemic models, including those which we study in this thesis, omitting mathematical details and discussing their connection to our work. We begin in Section 1.1 by discussing some of the most famous epidemics in history and the impact they had on the suffering population, providing motivation to better understand disease spread. In Section 1.2 we discuss early mathematical models of epidemics, noting the distinction between deterministic and stochastic epidemic models. We also describe the introduction of threshold parameters for epidemic models, which form a central interest of this thesis. In Section 1.3 we consider the development of models for which not all individuals behave in the same way; examples include multitype models, household models and network models. A non-technical definition of herd immunity is provided in Section 1.4, where we also discuss literature pertaining to mathematical models of vaccine action. In Section 1.5 we discuss the estimation of parameters of epidemic models from a statistical perspective, both for emerging epidemics and for epidemics where final outcome data are available. We conclude the background discussion of this thesis in Section 1.6 by considering recent literature regarding herd immunity, in particular literature resulting from the COVID-19 pandemic. Finally, we give an outline of the structure of the remainder of this thesis in Section 1.7.

1.1 Epidemics throughout history

Mankind has been afflicted by epidemics in various forms for thousands of years, ranging from the plague of Athens and the Antonine plague of the past to COVID-19, which we continue to combat in the present day. It is clear that advances in understanding of transmission and treatment of diseases have allowed for better mitigation of large epidemic outbreaks. Despite these advances, the ongoing COVID-19 pandemic is estimated to have taken tens of millions of lives – see Mathieu et al. [2020]. Being able to understand and predict the transmission of disease thus remains of great importance and value.

As scientific understanding of disease treatment and transmission has evolved, our ability to mitigate the impact of infectious disease outbreaks has vastly improved. For example, treatment methods such as bloodletting, which involved removing blood in order to allegedly alleviate illness, are now merely considered pseudoscience, having been replaced by far more sophisticated medical practices. It is natural to question how many lives would have been saved in epidemics throughout history with better understanding of the nature of their spread and the effectiveness of treatments. In conjunction with medical and scientific advances through the years, mathematicians have developed models which attempt to describe the spread of infection. Early examples of work using mathematical models include John Snow's investigations into Cholera in London in 1854, where Snow showed that contaminated water was triggering epidemics. Similarly, William Budd was, in 1873, able to successfully establish the source of typhoid fever. In these cases, the epidemic could be dealt with at source, rendering further mathematical study obsolete. The emergence of germ theory laid foundations from which mathematical models could be developed, as well as aiding identification of organisms that cause disease – see, for example, Susser and Stein [2009] for an overview. In cases where an epidemic cannot be so easily isolated, mathematical models can be used to study how quickly the epidemic will spread and what fraction of the population we may reasonably expect it to infect.

Major epidemic outbreaks subject the afflicted population to several major issues. The primary and most obvious of these are deaths; the Black Death of the fourteenth century is estimated to have killed anywhere from $30-60$ percent of Europe's population – see Alchon [2003], Table 1.1. Another stark example is the so-called Spanish flu, which began around the end of World War 1, estimated to have resulted in more deaths than even the Great War itself – see World Health Organisation interim guidance [2013], Table 3. These examples highlight the catastrophic impact epidemics can have on an unprepared population.

The speed of spread of an outbreak also has important consequences for its effect on the population; during the height of COVID-19, infection rates in the UK were very high, leading to drastically higher hospitalisation rates than

normal and huge pressures on hospitals and healthcare services. This suggests that actions to slow the spread of infection, even if they do not lower the number ultimately infected, can still be beneficial to the population. Moreover, if measures such as global restrictions and social distancing have to be taken in order to combat a disease, they incur their own costs socially and economically. It is therefore of interest to determine how long such restrictions should be in place whilst still providing adequate protection to the population. Through the height of the COVID-19 pandemic, mathematical models received great attention as attempts were made to predict the growing number of hospitalisations and deaths. Moreover, strong consideration was given to estimating the point at which health services might become overwhelmed, as well as the consequences of various intervention strategies. This clearly highlights the important role mathematical models have to play in epidemiological study.

Expecting to be able to accurately model every interaction in a population is extremely ambitious and proves, mathematically, to be very unwieldy. Moreover, complex models which attempt to model such interactions often suffer from, inter alia, issues regarding identifiability and overfitting. In this thesis we assume that the population of interest is closed, in the sense that no individuals join or leave the population; whilst not fully accurate, this assumption is reasonable for the present purposes owing to the fact that epidemics typically spread at a much faster rate than individuals join or leave the population. Such an assumption is not, however, reasonable for endemic diseases. The models we use are of the form susceptible $\rightarrow$ exposed $\rightarrow$ infectious $\rightarrow$ removed (SEIR), in which an individual begins susceptible to the disease and, upon contact with an infective, has a period in which they are infected but are unable to infect others (latent period). When their latent period ends, this individual is able to infect other susceptible individuals until the end of their infectious period. When they recover, i.e. at the end of their infectious period, such an individual is assumed to play no further role in the epidemic. Already this model represents a departure from many real-world epidemics, in which subsequent reinfection can occur after recovery. A central model of this thesis partitions the population into households. Individuals are assumed to mix globally with all individuals in the

population at the same rate and, typically at a higher rate, mix with individuals in their household. We develop this further into a model in which some individuals mix at a higher rate outside of their household than others. Despite the models we use containing departures from reality, they represent a compromise in which we attempt to model key features of an epidemic among a structured population whilst maintaining mathematical tractability.

During the first wave of COVID-19 in the UK, there was considerable discussion regarding delaying "the peak" of infection, referring to the point where the number of infectives, and consequently the number of hospitalisations, would be at its highest, implying that there would only be a single wave of infection. Such discussion has strong links to herd immunity. In simple terms, an epidemic among a population which has achieved herd immunity will not suffer a further major epidemic outbreak, but a population which has not reached herd immunity is susceptible to subsequent waves of infection. In this thesis we devote considerable attention to describing a mathematical framework in which we can study herd immunity.

In the remainder of this section we outline the development of mathematical models for epidemics with a particular focus on models with heterogeneous mixing. We also describe herd immunity in more detail and comment on medical and mathematical studies regarding how a population can gain immunity from infection.

1.2 Early mathematical models of epidemics

The first notable contribution to mathematical models of epidemics is due to the work of Daniel Bernoulli in 1760, in order to justify inoculation as a means to combat smallpox. In the late nineteenth century, P.D. En'ko collected data on the spread of measles and sought a mathematical model by which the process of infection could be understood. The work is written in Russian, but owing to Dietz an abridged translation is available - see Dietz [1988]. In En'ko's work the chain-binomial model is used, foreshadowing the work of Reed and Frost some forty years later.

The epidemic models which we study can be separated into two categories

– deterministic and stochastic. (Hybrid models, which combine deterministic and stochastic elements together in a single model, have been studied – see Hwang et al. [2022], but we do not consider such models in this thesis.) Stochastic models describe the probabilities with which events, such as the infection or recovery of an individual, occur. Deterministic models, however, describe the rates of transition between different states, often operating under the assumption of mass action, in which the rate of infection is proportional to the current numbers of susceptibles and infectives. Deterministic models are often described by a system of ordinary differential equations. In this thesis, we often obtain a deterministic model by taking the limit of a suitably scaled stochastic model as the population size becomes arbitrarily large.

Deterministic and stochastic epidemic models both received great attention in the early twentieth century, from both a probabilistic and statistical perspective. Hamer [1906] supposed, in a discrete-time setting, that the probability an individual becomes infected at the next time step is proportional to the number of susceptibles and infectives at the current time. Ross [1908] considered the same idea in a continuous time setting. An example of such a statistical study is the work of Brownlee [1906], in which the Pearson family of distributions was applied to epidemics. Perhaps the most revolutionary work to arise regarding mathematical models of epidemics in the early 1900's is the work on compartmental epidemic models. Ross, in 1916, considered such a compartmental model with two groups labelled "affected" and "unaffected", and used differential equations in order to study the number of affected individuals – see Ross [1916]. This work was later developed further in Ross and Hudson [1917] with the help of Hudson.

Arguably the most famous development of compartmental models is thanks to the much-celebrated work of Kermack and McKendrick, who used the susceptible, infectious, removed (SIR) framework which is still used to this day. McKendrick [1926] proposed one of the earliest stochastic epidemic models. Soon after, Kermack and McKendrick [1927] analysed a deterministic SIR model and noted that, under their model, the epidemic would not necessarily end by exhausting the entire susceptible population. As part of this work, they also

noted a "critical population density", below which no epidemic can occur. The deterministic models used in this thesis can be viewed as an extension of their model. Progress on stochastic epidemic models continued in the late 1920's with the work of Reed and Frost, who proposed a model involving generations of infectives, viz. the Reed-Frost model. This work was presented soon after its completion, but was only published much later thanks to Abbey [1952]. A similar model – the Greenwood model – was proposed independently in 1931 by Major Greenwood. Over a decade later, the work of Bartlett [1949] studied a continuous-time stochastic SIR epidemic model. In Bailey [1953], a stochastic SIR epidemic with infection and recovery rates was defined in continuous time, commonly referred to as the general stochastic epidemic, with final size of the epidemic estimated by statistical methods. The book by Bailey [1975], the first edition of which was published in 1957, concerns both deterministic and stochastic epidemic models, discussing parameter estimation for quantities such as transmission rates.

A crucial cornerstone in any epidemic analysis involves whether or not a major outbreak, in which a non-negligible fraction of the population ever become infected, can occur. Threshold theorems seek to quantify when such a major outbreak can occur; the quantities which the occurrence of a major outbreak depend on are often referred to as threshold parameters. Perhaps the most well known of these in the present day is the basic reproduction number R_0, often referring to the mean number of infections which will ensue from a typical infected individual in an otherwise susceptible population. The reproduction number R_0 has a rich history in epidemic study, although under several different names. The note of Kermack and McKendrick [1927] regarding critical population density is an early such example of the use of R_0 to establish a threshold theorem for the deterministic SIR model. A similar concept was first introduced in Dublin and Lotka [1925] regarding the ratio of births and deaths in a population. The central idea behind R_0 is that a major epidemic outbreak can occur if and only if $R_0 > 1$; in such a scenario infection can be sustained in the population. The work of Macdonald [1955] on Malaria noted such threshold behaviour when referring to a "critical level", denoted z_0 in his work. Many credit

Dietz [1975] as the first instance in which a threshold parameter, with threshold value one, is clearly defined for an epidemic model. The first threshold theorem for stochastic SIR epidemics is thanks to Whittle, who used a removal ratio introduced in Bailey [1953] to describe whether the final size of the epidemic could exceed some predetermined fraction with positive probability – see Whittle [1955].

The works of Bartlett [1955] and Kendall [1956] approximated the early stages of the general stochastic epidemic in a large population using a linear birth and death process. Such an approximation, sometimes referred to as Kendall's approximation, provides a threshold theorem in which the epidemic taking off corresponds to the approximating birth and death process not dying out. This has strong parallels with the fact that an epidemic can only take off if $R_0 > 1$. Bartoszyński [1967] noted that, in the early stages of an epidemic, infections can be equated to births of a branching process and studied the epidemic process via the associated branching process. In this thesis we derive and use threshold parameters, including R_0, making use of such a branching process approximation in order to do so.

The models highlighted in this section demonstrate the rapid progress of mathematical epidemiology in the first half of the twentieth century. These models typically assume that all individuals have the same infectivity and susceptibility as each other, an assumption often referred to as homogeneous mixing. In Section 1.3 we note a flurry of epidemic models with heterogeneous mixing which were developed in the latter half of the twentieth century and the early twenty-first century; such models assume a more complex mixing structure on the population of study.

1.3 Development of models with heterogeneous mixing

The homogeneously mixing epidemic model assumes that each susceptible is equally likely to be contacted by a given infectious individual. Epidemic models which assume no structure or heterogeneity on the population are relatively easy to analyse, but often represent a large oversimplification of real-world populations, which typically are not homogeneously mixing. There are two types

of heterogeneity which this thesis is primarily concerned with - that of household structure and multiple types of individuals. Both of these involve dividing the population into groups; for multitype models, individuals in different groups can contact, and are contacted by, other individuals at typically different rates. A model with household structure, however, typically assumes homogeneous mixing between all individuals but with extra (local) mixing occurring between individuals belonging to the same household. We highlight the development of both such models in the sequel.

Perhaps the earliest epidemic model which does not assume homogeneous mixing between all individuals is thanks to Rushton and Mautner [1955], who considered a deterministic epidemic model among m communities, in the absence of removals and deaths. Communities in that model mix homogeneously, with further homogeneous mixing occurring within a given community. Whilst the assumption of no recoveries is unrealistic in practice, their work formed a basis from which epidemic models with heterogeneous mixing could be studied. Shortly afterwards, Haskey [1957] considered a stochastic epidemic model with two classes. Watson [1972] extended the work of Rushton and Mautner, studying a stochastic SIR version of their model. Billard [1976] provided an alternative formulation of Watson's model and introduced a transformation technique in an attempt to give the model more analytical tractability. Such a technique involved an ordering of the possible states such that transitions happen in only one direction, leading to a matrix-vector differential equation in which the matrix present is lower triangular and thus the associated equation is more amenable to analysis.

The models described previously work well for modelling a small number of large groups or communities. In practice, however, a population may consist of a large number of small groups. This is particularly true in the case of a population partitioned into households - an often very important and realistic assumption. The work of Bartoszyński [1972] was the first to consider a large number of groups, in his case households, which need not be large themselves. For a while a version of the threshold theorem for household models was not forthcoming. Ball [1986] developed a stochastic epidemic model, in which the

duration of the infectious period could have an arbitrary but specified distribution, and derived results pertaining to the final size distribution of that model. This work included the proof of a version of Wald's identity for epidemics, which we apply several times in this thesis and provide in Appendix B. Addy et al. [1991] extended the work of Ball to account for outside infection and also derived equations for final size probabilities.

A particularly sought after result was the threshold theorem for the households model. Whilst the basic reproduction number R_0 works well for describing whether an epidemic can take off in a homogeneously mixing population, it relies on the assumption that infective individuals are not contacted by other infective individuals in the early stages of an epidemic; such an assumption does not hold for models with small mixing groups such as households, where repeated local contacts between individuals are likely to occur. This issue is noted by Ball in his discussion in Mollison et al. [1994] and an alternative threshold parameter, called there R_T, was introduced. Becker and Dietz [1995] considered such a threshold parameter, which they referred to as R_{H0}, for the stochastic SIR households model under the assumption of highly locally infectious disease. The quantity R_{H0}, labelled R_* in this thesis, parallels R_0 in the sense that $R_* > 1$ is necessary for a major outbreak (i.e. an outbreak in which a non-negligible fraction of the total number of households ever become infected) to occur. The crucial idea regarding R_* is to consider the proliferation of infected households rather than of infected individuals. In a situation parallel to repeat infection in the homogeneously mixing epidemic, it is unlikely that, in a population with a large number of households, an infective individual will contact a previously contacted household in the early stages of an epidemic. Independent work by Ball [1996] proved a threshold theorem for the stochastic SIR households model in which all households are the same size. Ball et al. [1997] also analysed the SIR households model in detail, providing a version of the threshold theorem.

The ideas of having an epidemic such as the households model, with two levels of mixing, as well as multiple types of individuals, need not be mutually exclusive. Ball and Lyne [2001] provided a fully rigorous proof of the threshold theorem for the multitype SIR households model, by coupling the epidemic to

an appropriate branching process, and Ball et al. [2004] estimated secure vaccination coverage for that model. The threshold parameter R_* for the multitype SIR households model can be derived using similar arguments to those used in the derivation of R_* for the households model. We consider a special case of this model, in the context of herd immunity, in this thesis. Epidemic models have now broadened to include many other assumptions based on real-world factors. Examples include the works of Ball and Neal [2002] and Pellis et al. [2011], which pertained to a model with households and workplaces, and Neal [2016] in which individuals were allowed to move between contact groups based on the time of day. Several reproduction numbers can be defined for these models, each having the threshold property, and a broad overview of these can be found in Pellis et al. [2012] and Ball et al. [2016].

Network epidemic models have received considerable attention in recent years. Such models use an undirected graph, with nodes corresponding to individuals, with disease being able to be spread along the edges of that graph. We note that Newman [2002], Danon et al. [2011] and Pellis et al. [2015] provide good overviews of epidemic models on networks. It is clear that the choice of the degree of each individual will affect how an epidemic plays out on that network, as some individuals may have far more contacts than others. The configuration model, introduced by Bollobás [1980], is widely used and assumes either that the degrees are determined deterministically (see Molloy and Reed [1998]) or that the degrees are each the realisation of a random variable, independent and identically distributed for each individual. Ball and Neal [2008] considered a stochastic epidemic on a network model incorporating casual contacts, defining R_0 for that model and deriving a deterministic model as the population size tends to infinity. Ball et al. [2019] considered a network model incorporating preventive dropping of edges, mimicking the behaviour of social distancing upon becoming infected. Further work on epidemics on networks include Trapman [2007], House [2012] and Kiss et al. [2017].

Network models benefit from having a great deal of flexibility in the way in which they can be constructed. A drawback, however, lies in the complexity of constructing network models in terms of choosing the degree of each indi-

vidual, as it is not necessarily always clear how to do this in a realistic manner. Moreover, it is often difficult to replicate some aspects of real-world networks, such as clustering of individuals and correlation of degrees between individuals. The models used in this thesis can be seen as "fully complete" from a network perspective, since all individuals are able to contact one another, albeit at a much lower rate than at which they can contact members of their own household.

We conclude the review of models with heterogeneous mixing by providing a hierarchy for the models which are considered in this thesis. All models in this thesis can be viewed as an extension of the homogeneously mixing SIR epidemic and, in most cases, as a simplification of the multitype households epidemic model. Throughout this thesis, we strongly encourage the reader to note and have in mind Figure 1.1 during model definition and model discussion.

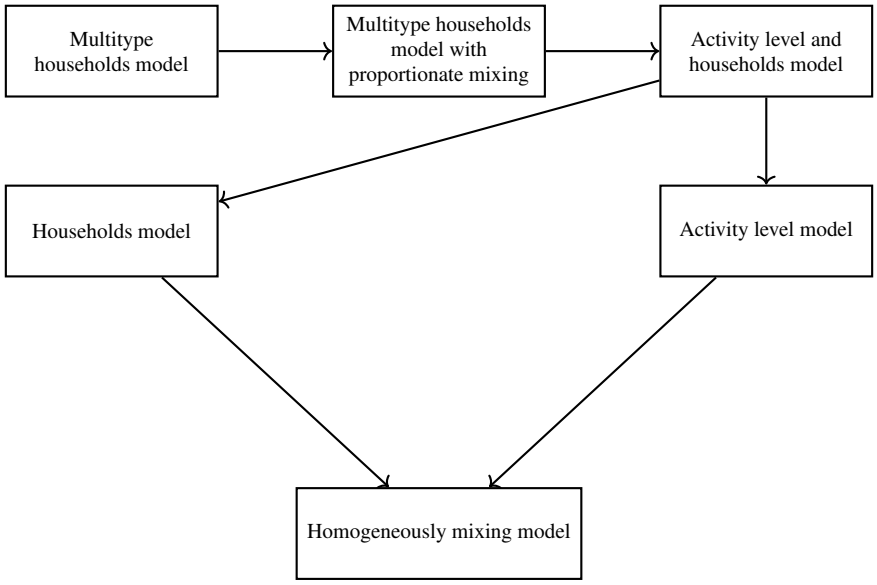

Figure 1.1: Hierarchy of models used in this thesis. An arrow from one model to another indicates that the latter model can be obtained (from the former model) by making certain parameter choices. For example, the households model becomes homogeneously mixing if the common household size $n = 1$.

1.4 Disease control and herd immunity

A natural question that arises when confronted by a disease outbreak in which $R_0 > 1$ is how it can be mitigated or contained – see, for example, World Health Organisation [2023]. If initial attempts such as quarantine of early cases are unsuccessful, attention typically turns to attempts to restrict the transmission rate

of the disease, thus reducing R_0. These measures range from basic attempts such as maintaining good hygiene and wearing masks, to more drastic measures such as social distancing and lockdown. Throughout this thesis, when restrictions are applied, we assume that they affect the ability of individuals to mix globally, but not the ability for an individual to mix with members of their own household. When we refer to preventative measures which cause a reduction in global mixing, we have in mind measures such as masks, social distancing or lockdown as opposed to vaccination.

In cases where reduction of disease transmission is not enough (or indeed not feasible) to prevent an outbreak, the susceptible population must seek protection from the disease elsewhere. Owing to the threshold property of R_0 we see that, if the population susceptibility is reduced such that $R_0 < 1$, a major outbreak can no longer occur, thus protecting the population. This is true for both the deterministic model, as well as the stochastic model when the population size $N \to \infty$. The herd immunity level is then the proportion of the population that must be immunised in order to prevent a major outbreak. We discuss the two such forms of immunisation that are central to this thesis, beginning with disease-induced herd immunity.

In the early 1900's, American farmers and veterinarians struggled to deal with epidemics of spontaneous miscarriage in their livestock. Their approach was to destroy or sell the affected livestock and have them replaced, which at first seems entirely reasonable. However, Eichorn [1917] noted that "Affected cows do not abort indefinitely. Much more than 50 per cent abort but once, relatively few abort twice... It is evident, therefore, that an immunity is produced". Contrary to initial beliefs, it was better to avoid taking in new livestock (who themselves may also be susceptible to the disease) and instead to persist with the current livestock, as suffering from the disease conferred immunity. It was here that the concept of herd immunity, as it is referred to today, was born. We note the distinction between herd immunity and acquired immunity; herd immunity protects an individual because the population susceptibility is, after some individuals have been infected and subsequently recovered, such that a major outbreak can no longer occur. On the other hand, acquired immunity refers to

an individual gaining immunity from infection by having been previously infected.

Disease-induced immunity later became the subject of medical experiments. Topley and Wilson [1923] performed experiments on rats in which herd immunity was conferred, finding that "It appears that a degree of immunity, which may save individual hosts when living among equally resistant companions, is rendered of no avail when they are surrounded by highly susceptible individuals of their own species". This work speculated about the difference between a population with many weakly immunised individuals and a population with a few highly resistant individuals. The concept of disease-induced immunity was then extended to human populations owing to the experiments of Sheldon Dudley in the 1920's, considering infection from diptheria bacilli in a population of boys in remote residence. In Dudley [1922] it was found that boys who had been in residence for longer were far less susceptible than the new boys. In addition to this, Dudley [1928] noted that "The production of human circulating antitoxin by the injection of manufactured diphtherial antigens causes a far more rapid increase in herd-immunity than can be produced by any immunizing stimuli which may exist in the natural human environment". Note that, in the examples above, herd immunity and acquired immunity are used interchangeably; in this thesis herd immunity always refers to protection of an individual owing to a reduction in overall population susceptibility. As a consequence, disease-induced herd immunity refers to a scenario in which enough of the population have recovered from infection such that the remaining susceptible population has $R_0 < 1$. Thus, disease-induced herd immunity is obtained by *the entire susceptible population*, rather than particular individuals benefiting from the immunity of their neighbours.

Vaccination against a disease is another widely used method in order to defend a population from infection. Whilst vaccination and, prior to that, inoculation, have a rich history in epidemiology, we focus here on mathematical models of vaccination, making a connection with disease-induced herd immunity when relevant. Suppose that $R_0 > 1$ and that, upon vaccination, a fraction c of the population is made immune to infection in a population which is

homogeneously mixing. Then, since a typical individual is unvaccinated with probability $1-c$, the value of R_0 is reduced to $R_V = (1-c)R_0$, implying that $c > 1 - R_0^{-1}$ will ensure $R_V < 1$. This formula for c, a version of which was introduced by Smith [1964], is sometimes called the critical vaccination coverage and is referred to in this thesis as the vaccine-induced herd immunity level. (The above critical value typically differs from $1 - R_0^{-1}$ for models with heterogeneous mixing.)

Smith's work is one of the earliest examples of mathematical models of vaccination (excluding that of Bernoulli's work on inoculation). Another of the earliest such examples is thanks to Neyman and Scott [1964], who used the theory of branching processes to investigate the reduction in the mean final size of a stochastic epidemic in discrete time. The use of a branching process to this end was suggested by Bharucha-Reid [1956] and later developed by Becker [1972] for a stochastic model, with the intention of providing a vaccination scheme that was sufficient, but vaccinated as few individuals as possible. At the time of Becker's work vaccinations were considered potentially harmful to the population, which motivated this minimisation aim; we note that minimising the number vaccinated in order to reduce R_0 to one remains of interest at present, particularly given vaccines are often difficult and expensive to produce. Hethcote and Waltman [1973] modified a deterministic model by allowing vaccination, with the aim of "prevention", referring in their context to keeping the total number ultimately infected, as well as the number currently infective at any one time, below fixed values. Anderson and May [1982] considered similar ideas to that of Hethcote and Waltman for a deterministic SEIR model. In doing so, they note the importance of the quantity $1 - R_0^{-1}$ in terms of protecting a population. The aforementioned studies all highlight the importance of taking measures to reduce R_0 to one in order to protect a population.

Our discussion has, thus far, only covered vaccination of a homogeneously mixing population, although the discussion regarding optimisation of vaccines holds for heterogeneous populations. Hethcote later extended his work to a deterministic epidemic model with large mixing groups - see Hethcote [1978]. Longini et al. [1978] considered a heterogeneous model for Influenza A with in-

dividuals stratified by age, forming optimal vaccine allocations for that model. The work of Anderson and May [1984] considered spatial heterogeneity, highlighting that random vaccination in a population which is heterogeneously mixing can fail to prevent an epidemic, even when vaccinating a proportion that would be sufficient for a homogeneously mixing population, since some individuals contribute more to infection than others. In conjunction with this, optimal allocation of the vaccine in such a population can protect that population using a lower coverage (proportion of the population which are vaccinated) than the homogeneously mixing model would suggest. This work has links to disease-induced herd immunity, which can be seen to act as a "targeted vaccine", in which the most active individuals are infected first, thus more quickly reducing the population susceptibility. One such heterogeneity, which we consider and later define in detail in this thesis, is that of proportionate mixing – see, for example, Anderson et al. [1986] who considered such an assumption for a model of HIV.

Households models have also received considerable attention in terms of modelling vaccination. Becker and Dietz [1995] considered a post-vaccination threshold parameter, R_v, in a model with households with the aim to reduce R_v to one; their model assumes a highly locally infectious disease, in which a household becomes fully infected as soon as one individual in that household becomes infected. Hall and Becker [1996] compared immunisation strategies for a model with different types of individuals as well as household structure. They compared immunisation of individuals at random with immunisation of whole households. Other allocations of a vaccine are available - one important such example is that of the equalising strategy. In this strategy, a household containing the largest number of susceptible individuals is the next to receive a vaccination. Thus, the equalising strategy attempts to make all households the same size, in terms of the remaining susceptible population in each household. This strategy, introduced by Ball et al. [1997], is conjectured to be optimal for the households model, with proof of optimality given in certain special cases along with numerical support of the conjecture otherwise. Becker and Starczak [1997] similarly considered the allocation of vaccines for the households

model, formulating this as a linear programming problem. The later work of Ball and Lyne [2002] and Ball and Lyne [2006] also formulated optimal vaccination in the households model as a linear programming problem.

We have outlined that there are multiple ways in which a population can gather immunity. In this thesis we compare vaccine-induced and disease-induced herd immunity and, in doing so, assume that these two immunities are not being gathered simultaneously in the population. The disease-induced herd immunity level h_D is the proportion of the population that must be infected by an epidemic such that a new epidemic among the remaining susceptible population has threshold parameter less than or equal to one. An analogous definition holds for the vaccine-induced herd immunity level h_C under the assumption of random vaccination, by a perfect vaccine, prior to an epidemic commencing. These herd immunity levels coincide for a population which is homogeneously mixing; we seek comparison of h_D and h_C for populations which are not homogeneously mixing, viz. models with household structure and multiple types of individuals.

Owing to the use of SIR and SEIR models, we assume that individuals are not susceptible to subsequent reinfection upon recovery. Moreover, we assume that vaccinated individuals are granted complete immunity to infection, often referred to as assuming a perfect vaccine. Becker and Starczak [1998] considered how vaccination strategies change under the assumption of random vaccine response, which confers partial immunity. This work is extended in Becker [2002], where the results were applied to data on mumps. Ball and Lyne [2002] provided detailed discussion of optimal vaccination in the case of an imperfect vaccine. We defer our discussion of the assumption of perfect vaccine and lack of subsequent reinfection to the concluding comments in Chapter 7.

1.5 Parameter estimation

We now discuss some of the vast body of recent literature around epidemic models, in particular regarding parameter estimation. We begin with a brief review of analysis of data for emerging epidemics, before discussing some of the work pertaining to analysis of final size data.

A first question when confronted by a novel disease, such as upon the discovery of COVID-19, is the rate at which the disease transmits, as well as how long infected individuals remain latent before being able to infect others. We note that quantities such as R_0 depend on the epidemic model being used and are usually estimated from epidemic data. We begin by discussing parameter estimation for an emerging epidemic – see, for example, Lipsitch et al. [2003], where the early stages of SARS in Singapore was analysed. Household data is often available in such an emerging epidemic scenario; Cauchemez et al. [2009] and House et al. [2012] provided analyses of such data for the transmission of Influenza A in the United States and England respectively. Ball and Shaw [2015], Ball and Shaw [2016] conducted inference for an emerging epidemic in the SIR households model. They developed an asymptotically unbiased estimate of the within-household infection rate, noting that data augmentation methods would become computationally expensive for large household sizes. Trapman et al. [2016] investigated the effect of population heterogeneity (such as network structure and household structure) and show that often the assumption of a homogeneously mixing population leads to overestimates of R_0, thus providing conservative estimates for the herd immunity level. The very recent work of Ball and Neal [2025] offers fast likelihood methods for emerging SEIR epidemics without the requirement for the aforementioned (computationally expensive) data augmentation methods.

Another quantity of interest for an emerging epidemic is the exponential growth rate r. Ball et al. [2015] noted the calculation of r for an epidemic as an open problem, although Fraser [2007] provided a closed-form approximation for calculating r for households models and further progress is made in Ball et al. [2016]. Fraser's work notes that "the exponential growth rate r for an exponentially growing epidemic is the same whether measured for individual or household incidence". This fact is useful when attempting to calibrate household models with different household structures. We note that, for homogeneously mixing models, R_0 is determined by r and the distribution of the time between an infection and the infections that result from it (the so-called generation interval), so there is a connection between estimation of R_0 and r;

see, for example, Ma et al. [2014] and Wallinga and Lipsitch [2007]. Indeed, an estimate of r is often used to obtain an estimate of R_0 in an emerging epidemic – see Ball et al. [2016] for discussion of the connection of R_0 and r for households models. Ross et al. [2010] consider the calculation of R_0 and r for the households model for an SIRS disease framework. In this thesis we calculate herd immunity levels for a variety of model parameters, rather than estimating these model parameters directly. The exception to this is in Section 2.5.3, where we infer real-world household size distributions from data.

We now briefly discuss parameter estimation using final outcome data. The process of infection is typically very difficult to estimate, owing to the times of infection being unknown. Becker [1993] noted this problem and attempted to resolve it by treating missing data as parameters in the framework of the expectation-maximisation algorithm. Becker and Britton [1999] applied martingale methods in order to infer several epidemic parameters with both complete and incomplete data. The work of Gibson and Renshaw [1998] (see also O'Neill and Roberts [1999]) transformed this area by providing a Bayesian framework for parameter estimation for the Reed-Frost model as well as the general stochastic epidemic. Their approach involves Markov chain Monte Carlo methods and variations of such methods remain widely used. O'Neill et al. [2000] developed this further by considering data pertaining to final outcomes in households. Demiris and O'Neill [2005] and Knock and O'Neill [2014] are further such examples of inference pertaining to epidemic models with two levels of mixing (such as households) using final outcome data.

1.6 Recent herd immunity studies

Herd immunity has received considerable study in light of the recent COVID-19 pandemic. We provide a few examples of its study amidst a great deal of literature. Fontanet and Cauchemez [2020] provided an early overview of estimates of herd immunity levels for COVID-19. Kwok et al. [2020] provided further such herd immunity estimates for several countries, and discussed whether pursuing disease-induced herd immunity as a primary means to combat COVID-19 could lead to unacceptable death tolls. Bernal et al. [2022] estimated R_0 for

COVID-19 in the UK between January and March 2020, using a model with households and communities. Okell et al. [2020] discussed whether deaths due to COVID-19 had reduced (at their time of writing) due to the population achieving herd immunity, concluding that the reduction was consistent with lockdown being effective in curtailing transmission, rather than herd immunity having been achieved.

An important question regarding a novel epidemic outbreak is what fraction of the population must be infected in order to protect the remaining susceptible population from a major outbreak. Under the assumption of a homogeneously mixing epidemic this quantity is $1-R_0^{-1}$ independently of whether immunity is gathered by vaccination or by infection and subsequent recovery. Britton et al. [2020] considered a model in which the population is stratified by age and activity level, showing that the disease-induced herd immunity level can be much lower than the vaccine-induced level for such a model. Similarly, Gomes et al. [2022] also found considerable reductions to the disease-induced herd immunity level for a model in which individual susceptibility varies. The work of Britton et al. [2020] considered a novel approximation to the herd immunity level which is obtained by applying restrictions, corresponding to reducing the mixing rate by a multiplicative factor, such that a first epidemic terminates with the population at herd immunity. We consider such an approximation for a population partitioned into households, and also for a model with activity levels and household structure, in Chapter 2 and Chapter 5 respectively. In these models, we assume that restrictions reduce the global mixing rate, but the within-house mixing rate is left unaffected. In Chapter 6 we extend this to the case where restrictions affect global and local mixing.

The multiplicative factor mentioned previously could, in practice, correspond to non-pharmaceutical measures such as social distancing and wearing masks. Flaxman et al. [2020] provided a broad overview of such interventions across Europe for the early stages of COVID-19, promoting continued intervention. Major intervention cannot last permanently owing to political, social and financial implications, so it is important to be cautious when relaxing restrictions where a population have not yet achieved herd immunity. Britton et al. [2021]

warned of a new wave of COVID-19 under these conditions. They also, perhaps counterintuitively, envisage a situation in which removal of strong restrictions can lead to more individuals becoming infected than removal of weaker restrictions. We consider a similar situation in the framework of the households model in Chapter 6, conjecturing that our findings generalise to a broader class of epidemic models.

This thesis primarily aims to develop a framework by which the disease-induced and vaccine-induced herd immunity levels h_D and h_C can be compared in a structured population. Whilst in practice vaccination is unlikely to be allocated uniformly at random, such a strategy provides a good baseline for comparison with disease-induced herd immunity. In any case, disease-induced herd immunity is of value to study; one may envisage a scenario where vaccination attempts prove unsuccessful, so that the only option for protecting the population (beyond constant, total lockdown) is that of disease-induced herd immunity.

We have provided a broad overview of this thesis in the context of wider mathematical epidemiology, as well as motivating the use of a model with individual types and household structure. The literature mentioned is a small subset of such a vast area of study, some of which contains more complex models than those used in this thesis. However, we note the benefit of models which impose realistic assumptions regarding the population of interest, whilst maintaining sufficient mathematical tractability that the models in question can still be analysed.

1.7 Thesis outline

The remainder of this thesis is structured as follows. In Chapter 2 we define the households epidemic model and give mathematical definitions of vaccine-induced and disease-induced herd immunity. We define also an approximation to the disease-induced herd immunity level, following Britton et al. [2020]. We then compare these herd immunity levels in detail, obtaining several explicit results in the case of a highly locally infectious epidemic. The main conclusion of the chapter is that, for the households model, we find that the disease-induced herd immunity level is typically higher than the vaccine-induced herd immunity

level. Chapter 2 is strongly based on our published work - see Ball et al. [2023].

In Chapter 3 we again consider the households model, as well as introducing the multitype model with proportionate mixing. Our attention is focused on establishing a Gaussian approximation to the disease-induced herd immunity level, from which we deduce the asymptotic variance of the disease-induced herd immunity level. In order to achieve this, we apply the theory of density dependent population processes described in Ethier and Kurtz [1986], Chapter 11. We find that the asymptotic variance is typically small, corroborating this with stochastic simulations.

We combine the multitype model with proportionate mixing with the households model in Chapter 4, in which we study the stochastic SIR multitype households model with proportionate global mixing. We first outline how the assumption of proportionate mixing simplifies the calculation of reproduction numbers, such as R_*. Our main result concerns a joint central limit theorem for random variables associated with the final outcome of the epidemic. A result of this form for the multitype households model is previously established in Ball and Lyne [2001]. However, we show how the assumption of proportionate global mixing greatly simplifies the both calculation and the resulting central limit theorem. We also provide a new method for analysing a major outbreak in this model by modifying the embedding construction which underpins the analysis of the epidemic process.

In Chapter 5 we define the activity level and households model as an important special case of the model studied in Chapter 4 and study it in the context of herd immunity. Since heterogeneous mixing typically decreases the disease-induced herd immunity level and household structure increases it, we investigate how these two factors compare in a model which includes them both. We show that the rate at which highly active individuals mix locally with less active individuals can be crucial in determining which of the vaccine-induced and disease-induced herd immunity levels is greater.

The impact of global restrictions on disease-induced herd immunity is investigated for the households model in Chapter 6. We determine a new disease-induced herd immunity level under the assumption that global restrictions lead

to heightened levels of local mixing. We also consider the scenario in which global restrictions are lifted before herd immunity has been achieved, leading to two waves of infection. In this context we prove that, in the homogeneously mixing model, the optimal control, in terms of minimising the number of individuals infected, is to allow the first epidemic to end just as herd immunity is achieved. We then generalise this result to the multitype households model with proportionate global mixing. Finally, in Chapter 7 we provide some concluding comments, discuss model limitations and suggest extensions of this work as well as potential avenues for future research.

We provide a definition of Multivariate Gontcharoff polynomials, which are used several times in our analysis, in Appendix A, where we also define the vector notation which is used throughout this thesis. In Appendix B we give a definition of Wald's identity for epidemics, which is used in several proofs in this thesis. In Appendix C we provide details underpinning the numerical results in this thesis, including stochastic simulations and solutions to ordinary differential equations.

2 Impact of household structure on disease-induced herd immunity

2.1 Introduction

During the ongoing COVID-19 pandemic there has been considerable discussion of herd immunity. For a very wide range of epidemic models, specifically models for which the basic reproduction number R_0 is given by the maximal eigenvalue of a next-generation matrix, if R_0 is greater than one, then vaccinating a fraction $h_C = 1 - R_0^{-1}$ of the population, chosen uniformly at random, with a perfect vaccine (i.e. one that necessarily renders its recipient immune to the disease) in advance of an outbreak reduces the reproduction number to one and thus prevents a large outbreak (see, for example, Diekmann et al. [2013], page 199). The quantity h_C is the classical (or vaccination-induced) herd immunity level. For a disease in which infection confers immunity to subsequent infection, herd immunity can also be attained by letting an epidemic run its natural course, possibly with some restrictions in place, for example, lockdown or other non-pharmaceutical interventions. The disease-induced herd immunity level h_D is the fraction of the population that needs to be infected before the effective basic reproduction number (i.e. R_0 for an epidemic among the remaining susceptible individuals) is reduced to one. For definiteness, we define h_D assuming no restrictions are in place and the epidemic simply runs its natural course.

For an epidemic among a homogeneously mixing population, the classical and disease-induced herd immunity levels are equal. However, that typically is not the case for epidemics among heterogeneous populations. For example, Britton et al. [2020] showed that in a model for COVID-19 in which the population was structured by age and activity level, when $R_0 = 2.5$, the disease-induced herd immunity level $h_D = 0.43$, which is substantially lower than $h_C = 0.6$, and Gomes et al. [2022] obtained even lower values for h_D in a model where individuals varied in susceptibility. These observations have a simple intuitive explanation. For example, in the model with varying susceptibility, individuals with higher susceptibility are more likely to be infected early in the epidemic

and consequently the average susceptibility of the remaining susceptibles decreases as the epidemic progresses leading to $h_D < h_C$. It seems likely that similar arguments hold for many other forms of heterogeneities, with the general conclusion that introducing heterogeneity into a model has the effect of reducing the disease-induced herd immunity level h_D.

An important population structure for epidemics among human populations, which can have a significant impact on disease dynamics and the performance of vaccination strategies, is that induced by households. The aim of this chapter is to investigate the impact of household structure on the disease-induced herd immunity level. We use an extension of the SIR (susceptible $\rightarrow$ infective $\rightarrow$ recovered) model introduced by Ball et al. [1997] to include an exposed (latent) period. In this model, infective individuals make two types of infectious contacts: *local* contacts with individuals chosen uniformly at random from their household and, at a much lower rate, *global* contacts with individuals chosen uniformly at random from the whole population. In sharp contrast to most other forms of heterogeneity, we find that the effect of household structure is generally to *increase* h_D. Other examples of heterogeneity for which $h_D < h_C$ include spatial epidemics – see Rass and Radcliffe [2003] and network epidemic models with significant transitivity – see Kiss et al. [2017].

For most models it is difficult to calculate h_D as it requires knowledge of the trajectory of the epidemic, which typically is not available in closed form. Moreover, in a stochastic model the disease-induced herd immunity level is in fact a random variable, as it depends on the realised trajectory, which converges to h_D as the population size converges to infinity. The following approximation to h_D, which we adapt to our model, is used in Britton et al. [2020] in the context of a multitype SIR epidemic model. A new model is considered in which *all* transmission rates are multiplied by a factor $\kappa < 1$ and its (limiting deterministic) final outcome is determined. (Note that this factor is denoted by α in Britton et al. [2020].) Let $\hat{\kappa}$ be the value of κ so that the effective R_0 among the remaining susceptibles is one. Then the fraction of the population ultimately infected by the epidemic with $\kappa = \hat{\kappa}$ gives an approximation to h_D, which we denote by $\tilde{h}_D$. Note that $\tilde{h}_D$ is not affected by the introduction of a latent pe-

riod into the model, as the distribution of the final size of a stochastic SEIR (susceptible $\rightarrow$ exposed $\rightarrow$ infective $\rightarrow$ recovered) model is invariant to very general assumptions concerning the latent period. We adopt a similar approach to obtain an approximation $\tilde{h}_D$ to h_D for the above households model, except that only global transmission rates are multiplied by κ, with local transmission rates unchanged. (The modification of the global infection rate is used to mimic global restrictions being imposed, which do not affect local mixing rates since individuals remain free to mix within their household at their usual rate.)

The above definition of h_D assumes that no restrictions are in place. Let $\hat{h}_D$ be a generic notation for the disease-induced herd immunity level under restrictions. In practice, $\hat{h}_D$ may depend on the precise pattern of restrictions imposed prior to herd immunity being reached (see, for example, Di Lauro et al. [2021]). A commonly-made assumption in modelling restrictions is that at time $t \geq 0$ *all* transmission rates are multiplied by a factor $\kappa(t)$. Under this assumption, Britton et al. [2021] show that $\hat{h}_D$ is independent of $\{\kappa(t) : t \geq 0\}$ under the assumption of proportionate mixing. Moreover, for the examples in Britton et al. [2020] and Britton et al. [2021], numerical studies showed that the precise timings of restrictions had minimal, if any, effect on $\hat{h}_D$. The situation is more subtle if restrictions are not applied uniformly, where for some models $\hat{h}_D$ can be highly dependent on the pattern of restrictions (Di Lauro et al. [2021]). However, numerical studies indicate that is not the case for the present households model, with restrictions affecting only global transmission rates. Note that $\tilde{h}_D = \hat{h}_D$ when such restrictions with factor $\hat{\kappa}$ are applied throughout the epidemic. Numerical studies suggest that, under many restrictions, $\tilde{h}_D$ is a better approximation than h_D to $\hat{h}_D$.

The usual definition of R_0 as the maximal eigenvalue of a next-generation matrix, or in non-mathematical terms as the mean number of infectious contacts made by a typical infective in an otherwise susceptible population, does not hold for the present households SEIR model, since even in the early stages of an epidemic there are likely to be repeat local contacts within a household. Pellis et al. [2012] give an alternative definition of R_0 via a linear approximation of the early phase of an epidemic in terms of generations of infections, which

coincides with the usual definition when it is applicable but can also be extended to models with small mixing groups such as the households SEIR model. Calculation of R_0 for the households SEIR model is quite complex. A simpler to calculate reproduction number for the households SEIR model is R_* (see Ball et al. [1997]), which is based on the proliferation of infected households (rather than infected individuals). The threshold values of R_0 and R_* are both one. The reproduction number R_* is useful for determining herd immunity levels, owing to its ease of calculation. However, it is not comparable between different household structures, unlike R_0, because it is a households-based rather than an individual-based reproduction number.

Before describing the main results, we need some more notation. Let λ_L denote the individual-to-individual local infection rate and λ_G denote the overall rate that an infective makes global contacts. Further, let H be a random variable describing the size of a household chosen uniformly at random and $\tilde{H}$, the size-biased version of H, be a random variable describing the size of the household to which an individual chosen uniformly at random from the population belongs (see Section 2.2.1). Let $\mu_{\tilde{H}} = \mathrm{E}[\tilde{H}]$. For $i = 1, 2, \ldots$, let $\mu_{\tilde{H}}^{[i]} = \mathrm{E}[\tilde{H}(\tilde{H}-1)\ldots(\tilde{H}-i+1)]$ denote the ith factorial moment of $\tilde{H}$ and $\hat{\mu}_{\tilde{H}}^{[i]} = i!\mu_{\tilde{H}}(\mu_{\tilde{H}}-1)^{i-1}$ be the ith factorial moment of a geometric distribution with success probability $\mu_{\tilde{H}}^{-1}$.

The complexities of the households model render analytical results comparing $\tilde{h}_D$ and h_C hard to obtain in general. First, we consider epidemics which are highly locally infectious, in that if one individual in the household becomes infected then the whole household becomes infected. This assumption, which was introduced in Becker and Dietz [1995], is obtained by letting $\lambda_L = \infty$ in our model. Under this assumption, the following are our main results.

- Theorem 2.1. If all households have size $n > 1$ and $R_* > 1$, then $\tilde{h}_D > h_C$.
- Theorem 2.2. Under the conditions of Theorem 2.1, $\tilde{h}_D - h_C$ is maximised as a function of λ_G when $R_0 = 2$.
- Theorem 2.4. $\tilde{h}_D = h_C$ for all λ_G such that $R_* > 1$ if and only if $\tilde{H}$ follows a geometric distribution, so H follows a logarithmic distribution.

- Theorem 2.5. Suppose that the epidemic is only just above threshold (i.e. R_* is only just above 1) and $l^* = \inf_{k\geq 2}\{k : \mu_{\tilde{H}}^{[k]} \neq \hat{\mu}_{\tilde{H}}^{[k]}\} < \infty$. Then $\tilde{h}_D > h_C$ if $\mu_{\tilde{H}}^{[l^*]} < \hat{\mu}_{\tilde{H}}^{[l^*]}$ and $\tilde{h}_D < h_C$ if $\mu_{\tilde{H}}^{[l^*]} > \hat{\mu}_{\tilde{H}}^{[l^*]}$.

- Corollary 2.6. Under the conditions of Theorem 2.5, $\tilde{h}_D > h_C$ if $\mathrm{var}(\tilde{H}) < \mathrm{E}[\tilde{H}]\mathrm{E}[\tilde{H}-1]$ and $\tilde{h}_D < h_C$ if $\mathrm{var}(\tilde{H}) > \mathrm{E}[\tilde{H}]\mathrm{E}[\tilde{H}-1]$.

- Theorem 2.7 gives an ordering of $\tilde{h}_D$ and h_C for epidemics which are both highly locally and highly globally infectious. The result is not given explicitly here as it requires appreciable further notation.

- Theorem 2.8. Suppose that $n > 1$ and $\mathrm{P}(\tilde{H} = n) = p = 1 - \mathrm{P}(\tilde{H} = 1)$, where $0 < p < 1$, so a fraction p of individuals reside in households of size n and the remainder in households of size 1. If $n = 2$, then $\tilde{h}_D > h_C$ for all p. For $n \geq 3$, if $p \leq \frac{n-2}{2(n-1)}$ then $\tilde{h}_D < h_C$ for all λ_G. If $p > \frac{n-2}{2(n-1)}$ then there exists $\lambda_G^*(n,p)$ such that $\tilde{h}_D > h_C$ for $\lambda_G < \lambda_G^*(n,p)$ and $\tilde{h}_D < h_C$ for $\lambda_G > \lambda_G^*(n,p)$.

- An expression for $\lambda_G^*(n,p)$ involving the root of an algebraic equation is given in Theorem 2.8. If p is close to 1 (i.e. the households are nearly all of size n) then λ_G, and thus R_0, must be exceedingly large in order to obtain $\tilde{h}_D < h_C$.

Analysis is also possible for epidemics that are weakly locally infectious, i.e. when λ_L is close to 0. We assume without loss of generality that the infectious period T_I has mean 1, and write $\tilde{h}_D(\lambda_L)$ and $h_C(\lambda_L)$ to show explicitly the dependence of these herd immunity levels on λ_L. Note that the model reduces to a standard homogeneously mixing SEIR epidemic when $\lambda_L = 0$, for which $R_0 = \lambda_G$ since $\mathrm{E}[T_I] = 1$. Thus, $\tilde{h}_D(0) = h_C(0) = 1 - R_0^{-1} = 1 - \lambda_G^{-1}$. Suppose that $\lambda_G > 1$, so $R_* > 1$, and let $\pi(0) = \frac{1}{\lambda_G}$.

- Theorem 2.12. As $\lambda_L \downarrow 0$,

$$\tilde{h}_D(\lambda_L) - h_C(\lambda_L) = 2\lambda_L^2 \pi(0)^2 (1-\pi(0)) \left[\mathrm{E}[\tilde{H}-1] - \mathrm{var}(\tilde{H})\right] + o(\lambda_L^2).$$

- Corollary 2.13. If $\mathrm{var}(\tilde{H}) < \mathrm{E}[\tilde{H}-1]$ then $\tilde{h}_D > h_C$ for all sufficiently

small $\lambda_L > 0$. If $\mathrm{var}(\tilde{H}) > \mathrm{E}[\tilde{H} - 1]$ then $\tilde{h}_D < h_C$ for all sufficiently small $\lambda_L > 0$.

- Corollary 2.14. Suppose all households are the same size $n > 1$. Then, for all sufficiently small $\lambda_L > 0$, we have $\tilde{h}_D > h_C$.

The final theorem concerns the case when $0 < \lambda_L < \infty$ and all households have the same size n.

- Theorem 2.15. For a common household size $n = 2$ or $n = 3$, and for any λ_G and $\lambda_L > 0$ such that $R_* > 1$, we have $\tilde{h}_D > h_C$.

We conjecture, supported by numerical studies, that Theorem 2.15 holds for all $n > 1$.

In Section 2.2, we define the stochastic SEIR households model underlying our analysis, describe briefly its threshold behaviour, calculation of the reproduction numbers, R_* and R_0, and of the final outcome in the event of an epidemic taking off. We also present a deterministic model which approximates epidemics that take off. In Section 2.3, we describe calculation of the vaccine-induced herd immunity level h_C, discuss the definition of the disease-induced herd immunity level h_D and describe in detail its approximation $\tilde{h}_D$. Theorems concerning comparison of $\tilde{h}_D$ and h_C are given in Section 2.4, with some of the longer proofs being deferred to Section 2.7 on account of their length. Numerical comparisons of herd immunity levels are given in Section 2.5, including illustration of theorems and study of herd immunity levels for real-world household size distributions. In Section 2.6, we give some concluding comments and discuss possible directions for future research.

2.2 SEIR households model

2.2.1 Model definition

We consider an SEIR (susceptible $\to$ exposed $\to$ infective $\to$ recovered) model for an epidemic among a closed and finite population separated into households. This is similar to the model in Ball et al. [1997] with an extra (exposed) state. The household structure is given as follows. We suppose, for $n = 1, 2, \ldots$, there

are m_n households of size n. There are $m = \sum_{n=1}^{\infty} m_n$ households (with $m < \infty$), and the total population size is $N = \sum_{n=1}^{\infty} n m_n$.

The epidemic begins at time $t = 0$ with one initial infective (chosen uniformly at random from the population) and with all other members of the population susceptible. When a given susceptible is contacted by an infective, they become exposed (latent infective) for a time that is distributed according to a non-negative random variable T_E, with an arbitrary but specified distribution having finite mean. When their exposed period ends, an individual becomes infectious for a time distributed according to a non-negative random variable T_I, with an arbitrary but specified distribution having finite mean. During their infectious period any given infective makes global contacts with any given susceptible according to a Poisson process with rate $\frac{\lambda_G}{N}$. Further, any given infective makes local contacts with any given susceptibles member of their household according to a Poisson process with rate λ_L. Once their infectious period ends, an infective recovers and has no further role in the epidemic. When there are no infectives or exposed infectives remaining, the epidemic terminates. Finally, all Poisson processes describing infectious contacts (whether or not either or both of the individuals involved are the same), as well as the random variables for exposed and infectious periods, are assumed to be mutually independent.

Many of the results in this chapter are based on approximations which become exact in the limit as the number of households $m \to \infty$ in an appropriate fashion. For $n = 1, 2, \ldots$, let $\alpha_n^{(m)} = \frac{m_n}{m}$ be the (deterministic) fraction of households that have size n. Precise conditions for such asymptotic results are beyond the scope of this chapter. We assume that $\lim_{m\to\infty} \alpha_n^{(m)} = \alpha_n$ $(n = 1, 2, \ldots)$, where $\sum_{n=1}^{\infty} \alpha_n = 1$ and $\sum_{n=1}^{\infty} n\alpha_n < \infty$. To ease the presentation we suppress the dependence on m of parameters such as $\alpha_n^{(m)}$ and just use their asymptotic values.

2.2.2 Threshold behaviour

Suppose that the number of households (m) is large. Since the epidemic begins with one initial infective, the probability that an individual contacted globally belongs to a previously infected household is small during the early stages of the epidemic. Thus the early stages of the epidemic can be approximated by a

branching process, describing the proliferation of infected households, in which every global contact is with a previously uninfected household (see Ball [1996]). The offspring mean R_* of this branching process, i.e. the expected number of global contacts occurring from a typical contacted household, is a threshold parameter for the households model. Standard branching process theory implies that, in the limit as $m \to \infty$, the epidemic takes off with strictly positive probability if and only if $R_* > 1$. In the event the epidemic takes off, a non-negligible fraction (a large number of households) of the population becomes infected.

The derivation of R_* is as follows. For $n = 1, 2, \ldots$, let $\tilde{\alpha}_n = \frac{nm_n}{N}$ be the probability an individual chosen uniformly at random from the population resides in a household of size n. Consider a globally contacted individual in an otherwise fully susceptible household of size n. This individual begins a local outbreak within their household with dynamics determined by local infection since, in the branching process, global contacts are with previously fully susceptible households. Let $\mu_n(\lambda_L)$ denote the mean size, including the initial infective, of a single-household epidemic with n members and local infection rate λ_L with only the initial infective infected globally. Global contacts from a given individual occur at rate λ_G, and such an individual has mean infectious period $\mathrm{E}[T_I]$. Wald's identity for epidemics (Lemma B.1) then gives the mean number of global contacts from a given contacted household of size n to be $\mu_n(\lambda_L)\lambda_G\mathrm{E}[T_I]$. By conditioning on the size of a household of a contacted individual, we have that

$$R_* = \sum_{n=1}^{\infty} \tilde{\alpha}_n \mu_n(\lambda_L)\lambda_G \mathrm{E}[T_I]. \tag{2.1}$$

In Ball [1986] it is shown that

$$\mu_n(\lambda_L) = n - \sum_{k=1}^{n-1} \binom{n-1}{k} \beta_k(\lambda_L)\phi(k\lambda_L)^{n-k}, \quad n = 1, 2, \ldots, \tag{2.2}$$

where $\phi(\theta) = \mathrm{E}[\mathrm{e}^{-\theta T_I}]$ and $\beta_k(\lambda_L)$ $(k = 1, 2, \ldots)$ are defined recursively by

$$\sum_{i=1}^{k} \binom{k}{i} \beta_i(\lambda_L)\phi(i\lambda_L)^{k-i} = k, \quad k = 1, 2, \ldots.$$

As explained in Section 2.1, a drawback of R_* is that it is not comparable between models with different household structures. An alternative threshold parameter, which does not suffer from that defect, is the basic reproduction number R_0. As also explained in Section 2.1, the usual definition of R_0 does not hold for households models. Instead, R_0 can be defined by considering generations of infectives, via a directed graph associated with an epidemic (Pellis et al. [2012] and Ball et al. [2016]). Such a graph is constructed by having population members as the vertices. If, during their infectious period, individual x would contact x', a directed edge is drawn from x to x'. The initial infective is the only member of generation 0. The generation of a given individual x is the shortest path length from the initial infective to x. Note that this may not coincide with real-time generations of infectives. Then R_0 is defined by the limit, as the population size goes to infinity, of the asymptotic geometric growth rate of the mean generation size (for a full definition, see Ball et al. [2016], Section 1). For $k = 1, 2, \ldots$ and $i = 0, 1, \ldots, k-1$, let $\mu_i^{(k)}$ be the mean size of the i^{th} generation for an epidemic in a household of size k with 1 initial infective. (For the present SEIR model, these quantities can be computed using methods described in Appendix A of Pellis et al. [2012].) Then R_0 in the households model is the unique positive solution λ of

$$1 - \lambda_G \mathrm{E}[T_I] \sum_{i=0}^{\infty} \frac{\mu_i}{\lambda^{i+1}} = 0, \tag{2.3}$$

where $\mu_i = \sum_{n=1}^{\infty} \tilde{\alpha}_n \mu_i^{(n)}$; see Ball et al. [2016], Section 2.2 for details.

Note that, like R_*, the critical value of R_0 is 1. More precisely, $R_0 = 1$ if and only if $R_* = 1$; $R_0 > 1$ if and only if $R_* > 1$; and $R_0 < 1$ if and only if $R_* < 1$. We use R_* for calculating or proving results pertaining to herd immunity levels, as it is far simpler to determine than R_0. We use R_0 when making comparisons between models, owing to its improved interpretability. (For further discussion of how R_* is affected by changing household structures, see Ball et al [2010], Section 4.4, which considers a random network model incorporating household structure.)

2.2.3 Early exponential growth rate r

The early exponential growth rate (Malthusian parameter) r of the SEIR households model is defined as follows. Consider the branching process of infected households described in Section 2.2.2. Then the Malthusian parameter, r, for the branching process satisfies

$$\int_0^\infty \mathrm{e}^{-rt}\beta(t)\,\mathrm{d}t = 1, \tag{2.4}$$

where $\beta(t)$ is the mean rate of global contacts emanating from a typical single-household epidemic, t time units after the household is infected. For many choices of distributions for T_E and T_I, the left-hand-side of (2.4) is not tractable; in Ball et al. [2016], Section 2.8, ways to approximate it are considered. We restrict attention to the case $T_E \sim \mathrm{Exp}(\delta)$ and $T_I \sim \mathrm{Exp}(\gamma)$, in which case the dynamics are Markovian and the left-hand side of (2.4) can be computed as outlined below, cf. Pellis et al. [2011], Section 4.2, which considers an SIR model. This problem is treated, in a more general setting, by Pollett and Stefanov [2002].

In the branching process in Section 2.2.2, individuals correspond to infected households, and an individual gives birth whenever a global contact arises from the corresponding single-household epidemic. We extend Ball and Shaw [2015], Section 4.1, to include an exposed state. For $n = 1,2,\ldots$, let $\tilde{E}_H^{(n)}$ denote a typical single-household epidemic in a household of size n, initiated by one member becoming exposed at time $t = 0$. For $t \geq 0$, let $S_H^{(n)}(t)$, $E_H^{(n)}(t)$ and $I_H^{(n)}(t)$ denote respectively the number of susceptibles, exposed and infected individuals in $\tilde{E}_H^{(n)}$ at time t. Let $\mathscr{F}^{(n)} = \{(s,e,i) \in \mathbb{Z}_+^3 : s+e+i \leq n\}$ be the set of possible household states for a household of size n. For $(s,e,i) \in \mathscr{F}^{(n)}$ and $t \geq 0$, let $p_{s,e,i}^{(n)}(t) = \mathrm{P}\left((S_H^{(n)}(t), E_H^{(n)}(t), I_H^{(n)}(t)) = (s,e,i)\right)$. Let $S_n = \left|\mathscr{F}^{(n)}\right| = \frac{n}{6}\left(n^2+6n+11\right)$. For a given household of size n, we have, in an obvious notation,

$$\beta^{(n)}(t) = \lambda_G \sum_{(s,e,i)\in\mathscr{F}^{(n)}} i p_{s,e,i}^{(n)}(t),$$

which after conditioning on the size of a typical household in the approximating

branching process yields

$$\beta(t) = \lambda_G \sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{(s,e,i)\in\mathscr{F}^{(n)}} i p_{s,e,i}^{(n)}(t).$$

Hence, using (2.4), r satisfies

$$\lambda_G \sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{(s,e,i)\in\mathscr{F}^{(n)}} i \tilde{p}_{s,e,i}^{(n)}(r) = 1, \tag{2.5}$$

where

$$\tilde{p}_{s,e,i}^{(n)}(r) = \int_0^{\infty} \mathrm{e}^{-rt} p_{s,e,i}^{(n)}(t)\,\mathrm{d}t.$$

To calculate $\tilde{p}_{s,e,i}^{(n)}(r)$, let $Q^{(n)} = \left[q_{i,j}^{(n)}\right]$ be the $S_n \times S_n$ transition-rate matrix of $\tilde{E}_H^{(n)}$, with states labelled such that $(n-1,1,0)$ is state 1. Suppose $k \in \{1,2,\ldots,S_n\}$ corresponds to the state $(s,e,i) \in \mathscr{F}^{(n)}$. Then

$$p_{s,e,i}^{(n)}(t) = \left(\mathrm{e}^{tQ^{(n)}}\right)_{1,k},$$

where $\mathrm{e}^A = \sum_{i=0}^{\infty} \frac{A^i}{i!}$ denotes the matrix exponential, which always converges – see Bhatia [2013]. Hence, for $r > 0$,

$$\tilde{p}_{s,e,i}^{(n)}(r) = \left([rI_{S_n} - Q^{(n)}]^{-1}\right)_{1,k}, \tag{2.6}$$

which exists since the matrix $\left[rI_{S_n} - Q^{(n)}\right]$ is diagonally dominant for all $r > 0$. We then compute r using using (2.5) and (2.6).

Further, if $\delta, \gamma, \lambda_L$ and the household structure are known and fixed then, for a given value of the early growth rate r, the corresponding λ_G is readily obtained using (2.5) and (2.6).

2.2.4 Final outcome

Consider an epidemic initiated by one initial infective in a population of size N (m households). Let Z_m denote the proportion of individuals infected in the epidemic. Provided $R_* > 1$, as $m \to \infty$, Z_m converges in probability to a discrete

random variable Z with probability mass function

$$\mathrm{P}(Z=z) = 1 - \mathrm{P}(Z=0),$$

for some $0 < z < 1$ defined below. (The mass at zero in the random variable Z corresponds to the branching process in the previous section going extinct.) We define a major outbreak to have occurred if $Z_m \approx z$ and it follows that the sum of the infectious periods of all infected individuals in a major outbreak, S_m, is approximately $NzE[T_I]$. Hence, the probability that a randomly chosen individual avoids global infection during the course of a major outbreak is approximately $\pi = \exp(-\lambda_G E[T_I]z)$, since to avoid global infection there must be no points in a Poisson process of intensity λ_G/N run for time $S_m \approx NzE[T_I]$.

Let $\tilde{\mu}_n(\lambda_L, \pi)$ denote the mean size of a single-household epidemic in a household of size n with local infection rate λ_L and $\mathrm{Bin}(n, 1-\pi)$ initial (globally infected) infectives; using the standard $\mathrm{Bin}(n,p)$ notation to denote the binomial distribution. Denote this epidemic model, which is considered in Addy et al. [1991], by $\tilde{E}_n(\lambda_L, \pi)$. Returning to the households model, suppose that a major outbreak occurs and let $\tilde{T}_n$ denote the total number of individuals infected in a typical household of size n, all of whom are initially susceptible. In the limit as $m \to \infty$, individuals independently avoid global infection with probability π. Thus in a household of size n, $\mathrm{Bin}(n, 1-\pi)$ will be infected globally and hence $\mathrm{E}[\tilde{T}_n] = \tilde{\mu}_n(\lambda_L, \pi)$. Then equation (3.10) of Ball et al. [1997] yields

$$\tilde{\mu}_n(\lambda_L, \pi) = n - \sum_{k=1}^{n} \binom{n}{k} \phi(k\lambda_L)^{n-k} \pi^k \beta_k(\lambda_L).$$

The probability that a given individual in an initially susceptible household of size n is infected during the epidemic is approximately $\tilde{\mu}_n(\lambda_L, \pi)/n$. Conditioning on the household size of a randomly chosen individual then establishes

$$z = \sum_{n=1}^{\infty} \tilde{\alpha}_n \frac{\tilde{\mu}_n(\lambda_L, \pi)}{n}. \tag{2.7}$$

Note that for large m the effect of the atypical behaviour of the household containing the initial infective becomes negligible and disappears in the limit as

$m \to \infty$. Thus, since $\pi = \exp(-\lambda_G \mathrm{E}[T_I]z)$, (2.7) admits an implicit equation for z, whereby $z = 0$ is always a solution and a second solution $z \in (0,1)$ exists if and only if $R_* > 1$. A similar argument can be used to establish, in the event of a major outbreak, the proportions $(P_{n,v})$ $(n = 1,2,\ldots; v = 0,1,\ldots,n)$ of households of size n with v members ultimately infected. For $n = 1,2,\ldots$, $(P_{n,v})$ satisfies the system of equations (see Addy et al. [1991], equation 4)

$$\sum_{i=0}^{v} \binom{n-i}{v-i} \frac{P_{n,i}}{\phi((n-v)\lambda_L)^i \pi^{n-v}} = \binom{n}{v}, \qquad v = 0,1,\ldots,n. \tag{2.8}$$

The above arguments are made rigorous in Ball et al. [1997], Section 4.2 and hold with or without the inclusion of a latent period, see Ball et al. [1997], Section 3.1.

2.2.5 Deterministic model

In House and Keeling [2008], Section 2, a system of ordinary differential equations (ODEs) is derived for the evolution over time of the SIR epidemic model with households, assuming that $T_I \sim \mathrm{Exp}(\gamma)$, i.e. the infectious period distribution is exponential with mean γ^{-1}. House and Keeling's ODEs represent the deterministic limit of the stochastic process defined in Section 2.2.1 as $m \to \infty$, under the assumptions that all households are the same size and there is no latent period. We extend the system of ODEs to allow for variable household size and a latent period $T_E \sim \mathrm{Exp}(\delta)$.

Consider the model in Section 2.2.1 with maximum household size $n_{\max}$. Let

$$\mathscr{H}^{(n_{\max})} = \{(s,e,i,r) \in \mathbb{Z}_+^4 : 1 \le s+e+i+r \le n_{\max}\}, \tag{2.9}$$

where $\mathbb{Z}_+ = \{0,1,\ldots\}$, and for $t \ge 0$ and $(s,e,i,r) \in \mathscr{H}^{(n_{\max})}$, denote by $H^{(m)}_{s,e,i,r}(t)$ the number of households with s susceptible, e exposed, i infectious and r recovered members at time t. For $n = 1,2,\ldots,n_{\max}$, let

$$\mathscr{H}_n = \{(s,e,i,r) \in \mathscr{H}^{(n_{\max})} : s+e+i+r = n\}.$$

Then, $\sum_{(s,e,i,r)\in\mathscr{H}_n} H^{(m)}_{s,e,i,r}(t) = m_n$ for all $t \geq 0$. Let

$$\mathscr{H}_+^{(n_{\max})} = \{(s,e,i,r) \in \mathscr{H}^{(n_{\max})} : e+i > 0\} \tag{2.10}$$

be the set of states in which there is at least one infective or exposed individual. For $(s,e,i,r) \in \mathscr{H}^{(n_{\max})}$, we assume the deterministic initial condition $\frac{1}{m}H^{(m)}_{s,e,i,r}(0) \to h_{s,e,i,r}(0)$ as $m \to \infty$, where $\sum_{(s,e,i,r)\in\mathscr{H}_+^{(n_{\max})}} h_{s,e,i,r}(0) > 0$, so a strictly positive fraction of the population is initially either exposed or infective in the limit as $m \to \infty$. Then, under the Markovian assumption, we have that $\frac{1}{m}H^{(m)}_{s,e,i,r}(t)$ converges in probability to a deterministic process $h_{s,e,i,r}(t)$ as $m \to \infty$; see Ethier and Kurtz [1986], Theorem 11.2.1. Clearly, for $n = 1,2,\ldots,n_{\max}$, we have $\sum_{(s,e,i,r)\in\mathscr{H}_n} h_{s,e,i,r}(t) = \alpha_n$ for all t. Let $\bar{i}(t) = \sum_{(s,e,i,r)\in\mathscr{H}^{(n_{\max})}} i h_{s,e,i,r}(t)$. The deterministic model (cf. Black et al [2014], equation (B.5)) can be obtained by considering the possible transition rates between household states and yields, for $(s,e,i,r) \in \mathscr{H}^{(n_{\max})}$,

$$\begin{aligned}
\frac{d}{dt}h_{s,e,i,r} =& \delta\left(-eh_{s,e,i,r} + (e+1)h_{s,e+1,i-1,r}\right) \\
&+ \gamma\left(-ih_{s,e,i,r} + (i+1)h_{s,e,i+1,r-1}\right) \\
&+ \lambda_G\bar{i}(t)\left(-sh_{s,e,i,r} + (s+1)h_{s+1,e-1,i,r}\right) \\
&+ \lambda_L\left(-sih_{s,e,i,r} + (s+1)ih_{s+1,e-1,i,r}\right),
\end{aligned} \tag{2.11}$$

where, on the right-hand side, $h_{s',e',i',r'}(t) = 0$ if $(s',e',i',r') \notin \mathscr{H}^{(n_{\max})}$.

2.3 Herd immunity in SEIR households model

In this section we outline the various versions of herd immunity that we consider. We start by recapping vaccine-induced herd immunity; we then describe and explore the details of the disease-induced herd immunity level h_D and its approximation $\tilde{h}_D$ which are outlined in Section 2.1. In this section, and throughout the remainder of the manuscript, we assume that $R_* > 1$, since if this is not the case then herd immunity is already achieved.

2.3.1 Vaccine-induced herd immunity level h_C

Suppose some members of the population are vaccinated before an epidemic occurs. Assume that such a vaccine is given to each member of the population independently with probability c, and is perfect, so that vaccinated individuals are completely immune to infection. Then, for $v = 0, 1, \ldots, n$, a given household of size n has v members vaccinated according to the (binomial) probability $\binom{n}{v} c^v (1-c)^{n-v}$. We obtain a post-vaccination threshold parameter $\hat{R}_U(c)$ by considering a branching process of potential global contacts. If a potential global contact is with a susceptible individual then it triggers a local epidemic; if the contact is with a vaccinated individual then nothing happens.

The mean number of potential global contacts emanating from a single-household epidemic for a household in state (n, v) that is contacted globally, with the initial infective chosen uniformly at random from members of the household ($\mu_{n,v}$, say) is given by

$$\mu_{n,v} = \left(\frac{n-v}{n} \right) \mu_{n-v}(\lambda_L) \lambda_G \mathrm{E}[T_I],$$

for $n = 1, 2, \ldots$ and $v = 0, 1, \ldots, n$. This is because a vaccinated member being contacted leads to no global contacts at all, and an unvaccinated member being contacted initiates a single-household epidemic amongst the $n - v$ non-immune members. Such an unvaccinated individual is contacted with probability $\frac{n-v}{n}$. Conditioning on the vaccination status of an individual's household, as well as the individual's household size, and using the same argument as for equation (2.1) yields a post-vaccination threshold parameter

$$\hat{R}_U(c) = \sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{v=0}^{n} \binom{n}{v} c^v (1-c)^{n-v} \left(\frac{n-v}{n} \right) \mu_{n-v}(\lambda_L) \lambda_G \mathrm{E}[T_I], \qquad (2.12)$$

with $\mu_0(\lambda_L) = 0$. The function $\hat{R}_U(c)$ is continuous and strictly decreasing, with $\hat{R}_U(0) = R_* > 1$ and $\hat{R}_U(1) = 0$. (To show that $\hat{R}_U(c)$ is strictly decreasing, let $f_n(c) = \sum_{v=0}^{n} \binom{n}{v} c^v (1-c)^{n-v} (n-v) \mu_{n-v}(\lambda_L)$. Then $f_n(c) = \mathrm{E}[g(X)]$, where $g(x) = x\mu_x(\lambda_L)$ and $X \sim \mathrm{Bin}(n, 1-c)$. The function g is strictly increasing and X is stochastically decreasing in c. Thus, $f_n(c)$ is strictly decreasing in c, whence

so is $\hat{R}_U(c)$.) This implies there is a critical value, h_C say, such that $\hat{R}_U(h_C) = 1$ and a major outbreak can be avoided. The quantity h_C is the vaccine-induced herd immunity level. Note that by ensuring $\hat{R}_U(c) \leq 1$, the whole population is considered protected as a major outbreak is no longer possible. This argument regarding uniform vaccination is considered (among other vaccination strategies) in Ball and Lyne [2006].

As noted at the start of Section 2.1, for epidemic models in which R_0 is given by the maximal eigenvalue of a next generation matrix, $h_C = 1 - R_0^{-1}$. For the present households model, R_0 is computed differently, see Section 2.2.2; if $\lambda_L \in (0, \infty)$ then it follows from Ball et al. [2016], Theorem 1, that $h_C \geq 1 - R_0^{-1}$ with equality if and only if $n_{\max} \leq 3$. In the highly locally infectious case ($\lambda_L = \infty$), $h_C = 1 - R_0^{-1}$ for all $n_{\max}$; see Remark 2 following the aforementioned Theorem 1. If $\lambda_L = 0$, the model reduces to a standard homogeneously mixing SEIR epidemic and $h_C = 1 - R_0^{-1}$.

2.3.2 Disease-induced herd immunity

Limiting disease-induced herd immunity level h_D

An alternative method of achieving herd immunity in a population arises from the spread of a first wave of infection, in which infected members from the first wave are considered immunised thereafter.

Consider the SIR version of the households model described in Section 2.2.1, with $T_I \sim \text{Exp}(\gamma)$ and assume this epidemic takes off. As the epidemic progresses some members of the population are infected, lowering the overall susceptibility of the population. Suppose that the first epidemic is stopped (i.e. all infectious spread, including that within households, is stopped) at time $t > 0$. Consider a second epidemic initiated at time t with one initial infective and all those individuals infected by time t in the first epidemic immune to infection in the second. Recalling that m is the number of households in the population, let $R_V^{(m)}(t)$ be the threshold parameter (R_*) for this second epidemic, which is a random variable owing to its dependence on the trajectory of the first epidemic. If $R_V^{(m)}(t) \leq 1$, then the second epidemic is not supercritical and a major outbreak cannot occur. Disease-induced herd immunity is achieved when

the trajectory of $R_V^{(m)}(t)$ crosses one. Write $T_*^{(m)} = \inf\{t \geq 0 : R_V^{(m)}(t) \leq 1\}$ and denote the fraction of the population that is still susceptible at time t in the first epidemic by $S^{(m)}(t)$. Then the disease-induced herd immunity level $H_D^{(m)}$ is given by $H_D^{(m)} = 1 - S^{(m)}\left(T_*^{(m)}\right)$ and is also a random variable determined by the trajectory of the first epidemic. We conjecture that $H_D^{(m)} \xrightarrow{\mathrm{p}} h_D$ as $m \to \infty$, where h_D is a constant, and in the presence of a latent period $T_E \sim \mathrm{Exp}(\delta)$, that $H_D^{(m)} \xrightarrow{\mathrm{p}} h_D^L$ as $m \to \infty$. Moreover, we conjecture that under suitable conditions, these convergence results hold also when T_I and T_E follow non-exponential distributions.

To compute h_D^L when $T_I \sim \mathrm{Exp}(\gamma)$ and $T_E \sim \mathrm{Exp}(\delta)$, we use the deterministic model in Section 2.2.5. Recall the definitions of $\mathscr{H}^{(n_{\max})}$ and $\mathscr{H}_+^{(n_{\max})}$ at (2.9) and (2.10). Let $\boldsymbol{h}(t) = \left(h_{s,e,i,r}(t) : (s,e,i,r) \in \mathscr{H}^{(n_{\max})}\right)$ and suppose that $\boldsymbol{h}(0) = \boldsymbol{\varepsilon}$. Then the deterministic model given by (2.11) can be used in the obvious fashion to define a disease-induced herd immunity level $h_D^L(\boldsymbol{\varepsilon})$. Let $\mathscr{H}_{0\mathrm{rec}} = \{(s,e,i,r) \in \mathscr{H}^{(n_{\max})} : r = 0\}$. We conjecture that $h_D^L(\boldsymbol{\varepsilon}^{(k)}) \to h_D^L$ as $k \to \infty$ for any sequence $(\boldsymbol{\varepsilon}^{(k)})$ satisfying $\sum_{(s,e,i,r) \in \mathscr{H}_+^{(n_{\max})}} \varepsilon_{s,e,i,r}^{(k)} \downarrow 0$ and $\sum_{(s,e,i,r) \in \mathscr{H}_{0\mathrm{rec}}} \varepsilon_{s,e,i,r}^{(k)} \uparrow 1$ as $k \to \infty$. Unless explicitly stated otherwise, when computing h_D^L, we assume that initially a fraction $\varepsilon = 10^{-5}$ of households are in state $(n_{\max} - 1, 0, 1, 0)$, with all other households being fully susceptible. An equivalent assumption is made when computing h_D. Note that, if the largest household is of size n, the system in (2.11) contains exact order $n^4/24$ equations (or $n^3/6$ in the SIR case) and becomes computationally expensive to solve when n is large.

Convergence of $H_D^{(m)}$ to h_D

The proofs of the conjectures in Section 2.3.2 require extending the theory of Barbour and Reinert [2013] to the households model and are beyond the intentions of this chapter. Instead, we comment briefly on a possible approach to proving the conjecture in Section 2.3.2 that $H_D^{(m)} \xrightarrow{\mathrm{p}} h_D^L$ as $m \to \infty$. We also present some numerical evidence in support of that conjecture for the SIR model and for the conjecture underlying the calculation of h_D in that model.

A possible proof of $H_D^{(m)} \xrightarrow{\mathrm{p}} h_D^L$ as $m \to \infty$ is to extend the theory of Barbour and Reinert [2013] to the households SEIR model. (Recall that in the calcu-

lation of $H_D^{(m)}$ there is one initial infective, so the proportion of initially infected individuals converges to zero as $m \to \infty$; the convergence of $H_D^{(m)}$ to h_D^L must be treated carefully as a consequence.) Briefly, such an extension would imply that, as $m \to \infty$, in the event of a major outbreak, the process $\left\{m^{-1}\boldsymbol{H}^{(m)}(t) : t \geq 0\right\}$, where

$$\boldsymbol{H}^{(m)}(t) = \left(H^{(m)}_{s,e,i,r}(t) : (s,e,i,r) \in \mathscr{H}^{(n_{\max})}\right)$$

(see Section 2.2.5), converges in probability to a random time translation of a deterministic process $\{\tilde{\boldsymbol{h}}(t) : -\infty < t < \infty\}$ in which the fraction of the population that is either exposed or infective converges to 0 as $t \to -\infty$. The random time translation arises from the randomness in the initial behaviour of the approximating branching process (see Section 2.2.2) before it settles into its exponentially growing regime. One can use $\tilde{\boldsymbol{h}}(t)$ to define (deterministic) analogues of $R_V^{(m)}(t)$ and $S^{(m)}(t)$ above, $\tilde{R}_V(t)$ and $\tilde{s}(t)$ say, and $h_D^L = 1 - \tilde{s}(\tilde{t}_*)$, where $\tilde{t}_* = \inf\{t \in (-\infty,\infty) : \tilde{R}_V(t) \leq 1\}$.

In Table 2.1 we present an example showing empirical evidence in support of this conjecture for the households SIR model when $T_E \sim \text{Exp}(\gamma)$. We also see in the bottom row of Table 2.1 evidence that $m\text{var}(H_D^{(m)})$ converges to a constant as $m \to \infty$, which is consistent with a central limit theorem for $H_D^{(m)}$.

m	10^2	10^3	10^4	10^5
$\bar{H}_D^{(m)}$	0.533495	0.532459	0.532260	0.532240
$\hat{\sigma}^{(m)}$	1.70×10^{-2}	1.67×10^{-3}	5.29×10^{-4}	1.67×10^{-4}
$m(\hat{\sigma}^{(m)})^2$	2.90×10^{-2}	2.78×10^{-3}	2.80×10^{-3}	2.80×10^{-3}

Table 2.1: Empirical evidence for the convergence of $H_D^{(m)}$ to a limiting value as $m \to \infty$, using 10000 simulations of major outbreaks with $(\lambda_G, \lambda_L, \gamma) = (2, 0.25, 1)$ and an equal number of households of size 1 and size 2. The sample mean ($\bar{H}_D^{(m)}$) and sample standard deviation ($\hat{\sigma}^{(m)}$) of $H_D^{(m)}$ are given for various total number of households (m). The mean appears to converge toward a fixed value, with the standard deviation appearing to decrease toward 0 as $m \to \infty$.

Recall from Section 2.3.2 that we calculate h_D^L by considering a sequence of deterministic epidemics in which the initial fraction of the population that is infected tends to 0. Further, we conjecture that $h_D^L(\boldsymbol{\varepsilon}^{(k)}) \to h_D^L$ as $k \to \infty$ for any sequence $(\boldsymbol{\varepsilon}^{(k)})$ satisfying $\sum_{(s,e,i,r)\in\mathscr{H}_+^{(n_{\max})}} \varepsilon^{(k)}_{s,e,i,r} \downarrow 0$ and $\sum_{(s,e,i,r)\in\mathscr{H}_{0\text{rec}}} \varepsilon^{(k)}_{s,e,i,r} \uparrow 1$ as $k \to \infty$. We give an example for an SIR model in Table 2.2.

	ε	10^{-2}	10^{-3}	10^{-4}	10^{-5}
Initially	Size 1	0.532274	0.532242	0.532238	0.532239
infected	Size 2	0.532032	0.532218	0.532237	0.532239
households	Sizes 1 & 2	0.532312	0.532246	0.532237	0.532239

Table 2.2: Empirical evidence of convergence of $h_D(\varepsilon)$ to h_D, using $(\lambda_G, \lambda_L, \gamma) = (2, 0.25, 1)$ and an equal number of households of size 1 and size 2, with an initial fraction ε of infected households, where $\varepsilon = \sum_{(s,e,i,r)\in\mathscr{H}_+^{(n_{\max})}} \varepsilon_{s,e,i,r}$. The different rows correspond to different choices of initial condition in terms of which household sizes contain initial infectives. In the first row, all initial infectives reside in size 1 households, and in the second row all initial infectives reside in size 2 households. In the third row, a fraction $\frac{\varepsilon}{2}$ households contain one initial infective and a fraction $\frac{\varepsilon}{2}$ households contain two initial infectives. As $\varepsilon \downarrow 0$, $h_D(\varepsilon)$ approaches a value consistent with Table 2.1. Smaller values of ε give the same value as $h_D(10^{-5})$ to this level of accuracy.

Tables 2.1 and 2.2 suggest that $\lim_{m\to\infty} H_D^{(m)}$ can be computed as the solution of the appropriate corresponding deterministic equations, along with the stopping condition $R_V(t) = 1$.

Figure 2.1 gives an example showing that, as m increases, the trajectories $\{S^{(m)}(t)\}$ and $\{R_V^{(m)}(t)\}$ become smoother, with a random time translation capturing the time it takes for the stochastic epidemic to take off. As a result, the trajectories of $R_V^{(m)}(t)$ and $1 - S^{(m)}(t)$ against t can, for large m, be thought of as random time shifts of the corresponding deterministic trajectories. The random time shift is absent, however, when considering $R_V^{(m)}(t)$ against $1 - S^{(m)}(t)$, since the time shifts of $R_V^{(m)}(t)$ and $1 - S^{(m)}(t)$ are the same for a given realisation of the process. In light of the definition above of $H_D^{(m)}$, we thus find, for large m, that $H_D^{(m)} \simeq \lim_{\varepsilon\to 0} h_D(\varepsilon)$. Owing to the random time shift, however, it is not the case that $T_*^{(m)} \simeq t_*$ even for large m.

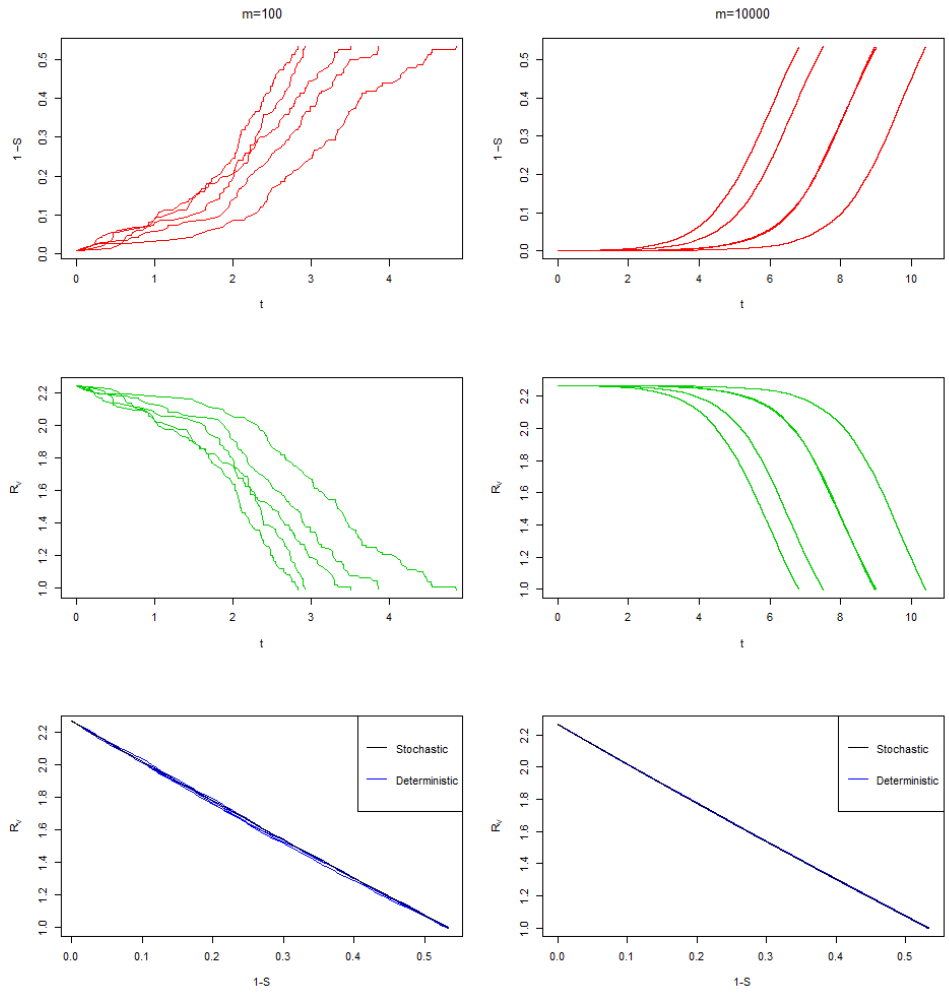

Figure 2.1: Realised trajectories of $R_V^{(m)}(t)$ and $1-S^{(m)}(t)$ for $m = 100$ (left column) and $m = 10000$ (right column), with $(\lambda_G, \lambda_L, \gamma) = (2, 0.25, 1)$ and an equal number of households of size 1 and size 2 with a single initial infective residing in a household of size 2. The top row plots the trajectories of $1-S^{(m)}(t)$ against t. The second row plots the trajectories of $R_V^{(m)}(t)$ against t. The final row plots the trajectories of $R_V^{(m)}(t)$ against $1-S^{(m)}(t)$, with the epidemic progressing from left to right and with the corresponding deterministic trajectory superimposed.

Approximate disease-induced herd immunity level $\tilde{h}_D$

Calculation of h_D requires deterministic limiting equations, which are not tractable in general; in the Markovian setting, for example, h_D can be found numerically. For other infectious period distributions, such a calculation is generally not available – such a calculation requires studying the trajectory of the epidemic in real time in order to calculate the time at which disease-induced herd immunity is achieved. We hence consider an approximation ($\tilde{h}_D$) to h_D. We

adapt the approach taken in Britton et al. [2020] to the households setting, using the final outcome of an epidemic with reduced global infection rate to approximate the state of the population at the time herd immunity is achieved. The method of approximation is as follows: Let $\kappa \in \left(R_*^{-1}, 1\right)$. Run to its conclusion an epidemic in which the global infection parameter λ_G is replaced by $\kappa\lambda_G$ and all other parameters are unchanged, i.e. an epidemic with prevention measure applied to global infection only. Then expose the population to a second epidemic with $\kappa = 1$, with members infected in the first epidemic now immune. The threshold parameter R_* for this second epidemic is a function of κ, which we denote by $\hat{R}_{DI}(\kappa)$. Determine $\hat{\kappa}$, the smallest value of κ such that $\hat{R}_{DI}(\kappa) \leq 1$ by solving $\hat{R}_{DI}(\hat{\kappa}) = 1$. Then $\tilde{h}_D$ is the fraction of the population infected in the first epidemic with $\kappa = \hat{\kappa}$. In summary, we adjust the global infection rate in the first epidemic to force criticality in the second, and then consider the final size of the first epidemic. Note that this method relies on final size results, which are more amenable to study than time-dependent results.

Accuracy of the approximation of h_D by $\tilde{h}_D$

The disparity between h_D and its approximation $\tilde{h}_D$ depends on the distribution of susceptibles inside households when herd immunity is achieved. When $\lambda_L = 0$, there is no within-household spread; this distribution is the same under h_D as under $\tilde{h}_D$. The same conclusion holds when $\lambda_L \to \infty$, since all single-household epidemics end immediately (everyone in a contacted household becomes infected as soon as that household is contacted). Thus, when $\lambda_L = 0$ or $\lambda_L = \infty$, we have $h_D = \tilde{h}_D$ and the approximation is exact, since the disparity between $\tilde{h}_D$ and h_D is driven by how local epidemics play out. For epidemics with $0 < \lambda_L < \infty$, local epidemics are run to termination under $\tilde{h}_D$, but not under h_D. This, coupled with a lower global infection rate under $\tilde{h}_D$, leads to $\tilde{h}_D$ being more strongly governed by within-household spread. A consequence of this is the distribution of susceptibles among households being more clumped under $\tilde{h}_D$ than h_D, leading to a larger proportion of households with no susceptible individuals than under h_D, which often results in both $\tilde{h}_D > h_D$ and the approximation becoming worse as $n_{\max}$ increases (cf. the discussion following

Theorem 2.1 in Section 2.4.2).

The above-mentioned clumping is illustrated in Figure 2.2, which considers the case of a common household size $n = 4$, with p_L varying in $[0,1]$ and λ_G being chosen so that $R_0 = 2$. When $p_L = 0$, there is no within-household spread and the distribution of the number of susceptibles in a typical household when herd immunity is reached is $\text{Bin}(4, 0.5)$ under both h_D and $\tilde{h}_D$. Note the agreement in the two distributions of susceptibles when $p_L = 1$. (When $p_L = 1$ households become fully infected upon being contacted globally; we need not use Poisson processes to model local infection in this case.) For $p_L \in (0,1)$, the distribution has greater mass at the extremes 0 and 4 under $\tilde{h}_D$ than h_D. We suspect that $\tilde{h}_D > h_D$ holds generally for a common household size $n > 1$ (with $0 < \lambda_L < \infty$) and give numerical examples supporting this claim in Section 2.5.2. It is possible (but atypical) for $\tilde{h}_D < h_D$ to occur, and the difference is small in the cases we have met. An example is a household structure comprised of households of size 1 and $n > 1$ only; see Figure 2.11 in Section 2.5.2.

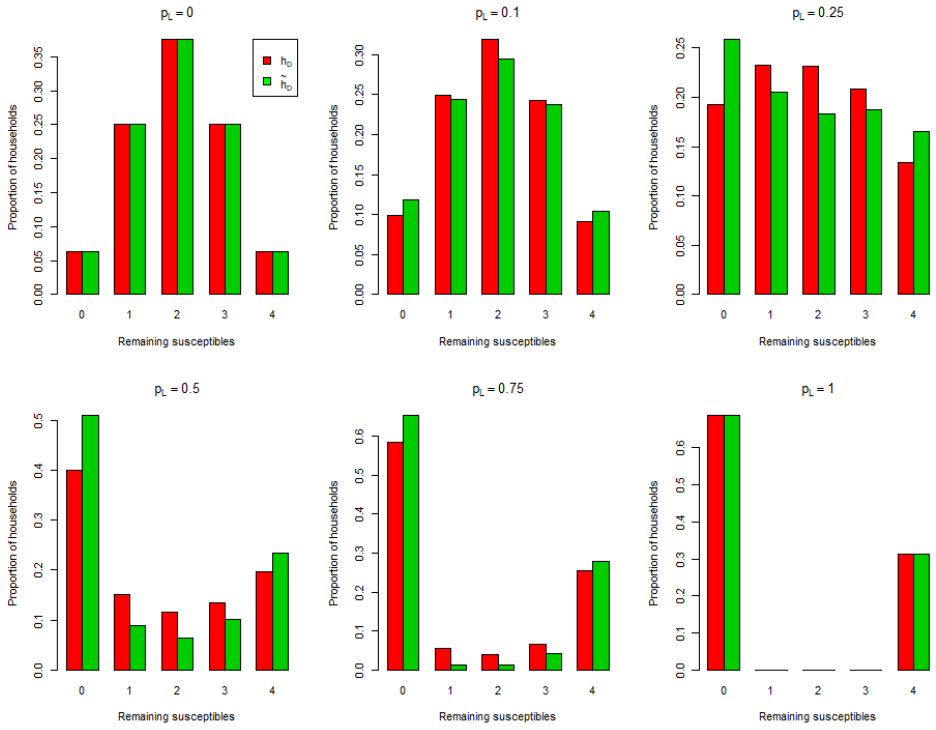

Figure 2.2: Distribution of the number of susceptibles in a typical household when herd immunity is achieved under h_D and $\tilde{h}_D$ when all households have size 4. For each p_L, λ_G is chosen so that $R_0 = 2$.

The accuracy of the approximation of h_D by $\tilde{h}_D$ is explored in Figure 2.3,

which shows heat maps of the percentage error $100\left|\tilde{h}_D - h_D\right|/h_D$ as a function of (p_L, R_0) for common household sizes $n = 2,3,4$ and 5. Observe that the percentage errors are all small, increase with n and are greatest for intermediate values of p_L. The maximum percentage error as (p_L, R_0) varies over $[0,1] \times (1,25]$ for each choice of n and for some other household size distributions are given in Table 2.3. Note that the value of p_L at which this maximum is attained tends to decrease with mean household size $\mu_H = \sum_{n=1}^{\infty} n\alpha_n$. This may be a consequence of the fact that for fixed p_L the fraction infected by a single-household epidemic increases with household size. The maximum percentage errors are small, except for countries with large mean household sizes. Moreover, these are maximum errors and even if they are not small, the error is small for many choices of parameter values, as illustrated by the $n = 5$ heat map in Figure 2.3. Thus, $\tilde{h}_D$ is generally a very good approximation of h_D.

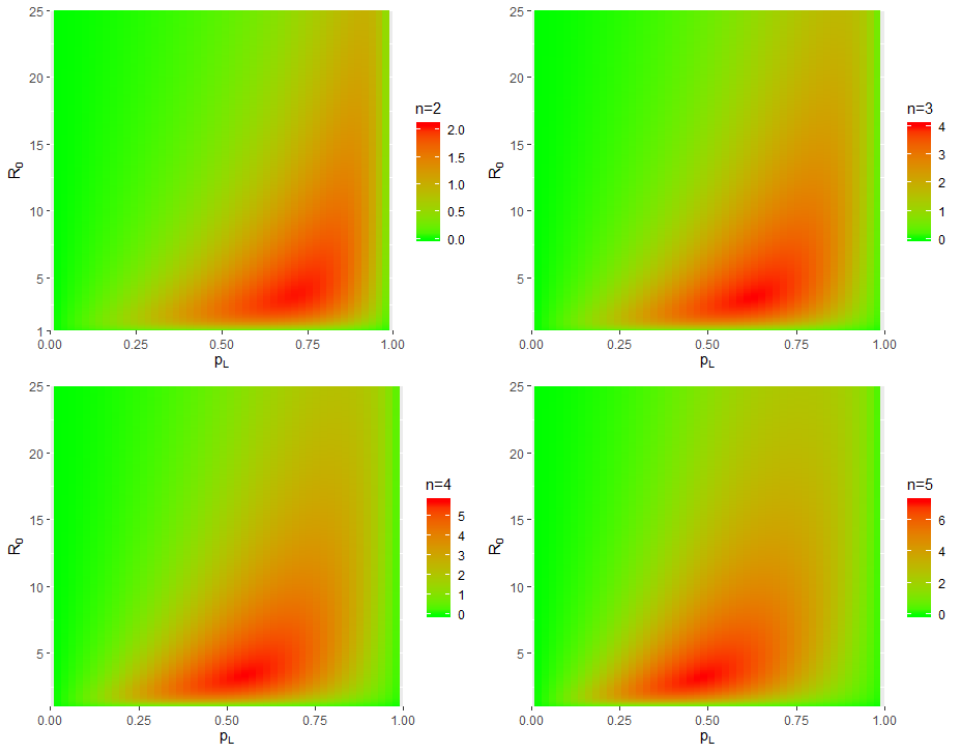

Figure 2.3: Heat maps of the percentage error $100\left|\tilde{h}_D - h_D\right|/h_D$ as a function of (p_L, R_0) for common household sizes $n = 2,3,4$ and 5.

Household	μ_H	Max error (%)	R_0	p_L	λ_G	λ_L
$n=2$	2	2.051	3.711	0.718	3.109	2.547
$n=3$	3	4.019	3.509	0.629	2.555	1.698
$n=4$	4	5.711	3.332	0.551	2.167	1.226
$n=5$	5	7.161	3.179	0.484	1.883	0.939
$\tilde{\alpha}_1=\tilde{\alpha}_4=0.5$	1.6	1.269	1.646	0.517	1.057	1.068
Sweden	2.0	1.859	2.135	0.566	1.418	1.306
UK	2.3	2.533	2.401	0.566	1.585	1.306
Argentina	3.3	4.236	2.479	0.495	1.386	0.979
Morocco	4.6	7.367	2.633	0.393	1.293	0.649
Chad	5.8	7.350	2.350	0.343	0.990	0.521
Pakistan	6.8	8.444	2.528	0.338	1.103	0.511

Table 2.3: The maximum percentage error for $\tilde{h}_D$ approximating h_D when $(p_L, R_0) \in [0,1] \times (1,25]$, together with the parameter values for which the maximum is attained. The first four rows correspond to a common household of size n. The fifth row corresponds to $\tilde{\alpha}_1 = \tilde{\alpha}_4 = 0.5$ and the remaining rows correspond to real-world household size distributions (see Section 2.5.3).

Overall, the approximation of h_D by $\tilde{h}_D$ is generally good. The approximation is exact in the limits $\lambda_L \to 0$ and $\lambda_L \to \infty$. We have observed that the approximation is typically worse when the mean household size μ_H increases, as well as for intermediate values of λ_L. The performance of the approximation is particularly poor in cases where the resulting household structures at the end of the first epidemic, under h_D and $\tilde{h}_D$ respectively, differ most. (Note the disparity between red and green bars in Figure 2.2, which illustrates an example in which $\tilde{h}_D$ and h_D differ.) Typically $\tilde{h}_D$ overestimates h_D, due to the inefficient effective household structures that result from the extra clumping of infectives under $\tilde{h}_D$. Such clumping arises since, under $\tilde{h}_D$, the epidemic is more strongly driven by local infection.

Impact of restrictions

Note that the parameter κ corresponds to restrictions being placed on the population which affect only the global infection rate; the severity of such restrictions increases as κ decreases. Since $\hat{\kappa}$ is chosen such that the second epidemic is at criticality, $\hat{\kappa}$ corresponds to the most severe restrictions that can be placed for the whole duration of the first epidemic, such that the second epidemic is not supercritical; more severe restrictions will leave the second epidemic supercritical, so

herd immunity will not be achieved. Recall that $\hat{h}_D$ denotes the disease-induced herd immunity level when restrictions are in place. Numerical investigations suggest that as κ increases from $\hat{\kappa}$ to 1, $\hat{h}_D$ decreases; see Figure 2.4. In this example, which uses the UK and Morocco household size distributions (see Section 2.5.3), such restrictions have only a small effect on the herd immunity level $\hat{h}_D$, with the effect being larger for Morocco. Moreover, when λ_L is fixed, we observe that $\hat{\kappa}$ decreases as R_0 increases. Repeating these calculations for other values of λ_L (not shown) reveals similar patterns and suggests that the effect is largest when λ_L is around 0.5. All of these observations regarding the UK and Morocco household size distribution are also seen with larger and more variable household size distributions: the effect of κ on $\hat{h}_D$ remains small but becomes slightly larger if the household size distribution is more variable, $\hat{\kappa}$ decreases with increasing R_0 and the effect of varying κ seems greatest when $\lambda_L \approx 0.5$.

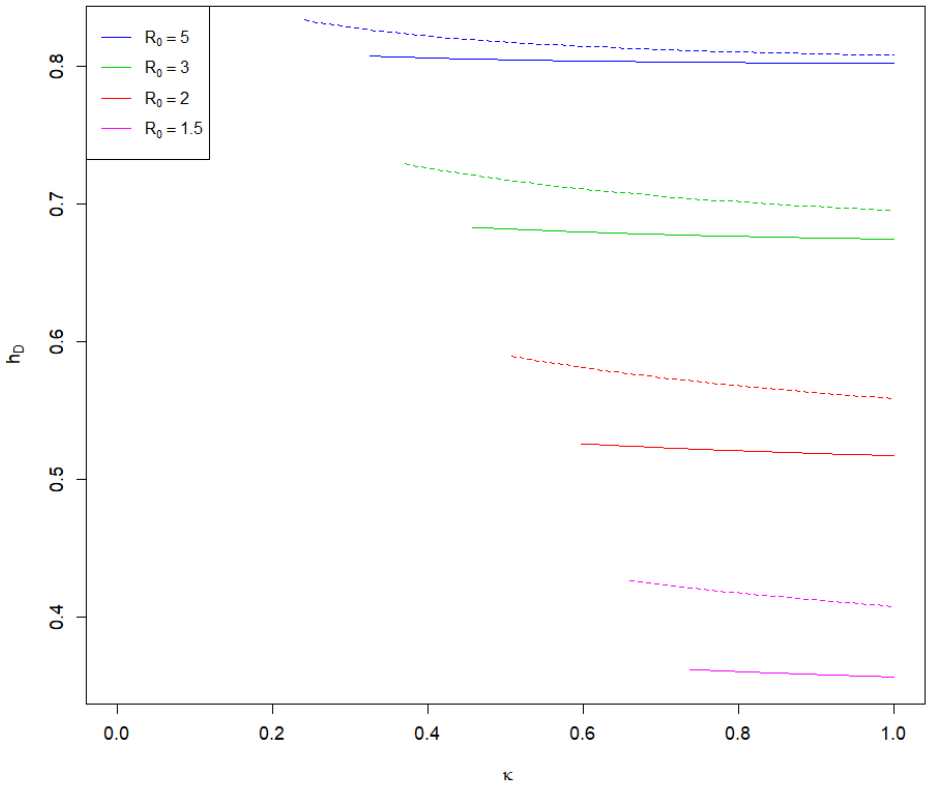

Figure 2.4: Values of $\hat{h}_D$ with global restrictions scaled by a factor κ for the duration of the first epidemic, using the UK household size distribution (solid lines) and Morocco's household size distribution (dashed lines), taking $\lambda_L = 0.5$ and considering several values of R_0.

2.4 Comparison of $\tilde{h}_D$ and h_C

2.4.1 Outline

This section presents results concerning orderings of $\tilde{h}_D$ and h_C. Since both of these quantities depend only on final outcome properties of the epidemic, they are invariant to the distribution of the latent period and we therefore take $T_E = 0$, corresponding to the SIR setting, in this section. The problem of solving for $\tilde{h}_D$ is not analytically tractable when $0 < \lambda_L < \infty$. We hence begin with the highly locally infectious case where $\lambda_L = \infty$, considered in Becker and Dietz [1995], for which a framework for comparison of $\tilde{h}_D$ and h_C is established and explicit progress is made. This is then applied to several household size distributions, beginning with all households being the same size, where $\tilde{h}_D > h_C$ is established (Theorem 2.1). Further, we show that for a common household size n, the maximum of $\tilde{h}_D - h_C$ as a function of λ_G occurs when $\lambda_G = \frac{4}{(1+n)\mathrm{E}[T_I]}$, corresponding to $R_0 = 2$ (Theorem 2.2). A necessary and sufficient condition for $\tilde{h}_D = h_C$ in the highly locally infectious case (Theorem 2.4) is also derived, leading to study of $\tilde{h}_D$ and h_C for epidemics just above criticality (Theorem 2.5, as well as highly locally and globally infectious epidemics (Theorem 2.7. We conclude the highly locally infectious case by deriving a result for household structures with only households of size 1 and $n > 1$ (Theorem 2.8). The weakly locally infectious case $\lambda_L \to 0$ is treated in Section 2.4.3 and a condition for $\tilde{h}_D > h_C$ is derived (Corollary 2.13).

We consider the general case $0 < \lambda_L < \infty$, both analytically and numerically, when all households are of the same size. In Section 2.4.4, we prove that $\tilde{h}_D > h_C$ for a common household size $n = 2$ and $n = 3$ (Theorem 2.15). We conjecture that $\tilde{h}_D > h_C$ holds for common household size $n \geq 4$; supporting evidence for this conjecture is provided in Section 2.5.2.

2.4.2 Highly locally infectious case

General framework

In the highly locally infectious case ($\lambda_L \to \infty$) explicit analytical progress is possible, as any infected individual will infect their whole household. We therefore

have $\mu_n(\lambda_L) = n$ for $n = 0, 1, \ldots$. Using (2.1), we find that $R_* = \lambda_G \mathrm{E}[T_I]\mu_{\tilde{H}}$, where $\mu_{\tilde{H}} = \mathrm{E}[\tilde{H}] = \sum_{n=1}^{\infty} n\tilde{\alpha}_n$ is the mean size of the household of an individual chosen uniformly at random from the population. Thus, $R_* > 1$ if and only if $\lambda_G > \frac{1}{\mu_{\tilde{H}}\mathrm{E}[T_I]}$. Substituting $\mu_n(\lambda_L) = n$ into (2.12) and solving $\hat{R}_U(c) = 1$ gives h_C as the unique solution of

$$\sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{v=0}^{n} \binom{n}{v} c^v (1-c)^{n-v} \frac{(n-v)^2}{n} \lambda_G \mathrm{E}[T_I] = 1. \tag{2.13}$$

The inner sum in (2.13) can be evaluated using the second moment of a $\mathrm{Bin}(n, 1-c)$ random variable. Using the definition of $\mu_{\tilde{H}}$, it follows that h_C is given by the unique solution in $(0,1)$ of the quadratic equation

$$h_C(1-h_C) + \mu_{\tilde{H}}(1-h_C)^2 - \frac{1}{\lambda_G \mathrm{E}[T_I]} = 0, \tag{2.14}$$

yielding

$$h_C = 1 - \frac{\sqrt{1 + \frac{4(\mu_{\tilde{H}}-1)}{\lambda_G \mathrm{E}[T_I]}} - 1}{2(\mu_{\tilde{H}} - 1)}. \tag{2.15}$$

As noted at the end of Section 2.3.1, $h_C = 1 - R_0^{-1}$ in the present highly locally infectious case.

Turning to the disease-induced herd immunity level $\tilde{h}_D$, consider the first epidemic with global infection rate $\hat{\kappa}\lambda_G$, where $\hat{\kappa}$ solves $\hat{R}_{DI}(\hat{\kappa}) = 1$ as described in Section 2.3.2. Let $z(\hat{\kappa})$ be the fraction of the population infected by that epidemic and $\pi = \exp(-\hat{\kappa}\lambda_G E[T_I] z(\hat{\kappa}))$, the probability that any given susceptible avoids global contact during that epidemic. For $n = 1, 2, \ldots$ and $v = 0, 1, \ldots, n$, let $x_{n,v}(\pi)$ be the proportion of households with n members which have v infected in this epidemic and thus immune to the second epidemic.

Letting $R_{DI}(\pi) = \hat{R}_{DI}(\hat{\kappa})$, we have

$$R_{DI}(\pi) = \sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{v=0}^{n} x_{n,v}(\pi) \frac{(n-v)^2}{n} \lambda_G \mathrm{E}[T_I]. \tag{2.16}$$

In the highly locally infectious case, a given individual escapes infection if and only if their whole household avoids global infection. Thus $x_{n,0}(\pi) = \pi^n$,

$x_{n,n}(\pi) = 1 - \pi^n$, and $x_{n,v}(\pi) = 0$ for $v \notin \{0, n\}$. Substitution into (2.16) yields

$$R_{DI}(\pi) = \sum_{n=1}^{\infty} n\tilde{\alpha}_n \pi^n \lambda_G \mathrm{E}[T_I]. \tag{2.17}$$

Note that $\tilde{\mu}_n(\infty, \pi) = n(1 - \pi^n)$, so equation (2.7) implies that the final proportion infected in the first epidemic is $\tilde{h}_D = z(\hat{\kappa}) = 1 - \sum_{n=1}^{\infty} \tilde{\alpha}_n \pi^n$. Thus,

$$\tilde{h}_D = 1 - f_{\tilde{H}}(\pi), \tag{2.18}$$

where $f_{\tilde{H}}(\pi) = \sum_{n=1}^{\infty} \tilde{\alpha}_n \pi^n$ is the probability-generating function of $\tilde{H}$. Setting $R_{DI}(\pi) = 1$ in (2.17) yields

$$\pi f'_{\tilde{H}}(\pi) = \frac{1}{\lambda_G \mathrm{E}[T_I]}. \tag{2.19}$$

Combined with (2.14), we have a framework to compare $\tilde{h}_D$ and h_C. Note, however, that the system given by (2.18) and (2.19) does not always allow closed-form calculation of $\tilde{h}_D$.

Note that in this subsection dealing with the highly local infectious case, the distribution of T_I only enters our results through its mean $E[T_I]$.

Common household size

Suppose that all households are of size n. When $n = 1$ the model reduces to the standard homogeneously mixing model, so $\tilde{h}_D = h_C = 1 - R_0^{-1}$. Therefore assume that $n > 1$. Using (2.15),

$$h_C = 1 - \frac{\sqrt{1 + \frac{4(n-1)}{\lambda_G \mathrm{E}[T_I]}} - 1}{2(n-1)}. \tag{2.20}$$

Note that $f_{\tilde{H}}(\pi) = \pi^n$, so (2.18) and (2.19) yield

$$\tilde{h}_D = 1 - \frac{1}{n\lambda_G \mathrm{E}[T_I]}. \tag{2.21}$$

Theorem 2.1. *Consider the highly locally infectious case with common household size $n > 1$. Then $\tilde{h}_D > h_C$ if $R_* > 1$.*

Proof. The claim is established by subtracting equation (2.20) from (2.21), and letting $x = \frac{1}{\lambda_G \mathrm{E}[T_I]}$ for ease of exposition, so $R_* = n\lambda_G \mathrm{E}[T_I] = \frac{n}{x}$. Expressing explicitly the dependence of $\tilde{h}_D$ and h_C on x, we obtain

$$\tilde{h}_D(x) - h_C(x) = \frac{\sqrt{1+4x(n-1)}-1}{2(n-1)} - \frac{x}{n} = \frac{n\sqrt{1+4x(n-1)}-n-2x(n-1)}{2n(n-1)}. \tag{2.22}$$

The result then follows by elementary manipulations of (2.22), since $R_* > 1$ implies $x \in (0,n)$. □

A heuristic justification for Theorem 2.1 is as follows. In disease-induced herd immunity, after the first epidemic, households contain either 0 or n susceptibles, depending on whether that household was infected. Consider a randomly chosen individual contacted globally in the second epidemic. If this individual is immune, this contact contributes no further infection. Otherwise, they begin an epidemic within their household which, in the highly locally infectious case, will infect all non-immune members. Hence, under disease-induced herd immunity, the potential for within-spread is as high as possible (the rest of the household is susceptible). Thus disease-induced herd immunity corresponds to the worst possible vaccination strategy for a given coverage, resulting in $\tilde{h}_D > h_C$. Note that, in this case, disease-induced herd immunity is equivalent to vaccination of whole households, which corresponds to the worst possible vaccination strategy.

A further result provides a link between the highly locally infectious case and R_0 for the households model, in which we treat $\tilde{h}_D - h_C$ as a function of λ_G.

Theorem 2.2. *Under the same assumptions as Theorem 2.1, $\tilde{h}_D - h_C$ has a unique maximum as a function of λ_G which is attained when $\lambda_G = \frac{4}{(1+n)\mathrm{E}[T_I]}$, corresponding to $R_0 = 2$.*

Proof. We show that $\tilde{h}_D(x) - h_C(x)$ has a unique stationary point, which must be a maximum since, from (2.22), $\tilde{h}_D(x) - h_C(x) \to 0$ as $x \downarrow 0$ and $x \uparrow n$, corresponding to $R_* \to \infty$ and $R_* \to 1$, respectively, and by Theorem 2.1, $\tilde{h}_D(x) - h_C(x) > 0$ for $x \in (0,n)$. Then we find the value of x which yields the maximum and compute the corresponding R_0 value.

Ignoring the denominator in (2.22), differentiation with respect to x and

equating to 0 leads us to solve

$$\frac{4n(n-1)}{2\sqrt{1+4x(n-1)}} - 2(n-1) = 0 \Longleftrightarrow \hat{x} = \frac{n+1}{4} \Longleftrightarrow \hat{\lambda}_G = \frac{4}{(1+n)\mathrm{E}[T_I]}.$$

In the highly locally infectious case, all secondary infections in a household are attributed to the primary case, so $\mu_0^{(k)} = 1$, $\mu_1^{(k)} = k-1$ and $\mu_j^{(k)} = 0$ for $j \notin \{0,1\}$. Setting $\lambda_G = \hat{\lambda}_G$ in (2.3) gives that R_0 is the unique positive root of $g_n(\lambda) = 0$, where

$$g_n(\lambda) = 1 - \frac{4}{1+n}\left(\frac{1}{\lambda} + \frac{n-1}{\lambda^2}\right).$$

Now $g_n(2) = 0$, so the maximum of $\tilde{h}_D - h_C$ is attained when $R_0 = 2$, as claimed. □

Intuitively, we would expect that for calibrated models (i.e. models with the same R_0) increasing the household size would increase the disease-induced herd immunity level. We consider this in the following result.

Theorem 2.3. *Under the same assumptions as Theorem 2.1, let $\tilde{h}_D^{(n)}$ denote the disease-induced herd immunity level, supposing that $R_0 > 1$ is held fixed. Then $\tilde{h}_D^{(n)}$ is increasing with n. Moreover, we have $\lim_{n\to\infty} h_D^{(n)} = 1 - R_0^{-2}$.*

Proof. Using the definition of R_0 in the highly locally infectious case in (2.3), we have

$$\lambda_G^{(n)} = \frac{R_0^2}{R_0 + n - 1}.$$

Then, since $\tilde{h}_D^{(n)} = 1 - \dfrac{1}{n\lambda_G^{(n)}}$, we have

$$\tilde{h}_D^{(n+1)} - \tilde{h}_D^{(n)} = \frac{R_0 - 1}{n(n+1)R_0^2} > 0,$$

which establishes the result. Direct computation yields $\lim_{n\to\infty} \tilde{h}_D^{(n)} = 1 - R_0^{-2}$, so that $\tilde{h}_D^{(n)} \in [1 - R_0^{-1}, 1 - R_0^{-2})$ for all $n \geq 1$. □

Necessary and sufficient condition for $\tilde{h}_D = h_C$ for all λ_G

The framework given in Section 2.4.2 for the highly locally infectious case enables proof of the following result. For $\theta \in (0,1]$, we write $\tilde{H} \sim \mathrm{Geom}(\theta)$ if $\tilde{H}$

has a geometric distribution with probability mass function

$$\mathrm{P}(\tilde{H} = x) = \theta(1-\theta)^{x-1}, \quad x = 1, 2 \ldots.$$

Theorem 2.4. *In the highly locally infectious case, $\tilde{h}_D = h_C$ for all λ_G such that $R_* > 1$ if and only if $\tilde{H} \sim \mathrm{Geom}(\mu_{\tilde{H}}^{-1})$.*

Proof. Recall that $R_* > 1$ if and only if $\lambda_G > \frac{1}{\mu_{\tilde{H}}\mathrm{E}[T_I]}$. We begin by assuming $\tilde{h}_D - h_C = 0$ for all $\lambda_G > \frac{1}{\mu_{\tilde{H}}\mathrm{E}[T_I]}$ and solving for $\tilde{H}$. Equations (2.14) and (2.18) give

$$f_{\tilde{H}}(\pi)(1 - f_{\tilde{H}}(\pi)) + \mu_{\tilde{H}}(f_{\tilde{H}}(\pi))^2 = \frac{1}{\lambda_G \mathrm{E}[T_I]}.$$

Equation (2.19) implies that

$$G(\pi) := f_{\tilde{H}}(\pi)(1 - f_{\tilde{H}}(\pi)) + \mu_{\tilde{H}}(f_{\tilde{H}}(\pi))^2 - \pi f'_{\tilde{H}}(\pi) = 0, \quad \pi \in [0, 1). \quad (2.23)$$

Further, $f_{\tilde{H}}(1) = 1$ since $f_{\tilde{H}}(\pi)$ is a probability-generating function. This separable ODE can be solved to yield, for $0 \leq \pi \leq 1$,

$$f_{\tilde{H}}(\pi) = \frac{\pi \mu_{\tilde{H}}^{-1}}{1 - \pi\left(1 - \mu_{\tilde{H}}^{-1}\right)}, \quad (2.24)$$

which is precisely the probability-generating function of a $\mathrm{Geom}(\mu_{\tilde{H}}^{-1})$ random variable. This establishes the only if part of the equivalence claim. For the converse, assume that $\tilde{H}$ follows a geometric distribution with parameter $\mu_{\tilde{H}}$. Then (2.23) holds and the logic for the above proof is reversible thereafter, so the result follows. □

If $\tilde{H} \sim \mathrm{Geom}(\theta)$, then H follows a logarithmic distribution having probability mass function

$$P(H = k) = \frac{-1}{\log \theta} \frac{(1-\theta)^k}{k}, \quad k = 1, 2, \ldots.$$

Note that real-life household size distributions will have a finite maximum size, so for any realistic household size distribution $\tilde{h}_D = h_C$ will not hold for all λ_G in the highly locally infectious case. We typically observe $\tilde{h}_D > h_C$ for real-life

household size distributions and comment upon this further in Section 2.5.2.

Just supercritical epidemics

We can use the framework provided in Section 2.4.2 to give an ordering of $\tilde{h}_D$ and h_C for epidemics which are just above threshold, i.e. when R_* is just above 1, so π is just below 1. We hence establish an ordering of $\tilde{h}_D$ and h_C by considering $G(\pi)$, given in (2.23), in the neighbourhood of $\pi = 1$. Assuming a fraction $z(\pi)$ of individuals are infected in the first epidemic gives a threshold parameter $R_{DI}(\pi)$ for the second epidemic, as given in (2.16). Vaccinating the same proportion uniformly at random gives a threshold parameter $R_U(\pi) = \hat{R}_U(z(\pi))$. Here, $R_U(\pi) - R_{DI}(\pi) = \lambda_G \mathrm{E}[T_I] G(\pi)$. Clearly we have $G(1) = 0$, since $f_{\tilde{H}}(1) = 1$ and $f'_{\tilde{H}}(1) = \mu_{\tilde{H}}$. Let $G^{(k)}$ be the k^{th} derivative of G and define

$$k^* = \inf_{k \geq 1} \{k : G^{(k)}(1) \neq 0\}. \tag{2.25}$$

Suppose that $G^{(k^*)}(1) > 0$. Then $G(\pi) < 0$ for π just below 1, so $R_U(\pi) < R_{DI}(\pi)$ for such π. Hence, $R_U(\pi) < 1$ if $R_{DI}(\pi) = 1$ and it follows that $\tilde{h}_D > h_C$ for epidemics which are just above threshold. A similar argument shows that $\tilde{h}_D < h_C$ if $G^{(k^*)}(1) < 0$. If $\tilde{H}$ follows a geometric distribution then $G^{(k)}(1) = 0$ for all $k \geq 1$, otherwise $\tilde{h}_D \neq h_C$. Determining the ordering of $\tilde{h}_D$ and h_C reduces to comparing factorial moments of $\tilde{H}$ to those of a geometric distribution with parameter $\mu_{\tilde{H}}^{-1}$. For a random variable $\tilde{H}$ define, for $i = 1, 2, \ldots$, the factorial moments $\mu_{\tilde{H}}^{[i]} = \mathrm{E}[\tilde{H}(\tilde{H}-1)\ldots(\tilde{H}-i+1)]$, with $\mu_{\tilde{H}}^{[0]} = 1$. Note that $\mu_{\tilde{H}}^{[1]} = \mu_{\tilde{H}}$.

Theorem 2.5. *Let $\tilde{H}$ be a given size-biased household size distribution with mean $\mu_{\tilde{H}}$ and factorial moments $\mu_{\tilde{H}}^{[i]}$ $(i = 0, 1, \ldots)$. Suppose that $l^* = \inf_{k \geq 2}\{k : \mu_{\tilde{H}}^{[k]} \neq k! \mu_{\tilde{H}} (\mu_{\tilde{H}} - 1)^{k-1}\} < \infty$. Then $\tilde{h}_D > h_C$ for highly locally infectious epidemics which are just above threshold if $\mu_{\tilde{H}}^{[l^*]} < l^*! \mu_{\tilde{H}} (\mu_{\tilde{H}} - 1)^{l^*-1}$, otherwise $\tilde{h}_D < h_C$ for such epidemics.*

Proof. For $i = 0, 1, 2 \ldots$, let $\hat{\mu}_{\tilde{H}}^{[i]}$ be the i^{th} factorial moment of $\tilde{H}$ when $\tilde{H} \sim \mathrm{Geom}(\mu_{\tilde{H}}^{-1})$. Then $\hat{\mu}_{\tilde{H}}^{[0]} = 1$ and

$$\hat{\mu}_{\tilde{H}}^{[n]} = n! \mu_{\tilde{H}} (\mu_{\tilde{H}} - 1)^{n-1}, \quad n = 1, 2, \ldots.$$

We compute $G^{(k)}(1)$ using the general Leibniz rule. For $k = 1, 2, \ldots$ we have

$$G^{(k)}(\pi) = (1-k) f_{\tilde{H}}^{(k)}(\pi) + (\mu_{\tilde{H}} - 1) \sum_{i=0}^{k} \binom{k}{i} f_{\tilde{H}}^{(i)}(\pi) f_{\tilde{H}}^{(k-i)}(\pi) - \pi f_{\tilde{H}}^{(k+1)}(\pi), \tag{2.26}$$

leading to

$$G^{(k)}(1) = (1-k)\mu_{\tilde{H}}^{[k]} + (\mu_{\tilde{H}} - 1) \sum_{i=0}^{k} \binom{k}{i} \mu_{\tilde{H}}^{[k-i]} \mu_{\tilde{H}}^{[i]} - \mu_{\tilde{H}}^{[k+1]}. \tag{2.27}$$

Note that $G^{(k)}(1) \equiv \delta_n - \mu_{\tilde{H}}^{[k+1]}$, where δ_k depends only on $\mu_{\tilde{H}}^{[0]}, \mu_{\tilde{H}}^{[1]}, \ldots, \mu_{\tilde{H}}^{[k]}$. Recall that $G^{(k)}(1) = 0$ for all $k = 0, 1, \ldots$ when $\tilde{H} \sim \text{Geom}(\mu_{\tilde{H}}^{-1})$. Now suppose that $l^* < \infty$ and $\mu_{\tilde{H}}^{[l^*]} < l^*! \mu_{\tilde{H}} (\mu_{\tilde{H}} - 1)^{l^*-1}$. Then $G^{(k)}(1) = 0$ for $k = 0, 1, \ldots l^* - 1$, since $\mu_{\tilde{H}}^{[k]} = \hat{\mu}_{\tilde{H}}^{[k]}$ for $k = 0, 1, \ldots, l^* - 1$, and $G^{(l^*)}(1) > 0$ since $\mu_{\tilde{H}}^{[l^*]} < \hat{\mu}_{\tilde{H}}^{[l^*]}$. Hence, $\tilde{h}_D > h_C$ by the observation following (2.25). A similar argument holds when $\mu_{\tilde{H}}^{[l^*]} > l^*! \mu_{\tilde{H}} (\mu_{\tilde{H}} - 1)^{l^*-1}$. □

In many cases (i.e. when the second factorial moment of $\tilde{H}$ differs from that of a geometric distribution with parameter $\mu_{\tilde{H}}^{-1}$) only the first derivative of G is required. The following corollary, which involves only the mean and variance of $\tilde{H}$, is an immediate consequence of Theorem 2.5 in the case $l^* = 2$.

Corollary 2.6. *If* $\text{var}(\tilde{H}) < \text{E}[\tilde{H}]\text{E}[\tilde{H} - 1]$ *then* $\tilde{h}_D > h_C$ *for highly locally infectious epidemics which are just above threshold. If* $\text{var}(\tilde{H}) > \text{E}[\tilde{H}]\text{E}[\tilde{H} - 1]$ *then* $\tilde{h}_D < h_C$ *for highly locally infectious epidemics which are just above threshold.*

Highly locally and globally infectious epidemics

The framework in Section 2.4.2 can also be used to consider highly locally and highly globally infectious epidemics. This corresponds to the case where π, the global escape probability, is small. Considering $\pi \downarrow 0$ yields the following theorem. (Recall that, for $n = 1, 2, \ldots$, $\tilde{\alpha}_n = \text{P}(\tilde{H} = n)$.)

Theorem 2.7. *Suppose that* $n^* = \inf_{n \geq 2}\{n : G^{(n)}(0) \neq 0\} < \infty$. *Then* $\tilde{h}_D > h_C$ *for all sufficiently small* $\pi > 0$ *if* $\tilde{\alpha}_{n^*} < \tilde{\alpha}_1^{n^*} (\mu_{\tilde{H}} - 1)^{n^*-1}$, *otherwise* $\tilde{h}_D < h_C$ *for such* π.

Proof. Note that, for $i = 0, 1, \ldots$, we have $f_{\tilde{H}}^{(i)}(0) = i!\tilde{\alpha}_i$, with $\tilde{\alpha}_0 = 0$. Substituting $\pi = 0$ into (2.26) yields, after elementary algebra, that $G^{(1)}(0) = 0$ and, for $n \geq 2$,

$$G^{(n)}(0) = n! \left((1-n)\tilde{\alpha}_n + (\mu_{\tilde{H}} - 1) \sum_{k=1}^{n-1} \tilde{\alpha}_{n-k}\tilde{\alpha}_k \right). \tag{2.28}$$

Suppose that $G^{(n)}(0) = 0$ for $n = 1, 2, \ldots$. Iterating (2.28) gives $\tilde{\alpha}_n = \tilde{\alpha}_1^n(\mu_{\tilde{H}} - 1)^{n-1}$. Then $\tilde{\alpha}_1 = \mu_{\tilde{H}}^{-1}$ (since $\sum_{n=1}^{\infty} \tilde{\alpha}_n = 1$), so $\tilde{H} \sim \text{Geom}(\mu_{\tilde{H}}^{-1})$. With n^* as in the statement of the theorem, we have that $G^{(n^*)}(0) > 0$ implies $\tilde{h}_D < h_C$, and $G^{(n^*)}(0) < 0$ implies $\tilde{h}_D > h_C$, from which the result follows.

□

Similarly to the just supercritical case, the only distribution for $\tilde{H}$ which has $G^{(n)}(0) = 0$ for all n is the geometric distribution with parameter $\mu_{\tilde{H}}^{-1}$. Theorem 2.7 then reduces the ordering of $\tilde{h}_D$ and h_C to iterative comparison of the probability mass function of $\tilde{H}$ with the relevant geometric distribution.

Households of size 1 and $n > 1$

Theorem 2.1 in Section 2.4.2 shows that, in the highly locally infectious case, $\tilde{h}_D > h_C$ for all λ_G when the households all have the same size. We now consider the simplest setting when there is variability in household size, i.e. the case where there are only two household sizes, 1 and $n > 1$. For $0 < p < 1$, let p denote the proportion of individuals who belong to a household of size n. Thus $\tilde{\alpha}_n = p$ and $\tilde{\alpha}_1 = 1 - p$. We consider how $\tilde{h}_D - h_C$ varies with p, with a view towards obtaining different orderings of $\tilde{h}_D$ and h_C as the household structure changes. The following theorem, proved in Section 2.7.1, shows that the ordering of $\tilde{h}_D$ and h_C is less straightforward when household size is variable.

Theorem 2.8. *Suppose that $\tilde{\alpha}_n = p = 1 - \tilde{\alpha}_1$, where $n > 1$ and $0 < p < 1$, and $R_* > 1$. If $n = 2$, then $\tilde{h}_D > h_C$ for all p. For $n \geq 3$, if $p \leq \frac{n-2}{2(n-1)}$ then $\tilde{h}_D < h_C$ for all λ_G. If $p > \frac{n-2}{2(n-1)}$ then there exists $\lambda_G^*(n,p)$ such that $\tilde{h}_D > h_C$ for $\lambda_G < \lambda_G^*(n,p)$ and $\tilde{h}_D < h_C$ for $\lambda_G > \lambda_G^*(n,p)$. Further, $\lambda_G^*(n,p) = \left[\mathrm{E}[T_I] \hat{\pi}_n(p) \left(1 - p + np\hat{\pi}_n(p)^{n-1} \right) \right]^{-1}$, where $\hat{\pi}_n(p)$ is the unique root in $(0,1)$*

of

$$\pi^{\frac{n}{2}-1} - p\pi^{n-1} - (1-p) = 0. \tag{2.29}$$

One can solve (2.29) for $\hat{\pi}$ numerically and thereby determine $\lambda_G^*(n,p)$, and the corresponding value $R_0^*(n,p)$ of R_0 (which is independent of $E[T_I]$), such that the change in the ordering of $\tilde{h}_D$ and h_C occurs. Figure 2.5 shows $R_0^*(n,p)$ as a function of p for various n, with $\tilde{h}_D < h_C$ above the plotted line and $\tilde{h}_D > h_C$ below it. If $p \leq (n-2)/2(n-1)$ then no change of sign occurs and $\tilde{h}_D > h_C$ for all values of $R_0 > 1$.

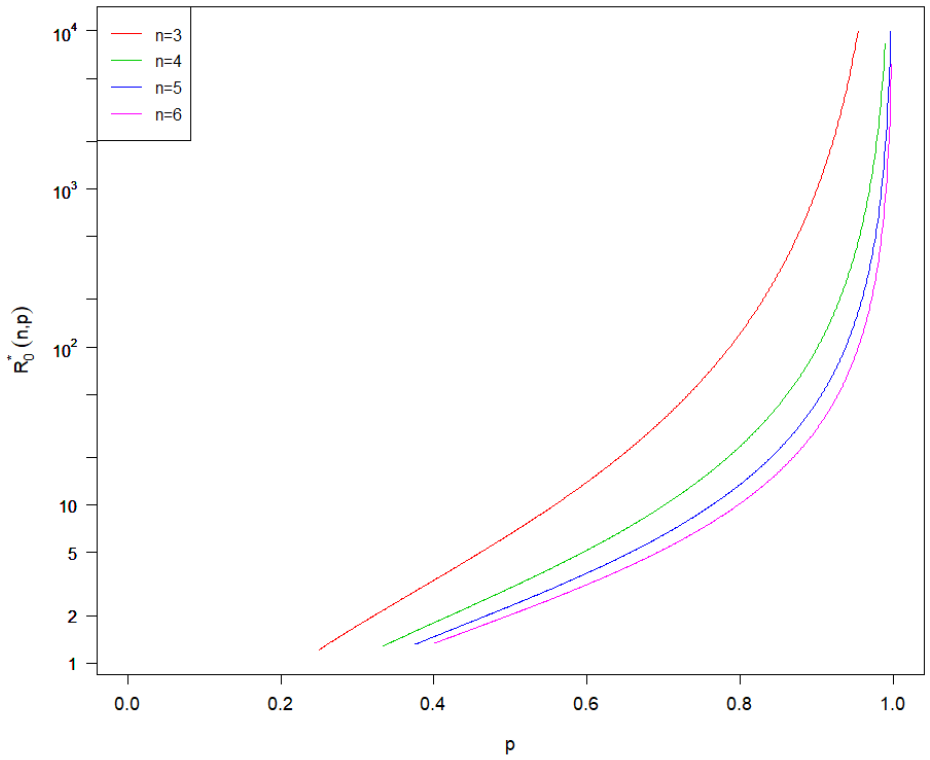

Figure 2.5: The value of $R_0^*(n,p)$ (on a logarithmic scale) as a function of p for $n \in \{3,4,5,6\}$.

We see immediately that $R_0^*(n,p)$ decreases with n and increases with p. One can also show that, for fixed n, $\lim_{p\uparrow 1} R_0^*(n,p) = \infty$. Further, $\mathrm{var}(\tilde{H}) = (n-1)^2 p(1-p)$, so the variability in household size is small when p is close to one. Thus Theorem 2.8 shows that, even with low variability in household size, we can have $\tilde{h}_D < h_C$; however, in this example, R_0 has to be unrealistically large for this to happen.

False infectives model

Consider the households epidemic model, with common household size $n > 1$ in the highly locally infectious case, adapted as follows. Suppose that individuals, upon being infected, have infectious period T_I satisfying $\mathrm{P}(T_I = 0) = 1 - \mathrm{P}(T_I = 1) = p$. Thus an infected individual is a "false infective" with probability $p \in (0,1)$. Denote this epidemic model by $\mathscr{E}_n(p)$. The mean size of a single-household outbreak in this model is given by $\mu_n(p) = p + n(1-p)$, since an individual is either a false infective (with probability p) or infects all n members of their household (with probability $1-p$). We first consider the final size of $\mathscr{E}_n(p)$, beginning with an elementary lemma.

Lemma 2.9. *Suppose $n > 1$. For $\pi, p \in (0,1)$, let $x = \pi + p(1-\pi)$. For $i \in \{0,1,2\}$, let*

$$a_i(p) = \sum_{v=0}^{n} \binom{n}{v} p^v (1-\pi)^v \pi^{n-v} v^i.$$

Then

$$\begin{aligned} a_0(p) &= x^n, \\ a_1(p) &= np(1-\pi)x^{n-1} \text{ and} \\ a_2(p) &= np(1-\pi)(np(1-\pi)+\pi)x^{n-2}. \end{aligned}$$

Proof. Consider the random variable $X \sim \mathrm{Bin}(n, 1-\pi)$, which has probability-generating function

$$\phi_X(p) = \mathrm{E}[p^X] = \sum_{v=0}^{n} \binom{n}{v} p^v (1-\pi)^v \pi^{n-v} = x^n = a_0(p), \quad 0 < p < 1.$$

Differentiation of $\phi_X(p)$ shows that $a_1(p) = pa_0'(p)$ and that $a_2(p) = p^2 a_0''(p) + a_1(p)$. The result follows by elementary manipulations of $a_i(p)$ ($i \in \{0,1,2\}$). □

We next consider the final size of the false infectives model under the assumption that a major outbreak occurs.

Lemma 2.10. *Assume a major outbreak occurs in the false infectives model $\mathscr{E}_n(p)$, where $p \in (0,1)$. Let π denote the probability an individual avoids global*

infection. The final size $z(p)$ of $\mathscr{E}_n(p)$ is given by

$$z(p) = 1 - \pi(\pi + p(1-\pi))^{n-1}.$$

Proof. We note that, in the notation of Section 2.2.2, we have $\lim_{\lambda_L \to \infty} \phi(\lambda_L) = p$ and $\lim_{\lambda_L \to \infty} \beta_i(\infty) = i(1-p)^{i-1}$ for $i = 0, 1, \ldots$. Then, using (2.7),

$$\begin{aligned} z(p) =& 1 - \frac{1}{n} \sum_{i=1}^{n} \binom{n}{i} (1-p)^{i-1} p^{n-i} \pi^i i \\ =& 1 - \frac{1}{n(1-p)} \sum_{i=1}^{n} \binom{n}{i} (1-p)^{i} p^{n-i} \pi^i i \\ =& 1 - \frac{\pi}{n(1-p)} \frac{d}{d\pi} \sum_{i=1}^{n} \binom{n}{i} (1-p)^{i} p^{n-i} \pi^i \\ =& 1 - \pi(\pi + p(1-\pi))^{n-1}, \end{aligned}$$

where the final line follows by applying Lemma 2.9 with p and π interchanged. □

We conclude this section by proving an ordering for $\tilde{h}_D$ and $\tilde{h}_C$ in the false infectives model.

Theorem 2.11. *Let $R_* > 1$ in the false infectives model. We have $\tilde{h}_D > h_C$.*

Proof. Let $p \in (0,1)$ be given. We proceed by comparison of reproduction numbers. If a fraction $c \in (0,1)$ of individuals are vaccinated uniformly at random with a perfect vaccine prior to the epidemic, the proportion of households with v members vaccinated $(P_U^{(v)}(c), v = 0, 1, \ldots)$ satisfies

$$P_U^{(v)}(c) = \binom{n}{v} c^v (1-c)^{n-v}.$$

Conversely, if a fraction z are infected in the false infectives model with global infection rate $\kappa\lambda_G$ and corresponding global escape probability $\pi(z)$, the proportion of households with v members vaccinated $(P_D^{(v)}, v = 0, 1, \ldots)$ is given by

$$P_D^{(v)}(z) = \binom{n}{v} p^v (1 - \pi(z))^v \pi(z)^{n-v}.$$

Hereafter we suppress the dependence of π on z. The corresponding threshold parameters are then given by

$$R_U(c) = \lambda_G \mathrm{E}[T_I] \sum_{v=0}^{n} P_U^{(v)}(c) \left(1 - \frac{v}{n}\right) \mu_{n-v}(p) \tag{2.30}$$

and

$$R_D(z) = \lambda_G \mathrm{E}[T_I] \sum_{v=0}^{n} P_D^{(v)}(z) \left(1 - \frac{v}{n}\right) \mu_{n-v}(p). \tag{2.31}$$

We assume that $\lambda_G \mathrm{E}[T_I] = 1$ without loss of generality. Expanding (2.30), we find that

$$R_U(c) = c^2(n-1)(1-p) + c[(1-2n)(1-p) - p] + n(1-p) + p. \tag{2.32}$$

Suppose now that $\pi \in (0,1)$ is chosen such that $z(\pi) = c$, so that both strategies leave the same proportion of the population immune. We consider the reproduction numbers as functions of π, which we denote by $\tilde{R}_U(\pi)$ and $\tilde{R}_D(\pi)$ respectively. Using Lemma 2.10 and substituting $z(\pi) = c$ into (2.32), we find

$$\tilde{R}_U(\pi) = (n-1)(1-p)\pi^2(\pi + p(1-\pi))^{2n-2} + \pi(\pi + p(1-\pi))^{n-1}. \tag{2.33}$$

Turning to $\tilde{R}_D(\pi)$, we recall the definition of $a_i(p)$ ($i \in \{0,1,2\}$) in Lemma 2.9. Then, after elementary manipulation of (2.31), we have

$$\begin{aligned}
\tilde{R}_D(\pi) &= \sum_{v=0}^{n} \binom{n}{v} p^v (1-\pi)^v \pi^{n-v} \left(1 - \frac{v}{n}\right) (p + (n-v)(1-p)) \\
&= \left(\frac{1-p}{n}\right) a_2(p) + \left(2p - 2 - \frac{p}{n}\right) a_1(p) + (n + p - np) a_0(p) \\
&= \pi(n\pi + p(1 - n\pi))[\pi + p(1-\pi)]^{n-2}.
\end{aligned} \tag{2.34}$$

Let $x = \pi + p(1-\pi)$ and note that $0 < x < 1$. Subtracting (2.33) from (2.34), we find that

$$\tilde{R}_D(\pi) - \tilde{R}_U(\pi) = (n-1)(1-p)\pi^2 x^{n-2}(1-x^n) > 0,$$

which establishes that $\tilde{h}_D > h_C$. □

2.4.3 Weakly locally infectious case

Consider now the case when the extra local infection is small but non-zero, corresponding to $\lambda_L \to 0$, with $R_* > 1$, $\mathrm{E}[T_I] = 1$ and $\mathrm{var}(T_I) < \infty$. Assume also that $n_{\max} < \infty$.

Theorem 2.12. *Let* $\pi(0) = \frac{1}{\lambda_G}$. *We have*

$$\tilde{h}_D(\lambda_L) - h_C(\lambda_L) = 2\lambda_L^2 \pi(0)^2 (1 - \pi(0)) \left[\mathrm{E}[\tilde{H} - 1] - \mathrm{var}(\tilde{H})\right] + o(\lambda_L^2).$$

The proof of Theorem 2.12 involves computing the first three terms of the Maclaurin expansion of $\tilde{h}_D(\lambda_L) - h_C(\lambda_L)$ and is given in Section 2.7.2, where the assumption that $n_{\max} < \infty$ is also explained. The assumption that $\mathrm{E}[T_I] = 1$ involves no loss of generality (since time can be rescaled appropriately) and is made to simplify the presentation of the proof. The assumption $\mathrm{var}(T_I) < \infty$ is required for the third term in the above-mentioned Maclaurin expansion. Note that when $\lambda_L = 0$, $R_* = R_0 = \lambda_G$, whence $h_C = \tilde{h}_D = 1 - \pi(0)$. The following corollary is an immediate consequence of Theorem 2.12.

Corollary 2.13. *If* $\mathrm{var}(\tilde{H}) < \mathrm{E}[\tilde{H} - 1]$ *then* $\tilde{h}_D > h_C$ *for all sufficiently small* $\lambda_L > 0$. *If* $\mathrm{var}(\tilde{H}) > \mathrm{E}[\tilde{H} - 1]$ *then* $\tilde{h}_D < h_C$ *for all sufficiently small* $\lambda_L > 0$.

If $\mathrm{var}(\tilde{H}) = \mathrm{E}[\tilde{H} - 1]$, higher terms in the Maclaurin expansion of $\tilde{h}_D(\lambda_L) - h_C(\lambda_L)$ are required in order to give an ordering. Note that Corollaries 2.6 and 2.13 produce contrasting orderings of $\tilde{h}_D$ and h_C if $\mathrm{E}[\tilde{H}] - 1 < \mathrm{var}(\tilde{H}) < \mathrm{E}[\tilde{H}](\mathrm{E}[\tilde{H}] - 1)$. (See Section 2.5.3 for a numerical exploration of this.)

A result for a common household size $n > 1$ follows immediately from Corollary 2.13.

Corollary 2.14. *Suppose all households are the same size* $n > 1$. *For all sufficiently small* $\lambda_L > 0$, *we have* $\tilde{h}_D > h_C$.

Proof. When the common household size is $n > 1$, we have $\mathrm{var}(\tilde{H}) = 0$ and $\mathrm{E}[\tilde{H} - 1] > 0$. Applying Corollary 2.13 then establishes the claim. □

2.4.4 Common household size with $0 < \lambda_L < \infty$

We have shown that, for a common household size n and any λ_G such that $R_* > 1$, we have $\tilde{h}_D > h_C$ when $\lambda_L \to 0$ and $\lambda_L = \infty$. The following theorem considers $\lambda_L \in (0, \infty)$.

Theorem 2.15. *For a common household size $n = 2$ or $n = 3$, and for any λ_G and $\lambda_L > 0$ such that $R_* > 1$, we have $\tilde{h}_D > h_C$.*

Proof. Suppose a fraction z of the population is infected by a first epidemic in the households model with the above parameters. This leads to a threshold parameter $\hat{R}_{DI}(z)$ for the second epidemic. Further, assuming the same proportion are vaccinated uniformly at random gives threshold parameter $\hat{R}_U(z)$. We show that $\hat{R}_{DI}(z) - \hat{R}_U(z) > 0$, from which $\tilde{h}_D > h_C$ is immediate.

Let P_i^D $(i = 0, 1, \ldots, n)$ be the proportion of households with i members immune owing to the first epidemic and let P_i^U $(i = 0, 1, \ldots, n)$ be the analogous quantity with uniformly at random vaccination, both assuming a fraction z of the population is immune. Then we can write

$$\hat{R}_U(z) = \lambda_G \mathrm{E}[T_I] \sum_{v=0}^{n} \left(1 - \frac{v}{n}\right) P_v^U \mu_{n-v}(\lambda_L)$$

and

$$\hat{R}_{DI}(z) = \lambda_G \mathrm{E}[T_I] \sum_{v=0}^{n} \left(1 - \frac{v}{n}\right) P_v^D \mu_{n-v}(\lambda_L).$$

Assuming $n = 2$ and considering the proportion of susceptibles remaining after vaccination also yields

$$2P_0^D + P_1^D = 2P_0^U + P_1^U.$$

Now $A := P_0^D - P_0^U = \pi^2 - (1-z)^2 > 0$, since the probability an individual avoids global infection π is larger than the overall probability it avoids infection $1 - z$. We then find

$$\begin{aligned}\hat{R}_{DI}(z) - \hat{R}_U(z) &= \lambda_G \mathrm{E}[T_I] \left[\left(P_0^D - P_0^U\right) \mu_2(\lambda_L) + \frac{1}{2}(P_1^D - P_1^U)\mu_1(\lambda_L) \right] \\ &= A\lambda_G \mathrm{E}[T_I] \left[\mu_2(\lambda_L) - \mu_1(\lambda_L)\right] > 0,\end{aligned}$$

since $\mu_2(\lambda_L) > \mu_1(\lambda_L)$ and $A > 0$. The result follows and the claim is established

for $n = 2$. The proof for $n = 3$ uses a similar (but more involved) argument and is deferred to Section 2.7.3.

□

A proof for $n > 3$ has not been forthcoming, but we make the following conjecture, which is supported by numerical evidence (Figure 2.10) in Section 2.5.2.

Conjecture 2.16. *For any common household size $n > 1$, and for any λ_G and λ_L such that $R_* > 1$, we have $\tilde{h}_D > h_C$.*

In Theorem 2.2 we show that for a common household size in the highly locally infectious case, the difference $\tilde{h}_D - h_C$ is maximised at $R_0 = 2$. We show numerically that this does not hold when $\lambda_L < \infty$. For ease of visualisation we work in terms of the probability that an infectious individual makes local infectious contact with a given individual in their household, $p_L = p_L(\lambda_L) = 1 - \phi(\lambda_L)$, instead of λ_L directly. Note that p_L is a monotonic function of λ_L, with $p_L(0) = 0$ and $p_L(\infty) = 1$. Taking $T_I \sim \text{Exp}(1)$, we have $p_L = \lambda_L(1+\lambda_L)^{-1}$. In Figure 2.6 we fix $n \in \{2,3,4,5,6\}$ and $p_L(\lambda_L)$, determine $\hat{\lambda}_G = \arg\max_{\lambda_G}(\tilde{h}_D - h_C)$, the global infection rate which maximises the difference (assumed to be positive on the basis of Conjecture 2.16) between the herd immunity levels, then calculate the resulting value of R_0.

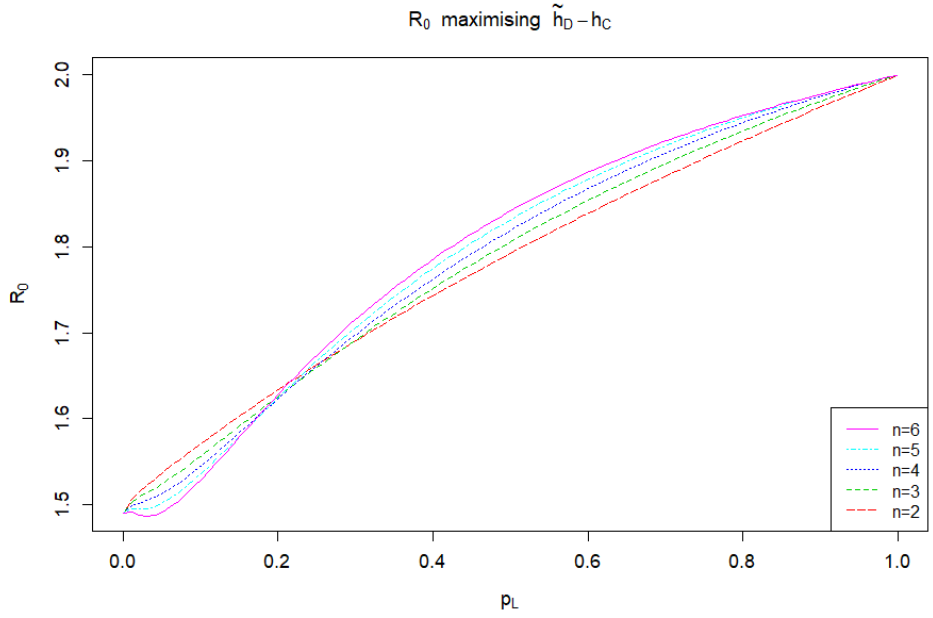

Figure 2.6: Plot of R_0 such that $\tilde{h}_D - h_C$ is maximised, where the common household size $n \in \{2,3,4,5,6\}$ and $T_I \sim \text{Exp}(1)$.

We see that the 'optimal' value of R_0 broadly increases with p_L and tends to 2 as $p_L \to 1$, consistent with Theorem 2.2. The dip near $p_L = 0$ becomes more pronounced as n increases. Similar observations regarding the 'optimal' value of R_0 and the dip at $p_L = 0$ hold for other choices of infectious period distribution which are not presented here.

When $p_L = 0$ we have $\tilde{h}_D = h_C$. As such, the value of R_0 when $\lambda_L = 0$ is not well-defined, since there is not a unique value of λ_G maximising $\tilde{h}_D - h_C$. This leads to instability when solving numerically. However, in the general setting with variable household sizes we can proceed analytically using Theorem 2.12. We have, as $\lambda_L \downarrow 0$,

$$\tilde{h}_D(\lambda_L) - h_C(\lambda_L) = 2\lambda_L^2 \pi(0)^2 (1-\pi(0)) \left[\mathrm{E}[\tilde{H}-1] - \mathrm{var}(\tilde{H})\right] + o(\lambda_L^2), \quad (2.35)$$

where $\pi(0) = \frac{1}{\lambda_G}$. If $\mathrm{E}[\tilde{H}-1] > \mathrm{var}(\tilde{H})$ ($\mathrm{E}[\tilde{H}-1] < \mathrm{var}(\tilde{H})$), the right-hand-side of (2.35), ignoring the $o(\lambda_L^2)$ term, is maximised (minimised) at $\hat{\pi}_0 = \frac{2}{3}$, yielding $\hat{\lambda}_G = 1.5$. The value of R_0 maximising $\left|\tilde{h}_D(\lambda_L) - h_C(\lambda_L)\right|$ then satisfies $R_0 \to 1.5$ as $\lambda_L \to 0$.

2.5 Numerical comparisons of herd immunity levels

2.5.1 Effect of infectious period

By taking $T_I \sim \exp(\gamma)$ in Section 2.3.2 we are able to write down ODEs, describing the deterministic epidemic, for the purposes of calculating h_D. In principle this can be extended to $T_I \sim \text{Gamma}(k,k)$ for $k \in \mathbb{N}$ using the method of stages, however this quickly gives rise to an unmanageable number of ODEs. Calculation of $\tilde{h}_D$ requires no such assumption on T_I, since $\tilde{h}_D$ relies only upon final size results. We briefly investigate how the choice of infectious period affects $\tilde{h}_D$, showing that the effect is very small for appropriately calibrated models.

We assume for the purposes of this exposition that $\mathrm{E}[T_I] = 1$. In order to study the effect of the infectious period, we consider $T_I \sim \text{Gamma}(k,k)$, where $k \geq 1$ is allowed to vary. When $k = 1$ we recover an exponential random variable with rate 1 and when $k \to \infty$ the infectious period distribution converges to a degenerate distribution with all its mass at one, so that large k closely resembles a constant infectious period; we write $k = \infty$ when referring to a constant infectious period with mean one.

In Figure 2.7 we plot $\tilde{h}_D$ and h_C, for several values of k, as functions of p_L for fixed R_0. That is, for a given value of p_L, we choose λ_G so that R_0 is fixed. In Figure 2.8 we fix R_0 but instead plot $\tilde{h}_D$ and h_C as functions of λ_L.

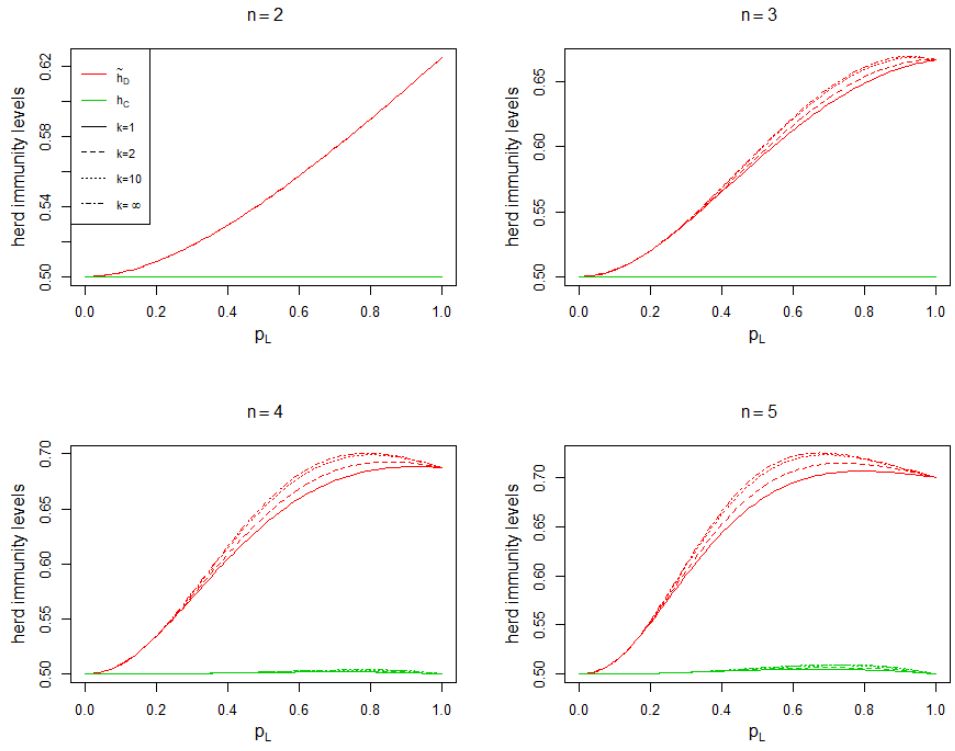

Figure 2.7: Herd immunity levels $\tilde{h}_D$ and h_C with $R_0 = 2$ as functions of p_L, taking $k \in \{1, 2, 10, \infty\}$ and with common household size $n \in \{2, 3, 4, 5\}$.

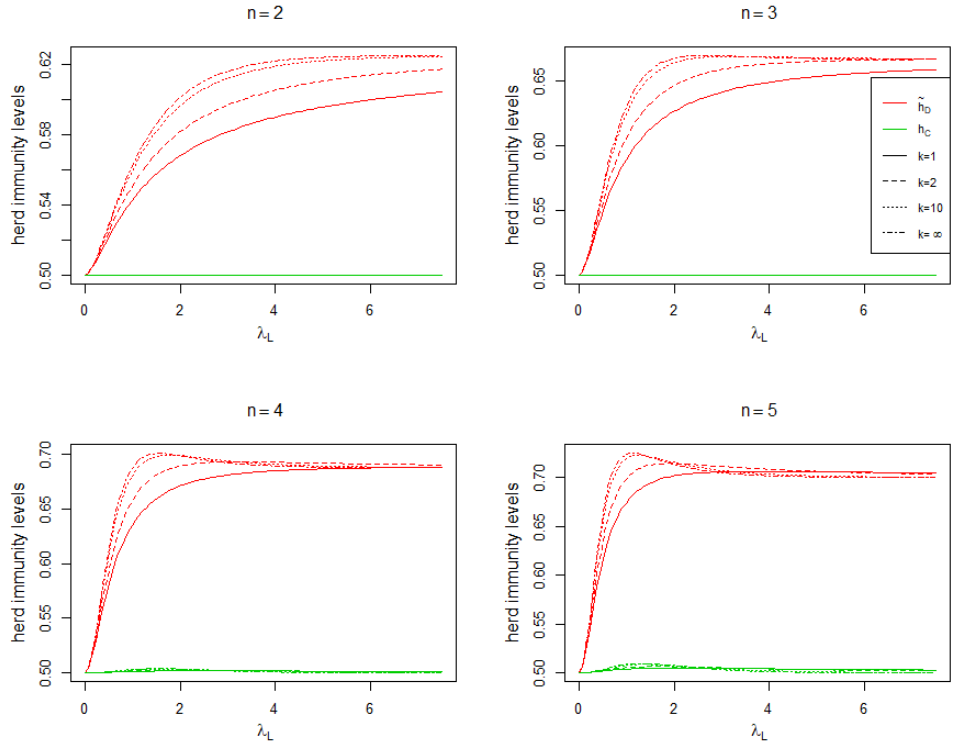

Figure 2.8: Herd immunity levels $\tilde{h}_D$ and h_C with $R_0 = 2$ as functions of λ_L, taking $k \in \{1, 2, 10, \infty\}$ and with common household size $n \in \{2, 3, 4, 5\}$.

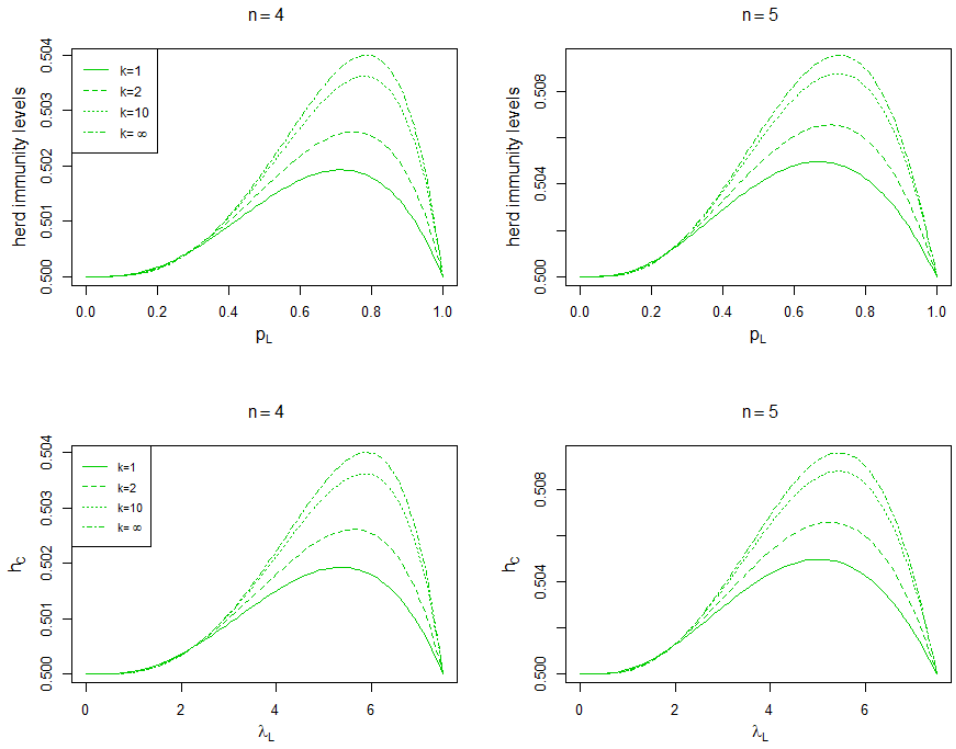

Figure 2.9: Comparison of h_C for various values of k, taking $R_0 = 2$, $k \in \{1,2,10,\infty\}$ and with common household size $n \in \{2,3,4,5\}$. Top panel: h_C as a function of p_L for fixed R_0. Bottom panel: h_C as a function of λ_L.

In an obvious notation, we have $\tilde{h}_D^{(k)} = \tilde{h}_D^{(1)}$ for fixed p_L when the common household size $n = 2$; this follows from the fact that $\mu_2(\lambda_L) = 1 + p_L$. In Figure 2.7 we observe that $\tilde{h}_D^{(\infty)} - \tilde{h}_D^{(1)}$ is typically small; the maximum percentage difference in this example when $n = 5$ is 4.18%. We also observe that the infectious period has more of an impact when the household sizes are larger.

In Figure 2.8 the discrepancy between $\tilde{h}_D^{(k)}$ for different values of k is larger, owing to the lack of calibration of the models used; when $T_I \sim \text{Gamma}(k,k)$ we have

$$p_L = 1 - \left(\frac{k}{k+\lambda_L}\right)^k > 1 - \frac{1}{1+\lambda_L}.$$

Thus, a given value of λ_L implies a smaller value of p_L when $k = 1$ compared to $k > 1$. The lack of calibration then leads to quite different values for $\tilde{h}_D^{(k)}$ as k varies.

In Figure 2.9 we provide a more detailed comparison of h_C from Figure 2.7 and Figure 2.8. The cases $n = 2$ and $n = 3$ are omitted, since $h_C = 1 - R_0^{-1}$ is constant in these cases – see Ball et al. [2016], Theorem 1. We note the scale on

the y-axis, which shows very small deviation from $y = 1 - R_0^{-1}$. Again using an obvious notation, the difference between $h_C^{(k)}$ and $1 - R_0^{-1}$ increases both with k and with the common household size n, although not by a substantial amount.

We have seen that, for models calibrated by p_L, the disease-induced herd immunity level $\tilde{h}_D$ is not strongly affected by the choice of infectious period distribution. Moreover, we have observed that typically $\tilde{h}_D > h_D$, with the above examples suggesting that this inequality would still hold, since $\tilde{h}_D^{(k)}$ increases with k. The lack of monotonicity in the above illustrations is not surprising, since in those examples R_0 is being held fixed, so λ_G is implicitly changing to account for the increase in λ_L or p_L.

In Section 2.5.2 we numerically illustrate some of the results from Section 2.4, then in Section 2.5.3 we explore how our findings play out in the context of realistic household size distributions. Owing to the above discussion we, throughout the remainder of this section, restrict attention the case where $T_I \sim \text{Exp}(\gamma)$, with $T_E \sim \text{Exp}(\delta)$ when a latent period is present.

2.5.2 Illustrative examples

In this subsection we assume that $\gamma = 1$ and, where applicable, $\delta = 1$ also. We begin by plotting in Figure 2.10 how h_D, h_D^L, $\tilde{h}_D$ and h_C vary with p_L for a common household size n when λ_G is chosen so that $R_0 = 2$ is fixed. (For details pertaining to the numerical solution of (2.11), see Appendix C.2.)

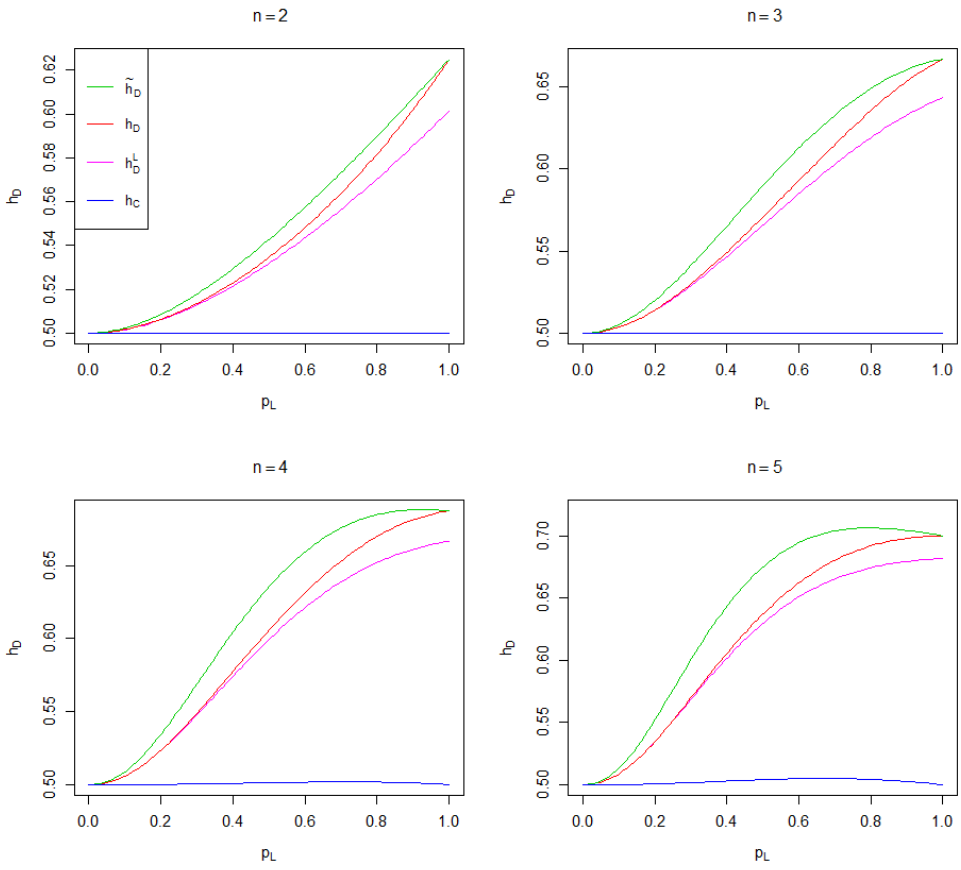

Figure 2.10: Herd immunity levels for fixed $R_0 = 2$ with common household size $n \in \{2,3,4,5\}$ and $\delta = \gamma = 1$.

Figure 2.10 provides an illustration of Theorem 2.15 and support for its extension in Conjecture 2.16; we observe $\tilde{h}_D > h_C$ throughout Figure 2.10. We also observe from Figure 2.10 that we have $h_D > h_D^L$. Analytical comparison of h_D^L and h_D is often not tractable, however we can show that $h_D > h_D^L$ for p_L sufficiently close to 1 as follows. When $p_L = 1$, under the SIR model disease-induced herd immunity leaves households either fully susceptible or fully non-susceptible. As noted in the discussion following Theorem 2.1, this corresponds to the worst possible vaccination strategy for a given coverage, implying $h_D > h_D^L$, since under the SEIR model there may be households in which only the initial case in that household is non-susceptible.

In Figure 2.11 we consider the same comparisons as in Figure 2.10, but with a variable household size distribution. Specifically, we take $\tilde{\alpha}_1 = \tilde{\alpha}_n = 0.5$ for some $n > 1$; half the individuals in the population are in households of size 1 and the other half are in households of size n. This implies that $\alpha_1 = n/(n+1)$

and $\alpha_n = 1/(n+1)$.

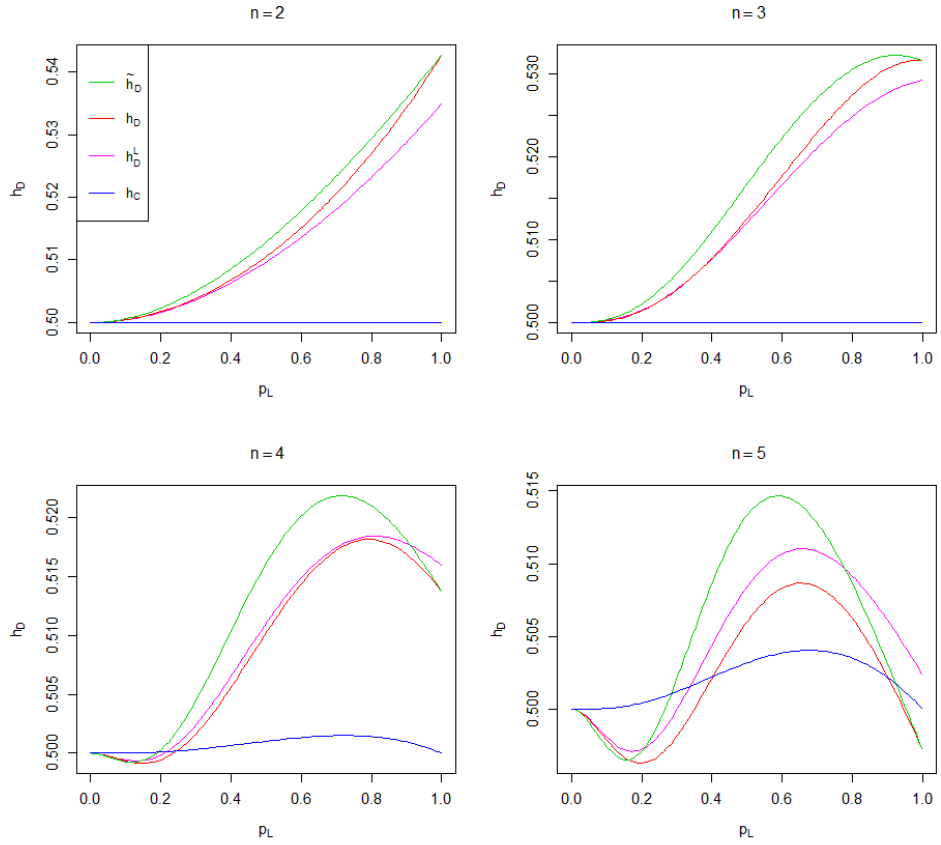

Figure 2.11: Herd immunity levels for fixed $R_0 = 2$ with household size distribution such that $\tilde{\alpha}_1 = \tilde{\alpha}_n = 0.5$ for $n \in \{2,3,4,5\}$.

A first observation based on Figure 2.11 is that the behaviour near $p_L = 0$ is consistent with the predictions of Section 2.4.3. Specifically, since $E[\tilde{H}] = (n-1)/2$ and $\text{var}(\tilde{H}) = (n-1)^2/4$, Theorem 2.12 predicts that $h_C > \tilde{h}_D$ near $p_L = 0$ if and only if $n \geq 4$. There is contrasting behaviour in terms of the shape of the herd immunity levels as n increases. When $n = 2$ and $n = 3$, h_C is the smallest of the considered herd immunity levels for all p_L. By contrast, when $n = 4$ or $n = 5$ there are values of p_L such that h_C is the largest of the considered herd immunity levels. As n increases in Figure 2.11, the approximation $\tilde{h}_D$ for h_D gets worse. Introducing a latent period does not necessarily lead to a reduction in the disease-induced herd immunity level; we observe that $h_D^L > h_D$ when $n = 4$ and $n = 5$.

Note that in Figures 2.10 and 2.11 we have $h_C \geq 1 - 1/R_0$, with strict inequality unless all households are of size 3 or less; this follows from Theorem

1 of Ball et al. [2016].

2.5.3 Real-world household size distributions

This section is motivated by the study in Britton et al. [2020], which considers the influence of population heterogeneity on the disease-induced herd immunity level for the COVID-19 pandemic. Britton et al. [2020] uses a Markov SEIR model in a population that is structured by age and activity level, in which for all individuals the exposed and infectious periods follow $\text{Exp}(1/3)$ and $\text{Exp}(1/4)$ distributions, respectively, with the unit of time being a day. Thus, the mean exposed and infectious periods are 3 and 4 days, respectively. Using the approximation to h_D described in Section 2.1, Britton et al. [2020] find that, when $R_0 = 2.5$, h_D for a homogeneously mixing model and h_C are both 60%; for the model with both age and activity structure, h_D is reduced to 43.0%.

The aim of the present numerical study is to investigate the effect of household structure on h_D, using a model with the above values of δ and γ and a range of real-world household size distributions. In order to achieve that we need a way of calibrating models with different choices of (λ_L, λ_G). One possibility is to keep the basic reproduction number R_0 fixed. However, R_0 is not uniquely defined for household models. The definition in Section 2.2.2 uses so-called rank generations and a different value for R_0 would typically be obtained if real-time generations were used instead, as for example in Neal and Therapod [2019]. In practice, for an emerging epidemic, R_0 is often estimated indirectly, via an estimate of the epidemic's early exponential growth rate r; see, for example, Wallinga and Lipsitch [2007]. For the multitype SEIR model used in Britton et al. [2020], R_0 and r satisfy

$$R_0 = \left(1 + \frac{r}{\delta}\right)\left(1 + \frac{r}{\gamma}\right); \tag{2.36}$$

see Sections 1.3.1 and 1.5 of the supplementary material of Trapman et al. [2016]. Note that the relationship (2.36) between R_0 and r is the same for all models in this class of multitype Markov SEIR epidemics and in particular matches that for the homogeneously mixing Markov SEIR model (Trapman et al. [2016]).

We adopt the following method of calibrating models with different (λ_L, λ_G), based on the early exponential growth rate r. For a given choice of R_0 in Britton *et al.*'s model, which we denote by R_0^{BBT}, we use (2.36) to calculate the corresponding value of the early exponential growth rate r under a multitype SEIR model. Then for a given value of $\lambda_L \in [0,\infty)$, we choose λ_G so that the early exponential growth rate of our households SEIR model equals r; see Section 2.2.3 for details. As previously, we use the local infection probability $p_L = 1-\phi(\lambda_L)$ in the figures.

We consider real-world household size distributions from demographically diverse countries. Note that the exact distribution is not available for some countries we consider and hence is estimated by maximum likelihood estimation using the available data (mean household size and summaries of the household size distribution). The household structures, their corresponding sources and estimation procedures are given in Section 2.5.4.

We begin by considering weakly locally infectious epidemics and just supercritical epidemics in Figure 2.12, which illustrates Corollaries 2.6 and 2.13. This implicitly gives orderings of $\tilde{h}_D$ and h_C for such epidemics, for a range of realistic household size distributions. Countries with $(\mu_{\tilde{H}}, \sigma^2_{\tilde{H}})$ below (above) the solid curve have $\tilde{h}_D > h_C$ ($\tilde{h}_D < h_C$) for just supercritical epidemics in the highly locally infectious case; those below (above) the dashed curve have $\tilde{h}_D > h_C$ ($\tilde{h}_D < h_C$) in the weakly locally infectious case.

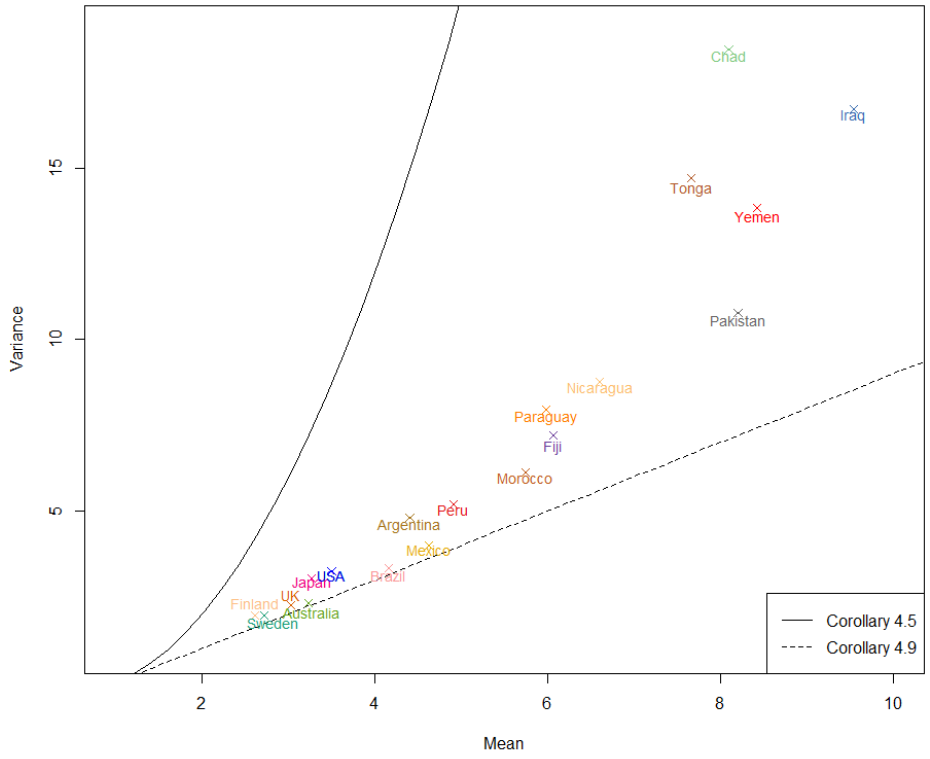

Figure 2.12: Critical values of $(\mu_{\tilde{H}}, \sigma^2_{\tilde{H}})$ from Corollaries 2.6 and 2.13, together with values of these quantities for several countries' household size distributions.

The region enclosed between the solid and dashed black curves in Figure 2.12 represents the set of values of $(\mu_{\tilde{H}}, \sigma^2_{\tilde{H}})$ for which Corollary 2.6 and Corollary 2.13 give different orderings of $\tilde{h}_D$ and h_C. We see that all countries considered have household size distributions in this set; though some are very close to the critical line $E[\tilde{H}-1] = \text{var}(\tilde{H})$ in the weakly locally infectious case. The scatterplot then indicates that the ordering of $\tilde{h}_D$ is indeed sensitive to the parameter choices, in terms of (λ_G, λ_L) and the household size distribution. Differing estimates of the global and local infection rates can lead to different conclusions in terms of whether vaccine-induced or disease-induced herd immunity are preferable.

We now explore the various herd immunity levels in our SIR and SEIR models, using the household size distributions of the UK (Figure 2.13) and Morocco (Figure 2.14) as exemplars. These countries are chosen because of their quite different household size distributions (cf. Figure 2.12). The computation

of h_D^L is omitted for Morocco as its calculation becomes numerically infeasible, since the dimension of the system of ODEs (2.11) becomes too large owing to the high maximum household size.

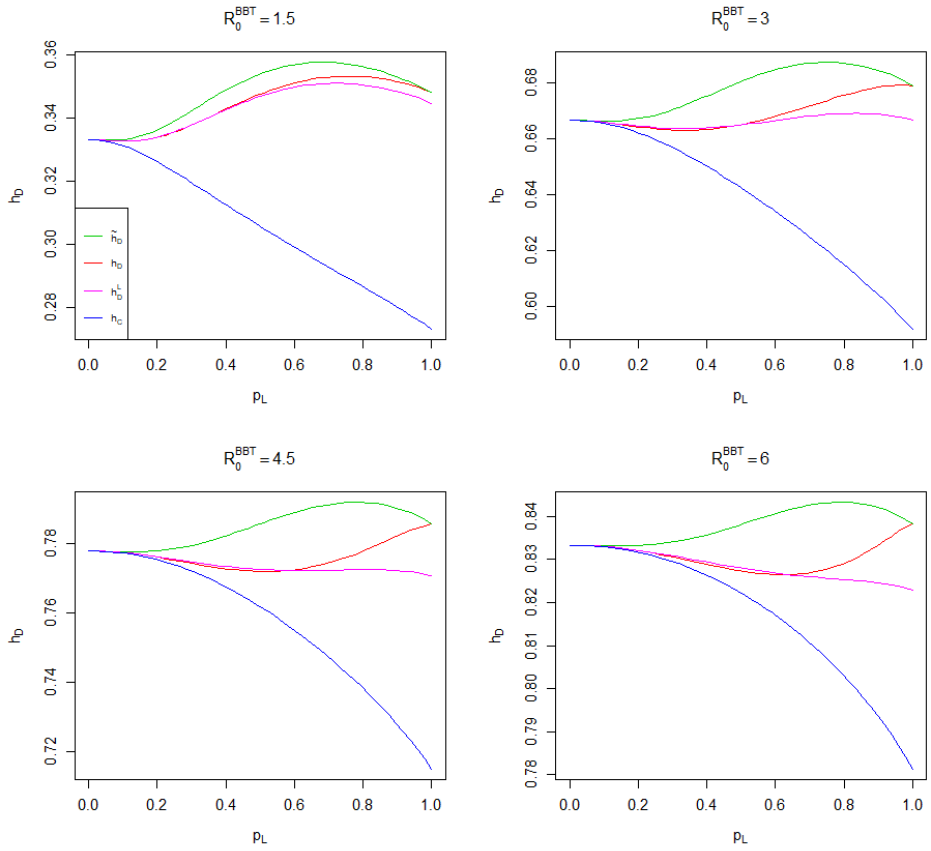

Figure 2.13: Herd immunity levels maintaining a fixed growth rate r implied by a given value of R_0^{BBT}, for the UK household size distribution.

Considering the UK household size distribution, which has $\mu_{\tilde{H}} = 3.02$ and $\sigma^2_{\tilde{H}} = 2.26$, we see that $h_D > h_C$, which is as expected given we have observed $h_D < h_C$ only in cases where household sizes have very high variability. We also observe less variation in h_D^L than in the other herd immunity levels. Increasing R_0^{BBT} leads to the growth rate r being fixed at a higher value, in turn causing higher herd immunity levels. We also observe that h_D^L and h_D are very close for fixed r as p_L increases from 0, until around $p_L = 0.6$.

We observe very similar qualitative behaviour for other household size distributions, as shown for the Morocco household size distribution (which has $\mu_{\tilde{H}} = 5.74$ and $\sigma^2_{\tilde{H}} = 6.12$) in Figure 2.14.

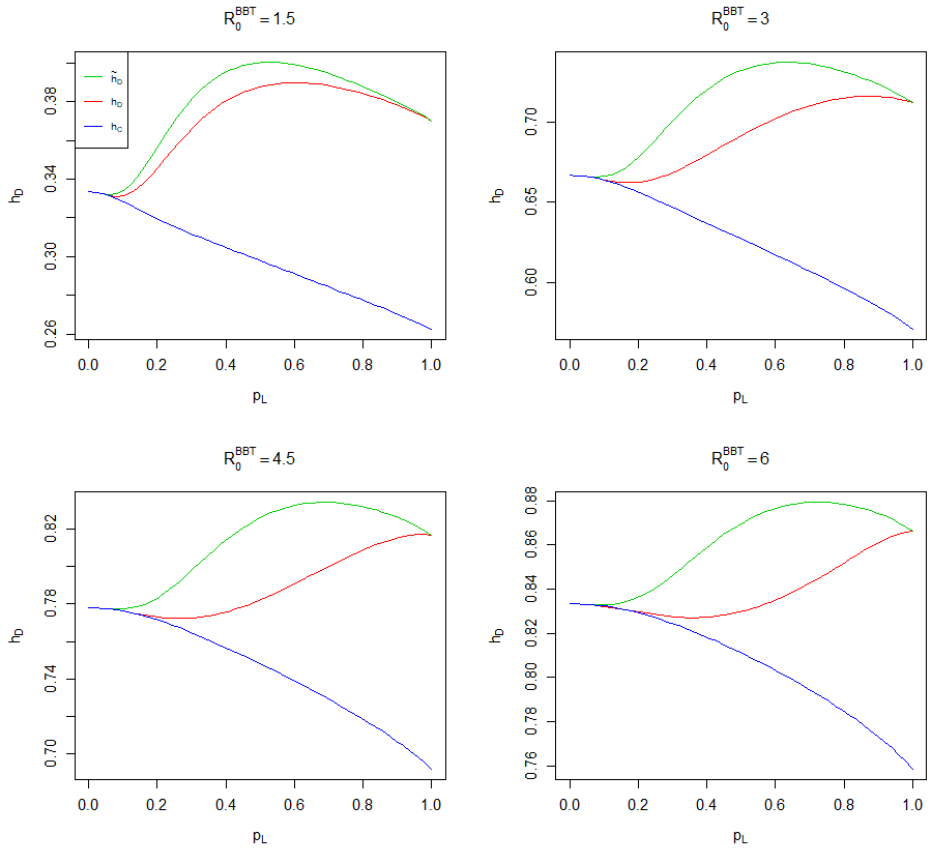

Figure 2.14: Herd immunity levels maintaining a fixed growth rate r implied by a given value of R_0^{BBT}, for Morocco's household size distribution.

We now explore the quantitative differences between the herd immunity levels in detail for a wider range of countries' household size distributions. Specifically, in Figure 2.15 we compare the various herd immunity levels between several countries in the absence of a latent period, with $R_0^{\mathrm{BBT}} = 3$. (Estimates of R_0 for COVID-19 vary greatly even for the same country, but other choices for R_0^{BBT} produce qualitatively similar results.)

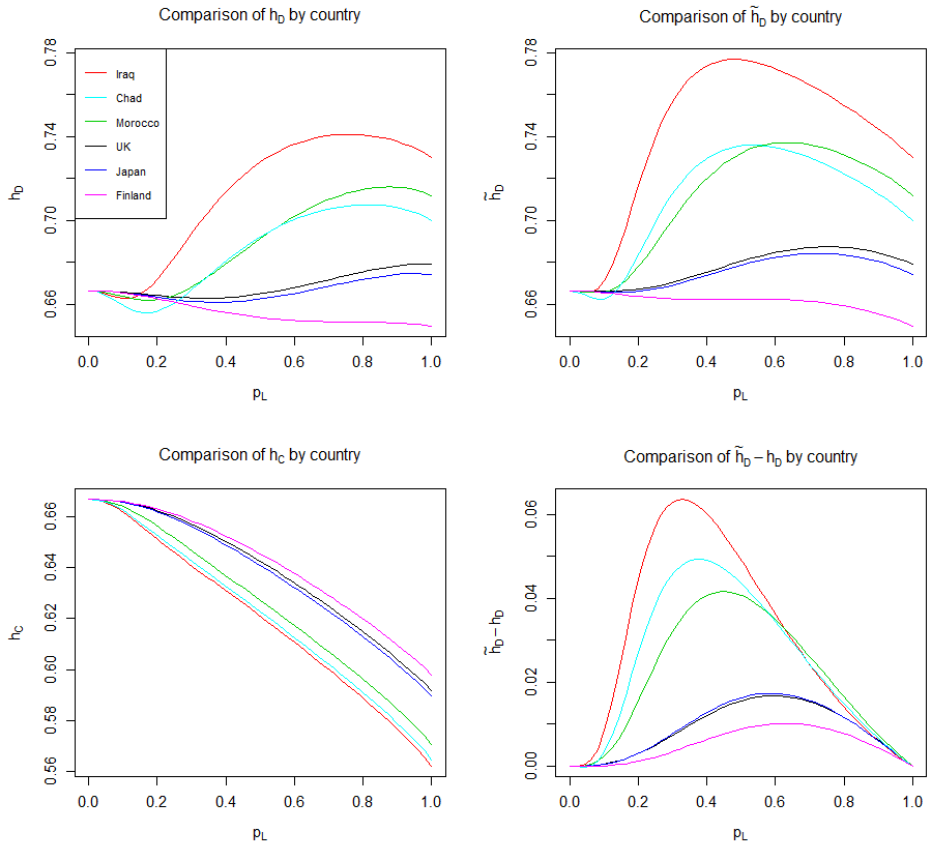

Figure 2.15: Comparison of h_D, $\tilde{h}_D$, $\tilde{h}_D - h_D$ and h_C respectively by country for $R_0^{\mathrm{BBT}} = 3$ (with r held fixed) comparing Iraq, Chad, Morocco, UK, Japan and Finland.

We observe $\tilde{h}_D > h_D$ in Figure 2.15, as well as $\tilde{h}_D = h_D$ at $p_L = 0$ and $p_L = 1$. For countries with generally smaller household sizes (i.e. Finland, Japan and the UK), $\tilde{h}_D$ and h_D are very close in value. Countries with a larger value of $\mu_{\tilde{H}}$ give larger values for h_D and $\tilde{h}_D$ but lower values of h_C. We generally observe $\tilde{h}_D > h_C$; the exceptions to this are for Morocco, Finland and Japan when p_L is close to zero, and even then the difference between $\tilde{h}_D$ and h_C is very small. We see h_C decreases monotonically with p_L, whereas $\tilde{h}_D$ and h_D are not monotone in their dependence on p_L. Finally, considering the last plot in Figure 2.15, we find that the difference $\tilde{h}_D - h_D$ is maximised at a smaller value of p_L, with a larger maximum difference, when $\mu_{\tilde{H}}$ is larger.

2.5.4 Household data

We briefly explain the data on which the analysis in Section 2.5.3 is based. The household data used for Finland, Sweden, Italy and the UK are taken from a Eurostat EU-SILC survey [2022]. The remaining data are from the United Nations household composition dataset [2017] and for these countries the proportion of households of each type is not readily available; the information available is $\mathrm{E}[H]$ as well as $P(H=1)$, $P(H=2 \text{ or } H=3)$, $\mathrm{P}(H=4 \text{ or } H=5)$ and $\mathrm{P}(H>5)$. For these countries we then wish to estimate, for $x=1,2,\ldots$, $\mathrm{P}(H=x)$, from which we can also estimate $\mathrm{P}(\tilde{H}=x)=\tilde{\alpha}_x$. To do so, we use maximum likelihood estimation assuming H has a shifted negative binomial distribution having probability mass function

$$f_{r,p}(x)=\frac{\Gamma(x)}{\Gamma(r)\Gamma(x-r+1)}p^r(1-p)^{x-1}, \qquad x=1,2,\ldots,$$

where $r\in(0,\infty)$ and $p\in(0,1)$ are parameters to be estimated. Since $\mathrm{E}[H]$ is known, this reduces to a one-parameter maximisation (for r, say) using $\mathrm{E}[H]=1+\frac{r(1-p)}{p}$. Then, letting $\tilde{p}=\tilde{p}(r)=\frac{r}{\mathrm{E}[H]+r-1}$ and x_i be the proportion of households of size i ($i=1,2,\ldots$), we have the likelihood

$$L(r)=[f_{r,\tilde{p}}(1)]^{x_1}\,[f_{r,\tilde{p}}(2)+f_{r,\tilde{p}}(3)]^{x_2+x_3}\,[f_{r,\tilde{p}}(4)+f_{r,\tilde{p}}(5)]^{x_4+x_5} \times\left[1-\sum_{i=1}^{5}f_{r,\tilde{p}}(i)\right]^{1-\sum_{i=1}^{5}x_i},$$

which can be optimised numerically over r to find the maximum likelihood estimate of r.

We note also that calculating any herd immunity level when the household size distribution has unbounded support requires truncation of that distribution. The truncation point must be chosen carefully to ensure that results are insensitive to the precise choice. For the household size distributions that we use we find that the approximation $\tilde{\alpha}_{15}=1-\sum_{n=1}^{14}\tilde{\alpha}_n$ is sufficient.

2.6 Discussion

We have presented a general framework for investigating disease-induced herd immunity in epidemic models with household structure. Calculating the disease-induced herd immunity level h_D for such models is not straightforward and we have introduced a useful approximation $\tilde{h}_D$, which is more amenable to analysis. In sharp contrast to most forms of heterogeneous mixing, for which h_D is *less* than the vaccine-induced herd immunity level h_C, the imposition of household structure generally leads to h_D being *greater* than h_C, unless the variability in the household size distribution is sufficiently large. This is proved using $\tilde{h}_D$ for epidemics which are either highly or weakly locally infectious, and numerical studies support the conjecture that it holds more generally.

The results in this chapter have shown that imposing heterogeneous population structures can increase the disease-induced herd immunity level. This should be kept in mind by organisations undertaking pandemic preparation, particularly among populations that contain large groups of individuals which mix locally. Whilst it is typically true that highly active individuals are infected more quickly, which helps to "speed up" disease-induced herd immunity, the opposing effect caused by household structure should also be kept in mind.

It would be worthwhile to consider more fully the impact of restrictions, such as lockdown, on $\hat{h}_D$, the disease-induced herd immunity level when restrictions are in place. In Section 2.3.2, we give an example where such restrictions affect only the global infection parameter λ_G, for which the impact of the restrictions on $\hat{h}_D$ is minimal; moreover, the approximation of $\hat{h}_D$ by $\tilde{h}_D$ improved with increasing restrictions. Similar results were found with other examples. However, in that example restrictions were applied uniformly with time which is unlikely to be the case in practice. Further, in practice restrictions may also affect the local infection rate λ_L; indeed it is not hard to envisage scenarios in which λ_L might increase. Another worthwhile avenue for future research is to consider models which combine household structure with other forms of heterogeneous mixing. This can be done using the multitype households model and a similar approximation to $\tilde{h}_D$ for the disease-induced herd immunity level can

be calculated using results in Ball and Lyne [2001]. In Chapter 5 we investigate this for a model with activity levels, as in Britton et al. [2020], and also household structure.

We have assumed throughout that the individual-to-individual local infection rate λ_L is independent of household size n. Although this assumption is often made with household models and is usually reasonable for small n, such as in the UK, Sweden and Finland household size distributions, it is less easily justified for countries with large household sizes, such as Iraq, Pakistan and Chad. One would expect λ_L to decrease with n and it would be interesting to explore the consequent impact on h_D. Note that the results of Section 2.4.2 concerning the highly locally infectious case are unaffected but other results may change.

Throughout a large part of this work we have used $\tilde{h}_D$ as an approximation to h_D. The only models in which we have computed h_D are those in which the infectious and latent periods follow exponential distributions. In real-life epidemics, the distributions of these quantities are usually far from exponential. Moreover, the impact of departures from exponential distributions on epidemic properties is usually greater in models incorporating small mixing groups, such as households. Although it is possible in principle to use the method of stages to extend the deterministic model in Section 2.2.5 to include Erlang distributed infectious and latent periods, and to allow for varied local and global infection rates between stages of infection, in practice, the number of ODEs soon becomes infeasible. However, it is straightforward to calculate $\tilde{h}_D$ for such models, and indeed for models in which individuals have infectivity profiles (for example, Goldstein et al. [2009]), since such calculation only requires final outcome properties of an epidemic. We have found that $\tilde{h}_D > h_D$ in most of our numerical studies with exponentially distributed infectious and latent periods, and that the difference is typically small unless the mean household size is large. We expect a similar conclusion to hold for models with other, more realistic, choices of infectious and latent period distributions.

2.7 Proofs

2.7.1 Proof of Theorem 2.8

Proof. Let $\pi \in (0,1)$. Using (2.17), $R_{DI}(\pi) = \lambda_G \mathrm{E}[T_I][(1-p)\pi + np\pi^n]$. Note that the proportion z infected in the first epidemic satisfies $z = 1-(1-p)\pi - p\pi^n$. We compare $R_{DI}(\pi)$ with the corresponding reproduction number $R_U(\pi)$, when this fraction z of the population is vaccinated uniformly at random. Using (2.13) (cf. (2.14)),

$$\begin{aligned} R_U(\pi) &= \lambda_G \mathrm{E}[T_I][1-z+p(n-1)(1-z)^2] \\ &= \lambda_G \mathrm{E}[T_I]\left\{(1-p)\pi + p\pi^n + p(n-1)[(1-p)\pi + p\pi^n]^2\right\}. \end{aligned}$$

Hence,

$$R_U(\pi) - R_{DI}(\pi) = p(n-1)\lambda_G \mathrm{E}[T_I]\left([(1-p)\pi + p\pi^n]^2 - \pi^n\right).$$

Let $h_n(\pi) = \pi^{\frac{n}{2}-1} - p\pi^{n-1}$. Elementary algebra shows that $R_U(\pi) - R_{DI}(\pi) < 0$ if $h_n(\pi) > 1-p$, $R_U(\pi) - R_{DI}(\pi) = 0$ if $h_n(\pi) = 1-p$, and $R_U(\pi) - R_{DI}(\pi) > 0$ if $h_n(\pi) < 1-p$. Now $h_2(\pi) = 1-p\pi > 1-p$ for all $\pi \in (0,1)$, so when $n=2$, $R_U(\pi) < R_{DI}(\pi)$ for all $\pi \in (0,1)$, whence $\tilde{h}_D > h_C$ for all λ_G (more precisely all λ_G such that the epidemic is supercritical). Suppose $n \geq 3$. Now $h_n(0) = 0$, $h_n(1) = 1-p$ and

$$h_n'(\pi) = \left(\frac{n}{2} - 1\right)\pi^{\frac{n}{2}-2} - p(n-1)\pi^{n-2}.$$

If $p \leq \frac{n-2}{2(n-1)}$ then $h_n'(\pi) > 0$ for all $\pi \in (0,1)$, so $h_n(\pi) < 1-p$ for all $\pi \in (0,1)$, leading to $\tilde{h}_D < h_C$ for all λ_G. Suppose that $p > \frac{n-2}{2(n-1)}$ and let $\pi_n^*(p) = \left(\frac{n-2}{2p(n-1)}\right)^{\frac{2}{n}}$. Then $h_n'(\pi) > 0$ for $\pi \in (0, \pi_n^*(p))$ and $h_n'(\pi) < 0$ for $\pi \in (\pi_n^*(p), 1)$. Hence, $h_n(\pi) = 1-p$ has a unique solution, $\hat{\pi}_n(p)$ say, in $(0,1)$. Further, $h_n(\pi) < 1-p$ for $\pi \in (0, \hat{\pi}_n(p))$, implying $R_U(\pi) > R_{DI}(\pi)$, and $h_n(\pi) > 1-p$ for $\pi \in (\hat{\pi}_n(p), 1)$, implying $R_U(\pi) < R_{DI}(\pi)$. It follows that there exists $\lambda_G^*(n,p)$ such that $\tilde{h}_D > h_C$ if $\lambda_G < \lambda_G^*(n,p)$, and $\tilde{h}_D < h_C$ if $\lambda_G > \lambda_G^*(n,p)$. The change in behaviour occurs when $R_{DI}(\hat{\pi}_n(p)) = R_U(\hat{\pi}_n(p)) = 1$. Substitut-

ing $\hat{\pi}_n(p)$ into the above expression for $R_{DI}(\pi)$ yields

$$\lambda_G^*(n,p) = \left[\mathrm{E}[T_I]\hat{\pi}_n(p)\left(1-p+np\hat{\pi}_n(p)^{n-1}\right)\right]^{-1},$$

as required. □

2.7.2 Proof of Theorem 2.12

For $k = 0,1,\ldots,n$, let $n_{[k]} = n!/(n-k)!$ denote the falling factorial. Consider the single-household epidemic $\tilde{E}_n(\lambda_L,\pi)$ described in Section 2.2.4. Let $\tilde{S}_n = n - \tilde{T}_n$ be the number of susceptibles remaining at the end of the epidemic. Define $\mu_n(\lambda_L)$ as in Section 2.2.2 and also define, for $k = 1,2$,

$$\hat{\mu}_{n,k}(\lambda_L,\pi) = \mathrm{E}[(\tilde{S}_n)_{[k]}].$$

Recall we assume that $\mathrm{E}[T_I] = 1$ and $\mathrm{var}(T_I) < \infty$. We start with a preliminary lemma.

Lemma 2.17. *For $n = 1,2,\ldots$, we have $\mu_n(0) = 1$ and $\mu_n'(0) = n-1$. Let $\pi = \pi(\lambda_L)$. For $n = 1,2,\ldots$ and $k = 1,2$, we have $\hat{\mu}_{n,k}(0,\pi(0)) = n_{[k]}\pi(0)^k$, $\frac{\partial}{\partial\lambda_L}\hat{\mu}_{n,k}(0,\pi(0)) = -kn_{[k+1]}\pi(0)^k(1-\pi(0))$ and $\frac{\partial}{\partial\pi}\hat{\mu}_{n,k}(0,\pi(0)) = n_{[k]}k\pi(0)^{k-1}$. Further, $\frac{\partial^2}{\partial\pi^2}\hat{\mu}_{n,1}(0,\pi(0)) = 0$ and $\frac{\partial^2}{\partial\lambda_L\partial\pi}\hat{\mu}_{n,1}(0,\pi(0)) = -n(n-1)(1-2\pi(0))$, where all derivatives are evaluated at $\lambda_L = 0$. For example, $\frac{\partial}{\partial\lambda_L}\hat{\mu}_{n,k}(0,\pi(0)) = \frac{\partial}{\partial\lambda_L}\hat{\mu}_{n,k}(\lambda_L,\pi)\Big|_{(\lambda_L,\pi)=(0,\pi(0))}$.*

Proof. We make use of Gontcharoff polynomials; see Picard and Lefèvre [1990], Section 2, for details. For a given sequence $U = u_0,u_1,\ldots$ of real numbers, the corresponding Gontcharoff polynomials, $G_0(x \mid U), G_1(x \mid U),\ldots$, are defined by

$$\sum_{i=0}^{n} n_{[i]}u_i^{n-i}G_i(x \mid U) = x^n \quad (n = 0,1,\ldots). \tag{2.37}$$

We consider the real sequence with $u_i = \phi(i\lambda_L)$. For $k = 1,2$ and $i = 0,1,\ldots$, let $G_{i,k}(\lambda_L) = G_i(1 \mid E^kU)$, where E^kU denotes the sequence $u_k,u_{k+1},\ldots$. Using

Ball [2019], Proposition 3.1 and Properties 2.1 and 2.2, gives

$$\mu_n(\lambda_L) = n - \sum_{i=1}^{n-1} (n-1)_{[i]} \phi(i\lambda_L)^{n-i} G_{i-1,1}(\lambda_L) \tag{2.38}$$

and

$$\hat{\mu}_{n,k}(\lambda_L, \pi) = \sum_{i=k}^{n} n_{[i]} \phi(i\lambda_L)^{n-i} \pi^i G_{i-k,k}(\lambda_L) \quad (k = 1,2). \tag{2.39}$$

Consider $G_{i,k}(0)$ for $k = 1,2$. Substituting $\lambda_L = 0$ into (2.37) implies that $\sum_{i=0}^{n} n_{[i]} G_{i,k}(0) = 1$. Recalling $n_{[0]} = 1$ then gives $G_{0,k}(0) = 1$, leading to $G_{i,k}(0) = \delta_{i,0}$ for $i = 0, 1, \ldots$, where

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

Differentiating (2.37) with respect to λ_L and setting $\lambda_L = 0$ yields

$$\sum_{i=0}^{n} n_{[i]} \left(G'_{i,k}(0) - G_{i,k}(0)(n-i)(i+k) \right) = 0.$$

Using $G_{i,k}(0) = \delta_{i,0}$ then gives $G'_{i,k}(0) = k\delta_{i,1}$, for $i = 0, 1, \ldots$ and $k = 1,2$. We now take the appropriate partial derivatives of (2.38), (2.39) and substitute $\lambda_L = 0$, noting that $\phi(0) = 1$ and $\phi'(0) = -\mathrm{E}[T_I] = -1$. Differentiating (2.38) with respect to λ_L gives

$$\begin{aligned} \mu'_n(\lambda_L) = &- \sum_{i=1}^{n-1} (n-1)_{[i]} (n-i) i \phi(i\lambda_L)^{n-i-1} \phi'(i\lambda_L) G_{i-1,1}(\lambda_L) \\ &- \sum_{i=1}^{n-1} (n-1)_{[i]} \phi(i\lambda_L)^{n-i} G'_{i-1,1}(\lambda_L). \end{aligned} \tag{2.40}$$

Substituting $\lambda_L = 0$ into (2.38), (2.40) and (2.39) respectively then establishes that $\mu_n(0) = 1$, $\mu'_n(0) = n-1$ and $\hat{\mu}_{n,k}(0, \pi(0)) = n_{[k]} \pi(0)^k$.

Differentiating (2.39) with respect to λ_L, we find

$$\begin{aligned} \frac{\partial}{\partial \lambda_L} \hat{\mu}_{n,k}(\lambda_L, \pi) = &\sum_{i=k}^{n} n_{[i]} (n-i) i \phi(i\lambda_L)^{n-i-1} \phi'(i\lambda_L) \pi^i G_{i-k,k}(\lambda_L) \\ &+ \sum_{i=k}^{n} n_{[i]} \phi(i\lambda_L)^{n-i} \pi^i G'_{i-k,k}(\lambda_L), \end{aligned} \tag{2.41}$$

from which letting $\lambda_L = 0$ gives $\frac{\partial}{\partial \lambda_L} \hat{\mu}_{n,k}(0, \pi(0)) = -k n_{[k+1]} \pi(0)^k (1 - \pi(0))$.

Next, taking the derivative of (2.39) with respect to π yields

$$\frac{\partial}{\partial \pi}\hat{\mu}_{n,k}(\lambda_L,\pi) = \sum_{i=k}^{n} n_{[i]}\phi(i\lambda_L)^{n-i} i\pi^{i-1} G_{i-k,k}(\lambda_L), \tag{2.42}$$

which gives $\frac{\partial}{\partial \pi}\hat{\mu}_{n,k}(0,\pi(0)) = n_{[k]}k\pi(0)^{k-1}$ by setting $\lambda_L = 0$. For the second derivatives, we require further differentiation. Firstly, differentiating (2.41) with respect to π, we have

$$\begin{aligned}\frac{\partial^2}{\partial \lambda_L \partial \pi}\hat{\mu}_{n,k}(\lambda_L,\pi) = &\sum_{i=k}^{n} n_{[i]}(n-i)i^2\phi(i\lambda_L)^{n-i-1}\phi'(i\lambda_L)\pi^{i-1}G_{i-k,k}(\lambda_L) \\ &+ \sum_{i=k}^{n} n_{[i]}\phi(i\lambda_L)^{n-i} i\pi^{i-1} G'_{i-k,k}(\lambda_L).\end{aligned} \tag{2.43}$$

Taking $\lambda_L = 0$ and $k = 1$ in (2.43), we find $\frac{\partial^2}{\partial \lambda_L \partial \pi}\hat{\mu}_{n,1}(0,\pi(0)) = -n(n-1)(1-2\pi(0))$. Finally, differentiating (2.42) with respect to π gives

$$\frac{\partial^2}{\partial \pi^2}\hat{\mu}_{n,k}(\lambda_L,\pi) = \sum_{i=k}^{n} n_{[i]}\phi(i\lambda_L)^{n-i} i(i-1)\pi^{i-2} G_{i-k,k}(\lambda_L). \tag{2.44}$$

Letting $\lambda_L = 0$ and $k = 1$ in (2.44) causes all terms to vanish, so that $\frac{\partial^2}{\partial \pi^2}\hat{\mu}_{n,1}(0,\pi(0)) = 0$, which then establishes the final result of Lemma 2.17. □

We are now in a position to prove Theorem 2.12. For ease of exposition in the following proof, we denote $h_C(\lambda_L)$ by $c(\lambda_L)$. Similarly, we denote $h_D(\lambda_L)$ by $z(\lambda_L)$. Recall that $n_{\max} < \infty$, so all sums in the proof contain only finitely many terms and hence are easily differentiated.

Proof. of Theorem 2.12 Suppose a fraction $c(\lambda_L)$ are vaccinated prior to an epidemic, such that the epidemic becomes critical. Note $c(0) = 1 - \frac{1}{\lambda_G}$ (recall $\mathrm{E}[T_I] = 1$). Now considering disease-induced herd immunity, assume that a fraction $z(\lambda_L)$ are infected in a first epidemic such that the second epidemic is critical. Let $\pi(\lambda_L)$ be the proportion who avoid global infection in the first epidemic. Then $z(0) = 1 - \frac{1}{\lambda_G}$ and $\pi(0) = \frac{1}{\lambda_G}$. We show that $c'(0) = z'(0) = \frac{\mu_{\tilde{H}-1}}{\lambda_G^2}$ and that

$$z''(0) - c''(0) = 4\pi(0)^2(1-\pi(0))\left(\mathrm{E}[\tilde{H}-1] - \mathrm{var}(\tilde{H})\right),$$

from which Theorem 2.12 follows immediately by Taylor's theorem.

We begin by considering the derivatives of $c(\lambda_L)$ at $\lambda_L = 0$. The post-vaccination threshold parameter $\hat{R}_U(c)$ defined at (2.3.1) satisfies $\hat{R}_U(c(\lambda_L)) = 1$, so by substituting $i = n - v$ in the inner sum in (2.3.1) we have that

$$\lambda_G \sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{i=1}^{n} \frac{i}{n} \binom{n}{i} (1 - c(\lambda_L))^i c(\lambda_L)^{n-i} \mu_i(\lambda_L) = 1. \tag{2.45}$$

Let $q_{n,i}(c) = \frac{i}{n}\binom{n}{i}(1-c)^i c^{n-i}$. Then $\sum_{i=1}^{n} q_{n,i}(c) = 1 - c$, $\sum_{i=1}^{n} q'_{n,i}(c) = -1$ and $\sum_{i=1}^{n} q''_{n,i}(c) = 0$, by exchanging the order of derivative and summation. We also have $\sum_{i=1}^{n}(i-1)q_{n,i}(c) = (n-1)(1-c)^2$, so $\sum_{i=1}^{n}(i-1)q'_{n,i}(c) = -2(n-1)(1-c)$. Differentiating (2.45) gives

$$\sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{i=1}^{n} \left[q'_{n,i}(c(\lambda_L)) c'(\lambda_L) \mu_i(\lambda_L) + q_{n,i}(c(\lambda_L)) \mu'_i(\lambda_L) \right] = 0. \tag{2.46}$$

Substituting $\lambda_L = 0$ in (2.46) and recalling $c(0) = 1 - \frac{1}{\lambda_G}$ yields $c'(0) = \frac{\mu_{\tilde{H}-1}}{\lambda_G^2}$. Differentiating (2.46) gives

$$\begin{aligned} \sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{i=1}^{n} \left[q''_{n,i}(c(\lambda_L)) c'(\lambda_L)^2 + q'_{n,i}(c(\lambda_L)) c''(\lambda_L) \right] \mu_i(\lambda_L) \\ + \sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{i=1}^{n} 2 q'_{n,i}(c(\lambda_L)) c'(\lambda_L) \mu'_i(\lambda_L) \\ + \sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{i=1}^{n} q_{n,i}(c(\lambda_L)) \mu''_i(\lambda_L) = 0. \end{aligned} \tag{2.47}$$

Let $A_n = \sum_{i=1}^{n} q_{n,i}(c(0)) \mu''_i(0)$. Then we set $\lambda_L = 0$ in (2.47) which yields

$$c''(0) = \sum_{n=1}^{\infty} \tilde{\alpha}_n A_n - 4c'(0)(1 - c(0)) \mu_{\tilde{H}-1}. \tag{2.48}$$

Turning now to $z(\lambda_L)$, let $\tilde{P}_{n,i}(\lambda_L, \pi(\lambda_L)) = \mathrm{P}(\tilde{S}_n = i)$ be the probability i members of a household avoid infection in the epidemic $\tilde{E}_n(\lambda_L, \pi(\lambda_L))$. Suppose all individuals infected (no longer susceptible) in the first epidemic are immune to infection in the second epidemic. Suppose the second epidemic is critical, so

that $R_{DI}(\pi(\lambda_L)) = 1$. Then considering the remaining susceptibles yields

$$\lambda_G \sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{i=1}^{n} \frac{i}{n} \tilde{P}_{n,i}(\lambda_L, \pi(\lambda_L)) \mu_i(\lambda_L) = 1. \tag{2.49}$$

Further, the proportion of the population infected in the first epidemic is given by (cf. (2.7))

$$z(\lambda_L) = 1 - \sum_{n=1}^{\infty} \tilde{\alpha}_n \frac{1}{n} \hat{\mu}_{n,1}(\lambda_L, \pi(\lambda_L)). \tag{2.50}$$

Differentiating (2.49) gives

$$\begin{aligned} \sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{i=1}^{n} \frac{i}{n} \left[\mu_i(\lambda_L) \left\{ \frac{\partial}{\partial \lambda_L} \tilde{P}_{n,i}(\lambda_L, \pi(\lambda_L)) + \frac{\partial}{\partial \pi} \tilde{P}_{n,i}(\lambda_L, \pi(\lambda_L)) \pi'(\lambda_L) \right\} \right] \\ + \sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{i=1}^{n} \frac{i}{n} \left[\mu_i'(\lambda_L) \tilde{P}_{n,i}(\lambda_L, \pi(\lambda_L)) \right] = 0, \end{aligned} \tag{2.51}$$

which can be used to solve for $\pi'(0)$ by setting $\lambda_L = 0$. Applying Lemma 2.17 and noting that $\hat{\mu}_{n,k}(\lambda_L, \pi(\lambda_L)) = \sum_{i=1}^{n} i_{[k]} \tilde{P}_{n,i}(\lambda_L, \pi(\lambda_L))$ yields $\pi'(0) = (\pi(0) - 2\pi(0)^2)\mu_{\tilde{H}-1}$. Differentiating (2.50), we have

$$z'(\lambda_L) = -\sum_{n=1}^{\infty} \tilde{\alpha}_n \frac{1}{n} \left\{ \frac{\partial}{\partial \lambda_L} \hat{\mu}_{n,1}(\lambda_L, \pi(\lambda_L)) + \frac{\partial}{\partial \pi} \hat{\mu}_{n,1}(\lambda_L, \pi(\lambda_L)) \pi'(\lambda_L) \right\}. \tag{2.52}$$

Substituting $\lambda_L = 0$ in (2.52), we find that $z'(0) = \frac{\mu_{\tilde{H}-1}}{\lambda_G^2} = c'(0)$. Before proceeding with further differentiation, let $B_n = \frac{1}{n} \frac{\partial^2}{\partial \lambda_L^2} \hat{\mu}_{n,1}(0, \pi(0))$. Differentiating (2.51), we reach

$$\begin{aligned} \sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{i=1}^{n} \frac{i}{n} \left[2\mu_i'(\lambda_L) \left\{ \frac{\partial}{\partial \lambda_L} \tilde{P}_{n,i}(\lambda_L, \pi(\lambda_L)) + \frac{\partial}{\partial \pi} \tilde{P}_{n,i}(\lambda_L, \pi(\lambda_L)) \pi'(\lambda_L) \right\} \right] \\ + \sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{i=1}^{n} \frac{i}{n} \left[\mu_i(\lambda_L) \left\{ \frac{\partial^2}{\partial \lambda_L^2} \tilde{P}_{n,i}(\lambda_L, \pi(\lambda_L)) + 2\pi'(\lambda_L) \frac{\partial^2}{\partial \lambda_L \partial \pi} \tilde{P}_{n,i}(\lambda_L, \pi(\lambda_L)) \right\} \right] \\ + \sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{i=1}^{n} \frac{i}{n} \left[\mu_i(\lambda_L) \left\{ \pi''(\lambda_L) \frac{\partial}{\partial \pi} \tilde{P}_{n,i}(\lambda_L, \pi(\lambda_L)) + [\pi'(\lambda_L)]^2 \frac{\partial^2}{\partial \pi^2} \tilde{P}_{n,i}(\lambda_L, \pi(\lambda_L)) \right\} \right] \\ + \sum_{n=1}^{\infty} \tilde{\alpha}_n \sum_{i=1}^{n} \frac{i}{n} \left[\mu_i''(\lambda_L) \tilde{P}_{n,i}(\lambda_L, \pi(\lambda_L)) \right] = 0, \end{aligned}$$

from which substituting $\lambda_L = 0$ and applying Lemma 2.17 as well as the defini-

tion of B_n gives

$$-\pi''(0) = \sum_{n=1}^{\infty} \tilde{\alpha}_n \left[A_n + B_n - 4(n-1)(n-2)\pi(0)^2(1-\pi(0))\right] + \sum_{n=1}^{\infty} \tilde{\alpha}_n \left[4(n-1)\pi(0)\pi'(0) + 2(n-1)\pi'(0)(2\pi(0)-1)\right]. \tag{2.53}$$

Differentiating (2.52), we find

$$z''(\lambda_L) = -\sum_{n=1}^{\infty} \tilde{\alpha}_n \frac{1}{n} \left\{ \frac{\partial^2}{\partial \lambda_L^2} \hat{\mu}_{n,1}(\lambda_L, \pi(\lambda_L)) + 2\pi'(\lambda_L) \frac{\partial^2}{\partial \lambda_L \partial \pi} \hat{\mu}_{n,1}(\lambda_L, \pi(\lambda_L)) \right\} - \sum_{n=1}^{\infty} \tilde{\alpha}_n \frac{1}{n} \left\{ [\pi'(\lambda_L)]^2 \frac{\partial^2}{\partial \pi^2} \hat{\mu}_{n,1}(\lambda_L, \pi(\lambda_L)) + \pi''(\lambda_L) \frac{\partial}{\partial \pi} \hat{\mu}_{n,1}(\lambda_L, \pi(\lambda_L)) \right\}.$$

We hence observe that

$$z''(0) = \sum_{n=1}^{\infty} \tilde{\alpha}_n \left[2(n-1)\pi'(0)(1-2\pi(0)) - B_n - \pi''(0)\right]. \tag{2.54}$$

Combining (2.53) and (2.54) establishes that

$$z''(0) = \sum_{n=1}^{\infty} \tilde{\alpha}_n \left[A_n + 4(n-1)\pi(0)\pi'(0) - 4(n-1)(n-2)\pi(0)^2(1-\pi(0))\right]. \tag{2.55}$$

Finally, noting that $c(0) = 1 - \pi(0)$ and that $c'(0) + \pi'(0) = \mu_{\tilde{H}-1}\pi(0)(1-\pi(0))$, we subtract (2.48) from (2.55) to reach

$$\begin{aligned} z''(0) - c''(0) &= 4\pi(0)^2(1-\pi(0)) \left[\mu_{\tilde{H}-1}^2 - \sum_{n=1}^{\infty} \tilde{\alpha}_n (n-1)(n-2)\right] \\ &= 4\pi(0)^2(1-\pi(0)) \left[(\mathrm{E}[\tilde{H}-1])^2 - \mathrm{E}[(\tilde{H}-1)(\tilde{H}-2)]\right] \\ &= 4\pi(0)^2(1-\pi(0)) \left[\mathrm{E}[\tilde{H}-1] - \mathrm{var}(\tilde{H})\right]. \end{aligned}$$

This establishes Theorem 2.12. □

2.7.3 Proof of Theorem 2.15 ($n = 3$)

We begin by making the notation in Theorem 2.15 explicit for the case $n = 3$. For $i \in \{0,1,2,3\}$, P_i^D is the probability of i members being infected in a house-

hold of size 3 during an epidemic in the households model in which a proportion z are infected in the first epidemic. Similarly, P_i^U is the probability of a household containing i vaccinated individuals, assuming vaccination uniformly at random with probability z. Note that P_i^D, P_i^U and z are considered as functions of $\pi \in (0,1)$. We begin with a preliminary lemma. Write $q_1 = \phi(\lambda_L)$ and $q_2 = \phi(2\lambda_L)$ and observe that $0 < q_2 < q_1 < 1$ for all $\lambda_L > 0$. Further, $P_0^U = (1-z)^3$, $P_1^U = 3z(1-z)^2$ and $P_2^U = 3z^2(1-z)$. The system in (2.8), or direct calculation, gives $P_0^D = \pi^3$, $P_1^D = 3\pi^2(1-\pi)q_2$ and $P_2^D = 3\pi(1-\pi)q_1\left(2\pi(q_1-q_2)+(1-\pi)q_1\right)$.

Lemma 2.18. *Let $A = 3(P_0^D - P_0^U)$, $B = 2(P_1^D - P_1^U)$ and $C = P_2^D - P_2^U$. Then $A - C > 0$.*

Proof. Considering the remaining susceptibles, note that

$$3P_0^D + 2P_1^D + P_2^D = 3(1-z) \qquad \text{and} \qquad 3P_0^U + 2P_1^U + P_2^U = 3(1-z). \quad (2.56)$$

Hence, $A + B + C = 0$, so

$$A - C = 2A + B = 6\left(\pi^3 + \pi^2(1-\pi)q_2 - (1-z)^2\right).$$

Using the first equation in (2.56),

$$1 - z = \pi\left(\pi^2 + 2\pi(1-\pi)[q_2(1-q_1) + q_1^2] + (1-\pi)^2 q_1^2\right) \equiv \pi h(\pi),$$

so $A > C$ if and only if $f(\pi) > 0$, where

$$f(\pi) = \pi + (1-\pi)q_2 - h(\pi)^2.$$

Jensen's inequality gives $q_2 > q_1^2$, so $f(0) = q_2 - q_1^4 > 0$. Further, $f(1) = 0$, so $f(\pi) > 0$ for $\pi \in (0,1)$ if f is concave on $[0,1]$. Now,

$$f''(\pi) = -2[h(\pi)h''(\pi) + h'(\pi)^2],$$

whilst we also have $h(\pi) > 0$ and

$$h''(\pi) = 2(1-q_1)(1+q_1-2q_2) > 0.$$

Thus $f''(\pi) < 0$ for $\pi \in [0,1]$, so f is concave on $[0,1]$ and $A > C$, as required.

□

We now prove Theorem 2.15 in the case $n = 3$.

Proof. We show that $\hat{R}_{DI}(z) > \hat{R}_U(z)$, from which the desired result follows. We have that

$$\begin{aligned}\frac{3}{\lambda_G \mathrm{E}[T_I]}\left(\hat{R}_{DI}(z) - \hat{R}_U(z)\right) =& A\mu_3(\lambda_L) + B\mu_2(\lambda_L) + C\mu_1(\lambda_L) \\ =& A[\mu_3(\lambda_L) - \mu_2(\lambda_L)] - C[\mu_2(\lambda_L) - \mu_1(\lambda_L)].\end{aligned} \tag{2.57}$$

Using (2.2) gives $\mu_1(\lambda_L) = 1$, $\mu_2(\lambda_L) = 2 - q_1$, and $\mu_3(\lambda_L) = 3 - 2q_1(q_1 - q_2) - 2q_2$. Therefore

$$\mu_3(\lambda_L) - \mu_2(\lambda_L) - [\mu_2(\lambda_L) - \mu_1(\lambda_L)] = 2(q_1 - q_2)(1 - q_1) > 0. \tag{2.58}$$

Since (by Lemma 2.18) $A > C$, it follows from (2.57) and (2.58) that $\hat{R}_{DI}(z) > \hat{R}_U(z)$.

□

3 Gaussian approximation of disease-induced herd immunity level

3.1 Introduction

For many epidemic models we can write down a system of equations (typically ODEs) for a suitably scaled epidemic process as the population size $N \to \infty$. In this chapter we consider approximating the epidemic process by Gaussian fluctuations about a deterministic process. In order to do so, we borrow from the ideas of density dependent population processes described in Ethier and Kurtz [1986], Chapter 11. Our interest lies in a Gaussian approximation of the disease-induced herd immunity level and its corresponding asymptotic variance, which we calculate by considering the distribution of appropriate hitting times. Throughout this chapter we assume that the deterministic epidemic of interest has threshold parameter greater than one, as otherwise herd immunity would already have been achieved.

This chapter is structured as follows. In Section 3.2 we outline the general framework for the calculations that follow by briefly introducing density dependent population processes, following Ethier and Kurtz [1986], Chapter 11. We apply the framework to the homogeneously mixing case in Section 3.3 as an initial example, showing in Section 3.4 that analytical progress is possible by applying a random time change to the epidemic process. In Section 3.5 we define the multitype model with proportionate mixing and compute a Gaussian approximation of h_D for that model. In Section 3.6 we apply the same methods to the households model with a common household size. For both models we find that asymptotic variance of the herd immunity level is small; the deterministic approximation to the herd immunity level is good for even modest population sizes. We demonstrate the small asymptotic variance by calculating approximate confidence intervals and verify our results with stochastic simulations. Finally, in Section 3.7 we briefly discuss possible extensions of this work.

3.2 General framework

We begin by outlining the framework of density dependent jump processes in which we will operate; we then interpret this framework in the present epidemic setting. Consider a population process with population size n and dimension d. Let $E \subset \mathbb{R}^d$ and let $\beta_{\boldsymbol{l}}$ ($\boldsymbol{l} \in \mathbb{Z}^d$) denote a collection of non-negative functions defined on E. We define the set

$$E_n = E \cap \{n^{-1}\boldsymbol{k} : \boldsymbol{k} \in \mathbb{Z}^d\}$$

and we assume that for all $\boldsymbol{x} \in E_n$, $\beta_{\boldsymbol{l}}(\boldsymbol{x}) > 0$ implies $\boldsymbol{x} + n^{-1}\boldsymbol{l} \in E_n$. A density dependent family refers to a sequence $\{\hat{\boldsymbol{X}}_n\}$ of jump Markov processes such that $\hat{\boldsymbol{X}}_n$ has state space E_n and the following transition law holds:

$$q^{(n)}_{\boldsymbol{x},\boldsymbol{y}} = n\beta_{n(\boldsymbol{y}-\boldsymbol{x})}(\boldsymbol{x}), \quad \boldsymbol{x}, \boldsymbol{y} \in E_n,$$

where $q^{(n)}_{\boldsymbol{x},\boldsymbol{y}}$ is the transition rate from state $\boldsymbol{x}$ to state $\boldsymbol{y}$. The quantity $\beta_{\boldsymbol{l}}(\boldsymbol{x})$ is the rate at which jump $\boldsymbol{l}$ occurs when the process is in state $\boldsymbol{x}$. In the present epidemic setting, n refers to either the number of individuals or the number of households in the population and is the quantity by which we scale the process; the states are vectors of dimension d which describe the epidemic. In the homogeneously mixing epidemic, for example, these state vectors are of dimension 2, and contain the number of susceptible and infectious individuals respectively.

In order to proceed with the desired calculations we require more notation. We define the drift function

$$\boldsymbol{F}(\boldsymbol{x}) = \sum_{\boldsymbol{l}} \boldsymbol{l}\beta_{\boldsymbol{l}}(x) \tag{3.1}$$

as the expected increment when the process is in state $\boldsymbol{x}$. We let $Y_{\boldsymbol{l}}$ ($\boldsymbol{l} \in \mathbb{Z}^d$) denote independent standard Poisson processes and $\tilde{Y}_{\boldsymbol{l}}(u) = Y_{\boldsymbol{l}}(u) - u$. Writing $\boldsymbol{X}_n = n^{-1}\hat{\boldsymbol{X}}_n$ and letting $t \geq 0$, we note the representation

$$\boldsymbol{X}_n(t) = \boldsymbol{X}_n(0) + \sum_{\boldsymbol{l}} \boldsymbol{l} n^{-1} \tilde{Y}_{\boldsymbol{l}}\left(n\int_0^t \beta_{\boldsymbol{l}}(\boldsymbol{X}_n(s))ds\right) + \int_0^t F(\boldsymbol{X}_n(s))ds. \tag{3.2}$$

We assume that $\boldsymbol{X}_n = \{\boldsymbol{X}_n(t) : t \geq 0\}$ satisfies (3.2). Further, we assume that $\lim_{n\to\infty} \boldsymbol{X}_n(0) = \boldsymbol{x}_0$ and that the (deterministic) process $\boldsymbol{x} = \{\boldsymbol{x}(t) : t \geq 0\}$ satisfies

$$\boldsymbol{x}(t) = \boldsymbol{x}_0 + \int_0^t F(\boldsymbol{x}(s))ds, \quad t \geq 0. \tag{3.3}$$

Theorem 3.1 (Ethier and Kurtz [1986], Theorem 11.2.1)**.** *Suppose that $\boldsymbol{F}$ is Lipschitz continuous on all compact $K \subset E$ and that*

$$\sum_l |\boldsymbol{l}| \sup_{\boldsymbol{x}\in K} \beta_l(\boldsymbol{x}) < \infty.$$

Then, with $\boldsymbol{X}_n$ as in (3.2) *and $\boldsymbol{x}$ as in* (3.3) *we have, for every $t \geq 0$,*

$$\lim_{n\to\infty} \sup_{s\leq t} |\boldsymbol{X}_n(s) - \boldsymbol{x}(s)| = 0 \quad a.s.$$

Theorem 3.1 provides a strong law of large numbers for the convergence of the process $\boldsymbol{X}_n$ to $\boldsymbol{x}$. It follows from (3.3) that the process $\boldsymbol{x}$ evolves according to

$$\frac{d\boldsymbol{x}}{dt} = \boldsymbol{F}(\boldsymbol{x}(t)), \quad \boldsymbol{x}(0) = \boldsymbol{x}_0, \tag{3.4}$$

so that the drift function corresponds to the right-hand side of the ODE system describing the limiting behaviour of $\boldsymbol{X}_n$ as $n \to \infty$. Our main interest is in a central limit theorem for the herd disease-induced herd immunity level, so we require a central limit theorem for $\boldsymbol{X}_n$. We define the infinitesimal variance function

$$\boldsymbol{G}(\boldsymbol{x}) = \sum_l \boldsymbol{l}\boldsymbol{l}^\top \beta_l(\boldsymbol{x}). \tag{3.5}$$

We also write $\boldsymbol{F} = (f_1, f_2, \ldots, f_d)$ and let $\partial\boldsymbol{F} = \left[\frac{\partial f_i}{\partial x_j}\right]$ $(i, j = 1, 2, \ldots, d)$ denote the $d \times d$ matrix of partial derivatives of $\boldsymbol{F}$. Finally, we let $\Rightarrow$ denote weak convergence in the space of right-continuous functions $f : [0,\infty) \to \mathbb{R}^d$ having limits from the left (i.e. càdlàg functions), endowed with the Skorohod metric – see Ethier and Kurtz [1986], Chapter 3.5.

Theorem 3.2 (Ethier and Kurtz [1986], Theorem 11.2.3)**.** *Suppose that, for each compact $K \subset E$, we have*

(i) $\sum_{l} |l|^2 \sup_{x \in K} \beta_l(x) < \infty$;

(ii) Each jump rate β_l *is continuous;*

(iii) All partial derivatives of $\boldsymbol{F}$ *are continuous.*

Suppose further that $\boldsymbol{X}_n$ *satisfies* (3.2) *and* $\boldsymbol{x}$ *satisfies* (3.3)*, and let* $\boldsymbol{V}_n(t) = \sqrt{n}(\boldsymbol{X}_n(t) - \boldsymbol{x}(t))$*, such that* $\lim_{n\to\infty} \boldsymbol{V}_n(0) = \boldsymbol{V}(0)$ *is constant. Then*

$$\boldsymbol{V}_n \Rightarrow \boldsymbol{V} \text{ as } n \to \infty,$$

where $\boldsymbol{V} = \{\boldsymbol{V}(t) : t \geq 0\}$ *is a Gaussian process with mean* $\boldsymbol{H}(t,0)\boldsymbol{V}(0)$ *and covariance function*

$$\operatorname{cov}(\boldsymbol{V}(t), \boldsymbol{V}(r)) = \int_0^{\min(t,r)} \boldsymbol{H}(t,s)\boldsymbol{G}(\boldsymbol{x}(s))\boldsymbol{H}(r,s)^\top ds, \tag{3.6}$$

with $\boldsymbol{H}$ *the solution to the matrix ODE*

$$\frac{\partial \boldsymbol{H}(t,s)}{\partial t} = \partial \boldsymbol{F}(\boldsymbol{x}(t))\boldsymbol{H}(t,s), \quad \boldsymbol{H}(s,s) = \boldsymbol{I}, \tag{3.7}$$

and where $\boldsymbol{I}$ *denotes the identity matrix of appropriate dimension.*

In our setting we are interested in the state of the population at the time when herd immunity is achieved, i.e. where the threshold parameter for the epidemic crosses one. We are thus interested in the distribution of the hitting time (τ_n, say) corresponding to the time at which herd immunity is achieved. Suppose $\varphi(\boldsymbol{x})$ is such that $\varphi(\boldsymbol{x}(0)) > 0$, and that $\varphi(\boldsymbol{x}(t)) = 0$ corresponds to herd immunity being achieved. Define

$$\tau_n = \inf\{t : \varphi(\boldsymbol{X}_n(t)) \leq 0\}$$

and

$$\tau = \inf\{t : \varphi(\boldsymbol{x}(t)) \leq 0\}$$

as the corresponding stopping times for the processes $\boldsymbol{X}_n(t)$ and $\boldsymbol{x}(t)$ respectively. We note the following theorem which underpins a Gaussian approximation for the disease-induced herd immunity level.

Theorem 3.3 (Ethier and Kurtz [1986], Theorem 11.4.1)**.** *Suppose that $\boldsymbol{X}_n$, $\boldsymbol{x}$, $\boldsymbol{V}_n$, $\boldsymbol{V}$ and $\boldsymbol{F}$ are as in Theorem 3.2, and that the conditions of Theorem 3.1 and Theorem 3.2 are satisfied. Suppose further that the following conditions hold:*

(i) $\tau < \infty$*;*

(ii) φ *is continuously differentiable on* $\mathbb{R}^d$*, with first partial derivatives given by* $\nabla\varphi$ *;*

(iii) $\varphi(\boldsymbol{x}(0)) > 0$*;*

(iv) $\nabla\varphi(\boldsymbol{x}(\tau)) \cdot \boldsymbol{F}(\boldsymbol{x}(\tau)) < 0$*.*

Then, as $n \to \infty$*,*

$$\sqrt{n}\,(\boldsymbol{X}_n(\tau_n) - \boldsymbol{x}(\tau)) \Rightarrow \boldsymbol{V}(\tau) - \frac{\nabla\varphi(\boldsymbol{x}(\tau)) \cdot \boldsymbol{V}(\tau)}{\nabla\varphi(\boldsymbol{x}(\tau)) \cdot \boldsymbol{F}(\boldsymbol{x}(\tau))}\boldsymbol{F}(\boldsymbol{x}(\tau)). \tag{3.8}$$

Note that conditions (iii) and (iv) of Theorem 3.3 ensure that τ corresponds to the time of a proper crossing of $\varphi(\boldsymbol{x}(t)) = 0$. In the present epidemic setting we are interested in the asymptotic distribution of the disease-induced herd immunity level, which is a linear combination of elements of $\boldsymbol{X}_n$. This motivates the following result.

Theorem 3.4. *Under the same assumptions as Theorem 3.3, let* $\boldsymbol{w} \in \mathbb{R}^d$ *and suppose that* $y_n(\tau_n) = \boldsymbol{w} \cdot \boldsymbol{X}_n(\tau_n)$ *and* $y(\tau) = \boldsymbol{w} \cdot \boldsymbol{x}(\tau)$*. Let* $\Sigma(t)$ *denote the covariance matrix of* $\boldsymbol{V}(t)$*. Then, as* $n \to \infty$*,*

$$\sqrt{n}\,(y_n(\tau_n) - y(\tau)) \xrightarrow{\mathrm{D}} N\left(0, \boldsymbol{w}\boldsymbol{A}\boldsymbol{\Sigma}(\tau)\boldsymbol{A}^\top\boldsymbol{w}^\top\right),$$

where

$$\boldsymbol{A} = \boldsymbol{I} - \frac{\boldsymbol{F}(\boldsymbol{X}(\tau)) \bigotimes \nabla\varphi(\boldsymbol{X}(\tau))}{\nabla\varphi(\boldsymbol{X}(\tau)) \cdot \boldsymbol{F}(\boldsymbol{X}(\tau))}, \tag{3.9}$$

with $\bigotimes$ *denoting the outer vector product.*

Proof. Using the fact that $y_n(\tau_n) - y(\tau) = \boldsymbol{w} \cdot (\boldsymbol{X}_n(\tau_n) - \boldsymbol{x}(\tau))$, we apply Theorem 3.3 to reach the desired asymptotic distribution. The variance term is readily obtained upon right-factorising $\boldsymbol{V}(\tau)$ in (3.8) and using standard properties of covariance matrices. □

Note that we can compute the covariance matrix $\Sigma(t)$ by using (3.6) and (3.7) to find that $\Sigma(t)$ satisfies the matrix ODE

$$\frac{d\Sigma}{dt} = G(\boldsymbol{X}) + \partial \boldsymbol{F}(\boldsymbol{X})\Sigma + \Sigma \partial \boldsymbol{F}(\boldsymbol{X})^{\top}, \quad \Sigma(0) = \mathbf{0}. \tag{3.10}$$

We then (typically numerically) solve (3.4) and (3.10) simultaneously in order to calculate $\Sigma(t)$. (For details pertaining to the numerical solution of (3.4) and (3.10), see Appendix C.2.) In Section 3.4 and Section 3.5 we give examples where $\Sigma(t)$ can be calculated analytically. Hence, for a suitably chosen epidemic process $\boldsymbol{X}_n$, we can use Theorem 3.4 to establish the asymptotic variance of the disease-induced herd immunity level.

3.3 Homogeneously mixing case

We briefly describe the application of the general framework outlined previously to the homogeneously mixing epidemic with infection rate $\lambda > 1$ and recovery rate γ. In this model, we have states in the scaled process of the form $\boldsymbol{x}(t) = (x(t), y(t))$ corresponding to the proportions of susceptibles and infectives respectively at time t. The possible jumps in the original process are $\boldsymbol{l}_1 = (-1, 1)$ with associated jump rate λxy, and $\boldsymbol{l}_2 = (0, -1)$ with rate γy. From this we can readily compute $\boldsymbol{F}$, $\partial \boldsymbol{F}$ and $\boldsymbol{G}$. The stopping rule is given by

$$\varphi(x, y) = \lambda x - \gamma,$$

since R_0 among the remaining susceptibles is equal to one when $\varphi(x, y) = 0$. Applying Theorem 3.4 with $\boldsymbol{w} = (1, 0)$ and solving numerically allows us to find that the asymptotic variance of the disease-induced herd immunity level is zero in this case. This is to be expected, as in this case the disease-induced herd immunity level is given by $1 - x(\tau)$ and the stopping condition is equivalent to $x = \gamma\lambda^{-1}$. We demonstrate this behaviour in the following section by considering a random time change for the epidemic, which enables analytical progress to be made.

3.4 Random time change for homogeneously mixing epidemic

The differential equations for $\boldsymbol{x}(t) = (x(t), y(t))$ in the homogeneously epidemic are given by

$$\begin{aligned} \frac{dx}{dt} &= -\lambda xy \\ \frac{dy}{dt} &= \lambda xy - \gamma y, \end{aligned} \tag{3.11}$$

subject to $y(0) = 1 - x(0) = \mu$, where $\mu > 0$ is small and corresponds to a small amount of initial infection. We consider a random time change of this process, as in Watson [1980], in which we divide the right-hand side of the above system by $y(t)$, yielding equations which are more amenable to study. In doing so, we speed up the clock by a factor equal to the current proportion of infectives $y(t)$. This results in the process $(\tilde{x}(t), \tilde{y}(t))$ which has the same final outcome as the original process. Moreover, we may consider initialising this process with the proportion of initial infectives equal to zero - we cannot do this with (3.11) as the system would remain at $(x(t), y(t)) = (1, 0)$ for all $t \geq 0$.

The original epidemic has infinite duration with $y(t) \to 0$ as $t \to \infty$; in the time-changed process, the epidemic concludes in finite time (i.e. $\tilde{y}(t) = 0$ for some $t < \infty$). The time-changed system is then

$$\begin{aligned} \frac{d\tilde{x}}{dt} &= -\lambda \tilde{x} \\ \frac{d\tilde{y}}{dt} &= \lambda \tilde{x} - \gamma, \end{aligned}$$

subject to $(\tilde{x}(0), \tilde{y}(0)) = (1, 0)$. Solving, we find

$$\begin{aligned} \tilde{x}(t) &= \exp(-\lambda t) \\ \tilde{y}(t) &= 1 - \gamma t - \exp(-\lambda t), \end{aligned}$$

for $t \geq 0$. The two possible jumps are the same as in the original process, and

we find that $\tilde{\boldsymbol{F}} = (-\lambda\tilde{x}, \lambda\tilde{x} - \gamma)$, so that

$$\partial\tilde{\boldsymbol{F}} = \begin{pmatrix} -\lambda & 0 \\ \lambda & 0 \end{pmatrix}$$

and

$$\tilde{\boldsymbol{G}} = \begin{pmatrix} \lambda\tilde{x} & -\lambda\tilde{x} \\ -\lambda\tilde{x} & \lambda\tilde{x}+\gamma \end{pmatrix}.$$

Substituting these into (3.10) we find, in an obvious notation, that

$$\frac{d\sigma_{11}}{dt} = \lambda\exp(-\lambda t) - 2\lambda\sigma_{11},$$

so that, after imposing the initial condition $\sigma_{11}(0) = 0$, we have

$$\sigma_{11}(t) = \exp(-\lambda t) - \exp(-2\lambda t).$$

Similar calculations yield that $\sigma_{12}(t) = \sigma_{21}(t) = -\sigma_{11}(t)$, and $\sigma_{22}(t) = \gamma t + \sigma_{11}(t)$.

Consider the asymptotic variance of the proportion of infectives in the homogeneously mixing epidemic when the proportion of remaining susceptibles reaches a certain attainable level, α, say, where $\alpha < 1$. The stopping rule is $\varphi_\alpha(\tilde{x}, \tilde{y}) = \tilde{x} - \alpha$, so that $\nabla\varphi_\alpha = (1, 0)$ and

$$\boldsymbol{A} = \begin{pmatrix} 0 & 0 \\ 1 - \frac{\gamma}{\lambda\alpha} & 1 \end{pmatrix}.$$

We thus have

$$\boldsymbol{A}\boldsymbol{\Sigma}(\tau)\boldsymbol{A}^\top = \begin{pmatrix} 0 & 0 \\ 0 & v_\alpha(\tau) \end{pmatrix},$$

where

$$\begin{aligned} v_\alpha(\tau) &= \left(1 - \frac{\gamma}{\lambda\alpha}\right)^2 \sigma_{11}(\tau) + \sigma_{12}(\tau)\left(1 - \frac{\gamma}{\lambda\alpha}\right) + \sigma_{21}(\tau)\left(1 - \frac{\gamma}{\lambda\alpha}\right) + \sigma_{22}(\tau) \\ &= \left(\frac{\gamma}{\lambda\alpha}\right)^2 \sigma_{11}(\tau) + \gamma\tau. \end{aligned}$$

In this case we can solve for the stopping time τ directly to find that

$$\tau = -\frac{1}{\lambda}\log(\alpha) < \infty.$$

It is clear that φ_α is continuously differentiable on $\mathbb{R}^2$. Moreover, we have $\varphi_\alpha(\tilde{\boldsymbol{x}}(0)) = 1-\alpha > 0$ and

$$\nabla\varphi_\alpha(\tilde{\boldsymbol{x}}(\tau))\cdot\boldsymbol{F}(\tilde{\boldsymbol{x}}(\tau)) = (1,0)\cdot(-\lambda\tilde{x}(\tau),\lambda\tilde{x}(\tau)-\gamma) = -\lambda\tilde{x}(\tau) < 0,$$

whence the conditions of Theorem 3.4 are satisfied. The asymptotic variance of the proportion of infectives when the susceptible proportion reaches level α is $\dfrac{\sigma^2}{n}$, where

$$\sigma^2 = \alpha - \alpha^2 - \frac{\gamma}{\lambda}\log(\alpha). \tag{3.12}$$

Substituting $\alpha = \gamma\lambda^{-1}$ into (3.12) yields the asymptotic variance of the proportion of infectives at the point where herd immunity is achieved and, as noted in Section 3.3, the asymptotic variance for the proportion of susceptibles at herd immunity is zero in this example.

3.5 Multitype model with proportionate mixing

We now consider the multitype model with proportionate mixing which is studied, for example, in Britton et al. [2021], Section 2. In a population of size n, the model has dynamics as follows. Each individual in the population is one of J types, with the possible types belonging to the set $\{1,2,\ldots,J\} = \mathscr{J}$. The population begin fully susceptible, excluding the initial infective. If a type-i individual becomes infectious they contact type-j individuals at rate $\dfrac{\beta_i\kappa_j}{n}$ $(i,j\in\mathscr{J})$, where β_i, $\kappa_i \in [1,\infty)$ are parameters controlling the rate of activity of each type of individual. All individuals who become infected are infectious for a random period of time, according to a realisation of an $\text{Exp}(\gamma)$ random variable, where we take $\gamma = 1$ without loss of generality. Individuals are removed when their infectious period ends, after which they play no further role in the epidemic. The epidemic terminates when there are no infectious individuals remaining. This epidemic is a special case of the more general multitype households model with

proportionate global mixing, later studied in detail in Chapter 4 , in the absence of household structure. It is also a generalisation of the homogeneously mixing epidemic.

We apply a random time change in order to reach a closed-form expression for the asymptotic variance. For the original process, we write $\boldsymbol{x}^* = (x_1, x_2, \ldots, x_J, y_1, \ldots, y_J)$, with the first J elements containing the proportion of susceptibles of each type and the final J elements the proportion of infectives of each type. For $i \in \mathscr{J}$, let $\boldsymbol{l}_i^*$ ($\tilde{\boldsymbol{l}}_i^*$) denote the jump corresponding to a type-i infection (recovery). Letting $\boldsymbol{e}_i$ denote the unit vector with 1 in the i^{th} position, we have $\boldsymbol{l}_i^* = \boldsymbol{e}_{J+i} - \boldsymbol{e}_i$ and $\tilde{\boldsymbol{l}}_i^* = -\boldsymbol{e}_{J+i}$. The associated jump rates are given by

$$\beta^*_{\boldsymbol{l}_k^*}(\boldsymbol{x}^*) = \kappa_k x_k \sum_{j=1}^{J} \beta_j y_j, \quad k \in \mathscr{J},$$

and

$$\beta^*_{\tilde{\boldsymbol{l}}_k^*}(\boldsymbol{x}^*) = y_k, \qquad k \in \mathscr{J}.$$

Instead of considering the above jump rates, we apply a random time change to the process, dividing each jump rate by $\sum_{j=1}^{J} \beta_j y_j$. Under this random time change, we recover independent linear death processes for the proportions of susceptibles of each type, which do not depend on the infectivity processes. Thus, as in Section 3.4, we may initialise the process with the entire population being susceptible. Moreover, the stopping rule is a function of the susceptibility processes only. As a result, we restrict attention to the time-changed process $\boldsymbol{x} = (\tilde{x}_1, \tilde{x}_2, \ldots, \tilde{x}_J)$ and apply the framework of Section 3.2 to this process in order to calculate the asymptotic variance of the disease-induced herd immunity level. Written explicitly, due to the random time change, we now consider only the infectious jumps $\boldsymbol{l}_i = -\boldsymbol{e}_i$ with associated jump rate $\kappa_i \tilde{x}_i$. This yields drift function $\boldsymbol{F}(\boldsymbol{x}) = (f_1(\boldsymbol{x}), f_2(\boldsymbol{x}), f_J(\boldsymbol{x}))$ with

$$f_k(\boldsymbol{x}) = -\kappa_k \tilde{x}_k, \quad k \in \mathscr{J}. \tag{3.13}$$

The matrix of partial derivatives of $\boldsymbol{F}$, $\partial \boldsymbol{F}$, has elements

$$\frac{\partial f_k}{\partial \tilde{x}_m} = -\delta_{km}, \quad k,m \in \mathscr{J}. \tag{3.14}$$

Using (3.5), the infinitesimal variance function $\boldsymbol{G}(\boldsymbol{x})$ is given by

$$\boldsymbol{G}(\boldsymbol{x}) = \text{diag}\{\kappa_1\tilde{x}_1, \kappa_2\tilde{x}_2, \ldots, \kappa_J\tilde{x}_J\}.$$

The stopping rule corresponding to the population reaching herd immunity is given by

$$\varphi(\boldsymbol{x}) = \sum_{i=1}^{J} \beta_i\kappa_i\tilde{x}_i - 1.$$

The following theorem demonstrates a Gaussian approximation to the disease-induced herd immunity level in the multitype model with proportionate mixing.

Theorem 3.5. *Let $H_D^{(n)}$ denote the disease-induced herd immunity level in the mulitype model with proportionate mixing with a population of size n with rates $\beta_1, \beta_2, \ldots, \beta_J$ and $\kappa_1, \kappa_2 \ldots, \kappa_J$ and with $R_0 > 1$. Then, as $n \to \infty$,*

$$\sqrt{n}\left(H_D^{(n)} - h_D\right) \xrightarrow{\text{D}} N(0, \sigma^2), \tag{3.15}$$

where

$$\sigma^2 = \sum_{i=1}^{J} \gamma_i \left(\exp\{-\kappa_i\tau\} - \exp\{-2\kappa_i\tau\}\right) \left[1 - \frac{\beta_i\kappa_i\sum_{k=1}^{J}\kappa_k\gamma_k\exp(-\kappa_k\tau)}{\sum_{k=1}^{J}\beta_k\kappa_k^2\exp(-\kappa_k\tau)}\right]^2$$

and where τ is the unique solution in $(0,\infty)$ of

$$\sum_{i=1}^{J} \beta_i\kappa_i\gamma_i\exp(-\kappa_i t) - 1 = 0. \tag{3.16}$$

Proof. We consider the time-changed process defined above, noting that

$$\text{var}\left(H_D^{(n)}\right) = \text{var}\left(1 - \mathbf{1}_J \cdot \boldsymbol{X}_n(\tau_n)\right) = \text{var}\left(\mathbf{1}_J \cdot \boldsymbol{X}_n(\tau_n)\right).$$

It is clear from (3.14) that $\boldsymbol{F}$ has bounded derivatives in this case, from which it follows that $\boldsymbol{F}$ is Lipschitz continuous on any compact subset of $[0,1]^J$. It is also

clear that all partial derivatives of $\boldsymbol{F}$ are continuous. There are J possible jumps in this model, which are all unit vectors. Recall that $\beta_{l_k} = \kappa_k \tilde{x}_k$ for $k \in \mathscr{J}$, from which it is evident that

$$\sum_{l} |l|^2 \sup_{x \in K} \beta_l(x) \leq J \max\{\kappa_1, \kappa_2 \ldots, \kappa_J\} < \infty,$$

so that the conditions of Theorem 3.1 and Theorem 3.2 are satisfied. We next consider the conditions of Theorem 3.3 in turn. The deterministic equations implied by (3.4) give that

$$\frac{d\tilde{x}_i}{dt} = -\kappa_i \tilde{x}_i, \quad \tilde{x}_i(0) = \gamma_i, \quad i \in \mathscr{J},$$

from which we find

$$\tilde{x}_i(t) = \gamma_i \exp(-\kappa_i t), \quad i \in \mathscr{J}, t \geq 0.$$

Then $\varphi(\boldsymbol{x}(t)) = 0$ is equivalent to (3.16), which one can easily show has a unique solution $\tau \in (0, \infty)$ when $R_0 > 1$. (We are implicitly assuming that $R_0 > 1$ at the beginning of the epidemic, otherwise herd immunity would have already been achieved.) Moreover, it is clear that $\varphi(\boldsymbol{x})$ is continuously differentiable on $\mathbb{R}^J$, with $\varphi(\boldsymbol{x}(0)) = R_0 - 1 > 0$. Lastly, we have

$$\nabla \varphi(\boldsymbol{x}(\tau)) \cdot \boldsymbol{F}(\boldsymbol{x}(\tau)) = -\sum_{i=1}^{J} \beta_i \kappa_i^2 \tilde{x}_i(\tau) < 0,$$

so that conditions (i)-(iv) of Theorem 3.3 (and hence of Theorem 3.4) are satisfied. Taking $\boldsymbol{w} = \mathbf{1}_J$ in Theorem 3.4 shows that (3.15) holds for some σ^2, which we now calculate.

In the notation of Theorem 3.4 we require $\boldsymbol{w}\boldsymbol{A}$ and $\boldsymbol{\Sigma}(t)$, where $\boldsymbol{w} = \mathbf{1}_J$. Letting $\boldsymbol{A} = [a_{ij}]$, we use (3.9) to find

$$a_{ij} = \delta_{ij} - \frac{\beta_j \kappa_j \kappa_i \tilde{x}_i(\tau)}{\sum_{k=1}^{J} \beta_k \kappa_k^2 \tilde{x}_k(\tau)}, \quad i, j \in \mathscr{J}.$$

Then

$$[\boldsymbol{w}\boldsymbol{A}]_i = 1 - \frac{\beta_i \kappa_i \sum_{k=1}^{J} \kappa_k \tilde{x}_k(\tau)}{\sum_{k=1}^{J} \beta_k \kappa_k^2 \tilde{x}_k(\tau)}, \quad i \in \mathscr{J}.$$

We solve (3.10) to find $\Sigma(t)$, noting that all off-diagonal elements of $\Sigma(t)$ are equal to zero. Letting $\sigma_k^2(t) = \Sigma_{kk}(t)$ ($k \in \mathscr{J}$), we have

$$\sigma_k^2(t) = \kappa_k \tilde{x}_k(t) - \kappa_k \sigma_k^2(t), \quad k \in \mathscr{J}, t \geq 0,$$

which can be solved subject to the initial condition $\sigma_k^2(0) = 0$ to give

$$\sigma_k^2(t) = \gamma_k \left(\exp\{-\kappa_k t\} - \exp\{-2\kappa_k t\}\right), \quad k \in \mathscr{J}, t \geq 0.$$

Then

$$\Sigma(\tau) = \operatorname{diag}\{\sigma_1^2(\tau), \sigma_2^2(\tau), \ldots, \sigma_J^2(\tau)\}.$$

The asymptotic variance σ^2 is then, by Theorem 3.5, equal to

$$\begin{aligned}
\sigma^2 &= \sum_{i=1}^{J} \sigma_i^2(\tau) \left[1 - \frac{\beta_i \kappa_i \sum_{k=1}^{J} \kappa_k \gamma_k \tilde{x}_k(\tau)}{\sum_{k=1}^{J} \beta_k \kappa_k^2 \tilde{x}_k(\tau)}\right]^2 \\
&= \sum_{i=1}^{J} \gamma_i \left(\exp\{-\kappa_k \tau\} - \exp\{-2\kappa_k \tau\}\right) \left[1 - \frac{\beta_i \kappa_i \sum_{k=1}^{J} \kappa_k \gamma_k \exp(-\kappa_k \tau)}{\sum_{k=1}^{J} \beta_k \kappa_k^2 \exp(-\kappa_k \tau)}\right]^2,
\end{aligned}$$

as required. □

In Britton et al. [2020], the above model is considered with $J = 3$ activity levels, with 50% of individuals having standard activity, and 25% of individuals having half and double activity respectively. This corresponds to $\kappa_1 = 0.5$, $\kappa_2 = 1$ and $\kappa_3 = 2$ and proportions $\gamma_1 = 0.25$, $\gamma_2 = 0.5$ and $\gamma_3 = 0.25$. We take $\beta_i = c\kappa_i$ ($i = 1, 2, 3$), where the constant c is chosen to fix R_0 to a desired value. In Table 3.1 we give the results of implementing Theorem 3.5 numerically, reporting the value of h_D, the asymptotic variance σ^2 and the width w_n of the corresponding 95% confidence interval. We also provide $\hat{H}_D$ and $\hat{\sigma}^2$, simulation-based estimates of mean disease-induced herd immunity level and asymptotic variance respectively.

R_0	h_D	$\hat{H}_D$	σ^2	$\hat{\sigma}^2$	w_{10^3}	w_{10^6}
1.5	0.24300	0.24257	0.09978	0.09884	0.03916	0.00124
2	0.37672	0.37661	0.13405	0.13393	0.04539	0.00144
2.5	0.46332	0.46328	0.14638	0.14659	0.04743	0.00150
3	0.52485	0.52488	0.14967	0.14970	0.04796	0.00152
4	0.60784	0.60791	0.14592	0.14611	0.04735	0.00150
5	0.66221	0.66222	0.13779	0.13771	0.04602	0.00146

Table 3.1: Herd immunity level h_D and corresponding asymptotic variance σ^2, with the width w_n of a 95% confidence interval for $n = 10^3$ and $n = 10^6$ respectively, considering the activity level model of Britton et al. [2020] for several values of R_0. The simulated mean disease-induced herd immunity level $\hat{H}_D$ and asymptotic variance $\hat{\sigma}^2$ are also given, where for each R_0 we take 10^6 simulations of a major outbreak among a population of size 10^3. Values are rounded to 5 decimal places.

Note that the first column of Table 3.1 simply gives the disease-induced herd immunity level (cf. Britton et al. [2020], Table 1). It is clear that the asymptotic variance is low for this model. The herd immunity level established from deterministic equations is very accurate even for modest population sizes, and the fluctuation about the deterministic herd immunity level is small. It is clear from comparing σ^2 and $\hat{\sigma}^2$ that stochastic simulations of this model agree well in terms of the asymptotic variance from Theorem 3.5.

3.6 Households model with common household size

We now consider the Gaussian approximation to the disease-induced herd immunity level for the households model of 2.2.1 in the case of a common household size $m > 1$ and infectious period distribution $T_I \sim \text{Exp}(1)$, with no latent period ($T_E = 0$). For this model we take n as the number of households in the population, rather than the number of individuals, during the exposition. This choice is consistent with the framework of Section 3.2, but contrasts the notation of, for example, Section 2.4.4, which used n to denote the common household size. We again provide the jumps, their associated rates, and the stopping rule, so that the methods of Section 3.2 can be applied. There are $H = \frac{1}{2}m(m+3)$ possible states that a household can be in. We order states analogously to Section 2.2.3, so that the first state is $(m,0)$ and the final state is $(1,0)$. The first (second) element s_k (i_k) of the state with linear index k corresponds to the num-

ber of susceptibles (infectives) in that state. The set of possible household states is given by $\mathscr{H} = \{(s_k, i_k) : k = 1, 2, \ldots, H\}$. It is useful to additionally define

$$\mathscr{H}_{\text{susc}} = \{k \in \{1, 2, \ldots, H\} : s_k > 0\}$$

and

$$\mathscr{H}_{\text{inf}} = \{k \in \{1, 2, \ldots, H\} : i_k > 0\}$$

as the set of states corresponding to households with at least one susceptible and at least one infective respectively. We assume that, initially, all but a small fraction of households are in state 1; we thus assume that the epidemic begins with a small fraction of households which are not fully susceptible. The process of interest is $\boldsymbol{x} = (x_1, x_2 \ldots, x_H)$ corresponding to the fractions of households in each in state. In this case a closed-form expression of the asymptotic variance is not available; we show that the conditions of Theorem 3.4 hold, before implementing it numerically.

A household in a given state can move to one of at most two other states, corresponding to infection and recovery respectively occurring in that household. In order to construct the jumps, we let $t(n) = \dfrac{n(n+1)}{2}$ denote the n^{th} triangular number ($n \in \mathbb{Z}_+$). We define $g : \mathbb{N} \to \mathbb{N}$ by

$$\begin{aligned} g(i) &= \min\{v \in \mathbb{N} : t(v) \geq i\} \\ &= \left\lceil -\frac{1}{2} + \sqrt{\frac{1}{4} + 2i} \right\rceil, \end{aligned}$$

with the property that an infection in a household with linear index i gives rise to a household with linear index $i + g(i)$. The infectious jump $\boldsymbol{l}_i$ associated with state i is

$$\boldsymbol{l}_i = \boldsymbol{e}_{i+g(i)} - \boldsymbol{e}_i, \quad i \in \mathscr{H}_{\text{susc}}, \tag{3.17}$$

with the recovery jumps given by

$$\tilde{\boldsymbol{l}}_i = \boldsymbol{e}_{i+1} - \boldsymbol{e}_i, \quad i \in \mathscr{H}_{\text{inf}}. \tag{3.18}$$

The infectious jump associated with state k, $\boldsymbol{l}_k$, has jump rate

$$\beta_{\boldsymbol{l}_k}(\boldsymbol{x}) = \lambda_G I s_k x_k + \lambda_L s_k i_k x_k, \quad k \in \mathscr{H}_{\text{inf}}, \tag{3.19}$$

where $I = \dfrac{1}{m}\sum_{k=1}^{H} i_k x_k$ is the proportion of infectives in the population. The rates associated to recovery jumps are given by

$$\beta_{\tilde{\boldsymbol{l}}_k}(\boldsymbol{x}) = \gamma i_k x_k, \quad k \in \mathscr{H}_{\text{rec}}. \tag{3.20}$$

Then

$$\boldsymbol{F}(\boldsymbol{x}) = \sum_{k\in\mathscr{H}_{\text{inf}}} \boldsymbol{l}_k \beta_{\boldsymbol{l}_k}(\boldsymbol{x}) + \sum_{k\in\mathscr{H}_{\text{rec}}} \tilde{\boldsymbol{l}}_k \beta_{\tilde{\boldsymbol{l}}_k}(\boldsymbol{x}), \tag{3.21}$$

and $\boldsymbol{G}(\boldsymbol{x})$ can be calculated using (3.5). The stopping rule corresponds to $R_*(\boldsymbol{x}) = 1$, where $R_*(\boldsymbol{x})$ denotes R_* among the remaining susceptible population. Now

$$R_*(\boldsymbol{x}) = \frac{1}{\gamma}\sum_{v=0}^{m} p_v(\boldsymbol{x}) \left(1 - \frac{v}{m}\right) \mu_{m-v}(\lambda_L)\lambda_G,$$

where $p_v(\boldsymbol{x})$ is the proportion of households with v non-susceptible members ($v = 0, 1, \ldots, m$). We note that

$$p_v(\boldsymbol{x}) = \sum_{k=1+t(v)}^{t(v+1)} x_k \quad (v = 0, 1, \ldots, m),$$

so that

$$\varphi(\boldsymbol{x}) = \frac{\lambda_G}{\gamma}\left[\sum_{v=0}^{m}\left(1 - \frac{v}{m}\right)\mu_{m-v}(\lambda_L) \sum_{k=1+t(v)}^{t(v+1)} x_k\right] - 1. \tag{3.22}$$

In order to apply Theorem 3.4 to the present households setting, we must confirm that the required conditions hold, which motivates the following sequence of lemmas. The bounds used in the following lemmas are coarse, but sufficient for the required results to hold.

Lemma 3.6. *All of the jump rates $\beta_{\boldsymbol{l}}(\boldsymbol{x})$ defined in* (3.19) *and* (3.20) *are contin-*

uous and, for each compact $K \subset [0,1]^J$, we have

$$\sum_{\boldsymbol{l}} |\boldsymbol{l}|^2 \sup_{\boldsymbol{x}\in K} \beta_{\boldsymbol{l}}(\boldsymbol{x}) < \infty.$$

Proof. The continuity of each jump rate is clear from the definition after recalling that $I = \frac{1}{m}\sum_{k=1}^{H} i_k x_k$. There are at most $2H$ possible transitions, although some of these have a jump rate of zero associated to them. Since each jump has at most two non-zero elements, and by appropriately bounding (3.19) and (3.20), we find

$$\begin{aligned}\sum_{\boldsymbol{l}} |\boldsymbol{l}|^2 \sup_{\boldsymbol{x}\in K} \beta_{\boldsymbol{l}}(\boldsymbol{x}) &\leq \sum_{i=1}^{2H} 2\max\{\lambda_G m + \lambda_L m^2, \gamma m\} \\ &= 4H\max\{\lambda_G m + \lambda_L m^2, \gamma m\} \\ &< \infty,\end{aligned}$$

as required. □

Lemma 3.7. *The function $\boldsymbol{F}(\boldsymbol{x})$ defined in* (3.21) *is Lipschitz continuous on any compact subset of $[0,1]^J$ and has continuous partial derivatives.*

Proof. We compute $\partial \boldsymbol{F}$ and show that all elements of $\partial \boldsymbol{F}$ are bounded and continuous, which is sufficient to establish the above claim. First, note that

$$\frac{\partial \beta_{\boldsymbol{l}_k}}{\partial x_j} = \lambda_G I s_k \delta_{jk} + \frac{\lambda_G}{m} i_j s_k x_k + \lambda_L s_k i_k \delta_{jk}, \quad k \in \mathscr{H}_{\text{inf}}, j = 1,2\ldots,H,$$

and

$$\frac{\partial \beta_{\tilde{\boldsymbol{l}}_k}}{\partial x_j} = \gamma i_k \delta_{jk}, \quad k \in \mathscr{H}_{\text{rec}}, j = 1,2\ldots,H,$$

from which we can calculate $\partial \boldsymbol{F}$. For ease of exposition, we write $\boldsymbol{l}_u$ for the infectious jump from state u, with the convention that $\boldsymbol{l}_u = \boldsymbol{0}_H$ if $u \notin \mathscr{H}_{\text{inf}}$. We

treat recovery jumps analogously. Then the columns of $\partial \boldsymbol{F}$ are given by

$$\begin{aligned}\frac{\partial \boldsymbol{F}}{\partial x_j} &= \sum_{u=1}^{H} \boldsymbol{l}_u \left(\lambda_G I s_u \delta_{uj} + \frac{\lambda_G}{m} i_j s_u x_u + \lambda_L i_u s_u \delta_{ju} \right) + \sum_{u=1}^{H} \tilde{\boldsymbol{l}}_u \gamma i_u \delta_{ju} \\ &= \boldsymbol{l}_j \left(\lambda_G s_j I + \lambda_L s_j i_j \right) + \frac{\lambda_G}{m} i_j \sum_{u=1}^{H} \boldsymbol{l}_u s_u x_u + \tilde{\boldsymbol{l}}_j \gamma i_j, \quad j = 1, 2, \ldots, H. \quad (3.23)\end{aligned}$$

Then $\partial \boldsymbol{F} = [f_{ij}]$ can be computed by considering the i^{th} element of (3.23) and recalling (3.17) and (3.18). It is clear that all of these partial derivatives are continuous. Moreover, each element f_{ij} of $\partial \boldsymbol{F}$ satisfies

$$|f_{ij}| \leq 2\lambda_G m + \lambda_L m^2 + \gamma m < \infty, \quad i, j = 1, 2, \ldots, H,$$

so that all first partial derivatives of $\boldsymbol{F}$ are bounded. This is sufficient to establish that $\boldsymbol{F}$ is Lipschitz continuous. □

Lemma 3.8. *Let $\varphi(\boldsymbol{x})$ be defined as in* (3.22) *and let τ be such that $\varphi(\boldsymbol{x}(\tau)) = 1$. Then $\varphi(\boldsymbol{x})$ is continuously differentiable on $\mathbb{R}^H$ and*

$$\nabla \varphi(\boldsymbol{x}(\tau)) \cdot \boldsymbol{F}(\boldsymbol{x}(\tau)) < 0.$$

Proof. Letting $\varphi'_j = \dfrac{\partial \varphi}{\partial x_j}$, we differentiate (3.22) to find

$$\begin{aligned}\varphi'_j &= \frac{\lambda_G}{\gamma} \sum_{v=0}^{m} \left(1 - \frac{v}{m}\right) \mu_{m-v}(\lambda_L) \sum_{k=t(v)+1}^{t(v+1)} \delta_{jk} \\ &= \frac{\lambda_G}{\gamma} \sum_{v=0}^{m} \left(1 - \frac{v}{m}\right) \mu_{m-v}(\lambda_L) \mathbb{1}_{\{t(v)+1 \leq j \leq t(v+1)\}}, \quad j = 1, 2, \ldots, H,\end{aligned}$$

from which it is clear that $\varphi(\boldsymbol{x})$ is continuously differentiable on $\mathbb{R}^H$. Note that $\varphi'_1 > \varphi'_2$ and $\varphi'_j \geq \varphi'_{j+1}$ for $j = 1, 2, \ldots, H-1$. Taking the required dot product

and using (3.21), we have

$$\begin{aligned}
\nabla\varphi(\boldsymbol{x}(\tau))\cdot\boldsymbol{F}(\boldsymbol{x}(\tau)) &= \sum_{k\in\mathscr{H}_{\text{inf}}}\beta_{l_k}(\boldsymbol{x}(\tau))\nabla\varphi(\boldsymbol{x}(\tau))\cdot\boldsymbol{l}_k + \sum_{k\in\mathscr{H}_{\text{rec}}}\beta_{\tilde{l}_k}(\boldsymbol{x}(\tau))\nabla\varphi(\boldsymbol{x}(\tau))\cdot\tilde{\boldsymbol{l}}_k \\
&= \sum_{k\in\mathscr{H}_{\text{inf}}}\beta_{l_k}(\boldsymbol{x}(\tau))\nabla\varphi(\boldsymbol{x}(\tau))\cdot(\boldsymbol{e}_{k+g(k)}-\boldsymbol{e}_k) \\
&\quad + \sum_{k\in\mathscr{H}_{\text{rec}}}\beta_{\tilde{l}_k}(\boldsymbol{x}(\tau))\nabla\varphi(\boldsymbol{x}(\tau))\cdot(\boldsymbol{e}_{k+1}-\boldsymbol{e}_k) \\
&= \sum_{k\in\mathscr{H}_{\text{inf}}}\beta_{l_k}(\boldsymbol{x}(\tau))\left\{\varphi'_{k+g(k)}-\varphi'_k\right\} + \sum_{k\in\mathscr{H}_{\text{rec}}}\beta_{\tilde{l}_k}(\boldsymbol{x}(\tau))\left\{\varphi'_{k+1}-\varphi'_k\right\} \\
&< 0,
\end{aligned}$$

as required.

□

We now apply the above lemmas with a view toward a Gaussian approximation for the disease-induced herd immunity level in the households model with common household size.

Theorem 3.9. *Consider the households epidemic model with n households of size m ($m > 1$) and with (λ_G, λ_L) such that $R_* > 1$. Let $H_D^{(n)}$ denote the disease-induced herd immunity level for this model and let h_D denote the corresponding quantity for the limiting deterministic model. Let $\tau \in (0,\infty)$ denote the unique solution to $\varphi(\boldsymbol{x}(t)) = 0$ and let $\Sigma(t)$ denote the solution to* (3.10)*. Then, as $n \to \infty$,*

$$\sqrt{n}\left(H_D^{(n)} - h_D\right) \xrightarrow{\text{D}} N(0,\sigma^2), \tag{3.24}$$

where

$$\sigma^2 = \frac{1}{m^2}\sum_{i\in\mathscr{H}_{inf}}\sum_{j\in\mathscr{H}_{inf}} s_i s_j \sigma_{ij}^2, \tag{3.25}$$

and

$$\sigma_{ij}^2 = \Sigma_{ij}(\tau), \quad i,j = 1,2,\ldots,H.$$

Proof. We apply the framework of Section 3.2 to the present model, using the jumps and jump rates defined by (3.17) - (3.20). It is clear that $\tau < \infty$, since any epidemic run to termination always leaves the remaining susceptible population subcritical. Since the population begin supercritical by assumption, we have

$\varphi(\boldsymbol{x}(0)) = R_* - 1 > 0$. The other conditions of Theorem 3.3 (and hence of Theorem 3.4) are satisfied owing to Lemmas 3.6 - 3.8, which establishes (3.24). Note that $\Sigma(\tau)$ must be computed numerically in this case. The proportion of susceptibles $S(t)$ at time t satisfies $S(t) = \sum_{i \in \mathscr{H}_{\text{inf}}} s_i x_i(t)$, and $h_D = 1 - S(\tau)$. Applying Theorem 3.4 with $[\boldsymbol{w}]_i = \frac{1}{m} s_i$ $(i = 1, 2, \ldots, H)$ then establishes the asymptotic variance given in (3.25). □

In this case we cannot derive an explicit expression for the asymptotic variance of the disease-induced herd immunity level, although we can still compute it numerically - see Table 3.2. When $\lambda_L \to \infty$, households jump rapidly from a single member being infected to all members being infected. Thus $\lambda_L \to \infty$, as well as $\lambda_L = 0$, can be viewed as homogeneously mixing epidemics, implying that the asymptotic variance is zero in both of these cases.

m	$\sigma^2_{\max}$	$\hat{\sigma}^2$	R_0^*	p_L^*
2	0.00755	0.00767	3.52	0.72
3	0.00999	0.01062	3.18	0.64
4	0.01132	0.01069	2.92	0.57
5	0.01221	0.01215	2.71	0.51

Table 3.2: The largest asymptotic variance $\sigma^2_{\max}$ of the disease-induced herd immunity level achieved in the case of a common household size $m \in \{2,3,4,5\}$ when $(p_L, R_0) \in [0,1] \times (1,25]$, together with the parameter values (p_L^*, R_0^*) at which this maximum is attained. Additionally provided are stochastic realisations $\hat{\sigma}^2$ of the variance of the disease-induced herd immunity level, taking 10^3 simulations of 10^3 households with $(p_L, R_0) = (p_L^*, R_0^*)$. Variances are rounded to 5 decimal places and parameter values are rounded to 2 decimal places.

We draw two main conclusions from Table 3.2. The first is that the asymptotic variance of the disease-induced herd immunity level is small, which can be seen in the first column (which gives the largest asymptotic variance as R_0 and p_L vary). Comparing the first and second columns of Table 3.2, we note good agreement between the asymptotic variance from numerical calculations and stochastic simulations; we expect these values to agree more closely for a larger population size, i.e. a larger number of households, although such simulations are still subject to Monte Carlo error.

3.7 Discussion

We have computed a Gaussian approximation to the disease-induced herd immunity level in the multitype model with proportionate mixing and in the households model with common household size. For the multitype model with proportionate mixing we have derived an explicit expression for the asymptotic variance, using a random time change. The calculated asymptotic variances agree with the corresponding Monte Carlo estimates from simulations and, for both models we consider, we have observed that the asymptotic variance is small.

Comparing Table 3.1 and Table 3.2, we observe that the asymptotic variance is small in both cases. In particular, the asymptotic variance for the households model is even smaller than that of the multitype model with proportionate mixing, for models calibrated by R_0. It appears, therefore, that the introduction of multiple types has a greater influence on the asymptotic variance of the disease-induced herd immunity level than the introduction of household structure. In any case, it is clear that the deterministic approximation to the disease-induced herd immunity level is good, even for modest population sizes (observe, for example, the confidence intervals provided in Table 3.1).

There are several natural extensions to the work outlined above. It is possible to extend the theory of Section 3.6 to the case of unequal household sizes, although the number of ODEs required would grow sharply. We expect that the asymptotic variance would be larger for a model with variable household size than for a model with common household size, although we still expect this variance to be small. It is also possible, in principle, to extend this calculation to the multitype households model with proportionate global mixing; note that the number of ODEs required would soon become very large, and we would again expect the asymptotic variance to be small. Whilst the use of a random time change to reach a closed-form expression for the asymptotic variance is not available, we could implement the calculations numerically and explore the asymptotic variance via that means.

4 Multitype households model with proportionate global mixing

4.1 Introduction

In this chapter we consider the stochastic SIR (susceptible $\rightarrow$ infective $\rightarrow$ recovered) multitype households epidemic model of Ball and Lyne [2001] with a further assumption on global contact rates, known as proportionate mixing. We assume that each individual in the population has a type, belonging to $\mathscr{J} = \{1,2,\ldots,J\}$, and mixes at two levels: globally and locally, the rates at which they mix being dependent on their type. The assumption of proportionate global mixing is made explicit in the model definition. Such an assumption gives rise to a reduction in the type space of associated branching processes and limiting equations, leading to more explicit expressions for quantities such as reproduction numbers. Using an embedding construction, we derive a central limit theorem for the final outcome of an epidemic which takes off. All of the calculations that follow are made simpler under the assumption of proportionate global mixing, owing to the underlying processes being effectively one-dimensional rather than J-dimensional.

In a model with proportionate global mixing, the type of an infectious individual does not affect the probability that they contact an individual of a given type (given that they make a global contact). The type of an infectious individual does, however, affect the rate at which these global contacts are made. Models with proportionate mixing are of interest for several reasons. A main motivation in the context of herd immunity is that, under proportionate global mixing, certain individuals mix more with the population at large than others. In this sense, a first wave of an epidemic can be thought of as a "targeted vaccine", in which more active individuals are immunised first, more quickly reducing the overall population susceptibility - see Britton et al. [2020]. Detailed study of the multitype households model with proportionate global mixing could, therefore, inform questions pertaining to herd immunity. Some vaccine action models, including that of Becker and Starczak [1997], Section 3.1, can be viewed in the

context of proportionate mixing. In such a model, individuals have their susceptibility and infectivity reduced by factors A and B respectively. In general (A,B) is a random vector but, under the non-random vaccine assumption (see Ball and Lyne [2006]), one assumes that all vaccinated individuals have their infectivity and susceptibility rates reduced by the same amount, i.e. $P((A,B)=(a,b))=1$ for some vector (a,b). Another example of interest is that of the leaky vaccine, in which $b=1$; vaccinating a given individual helps to protect them from infection, but does not limit their ability to infect others in the event that they become infected – see Halloran et al. [1992].

It has been established previously that, for deterministic epidemic models, model analysis becomes simpler under the assumption of proportionate mixing – see, for example, Gart [1968], Section 3. In this chapter, we show that the analysis of the *stochastic* multitype households SIR epidemic model is considerably simpler if the global (between-household) infection rates follow proportionate mixing. No such constraint is placed upon the local (within-household) infection rates. Under the assumption of proportionate global mixing, the branching process that approximates the early stages of an epidemic is effectively single-type, leading to considerable simplification in the calculation of reproduction numbers, the probability of a major outbreak and the early exponential growth rate of a major outbreak. (The branching process approximating the early stages of an epidemic becomes single-type by applying standard results pertaining to aggregation and superposition of the Poisson processes which govern global infection.) The assumption of proportionate global mixing also simplifies the calculation and proof of properties of the final outcome of a major epidemic, since the index set in key processes in the embedding argument is scalar rather than J-dimensional. We prove a multivariate central limit theorem for a vector of final state random variables (see Ball and O'Neill [1999]) in the event of a major outbreak. These final state random variables may be quite general and include, for example, the number of individuals of each type infected by the epidemic and the number of households infected (see Section 4.6 for further examples).

We also fill, in the present proportionate global mixing setting, a couple of gaps in Ball and Lyne [2001]. First, the definition of a major outbreak in

that paper, referred to there as a global epidemic, is not fully satisfactory in that it is based on the limiting branching process. Here, an epidemic among a population of m households is defined to be a major outbreak if it infects at least $\log(m)$ households. The choice of $\log(m)$ is just for convenience; $\log(m)$ can be replaced by any function $f(m)$ satisfying $f(m) \to \infty$ and $m^{-1}f(m) \to 0$ as $m \to \infty$. Indeed, for any specific parameter values, we can replace $\log(m)$ by cm but the choice of the constant $c > 0$ depends on those parameter values. Secondly, the proof of the central limit theorem for the outcome of a major outbreak in Ball and Lyne [2001] ignores the conditioning on a major outbreak when solving the crossing problem for the embedded process, though it yields the correct asymptotic mean and variance. Here, we exploit a lack-of-memory property in the process underlying the embedding, which gives a simple, novel and widely applicable solution to this conditioning problem.

This chapter is structured as follows. We begin in Section 4.2 with a description of the multitype households epidemic model, before defining the assumption on global mixing rates which represents proportionate global mixing. In Section 4.3 we consider the threshold behaviour of the model, demonstrating the type space reduction in the branching processes associated with the calculations of several reproduction numbers (viz. R_*, R_I and R_0), as well as the early exponential growth rate r. We then, in Section 4.4, find, in the event of a global outbreak, limiting equations for the fractions of individuals of each type that are ultimately infected as the population size goes to infinity. The key quantity we study is the severity (sum of infectious periods of those infected in the epidemic) in order to be able to consider a univariate process in this derivation. As a result, we reduce the calculation of the final size from a J-dimensional system of nonlinear equations to a single nonlinear equation.

In Section 4.5 we provide an embedding construction (following Sellke [1983]) of the epidemic process; such a construction differs from the "true" epidemic in how the epidemic is initiated. We derive convergence results for the severity process under this construction. In Section 4.6 these convergence results are used to establish a multivariate central limit theorem for so-called general final state random variables, first introduced in Ball and O'Neill [1999], for the embedded

process. In Section 4.7 we define a major outbreak for the multitype SIR households model and show that the probability of a major outbreak converges to the non-extinction probability of an appropriate approximating branching process. In Section 4.8 we connect the embedding construction to our model, deriving a central limit theorem (Theorem 4.23) for final outcome quantities conditional upon a major outbreak. The index set of the underlying processes are one-dimensional, owing to proportionate mixing. Moreover, the mean vector and covariance matrix of Theorem 4.23 are analogous to, but more explicit than, that of Ball and Lyne [2001], Theorem 5.3, and the related proofs are more easily presented. In Sections 4.9 and 4.10 we highlight the special cases of highly locally infectious disease and all households size one respectively, providing expressions for the asymptotic variance matrix in those cases in terms of the root of a nonlinear equation. We conclude this chapter in Section 4.11 by briefly discussing these results as well as possible extensions of this work.

4.2 Model definition

We consider a multitype SIR model, denoted by $\mathscr{E}$, with household structure in a closed population. We begin by defining the population structure. Suppose that households contain at most $n_{\max}$ individuals, where $n_{\max} < \infty$. Suppose further that there are J types of individuals in the population, with types belonging to $\mathscr{J} = \{1,2,\ldots,J\}$. Define the *household category* $\boldsymbol{n}$ as the J-tuple containing the number of members of each type in that household. Then, with $\mathbb{Z}_+ = \{0,1,\ldots\}$, let

$$\mathscr{N} = \{\boldsymbol{n} = (n_1, n_2, \ldots, n_J) \in \mathbb{Z}_+^J : 1 \leq \sum_{j=1}^{J} n_j \leq n_{\max}\} \tag{4.1}$$

denote the (finite) collection of possible household categories. Suppose that there are $m_{\boldsymbol{n}}$ households of category $\boldsymbol{n}$ ($\boldsymbol{n} \in \mathscr{N}$) with $m = \sum_{\boldsymbol{n}\in\mathscr{N}} m_{\boldsymbol{n}}$ households in total. The collection $\boldsymbol{m} = \{m_{\boldsymbol{n}} : \boldsymbol{n} \in \mathscr{N}\}$ is referred to as the *household category distribution*. Writing $\|\boldsymbol{n}\| = \sum_{i=1}^{J} n_i$, the population size ($N$) is then given by

$$N = \sum_{\boldsymbol{n}\in\mathscr{N}} \|\boldsymbol{n}\| m_{\boldsymbol{n}},$$

with the number of type-i individuals given by

$$N_i = \sum_{\boldsymbol{n} \in \mathscr{N}} n_i m_{\boldsymbol{n}}, \qquad i \in \mathscr{J}.$$

The probability an individual chosen uniformly at random from the population is type i is then $\gamma_i = N_i/N$ ($i \in \mathscr{J}$). We assume that $\min_{i \in \mathscr{J}} \gamma_i > 0$. The proportion of households of category $\boldsymbol{n}$ given by $\theta_{\boldsymbol{n}} = m_{\boldsymbol{n}}/m$ ($\boldsymbol{n} \in \mathscr{N}$). Finally, we denote, by $\alpha_i(\boldsymbol{n})$, the probability an individual chosen uniformly at random among all type-i individuals resides in a household of category $\boldsymbol{n}$, which satisfies

$$\alpha_i(\boldsymbol{n}) = \frac{n_i m_{\boldsymbol{n}}}{N_i}, \qquad i \in \mathscr{J}, \boldsymbol{n} \in \mathscr{N}.$$

The model has infectious dynamics as follows. Individuals begin susceptible (other than a small number of initial infectives which we specify more precisely when necessary) until they are contacted by an infective, at which point they immediately become infective; there is no latent period in the model. (Most of our results are concerned with the final outcome, the distribution of which is invariant to the introduction of a latent period.) For $i \in \mathscr{J}$, a type-i individual, upon being infected, remains infectious for a random time $T_I^{(i)}$, having an arbitrary but specified distribution, with mean infectious period length $\mu_I^{(i)}$. During their infectious period, a type-i individual globally contacts any given type-j individual according to the points of a Poisson process with rate λ_{ij}^G/N (cf. λ_{ij}^G/N_j in Ball and Lyne [2001]). Local contacts occur from a type-i individual to any given type-j individual *within their household* according to the points of a Poisson process with rate λ_{ij}^L. Let $\Lambda^G = [\lambda_{ij}^G]$ and $\Lambda^L = [\lambda_{ij}^L]$. Individuals recover at the end of their infectious period and play no further role in the epidemic. Finally, the Poisson processes governing infections, as well as the random variables governing infectious period lengths, are all assumed to be mutually independent.

We make a further assumption on global mixing rates, by assuming that $\beta_1, \beta_2, \ldots, \beta_J > 0$ and $\kappa_1, \kappa_2, \ldots, \kappa_J > 0$ exist such that $\lambda_{ij}^G = \beta_i \kappa_j$. This is referred to as *proportionate mixing*; under this assumption certain derivations, of quantities of interest such as reproduction numbers, growth rates and the final

size of the epidemic, are greatly simplified. The β_i quantities can be interpreted as varying infectivity of individuals, with larger β_i giving rise to a higher rate of infecting others. Similarly, a higher value for κ_i indicates greater susceptibility to infection. We concatenate these susceptibility and infectivity rates into vectors $\boldsymbol{\kappa}$ and $\boldsymbol{\beta}$ respectively. We suppress the dependence of $\mathscr{E}$ on $\boldsymbol{m}$, $\boldsymbol{\beta}$ and $\boldsymbol{\kappa}$ for ease of exposition throughout this Chapter.

It is helpful to also introduce a single-household epidemic model for a category-$\boldsymbol{n}$ household ($\boldsymbol{n} \in \mathscr{N}$), in which there are $\text{Bin}(n_i, 1-\pi_i)$ initial (globally infected) infectives of type i ($i \in \mathscr{J}$) and with local infection governed by Λ^L. This epidemic, which we denote by $\mathscr{E}_{\boldsymbol{n}}(\Lambda^L, \boldsymbol{\pi})$, is studied in Addy et al. [1991] and the joint generating function for the size and severity of this epidemic is derived in Ball and Lyne [2001], Section 3. Letting $\boldsymbol{a} = (a_1, a_2, \ldots, a_J)$, we also introduce the epidemic model $\mathscr{E}_{\boldsymbol{n},\boldsymbol{a}}(\Lambda^L)$ in which there are a_i initial infectives of type i ($i \in \mathscr{J}$) in a category-$\boldsymbol{n}$ household with no outside infection and with local infection governed by Λ^L. Letting $\boldsymbol{e}_i$ denote the i^{th} standard basis vector of $\mathbb{R}^J$, we write $\mu_{\boldsymbol{n},i,j}(\Lambda^L)$ for the mean number of type-j individuals infected in $\mathscr{E}_{\boldsymbol{n},\boldsymbol{e}_i}(\Lambda^L)$, including the initial infective if $i = j$. The epidemic model $\mathscr{E}_{\boldsymbol{n},\boldsymbol{e}_i}(\Lambda^L)$ corresponds to the case of a single initial infective of type i.

We proceed by considering some properties and important quantities associated to $\mathscr{E}$, making the connection back to the general multitype households model where appropriate.

4.3 Threshold behaviour

We now consider several reproduction numbers associated to the epidemic, as well as the early exponential growth rate. The calculation of each of these reproduction numbers involves considering an appropriate branching process, coupled to the early stages of the epidemic process. In the following derivations, we assume there is one initial infective, chosen uniformly at random from the population, and that the number of households (m) is large. Before turning attention to the quantities of interest, we introduce a supporting lemma which will be applied regularly in the derivations that follow.

Lemma 4.1. *Assuming m is large, a typical individual who is contacted globally*

in the early stages of $\mathscr{E}$ has type j with probability P_j given by

$$P_j = \frac{\kappa_j \gamma_j}{\sum_{k=1}^{J} \kappa_k \gamma_k}, \qquad j \in \mathscr{J}.$$

Proof. Consider a typical type-i infective in the early stages of the epidemic. This individual will contact type-j individuals at rate $\rho_{ij} = \beta_i \kappa_j N_j / N = \beta_i \kappa_j \gamma_j$. Their total rate of infection, ρ_i, calculated by aggregating the Poisson processes associated with infection to type-j members of the population is then given by

$$\rho_{ij} = \sum_{j=1}^{J} \beta_i \kappa_j \gamma_j, \qquad i, j \in \mathscr{J}.$$

We have J Poisson processes which run concurrently, with the points in these Poisson processes corresponding to the type-i infective infecting individuals of each type. For $j \in \mathscr{J}$, the probability that a type-j individual is infected (P_j, say) is given by the probability that the j^{th} Poisson process is the first to produce a point. Thus, by the superposition of Poisson processes (see Kingman [1993], Section 2.2) we have

$$\begin{aligned}
P_j &= \int_0^\infty \rho_{ij} e^{-\rho_{ij} s} \times \prod_{k \neq j} e^{-\rho_{ik} s} ds \\
&= \int_0^\infty \rho_{ij} e^{-\rho_{ij} s} \times e^{-\sum_{k \neq j} \rho_{ik} s} ds \\
&= \int_0^\infty \rho_{ij} e^{-\sum_{k=1}^{J} \rho_{ik} s} ds \\
&= \frac{\rho_{ij}}{\sum_{k=1}^{J} \rho_{ik}} \\
&= \frac{\beta_i \kappa_j \gamma_j}{\sum_{k=1}^{J} \beta_i \kappa_k \gamma_k} \\
&= \frac{\kappa_j \gamma_j}{\sum_{k=1}^{J} \kappa_k \gamma_k},
\end{aligned}$$

which is independent of i and establishes the lemma. □

An immediate consequence of Lemma 4.1 is that we may assume $\sum_{j=1}^{J} \kappa_j \gamma_j = 1$ without loss of generality - an assumption we make in the sequel. In the original statement for proportionate mixing λ_{ij}^{G} is defined up to a multiplicative constant; the above assumption ensures that the model is fully parameterised and

specifies the scaling used.

4.3.1 Households-based reproduction number R_*

We consider a single-type branching process approximation for the proliferation of infected households in the early stages of the epidemic as follows. Each individual in the branching process corresponds to an infected household in the epidemic. Provided m is sufficiently large, every global contact made in the early stages of $\mathscr{E}$ is with a previously uninfected household. The mean of the offspring distribution of this branching process, R_*, is a threshold parameter for the present epidemic model. This is because, analogously to the households epidemic model, in the limit $m \to \infty$, the epidemic takes off (infecting at least $\log(m)$ households) with non-zero probability if and only if $R_* > 1$. We henceforth refer to this situation as a major outbreak.

Each individual in the branching process corresponds to an infected household in the epidemic, with the first generation of the branching process corresponding to the household in the epidemic in which the initial infective resides. Consider a fully susceptible household which is contacted globally by one of its individuals becoming infected. The probability this individual is type j is $\kappa_j\gamma_j$, by Lemma 4.1 (which facilitates the use of a single-type branching process and requires the proportionate mixing assumption in order to hold). Given that the individual is type j, they will reside in a household of category $\boldsymbol{n}$ with probability $\alpha_j(\boldsymbol{n})$. In the approximating branching process, every global contact is assumed to be with an individual in an otherwise fully susceptible household. This infected individual then begins a single-household epidemic which infects, on average, $\mu_{\boldsymbol{n},j,k}(\Lambda^L)$ type-k individuals. These type-k individuals then each mix globally for random time with mean $\mu_I^{(k)}$ at rate $\sum_{l=1}^{J}\beta_k\kappa_l\gamma_l = \beta_k$. Putting this together and applying Wald's identity for multitype SIR epidemics (Lemma B.2) yields that the expected number of households arising from a single newly-infected household (corresponding to the mean number of offspring

from a given individual in the approximating branching process) is

$$\begin{aligned} R_* &= \sum_{j=1}^{J} \kappa_j \gamma_j \sum_{\boldsymbol{n} \in \mathscr{N}} \alpha_j(\boldsymbol{n}) \sum_{k=1}^{J} \mu_{\boldsymbol{n},j,k}(\Lambda^L) \sum_{l=1}^{J} \beta_k \kappa_l \gamma_l \mu_I^{(k)} \\ &= \sum_{j=1}^{J} \kappa_j \gamma_j \sum_{\boldsymbol{n} \in \mathscr{N}} \alpha_j(\boldsymbol{n}) \sum_{k=1}^{J} \mu_{\boldsymbol{n},j,k}(\Lambda^L) \mu_I^{(k)} \beta_k \sum_{l=1}^{J} \kappa_l \gamma_l \\ &= \sum_{j=1}^{J} \kappa_j \gamma_j \sum_{\boldsymbol{n} \in \mathscr{N}} \alpha_j(\boldsymbol{n}) \sum_{k=1}^{J} \mu_{\boldsymbol{n},j,k}(\Lambda^L) \mu_I^{(k)} \beta_k, \end{aligned} \tag{4.2}$$

where the final line follows from $\sum_{j=1}^{J} \kappa_j \gamma_j = 1$. This expression for R_* coincides exactly with the trace of the matrix defined in Ball et al. [2004], Section 2.2, which becomes a matrix of rank one under the assumption of proportionate global mixing. We thus reach the same expression for R_*, but using a single-type branching process rather than a J-type branching process.

4.3.2 Individual-based reproduction number R_I

Define the primary case in a household as the initial (globally contacted) infective and define secondary cases as any individuals infected locally in the ensuing single-household epidemic. We next calculate the individual-based reproduction number R_I, in which we assume that there is one primary case in any infected household, so all other individuals infected in that household are secondary cases and are attributed to the primary case. This approach is used to calculate R_I in the households model in Becker and Dietz [1995], as well as in Ball et al. [1997]. In the general multitype households model, such an approach requires a multitype branching process and, consequently, a mean matrix with dimension $2J \times 2J$, corresponding to primary and secondary infectives of each type. Under proportionate global mixing, however, this reduces to a matrix with dimension $(J+1) \times (J+1)$ and sufficient structure such that the eigenvalues can be calculated explicitly.

We begin by defining the multitype branching process of interest in more detail. Class-0 individuals in the branching process correspond to primary cases within a household (viz. the first member of the household to be infected globally). For $i \in \mathscr{J}$, class-i individuals in the branching process correspond to

secondary cases in the epidemic who are type i. We calculate the mean matrix ($\boldsymbol{M}_I$) for this multitype branching process. As convention, we assume that the first row and column of $\boldsymbol{M}_I$ refer to primary cases. The remaining J rows and columns refer to secondary cases, which must be typed as the proportionate mixing assumption applies only to global infection. Let $\boldsymbol{M}_I = [m_{ij}]$. Since secondary cases do not result in further secondary cases, we have $m_{ij} = 0$ when $i, j \geq 2$. Then $\boldsymbol{M}_I$ takes the partitioned form

$$\boldsymbol{M}_I = \begin{pmatrix} a & \boldsymbol{u}^\top \\ \boldsymbol{v} & \mathbf{0}_{J\times J} \end{pmatrix}.$$

Here a is the mean number of primary cases generated by a typical primary case. The vector $\boldsymbol{u} = (u_1, u_2, \ldots, u_J)^\top$ has i^{th} element given by the mean number of secondary cases of type i generated by a typical primary case. Similarly, the i^{th} element of $\boldsymbol{v} = (v_1, v_2, \ldots, v_J)^\top$ contains the mean number of primary cases generated by a typical secondary case of type i.

We now calculate the quantities in $\boldsymbol{M}_I$ in turn, beginning with a. A type-j individual will infect type-k individuals globally according to points of a Poisson process, with rate $\beta_j \kappa_k \gamma_k$, run for mean time $\mu_I^{(j)}$. The mean number of type-k individuals created by a typical type-j individual is thus $\beta_j \kappa_k \gamma_k \mu_I^{(j)}$. Each globally contacted individual is type j with probability $\kappa_j \gamma_j$ (owing to Lemma 4.1), whence

$$\begin{aligned} a &= \sum_{j=1}^{J} \kappa_j \gamma_j \sum_{k=1}^{J} \beta_j \kappa_k \gamma_k \mu_I^{(j)} \\ &= \sum_{j=1}^{J} \beta_j \kappa_j \gamma_j \mu_I^{(j)} \sum_{k=1}^{J} \kappa_k \gamma_k \\ &= \sum_{j=1}^{J} \beta_j \kappa_j \gamma_j \mu_I^{(j)}. \end{aligned}$$

We calculate $\boldsymbol{v}$ similarly. Consider a typical type-k secondary infective, who will generate primary cases by mixing globally. Then

$$\begin{aligned} v_k &= \sum_{j=1}^{J} \beta_k \kappa_j \gamma_j \mu_I^{(k)} \\ &= \beta_k \mu_I^{(k)}, \qquad k \in \mathscr{J}. \end{aligned}$$

We next compute $\boldsymbol{u}$ by considering the mean number of secondary cases generated by a typical primary case. A typical type-j individual in a category-$\boldsymbol{n}$ household will have all secondary cases attributed to them; such an individual contributes $\mu_{\boldsymbol{n},j,k}(\Lambda^L) - \delta_{jk}$ type-k secondary cases on average. (Here δ_{jk} denotes the usual Kronecker delta and accounts for the case where the initial infective is also type k.) Applying Lemma 4.1 and the definition of $\alpha_j(\boldsymbol{n})$ then yields

$$u_k = \sum_{j=1}^{J} \kappa_j \gamma_j \sum_{\boldsymbol{n} \in \mathscr{N}} \alpha_j(\boldsymbol{n}) \{\mu_{\boldsymbol{n},j,k}(\Lambda^L) - \delta_{jk}\}, \qquad k \in \mathscr{J},$$

which concludes the calculation of the mean matrix $\boldsymbol{M_I}$. We make use of this matrix to calculate the individual-based reproduction number R_I, recalling that $\boldsymbol{u} = (u_1, u_2, \ldots, u_J)^\top$ and $\boldsymbol{v} = (v_1, v_2, \ldots, v_J)^\top$.

Theorem 4.2. *The dominant eigenvalue R_I of $\boldsymbol{M_I}$ is given by*

$$R_I = \frac{a + \sqrt{a^2 + 4\boldsymbol{u}^\top \boldsymbol{v}}}{2}.$$

Proof. Note that $a \neq 0$ so that $\boldsymbol{M_I}$ is of rank at least one. Further, since $v_k > 0$ for all $k \in \mathscr{J}$, the first column of $\boldsymbol{M_I}$ is linearly independent from the remaining columns, whence $\boldsymbol{M_I}$ is of rank at least two. The remaining columns can all be written as linear combinations of one another, since $u_k > 0$ for $k \in \mathscr{J}$. As a result, none of the final J columns are linearly independent from one another. We conclude that $\boldsymbol{M_I}$ is of rank 2 and that $\boldsymbol{M_I}$ has at most 2 non-zero eigenvalues.

In order to compute the eigenvalues of $\boldsymbol{M}_I$, we notice the decomposition

$$\boldsymbol{M}_I = \begin{pmatrix} a & u_1 & \dots & u_J \\ v_1 & 0 & \dots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ v_J & 0 & \dots & 0 \end{pmatrix} = \begin{pmatrix} a & 0 \\ v_1 & -v_1 \\ \vdots & \vdots \\ v_J & -v_J \end{pmatrix} \begin{pmatrix} 1 & \frac{u_1}{a} & \dots & \frac{u_J}{a} \\ 0 & \frac{u_1}{a} & \dots & \frac{u_J}{a} \end{pmatrix} = \boldsymbol{AB}, \text{ say.}$$

The non-zero eigenvalues of $\boldsymbol{AB}$ and $\boldsymbol{BA}$ are identical – see Williamson [1954]. Computing $\boldsymbol{BA}$, we find

$$\boldsymbol{BA} = \begin{pmatrix} a + \frac{\boldsymbol{u}^\top \boldsymbol{v}}{a} & -\frac{\boldsymbol{u}^\top \boldsymbol{v}}{a} \\ \frac{\boldsymbol{u}^\top \boldsymbol{v}}{a} & -\frac{\boldsymbol{u}^\top \boldsymbol{v}}{a} \end{pmatrix}.$$

It follows that the eigenvalues must satisfy

$$x^2 - ax - \boldsymbol{u}^\top \boldsymbol{v} = 0. \tag{4.3}$$

Selecting the largest solution of (4.3), we find that

$$R_I = \frac{a + \sqrt{a^2 + 4\boldsymbol{u}^\top \boldsymbol{v}}}{2},$$

as required. □

We connect R_* and R_I for the present model in the following result.

Theorem 4.3. *We have $R_* = 1$ if and only if $R_I = 1$. Further, if $R_* > 1$ $(R_* < 1)$ then $R_* > R_I$ $(R_* < R_I)$.*

Proof. The reproduction number R_I is the unique positive solution to $g_I(x) = 0$, where

$$g_I(x) = 1 - \frac{a}{x} - \frac{\boldsymbol{u}^\top \boldsymbol{v}}{x^2},$$

and where a, $\boldsymbol{u}$, and $\boldsymbol{v}$ are defined in Section 4.3.2. Recalling the definition of

R_* in (4.2), we have

$$\begin{aligned}
\boldsymbol{u}^\top \boldsymbol{v} &= \sum_{l=1}^{J} u_l v_l \\
&= \sum_{l=1}^{J} \beta_l \mu_I^{(l)} \sum_{j=1}^{J} \kappa_j \gamma_j \sum_{\boldsymbol{n}\in\mathscr{N}} \alpha_j(\boldsymbol{n}) \{\mu_{\boldsymbol{n},j,l}(\Lambda^L) - \delta_{jl}\} \\
&= \sum_{j=1}^{J} \kappa_j \gamma_j \sum_{\boldsymbol{n}\in\mathscr{N}} \alpha_j(\boldsymbol{n}) \sum_{l=1}^{J} \{\mu_{\boldsymbol{n},j,l}(\Lambda^L) - \delta_{jl}\} \mu_I^{(l)} \beta_l \\
&= R_* - \sum_{j=1}^{J} \kappa_j \gamma_j \sum_{\boldsymbol{n}\in\mathscr{N}} \alpha_j(\boldsymbol{n}) \sum_{l=1}^{J} \delta_{jl} \mu_I^{(l)} \beta_l \\
&= R_* - \sum_{j=1}^{J} \beta_j \kappa_j \gamma_j \mu_I^{(j)} \\
&= R_* - a.
\end{aligned}$$

We define the function $\text{sign}(x)$ to be -1, 0 and 1 for $x < 0$, $x = 0$ and $x > 0$ respectively. Now $g_I(x)$ is an increasing function on $(0,\infty)$ and

$$\text{sign}(g_I(1)) = \text{sign}(1 - a - \boldsymbol{u}^\top \boldsymbol{v}) = \text{sign}(1 - R_*).$$

It follows, since R_I is the unique positive root of $g_I(x)$, that $R_* = 1$ if and only if $R_I = 1$, and $R_* > 1 (R_* < 1)$ if and only if $R_I > 1$ ($R_I < 1$). Suppose that $R_* > 1$. Then

$$\begin{aligned}
g_I(R_*) &= 1 - \frac{a}{R_*} - \frac{\boldsymbol{u}^\top \boldsymbol{v}}{R_*^2} \\
&> 1 - \frac{a}{R_*} - \frac{\boldsymbol{u}^\top \boldsymbol{v}}{R_*} \\
&= 0,
\end{aligned}$$

which implies that $R_* > R_I$ as $g_I(x)$ is increasing on $(0,\infty)$ and $g_I(R_I) = 0$. When

$R_* < 1$, we have

$$\begin{aligned} g_I(R_*) &= 1 - \frac{a}{R_*} - \frac{\boldsymbol{u}^\top \boldsymbol{v}}{R_*^2} \\ &< 1 - \frac{a}{R_*} - \frac{\boldsymbol{u}^\top \boldsymbol{v}}{R_*} \\ &= 0. \end{aligned}$$

Applying the same argument as above results in $R_* < R_I$ when $R_* < 1$, concluding the proof. □

4.3.3 Basic reproduction number R_0

We now derive the basic reproduction number R_0 for this model. This requires a generation-based branching process similar to that of Ball et al. [2016], Section 2.2, extended to the present multitype setting. Owing to the proportionate global mixing assumption, we require only a single-type branching process for the derivation. Consider a discrete-time branching process using the rank generations of infectives. Each individual in the branching process corresponds to an infected household, whose generation is given by the global generation of the corresponding primary infective in that household. If infected at "time" k, such a household produces further infected households at "times" $k+1, k+2, \ldots$. A typical household produces $\tilde{v}_k$ further infected households (on average) at time $k+1$. The basic reproduction number R_0 is then given by the unique positive solution ψ of the discrete-time Lotka-Euler equation

$$1 - \sum_{k=0}^{\infty} \frac{\tilde{v}_k}{\psi^{k+1}} = 0. \tag{4.4}$$

(To show that ψ is unique define $g : (0,\infty) \to \mathbb{R}$ by $g(x) = 1 - \sum_{k=0}^{\infty} \frac{\tilde{v}_k}{x^{k+1}}$. Then $g(x)$ is continuous with $g(x) \to -\infty$ as $x \downarrow 0$, $g(x) \to 1$ as $x \uparrow \infty$ and $g'(x) > 0$.)

Note that R_0 is the asymptotic geometric growth rate of the above branching process, augmented to include within-spread in households. This definition of R_0 coincides with the usual definition (see, for example, Heesterbeek and Dietz [1996]) when all households have size 1. We assume, in the derivation that

follows, that $\lambda_{ij}^L = \lambda_L$ for all $i, j \in \mathscr{J}$. This corresponds to the case where individuals are heterogeneous in terms of global contacts, but mix homogeneously within their household - a sensible assumption, for example, for calculations pertaining to herd immunity levels. We also assume, for ease of exposition, that $T_I^{(i)} \overset{\mathrm{D}}{=} T_I^{(j)}$ for all $i, j \in \mathscr{J}$.

It remains to compute $\tilde{\nu}_k$ for $k \geq 0$. When $k = 0$ we need only consider the offspring of the primary case of a typical infected household. A typical type-j individual in a category-$\boldsymbol{n}$ household will produce, on average, $\beta_j \mu_I^{(j)}$ global infections. Applying Lemma 4.1 then gives

$$\tilde{\nu}_0 = \sum_{j=1}^{J} \beta_j \kappa_j \gamma_j \mu_I^{(j)}.$$

We now turn attention to the case $k \geq 1$. It is beneficial to define the set of all household categories with at least two members as

$$\mathscr{N}_2 = \{\boldsymbol{n} = (n_1, n_2, \ldots, n_J) \in \mathbb{Z}_+^J : 2 \leq \sum_{j=1}^{J} n_j \leq n_{\max}\}.$$

If, in the process described above, the globally contacted individual is in a household of size 1, then $\nu_k = 0$ for $k \geq 1$. Suppose now that the household category of such a contacted individual is $\boldsymbol{n} \in \mathscr{N}_2$, and that the initial infective is type $j \in \mathscr{J}$. For $k \geq 1$, let $\mu_k^{(\boldsymbol{n})}(\lambda_L)$ denote the mean number of members in generation k of a single-household epidemic in a category-$\boldsymbol{n}$ household. By the assumption regarding Λ_L, this can be computed via $n = \|\boldsymbol{n}\|$ along with the methods Ball et al. [2016], Appendix A. For $k \geq 1$, let $\tilde{\mu}_{k,i}^{(\boldsymbol{n})}(\lambda_L)$ denote the mean number of type-i individuals in generation k in this household. Let u denote a typical individual who does not belong to generation 0. Since local infection happens uniformly at random within the household, we have

$$\begin{aligned}\tilde{\mu}_{k,i}^{(\boldsymbol{n})}(\lambda_L) =& \mathrm{E}\left[\sum_{l=1}^{n_i - \delta_{ij}} \mathbb{1}_{\{\text{Individual } l \text{ belongs to generation } k\}}\right] \\ =& (n_i - \delta_{ij})\mathrm{P}(\text{Individual } u \text{ belongs to generation } k).\end{aligned}$$

We also have

$$\begin{aligned}\mu_k^{(\boldsymbol{n})}(\lambda_L) =& \mathrm{E}\left[\sum_{l=1}^{\|\boldsymbol{n}\|-1} \mathbb{1}_{\{\text{Individual } l \text{ belongs to generation } k\}}\right] \\ =& (\|\boldsymbol{n}\|-1)\mathrm{P}(\text{Individual } u \text{ belongs to generation } k).\end{aligned}$$

Then, since $\|\boldsymbol{n}\| \geq 2$, it follows that

$$\tilde{\mu}_{k,i}^{(\boldsymbol{n})}(\lambda_L) = \frac{n_i - \delta_{ij}}{\|\boldsymbol{n}\|-1}\mu_k^{(\boldsymbol{n})}(\lambda_L).$$

Applying Lemma 4.1 as well as the definition of $\alpha_j(\boldsymbol{n})$ yields

$$\tilde{\nu}_k = \sum_{j=1}^{J} \kappa_j \gamma_j \sum_{\boldsymbol{n}\in\mathscr{N}_2} \alpha_j(\boldsymbol{n})\mu_k^{(\boldsymbol{n})}(\lambda_L) \sum_{i=1}^{J} \frac{n_i - \delta_{ij}}{\|\boldsymbol{n}\|-1}\beta_i \mu_I^{(i)}, \qquad k \geq 1,$$

where households of size 1 are excluded in order to make the definition of $\tilde{\nu}_k$ robust. We can then compute R_0 by taking the unique positive solution of (4.4).

The basic reproduction number R_0 does not typically attribute all secondary cases in a household to the primary case in that household; the following result connects R_0 and R_I in the highly locally infectious case, where the initial infective infects the whole household.

Theorem 4.4. *In the highly locally infectious case, we have $R_I = R_0$.*

Proof. In the highly locally infectious case, all local epidemics infect the entire household, and all secondary infection in the household is attributed to the primary infective in that household. As a result, we have $\mu_0^{(\boldsymbol{n})} = 1$, $\mu_1^{(\boldsymbol{n})} = \|\boldsymbol{n}\| - 1$ and $\mu_k^{(\boldsymbol{n})} = 0$ for $k \geq 2$. The defining equation for R_0 then reduces to

$$\psi^2 - \psi\tilde{\nu}_0 - \tilde{\nu}_1 = 0, \tag{4.5}$$

with $\tilde{\nu}_0 = \sum_{i=1}^{J} \beta_i \kappa_i \gamma_i \mu_I^{(i)}$ and

$$\tilde{\nu}_1 = \sum_{j=1}^{J} \kappa_j \gamma_j \sum_{\boldsymbol{n}\in\mathscr{N}} \alpha_j(\boldsymbol{n}) \sum_{i=1}^{J} (n_i - \delta_{ij})\beta_i \mu_I^{(i)}.$$

Note that equations (4.3) and (4.5) are equivalent; we have $R_0 = R_I$ in the highly

locally infectious case. □

It is clear from the proof of Theorem 4.4 that the result extends to a more general model. Indeed, the assumption of proportionate mixing and the restrictions on $T_I^{(j)}$ ($j \in \mathscr{J}$) are not necessary in order for Theorem 4.4 to hold – see Ball et al. [2016], Remark 2.

4.3.4 Perfect vaccine-associated reproduction number R_V

We conclude our discussion of reproduction numbers by defining the perfect vaccine-associated reproduction number R_V, introduced for the single-type households model by Goldstein et al. [2009]. Letting $R_*(c)$ denote the value of R_* after a proportion $c \in (0,1)$ of the population are vaccinated uniformly at random and assuming p_C is such that $R_*(p_C) = 1$, we define $R_V = (1-p_C)^{-1}$. The critical vaccination coverage is then $p_C = 1 - R_V^{-1}$, in an analogous manner to $p_C = 1 - R_0^{-1}$ for the homogeneously mixing epidemic. We note that, by an identical argument to that of Ball et al. [2016], Theorem 1, we have $R_V = R_0$ if $\tilde{v}_4 = 0$ and $R_V > R_0$ otherwise. It follows immediately that $R_V = R_0$ in the highly locally infectious case. In the non-highly-locally-infectious case, $p_C = 1 - R_0^{-1}$ if $n_{\max} \leq 3$, otherwise $1 - R_0^{-1} < p_C \leq 1 - R_I^{-1}$.

4.3.5 Early exponential growth rate r

The early exponential growth rate r is derived by calculating the Malthusian parameter of the branching process, which approximates the proliferation of infected households, outlined in Section 4.3.1. In the absence of the assumption regarding proportionate global mixing, calculating r requires calculating the Malthusian parameter for a multitype branching process, following Doney [1976]. Such a calculation is rather unwieldy, as well as being difficult to implement computationally. Using the single-type process, however, makes the calculation of r more convenient and presentable – see the discussion following Pellis et al. [2011], Section 3.1. The computation we give is an extension of the calculations in Ball and Shaw [2015], Section 4.1, and Ball et al. [2023], Appendix C, to the present setting: a multitype households epidemic model with proportionate global mixing and no latent period.

The early exponential growth rate (r) for the process defined in Section 4.3.1 satisfies

$$\int_0^\infty e^{-rt}\beta(t)dt = 1, \tag{4.6}$$

where $\beta(t)$ is the (instantaneous) mean number of global contacts arising from a typical household in the branching process, t time units after it was contacted globally. The left-hand side of (4.6) corresponds to the Laplace transform of $\beta(t)$ and is not tractable in general; we restrict attention to the case where $T_I^{(j)} \sim \text{Exp}(\chi_j)$ $\left(\text{with } \chi_j = 1/\mu_I^{(j)}\right)$ so that a Markov chain approach can be used.

We follow a method similar to that of Ball and Shaw [2015], Section 4.1, in order to calculate the left-hand side of (4.6), beginning with the necessary notation for a single household of category $\boldsymbol{n} \in \mathscr{N}$. Consider the single-household epidemic model $\mathscr{E}_{\boldsymbol{n},\boldsymbol{a}}(\boldsymbol{e}_j)$ defined in Section 4.2, with $j \in \mathscr{J}$. Let $\mathscr{F}_j^{(\boldsymbol{n})}$ be the set of possible states that the household can reach as the epidemic progresses. The possible states can be characterised by $(\boldsymbol{s},\boldsymbol{i})$, where $\boldsymbol{s} = (s_1, s_2, \ldots, s_J)$ is a vector of the number of susceptibles of each type. We define $\boldsymbol{i}$ analogously for the number of infectives of each type and note that, for every $k \in \mathscr{J}$, we have $s_k + i_k \leq n_k$. Let $S_j^{(\boldsymbol{n})} = |\mathscr{F}_j^{(\boldsymbol{n})}|$ be the number of possible states. Since the initial infective is type j, the states including $s_j = n_j$ are not possible and are excluded. It follows, for $\boldsymbol{n} \in \mathscr{N}$ and $j \in \mathscr{J}$, that

$$S_j^{(\boldsymbol{n})} = \frac{n_j(n_j+3)}{2} \times \prod_{i \neq j} \left(\frac{(n_i+1)(n_i+2)}{2} \right).$$

Let $p_{\boldsymbol{s},\boldsymbol{i},j}^{(\boldsymbol{n})}(t)$ be the probability that, at time $t \geq 0$, the within-household epidemic among a household of category $\boldsymbol{n}$, having an initial infective of type j, is in state $(\boldsymbol{s},\boldsymbol{i}) \in \mathscr{F}_j^{(\boldsymbol{n})}$. The mean number of global contacts at time t to type-k individuals, which emanate from a typical household of category $\boldsymbol{n}$ with an initial infective of type j, is then given by

$$\beta_{j,k}^{(\boldsymbol{n})}(t) = \sum_{(\boldsymbol{s},\boldsymbol{i}) \in \mathscr{F}_j^{(\boldsymbol{n})}} p_{\boldsymbol{s},\boldsymbol{i},j}^{(\boldsymbol{n})}(t) \sum_{l=1}^{J} i_l \beta_l \mu_I^{(l)} \kappa_k \gamma_k, \quad t \geq 0.$$

Totalling the rates of infection to each type, we have

$$\beta_j^{(\boldsymbol{n})}(t) = \sum_{(\boldsymbol{s},\boldsymbol{i})\in\mathscr{F}_j^{(\boldsymbol{n})}} p_{\boldsymbol{s},\boldsymbol{i},j}^{(\boldsymbol{n})}(t) \sum_{l=1}^{J} i_l \beta_l \mu_I^{(l)}, \quad t \geq 0.$$

Applying Lemma 4.3 as well as the definition of $\alpha_j(\boldsymbol{n})$ yields

$$\beta(t) = \sum_{j=1}^{J} \kappa_j \gamma_j \sum_{\boldsymbol{n}\in\mathscr{N}} \alpha_j(\boldsymbol{n}) \sum_{(\boldsymbol{s},\boldsymbol{i})\in\mathscr{F}_j^{(\boldsymbol{n})}} p_{\boldsymbol{s},\boldsymbol{i},j}^{(\boldsymbol{n})}(t) \sum_{k=1}^{J} i_k \beta_k \mu_I^{(k)}, \quad t \geq 0.$$

With a view toward calculating r via (4.6), we make use of the Markovian assumption in order to calculate

$$\tilde{p}_{\boldsymbol{s},\boldsymbol{i},j}^{(\boldsymbol{n})}(r) = \int_0^\infty e^{-rt} p_{\boldsymbol{s},\boldsymbol{i},j}^{(\boldsymbol{n})}(t)dt$$

as follows. Let state 1 refer to the initial state with $s_k = n_k - \delta_{kj}$ and $i_k = \delta_{kj}$ for all $k \in \mathscr{J}$ (the ordering of the remaining states is not crucial). Then, for each $j \in \mathscr{J}$ there exists a bijection

$$g_j : \mathscr{F}_j^{(\boldsymbol{n})} \to \left\{1, 2, \ldots, S_j^{(\boldsymbol{n})}\right\},$$

so each $(\boldsymbol{s},\boldsymbol{i}) \in \mathscr{F}_j^{(\boldsymbol{n})}$ corresponds to a unique state $d \in \left\{1, 2, \ldots, S_j^{(\boldsymbol{n})}\right\}$. The within-household epidemic is a Markov chain with state space $\mathscr{F}_j^{(\boldsymbol{n})}$ and transition matrix $Q_j^{(\boldsymbol{n})}$, say. Thus

$$p_{\boldsymbol{s},\boldsymbol{i},j}^{(\boldsymbol{n})}(t) = \left(e^{tQ_j^{(\boldsymbol{n})}}\right)_{1,d}, \tag{4.7}$$

where $e^A = \sum_{k=0}^{\infty} A^k/k!$ denotes the usual matrix exponential. We take the Laplace transform of (4.7) to find

$$\tilde{p}_{\boldsymbol{s},\boldsymbol{i},j}^{(\boldsymbol{n})}(r) = \left(\left[rI_{S_{\boldsymbol{n}}^j} - Q_j^{(\boldsymbol{n})}\right]^{-1}\right)_{1,d}. \tag{4.8}$$

Then, by substitution into (4.6), we have

$$\sum_{j=1}^{J} \kappa_j \gamma_j \sum_{\boldsymbol{n} \in \mathscr{N}} \alpha_j(\boldsymbol{n}) \sum_{(\boldsymbol{s},\boldsymbol{i}) \in \mathscr{F}_j^{(\boldsymbol{n})}} \tilde{p}_{\boldsymbol{s},\boldsymbol{i},j}^{(\boldsymbol{n})}(r) \sum_{k=1}^{J} i_k \beta_k \mu_I^{(k)} = 1, \tag{4.9}$$

so that r can be computed, typically only numerically, using (4.8) and (4.9).

4.4 Final outcome

We now turn attention to the final outcome of $\mathscr{E}$, supposing that N is large, there are few initial infectives, and a major outbreak occurs. In Ball and Lyne [2001] a system of J equations are presented for the final outcome. These equations couple the global escape probabilities for each type, which we denote by

$$\boldsymbol{\pi} = (\pi_1, \pi_2, \ldots, \pi_J),$$

with the probability of ultimate infection for an individual of each type, which we denote by

$$\tilde{\boldsymbol{z}} = (\tilde{z}_1, \tilde{z}_2, \ldots, \tilde{z}_J).$$

In the context of proportionate global mixing, we can instead consider a single univariate equation for the (weighted) sum of the infectious periods of those ultimately infected in the epidemic, referred to as the *severity* of the epidemic. The weights are given by $\beta_1, \beta_2, \ldots, \beta_J$. As a consequence, this definition of severity differs from the standard definition (viz. the sum of infectious periods of those ultimately infected – see, for example, Ball and Lyne [2001], Section 3). Upon calculating the severity, $\boldsymbol{\pi}$ and $\tilde{\boldsymbol{z}}$ are readily computed. To proceed, we calculate the contribution to the severity from each household in order to establish a univariate equation for the severity, which we scale by the population size N. Explicit definitions for terms including products and powers of vectors are provided in Appendix A.

Theorem 4.5. *Supposing a major outbreak occurs, the scaled severity* δ *satis-*

fies the implicit equation

$$\delta = \sum_{\boldsymbol{n}\in\mathscr{N}} \tilde{\alpha}_{\boldsymbol{n}} \|\boldsymbol{n}\|^{-1} \sum_{\boldsymbol{r}=\boldsymbol{0}}^{\boldsymbol{n}} \binom{\boldsymbol{n}}{\boldsymbol{r}} (1-\pi)^{r} \pi^{n-r} \sum_{j=1}^{J} \mu_{\boldsymbol{n}-\boldsymbol{r},\boldsymbol{r},j} \mu_I^{(j)} \beta_j, \tag{4.10}$$

where $\pi_j = \exp(-\delta\kappa_j)$ *and* $\tilde{\alpha}_{\boldsymbol{n}}$ *(*$\boldsymbol{n} \in \mathscr{N}$*) is the proportion of individuals which reside in a household of category* $\boldsymbol{n}$*.*

Proof. Let Υ_i ($i \in \mathscr{J}$) be the sum of the infectious periods of infected type-i individuals (the severity owing to type-i individuals). The weighted severity of the epidemic is given by $\Upsilon = \sum_{j=1}^{J} \Upsilon_j \beta_j$ and $\delta = \Upsilon/N$ is the scaled weighted severity; we derive a single balance equation for δ. Firstly, let $j \in \mathscr{J}$ and note that a typical type-j individual will avoid infection from all type-i individuals if there are no points in a Poisson process with rate λ_{ij}^{G}/N run until time Υ_i. Then, using independence of the different Poisson processes giving rise to contacts with a given individual of type j, we have

$$\begin{aligned}
\pi_j =& \text{P(typical type-}j\text{ individual avoids global infection)} \\
=& \prod_{i=1}^{J} \text{P(typical type-}j\text{ individual avoids global infection from all type-}i\text{ members)} \\
=& \prod_{i=1}^{J} \exp\left(-\frac{\lambda_{ij}^{G}}{N}\Upsilon_i\right) \\
=& \prod_{i=1}^{J} \exp\left(-\frac{\beta_i \kappa_j \Upsilon_i}{N}\right) \\
=& \exp\left(-\kappa_j \sum_{i=1}^{J} \frac{\beta_i \Upsilon_i}{N}\right) \\
=& \exp(-\delta\kappa_j),
\end{aligned}$$

which expresses the global escape probabilities in terms of the severity. Now consider calculating the total severity by aggregating the contribution from each household category, recalling that there are $m_{\boldsymbol{n}}$ households of category $\boldsymbol{n}$. In the limit $N \to \infty$, individuals of type i avoid global infection independently with probability π_i. Thus the ultimate spread of infection in a category-$\boldsymbol{n}$ household is that of the single-household outbreak $\mathscr{E}_{\boldsymbol{n}}(\Lambda^L, \boldsymbol{\pi})$. Let $\Upsilon_{\boldsymbol{n},\boldsymbol{r}}$ be the total severity (weighted sum of infectious periods among all types) from a single-household

outbreak with $\boldsymbol{n}$ initial susceptibles and $\boldsymbol{r}$ initial infectives. Then we have

$$\Upsilon = \sum_{\boldsymbol{n}\in\mathscr{N}} m_{\boldsymbol{n}} \sum_{\boldsymbol{r}=\boldsymbol{0}}^{\boldsymbol{n}} \binom{\boldsymbol{n}}{\boldsymbol{r}} (1-\boldsymbol{\pi})^{\boldsymbol{r}} \boldsymbol{\pi}^{\boldsymbol{n}-\boldsymbol{r}} \Upsilon_{\boldsymbol{n}-\boldsymbol{r},\boldsymbol{r}}.$$

Applying Lemma B.2, we find

$$\Upsilon = \sum_{\boldsymbol{n}\in\mathscr{N}} m_{\boldsymbol{n}} \sum_{\boldsymbol{r}=\boldsymbol{0}}^{\boldsymbol{n}} \binom{\boldsymbol{n}}{\boldsymbol{r}} (1-\boldsymbol{\pi})^{\boldsymbol{r}} \boldsymbol{\pi}^{\boldsymbol{n}-\boldsymbol{r}} \sum_{j=1}^{J} \mu_{\boldsymbol{n}-\boldsymbol{r},\boldsymbol{r},j} \mu_I^{(j)} \beta_j.$$

Recalling that $N = \sum_{\boldsymbol{n}\in\mathscr{N}} m_{\boldsymbol{n}} \|\boldsymbol{n}\|$, letting $\tilde{\alpha}_{\boldsymbol{n}}$ denote the probability that a randomly chosen individual resides in a household of category $\boldsymbol{n}$, and dividing by the population size N gives

$$\delta = \sum_{\boldsymbol{n}\in\mathscr{N}} \tilde{\alpha}_{\boldsymbol{n}} \|\boldsymbol{n}\|^{-1} \sum_{\boldsymbol{r}=\boldsymbol{0}}^{\boldsymbol{n}} \binom{\boldsymbol{n}}{\boldsymbol{r}} (1-\boldsymbol{\pi})^{\boldsymbol{r}} \boldsymbol{\pi}^{\boldsymbol{n}-\boldsymbol{r}} \sum_{j=1}^{J} \mu_{\boldsymbol{n}-\boldsymbol{r},\boldsymbol{r},j} \mu_I^{(j)} \beta_j, \tag{4.11}$$

as required. □

Note that $\delta = 0$ is always a solution to (4.11) and a second solution $\delta^* \in (0,\infty)$ exists if and only if $R_* > 1$. We show this fact later by considering an equivalent equation – see Theorem 4.8. Solving numerically then allows $\boldsymbol{\pi}$ to be calculated, and thus the final size $\tilde{\boldsymbol{z}}$ can be determined. By the above argument regarding the ultimate spread of infection, a typical category-$\boldsymbol{n}$ household will have, on average, $\mu_{\boldsymbol{n},i}(\Lambda^L, \boldsymbol{\pi})$ type-i members infected by the epidemic. Conditioning on the household category of a typical type-i individual then gives

$$\tilde{z}_i = \sum_{\boldsymbol{n}\in\mathscr{N}:n_i>0} \alpha_i(\boldsymbol{n}) \mu_{\boldsymbol{n},i}(\Lambda^L, \boldsymbol{\pi})/n_i, \quad i \in \mathscr{J}, \tag{4.12}$$

with $\pi_j = \exp(-\delta\kappa_j)$ for $j \in \mathscr{J}$.

The derivation of the univariate equation at (4.11) is only possible owing to the proportionate mixing assumption; in general $\tilde{z}_i$ must be computed by solving a J-dimensional system of equations – see Ball et al. [2004], Section 2.2.

4.5 Severity process

We next derive results with a view toward a central limit theorem for the severity of the multitype households model with proportionate global mixing. We make use of a construction introduced by Sellke – see Sellke [1983]. As a consequence of the proportionate mixing assumption, the resulting severity process is a single-type process (cf. the multitype severity process in Ball and Lyne [2001]). We also make the assumption of a finite number of household categories, which is not made in Ball and Lyne [2001]. The single-type severity process then yields more explicit covariance matrices, as well as more easily implemented numerical calculations, which are not immediately apparent from Ball and Lyne [2001]. We study the severity process in detail before making the connection to $\mathscr{E}$.

4.5.1 Sellke construction

In order to establish the desired convergence results, we use a Sellke construction (see Sellke [1983]) for calculating the final size of an epidemic, extended to the present setting of the multitype households model with proportionate global mixing. To do so, we first establish a construction of the epidemic $\mathscr{E}_{\boldsymbol{n}}(\Lambda^L, \boldsymbol{\pi})$ defined in Section 4.2. We begin the construction by introducing the relevant random variables, closely following Ball and Lyne [2001], Section 4.1. These random variables are all mutually independent and defined on an underlying probability space $(\Omega, \mathscr{F}, P)$. Let L_{ij} ($i \in \mathscr{J}$, $j = 1, 2, \ldots, n_i$) be independent random variables having an exponential distribution with rate κ_i. Define $\tilde{L}_{ij}$ ($i \in \mathscr{J}$, $j = 1, 2, \ldots, n_i$) similarly, but instead having an exponential distribution with rate 1. Finally, let $I_i^{(j)}$ ($i \in \mathscr{J}$, $j = 1, 2, \ldots, n_i$) each be realisations of $T_I^{(i)}$.

Let (i, j) denote the j^{th} individual of type i ($i \in \mathscr{J}$, $j = 1, 2, \ldots, n_i$). We use the triple $(L_{ij}, \tilde{L}_{ij}, I_i^{(j)})$ associated with each individual (i.e. for $i \in \mathscr{J}$, $j = 1, 2, \ldots, n_i$) in order to form the desired construction. Initially, all individuals in the population are susceptible. The epidemic is initiated by each individual being exposed to t units of external (global) infectious pressure. Individual (i, j) becomes infected globally if and only if $L_{ij} \leq t$. The globally infected individu-

als begin local epidemics, in which a typical susceptible (i', j'), say, accumulates exposure to local infection at rate $\sum_{k=1}^{J} \lambda^L_{ki'} y_k$, where y_k is the number of type-k infectives in the household at the given time. If this exposure exceeds $\tilde{L}_{i'j'}$, then (i', j') is infected locally. If individual (i, j) is infected, locally or globally, then their infectious duration is given by $I_i^{(j)}$. This construction gives a realisation of $\mathscr{E}_{\boldsymbol{n}}(\Lambda^L, \boldsymbol{\pi})$ with $\pi_i = \exp(-\kappa_i t)$ $(i \in \mathscr{J})$. Once there are no infectives present in the household, the process terminates.

The epidemic $\hat{\mathscr{E}}(t)$, with t units of initial global infectious pressure applied to each individual, is then defined by taking, for each $\boldsymbol{n} \in \mathscr{N}$, $m_{\boldsymbol{n}}$ independent copies of $\mathscr{E}_{\boldsymbol{n}}(\Lambda^L, \boldsymbol{\pi})$, which are identically distributed for the same $\boldsymbol{n}$, with $\pi_i = \exp(-\kappa_i t)$ $(i \in \mathscr{J})$. Note that, in this construction of $\hat{\mathscr{E}}(t)$, individuals do not make global contacts.

4.5.2 The severity process - description

We now outline the (single-type) severity process of interest. Note that in the sequel $t \geq 0$ refers to global infectious pressure and not to time. Let $\tilde{A}^{(j,\boldsymbol{n})}(t)$ denote the sum of the infectious periods of all type-j individuals ever infected in $\mathscr{E}_{\boldsymbol{n}}(\Lambda^L, \boldsymbol{\pi}(t))$, with $\pi_i(t) = \exp(-\kappa_i t)$. The severity of $\mathscr{E}_{\boldsymbol{n}}(\Lambda^L, \boldsymbol{\pi}(t))$ is then given by

$$A^{(\boldsymbol{n})}(t) = \sum_{j=1}^{J} \beta_j \tilde{A}^{(j,\boldsymbol{n})}(t), \quad t \geq 0, \boldsymbol{n} \in \mathscr{N}.$$

Consider now the multitype households epidemic $\hat{\mathscr{E}}(t)$ which is initiated by exposing each individual to t units of global infectious pressure. Writing $A^{(\boldsymbol{n},k)}(t)$ $(k = 1, 2, \ldots, m_{\boldsymbol{n}})$ for the $m_{\boldsymbol{n}}$ independent and identically distributed copies of $A^{(\boldsymbol{n})}(t)$, we define the total severity $A_\bullet(t)$ by

$$A_\bullet(t) = \sum_{\boldsymbol{n} \in \mathscr{N}} \sum_{k=1}^{m_{\boldsymbol{n}}} A^{(\boldsymbol{n},k)}(t), \qquad t \geq 0,$$

where we have summed the contributions to the severity from each household. This defines a process $A_\bullet = \{A_\bullet(t) : t \geq 0\}$.

Now consider the epidemic $\tilde{\mathscr{E}}(t)$, defined similarly to $\hat{\mathscr{E}}(t)$, but without disregarding subsequent global infection. Following Scalia-Tomba [1985], we

embed the epidemic in the $A_\bullet(t)$ process using sampling "times" $T_0, T_1, \ldots$ as follows. Assume that the population as a whole are exposed to T_0 units of initial global infectious pressure, with each individual exposed to $\bar{T}_0 = T_0/N$ units. This will cause some individuals to be globally infected, and they may also generate an infectious clump within their household by infecting other members of their household locally. This will give rise to $A_\bullet(\bar{T}_0)$ further units of global infection, so that the total amount of global infection is $T_1 = T_0 + A_\bullet(\bar{T}_0)$. Applying the same argument to the following generation then yields $T_2 = T_0 + A_\bullet(\bar{T}_1)$, and further iteration of this argument gives a sequence $T_0, T_1, \ldots$ satisfying $T_{k+1} = T_0 + A_\bullet(\bar{T}_k)(k = 0, 1, \ldots)$. Since the population is finite, there must be an iterative step which yields no further pressure. We define

$$k^* = \min\{k \geq 0 : T_{k+1} = T_k\}$$

to be the first generation at which no further subsequent infection is created, i.e., where the process terminates. Letting $T_\infty = T_{k^*}$ denote the terminal infectious pressure, observe that

$$T_\infty = \inf\{t > 0 : t = T_0 + A_\bullet(t)\}.$$

Scaling by the population size, we let $\bar{T}_k = T_k/N$ for $k = 0, 1, \ldots$. Then

$$\bar{T}_\infty = \inf\{t > 0 : t = \bar{T}_0 + N^{-1} A_\bullet(t)\} \tag{4.13}$$

corresponds to the scaled terminal pressure. If $Z_\bullet(t)$ $(\tilde{Z}_E(t))$ is the number of households infected in $\hat{\mathscr{E}}(t)$ $(\tilde{\mathscr{E}}(t))$, then $\tilde{Z}_E = Z_\bullet(\bar{T}_\infty)$. Other properties of the final outcome of $\tilde{\mathscr{E}}(t)$ are given by the corresponding properties of $\hat{\mathscr{E}}(\bar{T}_\infty)$

We now consider the sequences of epidemics $\mathscr{E}^{(v)}$, $\tilde{\mathscr{E}}^{(v)}$ and $\hat{\mathscr{E}}^{(v)}$ for $v = 1, 2, \ldots$ as follows. There are $N^{(v)}$ individuals in the population, and the infectious pressure on each individual used to initiate $\tilde{\mathscr{E}}^{(v)}$ is given by $\bar{T}_0^{(v)} = T_0/N^{(v)}$. There are $m^{(v)}$ households in total, comprised of $m_{\boldsymbol{n}}^{(v)}$ category-$\boldsymbol{n}$ households, with $m_H^{(v)} = N^{(v)}/m^{(v)}$ denoting the mean household size. The proportion of category-$\boldsymbol{n}$ households is given by $\theta_{\boldsymbol{n}}^{(v)}$ and, for $i \in \mathscr{J}$, the proportion of type-

i individuals is given by $\gamma_i^{(v)} > 0$ with $\gamma_i = \lim_{v\to\infty} \gamma_i^{(v)} > 0$. Values not indexed by v are assumed to be their corresponding asymptotic values (for example, $m_H = \lim_{v\to\infty} m_H^{(v)}$). Finally, we let $a_{\boldsymbol{n}}(t) = \mathrm{E}[A^{(\boldsymbol{n},1)}(t)]$, $a(t) = \sum_{\boldsymbol{n}\in\mathscr{N}} \theta_{\boldsymbol{n}} a_{\boldsymbol{n}}(t)$ and $a^{(v)}(t) = \sum_{\boldsymbol{n}\in\mathscr{N}} \theta_{\boldsymbol{n}}^{(v)} a_{\boldsymbol{n}}(t)$.

The corresponding scaled terminal infectious pressure of $\tilde{\mathscr{E}}^{(v)}$ is then given by $\bar{T}_\infty^{(v)}$, defined analogously to $\bar{T}_\infty$ – see (4.13). We define the severity process for $\hat{\mathscr{E}}^{(v)}$, denoted $A_\bullet^{(v)}(t)$, by

$$A_\bullet^{(v)}(t) = \sum_{\boldsymbol{n}\in\mathscr{N}} \sum_{k=1}^{m_{\boldsymbol{n}}^{(v)}} A^{(\boldsymbol{n},k)}(t), \qquad t \geq 0.$$

We note at this stage the disparity between $\mathscr{E}^{(v)}$ and $\tilde{\mathscr{E}}^{(v)}$, viz. the way in which the epidemic is initiated. It is fruitful to first consider the embedding construction in detail; in Section 4.8 we make explicit the connection between $\mathscr{E}^{(v)}$ and $\tilde{\mathscr{E}}^{(v)}$.

4.5.3 The severity process - analysis

In this section we prove a series of results pertaining to the behaviour of $A_\bullet^{(v)}(t)$ and $\bar{T}_\infty^{(v)}$ as $t \to \infty$. We make use of multivariate Gontcharoff polynomials – see Appendix A for further details. We derive limiting results for the severity process, beginning with a supporting lemma regarding properties of $a(t)$.

Lemma 4.6. *The function $a(t)$ is a non-decreasing and concave function on $[0,\infty)$.*

Proof. Recall that $a(t) = \sum_{\boldsymbol{n}\in\mathscr{N}} \theta_{\boldsymbol{n}} a_{\boldsymbol{n}}(t)$. It is sufficient to show, for all $t \geq 0$, that $a'_{\boldsymbol{n}}(t) > 0$ and $a''_{\boldsymbol{n}}(t) < 0$ for every household category $\boldsymbol{n} \in \mathscr{N}$. Consider the epidemic $\mathscr{E}_{\boldsymbol{n}}(\Lambda^L, \boldsymbol{\pi})$ with $\boldsymbol{\pi}(t) = (\pi_1(t), \pi_2(t), \ldots, \pi_J(t))$ such that $\pi_i(t) = \exp(-\kappa_i t)$ ($i \in \mathscr{J}$). Let $a_{\boldsymbol{n}}^{(i)}(t)$ denote the mean severity from type-i individuals in $\mathscr{E}_{\boldsymbol{n}}(\Lambda^L, \boldsymbol{\pi}(t))$, so $a_{\boldsymbol{n}}(t) = \sum_{i=1}^J a_{\boldsymbol{n}}^{(i)}(t)$. Let $z_i(t)$ denote the mean number of type-i individuals infected in $\mathscr{E}_{\boldsymbol{n}}(\Lambda^L, \boldsymbol{\pi}(t))$, with $s_i(t)$ the mean number of remaining type-i susceptibles. Applying Lemma B.2, we have $a_{\boldsymbol{n}}^{(i)}(t) = \beta_i \mu_I^{(i)} z_i(t) = \beta_i \mu_I^{(i)} (n_i - s_i(t))$. It remains to show that $s_i'(t) < 0$ and $s_i''(t) > 0$. Denote by $q_i^{(k)}$ the probability a type-k infective fails to infect anyone in a group

of $\boldsymbol{i}$ susceptibles (i_1 type-1 susceptibles, i_2 type-2 susceptibles,$\ldots, i_J$ type-J susceptibles) and let $\boldsymbol{q_i} = \left(q_{\boldsymbol{i}}^{(1)}, q_{\boldsymbol{i}}^{(2)}, \ldots, q_{\boldsymbol{i}}^{(J)}\right)$. Using Ball [2019], Theorem 4.1, we have

$$\mathrm{E}\left(\boldsymbol{S}_{[\boldsymbol{j}]}\right) = \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} \boldsymbol{\pi}^{\boldsymbol{i}}(t) G_{\boldsymbol{i}}^{(\boldsymbol{j})}(\mathbf{1}|\boldsymbol{U}) \quad (\boldsymbol{j} \in \mathbb{Z}_+^J), \tag{4.14}$$

where $\boldsymbol{U}$ is given by $\boldsymbol{U} = (\boldsymbol{q_k}, \boldsymbol{k} \in \mathbb{Z}_+^J)$ and $\boldsymbol{n}_{[\boldsymbol{k}]}$, denotes the vector falling factorial - see Appendix A. We now consider the right-hand side of (4.14) as a function of t. Extending Ball [2019], Remark 3.2, to the multivariate case, we have $G_{\boldsymbol{i}}^{(\boldsymbol{j})}(\mathbf{1}|\boldsymbol{U}) \geq 0$. Now $\boldsymbol{\pi}^{\boldsymbol{k}}(t) = \exp(-t\sum_{i=1}^J \kappa_i k_i)$, which clearly has a negative first derivative and positive second derivative. Then, since $s_1(t) = \mathrm{E}\left(\boldsymbol{S}_{[(1,0,\ldots,0)]}\right)$, we have $s_1'(t) < 0$ and $s_1''(t) > 0$; a similar argument holds for each $i \in \mathscr{J}$. Consequently, $a_{\boldsymbol{n}}(t)$ is non-decreasing and concave for every $\boldsymbol{n} \in \mathscr{N}$, so $a(t)$ is non-decreasing and concave also. □

Our next result concerns the almost sure convergence of the scaled severity process as $v \to \infty$; we show that this convergence occurs uniformly in t.

Theorem 4.7. *We have*

$$\sup_{t \geq 0} \left| \frac{1}{m^{(v)}} A_{\bullet}^{(v)}(t) - a(t) \right| \xrightarrow{\text{a.s.}} 0 \text{ as } v \to \infty.$$

Proof. The strong law of large numbers can be applied to each household category separately to give that, for each $t \in [0, \infty)$,

$$\frac{1}{m^{(v)}} \sum_{k=1}^{m_{\boldsymbol{n}}^{(v)}} A^{(\boldsymbol{n},k)}(t) = \theta_{\boldsymbol{n}}^{(v)} \frac{1}{m_{\boldsymbol{n}}^{(v)}} \sum_{k=1}^{m_{\boldsymbol{n}}^{(v)}} A^{(\boldsymbol{n},k)}(t) \xrightarrow{\text{a.s.}} \theta_{\boldsymbol{n}} a_{\boldsymbol{n}}(t) \text{ as } v \to \infty$$

for every $\boldsymbol{n} \in \mathscr{N}$. Since there are a finite number of household categories, we sum these to find that

$$\frac{1}{m^{(v)}} A_{\bullet}^{(v)}(t) \xrightarrow{\text{a.s.}} a(t) \text{ as } v \to \infty. \tag{4.15}$$

It remains to show that this convergence holds uniformly in t. Firstly, note that $A_{\bullet}^{(v)}(t)$ is non-decreasing in t and that $A_{\bullet}^{(v)}(\infty)$ is well-defined. In particular, since $A_{\bullet}^{(v)}(\infty)$ is a weighted sum of the infectious periods of all individuals in the population, we have that $A_{\bullet}^{(v)}(\infty)$ is almost surely finite and $a(\infty) < \infty$, so

(4.15) holds when $t = \infty$ also. Then (c.f Ball and Britton [2005], Lemma 1) we have that, for each $t \in [0,\infty]$, there exists a set $F_t \in \mathscr{F}$ with $\mathrm{P}(F_t) = 1$ such that, for all $\omega \in F_t$, we have

$$\lim_{v\to\infty} \frac{1}{m^{(v)}} A_\bullet^{(v)}(t,\omega) = a(t).$$

Here $A_\bullet^{(v)}(t,\omega)$ denotes the random variable $A_\bullet^{(v)}(t)$ evaluated at ω. Now take $F = \bigcap_{t\in\mathscr{T}} F_t$, where $\mathscr{T} = (\mathbb{Q}\cap[0,\infty))\cup\{\infty\}$. Then $F \in \mathscr{F}$ (the intersection taken is countable) and

$$\mathrm{P}(F^c) = \mathrm{P}\left(\bigcup_{t\in\mathscr{T}} F_t^c\right) \leq \sum_{t\in\mathscr{T}} \mathrm{P}(F_t^c) = 0.$$

Consequently, there exists a set $F \in \mathscr{F}$ with $P(F) = 1$ such that, for all $\omega \in F$, we have

$$\lim_{v\to\infty} \frac{1}{m^{(v)}} A_\bullet^{(v)}(t,\omega) = a(t), \qquad t \in \mathscr{T}.$$

Now $a(t)$ is non-decreasing in t (by Lemma 3.1) with $a(\infty) < \infty$. Fix an $\omega \in F$ and an $\varepsilon > 0$. We can partition the set $[0,\infty]$ by selecting $k \in \mathbb{N}$ and $t_0 = 0 < t_1 < t_2 \cdots < t_k < t_{k+1} = \infty$ such that $t_1, t_2, \ldots, t_k \in \mathbb{Q}$ and

$$a(t_{i+1}) - a(t_i) < \frac{\varepsilon}{2}, \qquad i = 0,1,\ldots,k.$$

The almost sure convergence of $A_\bullet^{(v)}(t)$ implies that there exists $v_0 \in \mathbb{N}$ such that

$$\left|\frac{1}{m^{(v)}} A_\bullet^{(v)}(t_i,\omega) - a(t_i)\right| < \frac{\varepsilon}{2}, \qquad i = 0,1,\ldots,k+1; v \geq v_0.$$

Let $t_i \leq t < t_{i+1}$. Then, assuming that $\frac{1}{m^{(v)}}A_\bullet^{(v)}(t,\omega) - a(t) > 0$, we have

$$\begin{aligned}\left|\frac{1}{m^{(v)}}A_\bullet^{(v)}(t,\omega) - a(t)\right| &= \frac{1}{m^{(v)}}A_\bullet^{(v)}(t,\omega) - a(t) \\ &\leq \frac{1}{m^{(v)}}A_\bullet^{(v)}(t_{i+1},\omega) - a(t_i) \\ &= \frac{1}{m^{(v)}}A_\bullet^{(v)}(t_{i+1},\omega) - a(t_{i+1}) + a(t_{i+1}) - a(t_i) \\ &< \frac{\varepsilon}{2} + \frac{\varepsilon}{2} = \varepsilon.\end{aligned}$$

A similar argument holds for the case where $\frac{1}{m^{(v)}}A_\bullet^{(v)}(t,\omega) - a(t) < 0$. Consequently, we have

$$\left|\frac{1}{m^{(v)}}A_\bullet^{(v)}(t,\omega) - a(t)\right| \leq \varepsilon, \qquad v \geq \nu_0, t \geq 0$$

and therefore

$$\sup_{t\geq 0}\left|\frac{1}{m^{(v)}}A_\bullet^{(v)}(t,\omega) - a(t)\right| \leq \varepsilon, \qquad v \geq \nu_0.$$

Then

$$\lim_{v\to\infty}\sup_{t\geq 0}\left|\frac{1}{m^{(v)}}A_\bullet^{(v)}(t,\omega) - a(t)\right| = 0,$$

since the choice of $\varepsilon > 0$ is arbitrary. Then, since $\mathrm{P}(F) = 1$, we have

$$\sup_{t\geq 0}\left|\frac{1}{m^{(v)}}A_\bullet^{(v)}(t) - a(t)\right| \xrightarrow{\text{a.s.}} 0 \text{ as } v \to \infty,$$

as required. □

In the remainder of this section we assume that $\bar{T}_0^{(v)} \xrightarrow{\text{a.s.}} 0$ as $v \to \infty$, noting stronger assumptions (on $\bar{T}_0^{(v)}$ or other quantities) where they are necessary. There are two possibilities for the scaled terminal infectious pressure $\bar{T}_\infty^{(v)}$, depending on whether a major outbreak occurs. We first consider the possible solutions of a related equation, before connecting these solutions to the behaviour of $\bar{T}_\infty^{(v)}$.

Theorem 4.8. *If $R_* \leq 1$, then $t = 0$ is the only solution in $[0,\infty)$ of the equation*

$$t = m_H^{-1}a(t).$$

If $R_* > 1$ then there is a unique solution, τ say, in $(0,\infty)$. Moreover, we have $R_* = m_H^{-1} a'(0)$.

Proof. If $t = 0$ then there is no global infection ($\pi_i = 1$ for all $i \in \mathscr{J}$) and so clearly $m_H^{-1} a(0) = 0$. Now $m_H^{-1} a(t)$ is concave, which follows from Lemma 4.6, so the equation $t = m_H^{-1} a(t)$ has at most two solutions. Since $a(\infty) < \infty$, a second solution exists if and only if $m_H^{-1} a'(0) > 1$, which we show is equivalent to $R_* > 1$.

We first calculate $a'_{\boldsymbol{n}}(0)$ for arbitrarily chosen $\boldsymbol{n} \in \mathscr{N}$. Consider a typical household of category $\boldsymbol{n}$. The probability of a single type-j individual being infected globally in $\mathscr{E}_{\boldsymbol{n}}(\Lambda^L, \boldsymbol{\pi})$ (denoted $P_j(t)$) satisfies

$$\begin{aligned} P_j(t) &= n_j (e^{-t\kappa_j})^{n_j - 1} (1 - e^{-t\kappa_j}) \times \prod_{i \neq j} e^{-t\kappa_i n_i} \\ &= n_j \kappa_j t + o(t), \end{aligned}$$

where $o(t)$ denotes any function satisfying $t^{-1} o(t) \to 0$ as $t \to 0$. The probability that more than one individual is infected globally is given by $o(t)$. If no individuals are infected globally, then there will be no contribution to the severity. Then, by Lemma B.2, the mean severity arising from a typical epidemic $\mathscr{E}_{\boldsymbol{n}}(\Lambda^L, \boldsymbol{\pi})$ satisfies

$$a_{\boldsymbol{n}}(t) = t \sum_{j=1}^{J} n_j \kappa_j \sum_{k=1}^{J} \mu_{\boldsymbol{n},j,k}(\Lambda^L) \mu_I^{(k)} \beta_k + o(t).$$

Then, using the definition of the derivative, we have

$$a'_{\boldsymbol{n}}(0) = \lim_{t \to 0} \frac{a_{\boldsymbol{n}}(t)}{t} = \sum_{j=1}^{J} n_j \kappa_j \sum_{k=1}^{J} \mu_{\boldsymbol{n},j,k}(\Lambda^L) \mu_I^{(k)} \beta_k.$$

Then, recalling the definition of R_* in (4.2) and noting that $m_H^{-1} n_j \theta_{\boldsymbol{n}} = \gamma_j \alpha_j(\boldsymbol{n})$

($i \in \mathscr{J}$, $\boldsymbol{n} \in \mathscr{N}$), we have

$$\begin{aligned}
\frac{a'(0)}{m_H} &= \frac{1}{m_H} \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} a'_{\boldsymbol{n}}(0) \\
&= \frac{1}{m_H} \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \sum_{j=1}^{J} \kappa_j n_j \sum_{k=1}^{J} \mu_{\boldsymbol{n},j,k}(\Lambda^L) \mu_I^{(k)} \beta_k \\
&= \sum_{j=1}^{J} \kappa_j \sum_{\boldsymbol{n} \in \mathscr{N}} \frac{\theta_{\boldsymbol{n}} n_j}{m_H} \sum_{k=1}^{J} \mu_{\boldsymbol{n},j,k}(\Lambda^L) \mu_I^{(k)} \beta_k \\
&= \sum_{j=1}^{J} \kappa_j \gamma_j \sum_{\boldsymbol{n} \in \mathscr{N}} \alpha_j(\boldsymbol{n}) \sum_{k=1}^{J} \mu_{\boldsymbol{n},j,k}(\Lambda^L) \mu_I^{(k)} \beta_k \\
&= R_*.
\end{aligned}$$

Hence $a'(0)/m_H > 1$ if and only if $R_* > 1$, which establishes the result. □

The next result connects R_* to the possible limiting values of $\bar{T}_\infty^{(v)}$.

Theorem 4.9. *If $R_* \leq 1$, then $\bar{T}_\infty^{(v)} \xrightarrow{\text{a.s.}} 0$ as $v \to \infty$. If $R_* > 1$, then*

$$\min\{\bar{T}_\infty^{(v)}, |\bar{T}_\infty^{(v)} - \tau|\} \xrightarrow{\text{a.s.}} 0 \text{ as } v \to \infty,$$

where $\tau > 0$ satisfies

$$\tau = m_H^{-1} a(\tau). \tag{4.16}$$

Proof. The result of Theorem 4.7 clearly holds for ${m_H^{(v)}}^{-1} \frac{1}{m^{(v)}} A_\bullet^{(v)}(t)$. Consequently, there exists an $F \in \mathscr{F}$ with $\mathrm{P}(F) = 1$ such that, for all $\omega \in F$, we have

$$\begin{aligned}
&\lim_{v \to \infty} \sup_{t \geq 0} \left| \frac{1}{m^{(v)}} A_\bullet^{(v)}(t, \omega) {m_H^{(v)}}^{-1} - a(t) m_H^{-1} \right| = 0 \text{ and} \\
&\lim_{v \to \infty} \bar{T}_0^{(v)}(\omega) = 0.
\end{aligned} \tag{4.17}$$

Writing $\bar{T}_\infty^{(v)} = \bar{T}_0^{(v)} + \frac{1}{N^{(v)}} A_\bullet^{(v)}(T_\infty^{(v)})$ (cf. (4.13)), we use $m_H^{(v)} = \frac{N^{(v)}}{m^{(v)}}$ to find

$$\bar{T}_\infty^{(v)} = \bar{T}_0^{(v)} + \frac{1}{m^{(v)}} A_\bullet^{(v)}(\bar{T}_\infty^{(v)}) {m_H^{(v)}}^{-1}.$$

Consider first the case where $R_* \leq 1$. Then, by Theorem 4.8, the only crossing point (where $t = m_H^{-1} a(t)$) occurs at $t = 0$. Fix an $\omega \in F$ and let $t > 0$ be arbitrary, with $\Delta_t = t - m_H^{-1} a(t) > 0$. Since $\bar{T}_0^{(v)} \xrightarrow{\text{a.s.}} 0$ as $v \to \infty$, there exists $\nu_1 \in \mathbb{N}$ such

that $\bar{T}_0^{(v)}(\omega) < \Delta_t/4$ for all $v \geq v_1$. By Theorem 4.7 there exists $v_2 \in \mathbb{N}$ such that, for all $v \geq v_2$,

$$\frac{1}{m^{(v)}} A_\bullet^{(v)}(t,\omega) m_H^{(v)^{-1}} \leq m_H^{-1} a(t) + \frac{\Delta_t}{2}.$$

Then, for all $v \geq v_3 = \max\{v_1, v_2\}$, we have

$$\begin{aligned} \bar{T}_0^{(v)}(\omega) + \frac{1}{m^{(v)}} A_\bullet^{(v)}(t,\omega) m_H^{(v)^{-1}} &\leq \frac{\Delta_t}{4} + m_H^{-1} a(t) + \frac{\Delta_t}{2} \\ &= t - \Delta_t + \frac{3\Delta_t}{4} \\ &= t - \frac{\Delta_t}{4} < t, \end{aligned}$$

which implies that $\bar{T}_\infty^{(v)}(\omega) < t$ for all $v \geq v_3$. Since $t > 0$ is arbitrary, it follows that $\bar{T}_\infty^{(v)}(\omega) \to 0$ as $v \to \infty$; this holds for all $\omega \in \mathscr{F}$. Noting that $\mathrm{P}(F) = 1$ then gives $\bar{T}_\infty^{(v)} \xrightarrow{\text{a.s.}} 0$ as $v \to \infty$, which proves the result for the case $R_* \leq 1$.

We next consider the case $R_* > 1$ in which, by Theorem 4.8, there are two crossing points (at $t = 0$ and $t = \tau$). Let $\varepsilon > 0$ be small, such that the set $[\varepsilon, \tau - \varepsilon]$ is non-empty, and let t be an arbitrary member of $[\varepsilon, \tau - \varepsilon]$, with $\bar{\Delta}_t = m_H^{-1} a(t) - t$. By Theorem 4.7, there exists $v_4 \in \mathbb{N}$ such that, for all $v \geq v_4$, we have

$$\frac{1}{m^{(v)}} A_\bullet^{(v)}(t,\omega) m_H^{(v)^{-1}} \geq m_H^{-1} a(t) - \frac{\bar{\Delta}_t}{2}.$$

Then

$$\begin{aligned} \bar{T}_0^{(v)}(\omega) + \frac{1}{m^{(v)}} A_\bullet^{(v)}(t,\omega) m_H^{(v)^{-1}} - t &\geq m_H^{-1} a(t) - \frac{\bar{\Delta}_t}{2} - t \\ &= \bar{\Delta}_t - \frac{\bar{\Delta}_t}{2} \\ &= \frac{\bar{\Delta}_t}{2}. \end{aligned}$$

Noting that $\min_{t \in [\varepsilon, \tau - \varepsilon]} \bar{\Delta}_t = \min\{m_H^{-1} a(\tau - \varepsilon) - \tau + \varepsilon, m_H^{-1} a(\varepsilon) - \varepsilon\} > 0$, we therefore have

$$\min_{t \in [\varepsilon, \tau - \varepsilon]} \left\{ \bar{T}_0^{(v)}(\omega) + \frac{1}{m^{(v)}} A_\bullet^{(v)}(t,\omega) m_H^{(v)^{-1}} - t \right\} > 0 \tag{4.18}$$

for $v \geq v_4$.

Now consider the case where $t > \tau + \varepsilon$ is arbitrary. Let $\hat{\Delta}_t = t - m_H^{-1} a(t)$, which is positive since $t > \tau$. Applying Theorem 4.7, there exists $\nu_5 \in \mathbb{N}$ such that, for all $\nu \geq \nu_5$, we have

$$\frac{1}{m^{(\nu)}} A_\bullet^{(\nu)}(t,\omega) {m_H^{(\nu)}}^{-1} \leq m_H^{-1} a(t) + \frac{\hat{\Delta}_t}{2}.$$

The fact that $\bar{T}_0^{(\nu)} \xrightarrow{\text{a.s.}} 0$ establishes the existence of $\nu_6 \in \mathbb{N}$ such that, for all $\nu \geq \nu_6$, we have $\bar{T}_0^{(\nu)}(\omega) \leq \dfrac{\hat{\Delta}_t}{4}$. Then, for $\nu \geq \nu_7 \coloneqq \max\{\nu_5, \nu_6\}$, we have

$$\begin{aligned} t - \left(\frac{1}{m^{(\nu)}} A_\bullet^{(\nu)}(t,\omega) {m_H^{(\nu)}}^{-1} + \bar{T}_0^{(\nu)}(\omega) \right) &\geq t - m_H^{-1} a(t) - \frac{\hat{\Delta}_t}{2} - \frac{\hat{\Delta}_t}{4} \\ &= \frac{\hat{\Delta}_t}{4}. \end{aligned}$$

Now $\inf\limits_{t > \tau+\varepsilon} \{\hat{\Delta}_t\} = \tau + \varepsilon - m_H^{-1} a(\tau+\varepsilon) > 0$ so, for $\nu \geq \nu_7$, we have

$$\inf_{t > \tau+\varepsilon} \left\{ t - \left(\bar{T}_0^{(\nu)}(\omega) + \frac{1}{m^{(\nu)}} A_\bullet^{(\nu)}(t,\omega) {m_H^{(\nu)}}^{-1} \right) \right\} > 0. \tag{4.19}$$

Combining (4.18) and (4.19), we find that

$$\min\left\{ \bar{T}_\infty^{(\nu)}(\omega), \left| \bar{T}_\infty^{(\nu)}(\omega) - \tau \right| \right\} \leq \varepsilon$$

for $\nu \geq \nu_7$. Then, as $\varepsilon > 0$ can be made arbitrarily small, we have

$$\lim_{\nu \to \infty} \min\left\{ \bar{T}_\infty^{(\nu)}(\omega), \left| \bar{T}_\infty^{(\nu)}(\omega) - \tau \right| \right\} = 0,$$

for arbitrary $\omega \in F$. Finally, noting that $\mathrm{P}(F) = 1$ gives

$$\min\left\{ \bar{T}_\infty^{(\nu)}, \left| \bar{T}_\infty^{(\nu)} - \tau \right| \right\} \xrightarrow{\text{a.s.}} 0 \text{ as } \nu \to \infty,$$

establishing the claim in the case $R_* > 1$. □

We next consider the case where the terminal infectious pressure is greater than some positive constant; for $\varepsilon \in (0, \tau)$, define $\bar{T}_{\infty,\varepsilon}^{(\nu)}$ as the smallest solution in $[\varepsilon, \infty)$ of $t = \bar{T}_0^{(\nu)} + \dfrac{1}{N^{(\nu)}} A_\bullet^{(\nu)}(t)$, provided one exists, with $\bar{T}_{\infty,\varepsilon}^{(\nu)} = \bar{T}_\infty^{(\nu)}$ otherwise. Theorem 4.9 gives rise to the following corollary.

Corollary 4.10. *Suppose that $R_* > 1$. Then*

$$\bar{T}_{\infty,\varepsilon}^{(v)} \xrightarrow{\text{a.s.}} \tau \text{ as } v \to \infty.$$

Proof. By the argument in the proof of Theorem 4.9, the solutions to

$$t = \bar{T}_0^{(v)} + \frac{1}{N^{(v)}} A_\bullet^{(v)}(t)$$

get arbitrarily close to 0 or τ as v becomes sufficiently large; for $\delta > 0$ the solutions are contained in $[0,\delta) \cup (\tau-\delta, \tau+\delta)$ for sufficiently large v. But δ can be arbitrarily small, in particular smaller than ε, so that, in the same notation as the proof of Theorem 4.9, $\bar{T}_{\infty,\varepsilon}^{(v)}(\omega) \in (\tau-\delta, \tau+\delta)$, from which the result follows. □

4.6 Joint central limit theorem for final state random variables

We now consider a multivariate central limit theorem for final state random variables defined on $\tilde{\mathscr{E}}^{(v)}$, first introduced for multitype SIR epidemics in Ball and O'Neill [1999]. For $t \geq 0$ and $\boldsymbol{n} \in \mathscr{N}$ denote, by $R_1^{(\boldsymbol{n})}(t), R_2^{(\boldsymbol{n})}(t), \ldots, R_p^{(\boldsymbol{n})}(t)$, a collection of p final outcome quantities associated to the epidemic $\mathscr{E}_{\boldsymbol{n}}(\Lambda^L, \boldsymbol{\pi})$ with $\pi_i = \exp(-\kappa_i t)$ ($i \in \mathscr{J}$). These may be any quantities which are summable over households; examples include the number of type-1 individuals infected and the number of households with more than 2 members infected. Further examples may, in an inferential setting, include quantities such as score statistics for parameters of interest, such as λ_{11}^L. Note that the severity is another such example, although we often denote this quantity separately due to the important role it plays in the analysis; we make use of the severity in the sequel in order to establish a multivariate central limit theorem for these final state random variables.

We consider the sequence of epidemics $\tilde{\mathscr{E}}^{(v)}$, as in Section 4.5.2. For $j = 1, 2, \ldots, p$ and $k = 1, 2, \ldots, m_{\boldsymbol{n}}^{(v)}$ let $R_j^{(\boldsymbol{n},k)}(t)$ ($t \geq 0$) denote independent and

identically distributed copies of $R_j(t)$. We then define, for $t \geq 0$,

$$R_{\bullet j}^{(v)}(t) = \sum_{\boldsymbol{n} \in \mathscr{N}} \sum_{k=1}^{m_{\boldsymbol{n}}^{(v)}} R_j^{(\boldsymbol{n},k)}(t), \qquad j = 1,2,\ldots,p. \tag{4.20}$$

Of interest is a joint central limit theorem for

$$\tilde{\boldsymbol{R}}_{\bullet}(\bar{T}_{\infty}^{(v)}) = (R_{\bullet 1}^{(v)}(\bar{T}_{\infty}^{(v)}), R_{\bullet 2}^{(v)}(\bar{T}_{\infty}^{(v)}), \ldots, R_{\bullet p}^{(v)}(\bar{T}_{\infty}^{(v)}), A_{\bullet}^{(v)}(\bar{T}_{\infty}^{(v)})).$$

In order to establish this, we require further notation. For $j = 1,2,\ldots,p$ and $\boldsymbol{n} \in \mathscr{N}$, suppose that $r_j^{(\boldsymbol{n})}(t) = \mathrm{E}[R_j^{(\boldsymbol{n},1)}(t)] < \infty$. This corresponds to the finiteness of the mean contribution from a category-$\boldsymbol{n}$ household (in which each individual is exposed to t units of global infectious pressure) to the j^{th} final state random variable. Write $r_j^{(v)}(t) = \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}}^{(v)} r_j^{(\boldsymbol{n})}(t)$ and $r_j(t) = \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} r_j^{(\boldsymbol{n})}(t)$. Let $\boldsymbol{C}^{(\boldsymbol{n})}(t,s) = \left[C_{ij}^{(\boldsymbol{n})}(t,s)\right]$ be the (symmetric) covariance function such that

$$C_{ij}^{(\boldsymbol{n})}(t,s) = \mathrm{cov}\left(R_i^{(\boldsymbol{n},1)}(t), R_j^{(\boldsymbol{n},1)}(s)\right), \qquad i,j = 1,2,\ldots,p,$$

and

$$C_{i,p+1}^{(\boldsymbol{n})}(t,s) = C_{p+1,i}^{(\boldsymbol{n})}(t,s) = \mathrm{cov}\left(R_i^{(\boldsymbol{n},1)}(t), A^{(\boldsymbol{n},1)}(s)\right), \qquad i = 1,2,\ldots,p,$$

with

$$C_{p+1,p+1}^{(\boldsymbol{n})}(t,s) = \mathrm{cov}\left(A^{(\boldsymbol{n},1)}(t), A^{(\boldsymbol{n},1)}(s)\right).$$

We assume that, for $j = 1,2,\ldots,p$, the process $R_j^{(\boldsymbol{n},1)}(t)$ only has jumps when an individual becomes infected. Finally, we assume the existence of $\zeta > 0$ such that

$$\mathrm{E}\left[\left(\sup_{t \geq 0} \left|R_j^{(\boldsymbol{n},1)}(t)\right|\right)^{2+\zeta}\right] < \infty \tag{4.21}$$

for every $\boldsymbol{n} \in \mathscr{N}$ and $j = 1,2,\ldots,p$. The condition in (4.21) ensures that all elements of $\boldsymbol{C}^{(\boldsymbol{n})}(t,s)$ are finite for any $t,s \geq 0$. We begin with convergence results for final state random variables.

Theorem 4.11. *For $j = 1,2,\ldots,p$ and $t \geq 0$, we have*

$$\frac{1}{m^{(v)}} R_{\bullet j}^{(v)}(t) \xrightarrow{\text{a.s.}} r_j(t) \text{ as } v \to \infty.$$

Proof. The result follows by applying the strong law of large numbers as well as noting that there are a finite number of household categories. □

In order to establish the following result, we let $T > 0$ and write

$$\tilde{\boldsymbol{R}}_{\bullet}^{(v)}(t) = (R_{\bullet 1}^{(v)}(t), R_{\bullet 2}^{(v)}(t), \ldots, R_{\bullet p}^{(v)}(t), A_{\bullet}^{(v)}(t)),$$

where the severity process is included explicitly due to the important role it plays in the analysis. We let $\xrightarrow{\text{w}}$ denote weak convergence on the space of bounded functions from $[0,T]$ to $\mathbb{R}^{p+1}$, endowed with the supremum metric. We use this topology, rather than the weaker Skorohod topology, since in the present application more easily checkable conditions for asymptotic tightness are available.

Theorem 4.12. *Let $T > 0$ and $\tilde{\boldsymbol{R}}_{\bullet,T}^{(v)} = \{\tilde{\boldsymbol{R}}_{\bullet}^{(v)}(t) : 0 \leq t \leq T\}$. Then*

$$\frac{1}{\sqrt{m^{(v)}}} \left(\tilde{\boldsymbol{R}}_{\bullet,T}^{(v)} - \mathrm{E}[\tilde{\boldsymbol{R}}_{\bullet,T}^{(v)}] \right) \xrightarrow{\text{w}} \boldsymbol{X}_T \text{ as } v \to \infty,$$

where $\boldsymbol{X}_T = \{(X_1(t), X_2(t), \ldots, X_p(t), X_A(t)) : 0 \leq t \leq T\}$ is a zero-mean Gaussian process with covariance function given by $\boldsymbol{C}(t,s) = \sum_{\boldsymbol{n} \in \mathcal{N}} \theta_{\boldsymbol{n}} \boldsymbol{C}^{(\boldsymbol{n})}(t,s)$ $(t,s \in [0,T])$.

Proof. To show that the finite-dimensional distributions of $\frac{1}{\sqrt{m^{(v)}}} \left(\tilde{\boldsymbol{R}}_{\bullet,T}^{(v)} - \mathrm{E}[\tilde{\boldsymbol{R}}_{\bullet,T}^{(v)}] \right)$ converge to those of $\boldsymbol{X}_T$, we use the Cramér-Wold device. For $i = 1,2,\ldots,p+1$,

let $q_i \in \mathbb{N}$ and, for $j = 1, 2, \ldots, q_i$, let $\lambda_{ij} \in \mathbb{R}$ and $t_{ij} \geq 0$. We have

$$\begin{aligned}
&\frac{1}{\sqrt{m^{(v)}}} \sum_{\boldsymbol{n} \in \mathscr{N}} \sum_{k=1}^{m_{\boldsymbol{n}}^{(v)}} \sum_{i=1}^{p+1} \sum_{j=1}^{q_i} \lambda_{ij} \left(\tilde{R}_i^{(\boldsymbol{n},k)}(t_{ij}) - \mathrm{E}[\tilde{R}_i^{(\boldsymbol{n},k)}(t_{ij})] \right) \\
&\quad = \sum_{\boldsymbol{n} \in \mathscr{N}} \sqrt{\theta_{\boldsymbol{n}}^{(v)}} \frac{1}{\sqrt{m_{\boldsymbol{n}}^{(v)}}} \sum_{k=1}^{m_{\boldsymbol{n}}^{(v)}} \left\{ \sum_{i=1}^{p+1} \sum_{j=1}^{q_i} \lambda_{ij} \left(\tilde{R}_i^{(\boldsymbol{n},k)}(t_{ij}) - \mathrm{E}[\tilde{R}_i^{(\boldsymbol{n},k)}(t_{ij})] \right) \right\} \\
&\quad \xrightarrow{\mathrm{D}} X, \text{ as } v \to \infty, \text{ where} \\
&X \sim N\left(0, \sum_{i=1}^{p+1} \sum_{j=1}^{q_i} \sum_{a=1}^{p+1} \sum_{b=1}^{q_a} \lambda_{ij} \lambda_{ab} \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \mathrm{cov}\left(\tilde{R}_i^{(\boldsymbol{n},1)}(t_{ij}), \tilde{R}_a^{(\boldsymbol{n},1)}(t_{ab}) \right) \right),
\end{aligned}$$

since all terms in the inner (braced) sum on the second line are independent and identically distributed. It is then clear by considering linear combinations of $\boldsymbol{X}_T(t_{ij})$ that the desired convergence holds.

It remains to check asymptotic tightness which, by Van Der Vaart and Wellner [1996], Lemma 1.4.3, can be checked for each component of $\tilde{\boldsymbol{R}}_T$ separately; we are required to prove tightness for a typical general final state random variable, R, say.

In order to establish asymptotic tightness, define

$$\|f\|_T = \sup_{0 \leq t \leq T} |f(t)|.$$

Further, for $\varepsilon > 0$, define the bracketing number $N_{[]}^{(v)}(\varepsilon, T)$ as the minimum number of sets N_ε in a partition $[0,T] = \bigcup_{i=1}^{N_\varepsilon} \mathscr{A}_{\varepsilon j}^{(v)}$ such that, for each set $\mathscr{A}_{\varepsilon j}^{(v)}$, we have

$$\sum_{\boldsymbol{n} \in \mathscr{N}} \sum_{k=1}^{m_{\boldsymbol{n}}^{(v)}} \mathrm{E}\left[\max_{t,s \in \mathscr{A}_{\varepsilon j}^{(v)}} \left| \frac{1}{\sqrt{m^{(v)}}} \left(R^{(\boldsymbol{n},k)}(t) - R^{(\boldsymbol{n},k)}(s) \right) \right|^2 \right] \leq \varepsilon^2. \tag{4.22}$$

Applying Van Der Vaart and Wellner [1996], Theorem 2.11.9, the sequence $(m^{(v)})^{-\frac{1}{2}} \left(R^{(v)} - \mathrm{E}\left[R^{(v)} \right] \right)$ $(v = 1, 2, \ldots)$ is asymptotically tight provided the following conditions hold:

(i) For any $d > 0$, we have

$$\sum_{\boldsymbol{n}\in\mathscr{N}}\sum_{k=1}^{m_{\boldsymbol{n}}^{(v)}} \mathrm{E}\left[\left\|\frac{1}{\sqrt{m^{(v)}}}R^{(\boldsymbol{n},k)}\right\|_T \mathbf{1}_{\left\{\left\|\frac{1}{\sqrt{m^{(v)}}}R^{(\boldsymbol{n},k)}\right\|_T > d\right\}}\right] \to 0 \text{ as } v\to\infty.$$

(ii) For any sequence (δ_v) satisfying $\delta_v \downarrow 0$ as $v\to\infty$, we have

$$\sup_{s,t\in[0,T]:|s-t|<\delta_v}\sum_{\boldsymbol{n}\in\mathscr{N}}\sum_{k=1}^{m_{\boldsymbol{n}}^{(v)}}\frac{1}{m^{(v)}}\mathrm{E}\left[\left(R^{(\boldsymbol{n},k)}(t)-R^{(\boldsymbol{n},k)}(s)\right)^2\right]\to 0 \text{ as } v\to\infty.$$

(iii) For any sequence (δ_v) satisfying $\delta_v \downarrow 0$ as $v\to\infty$, the bracketing number $N_{[]}^{(v)}(\varepsilon,T)$ satisfies

$$\int_0^{\delta_v}\sqrt{\log N_{[]}^{(v)}(\varepsilon,T)}d\varepsilon \to 0 \text{ as } v\to\infty.$$

To show (i), let $X_{\boldsymbol{n}} = \dfrac{1}{\sqrt{m^{(v)}}}\sup_{t\geq 0}\left|R^{(\boldsymbol{n},1)}(t)\right|$. Then

$$\begin{aligned}
&\sum_{\boldsymbol{n}\in\mathscr{N}}\sum_{k=1}^{m_{\boldsymbol{n}}^{(v)}} \mathrm{E}\left[\left\|\frac{1}{\sqrt{m^{(v)}}}R^{(\boldsymbol{n},k)}(t)\right\|_T \mathbf{1}_{\left\{\left\|\frac{1}{\sqrt{m^{(v)}}}R^{(\boldsymbol{n},k)}(t)\right\|_T > d\right\}}\right] \\
&= \sum_{\boldsymbol{n}\in\mathscr{N}}\sum_{k=1}^{m_{\boldsymbol{n}}^{(v)}}\mathrm{E}\left[X_{\boldsymbol{n}}\mathbf{1}_{\{X_{\boldsymbol{n}}>d\}}\right] \\
&= \sum_{\boldsymbol{n}\in\mathscr{N}} m_{\boldsymbol{n}}^{(v)}\mathrm{E}\left[X_{\boldsymbol{n}}\mathbf{1}_{\{X_{\boldsymbol{n}}>d\}}\right] \\
&= d^{-(1+\zeta)}\sum_{\boldsymbol{n}\in\mathscr{N}} m_{\boldsymbol{n}}^{(v)}\mathrm{E}\left[X_{\boldsymbol{n}}\mathbf{1}_{\{X_{\boldsymbol{n}}>d\}}d^{(1+\zeta)}\right] \\
&\leq d^{-(1+\zeta)}\sum_{\boldsymbol{n}\in\mathscr{N}} m_{\boldsymbol{n}}^{(v)}\mathrm{E}\left[X_{\boldsymbol{n}}^{2+\tilde{\zeta}}\right] \\
&\leq d^{-(1+\zeta)}\sum_{\boldsymbol{n}\in\mathscr{N}} \frac{m_{\boldsymbol{n}}^{(v)}}{{m^{(v)}}^{1+\frac{\zeta}{2}}}\mathrm{E}\left[\left(\sup_{t\geq 0}\left|R^{(\boldsymbol{n},1)}(t)\right|\right)^{2+\zeta}\right] \\
&= d^{-(1+\zeta)}\sum_{\boldsymbol{n}\in\mathscr{N}} \theta_{\boldsymbol{n}}^{(v)}{m^{(v)}}^{-\frac{\zeta}{2}}\mathrm{E}\left[\left(\sup_{t\geq 0}\left|R^{(\boldsymbol{n},1)}(t)\right|\right)^{2+\zeta}\right] \\
&\to 0 \text{ as } v\to\infty,
\end{aligned}$$

where the fifth line follows using the same argument as a proof of Markov's

inequality. Thus, condition (i) is satisfied.

Turning to condition (ii), let (δ_v) be any sequence satisfying $\delta_v \downarrow 0$ as $v \to \infty$. Assume, without loss of generality, that $0 < s < t$ and consider a typical household of category $\boldsymbol{n} \in \mathscr{N}$. We attach, to each type-i individual in this household, an independent Poisson process having rate κ_i. The first point in this individual's Poisson process corresponds to them becoming globally infected, and points thereafter are disregarded. Denote by $F_{\boldsymbol{n}}(s,t)$ the event that there is at least one point in the collection of Poisson processes, in a category-$\boldsymbol{n}$ household, occurring in the time interval $(s,t]$. Then, since $R^{(\boldsymbol{n},k)}(t)$ only jumps when an infection occurs, we have

$$\begin{aligned}
\mathrm{E}\left[\left(R^{(\boldsymbol{n},k)}(t) - R^{(\boldsymbol{n},k)}(s)\right)^2\right] &\leq 4\mathrm{E}\left[\left(\sup_{t\geq 0}\left|R^{(\boldsymbol{n},k)}(t)\right|\right)^2 \mathbf{1}_{\{F_{\boldsymbol{n}}(s,t)\}}\right] \\
&= 4\mathrm{E}\left[\left(\sup_{t\geq 0}\left|R^{(\boldsymbol{n},k)}(t)\right|\right)^2\right](1-\mathrm{P}(F^c_{\boldsymbol{n}}(s,t))) \\
&= 4\mathrm{E}\left[\left(\sup_{t\geq 0}\left|R^{(\boldsymbol{n},k)}(t)\right|\right)^2\right]\left(1-\exp\left(-\sum_{i=1}^{J} n_i\kappa_i(t-s)\right)\right) \\
&\leq 4\mathrm{E}\left[\left(\sup_{t\geq 0}\left|R^{(\boldsymbol{n},k)}(t)\right|\right)^2\right]\sum_{i=1}^{J} n_i\kappa_i(t-s) \\
&= (t-s)K_{\boldsymbol{n}}, \text{ say,}
\end{aligned}$$

from which it is clear that condition (ii) is satisfied. Moreover, a partition of $[0,T]$ into sets $\mathscr{A}^{(v)}_{\varepsilon j}$, intervals of length $L_\varepsilon = \frac{\varepsilon^2}{\sum_{\boldsymbol{n}\in\mathscr{N}} K_{\boldsymbol{n}}}$ (the final interval may have shorter length) is sufficient to ensure that (4.22) holds. Then $N^{(v)}_{[]}(\varepsilon,T) \leq \frac{c}{\varepsilon^2}$, where $c = 2T\sum_{\boldsymbol{n}\in\mathscr{N}} K_{\boldsymbol{n}}$, giving

$$\begin{aligned}
\int_0^{\delta_v}\sqrt{\log N^{(v)}_{[]}(\varepsilon,T)}\,d\varepsilon &\leq \int_0^{\delta_v}\sqrt{\log\left(\frac{c}{\varepsilon^2}\right)}\,d\varepsilon \\
&= \frac{\sqrt{c}}{2}\int_{\log\left(\frac{c}{\delta_v^2}\right)}^{\infty}\sqrt{u}\exp\left(-\frac{u}{2}\right)du \\
&\leq \frac{\sqrt{c}}{2}\int_{\log\left(\frac{c}{\delta_v^2}\right)}^{\infty}\exp\left(-\frac{u}{4}\right)du \to 0 \text{ as } v\to\infty,
\end{aligned}$$

which follows by taking the substitution $u = \log\left(\frac{c}{\varepsilon^2}\right)$ and noting that $\sqrt{u} \le \exp\left(\frac{u}{4}\right)$ for $u \ge 0$. Hence condition (iii) is satisfied, concluding the proof of asymptotic tightness and of the theorem. □

We next derive a joint central limit theorem for the general final state random variables and the severity in the embedding process.

Theorem 4.13. *Assume that $R_* > 1$ and, as $v \to \infty$,*

(i) $\sqrt{m^{(v)}}\left(\theta_{\boldsymbol{n}}^{(v)} - \theta_{\boldsymbol{n}}\right) \to 0 \quad (\boldsymbol{n} \in \mathscr{N})$,

(ii) $\sqrt{m^{(v)}}\bar{T}_0^{(v)} \xrightarrow{\mathrm{p}} 0$,

(iii) $\sqrt{N^{(v)}}\left(m_H^{(v)} - m_H\right) \to 0$.

Then, for any $\varepsilon \in (0, \tau)$,

$$(m^{(v)})^{-\frac{1}{2}} \begin{pmatrix} R_{\bullet 1}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)} r_1(\tau) \\ R_{\bullet 2}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)} r_2(\tau) \\ \vdots \\ R_{\bullet p}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)} r_p(\tau) \\ A_{\bullet}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)} a(\tau) \end{pmatrix} \xrightarrow{\mathrm{D}} N(\mathbf{0}_{p+1}, \boldsymbol{H}\boldsymbol{C}(\tau,\tau)\boldsymbol{H}^\top) \text{ as } v \to \infty,$$

where $\boldsymbol{r}'(\tau) = (r_1'(\tau), r_2'(\tau), \ldots, r_p'(\tau))^\top$ and

$$\boldsymbol{H} = \begin{pmatrix} \boldsymbol{I}_p & \frac{1}{m_H - a'(\tau)}\boldsymbol{r}'(\tau) \\ \mathbf{0}_p^\top & \frac{m_H}{m_H - a'(\tau)} \end{pmatrix}. \tag{4.23}$$

Proof. Consider first the severity process. Recalling that $a^{(v)}(t) = \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}}^{(v)} a_{\boldsymbol{n}}(t)$, we have

$$\begin{aligned} (m^{(v)})^{-\frac{1}{2}}\left[A_{\bullet}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)}a(\tau)\right] =& (m^{(v)})^{-\frac{1}{2}}\left[A_{\bullet}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)}a^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)})\right] \\ &+ (m^{(v)})^{\frac{1}{2}}\left[a^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - a(\bar{T}_{\infty,\varepsilon}^{(v)})\right] \\ &+ (m^{(v)})^{\frac{1}{2}}\left[a(\bar{T}_{\infty,\varepsilon}^{(v)}) - a(\tau)\right] \\ =& \tilde{A}^{(v)} + \tilde{B}^{(v)} + \tilde{C}^{(v)}, \text{ say.} \end{aligned} \tag{4.24}$$

Now $\bar{T}^{(v)}_{\infty,\varepsilon} \xrightarrow{\text{a.s.}} \tau$ as $v \to \infty$ by Corollary 4.10. Using Theorem 4.12 as well as the continuous mapping theorem (Van Der Vaart and Wellner [1996], Theorem 1.3.6) implies that $\tilde{A}^{(v)} \xrightarrow{\text{D}} X_A(\tau)$ as $v \to \infty$. Further, since $a_{\boldsymbol{n}}(t)$ is continuous for every $\boldsymbol{n} \in \mathscr{N}$, we have $a_{\boldsymbol{n}}(\bar{T}^{(v)}_{\infty,\varepsilon}) \xrightarrow{\text{a.s.}} a_{\boldsymbol{n}}(\tau)$ as $v \to \infty$. Combining this with condition (i) yields $\tilde{B}^{(v)} \xrightarrow{\text{a.s.}} 0$ as $v \to \infty$ and, in particular, that $B^{(v)} \xrightarrow{\text{p}} 0$ as $v \to \infty$. The mean value theorem implies that $\tilde{C}^{(v)}$ satisfies

$$\begin{aligned}\tilde{C}^{(v)} =& (m^{(v)})^{\frac{1}{2}} \left[a(\bar{T}^{(v)}_{\infty,\varepsilon}) - a(\tau) \right] \\ =& (m^{(v)})^{\frac{1}{2}} a'(\xi^{(v)}) \left(\bar{T}^{(v)}_{\infty,\varepsilon} - \tau \right)\end{aligned}$$

for some $\xi^{(v)}$ lying between $\bar{T}^{(v)}_{\infty,\varepsilon}$ and τ. Recalling that $\tau = m_H^{-1} a(\tau)$ as well as the definition of $\bar{T}^{(v)}_{\infty,\varepsilon}$ yields

$$\begin{aligned}(m^{(v)})^{\frac{1}{2}} \left[\bar{T}^{(v)}_{\infty,\varepsilon} - \tau \right] =& (m^{(v)})^{\frac{1}{2}} \bar{T}^{(v)}_0 + (m^{(v)})^{\frac{1}{2}} \left(\frac{1}{N^{(v)}} A^{(v)}_\bullet(\bar{T}^{(v)}_{\infty,\varepsilon}) - \tau \right) \\ =& (m^{(v)})^{\frac{1}{2}} \bar{T}^{(v)}_0 + (m^{(v)})^{-\frac{1}{2}} \left(\frac{m^{(v)}}{N^{(v)}} A^{(v)}_\bullet(\bar{T}^{(v)}_{\infty,\varepsilon}) - \tau m^{(v)} \right) \\ =& (m^{(v)})^{\frac{1}{2}} \bar{T}^{(v)}_0 + (m^{(v)})^{-\frac{1}{2}} \left(\frac{1}{m^{(v)}_H} A^{(v)}_\bullet(\bar{T}^{(v)}_{\infty,\varepsilon}) - a(\tau) m_H^{-1} m^{(v)} \right) \\ =& (m^{(v)})^{\frac{1}{2}} \bar{T}^{(v)}_0 + (m^{(v)})^{-\frac{1}{2}} \left(A^{(v)}_\bullet(\bar{T}^{(v)}_{\infty,\varepsilon}) - a(\tau) m^{(v)} \right) m_H^{-1} \\ &+ (m^{(v)})^{-\frac{1}{2}} \left(A^{(v)}_\bullet(\bar{T}^{(v)}_{\infty,\varepsilon}) \right) \left(\frac{1}{m^{(v)}_H} - \frac{1}{m_H} \right). \qquad (4.25)\end{aligned}$$

We denote by $o_p(1)$ any random variable X_v satisfying $X_v \xrightarrow{\text{p}} 0$ as $v \to \infty$. The first term of (4.25) is $o_p(1)$ by condition (ii). Considering the final term of (4.25), we have

$$\begin{aligned}(m^{(v)})^{-\frac{1}{2}} \left(A^{(v)}_\bullet(\bar{T}^{(v)}_{\infty,\varepsilon}) \right) \left(\frac{1}{m^{(v)}_H} - \frac{1}{m_H} \right) &= (m^{(v)})^{-1} A^{(v)}_\bullet(\bar{T}^{(v)}_{\infty,\varepsilon}) (m^{(v)})^{\frac{1}{2}} \left(\frac{m_H - m^{(v)}_H}{m_H m^{(v)}_H} \right) \\ &= (m^{(v)})^{-1} A^{(v)}_\bullet(\bar{T}^{(v)}_{\infty,\varepsilon}) \frac{\sqrt{N^{(v)}}(m_H - m^{(v)}_H)}{m_H (m^{(v)}_H)^{\frac{3}{2}}} \\ &= o_p(1).\end{aligned}$$

This follows by first using Theorem 4.7 to observe that $(m^{(v)})^{-1} A^{(v)}_\bullet(\bar{T}^{(v)}_{\infty,\varepsilon})$ is

bounded almost surely (and therefore bounded in probability) and then by applying condition (iii).

Combining (4.24) with (4.25) and using Slutsky's theorem (Van Der Vaart and Wellner [1996], Example 1.4.7) yields

$$\begin{aligned}(m^{(v)})^{-\frac{1}{2}}\left[A_{\bullet}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)})-m^{(v)}a(\tau)\right] &= (m^{(v)})^{-\frac{1}{2}}\left[A_{\bullet}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)})-m^{(v)}a^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)})\right] \\ &\quad + (m^{(v)})^{-\frac{1}{2}}\left[A_{\bullet}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)})-m^{(v)}a(\tau)\right] m_H^{-1}a'(\xi^{(v)}) \\ &\quad + o_p(1). \qquad (4.26)\end{aligned}$$

Further, for $j = 1,2,\ldots,p$, we have

$$\begin{aligned}(m^{(v)})^{-\frac{1}{2}}\left[R_{\bullet j}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)})-m^{(v)}r_j(\tau)\right] =& (m^{(v)})^{-\frac{1}{2}}\left[R_{\bullet j}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)})-m^{(v)}r_j^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)})\right] \\ &+ (m^{(v)})^{\frac{1}{2}}\left[r_j^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)})-r_j(\bar{T}_{\infty,\varepsilon}^{(v)})\right] \\ &+ (m^{(v)})^{\frac{1}{2}}\left[r_j(\bar{T}_{\infty,\varepsilon}^{(v)})-r_j(\tau)\right] \\ &= \hat{A}_j^{(v)}+\hat{B}_j^{(v)}+\hat{C}_j^{(v)}, \text{ say.}\end{aligned}$$

Now $\hat{A}_j^{(v)} \xrightarrow{\mathrm{D}} X_j(\tau)$ as $v \to \infty$, using Theorem 4.12, the continuous mapping theorem, and the fact that $\bar{T}_{\infty}^{(v)} \xrightarrow{\text{a.s.}} \tau$ as $v \to \infty$. We also have $\hat{B}_j^{(v)} \xrightarrow{\mathrm{p}} 0$ (cf. $\tilde{B}^{(v)} \xrightarrow{\mathrm{p}} 0$) as $v \to \infty$, since $r_j^{(\boldsymbol{n})}(t)$ is continuous for every $\boldsymbol{n} \in \mathscr{N}$ and $j = 1,2,\ldots,p$ and by applying condition (i). Rewriting $\hat{C}_j^{(v)}$ (cf. $\tilde{C}^{(v)}$) we use the mean value theorem to find

$$\begin{aligned}\hat{C}_j^{(v)} =& (m^{(v)})^{\frac{1}{2}}\left[r_j(\bar{T}_{\infty,\varepsilon}^{(v)})-r_j(\tau)\right] \\ =& (m^{(v)})^{\frac{1}{2}} r_j'(\xi_j^{(v)})\left(\bar{T}_{\infty,\varepsilon}^{(v)}-\tau\right), \qquad j = 1,2,\ldots,p,\end{aligned}$$

for some $\xi_j^{(v)}$ lying between $\bar{T}_{\infty,\varepsilon}^{(v)}$ and τ. Arguing as in the derivation of (4.26)

then yields, for $j = 1, 2, \ldots, p$,

$$
\begin{aligned}
&(m^{(v)})^{-\frac{1}{2}} \left[R_{\bullet j}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)} r_j(\tau) \right] \\
&\quad = (m^{(v)})^{-\frac{1}{2}} \left[R_{\bullet j}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)} r_j^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) \right] \\
&\qquad + (m^{(v)})^{-\frac{1}{2}} \left[A_{\bullet}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)} a(\tau) \right] m_H^{-1} r_j'(\xi_j^{(v)}) \\
&\qquad + o_p(1). \qquad (4.27)
\end{aligned}
$$

Using (4.26), (4.27) and Slutsky's theorem, we reach

$$
(m^{(v)})^{-\frac{1}{2}} \boldsymbol{G} \begin{pmatrix} R_{\bullet 1}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)} r_1(\tau) \\ R_{\bullet 2}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)} r_2(\tau) \\ \vdots \\ R_{\bullet p}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)} r_p(\tau) \\ A_{\bullet}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)} a(\tau) \end{pmatrix} \xrightarrow{\mathrm{D}} \begin{pmatrix} X_1(\tau) \\ X_2(\tau) \\ \vdots \\ X_p(\tau) \\ X_A(\tau) \end{pmatrix} \text{ as } v \to \infty,
$$

where $\boldsymbol{G} = \begin{pmatrix} \boldsymbol{I}_p & -m_H^{-1} \boldsymbol{r}'(\tau) \\ \boldsymbol{0}_p^\top & 1 - m_H^{-1} a'(\tau) \end{pmatrix}$. Then

$$
(m^{(v)})^{-\frac{1}{2}} \begin{pmatrix} R_{\bullet 1}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)} r_1(\tau) \\ R_{\bullet 2}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)} r_2(\tau) \\ \vdots \\ R_{\bullet p}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)} r_p(\tau) \\ A_{\bullet}^{(v)}(\bar{T}_{\infty,\varepsilon}^{(v)}) - m^{(v)} a(\tau) \end{pmatrix} \xrightarrow{\mathrm{D}} N(\boldsymbol{0}_{p+1}, \boldsymbol{H}\boldsymbol{C}(\tau,\tau)\boldsymbol{H}^\top) \text{ as } v \to \infty,
$$

with $\boldsymbol{H} = \boldsymbol{G}^{-1} = \begin{pmatrix} \boldsymbol{I}_p & \frac{1}{m_H - a'(\tau)} \boldsymbol{r}'(\tau) \\ \boldsymbol{0}_p^\top & \frac{m_H}{m_H - a'(\tau)} \end{pmatrix}$, which establishes the result. □

4.7 Branching process approximation and probability of a major outbreak

In this section we define a branching process approximation for the epidemic model $\mathscr{E}^{(v)}$ among $N^{(v)}$ members in which there is one individual initially infected, chosen in a manner which we specify in the sequel, as well as a limiting

branching process as $v \to \infty$. The initial construction of $\mathscr{E}^{(v)}$ and of the approximating branching processes follow Ball and Lyne [2001], Section 4.1; we include this construction below for completeness, beginning with the relevant quantities.

We begin by defining a linear ordering $\prec$ on $\mathscr{N}$ as follows. For $\boldsymbol{n}, \boldsymbol{n}' \in \mathscr{N}$, $\boldsymbol{n} \prec \boldsymbol{n}'$ provided

(i) $|\boldsymbol{n}| \leq |\boldsymbol{n}'|$ and,

(ii) if $|\boldsymbol{n}| = |\boldsymbol{n}'|$ and $\boldsymbol{n} \neq \boldsymbol{n}'$, then $n_i < n'_i$ with $i = \min\{j : n_j \neq n'_j\}$.

We use $\prec$ to list the household categories in increasing order. Let $\boldsymbol{n}^{(i)}$ denote the household category of household i ($i = 1, 2, \ldots, m^{(v)}$). We label the individuals in the i^{th} household $(i, j, 1), (i, j, 2), \ldots, (i, j, n^{(j)})$. We also, for $i = 1, 2, \ldots, m^{(v)}$, $j \in \mathscr{J}$ and $k = 1, 2, \ldots, n_j^{(i)}$ assign to individual (i, j, k) the population-based label $\sum_{i'=1}^{i-1} |\boldsymbol{n}^{(i')}| + \sum_{j'=1}^{j-1} n_{j'}^{(i)} + k$ and the type-j-based label $\sum_{i'=1}^{i-1} n_j^{(i')} + k$, with the relevant sums being zero if vacuous. Let $g_j^{(v)}$ ($j \in \mathscr{J}$) be the map which takes an individual's type-j-based label to their corresponding population-based label.

It is helpful to also define, for $v = 1, 2, \ldots$,

$$F_j^{(v)} = \frac{\sum_{i=1}^{j} \kappa_i \gamma_i^{(v)}}{\sum_{i=1}^{J} \kappa_i \gamma_i^{(v)}}, \qquad j \in \mathscr{J},$$

and

$$\tilde{F}_j^{(v)}(\boldsymbol{n}) = \sum_{\boldsymbol{r} \prec \boldsymbol{n}} \alpha_j^{(v)}(\boldsymbol{r}) \qquad \boldsymbol{n} \in \mathscr{N}, j \in \mathscr{J},$$

where, for completeness, $\tilde{F}_j^{(v)}(\mathbf{0}) = 0$ ($j \in \mathscr{J}$).

For $\boldsymbol{n} \in \mathscr{N}$ and for $j, k \in \mathscr{J}$, let $\eta_G^{(j,k)}$ denote a homogeneous Poisson process with intensity $\kappa_j \beta_k \gamma_k^{(v)}$. Such a process gives the times, relative to the time of infection of a given type-j individual, at which that individual makes contacts globally with type-k individuals. Similarly, let $\eta_L^{(\boldsymbol{n},i,j)}$ denote a homogeneous Poisson process, with intensity $n_k \lambda_{jk}^L$, analogous to $\eta_G^{(j,k)}$ but instead giving the times of local contacts with the individuals in the category-$\boldsymbol{n}$ household. Then define the infectious career of a typical type-j individual in a category-$\boldsymbol{n}$ house-

hold by writing

$$\mathscr{H}^{(\boldsymbol{n},j)} = \{T_I^{(j)}, \eta_G^{(j,k)}, \eta_L^{(\boldsymbol{n},j,k)} (k \in \mathscr{J})\}.$$

We now define, for $\boldsymbol{n} \in \mathscr{N}$ and $j \in \mathscr{J}$, the following independent sets of random quantities:

(i) $\mathscr{H}_{ik}^{(\boldsymbol{n},j)}$ $(i = 1,2,\ldots; k = 1,2,\ldots,n_j)$ independent and identically distributed according to $\mathscr{H}^{(\boldsymbol{n},j)}$;

(ii) $\mathscr{C}_{ik}^{(\boldsymbol{n},j)}$ $(i = 1,2,\ldots; k = 1,2,\ldots)$ independent and uniformly distributed on $\{1,2,\ldots,n_j\}$;

(iii) U_{jk} $(j \in \mathscr{J}, k = 0,1,\ldots)$ independent and uniformly distributed on $(0,1)$;

(iv) U_k and $\tilde{U}_k$ $(k = 0,1,\ldots)$ independent and uniformly distributed on $(0,1)$.

We then construct $\mathscr{E}^{(v)}$ as follows. The initial infective is chosen, for convenience, in a manner such that the initial generation of the approximating branching process is consistent with all of the subsequent generations of the branching process. Other choices of initial infective are possible, although they complicate the exposition. Thus, the initial infective is assumed to be type j with probability $P_j^{(v)}$ given by

$$P_j^{(v)} = \frac{\kappa_j \gamma_j^{(v)}}{\sum_{j'=1}^{J} \kappa_{j'} \gamma_{j'}^{(v)}}, \qquad j \in \mathscr{J}.$$

Consequently the initial infective, assuming they are type j, receives the type-j-based label $\lfloor N_j^{(v)} U_{j0} \rfloor + 1$ and population-based label $g_j^{(v)}(\lfloor N_j^{(v)} U_{j0} \rfloor + 1)$. Consider the k^{th} individual of type j to be infected in the i^{th} household of category $\boldsymbol{n}$ $(i = 1,2,\ldots,m^{(v)}, j \in \mathscr{J}, \boldsymbol{n} \in \mathscr{N},$ and $k = 1,2\ldots,n_j)$. This individual adopts the infectious career $\mathscr{H}_{i,k}^{(\boldsymbol{n},j)}$. The individual contacted by the k^{th} type-j local contact in household i' has household-based label $(i', j, \mathscr{C}_{i'k}^{(\boldsymbol{n},j)})$ $(j \in \mathscr{J}, k = 1,2,\ldots)$. Finally, for $j \in \mathscr{J}$ and $k = 1,2\ldots$, the individual contacted at the k^{th} type-j global contact receives the individual-j-based label $\lfloor N_j^{(v)} U_{jk} \rfloor + 1$. Denote by $Z_E^{(v)}$ the total number of households infected in $\mathscr{E}^{(v)}$.

The aforementioned collection of quantities can also be used to form realisations of a multitype single-household epidemic in a household of category

$\boldsymbol{n} \in \mathscr{N}$ with a single initial infective of type $j \in \mathscr{J}$ (provided $\boldsymbol{n}$ is such that $n_j > 0$). Denote such a typical realisation by $\mathscr{E}_i^{(\boldsymbol{n},j)}$, which is formed using the points of $\mathscr{H}_{ik}^{(\boldsymbol{n},j)}$ $(j \in \mathscr{J}; k = 1,2,\ldots,n_j)$ pertaining to local contacts to decide the times of local contacts. The l^{th} individual of type k is labelled (k,l), the initial infective being $(j,1)$. Which individuals become contacted is determined by $\mathscr{C}_{ik}^{(\boldsymbol{n},j)}$ $(j \in \mathscr{J}; k = 1,2,\ldots)$, i.e. by sampling among individuals of the relevant type uniformly at random. For the purposes of constructing this local epidemic, the points of the global contact process are ignored. Let $D_i^{(\boldsymbol{n},j)}$ denote the duration of $\mathscr{E}_i^{(\boldsymbol{n},j)}$. Turning to the global contacts, let $\eta_i^{(\boldsymbol{n},j)}$ denote the point process of all global contacts emanating from $\mathscr{E}_i^{(\boldsymbol{n},j)}$, i.e. the times (relative to the beginning of $\mathscr{E}_i^{(\boldsymbol{n},j)}$) at which individuals would make global contacts if permitted to do so.

We now define the approximating (single-type) branching process $\mathscr{B}^{(v)}$ $(v = 1,2,\ldots)$. All individuals in the process (including, by construction, the initial ancestor) have the same distribution. We outline this distribution for the initial individual. First, sample a type from $\mathscr{J}$ by choosing type j if and only if $U_0 \in [F_{j-1}^{(v)}, F_j^{(v)})$. Next, sample a household category among the households containing type-j individuals by choosing $\boldsymbol{n}$ if and only if $\tilde{U}_0 \in [\tilde{F}_j^{(v)}(\boldsymbol{n}'), \tilde{F}_j^{(v)}(\boldsymbol{n}))$ (here $\boldsymbol{n}'$ is the immediate predecessor of $\boldsymbol{n}$, with the convention that $\mathbf{0}$ immediately precedes $(1,0,\ldots,0)$). A typical individual, given the pair $(\boldsymbol{n},j)$, lives until age according to $D^{(\boldsymbol{n},j)}$ and reproduces at the points of $\eta^{(\boldsymbol{n},j)}$, cf. the duration and global contact points of $\mathscr{E}^{(\boldsymbol{n},j)}$. The collection of random variables $(U_k, \tilde{U}_k)$ $(k = 1,2,\ldots)$ can be used to describe the behaviour of all descendants of the initial ancestor in an analogous manner. The limiting branching process $\mathscr{B}$ is defined in the same fashion, but instead using the asymptotic values $F_j = \lim_{v\to\infty} F_j^{(v)}$ and $\tilde{F}(\boldsymbol{n}) = \lim_{v\to\infty} \tilde{F}^{(v)}(\boldsymbol{n})$. Let $Z^{(v)}$ (Z) and $D^{(v)}$ (D) denote respectively the total progeny and the offspring distribution of the branching process $\mathscr{B}^{(v)}$ $(\mathscr{B})$.

Lemma 4.14. *We have* $D^{(v)} \xrightarrow{\text{D}} D$ *as* $v \to \infty$.

Proof. Consider a typical individual in the branching process $\mathscr{B}^{(v)}$. It is clear that, given the same pair $(\boldsymbol{n},j)$, their offspring distribution will be the same as a corresponding individual (i.e. with the same pair) in $\mathscr{B}$. Let $X^{(v)}$ denote

a random variable which takes value j with probability $F_j^{(v)} - F_{j-1}^{(v)}$ ($j \in \mathscr{J}$). Ordering the household categories (omitting $\mathbf{0}$) according to $\prec$ and labelling them $\{1,2,\ldots,\}$, let $Y^{(v)}$ correspond to the label of a household chosen by the procedure in $\mathscr{B}^{(v)}$. Consider a typical $\boldsymbol{n} \in \mathscr{N}$ and suppose its associated label is i. Then

$$\begin{aligned} \mathrm{P}(Y^{(v)} \leq i) &= \sum_{j \in \mathscr{J}} \mathrm{P}(Y^{(v)} \leq i, X^{(v)} = j) \\ &= \sum_{j \in \mathscr{J}} \mathrm{P}(Y^{(v)} \leq i | X^{(v)} = j) \mathrm{P}(X^{(v)} = j) \\ &= \sum_{j \in \mathscr{J}} \tilde{F}_j^{(v)}(\boldsymbol{n})(F_j^{(v)} - F_{j-1}^{(v)}). \end{aligned} \tag{4.28}$$

Defining X and Y in the obvious fashion, an identical argument yields

$$\mathrm{P}(Y \leq i) = \sum_{j \in \mathscr{J}} \tilde{F}_j(\boldsymbol{n})(F_j - F_{j-1}).$$

Taking the limit of (4.28) as $v \to \infty$, it is clear that $Y^{(v)} \xrightarrow{\mathrm{D}} Y$ as $v \to \infty$, from which the result follows. □

We note the following corollary which follows from Lemma 4.14.

Corollary 4.15. *Let $p_{ext}^{(v)} = \mathrm{P}(Z^{(v)} < \infty)$ denote the extinction probability of $\mathscr{B}^{(v)}$ and define p_{ext} as the extinction probability of $\mathscr{B}$. We have $p_{ext}^{(v)} \to p_{ext}$ as $v \to \infty$.*

Proof. By Lemma 4.14 we have that $D^{(v)} \xrightarrow{\mathrm{D}} D$ as $v \to \infty$. Using Britton et al. [2007], Lemma 4.1, then establishes the result. □

We next consider a calculation which underpins the coupling of $\mathscr{B}^{(v)}$ to $\mathscr{E}^{(v)}$.

Lemma 4.16. *Suppose $m^{(v)}$ is such that $m^{(v)} \to \infty$ as $v \to \infty$. We have*

$$\mathrm{P}(Z^{(v)} \leq \log(m^{(v)})) \to p_{ext} \text{ as } v \to \infty.$$

Proof. Recalling the definition of p_{ext} and $p_{\text{ext}}^{(\nu)}$ in Corollary 4.15, we have

$$\begin{aligned}\limsup_{\nu\to\infty} \mathrm{P}(Z^{(\nu)} \leq \log(m^{(\nu)})) &\leq \limsup_{\nu\to\infty} \mathrm{P}(Z^{(\nu)} < \infty) \\ &= \limsup_{\nu\to\infty} p_{\text{ext}}^{(\nu)} = p_{\text{ext}}. \end{aligned} \tag{4.29}$$

Next, letting $k \in \mathbb{N}$, we have

$$\liminf_{\nu\to\infty} \mathrm{P}(Z^{(\nu)} \leq \log(m^{(\nu)})) \geq \liminf_{\nu\to\infty} \mathrm{P}(Z^{(\nu)} \leq k),$$

since $\log(m^{(\nu)}) > k$ for all sufficiently large ν. There are a finite number of paths of the branching process $\mathscr{B}^{(\nu)}$ along which $Z^{(\nu)} \leq k$. The probabilities of these paths converge to the probabilities of the corresponding paths in $\mathscr{B}$, by Lemma 4.14, whence

$$\liminf_{\nu\to\infty} \mathrm{P}(Z^{(\nu)} \leq k) = \mathrm{P}(Z \leq k).$$

Further, we have

$$\mathrm{P}(Z \leq k) \uparrow \mathrm{P}(Z < \infty) = p_{\text{ext}} \text{ as } k \uparrow \infty,$$

which together with (4.29) establishes the result. □

We next make the connection between the construction of the epidemic $\mathscr{E}^{(\nu)}$ to the approximating branching process $\mathscr{B}^{(\nu)}$. By the properties of Poisson processes which are also used in, for example, the proof of Lemma 4.1, it is clear that the method for choosing the type of the individual infected globally in $\mathscr{E}^{(\nu)}$ is the same as that of the process for choosing types in $\mathscr{B}^{(\nu)}$. Then the number of infected households in $\mathscr{E}^{(\nu)}$ and the number of individuals in the branching process $\mathscr{B}^{(\nu)}$ agree exactly until there is a repeat global contact (i.e. a global contact with a previously infected household) in $\mathscr{E}^{(\nu)}$. We let $K^{(\nu)}$ denote the number of global contacts in $\mathscr{E}^{(\nu)}$ that have occurred by the first time that a previously infected household in $\mathscr{E}^{(\nu)}$ is contacted globally, noting the following useful result regarding $K^{(\nu)}$.

Lemma 4.17. *We have*

$$\mathrm{P}\left(K^{(v)} \leq \log(m^{(v)})\right) \to 0 \text{ as } v \to \infty.$$

Proof. Let $H_i^{(v)}$ $(i = 1, 2, \ldots)$ denote the household contacted by the i^{th} global contact in $\mathscr{E}^{(v)}$, with the convention that $H_0^{(v)}$ is the household in which the initial infective resides. The probability that a global contact is with any given household is bounded above by

$$\max_{i \in \mathscr{J}} \max_{\boldsymbol{n} \in \mathscr{N}} \frac{n_i}{N_i^{(v)}} \leq \frac{n_{\max}}{N^{(v)} \min_{i \in \mathscr{J}} \gamma_i^{(v)}}$$

Then, noting the connection with the birthday problem, we have

$$\begin{aligned}
\mathrm{P}\left(K^{(v)} \leq \log(m^{(v)})\right) &= \mathrm{P}\left(\bigcup_{i=0}^{\lfloor \log(m^{(v)}) \rfloor} \bigcup_{j=i+1}^{\lfloor \log(m^{(v)}) \rfloor} \{H_i = H_j\}\right) \\
&\leq \sum_{i=0}^{\lfloor \log(m^{(v)}) \rfloor} \sum_{j=i+1}^{\lfloor \log(m^{(v)}) \rfloor} \mathrm{P}\left(H_i^{(v)} = H_j^{(v)}\right) \\
&\leq \frac{\left(\log(m^{(v)}) + 1\right)\left(\log(m^{(v)})\right) \mathrm{P}\left(H_1^{(v)} = H_2^{(v)}\right)}{2},
\end{aligned} \tag{4.30}$$

where

$$\mathrm{P}\left(H_1^{(v)} = H_2^{(v)}\right) \leq \frac{n_{\max}}{N^{(v)} \min_{i \in \mathscr{J}} \gamma_i^{(v)}} \tag{4.31}$$

Substituting (4.31) into (4.30) and taking the limit as $v \to \infty$ then establishes the result. □

Lemma 4.18. *We have*

$$P_E^{(v)} = \left|\mathrm{P}(Z_E^{(v)} \leq \log(m^{(v)})) - \mathrm{P}(Z^{(v)} \leq \log(m^{(v)}))\right| \to 0 \text{ as } v \to \infty.$$

Proof. We establish the result by conditioning on whether or not the event

$\{K^{(v)} \leq \log(m^{(v)})\}$ occurs. We have

$$\begin{aligned}\mathrm{P}(Z_E^{(v)} \leq \log(m^{(v)})) = \mathrm{P}(Z_E^{(v)} \leq \log(m^{(v)}), K^{(v)} \leq \log(m^{(v)})) \\ + \mathrm{P}(Z_E^{(v)} \leq \log(m^{(v)}), K^{(v)} > \log(m^{(v)}))\end{aligned}$$

and

$$\begin{aligned}\mathrm{P}(Z^{(v)} \leq \log(m^{(v)})) = \mathrm{P}(Z^{(v)} \leq \log(m^{(v)}), K^{(v)} \leq \log(m^{(v)})) \\ + \mathrm{P}(Z^{(v)} \leq \log(m^{(v)}), K^{(v)} > \log(m^{(v)})).\end{aligned}$$

By the discussion above, the epidemic $\mathscr{E}^{(v)}$ and the branching process $\mathscr{B}^{(v)}$ agree until we have a repeated global contact in a household. Thus

$$\begin{aligned}P_E^{(v)} &= \left|\mathrm{P}\left(Z_E^{(v)} \leq \log(m^{(v)}), K^{(v)} \leq \log(m^{(v)})\right) - \mathrm{P}\left(Z^{(v)} \leq \log(m^{(v)}), K^{(v)} \leq \log(m^{(v)})\right)\right| \\ &\leq \mathrm{P}\left(K^{(v)} \leq \log(m^{(v)})\right) \to 0,\end{aligned}$$

by applying Lemma 4.17. □

We denote the occurrence of a major outbreak by the event

$$G^{(v)} = \left\{Z_E^{(v)} > \log(m^{(v)})\right\}.$$

The probability that, in the limit $v \to \infty$, the event $G^{(v)}$ occurs is then readily obtained.

Theorem 4.19. *We have*

$$\mathrm{P}(G^{(v)}) \to 1 - p_{ext} \text{ as } v \to \infty.$$

Proof. The result follows immediately by applying Lemma 4.16 and Lemma 4.18. □

4.8 Connecting results

We now make the connection between the epidemic model $\mathscr{E}^{(v)}$ and the embedded process $\tilde{\mathscr{E}}^{(v)}$ to derive a multivariate central limit theorem for final state

random variables, conditional upon a major outbreak. We begin with a result that connects the solutions to the crossing problem in the embedding construction to the scaled severity δ of Section 4.4.

Theorem 4.20. *The equation $t = m_H^{-1} a(t)$ is equivalent to*

$$t = \sum_{\boldsymbol{n} \in \mathscr{N}} \tilde{\alpha}_{\boldsymbol{n}} \|\boldsymbol{n}\|^{-1} \sum_{r=0}^{n} \binom{\boldsymbol{n}}{r} (1-\pi)^r \pi^{n-r} \sum_{j=1}^{J} \mu_{n-r,r,j} \mu_I^{(j)} \beta_j,$$

where $\pi_j = \exp(-t\kappa_j)$ and $\tilde{\alpha}_{\boldsymbol{n}}$ ($\boldsymbol{n} \in \mathscr{N}$) is the proportion of individuals which reside in a household of category $\boldsymbol{n}$.

Proof. Throughout the proof, we suppress the explicit dependence of $\boldsymbol{\pi}$ on t for ease of exposition. Firstly note that, for $j \in \mathscr{J}$ and $\boldsymbol{n} \in \mathscr{N}$, the expected severity arising from type-j individuals in $\mathscr{E}_{\boldsymbol{n}}(\Lambda^L, \boldsymbol{\pi})$ is, by Lemma B.2, given by

$$s_{\boldsymbol{n},j}(t) = \mu_I^{(j)} \sum_{r=0}^{n} \binom{\boldsymbol{n}}{r} (1-\pi)^r \pi^{n-r} \mu_{n-r,r,j}.$$

Secondly, note that $m_H^{-1} \theta_{\boldsymbol{n}} = \tilde{\alpha}_{\boldsymbol{n}} \|\boldsymbol{n}\|^{-1}$ for each $\boldsymbol{n} \in \mathscr{N}$. Then

$$\begin{aligned}
m_H^{-1} a(t) &= m_H^{-1} \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} a_{\boldsymbol{n}}(t) \\
&= m_H^{-1} \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \mathrm{E}\left[A^{(\boldsymbol{n},1)}(t)\right] \\
&= m_H^{-1} \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \sum_{j=1}^{J} \beta_j \mathrm{E}\left[\tilde{A}^{(j,\boldsymbol{n})}(t)\right] \\
&= m_H^{-1} \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \sum_{j=1}^{J} \beta_j s_{\boldsymbol{n},j}(t) \\
&= m_H^{-1} \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \sum_{j=1}^{J} \beta_j \mu_I^{(j)} \sum_{r=0}^{n} \binom{\boldsymbol{n}}{r} (1-\pi)^r (\pi)^{n-r} \mu_{n-r,r,j} \\
&= \sum_{\boldsymbol{n} \in \mathscr{N}} \tilde{\alpha}_{\boldsymbol{n}} \|\boldsymbol{n}\|^{-1} \sum_{j=1}^{J} \beta_j \mu_I^{(j)} \sum_{r=0}^{n} \binom{\boldsymbol{n}}{r} (1-\pi)^r (\pi)^{n-r} \mu_{n-r,r,j} \\
&= \sum_{\boldsymbol{n} \in \mathscr{N}} \tilde{\alpha}_{\boldsymbol{n}} \|\boldsymbol{n}\|^{-1} \sum_{r=0}^{n} \binom{\boldsymbol{n}}{r} (1-\pi)^r \pi^{n-r} \sum_{j=1}^{J} \mu_{n-r,r,j} \mu_I^{(j)} \beta_j,
\end{aligned}$$

which completes the proof. □

Considering the original epidemic model $\mathscr{E}^{(\nu)}$, let $\Upsilon_i^{(\nu)}$ (cf. Υ_i in Section 4.4) denote the sum of the infectious periods of all type-i individuals ultimately

infected in $\mathscr{E}^{(v)}$ ($i \in \mathscr{J}$). Let $\Upsilon^{(v)} = \sum_{j=1}^{J} \Upsilon_i \beta_i$ and $\tilde{\Upsilon}^{(v)} = \Upsilon^{(v)}/N^{(v)}$, and suppose that $\boldsymbol{R}^{(v)} = (R_1^{(v)}, R_2^{(v)}, \ldots, R_p^{(v)})$ ($v = 1, 2, \ldots$) denotes a collection of p typical final state random variables associated to $\mathscr{E}^{(v)}$, with $\tilde{\boldsymbol{R}}^{(v)} = \boldsymbol{R}^{(v)}/m^{(v)}$.

We use an adapted embedding construction in order to achieve a joint central limit theorem for the severity and final state random variables, conditional upon a major outbreak. A similar method is used in Ball et al. [2024], who run an epidemic until $\log(m)$ communities are infected, before switching to the embedding construction and considering appropriate bounding constructions. Instead, we use the embedding construction throughout, defining the epidemic on all $m^{(v)}$ households as follows. Pick one individual to have threshold equal to zero, with all other individuals having thresholds as in Section 4.5.1, and set $T_0^{(v)} = 0$ ($v = 1, 2, \ldots$). The individual with threshold set to zero is the initial infective, and they can be chosen in the same manner as the initial infective in $\mathscr{E}^{(v)}$ is chosen. Denote by $Z_\bullet^{(v)}(t)$ the number of households infected in the embedding construction when each individual is exposed to t units of global infection. We run the epidemic until $\log(m^{(v)})$ households are infected. If the construction does not reach $\log(m^{(v)})$ households infected, we begin a new construction (independent of any previous constructions) until we reach an epidemic which does not die out before infecting $\log(m^{(v)})$ households. The above construction yields the correct final size for a major outbreak in $\mathscr{E}^{(v)}$ among $m^{(v)}$ households, so that $\boldsymbol{R}_\bullet^{(v)}(\bar{T}_\infty^{(v)})$ is a realisation of the final state random variables $\boldsymbol{R}^{(v)}$ conditional upon a major outbreak. We then define

$$T'^{(v)} = \inf\left\{t > 0 : Z_\bullet^{(v)}(t) \geq \log(m^{(v)})\right\},$$

and note that $T'^{(v)}$ is almost surely finite since $Z_\bullet^{(v)}(t) \to m^{(v)}$ as $t \to \infty$. The embedding construction continues after the population has been exposed to $T'^{(v)}$ units of global infection. In order to do this, we must account for the infected individuals and the accumulation of infectious pressure from the first construction. Let (i, j) correspond to the j^{th} type-i individual in the population. We set $\tilde{L}_{ij} = \infty$ as the threshold for individual (i, j) if they were infected in the first part of the construction, otherwise we set $\tilde{L}_{ij} = L_{ij} - T'^{(v)}$. These new thresholds

have the required distribution, owing to the memoryless property of the exponential distribution. We can study the remaining epidemic spread by considering an embedding construction with these new thresholds and with initial pressure given by setting

$$\bar{T}_0^{(v)} = \frac{1}{N^{(v)}} A_\bullet^{(v)} \left(\bar{T}'^{(v)} \right) - \bar{T}'^{(v)}. \tag{4.32}$$

We provide an upper bound for $\bar{T}_0^{(v)}$ by considering an upper bounding construction as follows. Let $\bar{T}_0^{(v,U)} = \bar{T}_0^{(v)} + \frac{1}{N^{(v)}} A_0^{(v,U)}$, where $A_0^{(v,U)}$ is defined as follows. Let $\boldsymbol{s}_1^{(v)}, \boldsymbol{s}_2^{(v)}, \ldots, \boldsymbol{s}_{\lceil \log(m^{(v)}) \rceil}^{(v)}$ denote vectors containing the number of remaining susceptibles of each type in the $\lceil \log(m^{(v)}) \rceil$ infected households, with $s_{ij}^{(v)}$ denoting the j^{th} element of $\boldsymbol{s}_i^{(v)}$ ($i = 1, 2, \ldots, \lceil \log(m^{(v)}) \rceil, j \in \mathscr{J}$). Then define

$$A_{0,i}^{(v,U)} = \sum_{j=1}^{\lceil \log(m^{(v)}) \rceil} s_{ij}^{(v)} I_j^{(i)}, \qquad i \in \mathscr{J},$$

independently of the rest of the construction, with

$$A_0^{(v,U)} = \sum_{i=1}^{J} A_{0,i}^{(v,U)}.$$

Thus $\bar{T}_0^{(v,U)}$ is the initial infectious pressure on each individual for an upper bounding process in which all individuals in the first $\log(m^{(v)})$ households become infected. Let $A^{(i,\boldsymbol{n})}$ denote the severity of $\mathscr{E}_{\boldsymbol{n},e_i}$ in the highly locally infectious case, with $\tilde{A}_j$ ($j = 1, 2, \ldots$) denoting independent and identically distributed copies of

$$\tilde{A} = \sum_{i \in \mathscr{J}} \sum_{\boldsymbol{n} \in \mathscr{N}} A^{(i,\boldsymbol{n})}.$$

It is clear that $\mathrm{E}[A^{(i,\boldsymbol{n})}] < \infty$ for each $i \in \mathscr{J}$ and $\boldsymbol{n} \in \mathscr{N}$, whence $\mathrm{E}[\tilde{A}] < \infty$.

Then

$$\begin{aligned}
\sqrt{m^{(v)}}\bar{T}_0^{(v)} &= \frac{\sqrt{m^{(v)}}}{N^{(v)}} T_0^{(v)} \\
&\leq \frac{1}{\sqrt{m^{(v)}}} T_0^{(v)} \\
&\leq \frac{1}{\sqrt{m^{(v)}}} T_0^{(v,U)} \\
&\leq \frac{1}{\sqrt{m^{(v)}}} \sum_{j=1}^{\lceil \log(m^{(v)}) \rceil} \tilde{A}_j \\
&= \frac{\lceil \log(m^{(v)}) \rceil}{\sqrt{m^{(v)}}} \frac{1}{\lceil \log(m^{(v)}) \rceil} \sum_{j=1}^{\lceil \log(m^{(v)}) \rceil} \tilde{A}_j \\
&\xrightarrow{\text{p}} 0 \text{ as } v \to \infty,
\end{aligned}$$

by using the law of large numbers and the fact that $\frac{\lceil \log(m^{(v)}) \rceil}{\sqrt{m^{(v)}}} \to 0$ as $v \to \infty$. Then $\sqrt{m^{(v)}}\bar{T}_0^{(v)} \xrightarrow{\text{p}} 0$ as $v \to \infty$, so that the conditions of Theorem 4.13 hold with this new $\bar{T}_0^{(v)}$.

We run the embedding process after the reset on all households, with individual thresholds suitably adjusted and with initial infectious pressure given by (4.32). Let $\hat{A}_\bullet^{(v)} = \left\{ \hat{A}_\bullet^{(v)}(t) : t \geq 0 \right\}$ denote the severity process for this reset epidemic, defining $\hat{R}_{\bullet j}^{(v)}$ $(j = 1, 2, \ldots, p)$ analogously. A consequence of using this approach is a discrepancy between $\hat{A}_\bullet^{(v)}$ and $A_\bullet^{(v)}$; the contributions from previously infected households will, due to the individual threshold changes, have a different distribution. For $\boldsymbol{n} \in \mathcal{N}$, let $\tilde{m}_{\boldsymbol{n}}^{(v)}$ denote the number of households of category $\boldsymbol{n}$ uninfected in the first construction (i.e. before the reset). Then

$$\hat{A}_\bullet^{(v)}(t) = \sum_{\boldsymbol{n} \in \mathcal{N}} \sum_{k=1}^{m_{\boldsymbol{n}}^{(v)} - \tilde{m}_{\boldsymbol{n}}^{(v)}} \hat{A}^{(\boldsymbol{n},k)}(t) + \sum_{\boldsymbol{n} \in \mathcal{N}} \sum_{k=1}^{\tilde{m}_{\boldsymbol{n}}^{(v)}} A^{(\boldsymbol{n},k)}(t),$$

where $\hat{A}^{(\boldsymbol{n},k)}(t)$ is the contribution from the k^{th} category-$\boldsymbol{n}$ household that contains a reset individual. We treat this discrepancy in the following lemma.

Lemma 4.21. *We have*

$$\frac{1}{\sqrt{m^{(v)}}} \sum_{\boldsymbol{n} \in \mathcal{N}} \sum_{k=1}^{m_{\boldsymbol{n}}^{(v)} - \tilde{m}_{\boldsymbol{n}}^{(v)}} A^{(\boldsymbol{n},k)}(t) \xrightarrow{\text{a.s.}} 0 \text{ as } v \to \infty.$$

Proof. We have

$$\begin{aligned}\frac{1}{\sqrt{m^{(v)}}}\sum_{\boldsymbol{n}\in\mathscr{N}}\sum_{k=1}^{m_{\boldsymbol{n}}^{(v)}-\tilde{m}_{\boldsymbol{n}}^{(v)}}A^{(\boldsymbol{n},k)}(t) &\leq \frac{1}{\sqrt{m^{(v)}}}\sum_{\boldsymbol{n}\in\mathscr{N}}\sum_{k=1}^{\lceil\log(m^{(v)})\rceil}A^{(\boldsymbol{n},k)}(t)\\ &= \sum_{\boldsymbol{n}\in\mathscr{N}}\frac{1}{\sqrt{m^{(v)}}}\sum_{k=1}^{\lceil\log(m^{(v)})\rceil}A^{(\boldsymbol{n},k)}(t)\\ &= \sum_{\boldsymbol{n}\in\mathscr{N}}\frac{\lceil\log(m^{(v)})\rceil}{\sqrt{m^{(v)}}}\frac{1}{\lceil\log(m^{(v)})\rceil}\sum_{k=1}^{\lceil\log(m^{(v)})\rceil}A^{(\boldsymbol{n},k)}(t)\\ &\xrightarrow{\text{a.s.}} 0 \text{ as } v\to\infty,\end{aligned}$$

which follows from the strong law of large numbers and the fact that $\frac{\lceil\log(m^{(v)})\rceil}{\sqrt{m^{(v)}}}\to 0$ as $v\to\infty$. □

An immediate consequence of Lemma 4.21 is that Theorem 4.7 and Theorem 4.12 apply to the $\hat{A}_{\bullet}^{(v)}$ process. Moreover, a similar result holds for each $\hat{R}_{\bullet j}^{(v)}$ process; we use this to derive the law of large numbers and central limit theorems of interest. In the following result, $\tilde{R}^{(v)}$ denotes a typical element of $\tilde{\boldsymbol{R}}^{(v)}$.

Theorem 4.22. *Suppose that $R_*>1$. We have, as $v\to\infty$,*

(i) $\tilde{\Upsilon}^{(v)}|G^{(v)}\xrightarrow{\text{p}}\tau$,

(ii) $\tilde{R}^{(v)}|G^{(v)}\xrightarrow{\text{p}}r(\tau)$.

Proof. We begin by showing that, for any $\varepsilon>0$, there exists $\delta>0$ and $\nu_2\in\mathbb{N}$ such that, for all $v\geq\nu_2$, we have

$$\mathrm{P}\left(Z_E^{(v)}>\delta m^{(v)}|G^{(v)}\right)\geq 1-\frac{\varepsilon}{2}. \tag{4.33}$$

Suppose that, for some $\delta\in(0,1)$, the number of households infected by $\mathscr{E}^{(v)}$ is not more than $\delta m^{(v)}$. Then, using (4.31), the probability that a global infectious contact is with a previously uninfected household is at least $1-\delta^*$, where

$$\delta^*=\min\left\{1,\frac{\delta n_{\max}}{\min_{i\in\mathscr{J}}\gamma_i^{(v)}}\right\}.$$

Now consider the branching process $\mathscr{B}^{(v)}(\delta)$, in which descendants are born as in $\mathscr{B}^{(v)}$, but are deleted at birth with probability δ^*. (Individuals who are deleted at birth have no offspring and do not contribute to the total progeny.) Define the branching process $\mathscr{B}(\delta)$ in a similar fashion. Let $Z^{(v)}(\delta)$ denote the total progeny of $\mathscr{B}^{(v)}(\delta)$ and denote by $p_{\text{ext}}^{(v)}(\delta)$ the extinction probability of $\mathscr{B}^{(v)}(\delta)$. Then, noting that $\mathscr{B}^{(v)}$ is a lower-bounding process for $\mathscr{E}^{(v)}$ whilst the number of infected households in $\mathscr{E}^{(v)}$ is not more than $\delta m^{(v)}$, we find

$$\mathrm{P}\left(Z_E^{(v)} \geq \delta m^{(v)}\right) \geq \mathrm{P}\left(Z^{(v)}(\delta) \geq \delta m^{(v)}\right) \geq 1 - p_{\text{ext}}^{(v)}(\delta).$$

We then have

$$p_{\text{ext}}^{(v)}(\delta) \to p_{\text{ext}}(\delta) \text{ as } v \to \infty,$$

where $p_{\text{ext}}(\delta)$ is the extinction probability of $\mathscr{B}(\delta)$ (see Britton et al. [2007], Lemma 4.1). Now $p_{\text{ext}}(\delta) \downarrow p_{\text{ext}}$ as $\delta \downarrow 0$, so δ can be chosen such that

$$\frac{1 - p_{\text{ext}}(\delta)}{1 - p_{\text{ext}}} \geq 1 - \frac{\varepsilon}{4}. \tag{4.34}$$

For any $\delta > 0$ there exists $\nu_0 \in \mathbb{N}$ such that $\delta m^{(v)} \geq \log(m^{(v)})$ for $v \geq \nu_0$. The facts that $p_{\text{ext}}^{(v)}(\delta) \to p_{\text{ext}}(\delta)$ and $\mathrm{P}(G^{(v)}) \to 1 - p_{\text{ext}}$ imply the existence of $\nu_1 \in \mathbb{N}$ such that, for all $v \geq \nu_1$,

$$\begin{aligned}
\mathrm{P}\left(Z_E^{(v)} > \delta m^{(v)} | G^{(v)}\right) &= \frac{\mathrm{P}\left(Z_E^{(v)} \geq \delta m^{(v)}, G^{(v)}\right)}{\mathrm{P}\left(G^{(v)}\right)} \\
&= \frac{\mathrm{P}\left(Z_E^{(v)} \geq \delta m^{(v)}\right)}{\mathrm{P}\left(G^{(v)}\right)} \\
&\geq \frac{1 - p_{\text{ext}}^{(v)}(\delta)}{\mathrm{P}\left(G^{(v)}\right)} \\
&\geq \frac{1 - p_{\text{ext}}(\delta)}{1 - p_{\text{ext}}} - \frac{\varepsilon}{4}.
\end{aligned} \tag{4.35}$$

Setting $\nu_2 = \max\{\nu_0, \nu_1\}$ and combining (4.34) and (4.35) then establishes (4.33).

We next show that, provided a major outbreak occurs, the scaled terminal

infectious pressure exceeds a positive fraction, i.e. we demonstrate that for any $\varepsilon > 0$ there exists $\varepsilon_1 > 0$ such that

$$\mathrm{P}\left(\bar{T}_\infty^{(v)} \geq \varepsilon_1 | G^{(v)}\right) \geq 1 - \varepsilon, \tag{4.36}$$

for sufficiently large v.

For $t \geq 0$, let $\tilde{Z}_{\boldsymbol{n},i}(t)$ denote the number of individuals infected in the embedding construction in the i^{th} category-$\boldsymbol{n}$ household ($i \in \mathscr{J}$, $\boldsymbol{n} \in \mathscr{N}$) and let

$$Y_\bullet^{(v)}(t) = \frac{1}{m^{(v)}} \sum_{\boldsymbol{n} \in \mathscr{N}} \sum_{i=1}^{m_{\boldsymbol{n}}^{(v)}} \mathbb{1}_{\{\tilde{Z}_{\boldsymbol{n},i}(t) > 0\}}$$

denote the proportion of households infected in the embedding construction with $m^{(v)}$ households. Then, for $t \geq 0$, (cf. Theorem 4.11)

$$Y_\bullet^{(v)}(t) \xrightarrow{\text{a.s.}} y(t) = 1 - \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \exp\left(-t \sum_{i=1}^{J} n_i \kappa_i\right) \text{ as } v \to \infty.$$

Let $\varepsilon_1 = -\dfrac{1}{J n_{\max} \kappa_J} \log\left(1 - \dfrac{\tilde{\delta}}{2}\right)$, where $\tilde{\delta}$ satisfies (4.33). Then

$$\begin{aligned}
\mathrm{P}\left(\bar{T}_\infty^{(v)} < \varepsilon_1 | G^{(v)}\right) &= \mathrm{P}\left(\bar{T}_\infty^{(v)} < \varepsilon_1, Z_E^{(v)} \geq \tilde{\delta}\tilde{m}^{(v)} | G^{(v)}\right) + \mathrm{P}\left(\bar{T}_\infty^{(v)} < \varepsilon_1, Z_E^{(v)} < \tilde{\delta}\tilde{m}^{(v)} | G^{(v)}\right) \\
&= A^{(v)} + B^{(v)}, \text{ say.}
\end{aligned}$$

Considering $A^{(v)}$, we have

$$\begin{aligned}
A^{(v)} = \mathrm{P}\left(\bar{T}_\infty^{(v)} < \varepsilon_1, Z_E^{(v)} \geq \tilde{\delta}\tilde{m}^{(v)} | G^{(v)}\right) &= \frac{\mathrm{P}\left(\bar{T}_\infty^{(v)} < \varepsilon_1, Z_E^{(v)} \geq \tilde{\delta}\tilde{m}^{(v)}, G^{(v)}\right)}{\mathrm{P}(G^{(v)})} \\
&\leq \frac{\mathrm{P}\left(Y_\bullet^{(v)}(\varepsilon_1) \geq \tilde{\delta}\right)}{\mathrm{P}(G^{(v)})},
\end{aligned}$$

since $Y_\bullet^{(v)}(t)$ is non-decreasing in t. We also have, as $v \to \infty$,

$$\begin{aligned} Y_\bullet^{(v)}(\varepsilon_1) &\xrightarrow{\text{a.s.}} 1 - \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \exp\left(-\varepsilon_1 \sum_{i=1}^{J} n_i \kappa_i\right) \\ &\leq 1 - \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \exp\left(-\varepsilon_1 J n_{\max} \kappa_J\right) \\ &= \frac{\tilde{\delta}}{2} < \tilde{\delta}. \end{aligned}$$

This, in conjunction with $\mathrm{P}(G^{(v)}) \to 1 - p_{\text{ext}}$ as $v \to \infty$, implies that $A^{(v)}$ can be made arbitrarily small for sufficiently large v; in particular, there exists $\nu_3 \in \mathbb{N}$ such that $A^{(v)} \leq \dfrac{\varepsilon}{2}$ for all $v \geq \nu_3$. From (4.33), there exists ν_4 such that $B^{(v)} \leq \dfrac{\varepsilon}{2}$ for all $v \geq \nu_4$. Setting $\nu_5 = \max\{\nu_3, \nu_4\}$, we have that, for all $v \geq \nu_5$,

$$\mathrm{P}\left(\bar{T}_\infty^{(v)} \geq \varepsilon_1 | G^{(v)}\right) \geq 1 - \varepsilon,$$

which establishes (4.36). Thus, conditional upon a major outbreak, we have $\bar{T}_\infty^{(v)} = \bar{T}_{\infty,\varepsilon_1}^{(v)}$ with large probability for sufficiently large v.

To prove (i), first note that $\tilde{\Upsilon}^{(v)} = {m_H^{(v)}}^{-1} \frac{1}{m^{(v)}} \Upsilon^{(v)}$ and that ${m_H^{(v)}}^{-1} \to m_H^{-1}$ as $v \to \infty$. Consider the embedding process beginning when $\lceil \log(m^{(v)}) \rceil$ households are infected. Then, for sufficiently large v, with probability at least $1 - \varepsilon$,

$$\frac{1}{m^{(v)}} \Upsilon^{(v)} = \frac{1}{m^{(v)}} \hat{A}_\bullet^{(v)}\left(\bar{T}_{\infty,\varepsilon_1}^{(v)}\right).$$

Now $\dfrac{1}{m^{(v)}} \hat{A}_\bullet^{(v)}\left(\bar{T}_{\infty,\varepsilon_1}^{(v)}\right) \xrightarrow{\text{p}} a(\tau)$, which follows immediately from Corollary 4.10. Then

$$\tilde{\Upsilon}^{(v)} | G^{(v)} \xrightarrow{\text{p}} m_H^{-1} a(\tau) = \tau \text{ as } v \to \infty$$

which establishes (i). A similar argument establishes (ii).

□

The final result of this section concerns a joint central limit theorem for the severity and the final state random variables $(R_1^{(v)}, R_2^{(v)}, \ldots, R_p^{(v)}, \Upsilon^{(v)})$, conditional upon a major outbreak. In the notation of Theorem 4.13, let $\boldsymbol{\Sigma} = \boldsymbol{H}\boldsymbol{C}(\tau,\tau)\boldsymbol{H}^\top$.

Theorem 4.23. *Suppose that $R_* > 1$. We have*

$$(m^{(v)})^{-\frac{1}{2}} \begin{pmatrix} R_1^{(v)} - m^{(v)} r_1(\tau) \\ R_2^{(v)} - m^{(v)} r_2(\tau) \\ \vdots \\ R_p^{(v)} - m^{(v)} r_p(\tau) \\ m_H \left({m_H^{(v)}}^{-1} \Upsilon^{(v)} - m^{(v)} \tau \right) \end{pmatrix} | G^{(v)} \xrightarrow{\mathrm{D}} N(\mathbf{0}_{p+1}, \Sigma) \text{ as } v \to \infty.$$

Proof. Firstly, recall that $\sqrt{m^{(v)}} \bar{T}_0 \xrightarrow{\text{a.s.}} 0$, and that Theorem 4.13 can be applied to the embedding process with $\bar{T}_0^{(v)}$ units of initial infectious pressure to each individual, replacing $A_\bullet^{(v)}(t)$ by $\hat{A}_\bullet^{(v)}(t)$ and $R_{\bullet j}^{(v)}(t)$ by $\hat{R}_{\bullet j}^{(v)}(t)$. Next, we have, for $v \geq v_0$ and with probability at least $1 - \varepsilon$,

$$\sqrt{m^{(v)}} \left(\tilde{\Upsilon}^{(v)} - \tau \right) = \sqrt{m^{(v)}} m_H^{-1} \left(\frac{1}{m^{(v)}} \hat{A}_\bullet^{(v)}(\bar{T}_{\infty,\varepsilon_1}^{(v)}) - a(\tau) \right) + o_p(1).$$

Finally, arguing as in the proof of Theorem 4.22, there exists $v_1 \in \mathbb{N}$ such that, for $v \geq v_1$, with probability at least $1 - \varepsilon$,

$$(m^{(v)})^{\frac{1}{2}} \begin{pmatrix} \tilde{R}_1^{(v)} - r_1(\tau) \\ \tilde{R}_2^{(v)} - r_2(\tau) \\ \vdots \\ \tilde{R}_p^{(v)} - r_p(\tau) \\ m_H \left(\tilde{\Upsilon}^{(v)} - \tau \right) \end{pmatrix} = (m^{(v)})^{-\frac{1}{2}} \begin{pmatrix} \hat{R}_{\bullet 1}^{(v)}(\bar{T}_{\infty,\varepsilon_1}^{(v)}) - m^{(v)} r_1(\tau) \\ \hat{R}_{\bullet 2}^{(v)}(\bar{T}_{\infty,\varepsilon_1}^{(v)}) - m^{(v)} r_2(\tau) \\ \vdots \\ \hat{R}_{\bullet p}^{(v)}(\bar{T}_{\infty,\varepsilon_1}^{(v)}) - m^{(v)} r_p(\tau) \\ \hat{A}_\bullet^{(v)}(\bar{T}_{\infty,\varepsilon_1}^{(v)}) - m^{(v)} a(\tau) + o_p(1) \end{pmatrix}.$$

The result follows by applying Theorem 4.13 to the aforementioned embedding process and using Slutsky's theorem. □

4.9 Highly locally infectious epidemics

We illustrate Theorem 4.23 by considering the special case of a highly locally infectious disease, in which infection of a member of a household necessarily results in the whole household becoming infected (see, for example, Becker and Dietz [1995] and in a multitype setting, Becker and Hall [1996]). For $v = 1, 2, \ldots$, let $\boldsymbol{Z}^{(v)} = (Z_1^{(v)}, Z_2^{(v)}, \ldots, Z_J^{(v)})^\top$, where $Z_i^{(v)}$ is the total number

of type-i individuals infected in an epidemic with $m^{(v)}$ households. We derive a central limit theorem for $\boldsymbol{Z}^{(v)}$, conditional upon a major outbreak ($G^{(v)}$), with an explicit expression for the (asymptotic) covariance matrix.

For $\boldsymbol{n} \in \mathscr{N}$, $t \geq 0$ and $i \in \mathscr{J}$, let $R_i^{(\boldsymbol{n})}(t)$ be the number of type-i individuals infected in the epidemic $\mathscr{E}_{\boldsymbol{n}}(\Lambda^L, e^{-t\boldsymbol{\kappa}})$. With this choice of final outcome quantities, Theorem 4.23 yields a central limit theorem for $\boldsymbol{Z}^{(v)}$ conditional upon $G^{(v)}$. The highly locally infectious assumption implies that, for $\boldsymbol{n} \in \mathscr{N}$,

$$(R_1^{(\boldsymbol{n})}(t), R_2^{(\boldsymbol{n})}(t), \ldots, A^{(\boldsymbol{n})}(t)) \stackrel{\mathrm{D}}{=} D^{(\boldsymbol{n})}(t) \left(n_1, n_2, \ldots, n_J, \sum_{j=1}^{J} \beta_j \sum_{k=1}^{n_j} I_k^{(j)} \right),$$

where $D^{(\boldsymbol{n})}(t) \sim \mathrm{Bin}(1, 1 - e^{t\boldsymbol{n}\boldsymbol{\kappa}^\top})$ and $I_k^{(j)} \stackrel{\mathrm{D}}{=} T_I^{(j)}$ $(j \in \mathscr{J}, k = 1, 2, \ldots, n_j)$ are independent random variables. For $\boldsymbol{n} \in \mathscr{N}$, let $b_1(\boldsymbol{n}) = \sum_{k=1}^{J} \beta_k n_k \mu_I^{(k)}$ and $b_2(\boldsymbol{n}) = \sum_{k=1}^{J} \beta_k^2 n_k \sigma_{I,k}^2$, where $\sigma_{I,k}^2 = \mathrm{var}(I^{(k)})$. Elementary calculations yield that, for $\boldsymbol{n} \in \mathscr{N}$ and $t \geq 0$,

$$\begin{aligned}
r_i^{(\boldsymbol{n})}(t) &= \mathrm{E}[R_i^{(\boldsymbol{n})}(t)] = n_i(1 - e^{-t\boldsymbol{n}\boldsymbol{\kappa}^\top}), \quad i \in \mathscr{J}, \\
a^{(\boldsymbol{n})}(t) &= \mathrm{E}[A^{(\boldsymbol{n})}(t)] = b_1(\boldsymbol{n})(1 - e^{-t\boldsymbol{n}\boldsymbol{\kappa}^\top}), \\
\mathrm{cov}(R_i^{(\boldsymbol{n})}(t), R_j^{(\boldsymbol{n})}(t)) &= n_i n_j (1 - e^{-t\boldsymbol{n}\boldsymbol{\kappa}^\top}) e^{-t\boldsymbol{n}\boldsymbol{\kappa}^\top}, \qquad i, j \in \mathscr{J}, \qquad (4.37) \\
\mathrm{cov}(R_i^{(\boldsymbol{n})}(t), A^{(\boldsymbol{n})}(t)) &= n_i b_1(\boldsymbol{n})(1 - e^{-t\boldsymbol{n}\boldsymbol{\kappa}^\top}) e^{-t\boldsymbol{n}\boldsymbol{\kappa}^\top}, \quad i \in \mathscr{J}, \qquad (4.38) \\
\mathrm{var}(A^{(\boldsymbol{n})}(t)) &= b_2(\boldsymbol{n})(1 - e^{-t\boldsymbol{n}\boldsymbol{\kappa}^\top}) + b_1^2(\boldsymbol{n})(1 - e^{-t\boldsymbol{n}\boldsymbol{\kappa}^\top}) e^{-t\boldsymbol{n}\boldsymbol{\kappa}^\top}. \qquad (4.39)
\end{aligned}$$

Then

$$a(t) = \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} a^{(\boldsymbol{n})}(t) = \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} b_1(\boldsymbol{n})(1 - e^{-t\boldsymbol{n}\boldsymbol{\kappa}^\top}), \quad t \geq 0$$

and, by Theorem 4.8, $R_* = m_H^{-1} a'(0) = \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} b_1(\boldsymbol{n}) \boldsymbol{n}\boldsymbol{\kappa}^\top$.

Suppose that $R_* > 1$ and, using Theorem 4.8, let τ be the unique solution

in $(0,\infty)$ of $t = m_H^{-1}a(t)$. For $t \geq 0$ and $i \in \mathscr{J}$, let

$$r_i(t) = \sum_{\boldsymbol{n}\in\mathscr{N}} \theta_{\boldsymbol{n}} r_i^{(\boldsymbol{n})}(t) = \sum_{\boldsymbol{n}\in\mathscr{N}} \theta_{\boldsymbol{n}} n_i (1 - e^{-t\boldsymbol{n}\boldsymbol{\kappa}^\top})$$

and, setting $p = J$, let

$$\boldsymbol{r}(t) = \sum_{\boldsymbol{n}\in\mathscr{N}} \theta_{\boldsymbol{n}} (1 - e^{-t\boldsymbol{n}\boldsymbol{\kappa}^\top})\tilde{\boldsymbol{n}},$$

where $\tilde{\boldsymbol{n}} = (n_1, n_2, \ldots, n_J, b_1(\boldsymbol{n}))^\top$. Let $\tilde{\boldsymbol{e}}$ denote the $(J+1)\times 1$ vector $(0,0,\ldots,1)^\top$. In the notation of Theorem 4.23, we have $\boldsymbol{C}(\tau,\tau) = \sum_{\boldsymbol{n}\in\mathscr{N}} \theta_{\boldsymbol{n}} \boldsymbol{C}^{(\boldsymbol{n})}(\tau,\tau)$ where, by (4.37)-(4.39),

$$\boldsymbol{C}^{(\boldsymbol{n})}(\tau,\tau) = (1 - e^{-t\boldsymbol{n}\boldsymbol{\kappa}^\top}) e^{-t\boldsymbol{n}\boldsymbol{\kappa}^\top} \tilde{\boldsymbol{n}}\tilde{\boldsymbol{n}}^\top.$$

Let $\boldsymbol{z} = (z_1, z_2, \ldots, z_J)^\top$, where $z_i = r_i(\tau)$ $(i \in \mathscr{J})$. Note that $z_i = \gamma_i \tilde{z}_i$, where $\tilde{z}_i$ is defined at (4.12). Let $\boldsymbol{H}$ be given by (4.23). Note that, for $\boldsymbol{n} \in \mathscr{N}$,

$$\boldsymbol{H}\tilde{\boldsymbol{n}} = \begin{pmatrix} \boldsymbol{n}^\top + b_1(\boldsymbol{n})(m_H - a'(\tau))^{-1}\tilde{\boldsymbol{r}}'(\tau) \\ b_1(\boldsymbol{n}) m_H (m_H - a'(\tau))^{-1} \end{pmatrix} \text{ and } \boldsymbol{H}\tilde{\boldsymbol{e}} = \begin{pmatrix} \tilde{\boldsymbol{r}}'(\tau) \\ m_H \end{pmatrix},$$

where $\tilde{\boldsymbol{r}}'(\tau) = (r_1'(\tau), r_2'(\tau), \ldots, r_J'(\tau))^\top$. It follows from Theorem 4.23 that

$$(m^{(v)})^{-\frac{1}{2}} \left(\boldsymbol{Z}^{(v)} - m^{(v)} \boldsymbol{z}^{(v)} \right) | G^{(v)} \xrightarrow{\mathrm{D}} N(\boldsymbol{0}_J, \boldsymbol{\Sigma}) \text{ as } v \to \infty, \tag{4.40}$$

where

$$\boldsymbol{\Sigma} = \sum_{\boldsymbol{n}\in\mathscr{N}} \theta_{\boldsymbol{n}} (1 - e^{-t\boldsymbol{n}\boldsymbol{\kappa}^\top})(\boldsymbol{S}_1^{(\boldsymbol{n})} + e^{-t\boldsymbol{n}\boldsymbol{\kappa}^\top} \boldsymbol{S}_2^{(\boldsymbol{n})}), \tag{4.41}$$

with

$$\boldsymbol{S}_1^{(\boldsymbol{n})} = \frac{b_2(\boldsymbol{n})}{(m_H - a'(\tau))^2} \tilde{\boldsymbol{r}}'(\tau)\tilde{\boldsymbol{r}}'(\tau)^\top,$$

$$\boldsymbol{S}_2^{(\boldsymbol{n})} = \boldsymbol{n}^\top\boldsymbol{n} + \frac{b_1(\boldsymbol{n})}{m_H - a'(\tau)} (\boldsymbol{n}^\top \tilde{\boldsymbol{r}}'(\tau)^\top + \tilde{\boldsymbol{r}}'(\tau)\boldsymbol{n}) + \left(\frac{b_1(\boldsymbol{n})}{m_H - a'(\tau)} \right)^2 \tilde{\boldsymbol{r}}'(\tau)\tilde{\boldsymbol{r}}'(\tau)^\top.$$

4.10 Standard SIR multitype epidemics

If all households have size 1, the model reduces to a special case of the standard SIR multitype epidemic epidemic model (see Andersson and Britton [2000], Chapter 6) in which mixing is proportionate. We apply the notation and results of Section 4.9 to this setting. There are J distinct household categories and $\mathscr{N} = \{\boldsymbol{e}_1, \boldsymbol{e}_2, \ldots, \boldsymbol{e}_J\}$. For $k \in \mathscr{J}$, $\theta_{\boldsymbol{e}_k} = \gamma_k$, $b_1(\boldsymbol{e}_k) = \beta_k \mu_I^{(k)}$, $b_2(\boldsymbol{e}_k) = \beta_k^2 \sigma_{I,k}^2$ and $r_i^{(\boldsymbol{e}_k)}(t) = \delta_{ki}(1 - e^{-\kappa_i t})$ ($i \in \mathscr{J}$). Hence, $r_i(t) = \gamma_i(1 - e^{-\kappa_i t})$ ($i \in \mathscr{J}$) and $a(t) = \sum_{k=1}^{J} \gamma_k \beta_k \mu_I^{(k)}(1 - e^{-\kappa_k t})$. Since $m_H = 1$, we have

$$R_0 = R_* = a'(0) = \sum_{k=1}^{J} \gamma_k \beta_k \kappa_k \mu_I^{(k)}.$$

Suppose that $R_0 > 1$ and let τ be the unique solution in $(0, \infty)$ of $t = a(t)$. For $i \in \mathscr{J}$, let $z_i = r_i(\tau) = \gamma_i(1 - e^{-\kappa_i \tau})$ and $\rho_i = \gamma_i - z_i$. Then $r_i'(\tau) = \kappa_i \rho_i$ ($i \in \mathscr{J}$) and $a'(\tau) = \sum_{k=1}^{J} \beta_k \kappa_k \rho_k \mu_I^{(k)}$. For $k \in \mathscr{J}$, note that $\theta_{\boldsymbol{e}_k}(1 - e^{-\boldsymbol{e}_k \boldsymbol{\kappa}^\top \tau}) = z_k$, $\theta_{\boldsymbol{e}_k}(1 - e^{-\boldsymbol{e}_k \boldsymbol{\kappa}^\top \tau}) e^{-\boldsymbol{e}_k \boldsymbol{\kappa}^\top \tau} = \gamma_k^{-1} z_k \rho_k$ and

$$\left[\boldsymbol{e}_k{}^\top \tilde{\boldsymbol{r}}'(\tau)^\top + \tilde{\boldsymbol{r}}'(\tau) \boldsymbol{e}_k \right]_{ij} = \delta_{ik} \kappa_j \rho_j + \delta_{jk} \kappa_i \rho_i, \quad i, j \in \mathscr{J}.$$

It then follows from (4.40) and (4.41), and a little algebra, that

$$(m^{(\nu)})^{-\frac{1}{2}} \left(\boldsymbol{Z}^{(\nu)} - m^{(\nu)} \boldsymbol{z} \right) | G^{(\nu)} \xrightarrow{\mathrm{D}} N(\boldsymbol{0}_J, \boldsymbol{\Sigma}) \text{ as } \nu \to \infty,$$

where $\boldsymbol{z} = (z_1, z_2, \ldots, z_J)^\top$ and $\boldsymbol{\Sigma} = [\sigma_{ij}]_{i,j \in \mathscr{J}}$ with

$$\sigma_{ij} = \delta_{ij} \frac{z_i \rho_i}{\gamma_i} + \frac{\rho_i \rho_j}{1 - \sum_{k=1}^{J} \beta_k \kappa_k \rho_k \mu_I^{(k)}} \left(\frac{\beta_i \mu_I^{(i)} z_i \kappa_j}{\gamma_i} + \frac{\beta_j \mu_I^{(j)} z_j \kappa_i}{\gamma_j} \right) + \frac{\kappa_i \rho_i \kappa_j \rho_j}{\left(1 - \sum_{k=1}^{J} \beta_k \kappa_k \rho_k \mu_I^{(k)} \right)^2} \sum_{k=1}^{J} \beta_k^2 z_k \left[\sigma_{I,k}^2 + \frac{\rho_k (\mu_I^{(k)})^2}{\gamma_k} \right].$$

4.11 Discussion

We have shown that the assumption of proportionate global mixing leads to considerable simplification in the proof and calculation of asymptotic properties of

stochastic multitype SIR epidemics among a population of households. For ease of exposition, we have not presented the results in full generality. For example, we can allow the overall rate that an individual makes contacts to depend also on household category, i.e. replace the proportionate mixing assumption $\lambda_{ij}^G = \beta_i \kappa_j$ by $\lambda_{ij}^G(\boldsymbol{n}) = \beta_i(\boldsymbol{n})\kappa_j$ and let $\lambda_{ij}^L = \lambda_{ij}^L(\boldsymbol{n})$. Under these generalisations, the approximating branching process $\mathscr{B}$ is still effectively single-type and the process $\{\boldsymbol{R}^{(n,k)}(t)\}$, defined at (4.20), still has a one-dimensional index set, so the given proofs and results continue to hold with minor modification. The assumption of a maximum household size $n_{\max} < \infty$ can also be relaxed, in which case $\mathscr{N}$ is countably finite. In order to do so, stronger assumptions are required on the convergence of $\theta_{\boldsymbol{n}}^{(v)}$ to $\theta_{\boldsymbol{n}}$ ($\boldsymbol{n} \in \mathscr{N}$) and on the moments of $R_j^{(\boldsymbol{n})}(t)$ ($\boldsymbol{n} \in \mathscr{N}, j = 1, 2, \ldots, p$), cf. (4.21).

The multitype households model is very general and by suitable choice of the type space includes, for example, models with more than two levels of mixing, such as a population of villages, each partitioned into households (cf. Britton et al. [2011] and Ouboter et al. [2016]). More complex structures can also be incorporated, provided the sizes of all structures except households converge to infinity as $v \to \infty$.

The central limit theorem for final state random variables is also very general and is applicable to more complex SIR models, following suitable modification of the type space of the individuals in the model. For example, in the carrier-borne model of Downton [1968], infected susceptibles are detected immediately independently with a specified probability, so in a single-type setting the effective infectious period distribution is a mixture of a point mass at zero and a strictly positive random variable I. This can be extended to the present multitype households model setting, with the detection probability possibly depending on both an individual's type and the category of their household. Suitably defined final state random variables can be used to obtain a multivariate central limit theorem for the numbers infected and the numbers detected immediately of the J types of individuals. The model can be extended further so that infected susceptibles are split into more than just two types of infectives, cf. Picard and Lefèvre [1990]. Another extension to the standard SIR model, con-

sidered in Picard and Lefèvre [1990], concerns models in which infectives pass through stages of infection, having possibly different levels of infection. Again this can be extended to the multitype households model setting and, under suitable conditions, Theorem 4.23 can be used to obtain for example a multivariate central limit theorem for the total severity in the different stages of infection for the J types of individuals. See Ball [2019] for further detail about such extensions in the context of exact results for the final outcome of SIR epidemics in populations without household structure.

5 Herd immunity in the activity level and households model

5.1 Introduction

In this chapter we consider herd immunity for a model with both activity levels and household structure. As discussed in Section 2.1, introducing individual heterogeneity typically causes the disease-induced herd immunity level to decrease. Examples can be seen in Britton et al. [2020] in the case of age and activity levels, and in Gomes et al. [2022] in the case of variable individual susceptibility. In contrast, the results of Chapter 2 show that household structure typically causes the disease-induced herd immunity level to increase. The aim of this chapter is therefore to consider, in the context of herd immunity, a model which combines activity level and household structure.

The model with activity levels and household structure is a slight simplification of the multitype households model described in Section 4.2; we borrow all of the relevant notation from that section, providing further details in Section 5.2. It is possible, in principle, to compute h_D for this model, although the number of ODEs required soon becomes infeasible owing to the large number of household categories, and thus the large number of potential states (in terms of the numbers of susceptible, infected and removed individuals) that each household can be in. We instead consider the approximation $\tilde{h}_D$ of h_D defined in Section 2.3.2. The calculation of $\tilde{h}_D$ in this chapter has strong parallels with the corresponding calculation for the households model.

As discussed in Section 2.4, the complexities of the households model make analytical progress comparing $\tilde{h}_D$ and h_C difficult to achieve. The introduction of individual activity levels into the model further complicates the analysis. As a result, we restrict our analytical results to the highly locally infectious case, where $\lambda_L \to \infty$. We show that the amount of local mixing which can occur between individuals of different types is a key factor controlling whether $\tilde{h}_D > h_C$, providing a heuristic justification for this behaviour. We also provide and discuss numerical comparisons of $\tilde{h}_D$ and h_C.

It is clear that, even under the assumption of proportionate global mixing, the model of Chapter 4 is quite general. As a result, it is not feasible to consider all parameter choices and household category distributions in our analysis. As such, we focus in the highly locally infectious case on distributions where orderings of $\tilde{h}_D$ and h_C are available. For our numerical analysis we consider extending the parameters used in Britton et al. [2020] to the households setting. We also consider briefly how herd immunity plays out in this model using real-world household size distributions, including the UK household size distribution. The parameters chosen are for the purposes of exposition and are not intended to replicate real-world mixing rates, but instead to demonstrate the sensitivity of $\tilde{h}_D$ and h_C to activity levels and household structure.

This chapter is structured as follows. In Section 5.2 we the model with activity levels and household structure. In Section 5.3 we provide a framework for comparison of $\tilde{h}_D$ and h_C, noting how the relevant quantities simplify in the highly locally infectious case. In Section 5.4 we show that $\tilde{h}_D < h_C$ in the activity level model in the absence of household structure, motivating further comparison of $\tilde{h}_D$ and h_C. We then, in Section 5.5, prove orderings of $\tilde{h}_D$ and h_C in some special cases, such as when all individuals in a given household have the same type or when all households are the same category. In Section 5.6 we remove the highly locally infectious assumption and provide further comparisons of $\tilde{h}_D$ and h_C, where we also consider some real-world household size distributions. Finally, in Section 5.7, we discuss how this model could be used to estimate real-world herd immunity levels, some possible extensions of our work, and how realistic activity rate parameters might be chosen.

5.2 Model definition

In order to obtain the (deterministic) activity level and households model we, using the model and notation of Section 4.2, take $\kappa_i = \lambda\beta_i$ for all $i \in \mathscr{J}$, where λ is a positive constant which can be chosen to set a threshold parameter (viz. R_*, R_0 or R_I) for the epidemic to a desired value. We consider the deterministic model obtained by allowing the number of households m to tend to infinity. We assume, without loss of generality, that $1 = \beta_1 \leq \beta_2 \cdots \leq \beta_J$ and $\beta_1 < \beta_J$, so

that type-1 individuals are the least active and type-J individuals are the most active. Note that, in terms of global mixing, individuals with higher activity rate associated to them both infect and are infected at a higher rate than those with lower activity. Finally, we assume that $\mu_I^{(i)} = 1$ and $\Lambda_{ij}^L = \lambda_L$ for all $i, j \in \mathscr{J}$. This corresponds to the case where differently typed individuals mix globally at different rates, but local epidemics are unaffected by the types of individuals among which they occur.

5.3 Herd immunity

5.3.1 General framework

Recall the vaccine-induced herd immunity level h_C, obtained by vaccinating uniformly at random with a perfect vaccine, such that the remaining susceptible population has $R_* = 1$. Recall also the disease-induced herd immunity level $\tilde{h}_D$, obtained by calculating the final size of an epidemic, with adjusted global infection rates, such that the epidemic terminates at $R_* = 1$. We outline the calculations of the herd immunity levels $\tilde{h}_D$ and h_C for the activity level and households model, which are extensions of Section 2.3.

In order to proceed, we first consider the effect of a vaccination profile on the remaining susceptible population. Suppose some individuals in the population are made immune from infection and let $v_{\boldsymbol{n},\boldsymbol{r}}$ denote the proportion of category-$\boldsymbol{n}$ households with $\boldsymbol{r}$ members immune from infection. The vaccination profile

$$v = \{v_{\boldsymbol{n},\boldsymbol{r}} : \boldsymbol{n} \in \mathscr{N}, \mathbf{0} \leq \boldsymbol{r} \leq \boldsymbol{n}\}$$

provides the effective household category distribution as a result of some individuals achieving immunity. We say that a household is in state $(\boldsymbol{n}, \boldsymbol{r})$ if it is a category-$\boldsymbol{n}$ household with $\boldsymbol{r}$ members immune. (Throughout this Chapter we write $\boldsymbol{r}$ members immune as short-hand for r_1 type-1, r_2 type-2, ..., r_J type-J members being immune.)

We calculate $R_*(v)$, the threshold parameter for an epidemic assuming the vaccination profile v. A randomly chosen type-j individual in a state-$(\boldsymbol{n}, \boldsymbol{r})$ household is susceptible with probability $\dfrac{n_j - r_j}{n_j}$ and, upon being infected, trig-

gers a local epidemic which infects $\mu_{\boldsymbol{n}-\boldsymbol{r},i,j}(\Lambda^L)$ type-i individuals on average. The mean number of global contacts ensuing from a type-i individual who was infected locally is $\lambda\beta_i$. Combining these, as well as conditioning on an individual's type and household category, as in Section 4.3.1, yields

$$R_*(v) = \lambda \sum_{j=1}^{J} \beta_j \gamma_j \sum_{\boldsymbol{n}\in\mathscr{N}} \alpha_j(\boldsymbol{n}) \sum_{\boldsymbol{r}=\boldsymbol{0}}^{\boldsymbol{n}} v_{\boldsymbol{n},\boldsymbol{r}} \left(\frac{n_j - r_j}{n_j}\right) \sum_{i=1}^{J} \mu_{\boldsymbol{n}-\boldsymbol{r},i,j}(\Lambda^L)\beta_i. \quad (5.1)$$

During vaccine-induced immunity we assume that vaccination occurs uniformly at random, so that $v_{\boldsymbol{n},\boldsymbol{r}} = \binom{\boldsymbol{n}}{\boldsymbol{r}} \boldsymbol{c}^{\boldsymbol{r}}(1-\boldsymbol{c})^{\boldsymbol{n}-\boldsymbol{r}}$, with $\boldsymbol{c} = (c, c, \ldots, c)$ for some vaccination coverage $c \in (0,1)$. Then h_C is the unique $c \in (0,1)$ which yields $R_*(v) = 1$. Calculating $\tilde{h}_D$ requires computing $v_{\boldsymbol{n},\boldsymbol{r}}$ (typically numerically) using the final size results in Section 4.4, by calculating the final size of the epidemic which terminates when $R_*(v) = 1$. In the sequel we denote by R_D (R_U) the threshold parameter corresponding to disease-induced herd immunity (uniform vaccination).

5.3.2 Simplification in highly locally infectious case

In the highly locally infectious case ($\lambda_L \to \infty$) any globally infected individual infects their whole household. As a result, both the vaccination profile and (5.1) can be simplified, as we now outline.

During disease-induced herd immunity, households are either rendered fully immune to infection or fully susceptible to infection from a subsequent epidemic. Throughout the remainder of this chapter, we let π denote the probability a type-1 individual avoids global infection in the first epidemic and write $\boldsymbol{\pi} = (\pi, \pi^{\beta_2}, \ldots, \pi^{\beta_J})$, noting the power law relationship between global avoidance probabilities of different types of individuals (see, for example, Gart [1968], Section 3). Then, using $\mu_{\boldsymbol{n}-\boldsymbol{r},i,j} = n_i$ in (5.1), we have

$$R_D(\pi) = \lambda \sum_{j=1}^{J} \beta_j \gamma_j \sum_{\boldsymbol{n}\in\mathscr{N}} \alpha_j(\boldsymbol{n}) \boldsymbol{\pi}^{\boldsymbol{n}} \sum_{l=1}^{J} n_l \beta_l. \quad (5.2)$$

Letting π_* be such that $R_D(\pi_*) = 1$, we can then, in an obvious notation, com-

pute

$$\tilde{h}_D = 1 - \sum_{j=1}^{J} \gamma_j \sum_{\boldsymbol{n}\in\mathscr{N}} \alpha_j(\boldsymbol{n})(1-\boldsymbol{\pi}_*^{\boldsymbol{n}}). \tag{5.3}$$

In most cases (5.2) cannot be solved analytically, in which case $\tilde{h}_D$ cannot be computed directly. Despite this, we can make progress for certain choices of parameters by comparing reproduction numbers. To this end, we now compute $R_U(c)$ in the highly locally infectious case. Using (5.1) we find

$$\begin{aligned} R_U(c) &= \lambda \sum_{j=1}^{J} \beta_j \gamma_j \sum_{\boldsymbol{n}\in\mathscr{N}} \alpha_j(\boldsymbol{n}) \sum_{\boldsymbol{r}=0}^{\boldsymbol{n}} \binom{\boldsymbol{n}}{\boldsymbol{r}} \boldsymbol{c}^{\boldsymbol{r}} (1-\boldsymbol{c})^{\boldsymbol{n}-\boldsymbol{r}} \left(\frac{n_j - r_j}{n_j}\right) \sum_{i=1}^{J} \beta_i (n_i - r_i) \\ &= \lambda \sum_{j=1}^{J} \beta_j \gamma_j \sum_{\boldsymbol{n}\in\mathscr{N}} \alpha_j(\boldsymbol{n}) f_{\boldsymbol{n},j}(c), \text{ say.} \end{aligned}$$

Observe that

$$f_{\boldsymbol{n},j}(c) = \mathrm{E}[g_i(X_1, X_2, \ldots, X_J)],$$

where, for given $\boldsymbol{n} \in \mathscr{N}$, $X_1, X_2, \ldots, X_J$ are independent random variables with $X_i \sim \mathrm{Bin}(n_i, c), i \in \mathscr{J}$ and

$$g_j(x_1, x_2, \ldots, x_J) = \left(1 - \frac{x_j}{n_j}\right) \sum_{l=1}^{J} \beta_l (n_l - x_l), \quad j \in \mathscr{J}.$$

Then

$$\begin{aligned} f_{\boldsymbol{n},j}(c) &= \mathrm{E}\left[\left(1 - \frac{X_j}{n_j}\right)\left(\sum_{l=1}^{J}(n_l - X_l)\beta_l\right)\right] \\ &= \mathrm{E}\left[\left(1 - \frac{X_j}{n_j}\right)\left(\sum_{l\neq j}^{J}(n_l - X_l)\beta_l + (n_j - X_j)\beta_j\right)\right] \\ &= (1-c)\left(\sum_{l\neq j}^{J}(1-c)n_l\beta_l\right) + \mathrm{E}\left[\left(1 - \frac{X_j}{n_j}\right)(n_j - X_j)\,\beta_j\right] \\ &= (1-c)\left(\sum_{l\neq j}^{J}(1-c)n_l\beta_l\right) + (1-c)^2 n_j\beta_j + c(1-c)\beta_j \\ &= (1-c)^2 \sum_{l=1}^{J} n_l\beta_l + c(1-c)\beta_j, \quad \boldsymbol{n} \in \mathscr{N}, j \in \mathscr{J}, \end{aligned}$$

leading to

$$R_U(c) = \lambda \sum_{j=1}^{J} \beta_j \gamma_j \sum_{\boldsymbol{n} \in \mathscr{N}} \alpha_j(\boldsymbol{n}) \left\{ (1-c)^2 \sum_{l=1}^{J} n_l \beta_l + c(1-c)\beta_j \right\}. \tag{5.4}$$

We conclude this section with an expression for h_C in the highly locally infectious case.

Theorem 5.1. *In the highly locally infectious case we have* $h_C = 1 - R_0^{-1}$.

Proof. Recall that in this case all individuals in the household are infected by the initial (globally contacted) infective. Then, from Section 4.3.4, we have that $R_V = R_0$, so the critical vaccination coverage is $h_C = 1 - R_V^{-1} = 1 - R_0^{-1}$. □

5.4 The activity level model without household structure

We begin our comparison of $\tilde{h}_D$ and h_C by considering the activity level model in the absence of household structure. This model is a special case of the standard multitype SIR model in Andersson and Britton [2000], Chapter 6, with proportionate global mixing, obtained by taking $\mathscr{N} = \{\boldsymbol{e}_1, \boldsymbol{e}_2, \ldots, \boldsymbol{e}_J\}$, so that all households contain a single member. Note that R_0 and R_* coincide for this model due to the lack of household structure; it follows from (4.2) that R_0 for this model is given by

$$R_0 = \lambda \sum_{j=1}^{J} \beta_j^2 \gamma_j. \tag{5.5}$$

In Britton et al. [2020], Table 1, the disease-induced herd immunity level is calculated for a specific set of parameters in the activity level model and $\tilde{h}_D < h_C$ in the examples presented there; the following result provides a more general comparison of $\tilde{h}_D$ and h_C in the activity level model.

Theorem 5.2. *We have* $\tilde{h}_D \leq h_C$ *in the activity level model, with equality if and only if* $J = 1$.

Proof. If $J = 1$ then the epidemic is homogeneously mixing and $\tilde{h}_D = h_C$; assume hereafter that $J > 1$. Suppose a first epidemic is run in the activity level

model. Then, in the framework of Section 5.3.1 and using (5.2), we have

$$R_D(\pi) = \lambda \sum_{j=1}^{J} \beta_j^2 \gamma_j \pi^{\beta_j},$$

Letting $\pi \in (0,1)$ denote the probability a typical type-1 individual avoids infection, note that the probability a type-j individual avoids infection is given by π^{β_j} ($j \in \mathscr{J}$). Thus, letting $S(\pi)$ denote the proportion of susceptibles remaining after the first epidemic, we have

$$S(\pi) = \sum_{j=1}^{J} \gamma_j \pi^{\beta_j}.$$

Supposing instead that a fraction $S(\pi)$ of the population are vaccinated uniformly at random prior to the epidemic, writing $\tilde{R}_U(\pi) = R_U(S(\pi))$, and using (5.5) yields

$$\tilde{R}_U(\pi) = S(\pi) R_0 = \lambda \left(\sum_{j=1}^{J} \gamma_j \pi^{\beta_j} \right) \sum_{j=1}^{J} \beta_j^2 \gamma_j.$$

In order to compare $\tilde{R}_U(\pi)$ and $R_D(\pi)$ we make use of Chebyshev's "other" inequality - see Hardy et al. [1952], p.168, which states that for a random variable X and functions f, g which are both increasing or both decreasing, we have

$$\mathrm{E}[f(X)g(X)] \geq \mathrm{E}[f(X)]\mathrm{E}[g(X)]. \tag{5.6}$$

With a view toward applying Chebyshev's other inequality, we let X be a random variable having probability mass function

$$\mathrm{P}(X = i) = \gamma_i, \quad i \in \mathscr{J}.$$

We also define $f, g : \mathscr{J} \to \mathbb{R}$ by

$$f(i) = \beta_i^2, \quad g(i) = -\pi^{\beta_i}.$$

Note that f, g are increasing functions with

$$\begin{aligned}\mathrm{E}[f(X)g(X)] &= -\sum_{j=1}^{J} \gamma_j \beta_j^2 \pi^{\beta_j} \\ &= -\frac{1}{\lambda} R_D(\pi)\end{aligned}$$

and

$$\begin{aligned}\mathrm{E}[f(X)]\mathrm{E}[g(X)] &= -\left(\sum_{j=1}^{J} \gamma_i \pi^{\beta_j}\right)\left(\sum_{j=1}^{J} \gamma_j \beta_j^2\right) \\ &= -\frac{1}{\lambda}\tilde{R}_U(\pi).\end{aligned}$$

The only cases of equality in (5.6) occur when X has all its probability mass on one point or when at least one of f and g are constant. It is clear that neither of these conditions hold, since $\gamma_i > 0$ for all $i \in \mathscr{J}$ and $\beta_1 < \beta_J$. Chebyshev's other inequality then implies that $R_D(\pi) < \tilde{R}_U(\pi)$, so that $\tilde{h}_D < h_C$, which establishes the claim. □

Theorem 5.2 establishes that $\tilde{h}_D < h_C$ when individuals are typed based on their activity level in the absence of household structure, which is considered in a more general setting in Bootsma et al. [2023], Section 7. The result of Theorem 5.2 contrasts the observations of $\tilde{h}_D > h_C$ which are typical in the households model. In the remainder of this chapter we impose activity level and household structure on the population and investigate how the ordering of $\tilde{h}_D$ and h_C changes.

5.5 Highly locally infectious epidemics, common household size

Typically it is not possible to obtain orderings of $\tilde{h}_D$ and h_C in a model with activity level and household structure, owing to the complicated form of (5.1). However, in the case $\lambda_L \to \infty$, some analytical progress can be made. We investigate how the distribution of individuals of different types affects this ordering by considering two extreme cases; we study the scenario in which all households contain an individual of each type, as well as the case in which all households

only contain one type of individual. These cases represent the extremes in which individuals of different types mix locally as much or as little as possible.

5.5.1 Single household category

In the case where all household categories are the same we can establish an ordering between $\tilde{h}_D$ and h_C. We consider, without loss of generality, the case where all households are size J and there is one individual of each type. Other configurations can be recovered by setting $\beta_i = \beta_j$ for some $i, j \in \mathscr{J}$ as appropriate.

Theorem 5.3. *Consider the activity level and households model with all households category $\boldsymbol{n} = (1,1,\ldots,1)$, in the highly locally infectious case, such that there are $J > 1$ individuals in each household. We have $\tilde{h}_D > h_C$.*

Proof. Using the framework of Section 5.3.1, we have

$$R_D(\boldsymbol{\pi}) = \frac{\lambda}{n}\pi_1\pi_2,\ldots,\pi_J\left(\sum_{i=1}^{J}\beta_i\right)^2,$$

with the proportion of individuals infected in the first epidemic given by $z(\boldsymbol{\pi}) = 1-\pi_1\pi_2\ldots\pi_J$. Setting $R_D(\boldsymbol{\pi}) = 1$, we find

$$\tilde{h}_D = 1 - \frac{n}{\lambda\left(\sum_{i=1}^{J}\beta_i\right)^2} = 1 - R_*^{-1}.$$

The result follows by recalling that $h_C = 1 - R_0^{-1}$ and that $R_* > R_0$ when $R_* > 1$; see Section 4.3.4. □

A heuristic justification of Theorem 5.3 is as follows. In the activity level model, the ideal method of spreading infection is to target highly active individuals, as this will more quickly reduce the overall population susceptibility. However, in the present model, it is not possible to infect a more active individual without also infecting their remaining (lower activity) household members, due to the highly locally infectious assumption. Consequently, disease-induced herd immunity is not effective at gathering herd immunity.

5.5.2 Fully clumped activity levels

We now turn attention to the case where all individuals in a household are the same type, whence individuals of different types do not mix locally. Such an assumption is not unreasonable, particularly during COVID-19 where individuals, who were unable to perform social distancing due to their work, tended to reside in households together. It is therefore of interest to study a model in which individual types are clumped together, and to observe the impact this has on the disease-induced herd immunity level.

We begin this section with the following result which considers fixing R_0 and increasing the household size, generalising Theorem 2.3.

Theorem 5.4. *Let $\tilde{h}_D^{(n)}$ denote the disease-induced herd immunity level when all households are size n, with all individuals in a given household the same type and with $\lambda = \lambda^{(n)}$ chosen such that $R_0 > 1$ is held fixed. Then $\tilde{h}_D^{(n)}$ is increasing with n.*

Proof. Note that $R_0 = R_I$ in the highly locally infectious case. We calculate $\lambda^{(n)}$ such that R_0 is held fixed. In the notation of Section 4.3.2, we have $R_0^2 = aR_0 + \boldsymbol{u}^\top \boldsymbol{v}$, where $a = \lambda \sum_{j=1}^J \beta_j^2 \gamma_j$. Further, we have $u_j = (n-1)\beta_j\gamma_j$ and $v_j = \lambda\beta_j$. Fixing $R_0 > 1$, this gives

$$\lambda^{(n)} = \frac{R_0^2}{(R_0 + n - 1)\sum_{j=1}^J \beta_j^2 \gamma_j}.$$

Further, letting π ($\tilde{\pi}$) denote the probability a type-1 individual avoids global infection when all households are size n (size $n+1$) we have, in an obvious notation, $z = 1 - \sum_{j=1}^J \gamma_j \pi^{n\beta_j}$ and $\tilde{z} = 1 - \sum_{j=1}^J \gamma_j \tilde{\pi}^{(n+1)\beta_j}$. Suppose that $\pi^n = \tilde{\pi}^{n+1} = q \in (0,1)$, so that the same fraction z ($= \tilde{z}$) are infected at the end of the respective epidemics. The threshold parameters for the second epidemic, denoted $R_D^{(n)}$, satisfy

$$R_D^{(n)}(q) = \sum_{j=1}^J \beta_j^2 \gamma_j q^{\beta_j} R_D^{(n)}(1),$$

where $R_D^{(n)}(1) = n\lambda^{(n)}$. An elementary calculation establishes that $R_D^{(n)}(1)$ is in-

creasing with n, so that the solution to $R_D^{(n)}(q) = 1$ decreases as n increases. This implies a greater fraction infected in the first epidemic as n increases, whence $\tilde{h}_D^{(n+1)} > \tilde{h}_D^{(n)}$, as required. □

We observe for this household category distribution that, for fixed R_0, the disease-induced herd immunity level $\tilde{h}_D^{(n)}$ increases with n whilst, by Theorem 5.1, $h_C = 1 - R_0^{-1}$ is fixed. It is therefore of interest to find the limit of $\tilde{h}_D^{(n)}$ as $n \to \infty$. In the case of no activity levels, we show in Theorem 2.3 that $\lim_{n\to\infty} \tilde{h}_D^{(n)} = 1 - R_0^{-2}$, so that $h_D^{(n)} \in \left[1 - R_0^{-1}, 1 - R_0^{-2}\right)$. The following theorem extends this to the multitype case, where the corresponding limit as $n \to \infty$ is the solution to a nonlinear equation.

Theorem 5.5. *Under the same conditions and assumptions as Theorem 5.4, we have* $\lim_{n\to\infty} \tilde{h}_D^{(n)} = \tilde{h}_D^{(\infty)}$, *where*

$$\tilde{h}_D^{(\infty)} = 1 - \sum_{j=1}^{J} \gamma_j q_\infty^{\beta_j}, \tag{5.7}$$

with q_∞ *the unique solution in* $(0,1)$ *of*

$$\sum_{j=1}^{J} \beta_j^2 \gamma_j q^{\beta_j} = \frac{1}{R_0^2} \sum_{j=1}^{J} \beta_j^2 \gamma_j. \tag{5.8}$$

Proof. Note that, for each $n \in \mathbb{N}$, the equation $R_D^{(n)}(q) = 1$ has a unique solution in (0,1), forming a sequence $(q_n)_{n\geq 1}$. Further, $R_D^{(n)} \to R_D^{(\infty)}$ uniformly as $n \to \infty$, where

$$R_D^{(\infty)}(q) = \frac{R_0^2 \sum_{j=1}^{J} \beta_j^2 \gamma_j q^{\beta_j}}{\sum_{j=1}^{J} \beta_j^2 \gamma_j}.$$

It is clear that the sequence $(q_n)_{n\geq 1}$ is non-decreasing and bounded below, implying that $q_\infty = \lim_{n\to\infty} q_n$ exists. Moreover, the uniform convergence of $R_D^{(n)}$ to $R_D^{(\infty)}$ implies that $R_D^{(\infty)}(q_\infty) = 1$; it can easily be shown that this is the unique root of $R_D^{(\infty)}(q) = 1$ in $(0,1)$. Then q_∞ can be computed by solving (5.8), from which $\tilde{h}_D^{(\infty)}$ satisfies (5.7), as required. □

Theorem 5.4 implies that, provided we can find parameters $(\beta_1, \beta_2, \ldots, \beta_J)$ and $(\gamma_1, \gamma_2, \ldots, \gamma_J)$ with $\tilde{h}_D^{(\infty)} \leq 1 - R_0^{-1}$, we will have a scenario in which $\tilde{h}_D^{(n)} \leq$

h_C for all n. We consider extending Britton et al. [2020], Table 1, to the present highly locally infectious households setting. In Britton et al. [2020] the parameters $(\gamma_1,\gamma_2,\gamma_3)=\left(\frac{1}{4},\frac{1}{2},\frac{1}{4}\right)$ and $(\beta_1,\beta_2,\beta_3)=(1,2,4)$ were used, corresponding to half, standard and double activity. We consider these parameters in Table 5.1, as well as $(\beta_1,\beta_2,\beta_3)=(1,3,9)$ in Table 5.2.

	$n=1$	$n=2$	$n=3$	$n=4$	$n=5$	$n\to\infty$	h_C
$R_0=2.0$	37.7	48.6	52.5	54.5	55.7	60.8	50.0
$R_0=2.5$	46.3	57.7	61.8	64.0	65.3	70.9	60.0
$R_0=3.0$	52.5	63.8	67.9	70.1	71.5	77.3	66.7

Table 5.1: Values of $\tilde{h}_D$ and h_C for a model with all households size n, and all members of a given household the same activity level in the highly locally infectious case, taking $(\gamma_1,\gamma_2,\gamma_3)=(\frac{1}{4},\frac{1}{2},\frac{1}{4})$ and $(\beta_1,\beta_2,\beta_3)=(1,2,4)$. Values correspond to percentages and are rounded to 1 decimal place.

	$n=1$	$n=2$	$n=3$	$n=4$	$n=5$	$n\to\infty$	h_C
$R_0=2.0$	14.9	37.0	40.0	42.1	43.0	47.1	50.0
$R_0=2.5$	18.0	44.4	47.9	50.0	51.2	56.4	60.0
$R_0=3.0$	20.0	49.7	53.5	55.5	56.7	62.8	66.7

Table 5.2: Values of $\tilde{h}_D$ and h_C for a model with all households size n, and all members of a given household the same activity level in the highly locally infectious case, taking $(\gamma_1,\gamma_2,\gamma_3)=(\frac{1}{4},\frac{1}{2},\frac{1}{4})$ and $(\beta_1,\beta_2,\beta_3)=(1,3,9)$. Values correspond to percentages and are rounded to 1 decimal place.

Table 5.1 illustrates the impact of household structure; observe that $\tilde{h}_D$ exceeds h_C once the common household size $n\geq 3$. Table 5.2 provides an example of activity rates for which $\tilde{h}_D < h_C$ regardless of the common household size n, although for this model some individuals are far more active than others. In complete contrast to the household category distribution in Section 5.5.1, highly active individuals in this model can be infected without infecting individuals of the other types. Thus disease-induced herd immunity is able to be achieved efficiently since the epidemic can effectively target highly active individuals, giving a heuristic justification for why $\tilde{h}_D < h_C$ can occur if the activity levels are contrasting enough.

5.5.3 Fully clumped, two types framework

In this section we consider the case in which $\gamma_1 = 1 - \gamma_2 = p$, with activity rates $(\beta_1, \beta_2) = (1, \beta)$. We maintain the highly locally infectious assumption, with all households size n and all individuals in a given household the same type. We provide a framework for comparing $\tilde{h}_D$ and h_C, which in general is difficult to analyse for $\beta > 1$. We then make direct progress in the cases $\beta = 2$ and $\beta = 3$ respectively.

We drop the explicit dependence of threshold parameters on p and n for ease of exposition. Consider first disease-induced herd immunity and let $\pi \in (0,1)$ denote the probability a typical type-1 individual avoids global infection, with $q = \pi^n$. Using (5.2), we find that the threshold parameter for the second epidemic is given by

$$R_D(q) = n\lambda q(p + \beta^2(1-p)q^{\beta-1}), \tag{5.9}$$

with the final size of the first epidemic given by

$$z(q) = 1 - pq - (1-p)q^{\beta}.$$

Then, using (5.4), we have

$$R_U(c) = \lambda\{p + \beta^2(1-p)\}(1-c)\{1 + (n-1)(1-c))\}$$

Writing $\tilde{R}_U(q) = R_U(z(q))$, corresponding to vaccinating a proportion $z(q)$ of the population uniformly at random, we have

$$\tilde{R}_U(q) = \lambda q\{p + \beta^2(1-p)\}(p + (1-p)q^2)\{1 + (n-1)(pq + (1-p)q^{\beta})\}$$

For a given value of β, we wish to study where the function

$$\begin{aligned} f_{n,p}^{(\beta)}(q) &= \frac{1}{\lambda}(R_D(q) - \tilde{R}_U(q)) \qquad (5.10) \\ &= nq(p + \beta^2(1-p)q^{\beta-1}) - \{p + \beta^2(1-p)\}(1-c)\{1 + (n-1)(1-z(q))\} \end{aligned}$$

changes sign in $(0,1)$ in order to determine whether $\tilde{h}_D > h_C$. The location of these sign changes will typically depend on both n and p. Observe that, for all $\beta \geq 1$, we have $f_{n,p}^{(\beta)}(0) = f_{n,p}^{(\beta)}(1) = 0$; in cases where β is an integer we can then use algebraic division to write $f_{n,p}^{(\beta)}(q) = q(1-q)g_{n,p}^{(\beta)}(q)$. If $g_{n,p}^{(\beta)}(q) > 0$ ($g_{n,p}^{(\beta)}(q) < 0$) for all $q \in (0,1)$ then $\tilde{h}_D > h_C$ ($\tilde{h}_D < h_C$). If $g_{n,p}^{(\beta)}(q)$ changes sign over $(0,1)$ then both orderings of $\tilde{h}_D$ and h_C can occur, depending on λ. We consider $\beta = 2$ and $\beta = 3$, corresponding to the cases in which $g_{n,p}^{(\beta)}$ is quadratic and quartic respectively. In the sequel we suppress the dependence of f and g on β for ease of exposition.

Standard and double activity

The following theorem relates the ordering of $\tilde{h}_D$ and h_C to the common household size and the proportion of type-1 individuals in the case of standard and double activity.

Theorem 5.6. *Suppose all members of a given household are the same type, in the highly locally infectious case, with common household size $n > 1$. Suppose there are two activity levels, with standard and double activity. Let p denote the proportion of individuals who are type 1 (standard activity). If $n \geq 4$, then $\tilde{h}_D > h_C$ independently of the value of p. When $n \in \{2,3\}$, if $p \geq \frac{4-n}{3}$ then $\tilde{h}_D > h_C$, otherwise there is a value $\lambda^*(n,p)$ such that $\tilde{h}_D > h_C$ ($\tilde{h}_D < h_C$) for $\lambda < \lambda^*(n,p)$ ($\lambda > \lambda^*(n,p)$). Moreover, we have*

$$\lambda^*(n,p) = \left[np\hat{q}_n(p) + 4n(1-p)\hat{q}_n^2(p)\right]^{-1}, \tag{5.11}$$

where $\hat{q}_n(p)$ is the unique solution in $(0,1)$ to

$$(n-1)(1-p)^2(4-3p)q^2 + (n-1)(1-p^2)(4-3p)q + 3p^2 - (4-n)p = 0.$$

Proof. Substituting $\beta = 2$ into (5.10), we have

$$f_{n,p}(q) = q(1-q)g_{n,p}(q), \tag{5.12}$$

where

$$g_{n,p}(q) = (n-1)(1-p)^2(4-3p)q^2 + (n-1)(1-p^2)(4-3p)q + 3p^2 - (4-n)p.$$

Note that $g_{n,p}(q)$ is quadratic with positive coefficients of q^2 and q respectively. There are no positive solutions to $g_{n,p}(q) = 0$ if and only if the constant term is non-negative, which is equivalent to

$$p \geq \frac{4-n}{3}.$$

If $n \geq 4$ then the above condition is satisfied for all $p \in (0,1)$, yielding $\tilde{h}_D > h_C$ independently of the value of p. If $p < \frac{4-n}{3}$ then $g(q)$ has two roots which differ in sign. Now

$$g'(1) = (n-1)(4-3p)(1-p)(3-p) > 0,$$

which implies that the positive root of $g_{n,p}(q)$ belongs to $(0,1)$. The change in sign of $g_{n,p}(q)$, and hence of $f_{n,p}(q)$, occurs when $g_{n,p}(q) = 0$ which is given, by definition, by $\hat{q}_n(p)$. The change of behaviour occurs when $R_D(\hat{q}_n(p)) = \tilde{R}_U(\hat{q}_n(p)) = 1$. Substituting $\hat{q}_n(p)$ and $\beta = 2$ into (5.9) then establishes (5.11), as required. □

Theorem 5.6 implies that, in the present setting of standard and double activity, we can have $\tilde{h}_D < h_C$ provided the global infection rates are high enough. In Figure 5.1 we consider how high the global infection rate has to be for this to occur, by plotting the critical value ($R_0^*(n,p)$, say) at which $\tilde{h}_D = h_C$. Thus $\tilde{h}_D > h_C$ below the curve and $\tilde{h}_D < h_C$ above it.

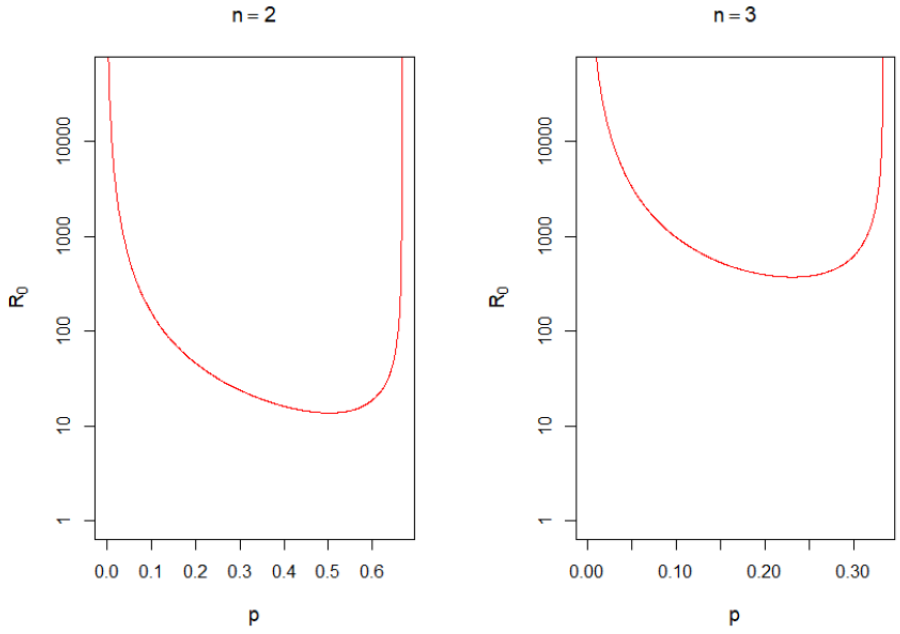

Figure 5.1: The value of $R_0^*(n,p)$ (on a logarithmic scale) as a function of p for $n \in \{2,3\}$.

Note that Figure 5.1 only considers $p \in (0, \frac{4-n}{3})$; for values of p outside of this interval there is no value of R_0 such that $\tilde{h}_D < h_C$. We observe that $R_0^*(3,p) > R_0^*(2,p)$, which is consistent with the fact that increasing household size makes a scenario in which $\tilde{h}_D < h_C$ more difficult to achieve. One can show, for $n = 2$ and $n = 3$, that $\lim_{p \uparrow \frac{4-n}{3}} R_0^*(n,p) = \infty$ and $\lim_{p \downarrow 0} R_0^*(n,p) = \infty$.

Standard and triple activity

We now consider the case of standard and triple activity, corresponding to taking $\beta = 3$. We begin with a supporting lemma regarding $g_{n,p}$, before restricting attention to the common household size $n = 2$ and $n = 3$ respectively.

Lemma 5.7. *When $\beta = 3$ and for all $p \in (0,1)$ and $n \geq 2$, we have $f_{n,p}(q) = q(1-q)g_{n,p}(q)$, where the function*

$$\begin{aligned} g_{n,p}(q) =& (n-1)(9-8p)(1-p)^2q^4 + (n-1)(9-8p)(1-p)^2q^3 \\ &+ (n-1)(9-8p)(1-p^2)q^2 \\ &- \{(n-1)(8p-9)p^2 + p(8p-9+n)\}q + p(8p-9+n) \end{aligned}$$

is convex on $\mathbb{R}$.

Proof. Let

$$\tilde{g}_{n,p}(q) = \frac{g_{n,p}(q)}{(n-1)(9-8p)(1-p)^2}.$$

We will show that $\tilde{g}''_{n,p}(q) > 0$, so that $g_{n,p}$ is convex. Appropriate differentiation yields

$$\begin{aligned}
\tilde{g}''_{n,p}(q) &= 12q^2 + 6q + \frac{2(1+p)}{(1-p)} \\
&= 12\left[\left(q+\frac{1}{4}\right)^2 - \frac{1}{16}\right] + \frac{2(1+p)}{(1-p)} \\
&= 12\left(q+\frac{1}{4}\right)^2 - \frac{3}{4} + \frac{2(1+p)}{(1-p)} \\
&> 0,
\end{aligned}$$

since $p \in (0,1)$. □

Theorem 5.8. *Suppose all members of a given household are the same type, in the highly locally infectious case, with common household size* $n = 2$. *Suppose there are two activity levels, with standard and triple activity. Let* p *denote the proportion of individuals who are type* 1 *(standard activity), and define*

$$p_\pm = \frac{37 \pm \sqrt{73}}{48}.$$

If $p \in [p_+, 1)$, *then* $\tilde{h}_D > h_C$. *If* $p \in (\frac{7}{8}, p_+)$ *then* $\tilde{h}_D < h_C$ $(\tilde{h}_D > h_C)$ *for* $\lambda < \lambda^*(p)$ $(\lambda > \lambda^*(p))$. *If* $p \in [p_-, \frac{7}{8}]$ *then* $\tilde{h}_D < h_C$. *If* $p \in (0, p_-)$ *then* $\tilde{h}_D > h_C$ $(\tilde{h}_D < h_C)$ *for* $\lambda < \lambda^*(p)$ $(\lambda > \lambda^*(p))$. *Moreover, we have*

$$\lambda^*(p) = \frac{1}{2}\left[p\hat{q}(p) + 9(1-p)\hat{q}^3(p)\right]^{-1},$$

where $\hat{q}(p)$ *is the unique solution in* $(0,1)$ *to*

$$\begin{aligned}
&(9-8p)(1-p)^2q^4 + (9-8p)(1-p)^2q^2 + (9-8p)(1-p^2)q^2 \\
&- p(1-p)(8p+7)q + p(8p-7) = 0.
\end{aligned}$$

Proof. Using the framework of Section 5.5.3 with $n = 2$ and $\beta = 3$ and letting

$f_{2,p} = f_p$ we have that $f_p(q) = q(1-q)g_p(q)$, where

$$g_p(q) = (9-8p)(1-p)^2q^4 + (9-8p)(1-p)^2q^2 + (9-8p)(1-p^2)q^2 \\ - p(1-p)(8p+7)q + p(8p-7).$$

Now g_p is convex by Lemma 5.7, so that g_p has at most two real roots. Note that $g_p(0) = p(8p-7)$ and $g_p(1) = 48p^2 - 74p + 27 = 0$ when $p = p_\pm$. Moreover,

$$g_p'(1) = (1-p)(32p^2 - 124p + 81).$$

We consider the possible values of p in turn. When $p \in (1, p_+]$, we have $g(1) \geq 0$, so that any real roots of g_p have the same sign. For such p, we have $g_p(0) > 0$, $g_p(1) > 0$ and $g_p'(1) < 0$, so that g_p has no real roots; it follows that $g_p(q) > 0$ for $q \in (0,1)$. When $p \in (\frac{7}{8}, p_+)$ we have $g_p(0) > 0$, so that any real roots of g_p have the same sign. Now $g_p(1) < 0$, so that g_p has a unique root in $(0,1)$.

Suppose now that $p \in [p, \frac{7}{8}]$. Then $g_p(0) \leq 0$ and $g_p(1) \leq 0$, so that $g_p(q) < 0$ for all $q \in (0,1)$. Finally, suppose that $p \in (0, p_-)$. We have $g_p(0) < 0$, which implies two roots that differ in sign. The fact that $g_p(1) > 0$ then establishes a unique root of g_p in $(0,1)$. The orderings regarding $\tilde{h}_D$ and h_C then follow from the discussion in Section 5.5.3. At points where behaviour changes from $\tilde{h}_D > h_C$ to $\tilde{h}_D < h_C$ we have $g_{n,p}(q) = 0$ which occurs, by definition, at $q = \hat{q}(p)$. At such a point, we have $R_D(\hat{q}(p)) = \tilde{R}_U(\hat{q}(p)) = 1$; substitution into (5.9) establishes the expression for $\lambda^*(p)$, which completes the proof. □

We next extend Theorem 5.8 to the case where all households are size 3.

Theorem 5.9. *Suppose all members of a given household are the same type, in the highly locally infectious case, with common household size $n = 3$. Suppose there are two activity levels, with standard and triple activity. Let p denote the proportion of individuals who are type* 1 (*standard activity*).

If $p \in [\frac{9}{10}, 1)$, then $\tilde{h}_D > h_C$. If $p \in (\frac{3}{4}, \frac{9}{10})$ then $\tilde{h}_D < h_C$ ($\tilde{h}_D > h_C$) for $\lambda < \lambda^*(p)$ ($\lambda > \lambda^*(p)$). If $p = \frac{3}{4}$ then $\tilde{h}_D < h_C$. If $p \in (0, \frac{3}{4})$ then $\tilde{h}_D > h_C$

$(\tilde{h}_D < h_C)$ *for* $\lambda < \lambda^*(p)$ $(\lambda > \lambda^*(p))$. *Moreover, we have*

$$\lambda^*(p) = \frac{1}{3}\left[p\hat{q}(p) + 9(1-p)\hat{q}^3(p)\right]^{-1},$$

where $\hat{q}(p)$ *is the unique solution in* $(0,1)$ *to*

$$\begin{aligned}&(9-8p)(1-p)^2q^4 + (9-8p)(1-p)^2q^3 + (9-8p)(1-p^2)q^2\\ &- p(1-p)(8p+3)q + p(4p-3) = 0.\end{aligned}$$

Proof. We use an argument analogous to that of the proof of Theorem 5.8, but taking $n = 3$ instead; using the notation of that theorem, we have

$$\begin{aligned}g_p(q) = 2\{&(9-8p)(1-p)^2q^4 + (9-8p)(1-p)^2q^3 + (9-8p)(1-p^2)q^2\\ &- p(1-p)(8p+3)q + p(4p-3)\},\end{aligned}$$

which is convex by Lemma 5.7. Now $g_p(0) = p(8p-6)$, $g_p(1) = 40p^2 - 66p + 27 = (4p-3)(10p-9)$, and $g_p'(1) = 2(1-p)(32p^2 - 120p + 81)$. If $p \in [\frac{9}{10}, 1)$ then $g_p(0) > 0$, $g_p(1) \geq 0$ and $g_p'(1) < 0$, so that $g_p(q) > 0$ for all $q \in (0,1)$. If $p \in (\frac{3}{4}, \frac{9}{10})$, then $g_p(0) > 0$ and $g_p(1) < 0$, implying g_p has a unique root in $(0,1)$. If $p = \frac{3}{4}$ then $g_p(0) = g_p(1) = 0$, and $g_p(q) < 0$ for all $q \in (0,1)$. Finally, if $p \in (0, \frac{3}{4})$ then $g_p(0) < 0$ and $g_p(1) > 0$, implying that g_p has a unique root in $(0,1)$. The ordering of $\tilde{h}_D$ and h_C, as well as the expression for $\lambda^*(p)$, follow from identical reasoning to that which is used in the proof of Theorem 5.8. □

Theorem 5.8 and Theorem 5.9 demonstrate the effect of increasing the activity rate of the second type, where we observe that $\tilde{h}_D > h_C$ is harder to achieve for fixed n when β is increased from $\beta = 2$ to $\beta = 3$. We expect that this ordering is "continuous" in β; supposing that $\tilde{h}_D > h_C$ for some fixed n and p, we would expect that this ordering still holds for $\beta' < \beta$. When $p \downarrow 0$ or $p \uparrow 1$ we find $\tilde{h}_D > h_C$; in this limiting case all households are size n with no typing, so this ordering is in agreement with the findings of Section 2.4.2.

5.6 Numerical analysis, $0 < \lambda_L < \infty$

In this section we consider numerical comparison of herd immunity levels without the highly locally infectious assumption. We take $T_I \sim \text{Exp}(1)$, recalling the discussion in Section 2.5.1, and, where we vary the local infection rate, we do this using p_L rather than λ_L. Where relevant, we note the connection between the numerical analysis when $p_L = 1$ and the results of Section 5.5. We thus consider $\tilde{h}_D$ and h_C as functions of p_L for various choices of household category distribution.

5.6.1 Variable clumping

In Section 5.5 we observed that the distribution of types among the households has an effect on the disease-induced herd immunity level. We now investigate the clumping of individuals of the same type as follows. Suppose that $J = 2$ and, initially, that all households are size 2. Suppose $\gamma_1 = 1 - \gamma_2 = \frac{1}{2}$. As in Section 4.2 we let $\theta_{\boldsymbol{n}}$ denote the proportion of households of category $\boldsymbol{n}$. We parameterise between full clumping and no clumping by letting $x_C \in [0,1]$ and setting

$$(\theta_{(2,0)}, \theta_{(1,1)}, \theta_{(0,2)}) = \left(\frac{x_C}{2}, 1 - x_C, \frac{x_C}{2}\right).$$

Thus $x_C = 0$ corresponds to the case where individuals of different types mix locally as much as possible, with $x_C = 1$ corresponding to maximal clumping of types, in which type-1 individuals only mix locally with type-1 individuals. The above idea can be extended to larger values of the common household size n, although such an extension is not unique without further parameterisation; we proceed in the case $n = 3$ by taking

$$(\theta_{(3,0)}, \theta_{(2,1)}, \theta_{(1,2)}, \theta_{(0,3)}) = \left(\frac{x_C}{2}, \frac{1 - x_C}{2}, \frac{1 - x_C}{2}, \frac{x_C}{2}\right).$$

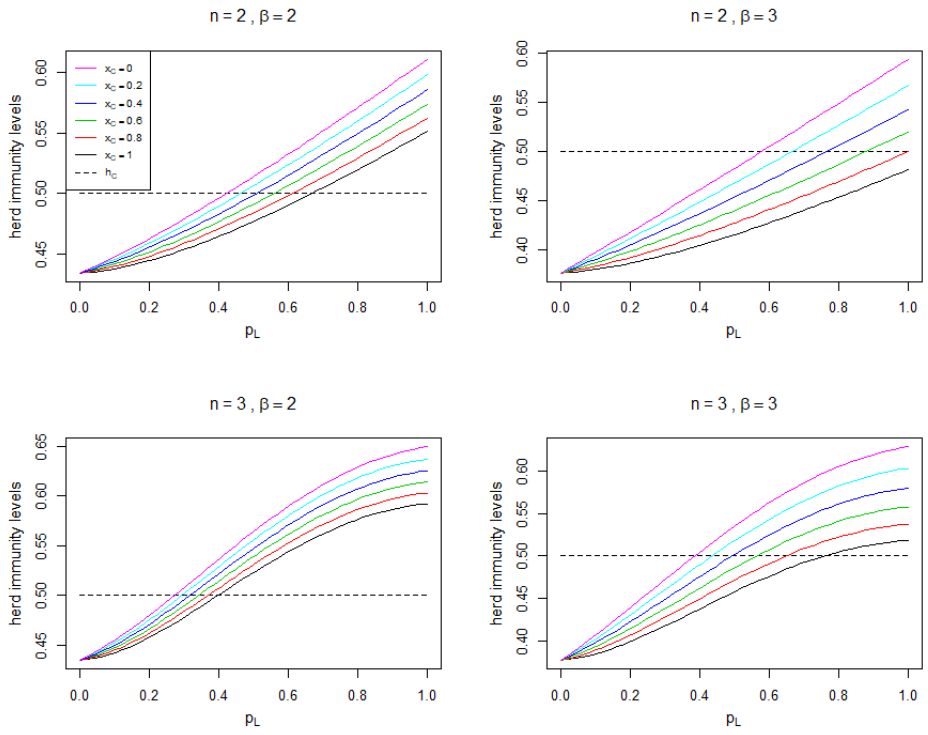

Figure 5.2: Herd immunity levels $\tilde{h}_D$ and h_C against p_L, when $\gamma_1 = \gamma_2 = 0.5$, for different values of the clumping parameter $x_C \in [0,1]$, $R_0 = 2$, common household size $n \in \{2,3\}$ and activity levels 1 and β, where $\beta \in \{2,3\}$.

In Figure 5.2 we plot $\tilde{h}_D$ as a function of p_L when $n = 2$ and $n = 3$ for fixed values of x_C, with $R_0 = 2$. Recall that, under these assumptions, $h_C = 1 - R_0^{-1} = \frac{1}{2}$ is constant. We also have $\tilde{h}_D < h_C$ when $p_L = 0$ by Theorem 5.2. Moreover, when $x_C = 0$, we have $\tilde{h}_D > h_C$ as $p_L \to 1$ owing to Theorem 5.3. We observe that $\tilde{h}_D$ increases with p_L, with lower values for $\tilde{h}_D$ as x_C increases (for fixed p_L). This illustrates the suspected trend that separating individuals of different types from mixing locally causes the disease-induced herd immunity level to decrease, as disease-induced herd immunity spreads more efficiently in that household structure. Moreover, in the plot corresponding to $n = 2$ and $\beta = 3$, we observe that $\tilde{h}_D < h_C$ for all p_L when $x_C = 1$. It is clear that, for intermediate values of p_L, the clumping affects the value of $\tilde{h}_D$, as well as whether $\tilde{h}_D > h_C$. The discrepancy between the values of $\tilde{h}_D$ obtained as x_C varies can be quite high; for example, the percentage increase in $\tilde{h}_D$ from $x_C = 1$ to $x_C = 0$ when $p_L = 1$ in the case $n = 3$, $\beta = 3$ is 21.2%.

We now consider a similar comparison when $\gamma_1 = 1 - \gamma_2 = p \neq \frac{1}{2}$. In this

case $x_C = 0$ loses its correspondence with the household structure of Theorem 5.3 in which all households have one individual of each type, and $x_C = 1$ does not correspond to fully clumped households. Letting $\tilde{p} = \min\{p, 1-p\}$, we take

$$(\theta_{(2,0)}, \theta_{(1,1)}, \theta_{(0,2)}) = (p - \tilde{p}(1-x_C), 2\tilde{p}(1-x_C), 1-p-\tilde{p}(1-x_C))$$

when all households are size 2. Note that this choice of parameterisation is such that $x_C = 1$ corresponds to type-1 and type-2 individuals being separated, i.e. clumped, as much as possible. The corresponding parameterisation when the common household size $n = 3$ is

$$(\theta_{(3,0)}, \theta_{(2,1)}, \theta_{(1,2)}, \theta_{(0,3)}) = (p - \tilde{p}(1-x_C), \tilde{p}(1-x_C), \tilde{p}(1-x_C), 1-p-\tilde{p}(1-x_C)).$$

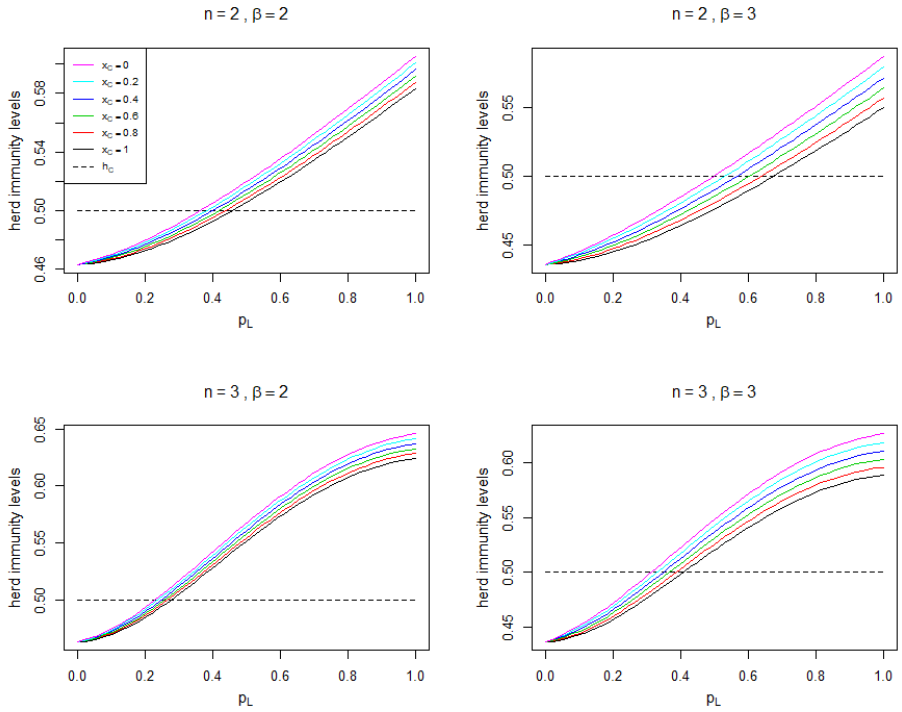

Figure 5.3: Herd immunity levels $\tilde{h}_D$ and h_C against p_L, when $\gamma_1 = \frac{1}{4}$, for different values of the clumping parameter $x_C \in [0,1]$, $R_0 = 2$, common household size $n \in \{2,3\}$ and activity levels 1 and β, where $\beta \in \{2,3\}$.

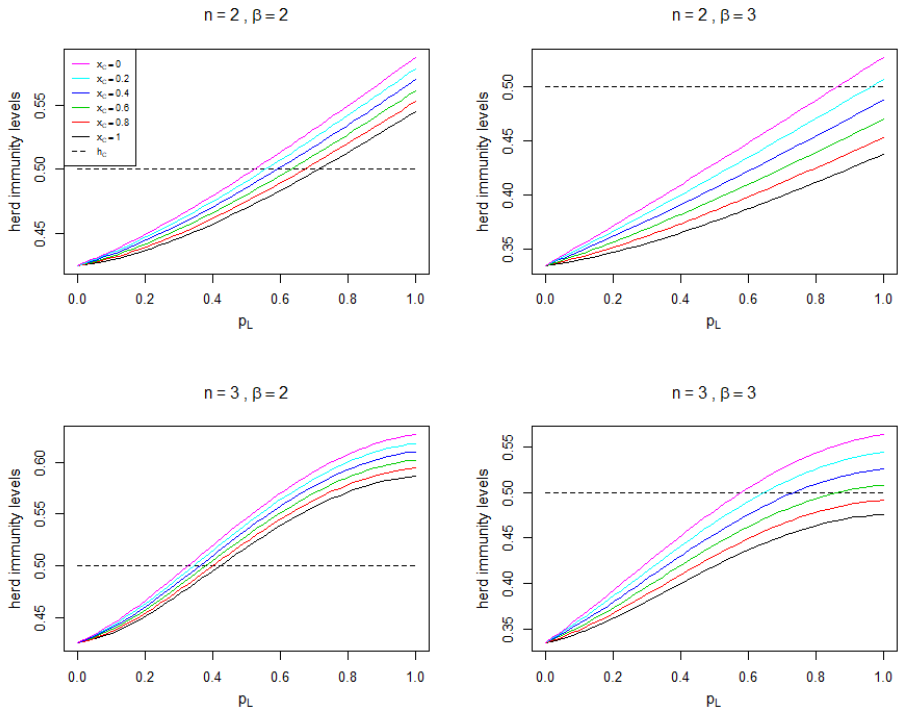

Figure 5.4: Herd immunity levels $\tilde{h}_D$ and h_C against p_L, when $\gamma_1 = \frac{3}{4}$, for different values of the clumping parameter $x_C \in [0,1]$, $R_0 = 2$, common household size $n \in \{2,3\}$ and activity levels 1 and β, where $\beta \in \{2,3\}$.

In Figure 5.3 (Figure 5.4) we take $p = \frac{1}{4}$ ($p = \frac{3}{4}$) and $R_0 = 2$, allowing x_C to vary. Broadly speaking, when $p = \frac{1}{4}$ we see $\tilde{h}_D > h_C$ for a larger range of values of p_L, likely owing to the fact that many type-1 individuals reside in households with a type-2 individual which, as discussed previously, is inefficient in terms of herd immunity. We again observe a large range of values for $\tilde{h}_D$ based on the value of the clumping parameter x_C.

5.6.2 Common household size with three types of individuals

In Section 5.6.1 we investigated the distribution of types using a clumping argument. As discussed there, such a method is not simple to parameterise when the common household size $n \geq 3$. Moreover, there are a vast number of choices for infection parameters and household category distributions, even for a fixed common household size n. In this section we focus on a specific subset of these choices and observe the behaviour of $\tilde{h}_D$ and h_C. In particular, we assume that there are three types of individual, in proportions $(\gamma_1, \gamma_2, \gamma_3) = (\frac{1}{4}, \frac{1}{2}, \frac{1}{4})$. We assume a common household size n, and that the activity levels are given by

$(1, \beta, \beta^2)$ for some $\beta \geq 1$. When $\beta = 2$ this parameterisation is analogous to that which was used by Britton et al. [2020], except incorporating household structure. We note that this choice of parameters is for the purposes of demonstrating how $\tilde{h}_D$ and h_C in a model with activity level and household structure, and other choices of parameter values are of interest – see Section 5.7 for further discussion of parameter selection.

The parameter β controls how different the three activity levels are from one another, with $\beta = 1$ corresponding to the single-type households model. In Figure 5.5 we consider an example in which we vary β, demonstrating the effect on $\tilde{h}_D$.

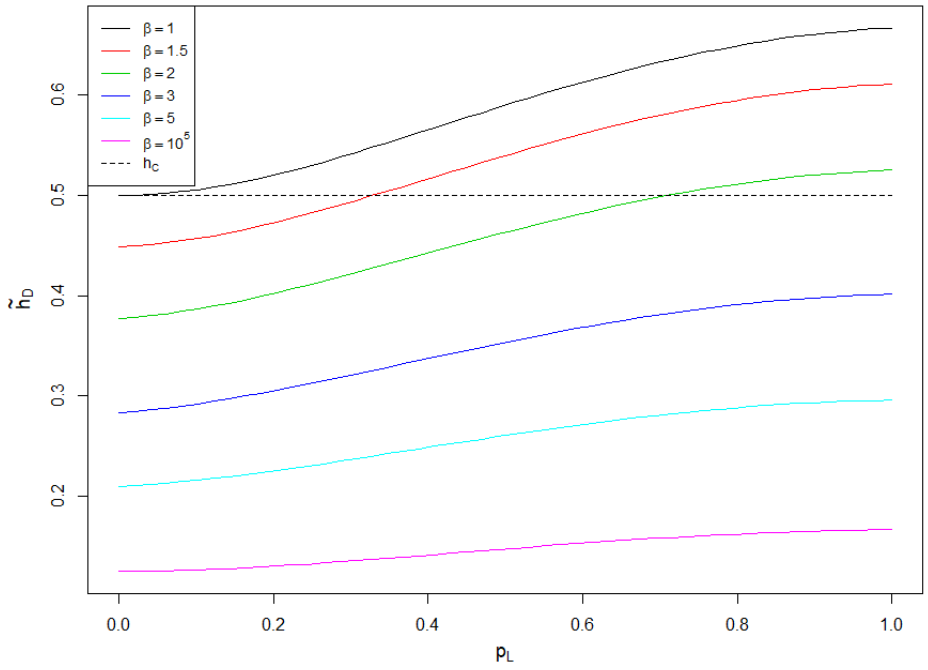

Figure 5.5: Values of $\tilde{h}_D$ for a population with common household size $n = 3$, activity levels $(1, \beta, \beta^2)$ for various values of β and $(\gamma_1, \gamma_2, \gamma_3) = (\frac{1}{4}, \frac{1}{2}, \frac{1}{4})$ and $R_0 = 2$.

In Figure 5.5 we assume that all individuals in a given household have the same type. When $\beta = 1$ we have $\tilde{h}_D > h_C$ (recall Theorem 2.15). The lower curves in Figure 5.5 correspond to increasing β, thus making the activity levels of different individuals differ more greatly. We observe, even for $\beta = 2$, a sharp reduction in $\tilde{h}_D$ compared to $\beta = 1$. As β increases further there are scenarios in

which $\tilde{h}_D < h_C$ for all $p_L \in (0,1)$. Moreover, when β is taken to be very large, we find that, for given values of R_0 and p_L, the value of $\tilde{h}_D$ differs from the corresponding value of $\tilde{h}_D$ in the households model (i.e. when $\beta = 1$) only by a multiplicative constant. We provide a justification of this as follows. As $\beta \to \infty$ there is one activity level which is far more active than the other two; when the epidemic begins the individuals with this activity level will get infected first. Since these individuals are far more active, achieving herd immunity among this group essentially achieves herd immunity for the population as well. This suggests that, as $\beta \to \infty$, the disease-induced herd immunity level is of the form $\gamma_3 \hat{\tilde{h}}_D$, where $\hat{\tilde{h}}_D$ is the disease-induced herd immunity level for a single type households epidemic with the given values of R_0 and p_L. If the assumption of individual types being fully clumped is removed, the above argument no longer holds, although it does provide a lower bound for $\tilde{h}_D$.

The above heuristic argument generalises to more than three types, provided one type is much more highly active than the others. (The parameterisation $(1, \beta, \beta^2)$ for the activity levels is not necessary for the above argument to hold.) The above argument also implies that, if β is large and γ_3 is small, $\tilde{h}_D$, which is bounded above by γ_3, will be small. This represents a case where disease-induced herd immunity is at its most efficient, because the population can be protected by a small number of the highly active individuals being infected. In particular, this is more efficient that uniform vaccination, which will vaccinate large proportions of individuals of other types when γ_3 is small.

The previous example considered varying the strength of activity levels. We next fix an activity level and allow the common household size n to vary. We persist with the same proportion of individuals of each type and consider two distributions of types. The first distribution of types is the fully clumped distribution in which all individuals in a given household are the same type. The second distribution assumes that the number of individuals of each type in a household of size n follows a multinomial distribution with probabilities $\boldsymbol{p} = (\frac{1}{4}, \frac{1}{2}, \frac{1}{4})$. In Figure 5.6 we plot $\tilde{h}_D$ and h_C for these choices of distribution when $\beta = 2$ and $R_0 = 2$ are fixed, where we vary both p_L and the common household size n. In Figure 5.7, we calculate, for each p_L, the corresponding

critical value ($\beta^*(p_L)$, say) of β such that $\tilde{h}_D = h_C$.

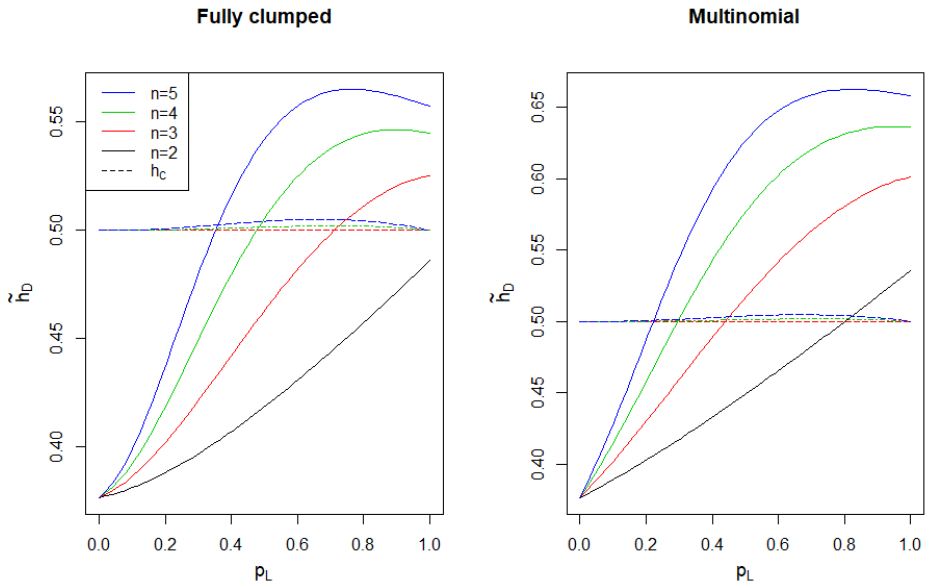

Figure 5.6: Values of $\tilde{h}_D$ and h_C for a population with common household size $n \in \{2,3,4,5\}$, activity levels $(1, \beta, \beta^2)$ with $\beta = 2$, $(\gamma_1, \gamma_2, \gamma_3) = (\frac{1}{4}, \frac{1}{2}, \frac{1}{4})$ and $R_0 = 2$. The left plot corresponds to fully clumped types and the right plot corresponds to a multinomial distribution for individual types.

Figure 5.6 is consistent with the previous discussions regarding clumping of individuals of the same type reducing the disease-induced herd immunity level – see the values on the y-axis. Broadly speaking, a larger local infection rate is required to achieve $\tilde{h}_D > h_C$ in the multinomial case than in the fully clumped case. In particular, we observe $\tilde{h}_D < h_C$ when all households are size 2 in the fully clumped case, but the same statement does not hold for a multinomial distribution of types. Note that $\tilde{h}_D$ is not monotone in p_L, since R_0 is being held fixed, and thus the global infection rates are implicitly changing. This behaviour is consistent with the households model in the absence of individual types – cf. Figure 2.10, which can be recovered by setting $\beta = 1$.

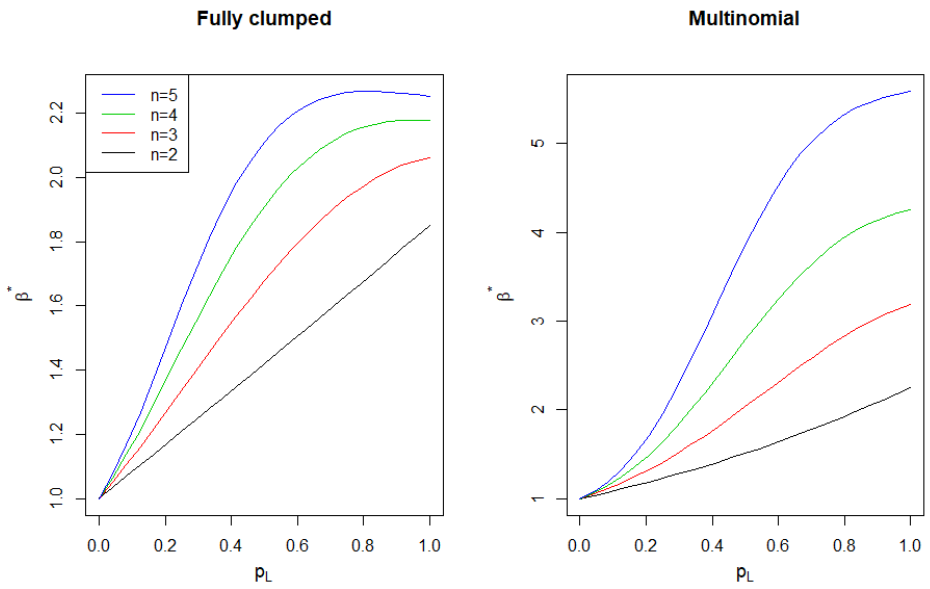

Figure 5.7: Critical value β^* of β such that $\tilde{h}_D = h_C$ for a population with common household size $n \in \{2,3,4,5\}$, activity levels $(1,\beta,\beta^2)$ with $(\gamma_1,\gamma_2,\gamma_3) = (\frac{1}{4},\frac{1}{2},\frac{1}{4})$ and $R_0 = 2$. The left plot corresponds to fully clumped types and the right corresponds plot to a multinomial distribution for individual types.

Comparison of different household sizes in Figure 5.7 illustrates that, for fixed R_0, a larger value of β is required in order to achieve $\tilde{h}_D = h_C$ as the household size n increases. (Note that if $n = 1$ then $\beta^*(p_L) = 1$ is constant, since any introduction of activity levels will lead to $\tilde{h}_D < h_C$.) Moreover, comparing the multinomial and fully clumped cases, we see that the required value of β for the fully clumped case is lower than the corresponding value for the multinomial case. The fact that $\beta^*(p_L)$ does not increase monotonically in p_L is suggested by the fact that $\tilde{h}_D$ is not monotonic in p_L (recall Figure 5.6).

5.6.3 Real-world household size distributions

We consider some brief numerical analysis of herd immunity levels using real-world household size distributions. We focus first on the UK household size distribution, before giving a comparison between countries with quite different mean household sizes – recall Figure 2.12.

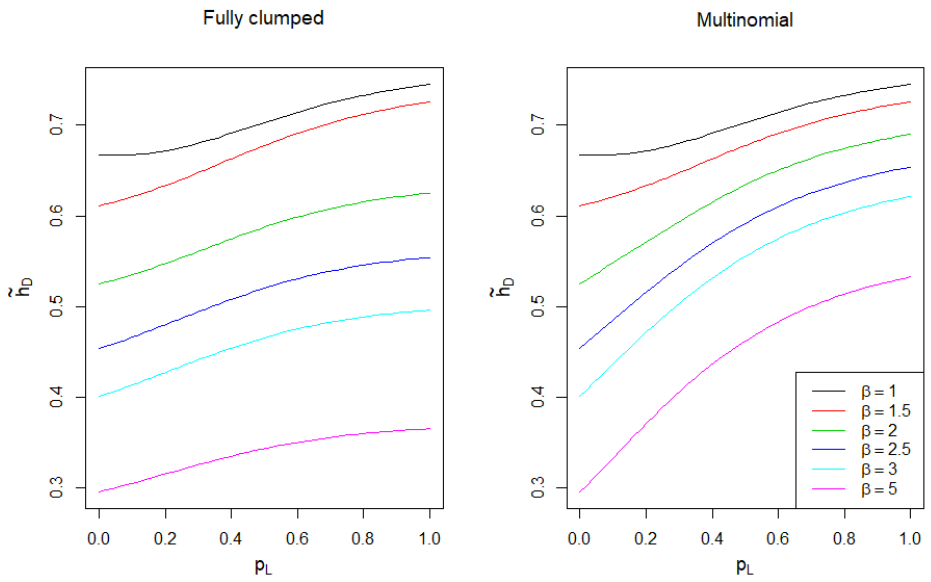

Figure 5.8: Values of $\tilde{h}_D$ against p_L for $\beta \in \{1, 1.5, 2, 2.5, 3, 5\}$, using the UK household size distribution and taking $R_0 = 3$. The left plot corresponds to fully clumped types and the right plot corresponds to a multinomial distribution of types; in both cases $(\gamma_1, \gamma_2, \gamma_3) = (\frac{1}{4}, \frac{1}{2}, \frac{1}{4})$.

Figure 5.8 provides another comparison of the disease-induced herd immunity levels when the individual types are fully clumped or follow a multinomial distribution. It is clear that the disease-induced herd immunity level can reduce drastically from the corresponding value in the households model (when $\beta = 1$) with this difference being even larger in the case when individual types are fully clumped. Moreover, the increase in $\tilde{h}_D$ as p_L increases is much less for the fully clumped case compared to the multinomial case. We suspect that this is because of the aforementioned impact of allowing highly globally active individuals to mix locally with individuals of a lower activity level, so that in the multinomial case the local mixing has more of an impact on the disease-induced herd immunity level.

We next consider the UK household size distribution with two types of individuals, with rates 1 and β, and let the number of individuals of type 1 follow a binomial distribution.

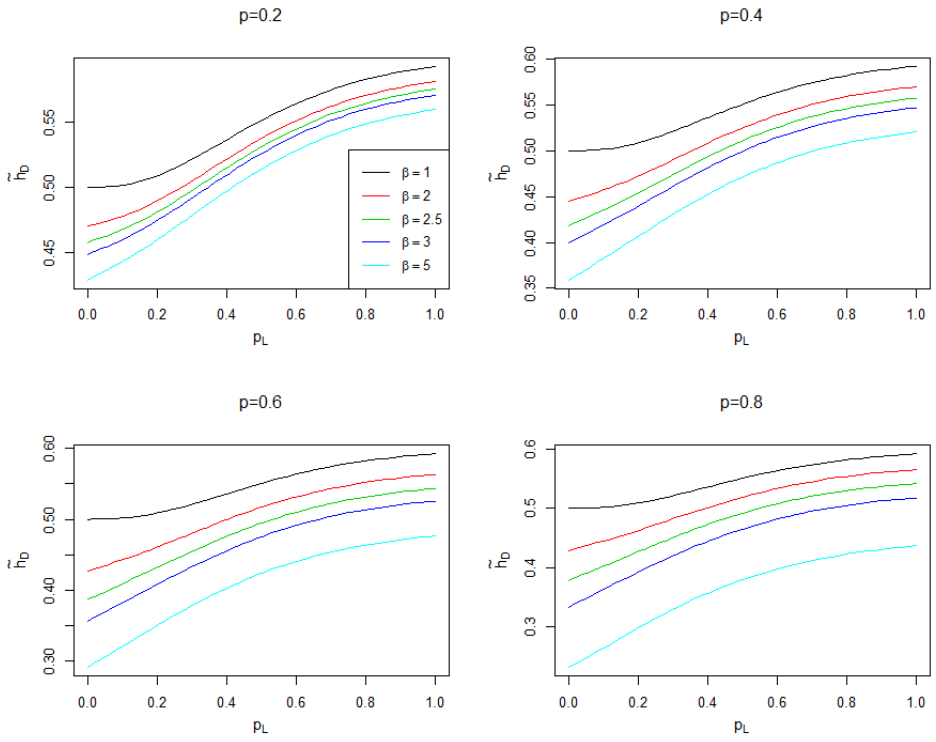

Figure 5.9: Values of $\tilde{h}_D$ against p_L for $\beta \in \{1, 2, 2.5, 3, 5\}$, using the UK household size distribution and taking $R_0 = 2$. The number of type-1 individuals in a household of size n is binomial with parameters (n, p).

The numerical results given by Figure 5.9 are qualitatively similar for different values of p; other choices of p, R_0 and real-world household size distribution produced similar results. The disease-induced herd immunity level again is highly variable and dependent on the activity rate β chosen. Broadly speaking, when the proportion p of type-1 individuals is small the values of $\tilde{h}_D$ are closer together, suggesting that the effect of the activity levels is less when most individuals belong to the higher activity level.

We conclude our numerical analysis by giving comparisons $\tilde{h}_D$ and h_C for a few different real-world household size distributions, viz. Mexico, Morocco, Pakistan, Sweden and UK – see Figure 5.10.

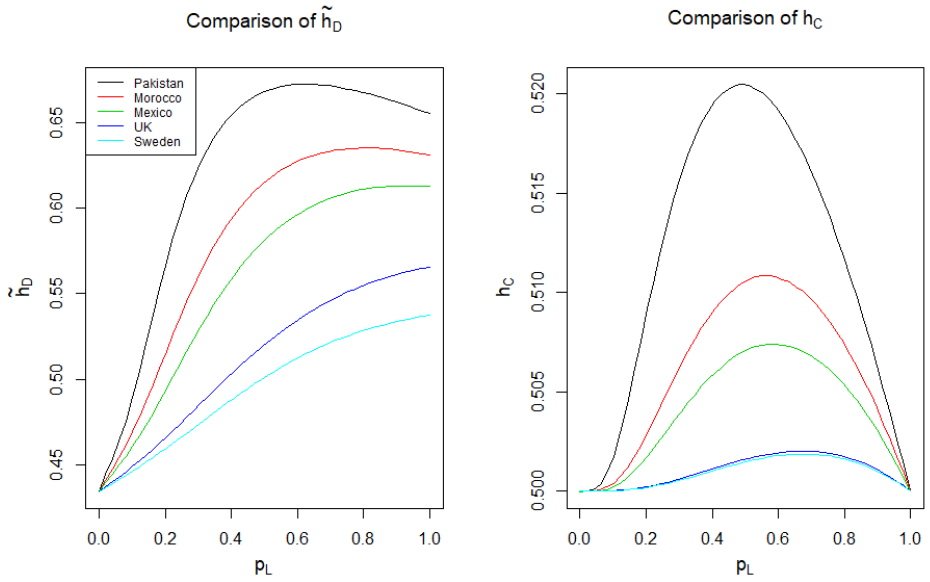

Figure 5.10: Values of $\tilde{h}_D$ against p_L for several real-world household size distributions, with activity levels $(\beta_1, \beta_2) = (1, 2)$. The number of type-1 individuals in a household of size n is binomial with parameters $(n, \frac{1}{2})$.

Whilst $h_C = 1 - R_0^{-1}$ when $p_L = 0$ and $p_L = 1$, we note that, in Figure 5.10, h_C does not change much for intermediate values of p_L; using $1 - R_0^{-1}$ as a baseline for comparison with $\tilde{h}_D$ is certainly reasonable. Note that both $\tilde{h}_D$ and h_C are larger in Figure 5.10 for countries with a larger mean household size. This is consistent with the findings of Chapter 2. Varying the proportions of individuals of type 1, as in Figure 5.9 leads to very similar observations, although they are omitted here for succinctness.

5.7 Discussion

We have provided a framework for studying $\tilde{h}_D$ and h_C in a model combining activity levels and household structure, as well as proving that $\tilde{h}_D < h_C$ for a model with activity levels only. In the highly locally infectious case we have derived conditions upon which $\tilde{h}_D > h_C$ for particular household category distributions. We have shown, and justified heuristically, that the rate at which individuals of different types mix locally is integral to the ordering of $\tilde{h}_D$ and h_C. We have also illustrated that, for models calibrated by R_0, increasing the household size tends to increase $\tilde{h}_D$. In contrast, when individual activity levels become more different from one another, we have observed that $\tilde{h}_D$ decreases.

From a healthcare perspective, we would like to provide a reliable estimate of the disease-induced herd immunity level. It is therefore, owing to the above calculations, important to ascertain how highly clumped the highly globally active individuals are. It is also of interest to study how much "more active" an active individual is – we have seen that this can drastically impact the disease-induced herd immunity level. These numerical calculations also support the idea of vaccinating first the most highly active individuals; doing so mimics the efficient spread of disease-induced herd immunity. As a result, the assumptions of activity level and household structure illustrate a more complex and changeable scenario in terms of herd immunity than that of Chapter 2.

As noted in Section 5.1 it is possible to compute h_D for the activity level and households model. Although the number of ODEs does quickly grow, one possible extension is to compute h_D for households of size 2 or 3, and to compare h_D to $\tilde{h}_D$ and h_C. We expect that h_D would be approximated well by $\tilde{h}_D$ for this model, as in the households model, with the approximation being worse as the maximum household size increases.

The progress made in the case of two fully clumped types in Section 5.5.3 allows for some orderings of $\tilde{h}_D$ and h_C, particularly when β is an integer because the resulting functions are polynomials. It may be possible to extend these results to non-integer β, provided a different method is used to study the resulting function $g_{n,p}^{(\beta)}$. It is likely that convexity still holds, although the fact that $g_{n,p}^{(\beta)}(0) = g_{n,p}^{(\beta)}(1) = 0$ can no longer be used to help factorise $g_{n,p}^{(\beta)}$. Despite this, we have been able to gain insight from integer values of β in terms of observing the effect of activity levels on disease-induced herd immunity.

It is clear that further numerical analysis could be conducted in order to investigate in more detail the interaction between activity level and household structure in the context of herd immunity. It is possible to do similar analysis to that of Section 5.6 when individuals are typed locally; we have omitted such analysis owing to a lack of obvious parameter choices for local infection rates, as well as for succinctness. Similar analysis could also be done for a model with only variable susceptibilities, by appropriate modification of the model of Chapter 4, as well as in the absence of proportionate mixing. Moreover, it would

be good to apply the analysis of this chapter to real-world data. We have already, in Section 2.5.4, given a method to estimate household size distributions from data. We would like be able to efficiently estimate the contact structure of the population of interest. Recall that, particularly in cases where a small proportion of individuals are much more active than all others, the disease-induced herd immunity level can be drastically reduced below the level suggested by a model with only household structure. Being able to accurately estimate contact rates is highly dependent on the data available. For example, Wallinga et al [2006] estimated contact rates among different age groups, using maximum likelihood estimation on survey data. Choosing a sensible number of activity levels, as well as how much these levels differ from one another, remains an open problem.

6 Impact of global restrictions on disease-induced herd immunity

6.1 Introduction

In this chapter we study disease-induced herd immunity, for a population under global restrictions, in more detail. Recall, in Chapter 2, the method to obtain $\tilde{h}_D$, in which we reduce global transmission rates such that a first epidemic terminates when R_* among the remaining susceptible population reaches one. In doing so we assume that local infection rates are unaffected. In practice, local infection rates are likely to increase as a result of global restrictions, as individuals would spend more time than usual in their household. We consider applying restrictions that decrease the rate of global mixing, but increase the rate of local mixing and observe the effect this has on the disease-induced herd immunity level.

As seen in Section 2.3.2, global restrictions in the households model can result in a scenario in which a first epidemic will terminate with the population having achieved herd immunity. If restrictions are too strong, however, there is a possibility that no major outbreak can occur until the restrictions are relaxed. In such a scenario, herd immunity is not achieved at the time of ending restrictions. Moreover, if global restrictions are too weak to prevent a first wave of infection, a scenario may occur in which the remaining susceptible population has not achieved herd immunity and is vulnerable to a second major outbreak. We prove that these behaviours are possible in the sequel. We then ascertain under what conditions two such major outbreaks are preferable to a single epidemic, in terms of the proportion of individuals ever infected. We also make a connection between the stopping of an unrestricted epidemic in real time and running a globally restricted epidemic to termination.

Imposing global restrictions on mixing rates is a type of epidemic control, which has been the subject of several studies, in particular in the case of deterministic SIR models. Morton and Wickwire [1974] consider such a problem and find that the optimal policies are of the form of "bang-bang" controls, where the

policy switches from no control to full control. Bolzoni et al. [2017] considered minimising the eradication time of the homogeneously mixing SIR epidemic via control, where the eradication time refers to the time at which the proportion of infectives falls to a specified level. Note that our control measure is transmission reduction, which is one of four measures outlined in that paper. Behncke [2000] sought optimal control in an attempt to minimise the total proportion infected, finding the optimal control to be "maximum effort on some time interval". They considered a deterministic SIR model with more general dynamics than the homogeneously mixing model. In these and many other examples, the typical approach is to apply control measures to the deterministic SIR epidemic model, usually by modifying the associated ODEs, and to use Pontryagin's Minimum Principle for linear time optimal control problems (see Pontryagin [2018]) to establish the optimal control. Optimal control in disease management is also considered by Bussell et al. [2019]. In contrast to the aforementioned methods, we proceed by using final size results, thus benefiting from the extra generality of not requiring the assumption that infectious period distributions are Markovian, as well as the reduced complexity of not having to study the epidemic in real time.

This chapter is structured as follows. We begin in Section 6.2 by defining a new disease-induced herd immunity level $\tilde{h}_D^*$ for the households model which accounts for the potential for greater local spread at a time of global restrictions, which we compare to the other disease-induced herd immunity levels defined in Section 2.3.2. (We are grateful to Tom Britton for suggesting this idea and for his insight during discussions regarding this chapter.) We then, in Section 6.3, consider in more depth the scenario where restrictions are applied throughout a first epidemic, before being completely removed for a second epidemic. If restrictions are too strong for the population to be able to achieve herd immunity during the first epidemic, two major outbreaks may occur. We show that a single, globally restricted, epidemic which terminates with the population at criticality always leads to fewer individuals infected than any other choice of global restriction that are applied uniformly throughout only the first epidemic. We show this for the multitype households model with proportionate global mixing, in-

troduced in Chapter 4, from which it is immediate that the result also holds for the households model and the multitype model with proportionate mixing.

6.2 Increased local infection during global restrictions: the disease induced herd immunity level $\tilde{h}_D^*$

As discussed in Section 6.1, it is likely that, in practice, the local infection rate will increase during a period of global restrictions, owing to individuals being unable to mix at their usual rate with individuals outside their household. We briefly investigate how this affects the disease-induced herd immunity level, by defining a new disease-induced herd immunity level $\tilde{h}_D^*$ and comparing it to the herd immunity levels defined in Chapter 2.

6.2.1 Common household size

Recall the definition of $\tilde{h}_D$ in Section 2.3.2. We extend this to the case where local infection is increased during global restrictions. We assume that the local infection rate increases in such a way that the mean total number of contacts made by an individual remains fixed. Consider first the model with common household size $n > 1$ and $\mathrm{E}[T_I] = 1$. Assume that $R_* > 1$, since otherwise herd immunity would already have been achieved. In that model the mean total number of contacts (λ, say) made by a typical individual during their infectious period satisfies

$$\lambda = \lambda_G + (n-1)\lambda_L.$$

It follows that λ will remain fixed if the first epidemic is run with infection parameters $(\alpha\lambda_G, \beta(\alpha)\lambda_L)$, where $\alpha \in (0,1)$ and

$$\beta(\alpha) = 1 + \frac{\lambda_G(1-\alpha)}{\lambda_L(n-1)}.$$

Let $\mathscr{E}_n(\lambda_G, \lambda_L)$ denote an epidemic in the households model with common household size n. In order to calculate $\tilde{h}_D^*$, we first run $\mathscr{E}_n(\alpha\lambda_G, \beta(\alpha)\lambda_L)$ to its conclusion, followed by an unrestricted epidemic (i.e. $\mathscr{E}_n(\lambda_G, \lambda_L)$) among the remaining susceptible population. We choose $\alpha = \alpha_*$ (cf. $\kappa = \kappa_*$ in Section 2.3.2)

such that the second epidemic is at criticality. (Note that α_* always exists since taking $\alpha = 0$ leaves the second epidemic supercritical and taking $\alpha = 1$ leaves the second epidemic subcritical.) Then $\tilde{h}_D^*$ is the final size of $\mathscr{E}_n(\alpha_*\lambda_G, \beta(\alpha_*))$. We anticipate that this modification, to increase the local infection rate as well as reduce the global infection rate during the first epidemic, will increase the disease-induced herd immunity level even further, owing to the extra clumping from the increased local infection rates; we expect that $\tilde{h}_D^* > \tilde{h}_D$ will typically be true. The following theorem considers the case $n = 2$.

Theorem 6.1. *Consider the households epidemic model with common household size $n = 2$, with $\lambda_L \in (0,\infty)$ and $R_* > 1$. We have $\tilde{h}_D^* > \tilde{h}_D$.*

Proof. We proceed by comparing reproduction numbers. Suppose that $\mathscr{E}_2(\alpha\lambda_G, \lambda_L)$ and $\mathscr{E}_2(\alpha'\lambda_G, \beta\lambda_L)$ infect the same proportion (z) of the population, with global escape probabilities π and π^* respectively. It follows that $\pi^* > \pi$, since the local infection is greater in the latter epidemic. Let P_i $(i = 0,1,2)$ denote the proportion of households with i immune members after $\mathscr{E}_2(\alpha\lambda_G, \lambda_L)$, and define P_i^* $(i = 0,1,2)$ analogously. In an obvious notation (cf. Section 2.4.4), we have

$$R_D(\lambda_L) = \lambda_G \mathrm{E}[T_I] \sum_{v=0}^{2} \left(1 - \frac{v}{2}\right) P_v \mu_{2-v}(\lambda_L)$$
$$R_D^*(\lambda_L) = \lambda_G \mathrm{E}[T_I] \sum_{v=0}^{2} \left(1 - \frac{v}{2}\right) P_v^* \mu_{2-v}(\lambda_L).$$

Note that, since both epidemics are assumed to infect the same proportion z of the population, we have

$$2P_0 + P_1 = 2P_0^* + P_1^*.$$

Note also that $A := 2\left(P_0^* - P_0\right) = 2(\pi^* - \pi) > 0$, so that

$$\begin{aligned} R_D^*(\lambda_L) - R_D(\lambda_L) &= \lambda_G \mathrm{E}[T_I] \left[(P_0^* - P_0)\,\mu_2(\lambda_L) + \frac{1}{2}(P_1^* - P_1)\mu_1(\lambda_L) \right] \\ &= A\lambda_G \mathrm{E}[T_I]\,[\mu_2(\lambda_L) - \mu_1(\lambda_L)] > 0, \end{aligned}$$

from which it follows that $\tilde{h}_D^* > \tilde{h}_D$. □

A proof of $\tilde{h}_D^* > \tilde{h}_D$ has not been forthcoming for $n > 2$; we make the

following conjecture.

Conjecture 6.2. *Consider the households epidemic model with common household size $n > 1$, with $\lambda_L \in (0,\infty)$ and $R_* > 1$. Then $\tilde{h}_D^* > \tilde{h}_D$.*

In Figure 6.1 we provide brief numerical support for Conjecture 6.2, observing that $\tilde{h}_D^* > \tilde{h}_D > h_D > h_C$. (Note that $n = 2$ in Figure 6.1 is consistent with Theorem 6.1.) As discussed previously, the clumping of the remaining susceptible population is greater under $\tilde{h}_D^*$ than under $\tilde{h}_D$ due to the extra local infection. When $\lambda_L = 0$ ($\lambda_L \to \infty$) the within-house epidemics infect none of (all of) the household, so $\tilde{h}_D^* = \tilde{h}_D$ in these cases. Similar behaviours occur for other choices of λ_G, with the difference between $\tilde{h}_D^*$ and h_C increasing as the common household size n increases.

In Figure 6.2 we illustrate the clumping of remaining susceptibles by considering the proportion of households with i members infected ($0 \leq i \leq n$) under $\tilde{h}_D^*$, $\tilde{h}_D$ and h_C respectively.

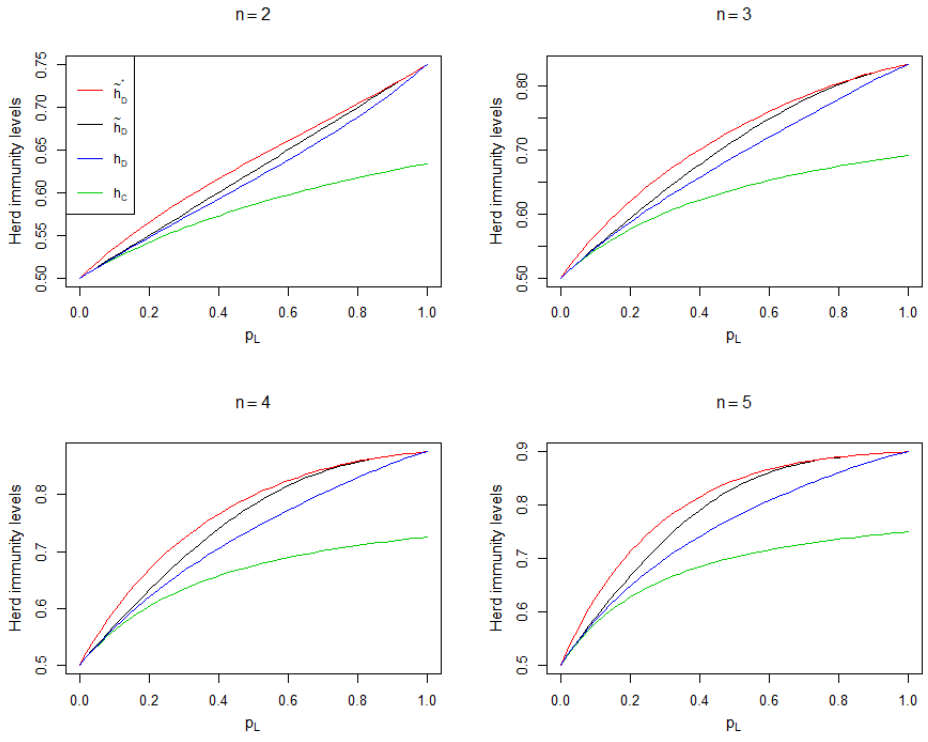

Figure 6.1: Comparison of herd immunity levels for common household size $n \in \{2,3,4,5\}$, with $\lambda_G = 2$, as p_L varies.

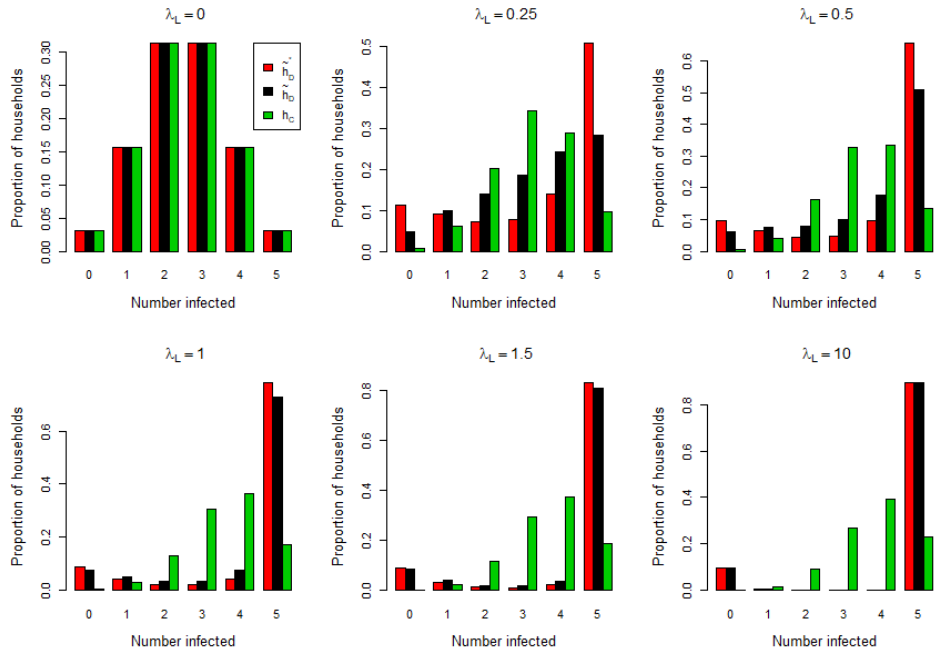

Figure 6.2: Comparison of household states under $\tilde{h}_D^*$, $\tilde{h}_D$ and h_C for various values of λ_L, with common household size $n = 5$ and $\lambda_G = 1.5$.

In Figure 6.2 we take $(n, \lambda_G) = (5, 1.5)$; note that other choices of (n, λ_G) produce qualitatively similar results. Under h_C individuals are vaccinated uniformly at random, so that the number of individuals infected in each household follows a binomial distribution with parameters n and h_C. It is clear in Figure 6.2 that the remaining susceptibles are more clumped under $\tilde{h}_D^*$ than $\tilde{h}_D$, further underlining the justification of Conjecture 6.2.

6.2.2 Unequal household sizes

In this section we briefly discuss the extension of $\tilde{h}_D^*$ to the households model with unequal household sizes, together with numerical examples of the typical ordering of the herd immunity levels. Assume that $\mathrm{E}[T_I] = 1$, without loss of generality, and consider an individual chosen uniformly at random from the population. Such an individual resides in a household of size n with probability $\tilde{\alpha}_n$, so the mean total number of contacts made by a typical individual during

their infectious period is given by

$$\begin{aligned}\lambda &= \sum_{n=1}^{\infty} \tilde{\alpha}_n \left(\lambda_G + (n-1)\lambda_L\right) \\ &= \lambda_G + \lambda_L \mathrm{E}[\tilde{H} - 1].\end{aligned}$$

This can be held fixed by running the first epidemic with infection parameters $(\alpha\lambda_G, \beta(\alpha)\lambda_L)$, where

$$\beta(\alpha) = 1 + \frac{\lambda_G(1-\alpha)}{\lambda_L \mathrm{E}[\tilde{H} - 1]}.$$

Then $\tilde{h}_D^*$ can be calculated in an analogous manner to the calculation for the households model with a common household size. As in the case of common household size, the typical behaviour is to have $\tilde{h}_D^* > \tilde{h}_D > h_D > h_C$; see Figure 6.3 for an example with real-world household size distributions. Thus the assumption of increased local infection rates during global restrictions typically increases the disease-induced herd immunity level even further.

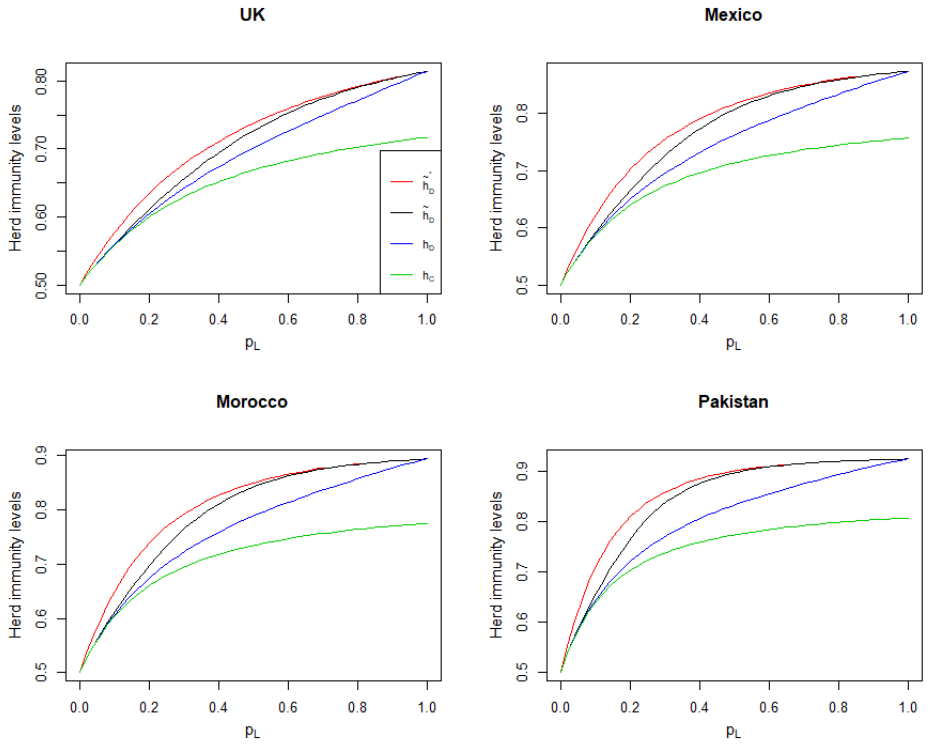

Figure 6.3: Comparison of herd immunity levels using real-world household size distributions for UK, Mexico, Morocco and Pakistan, with $\lambda_G = 2$ as p_L varies.

It is natural to question whether this ordering of herd immunity levels can be reversed. Whilst precise conditions for this inequality to hold are difficult to establish, we show in Figure 6.4 a numerical example with $\tilde{h}_D^* < \tilde{h}_D < h_D < h_C$.

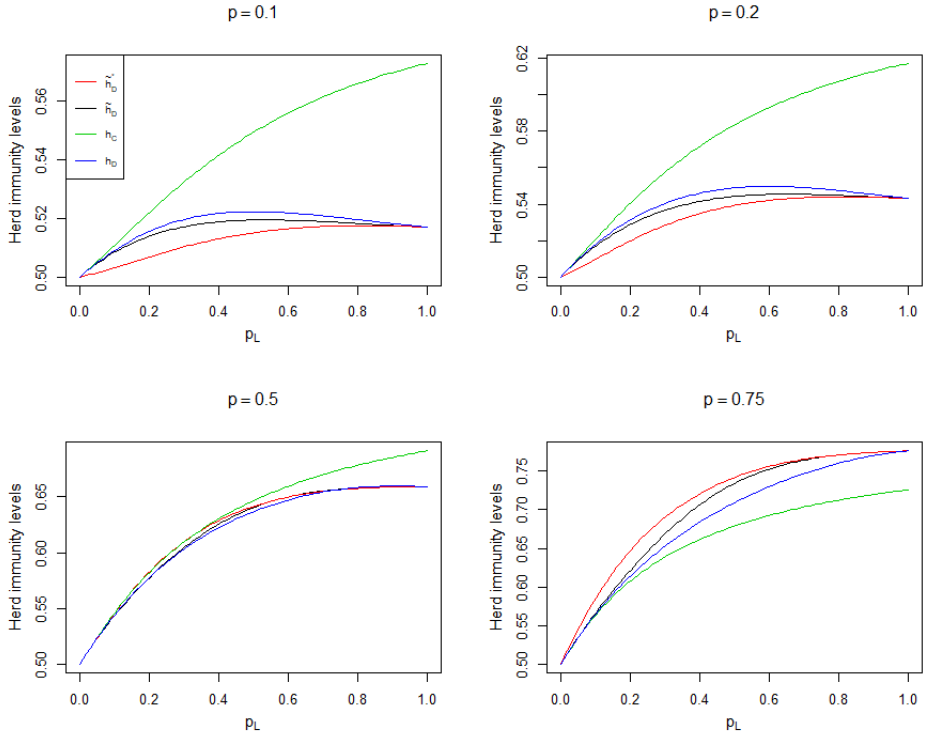

Figure 6.4: Comparison of herd immunity levels with $\lambda_G = 2$ and $\tilde{\alpha}_5 = 1 - \tilde{\alpha}_1 = p$.

Note that in Figure 6.4 the population comprises of households of size 1 and 5 only. We see that $\tilde{h}_D^* < \tilde{h}_D$ when the size-biased proportion p of individuals in households of size 5 is small, with the inequality changing as p varies. In this case the majority of individuals belong to single-member households which are not affected by the increase in local infection rates. Figure 6.4 is consistent with findings in Chapter 2 in which sufficiently high variance of the household size distribution can lead to $h_C < h_D$; it appears these findings extend, in the sense that $\tilde{h}_D^* < \tilde{h}_D < h_D < h_C$ when the variance of the household size distribution is sufficiently large.

6.3 Two waves of infection

Whilst in Section 2.4 we make comparisons between the disease-induced and vaccine-induced herd immunity levels, in reality it may be the case that, during

the early stages of an epidemic, non-pharmaceutical interventions (such as a lockdown) must be considered a primary option in order to limit disease spread. The present study considers the strength of possible global restrictions and finds that there is an optimal amount of global restrictions, beyond which restrictions can become harmful in terms of how much of the population ultimately become infected.

It seems natural to assume that the stronger global restrictions are, the more well-protected the population will be from an epidemic. However, lockdowns (or similar restrictions) are a finite resource which cannot be permanently maintained. If these restrictions are very strong and are then suddenly lifted, very little of the population will be immunised in a first wave, leaving a risk of a large second wave; in this case the final outcome will, in terms of the proportion of the population infected, be similar to that of not imposing global restrictions for the first epidemic. In this section we consider a first epidemic run with global infection rates reduced by a factor $\alpha \in (0,1)$, followed by a second, unrestricted epidemic among the remaining susceptibles.

The problem of optimal control is investigated numerically by Handel et al. [2007], where it is noted that the optimal choice is a scenario in which the first epidemic ends just as herd immunity is achieved. We prove this for the homogeneously mixing model, where we also make a connection with optimal stopping of a first epidemic. We then turn attention to the multitype households model with proportionate global mixing. Throughout the sequel we assume that all supercritical epidemics take off.

6.3.1 Homogeneously mixing epidemic

Consider the homogeneously mixing epidemic, denoted $\mathscr{E}_\lambda$, as the population size tends to infinity, assuming without loss of generality that $\mathrm{E}(T_I) = 1$. Then $R_0 = \lambda$ for this model and, if global restrictions are applied to a first epidemic, that first epidemic will have $R_0 = \alpha\lambda$, implying that $R_0 > 1$ if and only if $\alpha > \lambda^{-1}$. Letting $\alpha_0 = \lambda^{-1}$, the first wave can only take off provided $\alpha > \alpha_0$. Now suppose that we choose $\alpha = \alpha_*$ such that the population is at criticality at the end of the first epidemic. The threshold parameter R_0 for the second epidemic

satisfies $R_0 = \lambda(1-z_1)$, where z_1 is the proportion of individuals infected in the first epidemic. Using Andersson and Britton [2000], Theorem 4.2, we have that z_1 is the unique solution in $(0,1)$ of

$$1 - z_1 = \exp(-\alpha_* \lambda z_1).$$

In order to have criticality for the second epidemic we require $z_1 = 1 - \lambda^{-1}$, which implies that

$$\alpha_* = \frac{\log(\lambda)}{\lambda - 1}.$$

Consequently, no second epidemic can occur if $\alpha \geq \alpha_*$.

The above arguments demonstrate that there are three possibilities, dependent on the choice of α. If α is too small then the first epidemic will be subcritical, leaving all of the population susceptible to an unrestricted second epidemic. If α is too large then the first epidemic will be sufficient to achieve herd immunity and the second epidemic will fail to take off, but more individuals will be infected than necessary. For intermediate values of α, there will be two supercritical epidemics. Consider an epidemic model in which the population is exposed to $\mathscr{E}_{\alpha\lambda}$, with all remaining susceptibles then exposed to $\mathscr{E}_\lambda$ (recall our assumption that all supercritical epidemics take off). Denote this epidemic model by $\tilde{\mathscr{E}}_{\alpha,\lambda}$ and let $z(\alpha)$ denote the final size of $\tilde{\mathscr{E}}_{\alpha,\lambda}$. In Figure 6.5 we fix λ and investigate $z(\alpha)$ as α varies, observing the behaviour described above. Note that, when $\alpha_0 < \alpha < \alpha_*$, we have $z(\alpha) = z_1(\alpha) + z_2(\alpha)$, say, where $z_1(\alpha)$ is the unique solution in $(0,1)$ of

$$1 - z_1(\alpha) = \exp(-\alpha\lambda z_1(\alpha)). \tag{6.1}$$

Then $z_2(\alpha)$ is given by the unique solution in $(0, 1-z_1(\alpha))$ of

$$z_2(\alpha) = (1 - z_1(\alpha))(1 - \exp\{-\lambda z_2(\alpha)\}). \tag{6.2}$$

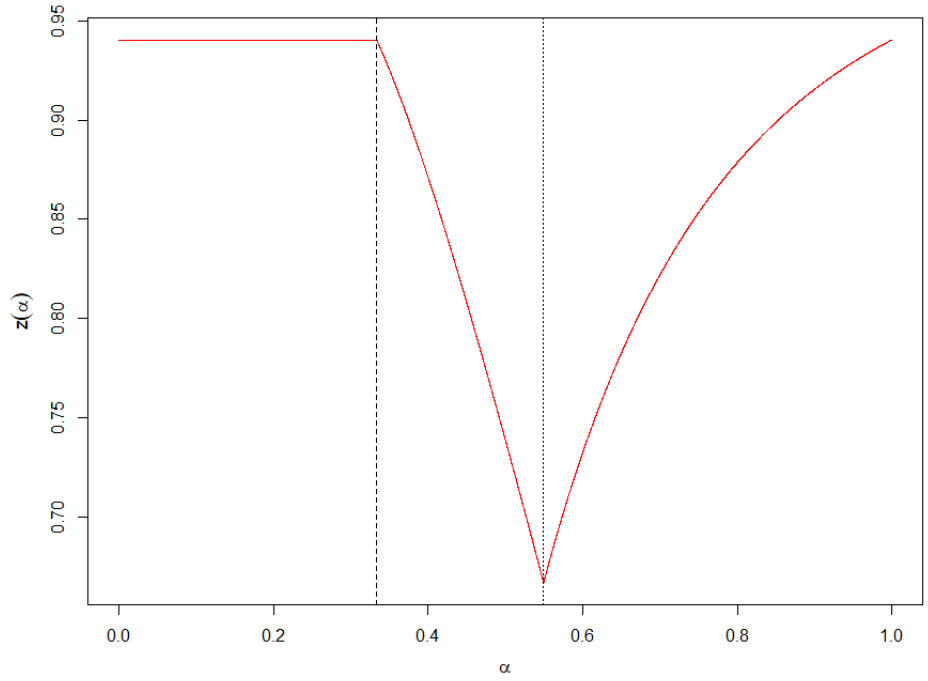

Figure 6.5: Plot of the final size $z(\alpha)$ as a function of $\alpha \in (0,1)$, taking $\lambda = 3$. The dashed (dotted) line corresponds to $\alpha = \alpha_0$ ($\alpha = \alpha_*$).

Figure 6.5 is consistent with the above calculations and is constructed, for $\alpha \in (\alpha_0, \alpha_*)$, by solving (6.1) and (6.2) numerically. Note that, in this example, $z(\alpha)$ is minimised by taking $\alpha = \alpha_*$, i.e. by restricting the first epidemic as much as possible whilst still achieving herd immunity for the second epidemic. The following theorem establishes that this minimisation always occurs at $\alpha = \alpha_*$. We note that $z_1(\alpha)$ is a non-decreasing function of α, so that $\alpha_0 < \alpha_*$.

Theorem 6.3. *Suppose $\lambda > 1$. The final size of $\tilde{\mathscr{E}}_{\alpha,\lambda}$ is minimised by taking $\alpha = \alpha_*$, that is,*

$$\arg\min_{\alpha \in [0,1]} z(\alpha) = \alpha_*.$$

Proof. Let $z_1(\alpha)$ ($z_2(\alpha)$) denote the final size of the first (second) epidemic. We divide $[0,1]$ into three regions, using α_0 and α_*. When $\alpha \geq \alpha_*$ the second epidemic cannot take off, in which case $z_2 = 0$. Then it is clear that, as α increases, $z_1(\alpha)$ will increase and thus $z(\alpha) \geq z(\alpha_*)$ when $\alpha \geq \alpha_*$. When $\alpha \leq \alpha_0$ the first epidemic is subcritical ($z_1(\alpha) = 0$) so this situation is equivalent to taking $\alpha = 1$ (a single unrestricted epidemic). Thus $z(\alpha) \geq z(\alpha_*)$ when $\alpha \leq \alpha_0$.

It remains to treat the case $\alpha_0 < \alpha < \alpha_*$, which implies $z_1(\alpha)$, $z_2(\alpha) > 0$. Recall that $z(\alpha_*) = 1 - \lambda^{-1}$ for this model. Suppose for the purpose of contradiction that $z_1 + z_2 \leq 1 - \lambda^{-1}$. Using (6.2), the final size $z_2(\alpha)$ of the second epidemic satisfies

$$z_2 = (1 - z_1)(1 - \exp(-\lambda z_2)), \tag{6.3}$$

where we have suppressed the dependence of z_1 and z_2 on α for ease of exposition. By assumption we have

$$(1 - z_1)(1 - \exp(-\lambda z_2)) \geq (z_2 + \lambda^{-1})(1 - \exp(-\lambda z_2)).$$

Letting $f(x) = (x + \lambda^{-1})(1 - \exp(-\lambda x)$, we have $f(0) = 0$ and

$$f'(x) = 1 + \lambda x \exp(-\lambda x) > 1, \quad x > 0,$$

so that $f(x) - x > 0$ for $x > 0$. It follows that (6.3) has no solution in $(0, 1)$, which is a contradiction; we find that $z(\alpha) > 1 - \lambda^{-1}$ for $\alpha_0 < \alpha < \alpha_*$ which, in addition to the above arguments, establishes the claim. □

Theorem 6.3 shows that the optimal control to minimise the final size is to have the first epidemic end with the remaining susceptible population at criticality. A heuristic argument for this is discussed in Handel et al. [2007], Section 4.

6.3.2 Optimal stopping in the homogeneously mixing model

We comment briefly on a heuristic connection between $\alpha = \alpha_*$ being the optimal choice to minimise final size and a problem regarding optimal stopping of an epidemic. Consider running the homogeneously mixing epidemic $\mathscr{E}_\lambda$ until a certain proportion (z, say) of the population become infected. At this point, the first epidemic is terminated (e.g. by a total lockdown) and a new homogeneously mixing epidemic begins among the remaining susceptible population. We call this epidemic the restart epidemic, denoting it by $\hat{\mathscr{E}}_{z,\lambda}$. Note that in $\hat{\mathscr{E}}_{z,\lambda}$ both epidemics are unrestricted, but the first epidemic is stopped early. In the context

of a homogeneously mixing epidemic it does not matter that the proportion z have received immunity through the epidemic; this idea of a first epidemic is simply used as a device in order to be able to compare final sizes.

Let $\alpha \in (0,1)$ be chosen such that z is the final size of $\mathscr{E}_{\alpha\lambda}$, and let $\tilde{z}$ be the final size of $\mathscr{E}_{\lambda}$. In this case the models $\tilde{\mathscr{E}}_{\alpha\lambda}$ and $\hat{\mathscr{E}}_{z,\lambda}$ give the same final size; they both run an unrestricted epidemic among the same proportion of remaining susceptibles. Moreover, infected individuals in the homogeneously mixing epidemic are chosen uniformly at random, so the composition of infected individuals is the same in both cases. We can then consider minimising the final size $\hat{z}$ of $\hat{\mathscr{E}}_{z,\lambda}$. Choosing a level z at which the restart occurs is equivalent to prescribing a value of $\alpha \in (0,1)$. It is clear that an immediate restart, given by taking $\alpha \downarrow 0$ and hence $z \downarrow 0$, will lead to the epidemic $\mathscr{E}_{\lambda}$, so that $\hat{z} \uparrow \tilde{z}$ as $z \downarrow 0$. Similarly, taking $\alpha \to 1$ leads to the epidemic $\mathscr{E}_{\lambda}$, so $\hat{z} \uparrow \tilde{z}$ as $z \uparrow \tilde{z}$. A natural choice is to take the restart level z such that the level of infection in the population at the time of the restart is as high as possible, so that the lockdown removes as much present infection as possible. Recalling (3.11), we find that the proportion of infectives at time t, denoted $y(t)$, is maximised when the proportion of susceptibles $x(t) = \lambda^{-1}$. It follows that the proportion of non-susceptibles under this strategy is then $1-\lambda^{-1}(=\tilde{h}_D)$. This provides the following interpretation of the global restriction level; choosing $\alpha = \alpha_*$ corresponds to stopping the epidemic at the optimal time (i.e. at its peak), whilst $\alpha < \alpha_*$ ($\alpha > \alpha_*$) corresponds to stopping the epidemic too early (too late) which leads to more of the population being infected than is necessary to achieve herd immunity.

6.3.3 Optimal stopping in models with heterogeneous mixing

The heuristic argument described in Section 6.3.2 is not immediately available in general for models that are not homogeneously mixing. In particular, in the households model, an epidemic with parameters (λ_G, λ_L) which is stopped early typically will have a different composition of infected individuals to that of an epidemic with parameters $(\alpha\lambda_G, \lambda_L)$ run to its conclusion, even if both of these epidemics infect the same overall fraction of the population. This is due to the fact that the local epidemics play out differently; the model with reduced global

infection rate is more driven by local infection. As a result, the population susceptibility will differ between these epidemics, even if they infect the same proportion of individuals, since the effective household structures are not the same at the end of the respective epidemics. However, in the multitype model with proportionate mixing, a power law relationship holds between the proportions of susceptibles of each type – see, for example, Gart [1968], Section 3. Thus fixing a proportion of the population to be infected in this model also fixes the proportions of each type that are infected. An analogous argument to that in the homogeneously mixing model can then be used to show that choosing $\alpha = \alpha_*$ corresponds to optimal stopping. The above reasoning also gives a justification as to why $\tilde{h}_D = h_D$ in the multitype model with proportionate mixing, as well as why $\tilde{h}_D = h_D$ is not true in general in the households model.

6.3.4 Multitype households model with proportionate global mixing

We consider extending Theorem 6.3 to the multitype households model with proportionate global mixing, recalling the model $\mathscr{E}_{\boldsymbol{m}}(\boldsymbol{\beta}, \boldsymbol{\kappa}, \Lambda^L)$, where we again borrow all of the relevant notation from Section 4.2 in what follows. (We now do not suppress the dependence of $\mathscr{E}$ on the model parameters.) For $\alpha \in (0,1)$, let $\tilde{\mathscr{E}}^{\alpha}_{\boldsymbol{m}}(\boldsymbol{\beta}, \boldsymbol{\kappa}, \Lambda^L)$ denote the epidemic $\mathscr{E}_{\boldsymbol{m}}(\alpha\boldsymbol{\beta}, \boldsymbol{\kappa}, \Lambda^L)$, followed by running $\mathscr{E}_{\boldsymbol{m}}(\boldsymbol{\beta}, \boldsymbol{\kappa}, \Lambda^L)$ among the remaining susceptible population. Let $z(\alpha)$ denote the final size of $\tilde{\mathscr{E}}^{\alpha}_{\boldsymbol{m}}(\boldsymbol{\beta}, \boldsymbol{\kappa}, \Lambda^L)$.

We denote by $q^{(j)}_{\boldsymbol{k}}$ the probability a type-j infective fails to infect anyone in a group of $\boldsymbol{k}$ susceptibles (k_1 type-1 susceptibles, k_2 type-2 susceptibles,$\ldots, k_J$ type-J susceptibles) in a single-household epidemic without outside infection and let $\boldsymbol{q}_{\boldsymbol{k}} = \left(q^{(1)}_{\boldsymbol{k}}, q^{(2)}_{\boldsymbol{k}}, \ldots, q^{(J)}_{\boldsymbol{k}}\right)$ with a view to establishing the following lemma.

Lemma 6.4. *Consider a typical type-l individual, denoted by χ, residing in a category-$\boldsymbol{n}$ household. Suppose that the second epidemic in $\tilde{\mathscr{E}}^{\alpha}_{\boldsymbol{m}}(\boldsymbol{\beta}, \boldsymbol{\kappa}, \Lambda^L)$ is triggered by a type-j initial infective, chosen uniformly at random among this household. The probability $\tilde{P}^{(jl)}_{\boldsymbol{n}}$ that χ is infected in the ensuing local epidemic within the household is given by*

$$\tilde{P}^{(jl)}_{\boldsymbol{n}} = \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \frac{1}{n_l} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G^{(\boldsymbol{e}_l)}_{\boldsymbol{i}}(\boldsymbol{1}|\boldsymbol{U}) \frac{i_j}{n_j} \boldsymbol{\pi}^{\boldsymbol{i}}, \quad j,l, \in \mathscr{J}, \tag{6.4}$$

where π_i ($i \in \mathscr{J}$) is the probability a typical type-i individual avoids global infection in the first epidemic in $\tilde{\mathscr{E}}_{\boldsymbol{m}}^{\alpha}(\beta, \boldsymbol{\kappa}, \Lambda^L)$, $\boldsymbol{\pi} = (\pi_1, \pi_2, \ldots, \pi_J)$ and where $G_{\boldsymbol{i}}^{(\boldsymbol{e}_l)}(\mathbf{1}|\boldsymbol{U})$ is defined in Appendix A, with $\boldsymbol{U}$ given by $\boldsymbol{u}_{\boldsymbol{k}} = \boldsymbol{q}_{\boldsymbol{k}}$ ($\boldsymbol{k} \in \mathbb{Z}_+^J$).

Proof. Assume that $l \in \mathscr{J}$ is given. We proceed using susceptibility sets, denoting the susceptibility set of individual χ by $\boldsymbol{S}$, which is defined to include χ. Thus χ avoids infection from the first epidemic if all members of their susceptibility set avoid global infection; we have

$$P_1 = \mathrm{P}(\chi \text{ avoids infection in the first epidemic}) = \sum_{\boldsymbol{i}=\mathbf{0}}^{\boldsymbol{n}} \mathrm{P}_l(\boldsymbol{S} = \boldsymbol{i})\boldsymbol{\pi}^{\boldsymbol{i}},$$

where the dependence on l is present owing to individual χ being a member of their own susceptibility set. Given they avoid infection in the first epidemic, individual χ will avoid infection in both epidemics if the initial infective in the second epidemic, which is chosen uniformly at random among the type-j individuals in the household, does not lie in χ's susceptibility set. Hence

$$P_2 = \mathrm{P}(\chi \text{ avoids infection in both epidemics}) = \sum_{\boldsymbol{i}=\mathbf{0}}^{\boldsymbol{n}} \mathrm{P}_l(\boldsymbol{S} = \boldsymbol{i})\boldsymbol{\pi}^{\boldsymbol{i}} \left(\frac{n_j - i_j}{n_j}\right),$$

which implies that

$$\begin{aligned} \tilde{P}_{\boldsymbol{n}}^{(jl)} &= P_1 - P_2 \\ &= \sum_{\boldsymbol{i}=\mathbf{0}}^{\boldsymbol{n}} \mathrm{P}_l(\boldsymbol{S} = \boldsymbol{i})\boldsymbol{\pi}^{\boldsymbol{i}} \frac{i_j}{n_j}. \end{aligned} \tag{6.5}$$

It remains to compute $\mathrm{P}_l(\boldsymbol{S} = \boldsymbol{i})$ for $\mathbf{0} \leq \boldsymbol{i} \leq \boldsymbol{n}$. In the notation of Ball [2019], Lemma 4.1, we have

$$\mathrm{P}_l(\boldsymbol{S} = \boldsymbol{i}) = \mathrm{P}_{\boldsymbol{jk}}(\boldsymbol{S}_A = \boldsymbol{i} - \boldsymbol{e}_l),$$

where $A = \{\chi\}$, $\boldsymbol{j} = \boldsymbol{e}_l$ and $\boldsymbol{k} = \boldsymbol{n} - \boldsymbol{e}_l$. Applying that lemma, we find

$$\begin{aligned}
\mathrm{P}_l(\boldsymbol{S} = \boldsymbol{i}) &= (\boldsymbol{n} - \boldsymbol{e}_l)_{[\boldsymbol{i}-\boldsymbol{e}_l]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_l)}(\boldsymbol{1}|\boldsymbol{U}) \\
&= \prod_{m \neq l} \frac{n_m!}{(n_m - i_m)!} \frac{(n_l - 1)!}{(n_l - 1 - (i_l - 1))!} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_l)}(\boldsymbol{1}|\boldsymbol{U}) \\
&= \frac{1}{n_l} \prod_{m \neq l} \frac{n_m!}{(n_m - i_m)!} \frac{n_l!}{(n_l - i_l)!} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_l)}(\boldsymbol{1}|\boldsymbol{U}) \\
&= \frac{1}{n_l} \prod_{m=1}^{J} \frac{n_m!}{(n_m - i_m)!} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_l)}(\boldsymbol{1}|\boldsymbol{U}) \quad (\boldsymbol{0} \leq \boldsymbol{i} \leq \boldsymbol{n}).
\end{aligned}$$

Substituting this expression into (6.5) then establishes (6.4).

□

We are now in a position to derive the threshold parameter for the second epidemic in $\tilde{\mathscr{E}}_{\boldsymbol{m}}^{\alpha}(\boldsymbol{\beta}, \boldsymbol{\kappa}, \Lambda^L)$.

Lemma 6.5. *The threshold parameter for the second epidemic in* $\tilde{\mathscr{E}}_{\boldsymbol{m}}^{\alpha}(\boldsymbol{\beta}, \boldsymbol{\kappa}, \Lambda^L)$*,* $R_{DI}(\boldsymbol{\pi})$*, satisfies*

$$R_{DI}(\boldsymbol{\pi}) = m_H^{-1} \sum_{k=1}^{J} \mu_I^{(k)} \beta_k \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_k)}(\boldsymbol{1}|\boldsymbol{U}) \boldsymbol{\kappa}\boldsymbol{i}\boldsymbol{\pi}^{\boldsymbol{i}},$$

where $\boldsymbol{\kappa}\boldsymbol{i} = \sum_{j=1}^{J} \kappa_j i_j$ *and with* $\boldsymbol{\pi}$ *as in the statement of Lemma 6.4.*

Proof. We construct the threshold parameter among the remaining susceptibles by considering the mean number of contacts that emanate from a typical globally contacted household in the early stages of the second epidemic. Consider a globally contacted individual in the early stages of the second epidemic. Such an individual is type j with probability $\gamma_j \kappa_j$ and, given their type, resides in a household of category $\boldsymbol{n}$ with probability $\alpha_j(\boldsymbol{n})$. The mean number of contacts that emanate from this household ($C_{\boldsymbol{n},j}$, say) is, by symmetry, given by

$$C_{\boldsymbol{n},j} = \sum_{k=1}^{J} n_k \mu_I^{(k)} \beta_k \mathrm{P}(\text{typical type-}k \text{ individual is infected by the ensuing local epidemic}),$$

where the above local epidemic is initiated by a type-j initial infective. Apply-

ing Lemma 6.4 gives

$$C_{\boldsymbol{n},j} = \sum_{k=1}^{J} \mu_I^{(k)} \beta_k \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_k)}(\boldsymbol{1}|\boldsymbol{U}) \frac{i_j}{n_j} \boldsymbol{\pi}^{\boldsymbol{i}}.$$

Conditioning on the type of the initial infective and their household category, as well as noting that $\dfrac{\alpha_j(\boldsymbol{n})\gamma_j}{n_j} = m_H^{-1}\theta_{\boldsymbol{n}}$, we have

$$\begin{aligned}
R_{\mathrm{DI}}(\boldsymbol{\pi}) &= \sum_{j=1}^{J} \gamma_j \kappa_j \sum_{\boldsymbol{n}\in\mathscr{N}} \alpha_j(\boldsymbol{n}) C_{\boldsymbol{n},j} \\
&= \sum_{j=1}^{J} \gamma_j \kappa_j \sum_{\boldsymbol{n}\in\mathscr{N}} \alpha_j(\boldsymbol{n}) \sum_{k=1}^{J} \mu_I^{(k)} \beta_k \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_k)}(\boldsymbol{1}|\boldsymbol{U}) \frac{i_j}{n_j} \boldsymbol{\pi}^{\boldsymbol{i}} \\
&= \sum_{j=1}^{J} \kappa_j \sum_{\boldsymbol{n}\in\mathscr{N}} \frac{\alpha_j(\boldsymbol{n})\gamma_j}{n_j} \sum_{k=1}^{J} \mu_I^{(k)} \beta_k \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_k)}(\boldsymbol{1}|\boldsymbol{U}) i_j \boldsymbol{\pi}^{\boldsymbol{i}} \\
&= m_H^{-1} \sum_{j=1}^{J} \kappa_j \sum_{\boldsymbol{n}\in\mathscr{N}} \theta_{\boldsymbol{n}} \sum_{k=1}^{J} \mu_I^{(k)} \beta_k \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_k)}(\boldsymbol{1}|\boldsymbol{U}) i_j \boldsymbol{\pi}^{\boldsymbol{i}} \\
&= m_H^{-1} \sum_{k=1}^{J} \mu_I^{(k)} \beta_k \sum_{\boldsymbol{n}\in\mathscr{N}} \theta_{\boldsymbol{n}} \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_k)}(\boldsymbol{1}|\boldsymbol{U}) \boldsymbol{\pi}^{\boldsymbol{i}} \sum_{j=1}^{J} \kappa_j i_j \\
&= m_H^{-1} \sum_{k=1}^{J} \mu_I^{(k)} \beta_k \sum_{\boldsymbol{n}\in\mathscr{N}} \theta_{\boldsymbol{n}} \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_k)}(\boldsymbol{1}|\boldsymbol{U}) \boldsymbol{\kappa}\boldsymbol{i}\boldsymbol{\pi}^{\boldsymbol{i}},
\end{aligned}$$

as required. □

We now consider minimising the final size of $\tilde{\mathscr{E}}_{\boldsymbol{m}}^{\alpha}(\boldsymbol{\beta},\boldsymbol{\kappa},\Lambda^L)$, using a generalisation of the argument in the proof of Theorem 6.3.

Theorem 6.6. *The final size of $\tilde{\mathscr{E}}_{\boldsymbol{m}}^{\alpha}(\boldsymbol{\beta},\boldsymbol{\kappa},\Lambda^L)$ is minimised by taking $\alpha = \alpha_*$, that is,*

$$\arg\min_{\alpha\in[0,1]} z(\alpha) = \alpha_*.$$

Proof. We proceed in a similar fashion to the proof of Theorem 6.3, using a proof by contradiction and considering severity. Let $z_1^{(1)}, z_2^{(1)}, \ldots, z_J^{(1)}$ denote the fraction of type-i individuals infected in the first epidemic, where dependence on α is suppressed for ease of exposition. Let

$$\delta_1 = \alpha \sum_{j=1}^{J} \mu_I^{(j)} \beta_j z_j^{(1)} \gamma_j$$

denote the severity of the first epidemic. Define $z_1^{(2)}, z_2^{(2)}, \ldots, z_J^{(2)}$ analogously and let

$$\delta_2 = \sum_{j=1}^{J} \mu_I^{(j)} \beta_j z_j^{(2)} \gamma_j$$

denote the severity of the second epidemic. Recalling (4.12), we have

$$z_i^{(j)} = \sum_{\boldsymbol{n} \in \mathscr{N} : n_i > 0} \frac{\alpha_i(\boldsymbol{n})}{n_i} \mu_{\boldsymbol{n},i} \left(\Lambda^L, \boldsymbol{\pi}^{(j)} \right), \quad i \in \mathscr{J}; j = 1, 2,$$

where $\pi_i^{(j)} = \exp(-\delta_j \kappa_i)$ for $i \in \mathscr{J}$ and $j = 1, 2$. Now, since

$$z_i^{(1)} + z_i^{(2)} = \sum_{\boldsymbol{n} \in \mathscr{N} : n_i > 0} \frac{\alpha_i(\boldsymbol{n})}{n_i} \mu_{\boldsymbol{n},i} \left(\Lambda^L, \exp\{-\boldsymbol{\kappa}(\delta_1 + \delta_2)\} \right)$$

we have

$$\begin{aligned}
\delta_2 &= \sum_{j=1}^{J} \mu_I^{(j)} \beta_j z_j^{(2)} \gamma_j \\
&= \sum_{j=1}^{J} \mu_I^{(j)} \beta_j \gamma_j \sum_{\boldsymbol{n} \in \mathscr{N} : n_j > 0} \frac{\alpha_j(\boldsymbol{n})}{n_j} \left[\mu_{\boldsymbol{n},j} \left(\Lambda^L, \hat{\boldsymbol{\pi}} \right) - \mu_{\boldsymbol{n},j} \left(\Lambda^L, \boldsymbol{\pi}^{(1)} \right) \right] \\
&= \sum_{j=1}^{J} \mu_I^{(j)} \beta_j \sum_{\boldsymbol{n} \in \mathscr{N}} m_H^{-1} \theta_{\boldsymbol{n}} \left[\mu_{\boldsymbol{n},j} \left(\Lambda^L, \hat{\boldsymbol{\pi}} \right) - \mu_{\boldsymbol{n},j} \left(\Lambda^L, \boldsymbol{\pi}^{(1)} \right) \right], \qquad (6.6)
\end{aligned}$$

where $\hat{\boldsymbol{\pi}} = \exp\{-\boldsymbol{\kappa}(\delta_1 + \delta_2)\}$. Taking $\boldsymbol{j} = \boldsymbol{e}_k$ in (4.14), we have

$$\mu_{\boldsymbol{n},k}(\Lambda^L, \boldsymbol{\pi}) = n_k - \mathrm{E}\left[\boldsymbol{S}_{[\boldsymbol{e}_k]} \right] = n_k - \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} \boldsymbol{\pi}^{\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_k)}(\boldsymbol{1}|\boldsymbol{U}), \quad k \in \mathscr{J}, \boldsymbol{n} \in \mathscr{N}.$$

Substitution into (6.6) gives

$$\begin{aligned}
\delta_2 &= m_H^{-1} \sum_{j=1}^{J} \mu_I^{(j)} \beta_j \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_j)}(\boldsymbol{1}|\boldsymbol{U}) \left[\exp\{-\boldsymbol{\kappa}\delta_1\}^{\boldsymbol{i}} - \exp\{-\boldsymbol{\kappa}(\delta_1 + \delta_2)\}^{\boldsymbol{i}} \right] \\
&= m_H^{-1} \sum_{j=1}^{J} \mu_I^{(j)} \beta_j \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_j)}(\boldsymbol{1}|\boldsymbol{U}) \exp\{-\boldsymbol{\kappa}\boldsymbol{i}\delta_1\} \left[1 - \exp\{\boldsymbol{\kappa}\boldsymbol{i}\delta_2\} \right]. \qquad (6.7)
\end{aligned}$$

Let δ_* denote the value of δ such that the second epidemic is at criticality. Suppose for the purpose of contradiction that $\delta_1 + \delta_2 \leq \delta_*$. Under this assumption,

the right-hand side of (6.7) is at least $f(\delta_2)$, where

$$f(\delta_2) = m_H^{-1} \sum_{j=1}^{J} \mu_I^{(j)} \beta_j \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_j)}(\boldsymbol{1}|\boldsymbol{U}) \left[\exp\{-\boldsymbol{\kappa i}(\delta_* - \delta_2)\} - \exp\{-\boldsymbol{\kappa i}\delta_*\}\right].$$

It is clear that $f(0) = 0$. Differentiation then gives

$$f'(\delta_2) = m_H^{-1} \sum_{j=1}^{J} \mu_I^{(j)} \beta_j \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_j)}(\boldsymbol{1}|\boldsymbol{U}) \boldsymbol{\kappa i} \exp\{-\boldsymbol{\kappa i}(\delta_* - \delta_2)\}$$

and

$$f''(\delta_2) = m_H^{-1} \sum_{j=1}^{J} \mu_I^{(j)} \beta_j \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_j)}(\boldsymbol{1}|\boldsymbol{U}) (\boldsymbol{\kappa i})^2 \exp\{-\boldsymbol{\kappa i}(\delta_* - \delta_2)\}.$$

As noted in Lemma 4.6, we have $G_{\boldsymbol{i}}^{(\boldsymbol{e}_k)}(\boldsymbol{1}|\boldsymbol{U}) \geq 0$ for all $\boldsymbol{i} \in \mathscr{N}$ and for all $k \in \mathscr{J}$. It follows that $f''(\delta_2) > 0$. By Lemma 6.5, we have

$$\begin{aligned} f'(0) &= m_H^{-1} \sum_{j=1}^{J} \mu_I^{(j)} \beta_j \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_j)}(\boldsymbol{1}|\boldsymbol{U}) \boldsymbol{\kappa i} \exp\{-\boldsymbol{\kappa i}\delta_*\} \\ &= m_H^{-1} \sum_{j=1}^{J} \mu_I^{(j)} \beta_j \sum_{\boldsymbol{n} \in \mathscr{N}} \theta_{\boldsymbol{n}} \sum_{\boldsymbol{i}=\boldsymbol{0}}^{\boldsymbol{n}} \boldsymbol{n}_{[\boldsymbol{i}]} \boldsymbol{q}_{\boldsymbol{i}}^{\boldsymbol{n}-\boldsymbol{i}} G_{\boldsymbol{i}}^{(\boldsymbol{e}_j)}(\boldsymbol{1}|\boldsymbol{U}) \boldsymbol{\kappa i} \boldsymbol{\pi}_*^{\boldsymbol{i}} \\ &= R_{\mathrm{DI}}(\boldsymbol{\pi}_*) \\ &= 1. \end{aligned}$$

Since $f(\delta_2)$ is convex, with $f(0) = 0$ and $f'(0) = 1$, it follows that there is no solution to (6.7) in $(0, \infty)$, which is a contradiction; we conclude that $\delta_1 + \delta_2 > \delta_*$. Then, in an obvious notation, we have

$$\hat{\pi}_i = \exp\{-\kappa_i(\delta_1 + \delta_2)\} < \exp\{-\kappa_i \delta_*\}, \quad i \in \mathscr{J}.$$

This implies that the total proportion of type-i individuals infected ($z_i(\alpha)$) satis-

fies

$$\begin{aligned} z_i(\alpha) &= \sum_{\boldsymbol{n}\in\mathscr{N}:n_i>0} \frac{\alpha_i(\boldsymbol{n})}{n_i}\mu_{\boldsymbol{n},i}(\Lambda^L,\hat{\boldsymbol{\pi}}) \\ &> \sum_{\boldsymbol{n}\in\mathscr{N}:n_i>0} \frac{\alpha_i(\boldsymbol{n})}{n_i}\mu_{\boldsymbol{n},i}(\Lambda^L,\boldsymbol{\pi}_*) \\ &= z_i(\alpha_*), \quad i\in\mathscr{J}. \end{aligned}$$

It is then clear that the total final size $z(\alpha)$ is minimised by taking $\alpha=\alpha_*$, as required. □

We note two special cases of Theorem 6.6 in the following corollaries.

Corollary 6.7. *Consider running two epidemics in the households model of Section 2.2.1, the first with all global mixing rates multiplied by α and the second unrestricted. The total final size of this pair of epidemics is minimised by taking $\alpha=\alpha_*$.*

Proof. Taking $J=1$ in Theorem 6.6, so that all individuals have the same type, establishes this result for the households model. □

Corollary 6.8. *Consider running two epidemics in the multitype epidemic model with proportionate mixing, the first with all mixing rates multiplied by α and the second unrestricted. The total final size of this pair of epidemics is minimised by taking $\alpha=\alpha_*$.*

Proof. Considering Theorem 6.6 with $\theta_{\boldsymbol{n}}=0$ for $\|n\|>1$, corresponding to considering households of size one only, establishes the result for this model. □

Note that the result for the homogeneously mixing model treated in Theorem 6.3 is also a special case of Theorem 6.6. In Figure 6.6 we demonstrate Corollary 6.7 with a numerical example for the UK household size distribution, noting the similarity to the behaviour observed in Figure 6.5. We also provide the corresponding plots of $z_1(\alpha)$ and $z_2(\alpha)$. Observe that $z(\alpha)$ only has contributions from both $z_1(\alpha)$ and $z_2(\alpha)$ when $\alpha_0<\alpha<\alpha_*$.

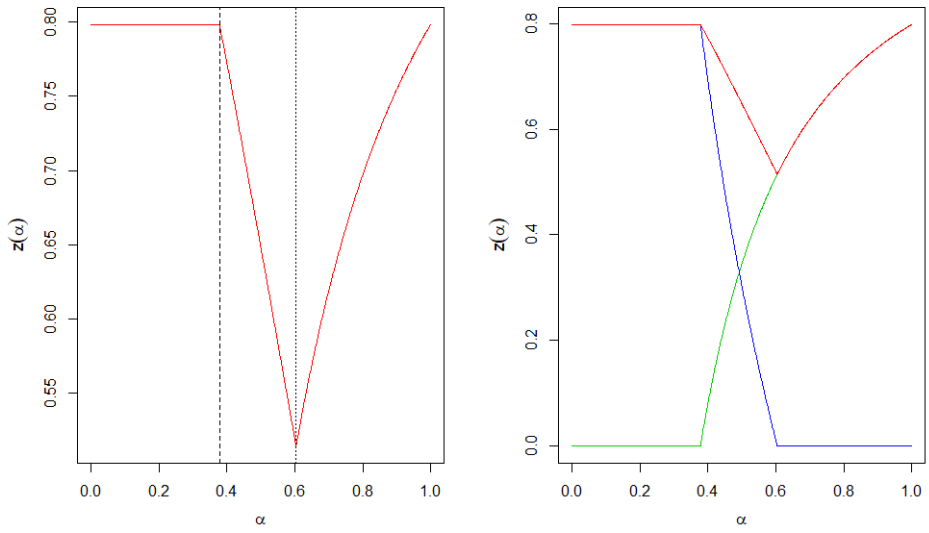

Figure 6.6: Left: plot of the total final size $z(\alpha)$ for two epidemics in the households model, the first with infection parameters $(\alpha\lambda_G, \lambda_L)$, the second with infection parameters (λ_G, λ_L), using the UK household size distribution and taking $(\lambda_G, \lambda_L, \gamma) = (1.25, 0.75, 1)$. The dashed (dotted) line corresponds to $\alpha = \alpha_0$ ($\alpha = \alpha_*$). Right: plot of $z(\alpha)$ (red), $z_1(\alpha)$ (blue), and $z_2(\alpha)$ (green).

6.3.5 Discussion

We have introduced a new approximation for the disease-induced herd immunity level which accounts for the fact that local infection increases when global restrictions are applied. We have also considered the case of two supercritical epidemics and shown that, in the models we consider, this scenario is always worse, in terms of the final size, than a single epidemic which leaves the remaining population at criticality. We have also commented on a connection between the imposition of global restrictions throughout an epidemic to the problem of optimal stopping of an unrestricted epidemic, providing a heuristic connection between $\tilde{h}_D$ and h_D.

There are several possible extensions to the work of this chapter. The assumption that the mean total rate at which an individual makes contacts remains constant may not be true in practice; we may find that even more contacts are made locally during periods of global restrictions than that which keeps the original rate constant. Moreover, the multiplication of local infection rates by a factor β has no effect on individuals in single-member households, who are

unable to mix locally anyway. Future work could incorporate the idea of "bubbles", in which single-member households are more highly globally connected to a single, other, household. We again expect to see the disease-induced herd immunity level being larger (often much larger) than the classical herd immunity level in this case.

In this chapter we have assumed that the local infection rate does not decrease as household size increases. It may be insightful to repeat the calculations of Section 6.2.2 when $\lambda_L = \lambda_L(n)$ depends on the household size, particularly due to the fact that, in our calculation of $\tilde{h}^*_D$, we assume that λ_L increases during global restrictions.

In the case of two supercritical epidemics, we have shown that there is an "optimal" global restriction for the first epidemic in order to minimise the total number infected across the two epidemics. We have shown this for the multitype households model with proportionate global mixing. Similar results hold for minimising other final outcome quantities which are increasing functions of the severity of the epidemic, using Theorem 6.6. It would be interesting to investigate whether the result of Theorem 6.6 holds for a wider class of epidemic models, as well as making the connection between global restrictions and optimal stopping. We have shown that α_* is optimal for the multitype model with proportionate mixing (Corollary 6.8). Numerical investigations suggest that a similar result is true in the multitype model in the absence of proportionate mixing, although a proof has not been forthcoming. It seems unlikely that a similar proof (i.e. by contradiction) will work for a wider class of models; finding a more widely applicable proof for the case of proportionate mixing may also be beneficial. We have also not shown that $z(\alpha)$ is decreasing between $\alpha = \alpha_0$ and $\alpha = \alpha_*$, but we believe it is possible to do so and are currently working on this problem.

We have assumed that restrictions are in place, and remain the same strength, throughout the duration of the first epidemic. In practice global restrictions have varying degrees of severity and, with a view toward cost, governments would be likely to try weaker restrictions first, increasing restrictions if a disease continued to grow. Moreover, as previously discussed, the use of global restrictions are

a finite resource; future research could consider how best to allocate restrictions subject to minimising the cost associated with implementing them.

In the homogeneously mixing model the optimal choice is to stop at the peak (i.e. when the proportion of infectives is at its highest) which coincides with herd immunity being achieved. In the mixing groups model of Ball and Neal [2022] these two points do not coincide; it may be fruitful to investigate the problem of optimal epidemic stopping for this model, as well as other models where the peak does not coincide with herd immunity being reached.

7 Concluding comments

We now provide a general overview of the results which we have established and discuss how these results could be extended. We discuss the underlying assumptions in our models, how they may be removed, and which results we might expect to change upon removing these assumptions. We also indicate possible directions for future work.

We have provided detailed comparison of h_D and h_C. In Chapter 2 we defined h_D (h_D^L when a latent period is present) and introduced an approximation $\tilde{h}_D$ of h_D for the households model, following Britton et al. [2020], which modifies the global infection rate so that the first epidemic terminates with the remaining susceptible population at criticality. We observed that the approximation is typically very good. Moreover, we have $\tilde{h}_D = h_D$ when the local infection rate $\lambda_L \to 0$ or $\lambda_L \to \infty$ and $\tilde{h}_D$, owing to its dependence on final outcome results, is more amenable to analysis, allowing for direct comparison of $\tilde{h}_D$ and h_C in these cases. We showed that, for the households model, we typically have $\tilde{h}_D > h_C$ unless the variability in households is sufficiently large. We also considered numerical comparisons of $\tilde{h}_D$, h_D, h_D^L, and h_C. During these comparisons we assumed an exponential distribution for the infectious period and latent period. We would expect to obtain qualitatively similar results for other, more realistic choices of infectious period distribution. We also provided comparison of herd immunity levels using real-world household size distributions, estimated from data. In our model we have assumed that the local infection rate is independent of the household size. For countries with large mean household size, such as Pakistan, it may be insightful to allow the local infection rate to depend on the household size, i.e. set $\lambda_L = \lambda_L(n)$ where $\lambda_L(n)$ is a decreasing function of n – see Cauchemez et al. [2004]. The results obtained in the highly locally infectious case are invariant to this assumption but the numerical results when $\lambda_L < \infty$ may change.

In Chapter 3 we obtained a Gaussian approximation to the disease-induced herd immunity level, for the multitype model with proportionate mixing and for the households model with common household size. We observed that the

asymptotic variance was small; brief numerical investigations suggest that this still holds when household size is variable. The asymptotic variance of h_D for the multitype model with proportionate mixing was compared to, and agreed well with, corresponding simulations. It is possible to extend this work to a multitype households model and to a model in the absence of proportionate mixing. However, obtaining an explicit expression for the asymptotic variance, as we have done for the multitype model with proportionate mixing, does not seem plausible in general owing to the lack of access to a random time change which will simplify the calculations. In any case we expect that the asymptotic variance of h_D would remain small.

In Chapter 4 we considered the multitype households model with proportionate global mixing. We used the assumption of proportionate mixing to simplify calculations of reproduction numbers and the final size of the epidemic. We also presented a central limit theorem for the final outcome of this model, conditional upon a major outbreak. In order to establish this central limit theorem, we used an embedding construction with index set which is one-dimensional, owing to the proportionate mixing assumption. We applied this central limit theorem to the special cases of a highly locally infectious disease and a standard SIR multitype epidemic, respectively, establishing essentially explicit mean vectors and asymptotic covariance matrices. These results easily extend to the case where the global and local infection rates are allowed to depend on an individual's household category. In deriving the central limit theorem we have assumed a finite maximum household size. A central limit theorem for this model can be derived in the absence of a finite maximum household size, provided stronger assumptions are placed on the moments of the final outcome quantities associated to the epidemic.

In Chapter 5 we studied herd immunity in a model with activity levels and household structure. (This model is a special case of the model analysed in Chapter 4.) We showed that the interaction between these two factors, one which typically causes $\tilde{h}_D$ to increase and the other causing $\tilde{h}_D$ to decrease, is rather complex. A key factor in determining an ordering for $\tilde{h}_D$ and h_C is the clumping of individuals of each type. Broadly speaking, we showed that individuals with

high global mixing rates who mix locally with less active individuals causes $\tilde{h}_D$ to increase. Thus, a situation where highly active individuals reside in households together leads to lower values of $\tilde{h}_D$. We provided analysis in the highly locally infectious case, where $\lambda_L \to \infty$, in which some explicit results were obtained. We also provided heuristic justification for these results in terms of how the epidemic spreads among the population structure in question. In doing so we emphasised the importance of population structure in disease-induced herd immunity. We have also noted how strongly disease-induced herd immunity levels can vary based on the underlying population structure – something which we must bear in mind when attempting to provide accurate estimates of the disease-induced herd immunity level.

The impact of global restrictions on disease-induced herd immunity was investigated in Chapter 6. We introduced a new approximation $\tilde{h}_D^*$ of h_D for the households model, where we accounted for the fact that local infection is typically greater at a time of global restrictions. We showed that $\tilde{h}_D^* > h_D$ when all households are size 2, providing evidence supporting a conjecture that this holds more generally for a common household size n. Returning to the multitype households model with proportionate mixing, we considered a scenario in which two supercritical epidemics occur, and sought to minimise the final size across these two epidemics by choosing the control measures for the first epidemic appropriately. We showed that, with this aim, the optimal choice is to infect precisely a proportion $\tilde{h}_D$ in the first epidemic, thus ending the first epidemic with the remaining susceptible population at criticality. We expect that this result regarding the minimisation of two epidemics, the first with control measures, would generalise to a wider class of epidemic models. We are currently working on such a generalisation by considering an alternative proof for the multitype households model with proportionate global mixing. Future work could assume that control measures are not applied uniformly during the first epidemic; it is not hard to envisage a scenario where control measures gradually increase, particularly when in the early stages of an outbreak they are deemed to be insufficient. Moreover, restrictions could be applied differently to different individuals. For example, individuals who have increased vulnerability upon

becoming infected could be placed under stronger global restrictions.

The models we have considered have, in terms of realism, the defect that an individual cannot become infected again once having recovered. In the case of endemic diseases, subsequent reinfection can occur. Whilst this certainly complicates the analysis, this thesis has shown that it is the state of the population when a new outbreak occurs that is most crucial; if many households have one or two individuals immune, this better prepares the population than a scenario in which households are either fully susceptible or fully immune. Moreover, if very active individuals become susceptible again, the overall population susceptibility can increase. Future work could consider herd immunity for a population in which individuals can become reinfected. It would also be of interest to study herd immunity in other population structures; examples include the overlapping groups model of Ball and Neal [2002] and a model involving time of day effects – see Neal [2016]. As we have demonstrated, the key lies in studying when the relevant threshold parameter among the remaining susceptible population reaches one.

We have not considered how herd immunity may play out in a network setting. Networks allow for a high amount of individual heterogeneity; it would be interesting to investigate further how herd immunity is affected by such a population structure – see Ball et al. [2024]. Moreover, the mixing of households could be assumed to take place on a network, such that each household corresponds to a node in the network. Ideas such as preventive dropping of edges, which mimics social distancing, considered by Ball et al. [2019], may also be of interest in the context of herd immunity.

We have assumed a perfect vaccine throughout this thesis. In practice vaccines are imperfect and it may be of interest to observe the impact of a non-perfect vaccine on the results we have obtained. A detailed analysis of a non-perfect vaccine in the households model is given by Ball and Lyne [2002]. Further, we have assumed that vaccination and herd immunity play out separately, rather than both impacting a population simultaneously. Whilst this appears to be a drawback, it is likely that in the early stages of a disease a vaccine is unavailable. Our work then gives insight into how disease-induced herd immunity

could play out in the absence of a vaccine, provided the population structure can be estimated well. The vaccine-induced herd immunity level provides a good baseline for comparison. In any case, vaccination could be accounted for in the associated ODEs governing a deterministic epidemic model by allowing individuals to transition directly from susceptible to recovered. We could also consider the case of waning immunity, in which individual susceptibility begins to increase again after recovery – see El Khalifi and Britton [2023].

The work of this thesis has shown that population structure can lead to a drastic increase or decrease in the disease-induced herd immunity level when compared to a model with homogeneous mixing of individuals. We have highlighted the importance of having a good model of the underlying population structure in order to properly understand herd immunity.

A Gontcharoff polynomials

We provide some vector notation which is used regularly throughout this thesis, before defining multivariate Gontcharoff polynomials. In this section we let $\boldsymbol{a}, \boldsymbol{b} \in \mathbb{R}^J$. We write

$$\boldsymbol{a}^{\boldsymbol{b}} = \prod_{j=1}^{J} a_j^{b_j} \text{ and } \sum_{\boldsymbol{r}=\boldsymbol{0}}^{\boldsymbol{a}} = \sum_{r_1=0}^{a_1} \dots \sum_{r_J=0}^{a_J}. \tag{A.1}$$

In the second definition of (A.1) we note that the sum is taken over all $\boldsymbol{r} \in \mathbb{Z}_+^J$ such that $\boldsymbol{r} \leq \boldsymbol{a}$, with the convention that $\boldsymbol{r} \leq \boldsymbol{a}$ if and only if $r_j \leq a_j$ for all $j \in \{1,2,\dots,J\}$. We write $\boldsymbol{r} < \boldsymbol{a}$ if $\boldsymbol{r} \leq \boldsymbol{a}$ and $\sum_{j=1}^J r_j < \sum_{j=1}^J a_j$. We also define the vector analogues to the factorial and falling factorial via

$$\boldsymbol{a}! = \prod_{j=1}^{J} a_j! \quad \text{and} \quad \boldsymbol{a}_{[\boldsymbol{b}]} = \prod_{i=1}^{J} a_{i[b_i]},$$

where $a_{[b]} = \dfrac{a!}{(a-b)!}$ denotes the falling factorial.

We now define multivariate Gontcharoff polynomials – see, for example, Picard and Lèfevre [1993], Definition 2.1. Let $\mathbb{N} = \{1,2\dots\}$ denote the set of natural numbers. For each $j \in \mathscr{J}$, let $U^{(j)} = \{u^{(j)}_{i_1,i_2,\dots,i_J} = u^{(j)}_{\boldsymbol{i}}(\boldsymbol{i} \in \mathbb{N}^J)\}$ be a given family of real numbers and define $\boldsymbol{U} = (U^{(1)}, U^{(2)}, \dots, U^{(J)})$. Let $\boldsymbol{k} = (k_1,k_2,\dots,k_J) \in \mathbb{Z}_+^J$ and let $\boldsymbol{x} = (x_1,x_2,\dots,x_J) \in \mathbb{R}^J$. The Gontcharoff polynomial $G_{\boldsymbol{k}}(\boldsymbol{x}|\boldsymbol{U})$, which is of degree k_i in x_i, is then defined by the following recursion: set $G_{\boldsymbol{0}}(\boldsymbol{x}|\boldsymbol{U}) = 1$ and, when $\sum_{i=1}^J k_i \geq 1$,

$$G_{\boldsymbol{k}}(\boldsymbol{x}|\boldsymbol{U}) = \frac{\boldsymbol{x}^{\boldsymbol{k}}}{\boldsymbol{k}!} - \sum_{\boldsymbol{i}<\boldsymbol{k}} \frac{\boldsymbol{u}_{\boldsymbol{i}}^{\boldsymbol{k}-\boldsymbol{i}}}{(\boldsymbol{k}-\boldsymbol{i})!} G_{\boldsymbol{i}}(\boldsymbol{x}|\boldsymbol{U}).$$

Define finally, for $\boldsymbol{i} \in \mathbb{Z}_+^J$, $G_{\boldsymbol{k}}^{(\boldsymbol{i})}(\boldsymbol{x}|\boldsymbol{U})$ as the partial derivative of $G_{\boldsymbol{k}}(\boldsymbol{x}|\boldsymbol{U})$ with derivatives of orders i_j in x_j ($j \in \mathscr{J}$), i.e.

$$G_{\boldsymbol{k}}^{(\boldsymbol{i})}(\boldsymbol{x}|\boldsymbol{U}) = \frac{\partial^{\|\boldsymbol{i}\|}}{\partial x_1^{i_1} \partial x_2^{i_2} \dots \partial x_J^{i_J}} G_{\boldsymbol{k}}(\boldsymbol{x}|\boldsymbol{U}).$$

B Wald's identity

We provide two identities, relating the mean final size and mean severity of epidemics among a group of individuals of size n, which are used regularly in this thesis. Let $\mathscr{E}_n$ denote an epidemic model in which there are n individuals and assume that the infectious period for each individual is independent and identically distributed according to the random variable T_I. Assume, without loss of generality, that there is one initial infective. Let N_* denote the total number of individuals ever infected in $\mathscr{E}_n$ and let T_A denote the sum of the infectious periods of these N_* individuals.

Lemma B.1 (Ball [1986], Corollary 2.2)**.** *We have*

$$\mathrm{E}[T_A] = \mathrm{E}[N_*]\mathrm{E}[T_I].$$

We next consider a multitype epidemic, with individual types belonging to $\{1,2,\ldots,J\}$. Suppose there are n_i type-i individuals and let $\boldsymbol{n} = (n_1, n_2, \ldots, n_J)$. Suppose that the infectious period of each type-i individual is independent and identically distributed according to the random variable $T_I^{(i)}$. Assume, without loss of generality, that there is one initial infective of type 1. Denote this epidemic model by $\mathscr{E}_{\boldsymbol{n}}$. Let $N_*^{(i)}$ be the total number of type-i individuals infected in $\mathscr{E}_{\boldsymbol{n}}$ and let $T_A^{(i)}$ denote the sum of the infectious periods of these $N_*^{(i)}$ individuals.

Lemma B.2 (Ball [1986], Corollary 3.2)**.** *We have*

$$\mathrm{E}\left[T_A^{(i)}\right] = \mathrm{E}\left[N_*^{(i)}\right]\mathrm{E}\left[T_I^{(i)}\right], \qquad i = 1,2,\ldots,J.$$

C Methods for numerical results

All numerical solutions and simulations were obtained using the R programming language (Version 4.4). Further details for particular numerical calculations and simulations are provided below.

C.1 Root-finding

In several instances in this thesis we wish to solve an equation of the form

$$f(x) = 0,$$

to find the root (x_*, say). (Examples include solving (2.3) to find R_0.) In many cases, such as in (2.3), the solution to such equations is unique; the "fzero" function in the package pracma (Version 1.9.9) is then, with a suitable choice of starting interval, used to find the desired root. The relative tolerance of this method is $\varepsilon = 1 \times 10^{-12}$. For further details of this method, see Alefeld et al. [1995].

In some cases we wish to solve an equation which has a unique root in a specific interval. Consider the equation

$$1 - z = \exp(-R_0 z), \tag{C.1}$$

where $R_0 > 1$. Whilst there is a unique solution to (C.1) in the interval $(0,1)$, care must be taken when searching for this root, since $z = 0$ is also a root. Moreover, it can be shown that the positive solution z_* of (C.1) satisfies $z_* \downarrow 0$ as $R_0 \downarrow 1$. However, a Taylor expansion of $\exp(-R_0 z)$ establishes that $z_* > 2(R_0^{-1} - R_0^{-2})$, which provides an adequate search interval. A similar method can be applied to the other root-finding problems that occur in this thesis.

C.2 Ordinary differential equations

The differential equations solved in this thesis can be written in the form

$$\frac{d\boldsymbol{y}}{dt} = f(\boldsymbol{y}, t), \quad \boldsymbol{y}(0) = \boldsymbol{y}_0. \tag{C.2}$$

Such equations are solved using an implementation of the Runge-Kutta algorithm from the package deSolve. The relative tolerance of this method is $\varepsilon = 1 \times 10^{-6}$. For more information on this method, consult Soetaert et al. [2010]. In some instances we wish to solve equations such as (C.2) until some "event"

time at which a given function of $\boldsymbol{y}$ takes a desired value. For example, we integrate (2.11) until R_* among the remaining susceptible population reaches one. In practice, we append a "rootfun" argument to the "ode" function used to integrate such equations, which terminates when a stopping condition is satisfied. Such stopping conditions are, in this thesis, always monotone, which ensures the integration has been stopped at the desired time.

C.3 Stochastic simulations

There are several simulations in this thesis which are used to provide realisations of the disease-induced herd immunity level, which is a random variable for a finite population, since it depends on the trajectory of the epidemic. For such simulations we assume that the infectious period $T_I \sim \text{Exp}(\gamma)$, in which case we can simulate a continuous-time Markov chain. As an example, the pseudocode to simulate realisations of $(H_D^{(m)}, T_*^{(m)})$, in the absence of a latent period, conditional on a major outbreak (recall Section 2.3.2) is provided in Algorithm 1. (We assume that the epidemic being simulated is initially supercritical.)

In order to perform simulations of the households model, we require more notation, including a labelling of the possible states a household can be in. First, we let $t(n)$ denote the n^{th} triangular number, so that $t(n) = \frac{1}{2}n(n+1)$ ($n \in \mathbb{N}$). Letting $n_{\max} < \infty$ denote the maximum household size, define $\tilde{\mathscr{H}}^{(n_{\max})}$ (cf. $\mathscr{H}^{(n_{\max})}$ at (2.9)) by

$$\tilde{\mathscr{H}}^{(n_{\max})} = \{(s,i,r) \in \mathbb{Z}_+^3 : 1 \leq s+i+r \leq n_{\max}\}.$$

Suppose $\boldsymbol{u}, \boldsymbol{v} \in \tilde{\mathscr{H}}^{(n_{\max})}$ are distinct. Then we define an ordering $\prec$ on $\tilde{\mathscr{H}}^{(n_{\max})}$ by writing $\boldsymbol{u} \prec \boldsymbol{v} \iff u_k < v_k$, where $k = \min_{i \geq 1}\{u_i \neq v_i\}$. We use $\prec$ to (uniquely) construct a list l of states ordered by $\prec$. (For completeness, the first state is $(1,0,0)$.) Letting N denote the number of possible states we can construct, in an obvious fashion, a bijection $b : \{1,2,\ldots,N\} \to l$ which provides a linear index for each state (s,i,r). In the sequel we write H_i for the number of households in state $b(i)$ ($i = 1,2,\ldots,N$). For ease of exposition, we also write $f(i) = \dfrac{i}{6}\left(i^2 - 6i + 11\right)$. Suppose the state $\boldsymbol{u} \in \tilde{\mathscr{H}}^{(n_{\max})}$ has linear index i. We

define $g : \{1,2,\ldots,N\} \to \{1,2,\ldots,N\}$ by

$$\begin{aligned} g(i) &= i + \min\{v \in \mathbb{N} : t(v) \geq i\} \\ &= i + \left\lceil -\frac{1}{2} + \sqrt{\frac{1}{4} + 2i} \right\rceil, \end{aligned}$$

with the property that an infection in a household with linear index i gives rise to a household with linear index $g(i)$. If a recovery occurs in a household with linear index i, that household becomes a household with linear index $i+1$. Finally, let X_i (Y_i) denote the number of susceptible (infective) individuals in a household with linear index i.

Algorithm 1 : Compute $H_D^{(m)}$ assuming no latent period

procedure HOUSEHOLDS SIMULATION($n_{\max}, m_1, m_2, \ldots, m_{n_{\max}}, \lambda_G, \lambda_L, \gamma$)
  Initialise: $N = f(n_{\max})$, $R_{\text{susc}} = R_*$, $\boldsymbol{H} = \mathbf{0}_N$, $\boldsymbol{x} = \mathbf{0}_{n_{\max} \times (n_{\max}+1)}$ and $M^{\text{Rates}} = \mathbf{0}_{N \times 2}$
  $m \leftarrow \sum_{i=1}^{n_{\max}} m_i$ and $\bar{n} \leftarrow \sum_{j=1}^{n_{\max}} j\alpha_j$ ▷ Mean household size
  for $i = 1$ to $n_{\max} - 1$ **do** $H_{1+f(i-1)} \leftarrow m_i$
  end for
  $H_{1+f(n_{\max}-1)} \leftarrow m_{n_{\max}} - 1$
  $H_{2+f(n_{\max}-1)} \leftarrow 1$ ▷ One initial infective in a household of size $n_{\max}$
  while $R_{\text{susc}} > 1$ **do**
    $I \leftarrow \frac{1}{\bar{n}} \sum_{i=1}^{N} H_i Y_i$
    if $I = 0$ **then**
      return $H_D^{(m)} = 0$ ▷ Infectives are all removed before herd immunity is achieved
    end if
    for $i = 1$ to N **do**
      $M_{i,1}^{\text{rates}} \leftarrow \lambda_G I H_i X_i + \lambda_L X_i Y_i$
      $M_{i,2}^{\text{rates}} \leftarrow \gamma H_i Y_i$
    end for
    Normalise M^{rates}
    Sample $(i, j) \in \{1, 2, \ldots, N\} \times \{1, 2\}$ with probability $M_{i,j}^{\text{rates}}$
    if $j = 1$ **then**
      $H_i \leftarrow H_i - 1$ ▷ Infection occurs
      $H_{g(i)} \leftarrow H_{g(i)} + 1$
    else if $j = 2$ **then**
      $H_i \leftarrow H_i - 1$ ▷ Recovery occurs
      $H_{i+1} \leftarrow H_{i+1} + 1$
    end if
    $\boldsymbol{z} \leftarrow m^{-1} \boldsymbol{H}$
    for $n = 1$ to $n_{\max}$ **do**
      for $v = 1$ to $n + 1$ **do**
        $x_{n,v} \leftarrow \sum_{k=1+f(n-1)+t(v-1)}^{f(n-1)+t(v)} z_k$
      end for
      Normalise $(x_{n,1}, x_{n,2}, \ldots, x_{n,n_{\max}})$
    end for
    $R_{\text{susc}} \leftarrow \frac{\lambda_G}{\gamma} \sum_{n=1}^{n_{\max}} \tilde{\alpha}_n \sum_{v=1}^{n} \left(1 - \frac{v}{n}\right) x_{n,v} \mu_{n-v}(\lambda_L)$
  end while
  return $H_D^{(m)} = 1 - \frac{1}{\bar{n}} \sum_{i=1}^{N} H_i X_i$
end procedure

Algorithm 1 can then be used to generate the desired number (n_{sim}, say)

of samples $\left(H_D^{(m,k)}\right)_{k=1}^{n_{\text{sim}}}$ from which Monte Carlo estimates of the mean and variance can be computed – see Table 2.1. To condition on a major outbreak we simply reject samples in which $H_D^{(m,k)} = 0$. Algorithm 1 can also be extended, in an obvious fashion, to account for the presence of a latent period $T_E \sim \text{Exp}(\delta)$. The Monte Carlo simulations provided in Table 3.1 and Table 3.2 can be generated using similar methods to that of Algorithm 1.

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