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Stochastic epidemic models for emerging diseases incorporating household structure and contact tracing

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Abstract

In this thesis, three stochastic epidemic models for intervention for emerging diseases are considered. The models are variants of real-time, responsive intervention, based upon observing diagnosed cases and targeting intervention towards individuals they have infected or are likely to have infected, be they housemates or named contacts. These models are:

(i) a local tracing model for a disease spreading amongst a community of households, wherein intervention (vaccination and/or isolation) is directed towards housemates of diagnosed individuals,

(ii) a contact tracing model for a disease spreading amongst a homogeneously-mixing population, with isolation of traced contacts of a diagnosed individual,

(iii) a local tracing and contact tracing model for a disease spreading amongst a community of households, with intervention directed towards housemates of both diagnosed and traced individuals.

These are quantified by deriving threshold parameters that determine whether the disease will infect a few individuals or a sizeable proportion of the population, as well as probabilities for such events occurring.

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CHAPTER 1

Introduction

1.1 Overview

Emerging (i.e. in the early stages of an outbreak) infectious diseases are a major concern to public health (e.g. SARS, Swine influenza) or agriculture (e.g. foot-and-mouth disease), so it is of great importance to evaluate possible outcomes and potential methods for their control. Often there might not be a large pool of historical outbreaks to use to directly make predictions, and carrying out experiments to create such data is obviously unethical and contrary to the reasons behind wanting to anticipate the outcome. Hence, mathematical modelling is a useful tool for quantifying the spread of a potential epidemic, and examine the effectiveness of control strategies.

In this thesis, the focus is on stochastic models, but epidemics may also be modelled deterministically. The deterministic modelling of epidemics assumes that given the initial conditions, the spread of the infection is determined entirely, and so, for instance, the epidemic will infect a given number of individuals with certainty. Modelling stochastically, we rather assume that, given the initial conditions, there is a probability of a given individual being infected. This stochastic variability is inherent in real life, and

the effect of this stochastic variability is particularly important in the initial stages of a real-life outbreak. Further, the final outcome of the epidemic is random, and hence it may be possible to calculate the probability that there will be a minor outbreak infecting only a few individuals, or that there will be a major outbreak infecting a reasonably deterministic proportion of individuals.

The main attention of this thesis is modelling responsive, targeted intervention, specifically of two forms: (i) targeting intervention towards the housemates of a diagnosed individual, and (ii) targeting intervention towards individuals a diagnosed individual has been in contact with, by asking the diagnosed individual to name these contacts. The intention of both of these forms is to direct intervention towards individuals who are already infected or are more likely to be in the near future, and reduce their chances of infection or the number of potentially-infectious contacts they make. The thesis looks at three models: (i) with household-level intervention, (ii) with contact tracing in a homogeneously-mixing population, and (iii) with household-level intervention, and tracing of between-household contacts.

The remainder of this chapter is structured follows. Section 1.2 describes the so-called standard SIR epidemic model, and some related models. Section 1.3 describes advances in modelling diseases in household-based populations, and gives some examples of intervention models exploiting such a structuring. Section 1.4 looks at some mathematical models for contact tracing, while Section 1.5 outlines the structure of the remainder of the thesis, and motivates the models in this thesis in the context of other models.

1.2 The Standard SIR Epidemic

This section describes a particular continuous-time epidemic model, the *standard SIR epidemic model*, and discusses some related models.

In the standard SIR epidemic model, at any given time each individual in the (fixed-size) population is in one of three states: Susceptible, Infective or Removed. Initially there are m infectives and n susceptibles. If a susceptible individual makes contact with an infective individual in a manner described below, then the susceptible individual becomes infective. Each infective individual remains infectious for a length of time, referred to as their *infectious period*, which are independent and identically distributed according to a random variable T_I, with an arbitrary but specified distribution. At the end of their infectious period, the infective individual is removed and plays no further part in the spread of the epidemic. Throughout their infectious period a given infective makes contacts with a given individual at the points of a Poisson process with rate $\frac{\lambda}{n}$. All Poisson processes are assumed independent of each other and the infectious periods.

The case where $T_I \sim \text{Exp}(\gamma)$, i.e. exponentially distributed with mean γ^{-1}, is referred to as the *general stochastic epidemic*, originated by McKendrick [37] and Bartlett [11].

The deterministic analogy of the general stochastic epidemic is the *general deterministic epidemic* of Kermack and McKendrick [33], in which $S(t)$, $I(t)$ and $R(t)$ (respectively, the number of susceptible, infectious and removed individuals at time t) are governed by the following equations:

$$S'(t) = -\lambda S(t) I(t),$$

$$I'(t) = \lambda S(t) I(t) - \gamma I(t),$$

$$R'(t) = \gamma I(t).$$

Kermack and McKendrick [33] also derived a threshold result for this model: the number of infectives is decreasing unless $S(0) > \frac{\gamma}{\lambda}$ (and $I(0) > 0$). It is a threshold result in the sense that different initial conditions ($S(0)$, $I(0)$) and parameter values (λ, γ) can result in different behaviour, as determined by these inequalities.

There are other SIR epidemic models, such as the discrete-time model of Reed and Frost (see, for example, Section 1.2 of Andersson and Britton [2]). The Reed-Frost model is a so-called chain-binomial model of the SIR epidemic, in which the number of susceptibles infected in the next discrete-time step has a binomial distribution with infection probability dependent on the number of infectives, who are infectious for one time step.

Modifications of the SIR epidemic include: the SI model, in which infected individuals never become removed; the SIRS model, in which removed individuals can lose their immunity and become susceptible again; and the SEIR model, in which infected individuals experience an exposed or latent period before becoming infectious.

1.3 Household Epidemics

This section gives a brief history and some examples of modelling epidemics in populations with individuals partitioned into households, in which they have a higher rate of mixing. This relates to the work in Chapters 2 and 4.

Many of the historical epidemic models have assumed that the population is composed of homogeneous individuals (i.e. the spread of disease is not affected by personal factors such as age or sex) who mix uniformly. These assumptions do not hold in real life

- factors such as age could affect a person's susceptibility, while individuals are more likely to come into contact with housemates, workmates or schoolmates than individuals picked at random from the population-at-large. As well as describing the spread of epidemics more accurately, departures from homogeneity in epidemic modelling are important as they offer frameworks in which intervention methods exploiting the heterogeneities can be considered (e.g. closing schools).

An early investigation of a heterogeneous model was by Rushton and Mautner [41], who studied a deterministic epidemic without removal of infectives spreading amongst a population divided into groups, with a higher infection rate within-groups than between. This model was extended to incorporate removals and considered in a deterministic and stochastic framework by Watson [45], though in the latter case approximations had to be made for tractability. Bartoszynski [12] modelled an epidemic as a discrete-time branching process of household ('family') subepidemics (these subepidemics are described in fairly general terms), with infectives having a Poisson-distributed number of contacts outside their households. This model is not strictly an epidemic model, but it does correspond to a limiting process used by, for example, Ball et al. [9] and in Chapters 2 and 4 of this thesis.

Important advances in household models were made concurrently by Becker and Dietz [13], Becker and Hall [14] and Ball et al. [9], all of which also considered interventions directed at household-level. Becker and Dietz [13] examined a highly infectious disease spreading amongst households (i.e. an infection in a household renders all susceptibles in that household infected) and consider strategies for vaccinating pre-outbreak. They suggest that for households of equal sizes it is better to randomly vaccinate individuals than households, but for households of varying sizes it is better to vaccinate larger

households. Becker and Hall [14] extended this to a population made up of different types of individuals. Ball et al. [9] considered a model with two levels of mixing (local and global, i.e. within-household and between-household respectively) and defined R_*, a household threshold parameter (in that its value determines whether the epidemic is certain to infect only a few households or a reasonably deterministic large proportion of households) which is analogous to the reproduction number R_0 (the expected number of secondary cases infected by a typical infective during their infectious period in an otherwise susceptible population). They also examined circumstances under which an *equalizing* vaccination strategy (one which leaves households with equal numbers of susceptibles) is optimal. Considering pre-outbreak optimal vaccination strategies has been treated as a linear programming problem by, for example, Becker and Starczak [16, 17] and Ball and Lyne [6, 7].

These pre-outbreak intervention models have had focus in the literature, particularly because they are easier to study than models in which the intervention is applied after the start of the epidemic. With pre-outbreak intervention, the effect is usually to alter the initial state of the epidemic and thereafter the epidemic will spread in a fairly standard manner. However, recent outbreaks such as that of SARS in 2002 and swine influenza in 2009 have shown that pre-outbreak intervention is not always possible, and that real-time, responsive intervention methods are needed and important to study too.

Motivated by the SARS outbreak, Becker et al. [18] examined various intervention policies for an epidemic spreading amongst a community of households, with individuals also separated into two types, *school attendees* and *others*, to account for increased mixing between school attendees during school hours. It is assumed that the lengths of latent and infectious periods are fixed, and that individuals are diagnosed at some

fixed time after infection. Intervention methods considered include taking steps to avoid exposure (e.g. wearing masks), isolating each case at diagnosis, closing schools, quarantining affected households and contact tracing (the latter is described further in Section 1.4). They found that quarantining affected households and contact tracing can be particularly effective in reducing the spread of an epidemic.

Ball et al. [10] considered a household epidemic model with a different dynamic intervention policy. Rather than vaccinate selected individuals before an outbreak, intervention is targeted towards individuals who are likely to become infectious by vaccinating housemates of removed individuals, or isolating their households. The vaccine is assumed to be *perfect*, in that it renders full immunity in all susceptibles (but has no effect on individuals who have already been infected). They focused attention on the exponential infectious period case, as this makes the household subepidemic process Markovian and makes the model more analytically tractable. Under these assumptions, it is seen that threshold behaviour and the probability of a large epidemic are independent of the latent period distribution. The effect of latent period length is studied for an infectious period distribution with increasing hazard rate (i.e. an individual who has been infectious longer is more likely to be removed sooner), and it is seen that longer latent periods reduce the spread. However, for an infectious period with decreasing hazard rate, the opposite is concluded.

The effect of the household-level interventions of Becker et al. [18] and Ball et al. [10] is to direct the intervention towards individuals who are more likely to be infected or already are infected, which can be particularly effective, compared with randomly-directed intervention, when within-household infection rates are high and latent periods are long. Other methods to direct intervention towards individuals with a higher

risk of infection include contact tracing (which is discussed in the next Section) and ring vaccination. Ring vaccination, which has been modelled by, for example, Müller et al. [39], involves vaccinating all individuals in a certain physical neighbourhood of a diagnosed individual, the bonus of which is that vaccinated individuals can form a barrier between infected and susceptible individuals.

1.4 Contact tracing

This section describes some of the models for contact tracing.

A real-time, responsive form of intervention that has received attention in the literature is contact tracing, in which, usually after diagnosis, individuals name a proportion of the other individuals that they have been in contact with, and these contacts are then traced and treated in some manner. The idea, of course, is to direct treatment towards individuals who have already been infected, even though they have not already been diagnosed. This relates to the work in Chapters 3 and 4.

Among other types of intervention (discussed in Section 1.3), Becker et al. [18] considered the following contact tracing model for an SEIR epidemic (with fixed-length latent and infectious periods) spreading amongst a community of households, motivated by SARS. When an individual is diagnosed (which is a fixed length of time after infection), their housemates are isolated, as are a fixed fraction of their infections outside the household (whose housemates in turn are not isolated until the traced individual is diagnosed). Note that the values of the time to diagnosis and latent and infectious periods, determine whether individuals are diagnosed during their latent period or infectious period, or after the end of their infectious period. They made the simplifying assumption that the traced contacts occur at the beginning of the infector's infectious

period, underestimating the reduction in the reproduction number as a result. They found that using a contact tracing strategy can be very successful in reducing transmission.

Pike [40] also considered a contact tracing model for a household-based population. At the first removal in a household, all remaining members of that household are vaccinated and isolated. Additionally, on removal, an individual names each of their contacts outside the household independently with fixed probability, and a named contact experiences a service time (distributed according to an exponential random variable Q), after which the named contact and their household are vaccinated. Infected individuals experience two exponentially-distributed latent periods, and all individuals who are susceptible or in their first latent period become immune when vaccinated (with no effect on other individuals). For analytical tractability, it was assumed that the infectious period is distributed according to an exponential random variable T_I, but then the model had to be approximated by assuming that contacts are named when they are infected (rather than when their infector is removed) and that the service times are distributed according to $Q + T_I$. Simulations suggest that this approximation is reasonable.

Klinkenberg et al. [34] considered a contact tracing model for an epidemic spreading amongst a homogeneously-mixing population. Infected individuals undergo fixed-length latent and infectious periods, while there is a detection time (from infection), which has a Gamma distribution. As in Becker et al. [18], an infected individual may be detected before, while or after they are infectious. In their analysis Klinkenberg et al. directed attention to the cases of infectious periods that are long (i.e. infinite) or short (i.e. all transmission occurs instantaneously upon becoming infective), and de-

tection times that are exponentially distributed or fixed (a special case and limiting case, respectively, of the Gamma distribution). Upon detection, an individual is isolated and names each of their contacts with fixed probability, and after a (constant) delay, the named contacts are isolated too. They assumed that tracing can be both 'forwards' (from infector to infectee) and 'backwards' (from infectee to infector). They also considered that tracing can be 'single-step' (in which traced individuals can name their own contacts only when they have been detected) and 'iterative' (in which traced individuals can name their contacts as soon as they have been traced). In the single-step case, they looked at the next-generation matrix (in which the element k_{ij} is the expected number of type-j individuals infected by a typical type-i infective, where a type-j individual is defined as having exactly j traceable ancestors in the transmission tree). The matrix is of infinite size but for calculation purposes is truncated. In the iterative tracing case they largely had to use simulations except in special cases. They concluded that generally single-step and iterative tracing are almost equally effective, except when the former has little effect at all.

Müller et al. [38] studied an SIRS epidemic (i.e. it is assumed that removed individuals can lose immunity and become susceptible again) with homogeneous mixing. The infectious periods and time until loss of immunity are exponentially distributed. The population is screened, and if an infected individual is detected they are treated and become immune. Further, the individual will name each of their contacts with fixed probability, and the named individual is traced and treated immediately (i.e. there is no delay). As in Klinkenberg et al. [34], they assumed that tracing can be forwards, backwards or both (full tracing) and that the tracing is iterative. They define a generation-based reproduction number, and study its asymptotic behaviour. They found that one may only need to trace a few steps from the detected individual to reduce spread, and

having longer chains of contacts may not be significantly more effective.

Shaban et al. [42] modelled contact tracing by using a network model for the social structure. The social network is represented by a simple random graph with a specified degree distribution. Modelling an SIR epidemic (i.e. no latent period) on the graph, after a delay time beginning at infection, an infected individual is detected and each friend (i.e. connected individual) of the detected individual is located with a fixed probability and vaccinated, with vaccination rendering susceptible individuals immune. The delay time is assumed to incorporate the time to detect symptoms and the delay in locating friends. A second model is considered in which the epidemic model is adapted to assume that there is an upper bound to the number of infections from a given infective and all friends of a detected individual are located. This model is seen to be more effective in reducing spread as it reduces the effect of people with large numbers of friends ('superspreaders'). This model differs from others in that tracing is directed towards individuals who are more likely to be infected, rather than have already been infected, and as such is a bit closer to the household intervention models discussed in Section 1.3.

Contact tracing has also been studied using simulation and deterministic models. For a bioterrorist smallpox attack, Kaplan et al. [30] compared a traced vaccination scheme against a mass vaccination scheme in a homogeneously-mixing population, with infected individuals undergoing a vaccine-sensitive latent period and then a vaccine-insensitive latent period prior to becoming infectious. In the traced vaccination scheme, a certain number of individuals are named by a symptomatic individual with a fixed fraction of true infectious contacts named, and enter a queue with a fixed number of servers (i.e. vaccinators) serving at a fixed rate. In the mass vaccination scheme, every-

one is placed in the queue immediately. These schemes are only initiated once a certain amount of infected people have exhibited symptoms. A third scheme is considered wherein traced vaccination is begun but mass vaccination is switched to after a fixed period of time. They concluded that mass vaccination is the best scheme (under certain conditions). However, the model assumed that there is a specific window of time during an individual's infection cycle (i.e. their vaccine-sensitive latent period), before vaccination of the individual is a waste of server time. This would not be so much the case if contact tracing were able to prevent infectious individuals from making further contacts (e.g. by isolating them) or if the tracing of an individual results in individuals likely to have come in contact with them (e.g. their housemates) being vaccinated too. Further if an epidemic is above threshold, a deterministic model assumes that there will be a major outbreak, but a stochastic model allows there to be a chance that only relatively few individuals are infected, in which case a mass vaccination could result in a large waste of vaccine resources compared with traced vaccination (since under mass vaccination a large number of individuals would be vaccinated whereas with traced vaccination this would only be a few).

Further deterministic studies of contact tracing include Armbruster and Brandeau [3], Eames [20], Eames and Keeling [21], Hyman et al. [29] and Tsimring and Huerta [44]. Armbruster and Brandeau [3] attached a cost to screening and contact tracing schemes, and suggested that contact tracing is cost-effective when disease prevalence is low, i.e. in the early stages of the epidemic. In the context of HIV, Hyman et al. [29] modelled contact tracing whereby traced contacts of individuals found through screening are counselled, which may or may not change their subsequent behaviour. They found that contact tracing is most effective when the infectives are divided into groups according to their infectiousness and infection is spread by a small group of highly in-

fectious individuals (so-called 'superspreaders'), rather than if infectives go through several stages of infection, with varying infectiousness. Eames [20], Eames and Keeling [21] and Tsimring and Huerta [44] considered contact tracing on a specified network-structure (similarly to Shaban et al. [42]), the former two via a pairwise approximation and the latter using a mean-field approach.

The simulation model for bioterrorist smallpox of Eichner [22] assumes that an individual will have 'close' and 'casual' contacts (analogous to the two levels of mixing of Ball et al. [9], but Eichner [22] assumed that the number of potential close contacts is not depleted by actual contacts) in a ratio of $3:1$. Detected individuals (there is a detection time which decreases as the epidemic spreads) are isolated and name all their close contacts and some of their casual contacts, who are then immediately vaccinated and put under surveillance. If they become symptomatic, they are then also isolated. It is seen that contact tracing is good at controlling the epidemic, with the vaccination of close contacts being a contributing factor to this.

1.5 Structure of thesis

Here we briefly outline the remainder of the thesis.

Chapter 2 concerns a model for an epidemic spreading amongst a population partitioned into households. Upon the first diagnosis in the household the remaining household members are vaccinated, with different models for the vaccine action considered, including whether or not the vaccine affects an individual during their latent period. A household may also be isolated after a diagnosis, with some probability. There is no contact tracing, except in this local, within-household sense. This sort of invention was studied by Ball et al. [10], but they focused on exponential infectious periods, and

assumed that only susceptible individuals were vaccine-sensitive, and that the vaccine is perfect in that it renders susceptibles immune with probability 1. In Chapter 2 different infectious and latent period distributions are considered, it is assumed that latent individuals may also be vaccine-sensitive and models for a non-perfect vaccine (such as random vaccine response or a vaccine that only *reduces* susceptibility and infectivity) is incorporated. The effects of these different assumptions are examined.

The model analysed in Chapter 3 is for an epidemic spreading amongst a homogeneously-mixing population (no household structure) with a contact tracing scheme (recall that other such contact tracing models include Müller et al. [38] and Klinkenberg et al. [34]). It is assumed that a diagnosed individual may name each of their infectious contacts independently with fixed probability, and then after some tracing delay the named contacts are isolated. Unlike the tracing delay in the model of Klinkenberg et al. [34] (which is of fixed length), in Chapter 3 individuals infected by the same infective are assumed to experience independent delays. However, the difference between results from independent and mutual delays is examined. There are two models for the contact tracing: one in which traced individuals are not allowed to name their own contacts, and one in which they are. The latter is like the 'multi-step' or 'iterative' tracing of Müller et al. [38] and Klinkenberg et al. [34], but the former differs from their 'single-step' tracing models.

In Chapter 4 a household-based epidemic is again modelled, this time incorporating a contact tracing scheme (recall that Becker et al. [18] and Pike [40] also modelled contact tracing for household-based populations). Upon the first diagnosis in the household the remaining household members are vaccinated. It is also assumed that, with a fixed probability, the household is isolated upon the first diagnosis within that household.

This differs from the models of Becker et al. [18] and Pike [40], in which isolation upon the first diagnosis in a household occurs for *all* households (the latter did assume that there is a vaccination at the first removal, but the isolation means this vaccination has no effect on the spread of the epidemic). Additionally, upon diagnosis an individual names each of their infectious contacts outside the household with a fixed probability, and after a tracing delay, these named individuals and their households are vaccinated (unlike in the contact tracing model of Becker et al. [18], in which the traced individual (and *only* the traced individual) is isolated). Infected individuals undergo two latent periods, and it is assumed that *all* individuals vaccinated while susceptible or during their first latent period become immune. Both Becker et al. [18] and Pike [40] made simplifying approximations regarding the time between a named individual's infection and their being named. The results in Chapter 4 are more exact, and different distributions for the latent periods and tracing delays are considered.

Chapter 5 gives some general conclusions and extensions.

CHAPTER 2

Local tracing

2.1 Introduction

Many stochastic epidemic models assume homogeneously mixing populations. However in reality individuals mix heterogeneously, as a result of population structures such as households, schools and workplaces. Further, there are outbreak control measures associated with these structures, such as vaccinating a whole school when a case is detected in that school, that cannot be considered in a homogeneous mixing framework. We focus on two-level mixing structures (see Ball et al. [9]) as a practically important departure from homogeneously mixing models, so that we can obtain analytical insights into the quantitative and qualitative behaviour of the models.

Ball et al. [10] considered an SEIR (susceptible $\rightarrow$ exposed $\rightarrow$ infective $\rightarrow$ removed) epidemic spreading amongst a population partitioned into households, with responsive vaccination and isolation policies. This model may be applicable to the outbreak of a disease for which a vaccine is available (for example, the spread of smallpox after a bioterrorist attack, see Halloran et al. [24] and Kaplan et al. [30]). However, the model of Ball et al. [10] has several limitations. First, only susceptible individuals are

assumed vaccine-sensitive. Second, a vaccine is assumed to render a susceptible immune with probability 1, i.e. it is a perfect vaccine. Third, the analysis focuses mainly on exponential infectious periods. We aim to address these limitations as follows: (i) we consider that latent individuals, as well as susceptibles, may be vaccine-sensitive; (ii) instead of a perfect vaccine response, we consider two specific non-perfect vaccine response models (all-or-nothing and non-random); and (iii) we consider both constant and exponential infectious and latent periods.

Under the various assumptions discussed above, we obtain important threshold parameters which give conditions under which an epidemic can become established and which can be used to determine whether or not a given intervention scheme necessarily prevents a large outbreak. We also derive methods for calculating the probability of a global epidemic (i.e. one that becomes established) under different possible intervention strategies. The theory is illustrated by a numerical study, using parameters previously estimated from data on an outbreak of *variola minor*, a virus which causes a mild form of smallpox.

The intervention model involves taking action upon the appearance of diagnosed cases in a household, by vaccinating and/or isolating the remaining members of that household, i.e. the intervention is directed towards the housemates of a diagnosed individual. Thus, we can consider this intervention to be a form of tracing on a *local* (within-household) level. In this chapter, there is no contact tracing of the form wherein diagnosed individuals name some of their infectious contacts outside the household, to whom intervention is then directed.

The chapter is structured as follows. In Section 2.2, the epidemic and vaccine action models are introduced. The concept of infectious intensity is defined and its use in de-

termining threshold behaviour is described. Some results from the theory of SIR (susceptible $\rightarrow$ infective $\rightarrow$ removed) epidemics needed in the sequel are also given. In the following three sections the threshold parameter and probability of a global epidemic are determined under various assumptions concerning the distributions of latent and infectious periods: in Section 2.3, exponential latent and infectious periods; in Section 2.4, exponential latent and constant infectious periods; and in Section 2.5, constant latent and infectious periods. Section 2.6 considers diseases which are highly infectious within households, for which more explicit results can be obtained. Section 2.7 contains some numerical illustrations of the theory, and Section 2.8 provides some concluding comments.

2.2 Background

2.2.1 Model

Consider the following SEIR (susceptible $\rightarrow$ exposed $\rightarrow$ infective $\rightarrow$ removed) epidemic model among a closed population of size N. At any time, each individual in the population is in one of four states: susceptible, exposed (i.e. latent), infective or removed. Initially a small number of individuals are infectives and the rest are susceptible. A susceptible individual becomes a latent individual if he/she makes contact with an infective in a manner described below. A latent individual remains latent for a period of time distributed according to a random variable T_L, having an arbitrary but specified distribution (i.e. no assumption is made about the form of its distribution, but the distribution has to be known), at the end of which he/she becomes infective. An infective individual remains infectious for a period of time distributed according to a random variable T_I, having an arbitrary but specified distribution with finite moment-

generating function (see Section 2.2.4), and then becomes removed. Once removed, an individual no longer plays a part in the epidemic process. The epidemic ends when there are no more latent or infective individuals left in the population.

The population of N individuals is partitioned into m households of size n. During his/her infectious period, a given infective makes *global* contacts with any given susceptible in the population at times given by the points of a homogeneous Poisson process with rate λ_G/N and, additionally, *local* contacts with any given susceptible in its household at times given by the points of a homogeneous Poisson process with rate λ_L. All of the Poisson processes, and the random variables describing latent and infectious periods, are assumed to be mutually independent. Note that, for ease of exposition, households are assumed to be of the *same* size, but the theory may be easily extended to consider households of unequal size, the details of which are given in Section 2.8.

There are *vaccination* and *isolation* policies incorporated in the model. At the time of the first removal within a household, all members of that household are vaccinated, with vaccine response described by the models in Section 2.2.2. Additionally, for $j = 1, 2, \ldots, n-1$, at the time of the jth removal in a given household there is, independently of all previous events, a probability p_j of that household being isolated, given that it has not already been isolated. After a household has been isolated, infective individuals of that household cannot make any further global contacts. Special cases of particular interest include $p_j = 0$ for all j (i.e. no isolation) and $p_1 = 1$ (i.e. isolation at the first removal). Note that we use the word 'isolation' here to describe the process of not just isolating the diagnosed case, but also quarantining their household.

2.2.2 Vaccine action models

Two different assumptions as to which types of individual may be affected by the vaccine (i.e. are vaccine-sensitive) are considered: (i) only susceptible individuals, and (ii) susceptible and exposed individuals. In both cases, infectives are considered vaccine-insensitive.

Following Becker and Starczak [17], the response of an individual to the vaccine is described by a random vector (A, B). Here, A is the relative susceptibility of a vaccinated individual compared with an unvaccinated individual and B the relative infectivity should he or she become infective. For example, the global contact rate between an unvaccinated infective and a vaccinated susceptible with $A = a$ is $a\lambda_G/N$ and the local contact rate between a vaccinated individual with $B = b$ who becomes infected and an unvaccinated susceptible in the same household is $b\lambda_L$. (The latter assumes implicitly that a vaccinated individual's infectivity is reduced (for $b < 1$, as is usual) by lowering his/her infectious rate without changing the infectious period. However, the same reduction could be achieved by keeping the infectious rate unchanged and shortening the infectious period. For simplicity we assume the former throughout the chapter and indicate where the results would change if reduction in infectivity is modelled differently.) Each individual responds independently. We consider two specific vaccine response models, both of which are common in the literature.

The first model describes an *all-or-nothing* vaccine response (see Halloran et al. [25]), where $P(A = B = 0) = \varepsilon = 1 - P(A = B = 1)$, i.e. complete immunity is rendered with probability ε, otherwise there is no effect. We denote the probability that the vaccine renders a susceptible individual immune by ε_S, and (when latents are vaccine-sensitive) a latent individual immune by ε_L.

The other model describes a *non-random* vaccine response, with $P(A = a, B = b) = 1$ for some (a, b), so all vaccine-sensitive individuals respond identically. In this case, after vaccination the epidemic becomes a two-type SEIR epidemic with types 1 and 2 corresponding to individuals who were vaccine-insensitive and vaccine-sensitive, respectively, when they were vaccinated. Let $\Lambda_L = \left[\lambda_{ij}^L\right]$ be the matrix in which λ_{ij}^L $(i, j = 1, 2)$ is the rate at which a given type-i infective infects a given type-j susceptible locally. It follows that

$$\Lambda_L = \begin{bmatrix} \lambda_L & a\lambda_L \\ b\lambda_L & ab\lambda_L \end{bmatrix}. \tag{2.2.1}$$

2.2.3 Threshold behaviour and infectious intensity

If the number of households m is large and the number of initial infectives is small, then during the early stages of an epidemic, there is only a small probability that a global contact is made with an individual from a household containing at least one non-susceptible individual. Thus, we can approximate the initial stages of the epidemic by a process in which all global contacts are made with individuals residing in completely susceptible households. In this approximation, the process of infected households follows a branching process.

Consider a single household epidemic: a completely susceptible household into which a global contact introduces infection, and suppose that no further global contact into the household occurs subsequently (thus initially there are $n - 1$ susceptibles and 1 infective). We call the number of global contacts emanating from this single household epidemic R. Then $R_* = \mathrm{E}[R]$ is a threshold parameter, since if $R_* \leq 1$ then a global epidemic cannot occur (a *global epidemic* occurs if, in the limit $m \to \infty$, the epidemic infects infinitely many households in the branching process approximation). Let $f(s) =$

$\mathrm{E}[s^R]$ be the probability generating function of R. When $R_* > 1$, and the epidemic is started by one initial infective, the probability of a global epidemic, p_G say, is $1 - \tau$, where τ is the root of $f(s) = s$ in $(0, 1)$. The parameter R_* is a households model equivalent of the basic reproduction number R_0; for R_0, see, for example, Heesterbeek and Dietz [26].

In order to calculate R_*, we use the concept of *infectious intensity*, which we now describe. The amount of time an individual is infectious for, whilst the household is not isolated, is called their active infectious period. An individual's active infectious period multiplied by their infectivity, relative to an unvaccinated individual, is called their effective infectious period. Specifically, if an individual's active infectious period is t, then their effective infectious period is t unless he/she is vaccine-sensitive with $B = b$, in which case it is bt. Denote the sum of the effective infectious periods of all infected individuals in a household by C_A, which we refer to as the infectious intensity of the single household epidemic. Define C_B and C_R as the infectious intensity generated before and after the first removal, respectively, so that $C_A = C_B + C_R$.

Global contacts are made by a given infective at rate λ_G during his/her effective infectious period, so the total number of global contacts, R, emanating from a single household epidemic has a Poisson distribution with random mean $\lambda_G C_A$. Thus, $R_* = \lambda_G \mathrm{E}[C_A]$ and $f(s) = \mathrm{E}\left[\mathrm{E}\left[s^R \,|C_A\right]\right] = \mathrm{E}\left[\exp\left(-\lambda_G C_A(1-s)\right)\right] = \psi(\lambda_G(1-s))$, where, for $\theta \geq 0$, $\psi(\theta) = \mathrm{E}\left[e^{-\theta C_A}\right]$. Unless specified otherwise, moment-generating functions are defined for $\theta \geq 0$.

2.2.4 Single-type SIR epidemics

Here we introduce notation and results we need from the theory of single-type SIR epidemics. Let $E(n_s, \phi, \phi_1, \beta)$ be a single-type SIR epidemic with n_s initial susceptibles, who, if they become infectious, will have an infectious period distributed as T with moment-generating function $\phi(\theta) = \mathrm{E}\left[e^{-\theta T}\right]$ and 1 initial infective, whose infectious period is distributed as T_1 with moment-generating function $\phi_1(\theta) = \mathrm{E}\left[e^{-\theta T_1}\right]$ (we assume moment-generating functions are finite for $\theta \geq 0$). The individual-to-individual contact rate is β. The *severity* of this epidemic, χ say, is the sum of the infectious periods of all the individuals who become infected, including the initial infective.

We now present results for this epidemic, obtained by modifying results from Ball [5], by (i) generalising results from $\beta = 1$ to the case of arbitrary but specified $\beta \geq 0$ and (ii) replacing equation (2.3) in Theorem 2.1 by equation (4.2) of Ball [5] and modifying all the relevant results accordingly. Thus, the expected severity of $E(n_s, \phi, \phi_1, \beta)$ is (by modifying Corollary 2.2 of Ball [5]),

$$\mathrm{E}[\chi] = \mathrm{E}\left[T_1\right] + \mu(n_s, \phi, \phi_1, \beta)\mathrm{E}\left[T\right],$$

where $\mu(n_s, \phi, \phi_1, \beta)$ is the mean number of susceptibles ultimately infected in $E(n_s, \phi, \phi_1, \beta)$, which, by modifying equation (2.25) of Ball [5], is given by

$$\mu(n_s, \phi, \phi_1, \beta) = n_s - \sum_{k=1}^{n_s} \binom{n_s}{k} \alpha_k \left(\phi(\beta k)\right)^{n_s - k} \phi_1(\beta k), \tag{2.2.2}$$

where $\alpha_1, \alpha_2, \ldots$ are defined recursively by

$$\sum_{\omega=1}^{k} \binom{k}{\omega} \alpha_\omega \left(\phi(\beta\omega)\right)^{k-\omega} = k \qquad (k = 1, 2, \ldots).$$

Let $\psi(n_s, \phi, \phi_1, \beta, \theta) = \mathrm{E}\left[e^{-\theta\chi}\right]$ be the moment-generating function of the severity of

$E(n_s, \phi, \phi_1, \beta)$. Then, by modifying Theorem 2.5 of Ball [5],

$$\psi(n_s, \phi, \phi_1, \beta, \theta) = \sum_{k=0}^{n_s} \binom{n_s}{k} \xi_k(\theta) \left(\phi(\beta k + \theta)\right)^{n_s - k} \phi_1(\beta k + \theta), \tag{2.2.3}$$

where $\xi_0(\theta), \xi_1(\theta), \ldots$ are defined by

$$\sum_{\omega=0}^{k} \binom{k}{\omega} \xi_\omega(\theta) \left(\phi(\beta\omega + \theta)\right)^{k-\omega} = 1 \qquad (k = 0, 1, \ldots).$$

We now describe how these results can be used here. Suppose there is no isolation in the response model and immediately after vaccination there are i_0 infectives, with infective j ($j = 1, 2, \ldots, i_0$) having remaining infectious period distributed as $T^{(j)}$.

First suppose that the vaccine response is all-or-nothing, and immediately after vaccination there are s_0 susceptibles and l_0 latents. Then C_R is distributed as the severity of an SEIR epidemic whose parameters depend on s_0, l_0, i_0 and the $T^{(j)}$s. Specifically, (i) the severity of an SEIR epidemic is distributed as the severity of an SIR epidemic, so initial latents may be regarded as initial infectives (see Section 4 of Ball [5]); (ii) moreover, one can equivalently assume there is just one initial infective, whose infectious period is equal to the sum of the infectious periods of all the initial latents and infectives. Thus C_R is distributed as the severity of $E(s_0, \phi, \phi_1, \lambda_L)$, with $\phi(\theta) = \mathrm{E}\left[e^{-\theta T_I}\right]$ and $\phi_1(\theta) = \left(\phi(\theta)\right)^{l_0} \prod_{j=1}^{i_0} \mathrm{E}\left[e^{-\theta T^{(j)}}\right]$, where the product is 1 when $i_0 = 0$.

Now suppose that the vaccine response is non-random, and that the vaccine is given to s_0 susceptibles (all vaccine-sensitive), l_{01} vaccine-insensitive latents and l_{02} vaccine-sensitive latents immediately after vaccination (at least one of l_{01} and l_{02} will be zero). By a similar argument to that above, C_R is distributed as the severity of $E(s_0, \phi_{NR}, \phi_1, a\lambda_L)$, where $\phi_{NR}(\theta) = \mathrm{E}\left[e^{-b\theta T_I}\right]$ and $\phi_1(\theta) = \left(\phi(\theta)\right)^{l_{01}} \left(\phi_{NR}(\theta)\right)^{l_{02}} \prod_{j=1}^{i_0} \mathrm{E}\left[e^{-\theta T^{(j)}}\right]$. Note that here *all* latents are incorporated into the single initial infective, so the ensuing epidemic is single-type.

2.2.5 Notation

For the single household epidemic introduced in Section 2.2.3 and $t \geq 0$, let $\boldsymbol{X}(t) = (S(t), L(t), I(t), R(t))$, where $S(t)$, $L(t)$, $I(t)$ and $R(t)$ are respectively the number of susceptible, latent, infective and removed individuals at time t. For the non-random vaccine model and $i = 1, 2$, let $S_i(t)$, $L_i(t)$ and $I_i(t)$ be the numbers of susceptibles, latents and infectives of type-i respectively at time $t > t_R$, where t_R is the time of the first removal. Noting that $S_1(t) = 0$ after vaccination, let $\boldsymbol{X}_{NR}(t) = (S_2(t), L_1(t), L_2(t), I_1(t), I_2(t), R(t))$, for $t > t_R$.

Denote by $\mathbf{0}$ a row vector of zeros and by $\mathbf{1}$ a column vector of ones, the dimensions of these being apparent from their context. Finally, let $t-$ and $t+$ denote left and right limit, respectively, for example, $S(t-) = \lim_{u \uparrow t} S(u)$ and $S(t+) = \lim_{u \downarrow t} S(u)$.

The most important parameters and functions appearing throughout this chapter are listed in Table 2.1, along with brief definitions.

2.3 Exponential latent and infectious periods

2.3.1 General theory

In this section $T_L \sim \text{Exp}(\delta)$ and $T_I \sim \text{Exp}(\gamma)$, i.e. T_L and T_I are exponentially distributed with means δ^{-1} and γ^{-1}, respectively. Under these assumptions, we derive expressions for the threshold parameter R_* and the probability of a global epidemic p_G, for various intervention models. Since infectious and latent periods are exponentially distributed, the single household epidemic model is Markovian.

For this case, there is a useful random time scale transformation of $\{\boldsymbol{X}(t)\}$ (cf. Watson [46] and Ball et al. [10]). For $t \geq 0$, let $\chi(t) = \int_0^t I(u)du$ be the severity of the household

Table 2.1: List of important parameters and functions for Chapter 2.

parameter	description
N	number of individuals in the population
n	number of individuals in each household
λ_L	local (i.e. within-household) individual-to-individual contact rate
λ_G/N	global (i.e. between-household) individual-to-individual contact rate
p_j	probability that a household is isolated at the jth removal, given that it is not already isolated
ε_S	all-or-nothing vaccine efficacy for susceptibles
ε_L	all-or-nothing vaccine efficacy for latents
a	non-random vaccine relative susceptibility
b	non-random vaccine relative infectivity
γ	rate parameter for exponentially distributed infectious period (i.e. mean=$\frac{1}{\gamma}$)
δ	rate parameter for exponentially distributed latent period (i.e. mean=$\frac{1}{\delta}$)
ι	length of constant infectious period
η	length of constant latent period
R_*	expected number of global contacts emanating from a typical single household epidemic
p_G	probability of a global epidemic
C_A	infectious intensity of a single household epidemic
C_B	infectious intensity generated before the first removal in a single household epidemic
C_R	infectious intensity generated after the first removal in a single household epidemic
$p_S(i,u)$	probability that an all-or-nothing vaccine has no effect on u out of i susceptibles
$p_L(j,v)$	probability that an all-or-nothing vaccine has no effect on v out of j latents

epidemic over $[0, t]$, and let $T_A = \chi(\infty)$. For $u \in [0, T_A]$, let $U(u) = \min\{t \geq 0 : \chi(t) = u\}$ and let $\tilde{\boldsymbol{X}}(u) = (\tilde{S}(u), \tilde{L}(u), \tilde{I}(u), \tilde{R}(u)) = \boldsymbol{X}(U(u))$. Thus the process $\{\tilde{\boldsymbol{X}}(u)\} = \{\tilde{\boldsymbol{X}}(u) : 0 \leq u \leq T_A\}$ is a random time scale transformation of $\{\boldsymbol{X}(t)\}$, obtained by running the clock at rate $I(t)^{-1}$ when $I(t) > 0$ and stopping the clock when $I(t) = 0$ (restarting it if and when $I(t) > 0$ again). In particular, time in $\{\tilde{\boldsymbol{X}}(u)\}$ corresponds to severity in $\{\boldsymbol{X}(t)\}$. In this transformed process we have that (i) removals occur at the points of a homogeneous Poisson process with rate γ; (ii) the times of these removals are the severity up until the corresponding removals in $\{\boldsymbol{X}(t)\}$; and (iii) independently of the removal process, susceptibles are infected independently at rate λ_L, provided there is at least one infective.

Let T_0 be the time of the first removal in $\{\tilde{\boldsymbol{X}}(t)\}$. Then (i) above implies that $T_0 \sim \text{Exp}(\gamma)$, and (ii) implies that $C_B = T_0$. By the lack-of-memory of the latent and infectious period distributions, we can condition on the state of the household epidemic when the first removal occurs (but before vaccination) to obtain the required results. For this, we require only $\tilde{S}(T_0-)$ and $\tilde{L}(T_0-)$, because for $t < T_0$, $\tilde{S}(t) + \tilde{L}(t) + \tilde{I}(t) = n$. Also, for $t < T_0$, $\tilde{I}(t) \geq 1$ and $\tilde{R}(t) = 0$, thus $\left(\tilde{S}(T_0-), \tilde{L}(T_0-)\right)$ takes values in the set $\Delta_0 = \{(i, j) : i = 0, 1, \ldots, n-1, j = 0, 1, \ldots, n-i-1\}$.

We now derive expressions for R_* and the generating function required to obtain p_G. We have that

$$\begin{aligned} R_* &= \lambda_G \mathrm{E}[C_A] = \lambda_G \mathrm{E}[T_0 + C_R] = \lambda_G \left(\mathrm{E}[T_0] + \mathrm{E}\left[\mathrm{E}\left[C_R \middle| \tilde{S}(T_0-), \tilde{L}(T_0-)\right]\right]\right) \\ &= \lambda_G \left(\frac{1}{\gamma} + \sum_{i=0}^{n-1} \sum_{j=0}^{n-i-1} \pi_{ij} H_{ij}\right), \end{aligned} \tag{2.3.1}$$

where, for $(i, j) \in \Delta_0$,

$$\pi_{ij} = \mathrm{P}\left(\tilde{S}(T_0-) = i, \tilde{L}(T_0-) = j\right)$$

and

$$H_{ij} = \mathrm{E}\left[C_R \,\middle|\, \tilde{S}(T_0-) = i, \tilde{L}(T_0-) = j\right].$$

Further, for $\theta \geq 0$,

$$\begin{aligned}\psi(\theta) &= \mathrm{E}\left[e^{-\theta C_A}\right] = \mathrm{E}\left[e^{-\theta T_0} e^{-\theta C_R}\right] = \mathrm{E}\left[\mathrm{E}\left[e^{-\theta T_0} e^{-\theta C_R} \,\middle|\, \tilde{S}(T_0-), \tilde{L}(T_0-)\right]\right] \\ &= \sum_{i=0}^{n-1} \sum_{j=0}^{n-i-1} \mathrm{E}\left[e^{-\theta T_0} 1_{\left\{\tilde{S}(T_0-)=i, \tilde{L}(T_0-)=j\right\}}\right] \mathrm{E}\left[e^{-\theta C_R} \,\middle|\, \tilde{S}(T_0-) = i, \tilde{L}(T_0-) = j\right] \\ &= \sum_{i=0}^{n-1} \sum_{j=0}^{n-i-1} g_{ij}(\theta) h_{ij}(\theta), \end{aligned} \tag{2.3.2}$$

where 1_A is the indicator function for the event A (i.e. if A occurs $1_A = 1$, otherwise $1_A = 0$), and, for $(i, j) \in \Delta_0$ and $\theta \geq 0$,

$$g_{ij}(\theta) = \mathrm{E}\left[e^{-\theta T_0} 1_{\left\{\tilde{S}(T_0-)=i, \tilde{L}(T_0-)=j\right\}}\right]$$

and

$$h_{ij}(\theta) = \mathrm{E}\left[e^{-\theta C_R} \,\middle|\, \tilde{S}(T_0-) = i, \tilde{L}(T_0-) = j\right].$$

In the following subsections expressions for π_{ij}, H_{ij}, $g_{ij}(\theta)$ and $h_{ij}(\theta)$ are derived under various intervention schemes. Note that, since there is no intervention prior to the first removal, π_{ij} and $g_{ij}(\theta)$ do *not* depend on the intervention model; calculation of these quantities is described in Section 2.3.2. Calculation of H_{ij} and $h_{ij}(\theta)$ under different intervention schemes is described in Sections 2.3.3 to 2.3.5.

2.3.2 Pre-intervention infectious intensity

For $t < T_0$, $\left\{\left(\tilde{S}(t), \tilde{L}(t), \tilde{I}(t)\right)\right\}$ is governed by the following transition rates:

from	to	rate
$(i,j,n-i-j)$	$(i-1,j+1,n-i-j)$	$\lambda_L i$
	$(i,j-1,n-i-j+1)$	$\frac{\delta j}{n-i-j}$
	$(i,j,n-i-j-1)$	γ

Let $\tilde{\Delta} = \left\{(i,j,k) \in \mathbb{Z}_+^3 : k \geq 1, i+j+k = n\right\}$ be the set of possible transient states of $\left\{\left(\tilde{S}(t), \tilde{L}(t), \tilde{I}(t)\right) : 0 \leq t < T_0\right\}$, n' be the cardinality (i.e. number of elements) of $\tilde{\Delta}$ and $h : \tilde{\Delta} \to \{1, 2, \ldots, n'\}$ be bijective (i.e. for every y in $\{1, 2, \ldots, n'\}$, there is exactly one x in $\tilde{\Delta}$ such that $h(x) = y$). The time-transformed household epidemic prior to the first removal can be represented by a process $\{Y(t) : 0 \leq t \leq T_0\}$, where $Y(t) = h\left(\tilde{S}(t), \tilde{I}(t), \tilde{L}(t)\right)$ $(0 \leq t < T_0)$ and $Y(T_0) = n' + 1$ (i.e. state $n'+1$ corresponds to all states of $\left\{\tilde{\boldsymbol{X}}(t)\right\}$ in which at least one removal has occurred, and hence is absorbing). The transition-rate matrix of $\{Y(t)\}$ has the form:

$$Q = \begin{bmatrix} Q_0 & -Q_0\mathbf{1} \\ \mathbf{0} & 0 \end{bmatrix},$$

where Q_0 is the matrix of transition rates among the transient states, which can be obtained using the above transition table. Note that all entries in $-Q_0\mathbf{1}$ (which gives the absorption rates) are γ.

The time to absorption is $T_0 = \min\{u > 0 : Y(u) = n' + 1\}$. For $y = 1, 2, \ldots, n'$ and $t \geq 0$, let $F_y(t) = P(T_0 \leq t \text{ and } Y(T_0-) = y | Y(0) = y_0)$ and $f_y(t) = F_y'(t)$. Then (see, for example, Asmussen [4], page 83),

$$f_y(t) = \left(e^{Q_0 t}\right)_{y_0,y} (-Q_0\mathbf{1})_y = \gamma \left(e^{Q_0 t}\right)_{y_0,y} \qquad (t \geq 0),$$

where $e^{Q_0 t} = \sum_{k=0}^{\infty} \frac{t^k Q_0^k}{k!}$ denotes the usual matrix exponential. Thus, for $y = 1, 2, \ldots, n'$,

$$\mathrm{P}(Y(T_0-) = y | Y(0) = y_0) = \int_0^\infty f_y(t)dt = \int_0^\infty \gamma \left(e^{Q_0 t}\right)_{y_0,y} dt = -\gamma \left(Q_0^{-1}\right)_{y_0,y}. \quad (2.3.3)$$

Note that Q_0 is non-singular since $\{1, 2, \ldots, n'\}$ is a transient class, so all the eigenvalues of Q_0 have strictly negative real parts (see Asmussen [4], page 83). Further, for $\theta \geq 0$

$$\begin{aligned}
\mathrm{E}\left[e^{-\theta T_0} 1_{\{Y(T_0-)=y\}} \middle| Y(0) = y_0\right] &= \mathrm{E}\left[e^{-\theta T_0} \mathrm{P}(Y(T_0-) = y | Y(0) = y_0, T_0)\right] \\
&= \mathrm{E}\left[e^{-\theta T_0} \left(e^{Q_0 T_0}\right)_{y_0,y}\right] \\
&= \left(\mathrm{E}\left[e^{-\theta T_0} e^{Q_0 T_0}\right]\right)_{y_0,y} \\
&= \left(\int_0^\infty \gamma e^{-\gamma t} e^{-\theta t} e^{Q_0 t} dt\right)_{y_0,y} \\
&= \gamma \left(\left((\theta + \gamma) I - Q_0\right)^{-1}\right)_{y_0,y}. \qquad (2.3.4)
\end{aligned}$$

For $(i, j) \in \Delta_0$, π_{ij} and $g_{ij}(\theta)$ are obtained by setting $y = h(i, j, n - i - j)$ in (2.3.3) and (2.3.4), respectively.

2.3.3 No isolation, vaccine-insensitive latents

In this case, intervention only affects individuals who are still susceptible when the first removal occurs. Since intervention does not affect latents, the infectious intensity of the household epidemic is invariant to the latent period distribution, and as a result, C_R depends on $\tilde{\boldsymbol{X}}(T_0-)$ only through $\tilde{S}(T_0-)$. Thus, for all j, $H_{ij} = H_{i0}$, and $h_{ij}(\theta) = h_{i0}(\theta)$. Recall that under the random time change described above, susceptibles are infected independently at rate λ_L provided there is at least one infective. Thus $\tilde{S}(T_0-)|T_0 \sim \mathrm{Bin}(n-1, e^{-\lambda_L T_0})$, i.e. it has a binomial distribution with $n-1$ trials and success probability $e^{-\lambda_L T_0}$. For $i = 0, 1, \ldots, n-1$, let $\pi_{i\bullet} = \sum_{j=0}^{n-i-1} \pi_{ij}$ and $g_{i\bullet}(\theta) = \sum_{j=0}^{n-i-1} g_{ij}(\theta)$ ($\theta \geq 0$) (recall (2.3.1) and (2.3.2)). Recall also that $T_0 \sim \mathrm{Exp}(\gamma)$,

so $\mathrm{E}\left[e^{-\theta T_0}\right] = \frac{\gamma}{\gamma+\theta}$. Then,

$$\begin{aligned}
\pi_{i\bullet} &= \mathrm{P}\left(\tilde{S}(T_0-) = i\right) = \mathrm{E}[\mathrm{P}(\tilde{S}(T_0-) = i|T_0)] \\
&= \mathrm{E}\left[\binom{n-1}{i} e^{-\lambda_L T_0 i}\left(1 - e^{-\lambda_L T_0}\right)^{n-i-1}\right] \\
&= \binom{n-1}{i}\mathrm{E}\left[\sum_{k=0}^{n-i-1}(-1)^k\binom{n-i-1}{k}e^{-\lambda_L T_0 i}e^{-\lambda_L T_0 k}\right] \\
&= \binom{n-1}{i}\sum_{k=0}^{n-i-1}(-1)^k\binom{n-i-1}{k}\frac{\gamma}{\gamma+\lambda_L(i+k)}
\end{aligned}$$

and, for $\theta \geq 0$

$$\begin{aligned}
g_{i\bullet}(\theta) &= \mathrm{E}\left[e^{-\theta T_0}1_{\{\tilde{S}(T_0-)=i\}}\right] = \mathrm{E}\left[\binom{n-1}{i} e^{-\lambda_L T_0 i}\left(1 - e^{-\lambda_L T_0}\right)^{n-i-1} e^{-\theta T_0}\right] \\
&= \binom{n-1}{i}\mathrm{E}\left[\sum_{k=0}^{n-i-1}(-1)^k\binom{n-i-1}{k}e^{-\lambda_L T_0 k}e^{-(\lambda_L i+\theta)T_0}\right] \\
&= \binom{n-1}{i}\sum_{k=0}^{n-i-1}(-1)^k\binom{n-i-1}{k}\frac{\gamma}{\gamma+(i+k)\lambda_L+\theta}.
\end{aligned}$$

These expressions are easier to compute than (2.3.3) and (2.3.4). Now (2.3.1) and (2.3.2) yield

$$R_* = \lambda_G\left(\frac{1}{\gamma} + \sum_{i=0}^{n-1}\pi_{i\bullet}H_{i0}\right) \quad \text{and} \quad \psi(\theta) = \sum_{i=0}^{n-1} g_{i\bullet}(\theta)h_{i0}(\theta).$$

It remains to calculate H_{i0} and $h_{i0}(\theta)$ ($i = 0, \ldots, n-1$) for the two vaccine action models. First, the lack-of-memory property of the exponential distribution implies that the remaining infectious periods of infectives just after the first removal follow independent Exp(γ) random variables. Further, if $\tilde{S}(T_0-) = i$, then $\tilde{L}(T_0) + \tilde{I}(T_0) = n-i-1$, so the combined remaining infectious periods of the $n-i-1$ latents and infectives has moment-generating function $\phi_1(\theta) = \left(\frac{\gamma}{\gamma+\theta}\right)^{n-i-1}$.

All-or-nothing vaccine

We now assume the vaccine response is all-or-nothing, as defined in Section 2.2.2. Let S_V denote the number of susceptibles for whom vaccination has no effect. Let $p_S(i, u)$

be the probability that $S_V = u$ when i susceptibles are vaccinated. Thus,

$$p_S(i,u) = \binom{i}{u}(1-\varepsilon_S)^u \varepsilon_S^{i-u} \qquad (u = 0, 1, \ldots, i). \tag{2.3.5}$$

If $\tilde{S}(T_0-) = i$ and $S_V = u$, C_R is distributed as the severity of $E(u, \phi, \phi_1, \lambda_L)$, with $\phi(\theta) = \frac{\gamma}{\gamma+\theta}$ and ϕ_1 as above. Hence, for $(i,j) \in \Delta_0$,

$$H_{ij} = H_{i0} = \frac{1}{\gamma}\sum_{u=0}^{i} p_S(i,u)\left(n - i - 1 + \mu\left(u, \phi, \phi_1, \lambda_L\right)\right)$$

and

$$h_{ij}(\theta) = h_{i0}(\theta) = \sum_{u=0}^{i} p_S(i,u)\psi\left(u, \phi, \phi_1, \lambda_L, \theta\right) \qquad (\theta \geq 0).$$

Non-random vaccine

Suppose now that the vaccine response is non-random, as defined in Section 2.2.2. Given $\tilde{S}(T_0-) = i$, the contributions to the severity after the first removal are distributed as those for $E(i, \phi_{NR}, \phi_1, a\lambda_L)$, where $\phi_{NR}(\theta) = \frac{\gamma}{\gamma+b\theta}$ and ϕ_1 as above, since the infectious periods of vaccinated individuals are essentially changed by a factor b. Hence, for $(i,j) \in \Delta_0$,

$$H_{ij} = H_{i0} = \frac{1}{\gamma}\left(n - i - 1 + b\mu\left(i, \phi_{NR}, \phi_1, a\lambda_L\right)\right)$$

and

$$h_{ij}(\theta) = h_{i0}(\theta) = \psi\left(i, \phi_{NR}, \phi_1, a\lambda_L, \theta\right) \qquad (\theta \geq 0).$$

These results are independent of the precise model used for reduction in infectivity.

2.3.4 No isolation, vaccine-sensitive latents

We assume now that latent individuals are vaccine-sensitive, and that the response model does not include isolation.

All-or-nothing vaccine

Let L_V be the number of latents left immediately after vaccination and $p_L(j,v)$ be the probability that the vaccine has no effect on v out of j latents. Then,

$$p_L(j,v) = \binom{j}{v}(1-\varepsilon_L)^v \varepsilon_L^{j-v} \qquad (v = 0, 1, \ldots, j). \tag{2.3.6}$$

If $\tilde{S}(T_0-) = i$, $\tilde{L}(T_0-) = j$, $S_V = u$ and $L_V = v$, then immediately after vaccination there are u susceptibles, v latents and $n-i-j-1$ infectives. Hence C_R is distributed as the severity of $E(u, \phi, \phi_1, \lambda_L)$, where $\phi(\theta) = \frac{\gamma}{\gamma+\theta}$ and $\phi_1(\theta) = (\phi(\theta))^{n-i-j-1+v}$. Thus, for $(i,j) \in \Delta_0$,

$$H_{ij} = \frac{1}{\gamma}\sum_{u=0}^{i}\sum_{v=0}^{j} p_S(i,u)p_L(j,v)\left(n-i-j-1+v+\mu\left(u,\phi,\phi_1,\lambda_L\right)\right)$$

and

$$h_{ij}(\theta) = \sum_{u=0}^{i}\sum_{v=0}^{j} p_S(i,u)p_L(j,v)\psi\left(u,\phi,\phi_1,\lambda_L,\theta\right) \qquad (\theta \geq 0).$$

Non-random vaccine

If $\tilde{S}(T_0-) = i$ and $\tilde{L}(T_0-) = j$, then immediately after vaccination there are $n-i-j-1$ infectives, having independent remaining infectious periods each following an Exp(γ) distribution. The effective infectious periods of the j vaccinated latents follow independent Exp($\frac{\gamma}{b}$) distributions. Thus C_R is distributed as the severity of $E(i, \phi_{NR}, \phi_1, a\lambda_L)$, with ϕ_{NR} as in Section 2.3.3 and $\phi_1(\theta) = (\phi(\theta))^{n-i-j-1}(\phi_{NR}(\theta))^j$. Hence for $(i,j) \in \Delta_0$,

$$H_{ij} = \frac{1}{\gamma}\left[n-i-j-1+b\left(j+\mu\left(i,\phi_{NR},\phi_1,a\lambda_L\right)\right)\right]$$

and

$$h_{ij}(\theta) = \psi\left(i,\phi_{NR},\phi_1,a\lambda_L,\theta\right) \qquad (\theta \geq 0).$$

As in Section 2.3.3, these results do not depend on the precise model used for reduction in infectivity.

2.3.5 Isolation

If $p_1 = 1$ (i.e. isolation at the first removal), there is no infectious intensity generated after the first removal (i.e. $C_R = 0$), so $C_A = T_0$, whence $R_* = \frac{\lambda_G}{\gamma}$ and $\psi(\theta) = \frac{\gamma}{\gamma+\theta}$. Moreover, these expressions hold for any arbitrary but specified latent period distribution, see Section 3.1 of Ball et al. [10].

If $p_1 < 1$ then $\mathrm{P}(C_R > 0) > 0$ and, unless $p_j = 0$ ($j = 2, 3, \ldots, n-1$), we can no longer use results for SIR epidemics to calculate properties of C_R as the household may be isolated *after* the first removal. However, we can use a random-time transformation to make C_R the absorption time of a transient continuous-time Markov Chain, and use results from the theory of *phase-type* distributions.

We now introduce notation and recall results for phase-type distributions used in the sequel. Let $\{W(t) : t \geq 0\}$ be a continuous-time Markov chain with state space $\{1, 2, \ldots, m+1\}$, where states $1, 2, \ldots, m$ are transient and state $m+1$ is absorbing, so the transition-rate matrix of $\{W(t) : t \geq 0\}$ has the form

$$Q_W = \begin{bmatrix} Q_{0W} & -Q_{0W}\mathbf{1} \\ \mathbf{0} & 0 \end{bmatrix},$$

where Q_{0W} is the $m \times m$ matrix of transition rates among the transient states. Suppose that $W(0) = w_0$ and let $T = \min\{t \geq 0 : W(t) = m+1\}$ be the absorption time. Then T has a phase-type distribution, denoted by $\mathrm{PT}(Q_{0W}, w_0)$, and from Proposition 4.1 on page 83 of Asmussen [4],

$$\mathrm{E}\left[T \,|W(0) = w_0\right] = -\left(Q_{0W}^{-1}\right)_{w_0} \mathbf{1}, \tag{2.3.7}$$

while

$$\mathrm{E}\left[e^{-\theta T}\middle| W(0)=w_0\right] = -\left((\theta I - Q_{0W})^{-1}\right)_{w_0} Q_{0W}\mathbf{1} \quad (\theta \geq 0), \tag{2.3.8}$$

using the notation $(A)_i$ to represent the ith row of A.

All-or-nothing vaccine

Suppose first that latents are vaccine-sensitive. If there is no latent or infective left after vaccination, then there is no contribution to the infectious intensity after the first removal, i.e. $C_R = 0$. Otherwise, if the household is not isolated at the first removal, there may be further contribution. In this case, after the first removal we use again the random-time transformation used before the first removal (running the clock at rate $(I(t))^{-1}$). Hence, if $\left(\tilde{S}(T_0+), \tilde{L}(T_0+), \tilde{I}(T_0+)\right) = (i,j,k)$, then for $t > T_0$, $\left\{\left(\tilde{S}(t), \tilde{L}(t), \tilde{I}(t)\right)\right\}$ is governed by the following transition table:

from	to	at rate
(u,v,w)	$(u-1, v+1, w)$	$\lambda_L u$
	$(u, v-1, w+1)$	$\delta \frac{v}{w}$
	$(u, v, w-1)$ $(w>1)$	$(1-p_{2+i+j+k-u-v-w})\gamma$
	$(u, v-1, 1)$ $(v>0, w=1)$	$(1-p_{2+i+j+k-u-v-w})\gamma$
	$(u, 0, 0)$ $(v=0, w=1)$	$(1-p_{2+i+j+k-u-v-w})\gamma$
	isolation	$p_{2+i+j+k-u-v-w}\gamma$

Note that $\left\{\left(\tilde{S}(t), \tilde{L}(t), \tilde{I}(t)\right)\right\}$ terminates if $v = w = 0$. Also, if $w = 1$ and a removal occurs in the untransformed process, then the clock stops in the transformed process and, provided $v > 0$, starts again when an exposed individual becomes an infective in the untransformed process.

For $i = 0, 1, \ldots, n-2$, $j = 0, 1, \ldots, n-i-2$ and $k = 1, 2, \ldots, n-i-j-1$, let $\tilde{\Delta}_{i,j,k} = \left\{(u, v, w) \in \mathbb{Z}_+^3 : u \leq i, v \leq i+j-u, 1 \leq w \leq i+j+k-u-v\right\}$ be the set of possible transient states of $\left\{\left(\tilde{S}(t), \tilde{L}(t), \tilde{I}(t)\right) : T_0 < t \leq C_A\right\}$ given that $\left(\tilde{S}(T_0+), \tilde{L}(T_0+), \tilde{I}(T_0+)\right) = (i, j, k)$, let $n_{i,j,k}$ be the cardinality of $\tilde{\Delta}_{i,j,k}$ and $h_{i,j,k} : \tilde{\Delta}_{i,j,k} \to \{1, 2, \ldots, n_{i,j,k}\}$ be bijective. Suppose that there are i susceptible, j latent and k infective individuals immediately after vaccination. Then this time-transformed household epidemic post-vaccination can be represented by the process $\{Y_{i,j,k}(t) : 0 \leq t \leq C_R\}$, where $Y_{i,j,k}(0) = h_{i,j,k}(i, j, k)$, $Y_{i,j,k}(t) = h_{i,j,k}\left(\tilde{S}(T_0+t), \tilde{L}(T_0+t), \tilde{I}(T_0+t)\right)$ $(0 < t < C_R)$ and $Y_{i,j,k}(C_R) = n_{i,j,k} + 1$. Recall that C_R is the infectious intensity generated after the first removal (i.e. after the household is vaccinated), so $\{Y_{i,j,k}(t)\}$ is absorbed in state $n_{i,j,k}+1$ and C_R is given by the corresponding time to absorption. Let $Q_{0;i,j,k}$ be the matrix of transition rates among the transient states for $\{Y_{i,j,k}(t)\}$, i.e. among states $1, 2, \ldots, n_{i,j,k}$. Note that the elements of $Q_{0;i,j,k}$ can be obtained using the above transition table. Let $\mu_{i,j,k} = \mathrm{E}\left[C_R \middle| \tilde{S}(T_0+) = i, \tilde{L}(T_0+) = j, \tilde{I}(T_0+) = k\right]$ and $\psi_{i,j,k}(\theta) = \mathrm{E}\left[e^{-\theta C_R} \middle| \tilde{S}(T_0+) = i, \tilde{L}(T_0+) = j, \tilde{I}(T_0+) = k\right]$ $(\theta \geq 0)$. Then, using (2.3.7),

$$\mu_{i,j,k} = -\left(Q_{0;i,j,k}^{-1}\right)_{h_{i,j,k}(i,j,k)} \mathbf{1}$$

and, using (2.3.8),

$$\psi_{i,j,k}(\theta) = -\left((\theta I - Q_{0;i,j,k})^{-1}\right)_{h_{i,j,k}(i,j,k)} Q_{0;i,j,k}\mathbf{1}.$$

Assuming that the household is not isolated when the first removal occurs, there is further active severity if there is at least one individual who is infective or latent immediately after the first removal. If there are only latents, under the random-time transformation, the clock will begin again when the next infective appears. So, in the vaccine-sensitive latents case we have, for $(i, j) \in \Delta_0$ (note that here i and j represent $\tilde{S}(T_0-)$

and $\tilde{L}(T_0-)$, respectively, i.e. the numbers of susceptibles and latents immediately before the first removal, not, as above, immediately after),

$$H_{ij} = (1-p_1)\sum_{u=0}^{i}\sum_{v=0}^{j} p_S(i,u)p_L(j,v)\left(1_{\{i+j<n-1\}}\mu_{u,v,n-i-j-1} + 1_{\{i+j=n-1,v>0\}}\mu_{u,v-1,1}\right),$$

and, for $\theta \geq 0$,

$$h_{ij}(\theta) = p_1 + (1-p_1)\sum_{u=0}^{i}\sum_{v=0}^{j} p_S(i,u)p_L(j,v)\left(1_{\{i+j<n-1\}}\psi_{u,v,n-i-j-1}(\theta) + 1_{\{i+j=n-1,v>0\}}\psi_{u,v-1,1}(\theta)\right) + 1_{\{i+j=n-1,v=0\}}\Big).$$

The above formulae reduce to the vaccine-insensitive latent case on setting $\varepsilon_L = 0$.

An alternative derivation of $\mu_{i,j,k}$: a modified process

In this case, we have an alternative method for obtaining $\mu_{i,j,k}$ (the expected time to absorption) by using the method of Hernández-Suárez and Castillo-Chavez [28], which was extended to an epidemic framework by Ball and Lyne [6]. The main idea of the method is to modify the (post-vaccination) process by removing the absorption state and replacing transitions to this state with transitions to the state that the process was in immediately after vaccination. Note that we use the same time-transformation as before.

The process $\{Y_{i,j,k}(t) : 0 \leq t \leq C_R\}$ has one absorption state, $n_{i,j,k}+1$. Thus we define a modified process $\left\{Y^M_{i,j,k}(t) : t \geq 0\right\}$, where the absorption state $n_{i,j,k}+1$ is removed, and transitions to this state are replaced by transitions (at the same rate) to the initial state, $h_{i,j,k}(i,j,k)$.

The modified process is irreducible and positively recurrent, and so it possesses an equilibrium distribution, $\boldsymbol{\pi}_{i,j,k}$. Since C_R was represented by the time to the absorption

of $\{Y_{i,j,k}(t) : 0 \leq t \leq C_R\}$, and absorptions now correspond to $\left\{Y^M_{i,j,k}(t) : t \geq 0\right\}$ returning to its initial state, C_R can now be considered as the time this modified process takes to return to the initial state. If $\boldsymbol{\alpha}_{i,j,k}$ is the vector representing the transition rates back to the initial state, then the expected time for a return to the initial state is given by $\left(\boldsymbol{\pi}^\top_{i,j,k}\boldsymbol{\alpha}_{i,j,k}\right)^{-1}$, and hence

$$\mu_{i,j,k} = \left(\boldsymbol{\pi}^\top_{i,j,k}\boldsymbol{\alpha}_{i,j,k}\right)^{-1}.$$

Non-random vaccine

If the vaccine response is non-random, there are two types of individual after the first removal as described in Section 2.2.2. The derivation of H_{ij} and $h_{ij}(\theta)$ proceeds similarly to before, except that the clock-rate used for the time-change is $(I_1(t) + bI_2(t))^{-1}$. Recalling the notation introduced in Section 2.2.5, let $\left\{\tilde{\boldsymbol{X}}_{NR}(t)\right\}$ be the random time-transformation of $\{\boldsymbol{X}_{NR}(t)\}$, obtained by running the clock at rate $(I_1(t) + bI_2(t))^{-1}$, stopping the clock when there is no infective and starting again when a new infective appears. Then, the times of the removals in $\left\{\tilde{\boldsymbol{X}}_{NR}(t)\right\}$ give the infectious intensity up until the corresponding removals in $\{\boldsymbol{X}_{NR}(t)\}$.

For $t > T_0$, the process $\left\{\left(\tilde{S}_2(t), \tilde{L}_1(t), \tilde{L}_2(t), \tilde{I}_1(t), \tilde{I}_2(t)\right)\right\}$ is governed by the transition table:

from	to	at rate
(i, j_1, j_2, k_1, k_2)	$(i-1, j_1, j_2+1, k_1, k_2)$	$a\lambda_L i$
	$(i, j_1-1, j_2, k_1+1, k_2)$	$\delta \frac{j_1}{k_1+bk_2}$
	$(i, j_1, j_2-1, k_1, k_2+1)$	$\delta \frac{j_2}{k_1+bk_2}$
	$(i, j_1, j_2, k_1-1, k_2)$ $(k_1+k_2>1)$	$(1-p_{n-i-j_1-j_2-k_1-k_2+1})\gamma \frac{k_1}{k_1+bk_2}$
	$(i, j_1, j_2, k_1, k_2-1)$ $(k_1+k_2>1)$	$(1-p_{n-i-j_1-j_2-k_1-k_2+1})\gamma \frac{k_2}{k_1+bk_2}$
	$(i, j_1-1, j_2, 1, 0)$	$(1-p_{n-i-j_1-j_2})\gamma \frac{j_1}{j_1+j_2}$
	$(j_1>0, k_1=1, k_2=0)$	
	$(i, j_1, j_2-1, 0, 1)$	$(1-p_{n-i-j_1-j_2})\gamma \frac{j_2}{j_1+j_2}$
	$(j_2>0, k_1=1, k_2=0)$	
	$(i, j_1-1, j_2, 1, 0)$	$(1-p_{n-i-j_1-j_2})\gamma \frac{j_1}{b(j_1+j_2)}$
	$(j_1>0, k_1=0, k_2=1)$	
	$(i, j_1, j_2-1, 0, 1)$	$(1-p_{n-i-j_1-j_2})\gamma \frac{j_2}{b(j_1+j_2)}$
	$(j_2>0, k_1=0, k_2=1)$	
	$(i, 0, 0, 0, 0)$	$(1-p_{n-i-j_1-j_2})\gamma$
	$(j_1=j_2=0, k_1+k_2=1)$	
	isolation	$p_{n-i-j_1-j_2-k_1-k_2+1}\gamma \frac{k_1+k_2}{k_1+bk_2}$

Let $\tilde{\Delta}_{NR} = \left\{(i, j_1, j_2, k_1, k_2, l) \in \mathbb{Z}_+^6 : k_1+k_2 \geq 1, i+j_1+j_2+k_1+k_2+l = n\right\}$ be the set of possible transient states of $\left\{\tilde{\boldsymbol{X}}_{NR}(t)\right\}$, n_1' be the cardinality of $\tilde{\Delta}_{NR}$ and $h_1 : \tilde{\Delta}_{NR} \to \{1, 2, \ldots, n_1'\}$ be bijective. Similarly to before, the post-vaccination, time-transformed household process can be represented by the process $\{Y_1(t) : 0 \leq t \leq C_R\}$, where $Y_1(0) = h_1\left(\tilde{\boldsymbol{X}}_{NR}(T_0+)\right)$, $Y_1(t) = h_1\left(\tilde{\boldsymbol{X}}_{NR}(T_0+t)\right)$ $(0 < t < C_R)$ and $Y_1(C_R) = n_1'+1$. It then follows that $C_R \left| \tilde{\boldsymbol{X}}_{NR}(T_0+) \right. \sim \text{PT}\left(Q_{0,1}, \tilde{\boldsymbol{X}}_{NR}(T_0+)\right)$, where $Q_{0,1}$ is the matrix of transition rates of $\{Y_1(t)\}$ among its transient states $1, 2, \ldots, n_1'$, which can be

obtained using the above transition table. Let $\mu^{NR}_{i,j_1,j_2,k_1,k_2,l} = \mathrm{E}\left[C_R \,\middle|\, \tilde{\boldsymbol{X}}_{NR}(T_0+) = (i, j_1, j_2, k_1, k_2, l)\right]$ and $\psi^{NR}_{i,j_1,j_2,k_1,k_2,l}(\theta) = \mathrm{E}\left[e^{-\theta C_R} \,\middle|\, \tilde{\boldsymbol{X}}_{NR}(T_0+) = (i, j_1, j_2, k_1, k_2, l)\right]$ $(\theta \geq 0)$ for $i + j_1 + j_2 + k_1 + k_2 + l = n$. Then, using (2.3.7),

$$\mu^{NR}_{i,j_1,j_2,k_1,k_2,l} = -\left(Q_{0,1}^{-1}\right)_{h_1(i,j_1,j_2,k_1,k_2,l)} \mathbf{1}$$

and, using (2.3.8),

$$\psi^{NR}_{i,j_1,j_2,k_1,k_2,l}(\theta) = -\left((\theta I - Q_{0,1})^{-1}\right)_{h_1(i,j_1,j_2,k_1,k_2,l)} Q_{0,1}\mathbf{1}.$$

If $\tilde{S}(T_0-) = i$ and $\tilde{L}(T_0-) = j$, where $i+j < n-1$, then if latent individuals are vaccine-insensitive, $\tilde{\boldsymbol{X}}_{NR}(T_0+) = (i, j, 0, n-i-j-1, 0, 1)$, while if they are vaccine-sensitive, $\tilde{\boldsymbol{X}}_{NR}(T_0+) = (i, 0, j, n-i-j-1, 0, 1)$. However if $\tilde{S}(T_0-) = i$ and $\tilde{L}(T_0-) = j > 0$, where $i + j = n - 1$ so there is no infective immediately after the first removal, then the clock will only restart after the first removal when one of the j latents becomes infectious and thus if latent individuals are vaccine-insensitive, $\tilde{\boldsymbol{X}}_{NR}(T_0+) = (i, j-1, 0, 1, 0, 1)$, while if they are vaccine-sensitive, $\tilde{\boldsymbol{X}}_{NR}(T_0+) = (i, 0, j-1, 0, 1, 1)$. Thus, in the vaccine-insensitive latents case, for $(i, j) \in \Delta_0$,

$$H_{ij} = (1-p_1)\left(1_{\{i+j<n-1\}}\mu^{NR}_{i,j,0,n-i-j-1,0,1} + 1_{\{i+j=n-1,j>0\}}\mu^{NR}_{i,j-1,0,1,0,1}\right)$$

and, for $\theta \geq 0$,

$$\begin{aligned} h_{ij}(\theta) = p_1 &+ (1-p_1)1_{\{i+j<n-1\}}\psi^{NR}_{i,j,0,n-i-j-1,0,1}(\theta) \\ &+ (1-p_1)1_{\{i+j=n-1,j>0\}}\psi^{NR}_{i,j-1,0,1,0,1}(\theta) \\ &+ (1-p_1)1_{\{i=n-1\}}, \end{aligned}$$

while in the vaccine-sensitive latents case, for $(i, j) \in \Delta_0$,

$$H_{ij} = (1-p_1)\left(1_{\{i+j<n-1\}}\mu^{NR}_{i,0,j,n-i-j-1,0,1} + 1_{\{i+j=n-1,j>0\}}\mu^{NR}_{i,0,j-1,0,1,1}\right)$$

and, for $\theta \geq 0$,

$$\begin{aligned} h_{ij}(\theta) = p_1 &+ (1-p_1)1_{\{i+j<n-1\}}\psi^{NR}_{i,0,j,n-i-j-1,0,1}(\theta) \\ &+ (1-p_1)1_{\{i+j=n-1,j>0\}}\psi^{NR}_{i,0,j-1,0,1,1}(\theta) \\ &+ (1-p_1)1_{\{i=n-1\}}. \end{aligned}$$

Note that these expressions would change if the reduction in infectivity of vaccinated individuals was modelled differently, as reducing infectious periods of vaccinated individuals would alter the times of removals.

An alternative derivation of $\mu^{NR}_{i,j_1,j_2,k_1,k_2,l}$: modified process

Once again we can obtain the mean absorption time also by using a modified process. In this case, $\{Y_1(t) : 0 \leq t \leq C_R\}$ is absorbed into state $n_1'+1$, so we define our modified process $\{Y^M_{i,j_1,j_2,k_1,k_2,l}(t) : t \geq 0\}$ by removing state $n_1' + 1$ and replacing transitions to this state with transitions (at the same rate) to the initial state, $h_1(i, j_1, j_2, k_1, k_2, l)$.

The modified process is irreducible and positively recurrent, and so it possesses an equilibrium distribution, $\boldsymbol{\pi}^{NR}_{i,j_1,j_2,k_1,k_2,l}$. Since C_R was represented by the time to the absorption of $\{Y_1(t) : 0 \leq t \leq C_R\}$, and absorptions now correspond to $\{Y^M_{i,j_1,j_2,k_1,k_2,l}(t) : t \geq 0\}$ returning to its initial state, C_R can now be considered as the time this modified process takes to return to the initial state. If $\boldsymbol{\alpha}^{NR}_{i,j_1,j_2,k_1,k_2,l}$ is the vector representing the transition rates back to the initial state, then the expected time for a return to the initial state is given by $\left(\left(\boldsymbol{\pi}^{NR}_{i,j_1,j_2,k_1,k_2,l}\right)^\top \boldsymbol{\alpha}^{NR}_{i,j_1,j_2,k_1,k_2,l}\right)^{-1}$, and hence

$$\mu^{NR}_{i,j_1,j_2,k_1,k_2,l} = \left(\left(\boldsymbol{\pi}^{NR}_{i,j_1,j_2,k_1,k_2,l}\right)^\top \boldsymbol{\alpha}^{NR}_{i,j_1,j_2,k_1,k_2,l}\right)^{-1}.$$

2.4 Exponential latent and constant infectious periods

2.4.1 General theory

If $T_L \sim \text{Exp}(\delta)$ and $T_I \equiv \iota$ (i.e. it is constant with value ι), the time of the first removal is ι. Since latent periods are exponentially distributed, for $t < \iota$ (i.e. before the first removal), $\{\boldsymbol{X}(t)\}$ is Markovian and $\{(S(t), L(t), I(t))\}$ is governed by the transition table:

from	to	at rate
$(i, j, n-i-j)$	$(i-1, j+1, n-i-j)$	$\lambda_L i(n-i-j)$
	$(i, j-1, n-i-j+1)$	δj

Define $\{Y_2(t)\} = \{Y_2(t) : 0 \leq t < \iota\}$ to be a relabelling of $\{\boldsymbol{X}(t) : 0 \leq t < \iota\}$, such that $Y_2(t) = h_0(S(t), L(t))$, where $h_0 : \Delta_0 \to \{1, 2, \ldots, n'\}$ (note that for $0 \leq t < \iota$, if $S(t) = i$ and $L(t) = j$, then $I(t) = n - i - j$). Thus, if $Y_2(0) = y_0 = h_0(n-1, 0)$,

$$P(Y_2(\iota-) = y | Y_2(0) = y_0) = \left(e^{Q_2 \iota}\right)_{y_0, y},$$

where Q_2 is the transition-rate matrix of $\{Y_2(t)\}$. Thus,

$$\begin{aligned} R_* &= \lambda_G \text{E}\left[C_A\right] = \lambda_G \text{E}\left[\text{E}\left[C_A \middle| S(\iota-), L(\iota-)\right]\right] \\ &= \lambda_G \sum_{i=0}^{n-1} \sum_{j=0}^{n-i-1} \left(e^{Q_2 \iota}\right)_{y_0, h_0(i,j)} G_{ij}, \end{aligned}$$

where, for $(i, j) \in \Delta_0$,

$$G_{ij} = \text{E}\left[C_A \middle| S(\iota-) = i, L(\iota-) = j\right],$$

and, for $\theta \geq 0$,

$$\begin{aligned} \psi(\theta) &= \text{E}\left[e^{-\theta C_A}\right] = \text{E}\left[\text{E}\left[e^{-\theta C_A} \middle| S(\iota-), L(\iota-)\right]\right] \\ &= \sum_{i=0}^{n-1} \sum_{j=0}^{n-i-1} \left(e^{Q_2 \iota}\right)_{y_0, h_0(i,j)} \phi_{ij}(\theta), \end{aligned}$$

where, for $(i,j) \in \Delta_0$ and $\theta \geq 0$,

$$\phi_{ij}(\theta) = \mathrm{E}\left[e^{-\theta C_A} \middle| S(\iota-) = i, L(\iota-) = j \right].$$

Thus to calculate R_* and $\psi(\theta)$ we need to calculate G_{ij} and $\phi_{ij}(\theta)$. Note that if $S(\iota-) = i$ and $L(\iota-) = j$, then $I(\iota) = n-i-j-1$. If we label these infectives $1, 2, \ldots, n-i-j-1$, then at time ι infective k will have a remaining infectious period, ι_k say, which takes some value in $[0, \iota]$, so the combined remaining infectious period of these infectives is $\sum_{i=1}^{n-i-j-1} \iota_k$. To proceed we introduce a reward process defined on a continuous-time Markov chain, see for example, Keilson and Rao [31] or, closer to the present notation, Ball et al. [8]. Define a reward process $\{Z(t) : 0 \leq t \leq \iota\}$ by letting $Z(t) = \int_0^t I(u)du$, the total severity up until time t (so $C_B = Z(\iota)$). Then $\sum_{k=1}^{n-i-j-1} \iota_k = (n-i-j)\iota - Z(\iota)$. Thus, for $0 \leq t < \iota$, if $Y_2(t)$ is in state $h_0(i,j)$, so there are $n-i-j$ infectives, reward is earned at rate $\rho_{h_0(i,j)} = n-i-j$ per unit time. Defining $D = \mathrm{diag}(\rho_1, \rho_2, \ldots, \rho_{n'})$, and using Theorem 3.3 of Ball et al. [8] yields

$$\begin{aligned} \mathrm{E}\left[e^{-\theta Z(t)} \middle| Y_2(t) = h_0(i,j) \right] &= \frac{\left(e^{(Q_2 - \theta D)t}\right)_{y_0, h_0(i,j)}}{P(Y_2(t) = h_0(i,j))} \\ &= \frac{\left(e^{(Q_2 - \theta D)t}\right)_{y_0, h_0(i,j)}}{\left(e^{Q_2 t}\right)_{y_0, h_0(i,j)}} \qquad (\theta \in \mathbb{R}), \end{aligned} \tag{2.4.1}$$

which we use to obtain the results we need.

2.4.2 All-or-nothing vaccine, no isolation

Assume latent individuals are vaccine-sensitive. If $S(\iota-) = i$, $L(\iota-) = j$, $S_V = u$ and $L_V = v$, then, at the first removal, the total remaining infectious period of the v latents and $n-i-j-1$ infectives is $v\iota + \sum_{k=1}^{n-i-j-1} \iota_k = (n-i-j+v)\iota - Z(\iota)$. Hence, given $Z(\iota)$, C_R is distributed as the severity of $E(u, \phi, \Phi_1, \lambda_L)$, where $\phi(\theta) = e^{-\iota\theta}$ and

$\Phi_1(\theta) = (\phi(\theta))^{n-i-j+v} e^{\theta Z(\iota)}$. Thus, for $(i,j) \in \Delta_0$,

$$\begin{aligned} G_{ij} &= \sum_{u=0}^{i}\sum_{v=0}^{j} p_S(i,u)p_L(j,v)\mathrm{E}\left[\, Z(\iota) + C_R \middle|\, Y_2(\iota) = h_0(i,j)\right] \\ &= \sum_{u=0}^{i}\sum_{v=0}^{j} p_S(i,u)p_L(j,v)\mathrm{E}\left[\, \iota\left(n-i-j+v+\mu(u,\phi,\Phi_1,\lambda_L)\right) \middle|\, Y_2(\iota) = h_0(i,j)\right] \\ &= \iota \sum_{u=0}^{i}\sum_{v=0}^{j} p_S(i,u)p_L(j,v)\left(n-i-j+v+\mu(u,\phi,\phi_1,\lambda_L)\right), \end{aligned}$$

where the last equality follows by noting that $\mu(u,\phi,\Phi_1,\lambda_L)$ is linear in Φ_1 and

$$\begin{aligned} \phi_1(\theta) &= \mathrm{E}\left[\, \Phi_1(\theta) \middle|\, Y_2(\iota) = h_0(i,j)\right] \\ &= (\phi(\theta))^{n-i-j+v}\mathrm{E}\left[e^{\theta Z(\iota)} \middle|\, Y_2(\iota) = h_0(i,j)\right] \\ &= (\phi(\theta))^{n-i-j+v} \frac{\left(e^{(Q_2+\theta D)\iota}\right)_{y_0,h_0(i,j)}}{\left(e^{Q_2\iota}\right)_{y_0,h_0(i,j)}}, \end{aligned}$$

and, for $(i,j) \in \Delta_0$ and $\theta \geq 0$,

$$\begin{aligned} \phi_{ij}(\theta) &= \sum_{u=0}^{i}\sum_{v=0}^{j} p_S(i,u)p_L(j,v)\mathrm{E}\left[e^{-\theta C_A} \middle|\, S(\iota-) = i, L(\iota-) = j, S_V = u, L_V = v\right] \\ &= \sum_{u=0}^{i}\sum_{v=0}^{j} p_S(i,u)p_L(j,v)\mathrm{E}\left[e^{-\theta Z(\iota)}\psi\left(u,\phi,\Phi_1,\lambda_L,\theta\right) \middle|\, Y_2(\iota-) = h_0(i,j)\right] \\ &= \sum_{u=0}^{i}\sum_{v=0}^{j} p_S(i,u)p_L(j,v)\sum_{k=0}^{u}\binom{u}{k}\xi_k(\theta)e^{-(\theta+\lambda_L k)(u+v+n-i-j-k)\iota} \\ &\qquad\qquad \times \mathrm{E}\left[e^{\lambda_L k Z(\iota)} \middle|\, Y_2(\iota-) = h_0(i,j)\right] \\ &= \sum_{u=0}^{i}\sum_{v=0}^{j} p_S(i,u)p_L(j,v)\sum_{k=0}^{u}\binom{u}{k}\xi_k(\theta)e^{-(\theta+\lambda_L k)(u+v+n-i-j-k)\iota} \\ &\qquad\qquad \times \frac{\left(e^{(Q_2+\lambda_L k D)\iota}\right)_{y_0,h_0(i,j)}}{\left(e^{Q_2\iota}\right)_{y_0,h_0(i,j)}}, \end{aligned}$$

where $\xi_0(\theta), \xi_1(\theta), \ldots$ are defined by

$$\sum_{\omega=0}^{k}\binom{k}{\omega}\xi_\omega(\theta)\left(\phi(\lambda_L\omega+\theta)\right)^{k-\omega} = 1 \qquad (k = 0, 1, \ldots).$$

In the above, $p_S(i,u)$ and $p_L(j,v)$ are given by equations (2.3.5) and (2.3.6). Expressions for the vaccine-insensitive latents case can be obtained by setting $\varepsilon_L = 0$.

2.4.3 Non-random vaccine, no isolation

If $S(\iota-) = i$ and $L(\iota-) = j$, then, given $Z(\iota)$, C_R is distributed as the severity of $E(i, \phi_{NR}, \Phi_1, a\lambda_L)$ if latents are vaccine-insensitive and $E(i, \phi_{NR}, \Phi_2, a\lambda_L)$ if they are vaccine-sensitive, where (with $\phi(\theta)$ as above) $\phi_{NR}(\theta) = e^{-b\iota\theta}$, $\Phi_1(\theta) = (\phi(\theta))^{n-i}e^{\theta Z(\iota)}$ and $\Phi_2(\theta) = (\phi(\theta))^{n-i-j}(\phi_{NR}(\theta))^j e^{\theta Z(\iota)}$. Thus if latents are vaccine-insensitive, we have (using similar arguments to the all-or-nothing case), for $(i,j) \in \Delta_0$,

$$G_{ij} = \iota\left[(n - i + b\mu(i, \phi_{NR}, \phi_1, a\lambda_L)\right],$$

where

$$\phi_1(\theta) = (\phi(\theta))^{n-i}\frac{\left(e^{(Q_2+\theta D)\iota}\right)_{y_0,h_0(i,j)}}{\left(e^{Q_2\iota}\right)_{y_0,h_0(i,j)}},$$

and, for $\theta \geq 0$

$$\phi_{ij}(\theta) = \sum_{k=0}^{u}\binom{k}{u}\xi_k(\theta)e^{-(\theta+a\lambda_L k)(b(i-k)+n-i)\iota}\frac{\left(e^{(Q_2+a\lambda_L kD)\iota}\right)_{y_0,h_0(i,j)}}{\left(e^{Q_2\iota}\right)_{y_0,h_0(i,j)}},$$

while if latents are vaccine-sensitive, then for $(i,j) \in \Delta_0$,

$$G_{ij} = \iota\left[n - i - j + b\left(j + \mu(i, \phi_{NR}, \phi_2, a\lambda_L)\right)\right],$$

where

$$\phi_2(\theta) = (\phi(\theta))^{n-i-j}(\phi_{NR}(\theta))^j\frac{\left(e^{(Q_2+\theta D)\iota}\right)_{y_0,h_0(i,j)}}{\left(e^{Q_2\iota}\right)_{y_0,h_0(i,j)}},$$

and, for $\theta \geq 0$

$$\phi_{ij}(\theta) = \sum_{k=0}^{u}\binom{k}{u}\xi_k(\theta)e^{-(\theta+a\lambda_L k)(b(i+j-k)+n-i-j)\iota}\frac{\left(e^{(Q_2+a\lambda_L kD)\iota}\right)_{y_0,h_0(i,j)}}{\left(e^{Q_2\iota}\right)_{y_0,h_0(i,j)}}.$$

In both cases, $\xi_0(\theta), \xi_1(\theta), \ldots$ are defined by

$$\sum_{\omega=0}^{k}\binom{k}{\omega}\xi_\omega(\theta)\left(\phi_{NR}(a\lambda_L\omega + \theta)\right)^{k-\omega} = 1 \qquad (k = 0, 1, \ldots).$$

2.4.4 Isolation at first removal

In this case, $C_A = Z(\iota)$ and thus to calculate R_*, we need to obtain $\mathrm{E}\left[Z(\iota)\right]$. Note that in $\{Y_2(t)\}$, the state which corresponds to all individuals being infective is absorbing (without loss of generality we shall assume that this is state n'), so Q_2 is singular. States $\{1, 2, \ldots, n'-1\}$ form a transient class, so Q_2 has the form

$$Q_2 = \begin{bmatrix} Q_T & -Q_T\mathbf{1} \\ \mathbf{0} & 0 \end{bmatrix},$$

where Q_T is non-singular. Now if we let $Z(\iota) = Z_T + Z_{n'}$, where Z_T is the reward contribution from the transient states and $Z_{n'}$ is the reward contribution from state n', and let $\boldsymbol{\rho}_T = (\rho_1, \rho_2, \ldots, \rho_{n'-1})^\top$, where $^\top$ denotes transpose, then

$$\mathrm{E}[Z_T] = \left(\int_0^\iota e^{Q_T u}\boldsymbol{\rho}_T du\right)_{y_0} = \left(Q_T^{-1}(e^{Q_T\iota} - I)\boldsymbol{\rho}_T\right)_{y_0}$$

and, conditioning on $\tau = \min\{t : Y_2(t) = n'\}$,

$$\begin{aligned}\mathrm{E}[Z_{n'}] = \mathrm{E}[\mathrm{E}[Z_{n'}|\tau]] &= \left(\int_0^\iota e^{Q_T u}\left(-Q_T\mathbf{1}\right)(\iota - u)\rho_{n'} du\right)_{y_0} \\ &= \left(\left(\iota\mathbf{1} - Q_T^{-1}\left(e^{Q_T\iota} - I\right)\mathbf{1}\right)\rho_{n'}\right)_{y_0}.\end{aligned}$$

Thus,

$$\begin{aligned}R_* &= \lambda_G \mathrm{E}\left[Z(\iota)\right] = \lambda_G \mathrm{E}\left([Z_T] + [Z_{n'}]\right) \\ &= \lambda_G\left(Q_T^{-1}(e^{Q_T\iota} - I)\boldsymbol{\rho}_T + \left(\iota\mathbf{1} - Q_T^{-1}\left(e^{Q_T\iota} - I\right)\mathbf{1}\right)\rho_{n'}\right)_{y_0} \qquad (2.4.2) \\ &= \lambda_G\left(Q_T^{-1}(e^{Q_T\iota} - I)\boldsymbol{\rho}_T + \left(\iota\mathbf{1} - Q_T^{-1}\left(e^{Q_T\iota} - I\right)\mathbf{1}\right)n\right)_{y_0},\end{aligned}$$

since $\rho_{n'} = n$. Also, for $\theta \geq 0$,

$$\begin{aligned}\psi(\theta) &= \mathrm{E}\left[e^{-\theta Z(\iota)}\right] = \mathrm{E}\left[\mathrm{E}\left[e^{-\theta Z(\iota)}\middle| Y_2(\iota-)\right]\right] \\ &= \sum_{i=0}^{n-1}\sum_{j=0}^{n-i-1}\left(e^{Q_2\iota}\right)_{y_0,h_0(i,j)}\frac{\left(e^{(Q_2-\theta D)\iota}\right)_{y_0,h_0(i,j)}}{\left(e^{Q_2\iota}\right)_{y_0,h_0(i,j)}} = \sum_{i=0}^{n-1}\sum_{j=0}^{n-i-1}\left(e^{(Q_2-\theta D)\iota}\right)_{y_0,h_0(i,j)} \\ &= \left(e^{(Q_2-\theta D)\iota}\mathbf{1}\right)_{y_0}.\end{aligned}$$

It is difficult to make analytical progress for a general isolation scheme, since the times of subsequent removals are not fixed.

2.5 Constant latent and infectious periods

2.5.1 Introduction

Assume now that latent and infectious periods are both constant, specifically $T_L \equiv \eta$ and $T_I \equiv \iota$. If $\eta < \iota$ it is difficult to make analytical progress unless n is small since there can be more than one infective before the first removal and the process is not Markov. So, here we assume that, $\eta \geq \iota$, and note that in this case, there is one infective up until the time of the first removal, i.e. $I(t) = 1$ for $t \leq \iota$. Hence, $S(\iota-) \sim \text{Bin}\left(n-1, e^{-\lambda_L\iota}\right)$ and $L(\iota-) = n - 1 - S(\iota-)$.

Thus

$$\begin{aligned}R_* &= \lambda_G \mathrm{E}\left[C_A\right] = \lambda_G\left(\iota + \mathrm{E}\left[C_R\right]\right) = \lambda_G\left(\iota + \mathrm{E}\left[\mathrm{E}\left[C_R\middle| S(\iota-)\right]\right]\right) \\ &= \lambda_G\left(\iota + \sum_{i=0}^{n-1}\binom{n-1}{i}e^{-\lambda_L\iota i}\left(1-e^{-\lambda_L\iota}\right)^{n-i-1}H_i\right),\end{aligned}$$

where, for $i = 0, 1, \ldots, n-1$

$$H_i = \mathrm{E}\left[C_R\middle| S(\iota-) = i\right],$$

while, for $\theta \geq 0$,

$$\begin{aligned}\psi(\theta) = \mathrm{E}\left[e^{-\theta C_A}\right] &= \mathrm{E}\left[e^{-\iota}e^{-\theta C_R}\right] = e^{-\iota}\mathrm{E}\left[\mathrm{E}\left[e^{-\theta C_R}\middle| S(\iota-)\right]\right] \\ &= e^{-\iota\theta}\left(\sum_{i=0}^{n-1}\binom{n-1}{i}e^{-\lambda_L\iota i}\left(1-e^{-\lambda_L\iota}\right)^{n-i-1}h_i(\theta)\right)\end{aligned}$$

where, for $i = 0, 1, \ldots, n-1$ and $\theta \geq 0$,

$$h_i(\theta) = \mathrm{E}\left[e^{-\theta C_R}\middle| S(\iota-) = i\right].$$

Note that if there is no isolation the latent period length η has no bearing on these calculations, the point being that here C_B is known explicitly, while C_R is invariant under changes of latent period.

As in Section 2.4, it is hard to make analytical progress for a general isolation scheme, but in the isolation at the first removal case ($p_1 = 1$), $C_A = \iota$, so $R_* = \lambda_G\iota$ and $\psi(\theta) = e^{-\iota\theta}$.

2.5.2 All-or-nothing vaccine, no isolation

Suppose that latents are vaccine-sensitive. If $S_V = u$ and $L_V = v$, then C_R has the same distribution as the severity of $E(u, \phi, \phi_1, \lambda_L)$, where $\phi(\theta) = e^{-\iota\theta}$ and $\phi_1(\theta) = (\phi(\theta))^v$. Hence,

$$H_i = \iota\sum_{u=0}^{i}\sum_{v=0}^{n-i-1} p_S(i,u)p_L(n-i-1,v)\left(v + \mu(u,\phi,\phi_1,\lambda_L)\right)$$

and, for $\theta \geq 0$,

$$h_i(\theta) = \sum_{u=0}^{i}\sum_{v=0}^{n-i-1} p_S(i,u)p_L(n-i-1,v)\psi(u,\phi,\phi_1,\lambda_L,\theta).$$

In the above, $p_S(i,u)$ and $p_L(j,v)$ are given by equations (2.3.5) and (2.3.6). Corresponding results for the vaccine-insensitive latent case are obtained by setting $\varepsilon_L = 0$.

2.5.3 Non-random vaccine, no isolation

If the vaccine is non-random and $S(\iota-) = i$, then C_R is distributed as the severity of $E(i, \phi_{NR}, \phi_1, a\lambda_L)$ if latents are vaccine-insensitive and $E(i, \phi_{NR}, \hat{\phi}_1, a\lambda_L)$ if they are vaccine-sensitive, where $\phi_{NR}(\theta) = e^{-b\iota\theta}$, $\phi_1(\theta) = (\phi(\theta))^{n-i-1}$ and $\hat{\phi}_1(\theta) = (\phi_{NR}(\theta))^{n-i-1}$. Hence if latents are vaccine-insensitive,

$$H_i = \iota\left(n - i - 1 + b\mu(i, \phi_{NR}, \phi_1, a\lambda_L)\right)$$

and

$$h_i(\theta) = \psi(i, \phi_{NR}, \phi_1, a\lambda_L\theta) \quad (\theta \geq 0),$$

while if they are vaccine-sensitive,

$$H_i = b\iota\left(n - i - 1 + \mu(i, \phi_{NR}, \hat{\phi}_1, a\lambda_L)\right)$$

and

$$h_i(\theta) = \psi(i, \phi_{NR}, \hat{\phi}_1, a\lambda_L, \theta) \quad (\theta \geq 0).$$

These results do not depend on the precise model used for reduction in infectivity.

2.6 Locally highly infectious diseases ($\lambda_L \to \infty$)

2.6.1 Introduction

In this section we consider locally highly infectious diseases, i.e. we consider what happens as $\lambda_L \to \infty$. In this limit, all the susceptibles in the single household epidemic are infected immediately after time zero. Of course, the limiting values of R_* and $\psi(\theta)$ can be obtained by letting $\lambda_L \to \infty$ in the expressions given in Sections 2.3-2.5. However,

for many of these limits more direct and general arguments are available and these are outlined below. The results are generally simpler than those in the previous sections, and provide a useful approximation when λ_L is large. Highly infectious diseases have previously been considered by, for example, Becker and Dietz [13].

Without intervention, or for any vaccine that only works on susceptibles (without isolation in the response model), we simply have $R_* \to \lambda_G n E[T_I]$, since everyone will become infectious, and also therefore $\psi(\theta) \to \left(E[e^{-\theta T_I}]\right)^n$. Thus now we look at response models with vaccine-sensitive latents and/or isolation.

2.6.2 No isolation, vaccine-sensitive latents

We label the initial infective as 1 and the susceptibles as $2, 3, \ldots, n$. Let X_i and Y_i be respectively the latent and infectious periods of individual i. Therefore, $X_1 = 0$, $X_i \sim T_L$ ($i = 2, 3, \ldots, n$) and $Y_i \sim T_I$ ($i = 1, 2, \ldots, n$).

The contribution of individual 1 to the infectious intensity is Y_1. For the other individuals, their contribution depends on whether or not they are infective at the time of the first removal. For $i = 2, 3, \ldots, n$, individual i is latent at the time of the first removal if $\min_{j \neq i} (X_j + Y_j) < X_i$, otherwise they are infectious. Thus, the expected number of latents at the time of the first removal is $\sum_{i=2}^{n} \mathrm{P}(\min_{j \neq i}(X_j + Y_j) < X_i) = (n-1)q$, by symmetry, where $q = \mathrm{P}\left(\min_{j \neq 2} (X_j + Y_j) < X_2\right)$. Thus if the vaccine response is non-random,

$$R_* \to \lambda_G E[T_I] \left[1 + (n-1)(bq + 1 - q)\right],$$

while for the all-or-nothing vaccine, for $i = 2, 3, \ldots, n$, individual i never becomes infectious with probability $\mathrm{P}(\min_{j \neq i}(X_j + Y_j) < X_i)\varepsilon_L$, so, using a similar symmetry

argument,

$$R_* \to \lambda_G E[T_I]\left[1 + (n-1)\left(1 - q\varepsilon_L\right)\right].$$

Hence, given the distributions of T_I and T_L, it is sufficient to calculate q, which we do now for the distributions considered earlier.

If $T_I \sim \text{Exp}(\gamma)$ and $T_L \sim \text{Exp}(\delta)$, then for $j \neq 1, 2$,

$$\text{P}\left(X_j + Y_j > t\right) = 1 - \int_0^t \delta e^{-\delta u}\left(1 - e^{-\gamma(t-u)}\right) du = \begin{cases} \frac{\delta}{\delta-\gamma}e^{-\gamma t} - \frac{\gamma}{\delta-\gamma}e^{-\delta t} & \text{if } \delta \neq \gamma; \\ (1+\gamma t)e^{-\gamma t} & \text{if } \delta = \gamma. \end{cases}$$

Thus

$$\begin{aligned} q &= 1 - \text{P}\left(\min_{j\neq 2}\left(X_j + Y_j\right) > X_2\right) = 1 - \int_0^\infty \delta e^{-\delta u}\text{P}\left(Y_1 > u\right)\left[\text{P}\left(X_3 + Y_3 > u\right)\right]^{n-2} du \\ &= \begin{cases} 1 - \int_0^\infty \delta e^{-\delta u} e^{-\gamma u}\left[\frac{\delta}{\delta-\gamma}e^{-\gamma u} - \frac{\gamma}{\delta-\gamma}e^{-\delta u}\right]^{n-2} du & \text{if } \delta \neq \gamma; \\ 1 - \int_0^\infty \gamma e^{-2\gamma u}(1+\gamma u)^{n-2} e^{-(n-2)\gamma u} du & \text{if } \delta = \gamma \end{cases} \\ &= \begin{cases} 1 - \frac{1}{(\delta-\gamma)^{n-2}}\sum_{k=0}^{n-2}\binom{n-2}{k}(-1)^k \frac{\delta^{n-k-1}\gamma^k}{\delta(k+1)+\gamma(n-k-1)} & \text{if } \delta \neq \gamma; \\ 1 - \sum_{k=0}^{n-2}\frac{(n-2)!}{(n-k-2)!}n^{-k-1} & \text{if } \delta = \gamma. \end{cases} \end{aligned}$$

If $T_I \equiv \iota$, and $T_L \sim \text{Exp}(\delta)$, then $\min_{j\neq 2}\left(X_j + Y_j\right) = Y_1 = \iota$, so

$$q = \text{P}(X_2 > \iota) = e^{-\delta\iota}.$$

For $\psi(\theta)$, results can no longer be obtained via symmetry, since the fates of different individuals are not independent. However, the distribution of the number of initial susceptibles who become infectious before the first removal occurs, W say, is sufficient, since in the all-or-nothing case

$$\psi(\theta) \to \sum_{j=0}^{n-1}\text{P}(W=j)\sum_{v=0}^{n-j-1} p_L(n-j-1, v)\left(\text{E}\left[e^{-\theta T_I}\right]\right)^{v+j+1}$$

and in the non-random case

$$\psi(\theta) \to \sum_{j=0}^{n-1}\text{P}(W=j)\left(\text{E}\left[e^{-\theta T_I}\right]\right)^{j+1}\left(\text{E}\left[e^{-b\theta T_I}\right]\right)^{n-j-1}.$$

If $T_I \equiv \iota$ and $T_L \sim \text{Exp}(\delta)$, then $W \sim \text{Bin}\left(n-1, 1-e^{-\delta\iota}\right)$.

If $T_I \sim \text{Exp}(\gamma)$ and $T_L \sim \text{Exp}(\delta)$, then before the first removal, when there are k infectives (and hence $n-k$ latents) the probability that the next event will be a latent becoming infective is $\frac{(n-k)\delta}{(n-k)\delta+k\gamma}$, and that it will be a removal is $\frac{k\gamma}{(n-k)\delta+k\gamma}$. Hence,

$$\text{P}(W=j) = \left(\prod_{k=1}^{j} \frac{(n-k)\delta}{(n-k)\delta+k\gamma}\right) \frac{(j+1)\gamma}{(j+1)\gamma+(n-j-1)\delta} \quad (j=0,1,\ldots,n-1),$$

where the product is 1 if $j=0$.

2.6.3 Isolation, vaccine-insensitive latents

In this case all the susceptibles will become infective at some point, though some or all of their infectious period may be experienced while the household is isolated. If $T_I \sim \text{Exp}(\gamma)$, then using the random-time change used in Section 2.3, it follows that the infectious intensity up until the kth removal is a sum of k independent $\text{Exp}(\gamma)$ random variables, i.e. it has a $\text{Gamma}(k,\gamma)$ distribution (so it has mean $k\gamma^{-1}$). (This holds for an arbitrary but specified latent period distribution – see Section 3.1 of Ball et al. [10]). The probability that the household is isolated at the kth removal is $p_k \prod_{j=1}^{k-1}(1-p_j)$ (where the product is 1 when $k=1$), and, setting $p_n = 1$ without loss of generality, the probability that the household is never isolated is $p_n \prod_{j=1}^{n-1}(1-p_j)$. Thus

$$R_* \to \frac{\lambda_G}{\gamma} \sum_{k=1}^{n} k p_k \prod_{j=1}^{k-1}(1-p_j),$$

and, for $\theta \geq 0$,

$$\psi(\theta) \to \sum_{k=1}^{n} p_k \left(\frac{\gamma}{\gamma+\theta}\right)^k \prod_{j=1}^{k-1}(1-p_j)\,.$$

2.6.4 Isolation, vaccine-sensitive latents

If $T_I \sim \text{Exp}(\gamma)$ and $T_L \sim \text{Exp}(\delta)$, for the all-or-nothing vaccine we have, as above, that the severity up until the kth removal has a Gamma(k, γ) distribution, but now, if $W = j$ and $L_V = v$, then there will be only $v + j + 1$ removals. Hence,

$$R_* \to \frac{\lambda_G}{\gamma}\left[\sum_{j=0}^{n-1}\text{P}(W=j)\sum_{v=0}^{n-j-1}p_L(n-j-1,v)\left(\sum_{k=1}^{j+v}kp_k\prod_{i=1}^{k-1}(1-p_i)\right.\right.$$
$$\left.\left.+(j+v+1)\prod_{i=1}^{j+v}(1-p_i)\right)\right]$$

and, for $\theta \geq 0$,

$$\psi(\theta) \to \sum_{j=0}^{n-1}\text{P}(W=j)\sum_{v=0}^{n-j-1}p_L(n-j-1,v)\left(\sum_{k=1}^{j+v}p_k\left(\frac{\gamma}{\gamma+\theta}\right)^k\prod_{i=1}^{k-1}(1-p_i)\right.$$
$$\left.+\left(\frac{\gamma}{\gamma+\theta}\right)^{j+v+1}\prod_{i=1}^{j+v}(1-p_i)\right).$$

If the vaccine response is non-random, then the random time-change cannot be used in a similar way here, since the two types of infectious individuals contribute differently to the infectious intensity. One can of course, though, use the results from Section 2.3.5, changing the initial conditions to $n-1$ latents and 1 infective.

2.6.5 Isolation at the first removal

Here we assume arbitrary but specified distributions for latent and infectious periods. With isolation at the first removal the initial infective will contribute $\min_j(X_j + Y_j)$ to the severity, while from the initial susceptibles, individual i will contribute $\min_j(X_j + Y_j) - X_i$ if $X_i < \min_{j\neq i}(X_j + Y_j)$ and zero otherwise. Hence, by symmetry,

$$R_* \to \lambda_G\left(\text{E}\left[\min_j(X_j+Y_j)\right] + (n-1)\text{E}\left[\left(\min_j(X_j+Y_j) - X_2\right)1_{\{X_2<\min_{j\neq i}(X_j+Y_j)\}}\right]\right).$$

If $T_I \sim \text{Exp}(\gamma)$ then note, from Section 2.3.5, that R_* and $\psi(\theta)$ are independent of λ_L.

In the $T_I = \iota$, $T_L \sim \text{Exp}(\gamma)$ case we have that $\min_j (X_j + Y_j) = Y_1 = \iota$, and hence $\text{E}[\min_j (X_j + Y_j)] = \iota$, while

$$\begin{aligned}\text{E}\left[\left(\min_j (X_j + Y_j) - X_2\right) 1_{\{X_2 < \min_{j\neq i}(X_j+Y_j)\}}\right] &= \int_0^\iota (\iota - t)\delta e^{-\delta t}\, dt \\ &= \iota - \delta^{-1}\left(1 - e^{-\delta\iota}\right).\end{aligned}$$

Further, since $\min_j(X_j + Y_j) = \iota$, the contributions of individuals to the infectious intensity are independent, and hence by symmetry

$$\psi(\theta) \to e^{-\iota\theta}\left(q + \text{E}\left[e^{-\theta(\iota - X_2)} 1_{\{X_2<\iota\}}\right]\right)^{n-1},$$

where, as before, $q = e^{-\delta\iota}$ is the probability that a given susceptible is latent at the time of the first removal, and, for $\theta \geq 0$,

$$\begin{aligned}\text{E}\left[e^{-\theta(\iota - X_2)} 1_{\{X_2<\iota\}}\right] &= \int_0^\iota e^{-\theta(\iota - t)}\delta e^{-\delta t} dt \\ &= \begin{cases} \frac{\delta}{\delta - \theta}\left(e^{-\iota\theta} - e^{-\iota\delta}\right) & \text{if } \delta \neq \theta; \\ \theta\iota e^{-\iota\theta} & \text{if } \delta = \theta. \end{cases}\end{aligned}$$

2.7 Numerical illustrations

In this section we illustrate the theory using some parameter estimates derived from data on an outbreak of *variola minor* (a virus which causes a mild form of smallpox) in São Paulo in 1956 (see Ball and Lyne [7]). Specifically, unless otherwise indicated, we set $\lambda_L = 0.3821$, $\lambda_G = 1.4159$, $a = 0.1182$, $b = 0.8712$, $\gamma = \iota = 1$ and $\delta = \frac{1}{\eta} = \frac{1}{4}$ (i.e. $\text{E}[T_L] = 4\text{E}[T_I]$, a reasonable assumption for smallpox; see Kaplan et al. [30].)

Figure 2.1 shows the impact of different vaccine response models on the probability of a global epidemic, p_G. Here $T_I \sim \text{Exp}(\gamma)$, $T_L \sim \text{Exp}(\frac{1}{4})$, the household size $n = 5$ (similar results hold for different n) and $R_* = 2$ in the absence of intervention. The parameters in the all-or-nothing and non-random vaccine models are matched using

$\varepsilon_S = 1 - ab = 0.8970$ and $\varepsilon_L = 1 - b = 0.1288$, these equations arising respectively from the vaccine efficacy measures $1 - \mathrm{E}[AB]$ and $1 - \mathrm{E}[AB]/\mathrm{E}[A]$ which describe efficacy in terms of susceptibility-infectivity and infectivity (see Becker et al. [19]). Six different intervention models are considered: (i) no intervention, (ii) vaccine-insensitive latents (VIL) without isolation, (iii) vaccine-sensitive latents (VSL) without isolation, (iv) VIL with isolation (here we assume $p_1 = 0$, $p_2 = 1$, so isolation occurs at the second removal within a household), (v) VSL with isolation and (vi) isolation at the first removal within a household ($p_1 = 1$).

All-or-nothing and non-random vaccine response models give similar results. A key point to note from Figure 2.1 is that the differences between the plots for all-or-nothing and non-random vaccine responses are minor. When compared with the perfect vaccine there is a notable difference in the vaccine-sensitive latent models which arises because $\varepsilon_L = 1$ for the perfect vaccine, but only 0.1288 for the imperfect vaccine. Figures 2.2 and 2.3 provide more comparisons of the all-or-nothing and non-random vaccine models, again with $n = 5$ and with parameters matched as for Figure 2.1. Again $T_I \sim \mathrm{Exp}(1)$, $T_L \sim \mathrm{Exp}(\frac{1}{4})$, while latents are assumed to be vaccine-insensitive. Figure 2.2 illustrates that the two models yield very similar values of R_* when the all-or-nothing model is chosen to match a given non-random model. Conversely, with ε_S fixed in the all-or-nothing model and (a, b) chosen such that $\varepsilon_S = 1 - ab$, markedly different R_* values can be obtained as illustrated in Figure 2.3. However, it should be noted that such discrepancies occur when b is small, so that a exceeds unity, which may be unrealistic in many scenarios. Assuming constant infectious periods yields similar plots. Finally, note that Figures 2.2 and 2.3 also give some indication of the impact of uncertainty regarding the parameters of the vaccine action models. For example, the parameter b is often hard to estimate precisely from outbreak data, but for the numerical examples

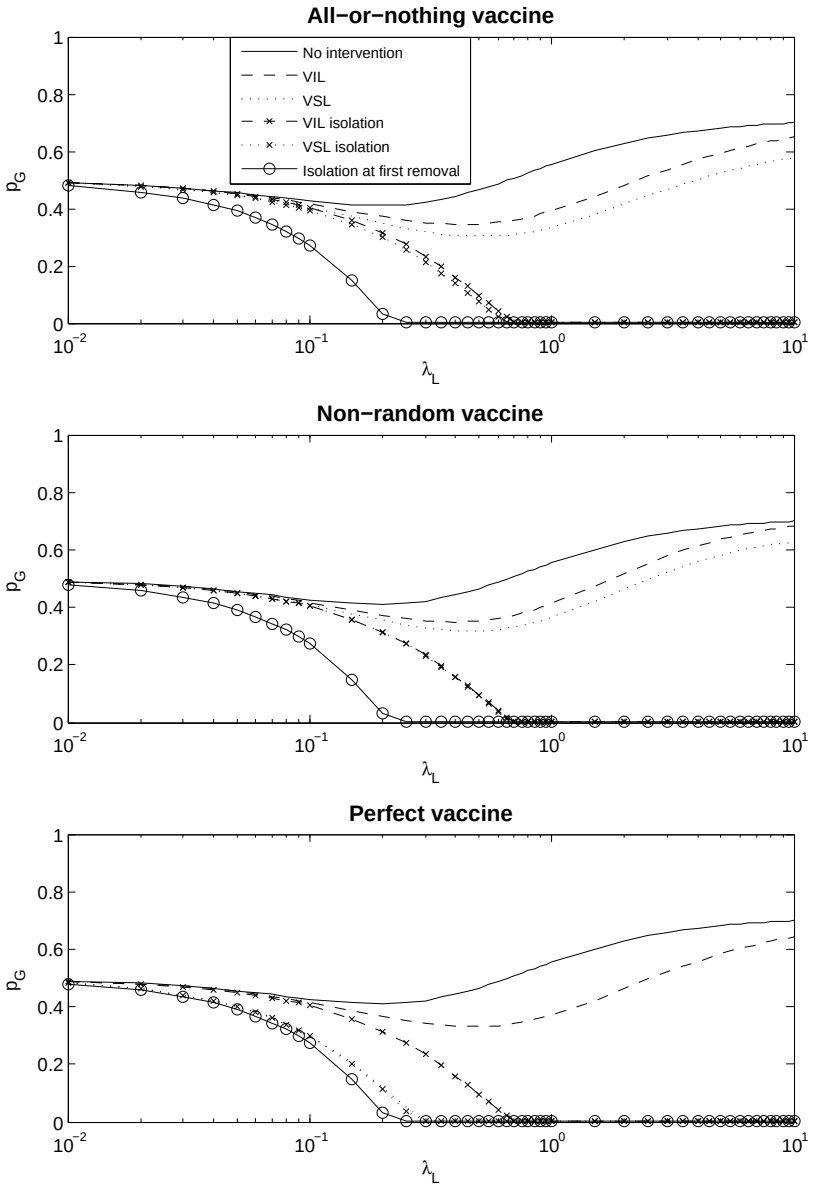

Figure 2.1: p_G as λ_L and λ_G vary so that $R_* = 2$ without intervention.

presented here it is clear that small changes in the value of b do not have a substantial impact on R_*.

A good isolation policy is more effective than vaccination. From Figure 2.1, we see that vaccination can reduce p_G and this is reduced further when latents are vaccine-sensitive, but isolation is much more effective (and with isolation, it makes little difference whether or not latents are vaccine-sensitive). The three plots in Figure 2.4 ((i) $\varepsilon_L = 0$, (ii) $\varepsilon_L = \frac{1}{2}\varepsilon_S$ and (iii) $\varepsilon_L = \varepsilon_S$) illustrate such findings in more detail. We assume iso-

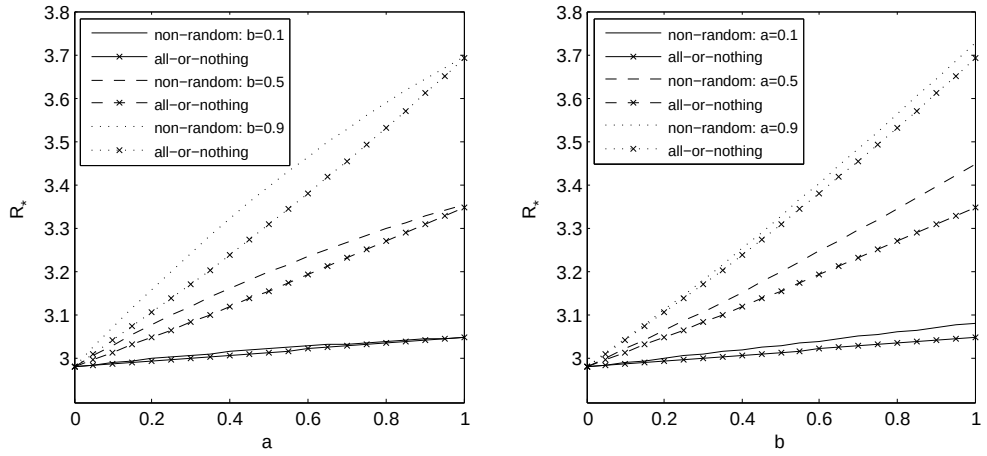

Figure 2.2: R_* varying with a and b, with matched efficacy all-or-nothing cases.

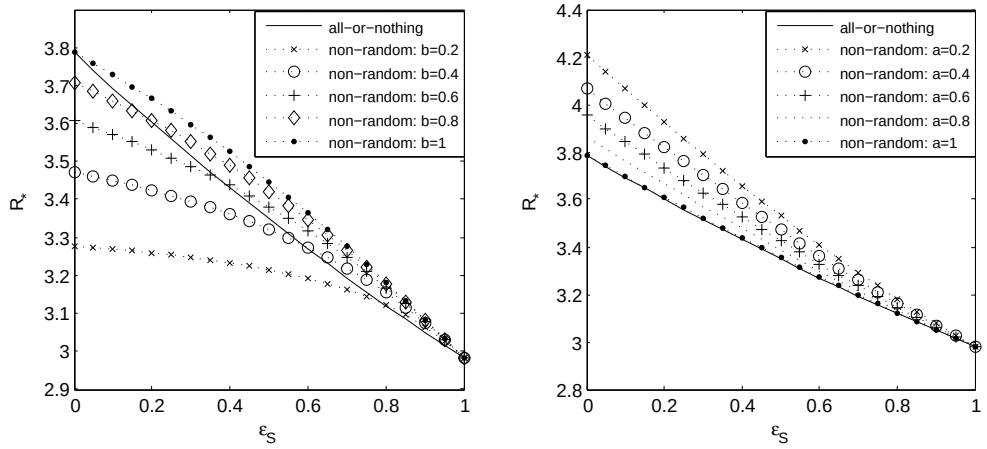

Figure 2.3: R_* varying with ε_S, with matched efficacy non-random cases.

lation at the ith removal ($i = 1, 2, 3, 4$), i.e. $p_i = 1$ and $p_j = 0$ for $j \neq i$. Note that with vaccine-insensitive latents and isolation at the second removal, ε_S has no effect on R_*, since T_I is exponentially distributed. This is because a second removal will only occur if there is at least one latent or infective immediately after the first removal, and thus the occurrence of the second removal is independent of the vaccine. This fact, combined with the Markov property, yields the flat curve. As isolation comes later, the vaccine has more of an effect. On the other hand, the more effective the vaccine is, the less difference isolation makes. However, it is clear that earlier isolation schemes offer significant improvement.

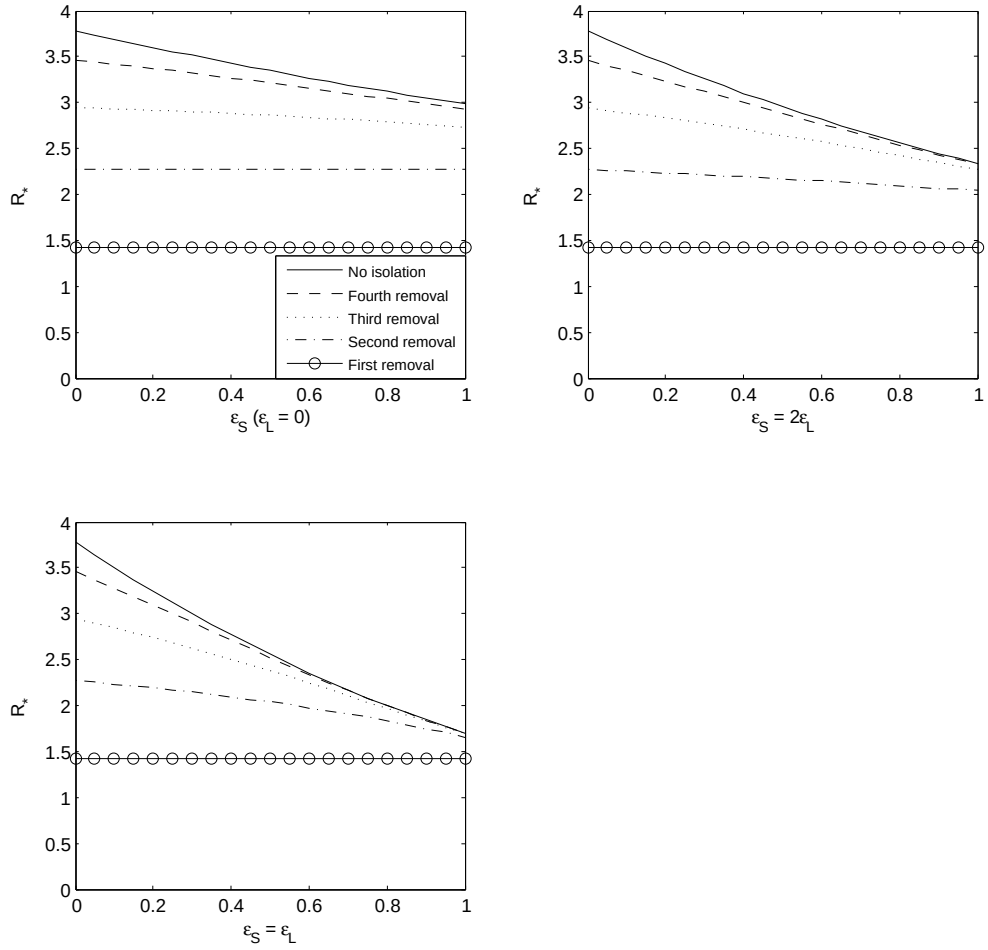

Figure 2.4: R_* varying with ε_S for various isolation policies and assumptions about ε_L.

Different infectious period distributions can yield materially different results. The effect of the choice of infectious period distribution on the results is illustrated in Figure 2.5. We compare exponential (E) and constant (C) infectious periods with the same mean (distributions are listed in the form latent period/infectious period). If latents are vaccine-sensitive latents then we set $\varepsilon_S = \varepsilon_L$. We might expect that constant infectious periods generally mean the epidemic is 'worse' (since exponential distributions have more probability mass below the mean than above it), but there is clearly a significant difference when we change the infectious period; particularly for p_G. The difference is

noticeable even as the household size gets much larger than 5, as it is here. The choice of latent period distribution has much less of an effect.

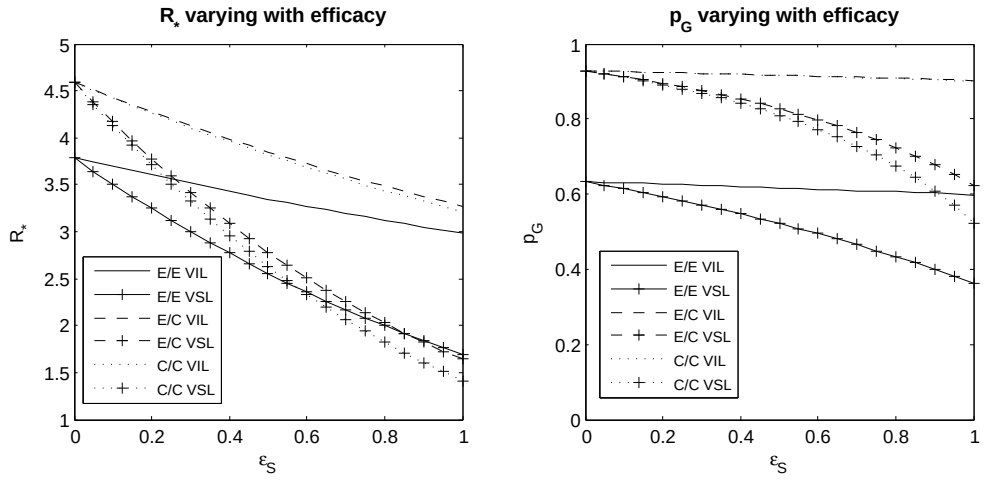

Figure 2.5: R_* and p_G varying with ε_S in VIL ($\varepsilon_L = 0$) and VSL ($\varepsilon_L = \varepsilon_S$) cases for different latent/infectious period distributions.

R_* alone is not sufficient to summarise the potential for an epidemic. The considerable variability seen in the plots of Figure 2.1 highlights the fact that R_* alone does not adequately summarise the epidemic model. Further, while with isolation we observe monotone behaviour, in that R_* decreases with increasing λ_L, this is not the case without isolation for reasons we now outline. Consider first the case of no intervention. If the mean of the offspring distribution of a branching process is held fixed then broadly the extinction probability increases with the variance of the offspring distribution. This phenomenon has been noted previously, see, for example, Becker and Marschner [15] and Lloyd-Smith et al. [36]. Thus, for fixed R_*, p_G is least when there is greatest variability in the infectious intensity generated by a single-household epidemic. From Figure 2.1, p_G is least when $\lambda_L \approx 0.25$, i.e. when the single-household epidemic is at its 'internal' threshold. This is intuitively plausible since when the single-household epidemic is well above its internal threshold most of the household will become infected and when it is well below its internal threshold only the index case in the household is

likely to become infected, both of which lead to less variability in infectious intensity. Moreover, there is less variability in infectious intensity in the limit $\lambda_L \to \infty$ than in the limit $\lambda_L \to 0$, since the whole household is infected in the former and only the index case in the latter, which explains why p_G is greatest in the limit $\lambda_L \to \infty$. A similar phenomenon is observed for the vaccination-only models. However, the behaviour of the models with isolation is less affected by λ_L, so as λ_L increases the decrease in λ_G (so that $R_* = 2$) dominates and p_G drops to 0. There are also clear qualitative differences between the R_* and p_G plots of Figure 2.5, reinforcing the need for caution in relying on only one of these quantities as a summary measure.

2.8 Concluding comments

We have derived expressions for R_* and p_G under various assumptions regarding the choice of latent and infectious period distribution, vaccine response model and isolation scheme. Our numerical illustrations indicate that R_* and p_G can be materially affected by using exponential infectious periods (which are implicitly assumed in most deterministic models) as opposed to constant infectious periods (which are often more realistic in practice). Similarly, R_* and p_G can be affected by isolation schemes and a vaccine which is effective for latent individuals. In contrast, suitably matched all-or-nothing and non-random vaccine response models seem to yield similar values of R_* and p_G.

We have restricted attention to populations partitioned into households of equal size, but the expressions obtained can be easily extended to unequal household sizes (cf. Ball et al. [9]) as follows. Suppose that, for $k = 1, 2, \ldots$, the proportion of households containing k susceptibles at the start of an epidemic is α_k. For $k = 1, 2, \ldots$, the probability

that a given global contact is with an individual residing in a household of size k is $\tilde{\alpha}_k = k\alpha_k / \sum_{i=1}^{\infty} i\alpha_i$, since such a contact is with an individual chosen uniformly at random from the entire population. It then follows that for this model,

$$R_* = \sum_{k=1}^{\infty} \tilde{\alpha}_k R_*^{(k)}, \quad f(s) = \sum_{k=1}^{\infty} \tilde{\alpha}_k f^{(k)}(s),$$

where, for $k = 1, 2, \ldots$, $R_*^{(k)}$ and $f^{(k)}(s)$ are R_* and $f(s)$, respectively, for a population partitioned into households of equal size k. If the epidemic is initiated by one individual, chosen uniformly at random from the population, becoming infected then p_G is $1 - \tau$, where τ is the smallest root of $f(s) = s$ in $[0, 1]$. (Note that $\tau = 1$ if $R_* \leq 1$). If instead the initial infective is known to reside in a household of size k, conditioning on the size of the first generation in the approximating branching process shows that the p_G is $1 - f^{(k)}(\tau)$.

In this chapter, intervention has been considered only at a local level: when diagnosed cases appear, intervention is taken only within the household of the diagnosed individual. Such local intervention methods may not be sufficient to prevent a global epidemic, in which case we need to reduce λ_G. One such method is by contact tracing, which involves directing intervention at named contacts of a diagnosed individual. We consider this in a homogeneously-mixing population model in Chapter 3 and in a household-based model in Chapter 4. Another extension one may consider is to have the vaccination at a later removal, which would be analytically tractable in the case of exponentially distributed infectious periods, but not for constant infectious periods. Further, in the models here there is one latent period, with latents being vaccine-sensitive or vaccine-insensitive. The model and methods used here could be adapted to incorporate individuals experiencing two latent periods after being infected: a vaccine-sensitive period, followed by a vaccine-insensitive one. The assumption of instanta-

neous vaccine response is perhaps unrealistic for most real-life vaccines, though it provides a bound by considering the best-possible scenario. A delay can be introduced into the vaccine response but that would make the model less tractable. However, the theory can be extended to consider other forms of intervention for which this assumption is more reasonable, for example the use of antivirals.

CHAPTER 3

Contact tracing

3.1 Introduction

Contact tracing is a form of intervention in which diagnosed individuals name the other individuals they have been in contact with and these named contacts are traced and treated in some manner. The idea is that, rather than randomly directing intervention, the treatment is given to individuals who are likely to have become infected, before they have been diagnosed. It has been studied in a stochastic environment by, for example, Becker et al. [18], Müller et al. [38] and Klinkenberg et al. [34]. However, there are difficulties in making analytical progress with contact tracing, particularly since infected individuals with the same infector do not act independently of one another, as noted by Müller et al. [38]. Becker et al. [18] approximated by assuming that the traced contact was infected at the beginning of their infector's infectious period. Except for a special case where individuals make their infectious contacts instantaneously, Klinkenberg et al. [34] used an infinite-size next generation matrix to obtain the threshold parameter, having to truncate it to obtain numerical results, or they used simulations. Müller et al. [38] had to study a reproduction number for the ith gener-

ation (of infected individuals) and studied the behaviour as $i \to \infty$. Generally, one of the attractive outcomes of modelling epidemics stochastically, the ability to affix a probability to the possibility of a major outbreak, is not examined due to the increased difficulty.

In this chapter, we look at two contact tracing models, in which, on diagnosis, individuals name their infections, and a named individual is found and isolated; if they have not ended their infectious period (i.e. they are not yet symptomatic) when this happens they are considered *traced*. The first, which we refer to as *single-step* tracing, assumes that traced individuals do not ever name contacts themselves. This differs from the definition used by, for example, Klinkenberg et al. [34] - in their definition of single-step tracing, a traced individual may name their contacts, but only once they have been diagnosed. The second, which we refer to as *iterative* tracing, assumes that traced individuals can name contacts, and that they do this immediately upon being traced. As such, this is in line with the definition of iterative (or 'recursive') tracing used by Klinkenberg et al. [34] and Müller et al. [38].

We derive results both with and without a delay in the tracing process. This delay may be considered the amount of time it takes to find and isolate a traced individual. The modelling of a specific tracing delay has been used by Klinkenberg et al. [34], who assumed this delay is fixed, while others, such as Shaban et al. [42], assumed that the time to detect an infected individual (from infection) incorporates a delay in tracing their contacts. As such, an implicit assumption about these delays is that individuals with the same infector experience a mutual delay. We assume that delays are independent instead, although we do note when results still hold if these delays were assumed to be the same.

We examine the process of unnamed individuals embedded in the full process, and use this to obtain R_U, a threshold parameter that can be used to determine whether, in a large population, the outbreak will be minor (infecting just a few individuals, effectively becoming 'extinct') or major (infecting a reasonably deterministic proportion of the population). To calculate R_U, an approach of looking at generations of named individuals is used, akin to that used by Klinkenberg et al. [34]. We calculate this for different distribution choices for infectious and latent periods and tracing delays. Notably, we show that in the iterative tracing case, without latent periods or delays, the threshold parameter R_U (and hence whether the process is super- or subcritical) depends upon the infectious period only through its mean, and not through the specific choice of distribution. Where possible, expressions for the probability of extinction are obtained, otherwise the framework allows for simulations to be used. In the case of Exponentially-distributed infectious periods, we suggest two approximations that make infected individuals independent, one simple and another more sophisticated (preserving the generational nature of named individuals), and examine the effect of these approximations.

This chapter is structured as follows. In Section 3.2, the epidemic and contact tracing models are introduced. Then, a modified birth-death process and how it is used to determine the threshold behaviour are described. In the following two sections, the threshold parameter is determined under single-step tracing, with constant infectious periods in Section 3.4 and Exponential in Section 3.5. In Section 3.6, the threshold parameter is obtained under iterative tracing without latent periods or tracing delays, for an arbitrary infectious period. This is extended to include latent periods and delays in the Exponential infectious period case in Section 3.7. Numerical results are used to illustrate the theory in Section 3.8, with simulations also used to test the theory, and

some concluding comments are given in Section 3.9.

3.2 Background

3.2.1 Epidemic and contact tracing models

We consider an SEIR (Susceptible $\rightarrow$ Exposed $\rightarrow$ Infective $\rightarrow$ Removed) epidemic spreading amongst a homogeneously mixing closed population of size N, with a contract tracing scheme applied to reduce spread. At any time, each individual in the population is in one of four states: susceptible, exposed (i.e. latent), infective or removed. Initially a small number of individuals are infectives and the rest are susceptible. A susceptible individual becomes a latent individual if he/she makes contact with an infective in a manner described below. A latent individual remains latent for a period of time distributed according to a random variable T_L, having an arbitrary but specified distribution (i.e. no assumption is made about the form of its distribution, but the distribution has to be known), at the end of which he/she becomes infective. An infective individual remains infectious for a period of time distributed according to a random variable T_I, having an arbitrary but specified distribution, and then becomes removed. Contacts between two given individuals in the population occur at times given by the points of a homogeneous Poisson process with rate $\frac{\lambda}{N}$. Once removed, an individual no longer plays a part in the epidemic process. The epidemic ends when there are no more latent or infective individuals left in the population. All of the Poisson processes, and the random variables describing latent and infectious periods, are assumed to be mutually independent.

This epidemic incorporates a contact tracing scheme as follows. Upon removal, an indi-

vidual (i.e. an infector) names each of the individuals they infected (i.e. their infectees) independently with probability p, and named infectees are removed after a delay period of time distributed according to a random variable T_D, having an arbitrary but specified distribution. An individual whose removal is a result of contact tracing (and not the natural end of their infectious period) is considered traced. Note that an individual is named, but not traced, if their infectious period ends during their infector's infectious period or the associated tracing delay. The naming process and random variables describing delay periods are assumed independent of the Poisson processes, and random variables describing latent and infectious periods. Generally, we assume that the random variables describing the delay periods of all individuals with the same infector (i.e. siblings) are mutually independent. However, in some cases it will be shown that results hold also when siblings are assumed to have delay periods of the same length. We consider two forms of tracing, (i) single-step, in which we assume traced individuals can not name their contacts, and (ii) iterative, in which we assume traced individuals can name their contacts.

3.3 Threshold behaviour and a modified birth-death process

If the population size N is large, then during the early stages of the epidemic, there is only a small probability that an infective makes contact with an already-infected individual. Thus we can approximate the early stages of the epidemic by a process in which all of an infective's contacts are made with susceptible individuals. In this approximation, the process of infected individuals follows a modified birth-death process.

This modified birth-death process, with births corresponding to new infections and deaths corresponding to removals, is as follows. Individuals give birth at rate λ over

their active lifetime which has a natural length distributed as T_I, which begins after a latent period distributed as T_L. Further, when an individual dies naturally, each of its offspring are named, independently and with probability p, and are traced after some delay distributed as T_D (the delays of siblings are independent of each other). An individual who is still alive at the time they are traced dies unnaturally. Note that, in the single-step case, offspring of individuals who die unnaturally necessarily die naturally. In the epidemic context, active lifetimes correspond to infectious periods, natural deaths correspond to untraced removals and unnatural deaths correspond to traced removals.

The threshold behaviour of this modified birth-death process can be obtained by considering the embedded single-type discrete-time Galton-Watson process describing unnamed individuals, in which the offspring of a given individual are either (a) their immediate unnamed offspring, or (b) unnamed descendants who are separated from the given individual, in the family tree, only by named individuals.

To distinguish between the processes and for conciseness, we shall refer to the modified birth-death process as the MBDP and the Galton-Watson process as the GWP.

Let a named individual who is separated from an unnamed individual in the family tree of the MBDP by $k-1$ named individuals be called a type-k individual. Hence, type-1 individuals are the named immediate offspring of unnamed individuals, type-2 individuals are the named immediate offspring of the named immediate offspring of unnamed individuals, and so on. Type-0 individuals are unnamed individuals. We shall refer to the type-k individuals who are separated from a given unnamed individual in the family tree only by named individuals as that unnamed individual's immediate type-k descendants.

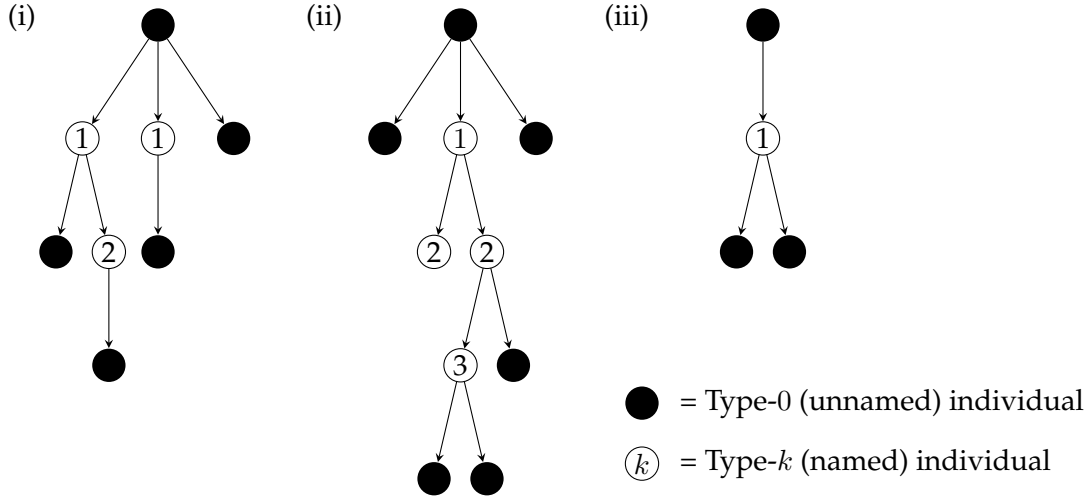

Figure 3.1: Some example realizations of R.

In (i) $R = 4$, $R_1 = 2$, $R_2 = 1$, $R^{(0)} = 1$, $R^{(1)} = 3$, $R^{(2)} = 4$.

In (ii) $R = 5$, $R_1 = 0$, $R_2 = 1$, $R_3 = 2$, $R^{(0)} = 2$, $R^{(1)} = 2$, $R^{(2)} = 3$, $R^{(3)} = 5$.

In (iii) $R = 2$, $R_1 = 2$, $R^{(0)} = 0$, $R^{(1)} = 2$.

To analyse the MBDP we focus attention on the offspring random variable, R say, in the GWP, by obtaining expressions for its mean, $R_U = E[R]$, which we call a type-reproduction number, following Heesterbeek and Roberts [27]. For some example realizations of R, see Figure 3.1. Standard results from branching process theory tell us that the GWP will die out with probability 1 if and only if $R_U \leq 1$, and that if $R_U > 1$ the extinction probability of the GWP, p_E say, is the smallest solution of $s = H(s)$ in $(0, 1)$, where $H(s) = \mathrm{E}\left[s^R\right]$.

We obtain R_U by considering a typical unnamed individual, A say, in the MBDP. Let R_i be the total number of unnamed immediate offspring of all the immediate type-i ($i = 1, 2, \ldots$) descendants of A and $R_{U,i} = \mathrm{E}\left[R_i\right]$. Let $R^{(k)}$ be the total number of unnamed immediate offspring of A and all of its immediate descendants of up to and

including type-k, then for $k = 1, 2, \ldots$,

$$R^{(k)} = R^{(0)} + \sum_{i=1}^{k} R_i,$$

noting that $R^{(0)}$ is the number of unnamed immediate offspring of A. Examples of R_i and $R^{(k)}$ are given in Figure 3.1. We have that $R^{(k)} \uparrow R$ as $k \to \infty$, and so by the monotone convergence theorem,

$$R_U = \lim_{k\to\infty} R_U^{(k)},$$

where $R_U^{(k)} = E\left[R^{(k)}\right]$.

Note that while we have shown that if $R_U^{(k)}$ has finite limit then this limit is R_U, it may be the case that as $k \to \infty$, $R_U^{(k)} \to \infty$, in which case $R_U = \infty$. In the sequel we shall derive this threshold parameter under two different distributions for the life-time/infectious period: (i) constant, and (ii) exponential. Further, we obtain necessary and sufficient conditions for R_U to be finite.

3.3.1 Notation

The most important parameters and functions appearing throughout this chapter are listed in Table 3.1, along with brief definitions.

3.4 Single-step tracing, constant infectious period

Suppose that the infectious period has a fixed value ι, i.e. $T_I \equiv \iota$.

3.4.1 Calculation of R_U

Consider again our typical unnamed individual in the MBDP, A, who gives birth to unnamed individuals over their lifetime at rate $\lambda(1-p)$. Thus, $R^{(0)} \sim$ Poisson$(\lambda(1-p)\iota)$

Table 3.1: List of important parameters and functions for Chapter 3.

parameter	description
N	number of individuals in the population
λ/N	individual-to-individual contact rate
p	probability of a contact being named, given that the infector is allowed to name contacts
ι	length of constant infectious period
γ	rate parameter for exponentially distributed infectious period (i.e. mean=$\frac{1}{\gamma}$)
ξ	rate parameter for exponentially distributed tracing delay (i.e. mean=$\frac{1}{\xi}$)
$\phi_L(\theta)$	moment-generating function of latent period ($\theta \geq 0$)
R	offspring random variable of embedded Galton-Watson process of unnamed individuals
R_U	expected value of R, type-reproduction number
$R^{(k)}$	total number of unnamed immediate offspring of an unnamed individual and their immediate descendants of up to and including type-k
$R_U^{(k)}$	expected value of $R^{(k)}$
R_i	total number of unnamed immediate offspring of all the immediate type-i descendants of a unnamed individual
p_E	probability of extinction

and

$$\mathrm{E}\left[R^{(0)}\right] = \lambda(1-p)\iota.$$

Similarly, if the number of type-1 (i.e. named immediate) descendants of A is N_1, then $N_1 \sim \text{Poisson}(\lambda p\iota)$, and if the number of offspring contributed to $\sum_{i=1}^{\infty} R_i$ by the ith (unordered) type-1 descendant of A is Z_i, then

$$\sum_{i=1}^{\infty} R_i = \sum_{i=1}^{N_1} Z_i,$$

where the sum on the right is zero if $N_1 = 0$. Conditional on $N_1 = n$, the birth times of these n individuals can be obtained by sampling n independent $\mathrm{U}(0, \iota)$ random variables, hence the Z_i are independent and identically distributed with common distribution Z, say, and so

$$\begin{aligned}\mathrm{E}\left[\sum_{i=1}^{\infty} R_i\right] &= \sum_{n=0}^{\infty} \mathrm{P}\left(N_1 = n\right) n\mathrm{E}\left[Z_1\right] \\ &= \lambda p\iota \mathrm{E}[Z].\end{aligned}$$

Thus

$$R_U = \mathrm{E}[R] = \lambda(1-p)\iota + \lambda p\iota \mathrm{E}[Z], \tag{3.4.1}$$

and it suffices to derive $\mathrm{E}[Z]$, which we do for separate cases in the later parts of this subsection.

Note that the Z_i depend upon the delays experienced by the corresponding name descendants of A, but these have been assumed to be independent. If we were to assume that these individuals all experience the *same* delay then the Z_i would no longer be independent, however the above results would be unchanged due to the linearity of expectations, i.e. R_U is the same whether sibling units experience independent (and identically distributed) or the same delays.

3.4.2 Calculation of extinction probability

If the number of immediate offspring of A is M, then $M \sim \text{Poisson}(\lambda\iota)$. Conditional on $M = k$, the birth times of these k individuals can be obtained by sampling k independent $\text{U}(0, \iota)$ random variables, and hence the contributions to R of these k individuals are independent and identically distributed. An unnamed individual (occurring with probability $1-p$) will contribute just themselves to R, while the ith (unordered) named (probability p) offspring will have a contribution Z_i distributed as Z, and hence

$$\begin{aligned}
\text{E}\left[s^R \middle| M = k\right] &= \text{E}\left[\prod_{i=1}^{k}(1-p)s + ps^{Z_i}\right] \\
&= \left[(1-p)s + p\text{E}\left[s^Z\right]\right]^k \\
&= \left[(1-p)s + pG(s)\right]^k,
\end{aligned}$$

since Z_i are independent and identically distributed with common distribution Z, and where $G(s) = \text{E}\left[s^Z\right]$. Hence

$$\begin{aligned}
H(s) &= \sum_{k=0}^{\infty} \frac{e^{-\lambda\iota}(\lambda\iota)^k}{k!} \text{E}\left[s^R \middle| M = k\right] \\
&= \sum_{k=0}^{\infty} \frac{e^{-\lambda\iota}(\lambda\iota)^k}{k!} \left[(1-p)s + pG(s)\right]^k \\
&= e^{-\lambda\iota} \sum_{k=0}^{\infty} \frac{1}{k!} \left[\lambda\iota\left((1-p)s + pG(s)\right)\right]^k \\
&= \exp\left\{-\lambda\iota\left(1 - (1-p)s - pG(s)\right)\right\}, \qquad (3.4.2)
\end{aligned}$$

which we can use to calculate p_E, and it suffices to derive $G(s)$, which we do for separate cases in the remainder of this subsection.

Note that if the delays of sibling units were assumed to be the same, the Z_i would become dependent, and since they appear here in a nonlinear expression, the above results would not hold.

3.4.3 No latent period, no delay

Suppose first that $T_L \equiv T_D \equiv 0$, i.e. individuals experience neither a latent period nor a delay.

In this case, all type-1 individuals must die unnaturally (since their parents necessarily predecease them as the lifetimes are all equal), and hence all their immediate offspring are unnamed (and therefore $R_i = 0$ for $i > 1$). Hence, if we consider a typical type-1 descendant, B, of A, born at a time V from the end of their parent's lifetime, then

$$V \sim \mathrm{U}(0, \iota)$$

and their contribution to R_1 has a Poisson(λV) distribution. Thus

$$\begin{aligned} \mathrm{E}[Z] &= \int_{v=0}^{\iota} \frac{1}{\iota} \lambda v \, dv \\ &= \frac{\lambda \iota}{2}, \end{aligned}$$

and therefore, using Eqn. (3.4.1),

$$R_U = \lambda(1-p)\iota + \frac{\lambda^2 p \iota^2}{2},$$

while

$$\begin{aligned} G(s) &= \int_0^{\iota} \frac{1}{\iota} \sum_{j=0}^{\infty} \frac{e^{-\lambda v}(\lambda v)^j}{j!} s^j \, dv \\ &= \int_0^{\iota} \frac{1}{\iota} e^{-\lambda v(1-s)} \, dv \\ &= \frac{1}{\lambda \iota (1-s)} \left(1 - e^{-\lambda \iota (1-s)}\right), \end{aligned}$$

and therefore, using Eqn. (3.4.2),

$$H(s) = \exp\left\{-\lambda \iota \left(1 - (1-p)s - \frac{p}{\lambda \iota (1-s)} \left(1 - e^{-\lambda \iota (1-s)}\right)\right)\right\}.$$

3.4.4 With latent period and delay

Let the distributions of T_L and T_D be arbitrary. We revisit our typical unnamed individual A and their typical named immediate offspring B, and recall that B is born at time length V before the end of A's lifetime. Suppose the latent period of B is $T_{L,B}$ and the delay associated with B is $T_{D,B}$.

The length of time after B's birth that B can be traced is $V + T_{D,B}$. However, for the first time $T_{L,B}$ time units, B is not active, i.e. they cannot have offspring over this period. After this latent period they are active until the end of their natural active lifetime or they are traced, whichever comes first. Hence, B's actual active lifetime is $\min\{\iota, \max\{0, V + T_{D,B} - T_{L,B}\}\}$. Note that if $\iota > V + T_{D,B} - T_{L,B}$, then B is traced and hence all their offspring are unnamed. On the other hand, if $\iota \leq V + T_{D,B} - T_{L,B}$, then B is not traced and hence lives a full active lifetime and can name individuals.

We shall now break this down into four cases: (i) $T_{D,B} - T_{L,B} > \iota$, (ii) $0 \leq T_{D,B} - T_{L,B} \leq \iota$, (iii) $-\iota \leq T_{D,B} - T_{L,B} < 0$ and (iv) $T_{D,B} - T_{L,B} < -\iota$.

In case (i), clearly $V + T_{D,B} - T_{L,B} > \iota$, so B's active lifetime is ended naturally, and hence B can name contacts made over a full natural lifetime, hence B's offspring has the same distribution as that of a type-0 individual's. We shall label the event that $T_{D,B} - T_{L,B} > \iota$ as C_1.

In case (ii), we have two subcases (a) $V < \iota - T_{D,B} + T_{L,B}$, in which case B is traced (we label this case as C_2, and (b) $V \geq \iota - T_{D,B} + T_{L,B}$, in which case B is untraced and has a full active lifetime (we label this case as C_3).

In case (iii), $V + T_{D,B} - T_{L,B} < \iota$ so B is traced. We have two subcases (a) $V \geq T_{L,B} - T_{D,B}$ in which case B can have offspring (we label this case as C_4), and (b)

$V < T_{L,B} - T_{D,B}$ in which case B has no offspring (we label this case as C_5).

In case (iv), $V + T_{D,B} - T_{L,B} < 0$ so B can have no offspring. We label the event that $T_{D,B} - T_{L,B} < -\iota$ as C_6.

Let the effective delay be $T_{D-L} = T_D - T_L$ (i.e. the delay minus the latent period) and let μ_{D-L} be the distribution measure of T_{D-L}.

Putting this all together,

$$\mathrm{E}[Z] = \sum_{k=1}^{4} \mathrm{E}\left[Z\mathbf{1}_{C_k}\right],$$

and we have that

$$\begin{aligned}
\mathrm{E}\left[Z\mathbf{1}_{C_1}\right] &= \int_{x=\iota}^{\infty} R_U \, d\mu_{D-L}(x) \\
&= \mathrm{P}\left(T_{D-L} > \iota\right) R_U, \\
\mathrm{E}\left[Z\mathbf{1}_{C_2}\right] &= \int_{x=0}^{\iota} \int_{v=0}^{\iota-x} \frac{1}{\iota}\lambda(v+x) \, dv \, d\mu_{D-L}(x) \\
&= \frac{\lambda\iota}{2}\mathrm{P}\left(0 \le T_{D-L} \le \iota\right) - \frac{\lambda}{2\iota}\mathrm{E}\left[T_{D-L}^2 1_{\{0 \le T_{D-L} \le \iota\}}\right], \\
\mathrm{E}\left[Z\mathbf{1}_{C_3}\right] &= \int_{x=0}^{\iota} \mathrm{P}(V > \iota - x) R_U \, d\mu_{D-L}(x) \\
&= \frac{R_U}{\iota}\mathrm{E}\left[\left(T_{D-L}\right) 1_{\{0 \le T_{D-L} \le \iota\}}\right], \\
\mathrm{E}\left[Z\mathbf{1}_{C_4}\right] &= \int_{x=-\iota}^{0} \int_{v=-x}^{\iota} \frac{1}{\iota}\lambda\left(v+x\right) \, dv \, d\mu_{D-L}(x) \\
&= \frac{\lambda}{2\iota}\mathrm{E}\left[\left(\iota + T_{D-L}\right)^2 1_{\{-\iota \le T_{D-L} \le 0\}}\right].
\end{aligned}$$

By using Eqn. (3.4.1), we then get an equation for R_U of the form

$$R_U = aR_U + b,$$

where

$$a = \lambda p\iota \mathrm{P}\left(T_{D-L} > \iota\right) + \lambda p \mathrm{E}\left[\left(T_{D-L}\right) 1_{\{0 \le T_{D-L} \le \iota\}}\right],$$
$$b = \lambda(1-p)\iota + \frac{\lambda^2 p}{2}\left(\mathrm{E}\left[\left(\iota + T_{D-L}\right)^2 1_{\{-\iota \le T_{D-L} \le 0\}}\right] + \iota^2 \mathrm{P}\left(0 \le T_{D-L} \le \iota\right) - \mathrm{E}\left[T_{D-L}^2 1_{\{0 \le T_{D-L} \le \iota\}}\right]\right).$$

Note that a is the expected number of named but untraced immediate offspring of a typical unnamed individual. Since lifetimes are constant, these named but untraced immediate offspring have offspring in the manner of unnamed individuals, which means

$$\begin{aligned} R_U &= b + aR_U \\ &= b + ab + a^2 R_U \\ &\vdots \\ &= \sum_{i=0}^{k} a^i b + a^{k+1} R_U \end{aligned}$$

and by letting $k \to \infty$ in the above we can see that,

$$R_U = \frac{b}{1-a}$$

for $a < 1$, otherwise R_U is infinite.

Meanwhile

$$G(s) = \sum_{k=1}^{6} \mathrm{E}\left[s^Z \mathbf{1}_{C_k}\right],$$

and we have that

$$\begin{aligned} \mathrm{E}\left[s^Z \mathbf{1}_{C_1}\right] &= \int_{x=\iota}^{\infty} H(s)\, d\mu_{D-L}(x) \\ &= \mathrm{P}\left(T_{D-L} > \iota\right) H(s), \end{aligned}$$

$$
\begin{aligned}
\mathrm{E}\left[s^Z \mathbf{1}_{C_2}\right] &= \int_{x=0}^{\iota} \int_{v=0}^{\iota-x} \frac{1}{\iota} e^{-\lambda(v+x)(1-s)}\, dv\, d\mu_{D-L}(x) \\
&= \frac{1}{\lambda\iota(1-s)} \left\{ \mathrm{E}\left[e^{\lambda(s-1)(T_{D-L})} 1_{\{0 \le T_{D-L} \le \iota\}} \right] - e^{\lambda\iota(s-1)} \mathrm{P}\left(0 \le T_{D-L} \le \iota\right) \right\}, \\
\mathrm{E}\left[s^Z \mathbf{1}_{C_3}\right] &= \int_{x=0}^{\iota} \mathrm{P}(V > \iota - x) H(s)\, d\mu_{D-L}(x) \\
&= \frac{H(s)}{\iota} \mathrm{E}\left[(T_{D-L})\, 1_{\{0 \le T_{D-L} \le \iota\}} \right], \\
\mathrm{E}\left[s^Z \mathbf{1}_{C_4}\right] &= \int_{x=-\iota}^{0} \int_{v=-x}^{\iota} \frac{1}{\iota} e^{-\lambda(v+x)(1-s)}\, dv\, d\mu_{D-L}(x) \\
&= \frac{1}{\lambda\iota(1-s)} \left\{ \mathrm{P}\left(0 \le -T_{D-L} \le \iota\right) - \mathrm{E}\left[e^{\lambda(s-1)(\iota+T_{D-L})} 1_{\{0 \le -T_{D-L} \le \iota\}} \right] \right\}, \\
\mathrm{E}\left[s^Z \mathbf{1}_{C_5}\right] &= \int_{x=-\iota}^{0} \int_{v=0}^{-x} \frac{1}{\iota} s^0\, dv\, d\mu_{D-L}(x) \\
&= \frac{1}{\iota} \mathrm{E}\left[T_{D-L} 1_{\{0 \le -T_{D-L} \le \iota\}} \right], \\
\mathrm{E}\left[s^Z \mathbf{1}_{C_6}\right] &= \int_{x=-\infty}^{\iota} s^0\, d\mu_{D-L}(x) \\
&= \mathrm{P}\left(-T_{D-L} > \iota\right),
\end{aligned}
$$

and $H(s)$ follows using Eqn. (3.4.2).

3.5 Single-step tracing, Exponential infectious period

We now assume that $T_I \sim \mathrm{Exp}(\gamma)$, i.e. the infectious period of each individual is exponentially distributed with mean $\frac{1}{\gamma}$.

3.5.1 Calculation of R_U

Let T denote the lifetime of our typical unnamed individual in the MBDP, A, ($T \sim \mathrm{Exp}(\gamma)$) and, for $k = 0, 1, \ldots$,

$$
h_k(t) = \mathrm{E}\left[R^{(k)} | T = t \right].
$$

Now, $R^{(0)}$ is just the number of unnamed immediate offspring. Hence, $\left(R^{(0)}|T=t\right) \sim$ Poisson$(\lambda(1-p)t)$, so

$$\mathrm{E}\left[R^{(0)}\middle|\, T=t\right] = \lambda(1-p)t.$$

Let N_1 denote the number of named immediate (i.e. type-1) offspring of our typical individual and, for $i = 1, 2, \ldots, N_1$, let $Z_i^{(k)}$ be the total number of descendants from the ith such (arbitrarily ordered) individual that contribute to $R^{(k)}$. Thus

$$\sum_{i=1}^{k} R_i = \sum_{i=1}^{N_1} Z_i^{(k)},$$

where the sum on the right is zero if $N_1 = 0$. Now $N_1|T=t \sim$ Poisson(λpt) and conditional upon $N_1 = n$, $T = t$, the birth times of these n individuals can be obtained by sampling n independent $U(0,t)$ random variables, and hence the $Z_i^{(k)}$ are independent and identically distributed, with common distribution $Z^{(k)}$, say. Thus

$$\begin{aligned}\mathrm{E}\left[\sum_{i=1}^{k} R_i|T=t\right] &= \sum_{n=0}^{\infty} \mathrm{P}\left(N_1=n|\, T=t\right) n\mathrm{E}\left[Z^{(k)}|T=t\right] \\ &= \lambda pt g_k(t),\end{aligned}$$

where $g_k(t) = \mathrm{E}\left[Z^{(k)}|T=t\right]$ $(k = 1, 2, \ldots)$ and $g_0(t) = 0$, and so, for $k = 1, 2, \ldots$

$$h_k(t) = \lambda(1-p)t + \lambda pt g_k(t) \qquad (t > 0) \tag{3.5.1}$$

and $h_0(t) = \lambda(1-p)t$.

In the later parts of this subsection, we obtain expressions for $g_k(t)$ and then use this to obtain an expression for R_U, first assuming no delays or latent periods, then assuming exponentially-distributed delays and arbitrarily distributed latent periods.

In a similar fashion to Section 3.4.1, it can be shown that R_U would be the same here if it were assumed that sibling units experience the same delay.

3.5.2 Calculation of extinction probability

Let

$$\hat{H}(s,t) = E\left[s^R \middle| T = t\right].$$

If the number of immediate offspring of A is M, then $M|T = t \sim \text{Poisson}(\lambda t)$. Conditional on $M = k$ and $T = t$, the birth times of these k individuals can be obtained by sampling k independent $\text{U}(0,t)$ random variables, and hence the contributions to R of these k individuals are independent and identically distributed. An unnamed individual (occurring with probability $1-p$) will contribute just themselves to R, while a named individual (probability p) will have a contribution distributed as Z, and hence

$$\begin{aligned}\text{E}\left[s^R \middle| M = k, T = t\right] &= \left[(1-p)s + p\text{E}\left[s^Z\right]\right]^k \\ &= \left[(1-p)s + pG(s,t)\right]^k,\end{aligned}$$

where $G(s,t) = \text{E}\left[s^Z \middle| T = t\right]$, and so

$$\begin{aligned}\hat{H}(s,t) &= \sum_{k=0}^{\infty} \frac{e^{-\lambda t}(\lambda t)^k}{k!} \text{E}\left[s^R \middle| M = k, T = t\right] \\ &= \sum_{k=0}^{\infty} \frac{e^{-\lambda t}(\lambda t)^k}{k!} \left[(1-p)s + pG(s,t)\right]^k \\ &= e^{-\lambda t} \sum_{k=0}^{\infty} \frac{1}{k!} \left[\lambda t\left((1-p)s + pG(s,t)\right)\right]^k \\ &= \exp\left\{-\lambda t\left(1 - (1-p)s - pG(s,t)\right)\right\}, \qquad (3.5.2)\end{aligned}$$

while

$$H(s) = \int_0^{\infty} \gamma e^{-\gamma t} \hat{H}(s,t)\, dt,$$

which, if we can evaluate it, can be used to calculate p_E

In a similar fashion to Section 3.4.2, it can be shown that $H(s)$ would be different if it were assumed that sibling units experience the same delay.

3.5.3 No latent period, no delay

First we look at the case without latent periods or delays, i.e. $T_D \equiv T_L \equiv 0$.

For $k = 1, 2, \ldots$, to compute $\mathrm{E}\left[Z^{(k)}|T = t\right]$, let V denote the excess lifetime of individual A when our typical named descendant of A, B, is born and T_B denote the natural lifetime of B. Then

$$V|T = t \sim \mathrm{U}(0, t).$$

Further if $T_B > V$ then none of B's immediate offspring are named, so

$$\mathrm{E}\left[Z^{(k)}|V = v, T_B > v\right] = \lambda v,$$

whilst, if $T_B < V$ then B dies before A and so B can name individuals,

$$(Z^{(k)}|V = v, T_B = t_B) \quad \stackrel{D}{=} \quad R^{(k-1)}|T = t_B \qquad\qquad (t_B < v).$$

Putting this all together yields

$$\begin{aligned} g_k(t) &= \int_{v=0}^{t} \frac{1}{t} \left\{ \mathrm{P}(T_B > v)\lambda v + \int_{u=0}^{v} \gamma e^{-\gamma u} h_{k-1}(u) du \right\} dv \\ &= \int_{v=0}^{t} \frac{1}{t} \left\{ \lambda v e^{-\gamma v} + \int_{u=0}^{v} \gamma e^{-\gamma u} h_{k-1}(u) du \right\} dv, \end{aligned}$$

whence, using Eqn. (3.5.1),

$$\begin{aligned} h_k(t) &= \lambda(1-p)t + \lambda p \int_0^t \lambda v e^{-\gamma v}\, dv + \lambda p \int_{v=0}^{t} \int_{u=0}^{v} \gamma e^{-\gamma u} h_{k-1}(u)\, du\, dv \\ &= \lambda(1-p)t + \frac{\lambda^2 p}{\gamma^2} \left\{ 1 - e^{-\gamma t} - \gamma t e^{-\gamma t} \right\} \\ &\qquad + \lambda p \int_{v=0}^{t} \int_{u=0}^{v} \gamma e^{-\gamma u} h_{k-1}(u)\, du\, dv, \end{aligned} \tag{3.5.3}$$

for $k = 1, 2, \ldots$, and $h_0(t) = \lambda(1-p)t$.

For $\theta \geq 0$, let $L_k(\theta) = \int_0^\infty e^{-\theta t} h_k(t)\, dt$ be the Laplace transform of $h_k(t)$. Taking the Laplace transform of Eqn. (3.5.3) yields, for $k = 1, 2, \ldots$,

$$L_k(\theta) = \frac{\lambda(1-p)}{\theta^2} + \frac{\lambda^2 p}{\theta(\theta+\gamma)^2} + \frac{\lambda p \gamma}{\theta^2} L_{k-1}(\gamma + \theta), \tag{3.5.4}$$

and $L_0(\theta) = \int_0^\infty e^{-\theta t}\lambda(1-p)tdt = \frac{\lambda(1-p)}{\theta^2}$.

Now, $R_U^{(k)} = \int_0^\infty \gamma e^{-\gamma t} h_k(t)\, dt = \gamma L_k(\gamma)$, and setting $\theta = j\gamma$ ($j = 1, 2\ldots$) in Eqn. (3.5.4) yields

$$L_k(j\gamma) = \frac{\lambda(1-p)}{j^2\gamma^2} + \frac{\lambda^2 p}{j(j+1)^2\gamma^3} + \frac{\lambda p}{j^2\gamma} L_{k-1}((j+1)\gamma). \tag{3.5.5}$$

This gives $L_1(j\gamma) = \frac{\lambda(1-p)}{j^2\gamma^2} + \frac{\lambda^2 p}{j(j+1)^2\gamma^3} + \frac{\lambda^2 p(1-p)}{j^2(j+1)^2\gamma^3}$. Suppose now that, for $k = 1, 2, \ldots, \kappa$ and $j = 1, 2, \ldots$,

$$L_k(j\gamma) = \frac{\lambda(1-p)}{j^2\gamma^2} + \frac{\lambda p}{\gamma^2} \sum_{i=1}^{k} \frac{\lambda^i p^{i-1}((1-p)+i+j-1)}{\gamma^i[(i+j)!]^2/[(j-1)!]^2}, \tag{3.5.6}$$

then using Eqn. (3.5.5),

$$\begin{aligned}
L_{\kappa+1}(j\gamma) &= \frac{\lambda(1-p)}{j^2\gamma^2} + \frac{\lambda^2 p}{j(j+1)^2\gamma^3} + \frac{\lambda p}{j^2\gamma} L_\kappa((j+1)\gamma) \\
&= \frac{\lambda(1-p)}{j^2\gamma^2} + \frac{\lambda^2 p}{j(j+1)^2\gamma^3} \\
&\qquad + \frac{\lambda p}{j^2\gamma}\left(\frac{\lambda(1-p)}{(j+1)^2\gamma^2} + \frac{\lambda p}{\gamma^2}\sum_{i=1}^{\kappa} \frac{\lambda^i p^{i-1}((1-p)+i+j)}{\gamma^i[(i+j+1)!]^2/[j!]^2}\right) \\
&= \frac{\lambda(1-p)}{j^2\gamma^2} + \frac{\lambda^2 p}{j(j+1)^2\gamma^3} + \frac{\lambda^2 p(1-p)}{j^2(j+1)^2\gamma^3} \\
&\qquad + \frac{\lambda p}{j^2\gamma^2}\sum_{i=1}^{\kappa} \frac{\lambda^{i+1} p^{i}((1-p)+i+j)}{\gamma^{i+1}[(i+j+1)!]^2/[j!]^2} \\
&= \frac{\lambda(1-p)}{j^2\gamma^2} + \frac{\lambda^2 p}{j(j+1)^2\gamma^3} + \frac{\lambda^2 p(1-p)}{j^2(j+1)^2\gamma^3} \\
&\qquad + \frac{\lambda p}{j^2\gamma^2}\sum_{i=2}^{\kappa+1} \frac{\lambda^{i} p^{i-1}((1-p)+i+j-1)}{\gamma^{i}[(i+j)!]^2/[j!]^2} \\
&= \frac{\lambda(1-p)}{j^2\gamma^2} + \frac{\lambda p}{\gamma^2}\sum_{i=1}^{\kappa+1} \frac{\lambda^{i} p^{i-1}((1-p)+i+j-1)}{\gamma^{i}[(i+j)!]^2/[(j-1)!]^2},
\end{aligned}$$

hence Eqn. (3.5.6) holds for all $k = 1, 2, \ldots$. Recall that $R_U^{(k)} = \gamma L_k(\gamma)$. Thus, setting $j = 1$ in Eqn. (3.5.6) yields

$$R_U^{(k)} = \frac{\lambda(1-p)}{\gamma} + \frac{\lambda p}{\gamma}\sum_{i=1}^{k} \frac{\lambda^i p^{i-1}((1-p)+i)}{[(i+1)!]^2\gamma^i}.$$

Letting $k \to \infty$ in the above expression gives

$$R_U = \frac{\lambda(1-p)}{\gamma} + \frac{\lambda p}{\gamma} \sum_{i=1}^{\infty} \frac{\lambda^i p^{i-1}((1-p)+i)}{[(i+1)!]^2 \gamma^i}.$$

Note that

$$\begin{aligned}
\frac{\lambda p}{\gamma} \sum_{i=1}^{\infty} \frac{\lambda^i p^{i-1}((1-p)+i)}{[(i+1)!]^2 \gamma^i} &< \frac{\lambda}{\gamma} \sum_{i=1}^{\infty} \frac{(\lambda p)^i (i+1)}{[(i+1)!]^2 \gamma^i} \\
&< \frac{\lambda}{\gamma} \sum_{i=1}^{\infty} \frac{(\lambda p)^i}{i! \gamma^i} \\
&< \frac{\lambda}{\gamma} \sum_{i=0}^{\infty} \frac{(\lambda p)^i}{i! \gamma^i} \\
&= \frac{\lambda}{\gamma} e^{\frac{\lambda p}{\gamma}}
\end{aligned}$$

and hence the expression for R_U is convergent. It can be rewritten as

$$R_U = \frac{\lambda(1-p)}{\gamma} \sum_{j=0}^{\infty} \frac{(\lambda p)^j}{[(j+1)!]^2 \gamma^j} + \frac{\lambda^2 p}{\gamma^2} \sum_{k=0}^{\infty} \frac{(k+1)(\lambda p)^k}{[(k+2)!]^2 \gamma^k}. \tag{3.5.7}$$

Now,

$$\begin{aligned}
\sum_{k=0}^{\infty} \frac{(k+1)t^k}{[(k+2)!]^2} &= \frac{d}{dt} \left[\sum_{k=0}^{\infty} \frac{t^{k+1}}{[(k+2)!]^2} \right] \\
&= \frac{d}{dt} \left[\sum_{k=0}^{\infty} \frac{t^k}{[(k+1)!]^2} - 1 \right] \\
&= \frac{d}{dt} \left[\frac{1}{t} \sum_{k=0}^{\infty} \frac{t^{k+1}}{[(k+1)!]^2} \right] \\
&= \frac{d}{dt} \left[\frac{1}{t} \left(I_0\left(2\sqrt{t}\right) - 1 \right) \right] \\
&= \frac{1}{t^2} \left(\sqrt{t} I_{-1}\left(2\sqrt{t}\right) - I_0\left(2\sqrt{t}\right) + 1 \right),
\end{aligned}$$

where $I_k(x)$ is the modified Bessel function of the first kind (and using equation 9.6.28 from Abramowitz and Stegun [1]: $\frac{d}{dx} I_0(x) = I_{-1}(x)$).

Thus (setting $\delta = \sqrt{\frac{\lambda p}{\gamma}}$),

$$\begin{aligned}
R_U &= \frac{1-p}{p} \left\{ I_0(2\delta) - 1 \right\} + \frac{1}{p} \left\{ \delta I_{-1}(2\delta) - I_0(2\delta) + 1 \right\} \\
&= 1 + \frac{\delta}{p} I_{-1}(2\delta) - I_0(2\delta).
\end{aligned}$$

Setting $\gamma = 1$ without loss of generality, and letting $p \to 1$ in the above yields

$$R_U = 1 + \sqrt{\lambda} I_{-1}\left(2\sqrt{\lambda}\right) - I_0\left(2\sqrt{\lambda}\right).$$

Consider the extinction probability, p_E, of the GWP. Recall that if $T_B > V$, then none of B's immediate offspring are named, so

$$\begin{aligned} \mathrm{E}\left[s^{Z_i} \middle| V = v, T_B > v\right] &= \sum_{j=0}^{\infty} \frac{(\lambda v)^j e^{-\lambda v}}{j!} s^j \\ &= \exp\left\{\lambda v\,(s-1)\right\}, \end{aligned}$$

whilst, if $T_B < V$ then B dies before A and

$$\left(s^{Z_i} \middle| V = v, T_B = t_B\right) =^D s^R \middle| T = t_B \qquad (t_B < v).$$

Hence,

$$\begin{aligned} G(s,t) &= \int_{v=0}^{t} \frac{1}{t} \left\{ \mathrm{P}\left(T_B > v\right) e^{\lambda v(s-1)} + \int_{u=0}^{v} \gamma e^{-\gamma u} \hat{H}(s,u)\, du \right\} dv \\ &= \frac{1}{t} \int_{v=0}^{t} e^{\lambda v(s-1)} e^{-\gamma v}\, dv + \frac{1}{t} \int_{v=0}^{t} \int_{u=0}^{v} \gamma e^{-\gamma u} \hat{H}(s,u)\, du\, dv, \end{aligned}$$

whence

$$\begin{aligned} \hat{H}(s,t) = \exp\Bigg\{ &\lambda(1-p)st + \lambda p \int_{v=0}^{t} e^{\lambda v(s-1)} e^{-\gamma v} dv \\ &+ \lambda p \int_{v=0}^{t} \int_{u=0}^{v} \gamma e^{-\gamma u} \hat{H}(s,u)\, du\, dv - \lambda t \Bigg\}. \end{aligned}$$

Hence

$$\begin{aligned} \log \hat{H}(s,t) = \lambda((1-p)s - 1)t &+ \lambda p \int_{v=0}^{t} e^{\lambda v(s-1)} e^{-\gamma v} dv \\ &+ \lambda p \int_{v=0}^{t} \int_{u=0}^{v} \gamma e^{-\gamma u} \hat{H}(s,u)\, du\, dv. \end{aligned} \tag{3.5.8}$$

Differentiating the above twice (and rearranging) gives

$$\frac{d^2 \hat{H}(s,t)}{dt^2} \hat{H}(s,t) - \left(\frac{d\hat{H}(s,t)}{dt}\right)^2 + f_1(s,t)\left[\hat{H}(s,t)\right]^2 - f_2(t)\left[\hat{H}(s,t)\right]^3 = 0,$$

where $f_1(s,t) = \lambda p(\lambda(1-s)+\gamma)e^{-(\lambda(1-s)+\gamma)t}$ and $f_2(t) = \lambda p\gamma e^{-\gamma t}$. This differential equation appears to have a fairly simple form, but it is unfortunately very difficult to solve. It may be possible to use solve it numerically, but $\hat{H}(s,t)$ is not the end result we require, since we need to find $H(s) = \int_0^\infty \gamma e^{-\gamma t}\hat{H}(s,t)\,dt$.

Alternatively, if we differentiate Eqn. (3.5.8) once and let $Y(s,t) = \int_{u=0}^{t} \gamma e^{-\gamma u}\hat{H}(s,u)du$, we obtain

$$\frac{d^2Y(s,t)}{dt^2} - \lambda pY(s,t)\frac{dY(s,t)}{dt} + f_3(s,t)\frac{dY(s,t)}{dt} = 0,$$

where $\gamma + \lambda - \lambda(1-p)s - \lambda pe^{-(\lambda(1-s)+\gamma)t}$. Now we require $H(s) = \lim_{t\to\infty} Y(s,t)$, but unfortunately this differential equation is also difficult to solve, even if we just require the asymptotic behaviour of its solution.

Note that later on in Section 3.8 we use simulations to obtain estimates for $H(s)$ and hence p_E. However, an exact method would require much less computation time.

A simplifying approximation: independence of sibling units

The sort of contact tracing we are considering here is one such that individuals are named and traced at the end of their parent's lifetime, and hence sibling units co-depend upon when their parent's lifetime ends, in other words upon the size of their parent's lifetime. Note that when the lifetime is constant, then the sibling units become independent, but what of the case where lifetimes are exponential? These inter-sibling dependencies make the model harder to analyse, but how much of an effect do they have?

Suppose that instead of being named at their end of their parent's lifetime, individuals (independently of their siblings) are named after a period of time beginning at their birth, which has an Exp(γ) distribution. The lifetime of a named individual would

then be the minimum of this naming period and their natural lifetime (both of which are Exp(γ) random variables) and so has an Exp(2γ) distribution. Further, named individuals would be traced with probability $\frac{1}{2}$, otherwise they would be untraced.

Let us approximate then by assuming we have a two-type process of unnamed and named individuals (which we shall refer to as type-U and type-N, respectively), with respective lifetimes distributed according to $T_U \sim \text{Exp}(\gamma)$ and $T_N \sim \text{Exp}(2\gamma)$ with birth rates represented by

$$\Lambda = \begin{bmatrix} \lambda_{UU} & \lambda_{UN} \\ \lambda_{NU} & \lambda_{NN}, \end{bmatrix} = \begin{bmatrix} \lambda(1-p) & \lambda p \\ \frac{\lambda}{2} + \frac{\lambda(1-p)}{2} & \frac{\lambda p}{2}, \end{bmatrix},$$

where λ_{ij} is the rate of birth of type-j offspring by type-i parents ($i, j = U, N$).

Then the matrix of mean offspring is

$$M = \begin{bmatrix} \frac{\lambda(1-p)}{\gamma} & \frac{\lambda p}{\gamma} \\ \frac{\lambda(2-p)}{4\gamma} & \frac{\lambda p}{4\gamma}, \end{bmatrix}.$$

The reproduction number, R_0, is given by the largest eigenvalue of M and is a threshold parameter in that extinction occurs with probability 1 if and only if $R_0 \leq 1$. So, setting $\gamma = 1$ without loss of generality,

$$R_0 = \frac{\lambda}{8}\left(4 - 3p + \sqrt{16 - 8p + 9p^2}\right).$$

If $f_U(\theta)$ and $f_N(\theta)$ ($\theta \geq 0$ are the moment-generating functions of T_U and T_N, then $q_U = f_U\left(\lambda_{UU}(1-q_U) + \lambda_{UN}(1-q_N)\right)$ and $q_N = f_N\left(\lambda_{NU}(1-q_U) + \lambda_{NN}(1-q_N)\right)$ ($q_U, q_N \in (0,1)$), where q_U and q_N are the extinction probabilities given the is one initial individual who is unnamed and named, respectively. So, we get (setting $\gamma = 1$ without loss of

generality)

$$q_U = \frac{1}{1+\lambda(1-p)(1-q_U)+\lambda p(1-q_N)},$$
$$q_N = \frac{2}{2+\frac{\lambda}{2}(2-p)(1-q_U)+\frac{\lambda p}{2}(1-q_N)},$$

which we can solve for q_U (which is the value of interest, since it is sensible to assume we begin with an unnamed individual).

In particular, when $p = 1$, we get

$$q_U = \begin{cases} \frac{3+\sqrt{9+4(\lambda+\lambda^2)}}{2(\lambda+\lambda^2)} & \text{if } \lambda > \frac{8}{1+\sqrt{17}} \\ 1 & otherwise \end{cases}$$

We consider how this approximating, dependence-free model compares with the true model in Section 3.8.

A heuristic derivation of the threshold parameter; a more sophisticated approximation

Here we offer an alternative, non-rigorous derivation of R_U by using an approximation that ignores the dependence of sibling units and incorporates lack-of-memory. In our approximation, instead of assuming that the offspring of an individual are named when the individual dies, we assume that the offspring (independent of each other) are named after a period of time beginning at their birth which has the same distribution as their parent's lifetime (this lack-of-memory assumption makes sense when we see that the resulting distributions are Exponential).

Let M_{ij} be the mean number of type-j offspring spawned by a type-i in this approximation. (Note that a type-i individual can only spawn type-0 or type-$(i+1)$ individuals.

Clearly, a type-0 individual has a lifetime with distribution Exp(γ) and has offspring at

rate λ throughout its lifetime, naming each one independently with probability p. So $M_{00} = \frac{\lambda(1-p)}{\gamma}$ and $M_{01} = \frac{\lambda p}{\gamma}$.

Consider a typical named child, B say, of a typical unnamed individual, A say. B has an Exp(γ) natural lifetime and under our approximation they will be named after a period of time with a Exp(γ) distribution (the distribution of A's excess lifetime after giving birth to B), which we assume to be independent of the corresponding periods of time for B's siblings. B has a lifetime distributed as the minimum of these, i.e. it has an Exp(2γ) distribution. Now, with probability $\frac{1}{2}$ they die naturally (and hence can name offspring), otherwise they die unnaturally (and cannot name offspring). Hence, $M_{10} = \frac{\lambda(1-p)}{4\gamma} + \frac{\lambda}{4\gamma}$ and $M_{12} = \frac{\lambda p}{4\gamma}$.

Consider now a typical named child of B, C say. Then individual C is type-2. C has an Exp(γ) natural lifetime and under our approximation they will be named after a period of time with a Exp(2γ) distribution (the distribution of B's excess lifetime after giving birth to C), which we assume to be independent of the corresponding periods of time for C's siblings. C has a lifetime distributed as the minimum of these, i.e. it has an Exp(3γ) distribution. Now, with probability $\frac{1}{3}$ they die naturally (and hence can name offspring), otherwise they die unnaturally (and cannot name offspring). Hence, $M_{20} = \frac{\lambda(1-p)}{9\gamma} + \frac{2\lambda}{9\gamma}$ and $M_{23} = \frac{\lambda p}{9\gamma}$.

Consider now a typical type-k individual. They have an Exp(γ) natural lifetime and under our approximation they will be named after a period of time with a Exp($k\gamma$) distribution (the distribution of the type-$(k-1)$ individual's excess lifetime after giving birth to the type-(k) individual), which we assume to be independent of the corresponding periods of time for the other type-k offspring. They have a lifetime distributed as the minimum of these, i.e. it has an Exp($(k+1)\gamma$) distribution. Now, with

probability $\frac{1}{k+1}$ they die naturally (and hence can name offspring), otherwise they die unnaturally (and cannot name offspring). Hence, $M_{k,0} = \frac{\lambda(1-p)}{(k+1)^2\gamma} + \frac{k\lambda}{(k+1)^2\gamma}$ and $M_{k,k+1} = \frac{\lambda p}{(k+1)^2\gamma}$.

Then

$$\begin{aligned} R_U &= M_{00} + M_{01}\left(M_{10} + M_{12}\left(M_{20} + M_{23}\left(M_{30} + \ldots\right)\right)\right) \\ &= M_{00} + M_{01}M_{10} + M_{01}M_{12}M_{20} + \ldots \\ &= M_{00} + \sum_{k=1}^{\infty}\left(\prod_{j=1}^{k} M_{j-1,j}\right) M_{k0} \\ &= \frac{\lambda(1-p)}{\gamma} + \sum_{k=1}^{\infty} \frac{(\lambda p)^k}{[k!]^2\gamma^k}\left(\frac{\lambda(1-p)}{(k+1)^2\gamma} + \frac{k\lambda}{(k+1)^2\gamma}\right) \\ &= \frac{\lambda(1-p)}{\gamma} + \frac{\lambda p}{\gamma}\sum_{k=1}^{\infty} \frac{\lambda^k p^{k-1}((1-p)+k)}{[(k+1)!]^2\gamma^k}, \end{aligned}$$

which agrees with Eqn. (3.5.7).

We can obtain an estimate for the extinction probability, p_E, by assuming a type-i individual has a lifetime distributed as $T_i \sim \text{Exp}((i+1)\gamma)$, and that a type-$i$ individual gives birth to type-j individuals at rate $\lambda_{i,j}$ over its lifetime, with

$$\lambda_{i,j} = \begin{cases} \frac{\lambda(i+1-p)}{i+1} & \text{if } j = 0 \\ \frac{\lambda p}{i+1} & \text{if } j = i+1 \\ 0 & \text{otherwise.} \end{cases}$$

Let q_j ($j = 0, 1, 2, \ldots$) be the probability of extinction for an epidemic started by a type-j individual. Calculating these using $q_k = \text{E}\left[e^{-\sum_i \lambda_{ki}(1-q_i)T_k}\right]$, where, for $k = 0, 1, 2, \ldots$, $T_k \sim \text{Exp}((k+1)\gamma)$, $\lambda_{k0} = \frac{\lambda(1-p+k)}{k+1}$, $\lambda_{k,k+1} = \frac{\lambda p}{k+1}$ and $\lambda_{kj} = 0$ ($j \neq 0, k+1$), gives the system of equations

$$q_k = \frac{(k+1)\gamma}{(k+1)\gamma + \frac{\lambda(1-p+k)}{k+1}(1-q_0) + \frac{\lambda p}{k+1}(1-q_{k+1})} \qquad (k = 0, 1, 2, \ldots). \tag{3.5.9}$$

Solving this for q_0 (the probability of extinction for an epidemic started by an unnamed individual) gives the continued fraction equation

$$q_0 = \cfrac{a_0}{b_0 - \cfrac{a_1}{b_1 - \cfrac{a_2}{b_2 - \cfrac{a_3}{b_3 - \dots}}}}$$

where

$$\begin{aligned} a_0 &= \gamma, \\ a_k &= \frac{(k+1)\gamma\lambda p}{k} && (k = 1, 2, 3, \dots), \\ b_k &= (k+1)\gamma + \lambda - \frac{\lambda(1-p+k)}{k+1} q_0 && (k = 0, 1, 2, \dots). \end{aligned}$$

3.5.4 Exponential delay and arbitrary latent period

Now we let the delays be exponentially distributed with mean $\frac{1}{\xi}$, i.e. $T_D \sim \text{Exp}(\xi)$, and we let the latent period have some arbitrary distribution T_L, with distribution measure μ_L and known moment-generating function $\phi_L(\theta)$ ($\theta \geq 0$).

We revisit our typical unnamed individual A with lifetime T and individual B, a typical named offspring of A, with natural lifetime T_B and born with time V left of A's lifetime. Now, we let $T_{B,D}$ and $T_{B,L}$ be the tracing delay and latent period of B, respectively.

The delay and latent period have opposing effects on B. The latent period reduces the amount of time that B could have offspring over before being traced, while the delay increases this. We shall let $W = \max\{0, V + T_{B,D} - T_{B,L}\}$.

Conditioning on $T = t$, $T_{B,D} = t_D$ and $T_{B,L} = t_L$, we consider three cases: (i) $t_D > t_L$, (ii) $t_D \leq t_L \leq t_D + t$ and (iii) $t_L > t_D + t$. In case (i), W is uniformly distributed

over $(t_D - t_L, t + t_D - t_L)$. In case (ii), W has a probability mass of $\frac{t_L - t_D}{t}$ at 0 with the remaining mass uniformly distributed over $(0, t + t_D - t_L)$. In case (iii), $W = 0$.

Further if $T_B > W$ then none of B's immediate offspring are named, so

$$\mathrm{E}\left[Z^{(k)} | W = w, T_B > w\right] = \lambda w,$$

whilst, if $T_B < W$ then B is not traced and so

$$(Z^{(k)} | W = w, T_B = t_B) \stackrel{D}{=} \left(R^{(k-1)} | T = t_B\right) \qquad (t_B < w).$$

Putting this all together yields

$$\begin{aligned} g_k(t) = &\int_{t_D=0}^{\infty} \xi e^{-\xi t_D} \int_{t_L=0}^{t_D} \int_{w=t_D-t_L}^{t+t_D-t_L} \frac{1}{t} \chi(w)\, dw\, d\mu_L(t_L)\, dt_D \\ &+ \int_{t_D=0}^{\infty} \xi e^{-\xi t_D} \int_{t_L=t_D}^{t_D+t} \int_{w=0}^{t+t_D-t_L} \frac{1}{t} \chi(w)\, dw\, d\mu_L(t_L)\, dt_D, \end{aligned} \tag{3.5.10}$$

where

$$\chi(t) = \left\{ \lambda t e^{-\gamma t} + \int_{u=0}^{t} \gamma e^{-\gamma u} h_{k-1}(u) du \right\}$$

and multiplying Equation (3.5.10) by t gives

$$\begin{aligned} t g_k(t) = &\int_{t_D=0}^{\infty} \xi e^{-\xi t_D} \int_{t_L=0}^{t_D} \int_{w=t_D-t_L}^{t+t_D-t_L} \chi(w)\, dw\, d\mu_L(t_L)\, dt_D \\ &+ \int_{t_D=0}^{\infty} \xi e^{-\xi t_D} \int_{t_L=t_D}^{t_D+t} \int_{w=0}^{t+t_D-t_L} \chi(w)\, dw\, d\mu_L(t_L)\, dt_D. \end{aligned}$$

Let $L_k(\theta) = \int_0^{\infty} e^{-\theta t} h_k(t) dt$ ($\theta \geq 0$), then using Eqn. (3.5.1),

$$\begin{aligned} L_k(\theta) &= \int_0^{\infty} e^{-\theta t} \left(\lambda(1-p)t + \lambda p t g_k(t)\right) dt \\ &= \frac{\lambda(1-p)}{\theta^2} + \lambda p \int_0^{\infty} e^{-\theta t} t g_k(t)\, dt. \end{aligned}$$

Noting that

$$\begin{aligned} &\int_{t=0}^{\infty} e^{-\theta t} \int_{t_D=0}^{\infty} \xi e^{-\xi t_D} \int_{t_L=0}^{t_D} \int_{w=t_D-t_L}^{t+t_D-t_L} \chi(w)\, dw\, d\mu_L(t_L)\, dt_D\, dt \\ &= \int_{t=0}^{\infty} e^{-\theta t} \int_{t_L=0}^{\infty} \int_{t_D=t_L}^{\infty} \xi e^{-\xi t_D} \int_{w=t_D-t_L}^{t+t_D-t_L} \chi(w)\, dw\, dt_D\, d\mu_L(t_L)\, dt \end{aligned}$$

$$
\begin{aligned}
&= \int_{t=0}^{\infty} e^{-\theta t} \int_{t_L=0}^{\infty} e^{-\xi t_L} \int_{t'_D=0}^{\infty} \xi e^{-\xi t'_D} \int_{w=t'_D}^{t+t'_D} \chi(w)\, dw\, dt'_D\, d\mu_L(t_L)\, dt \\
&= \phi_L(\xi) \int_{t=0}^{\infty} e^{-\theta t} \int_{t'_D=0}^{\infty} \xi e^{-\xi t'_D} \int_{w=t'_D}^{t+t'_D} \chi(w)\, dw\, dt'_D\, dt \\
&= \phi_L(\xi) \int_{t'_D=0}^{\infty} \xi e^{-\xi t'_D} \int_{w=t'_D}^{\infty} \chi(w) \int_{t=w-t'_D}^{\infty} e^{-\theta t}\, dt\, dw\, dt'_D \\
&= \frac{\phi_L(\xi)}{\theta} \int_{w=0}^{\infty} e^{-\theta w} \chi(w) \int_{t'_D=0}^{w} \xi e^{-(\xi-\theta)t'_D}\, dt'_D\, dw \\
&= \begin{cases} \frac{\xi\phi_L(\xi)}{\theta(\xi-\theta)} \int_{w=0}^{\infty} \left(e^{-\theta w} - e^{-\xi w}\right) \left\{\lambda w e^{-\gamma w} + \int_{u=0}^{w} \gamma e^{-\gamma u} h_{k-1}(u)\, du\right\} dw & \text{if } \theta \neq \xi \\ \phi_L(\xi) \int_{w=0}^{\infty} w e^{-\xi w} \left\{\lambda w e^{-\gamma w} + \int_{u=0}^{w} \gamma e^{-\gamma u} h_{k-1}(u)\, du\right\} dw & \text{if } \theta = \xi \end{cases} \\
&= \begin{cases} \frac{\xi\phi_L(\xi)}{\theta(\xi-\theta)} \left\{\frac{\lambda}{(\theta+\gamma)^2} - \frac{\lambda}{(\xi+\gamma)^2} + \frac{\gamma}{\theta} L_{k-1}(\theta+\gamma) - \frac{\gamma}{\xi} L_{k-1}(\xi+\gamma)\right\} & \text{if } \theta \neq \xi \\ \phi_L(\xi) \left\{\frac{2\lambda}{(\xi+\gamma)^3} - \frac{\gamma}{\xi} L'_{k-1}(\xi+\gamma)\right\} & \text{if } \theta = \xi, \end{cases}
\end{aligned}
$$

and further that

$$
\begin{aligned}
&\int_{t=0}^{\infty} e^{-\theta t} \int_{t_D=0}^{\infty} \xi e^{-\xi t_D} \int_{t_L=t_D}^{t_D+t} \int_{w=0}^{t+t_D-t_L} \chi(w)\, dw\, d\mu_L(t_L)\, dt_D\, dt \\
&= \int_{t_D=0}^{\infty} \xi e^{-\xi t_D} \int_{t_L=t_D}^{\infty} \int_{t_1=0}^{\infty} e^{-\theta(t_1-t_D+t_L)} \int_{w=0}^{t_1} \chi(w)\, dw\, dt_1\, d\mu_L(t_L)\, dt_D \\
&= \int_{t_D=0}^{\infty} \xi e^{-\xi t_D} \int_{t_L=t_D}^{\infty} \int_{w=0}^{\infty} \chi(w) \int_{t_1=w}^{\infty} e^{-\theta(t_1-t_D+t_L)}\, dt_1\, dw\, d\mu_L(t_L)\, dt_D \\
&= \frac{1}{\theta} \int_{t_L=0}^{\infty} e^{-\theta t_L} \int_{t_D=0}^{t_L} \xi e^{-(\xi-\theta)t_D} \int_{w=0}^{\infty} e^{-\theta w} \chi(w)\, dw\, dt_D\, d\mu_L(t_L) \\
&= \frac{1}{\theta} \int_{t_L=0}^{\infty} e^{-\theta t_L} \int_{t_D=0}^{t_L} \xi e^{-(\xi-\theta)t_D} \left(\frac{\lambda}{(\theta+\gamma)^2} + \frac{\gamma}{\theta} L_{k-1}(\theta+\gamma)\right) dt_D\, d\mu_L(t_L) \\
&= \begin{cases} \frac{\xi}{\theta(\xi-\theta)} \int_{t_L=0}^{\infty} \left(e^{-\theta t_L} - e^{-\xi t_L}\right) \left(\frac{\lambda}{(\theta+\gamma)^2} + \frac{\gamma}{\theta} L_{k-1}(\theta+\gamma)\right) d\mu_L(t_L) & \text{if } \theta \neq \xi \\ \int_{t_L=0}^{\infty} t_L e^{-\xi t_L} \left(\frac{\lambda}{(\xi+\gamma)^2} + \frac{\gamma}{\xi} L_{k-1}(\xi+\gamma)\right) d\mu_L(t_L) & \text{if } \theta = \xi \end{cases} \\
&= \begin{cases} \frac{\xi(\phi_L(\theta)-\phi_L(\xi))}{\theta(\xi-\theta)} \left(\frac{\lambda}{(\theta+\gamma)^2} + \frac{\gamma}{\theta} L_{k-1}(\theta+\gamma)\right) & \text{if } \theta \neq \xi \\ -\phi'_L(\xi) \left(\frac{\lambda}{(\xi+\gamma)^2} + \frac{\gamma}{\xi} L_{k-1}(\xi+\gamma)\right) & \text{if } \theta = \xi, \end{cases}
\end{aligned}
$$

it follows that, for $\theta \neq \xi$,

$$\begin{aligned}
L_k(\theta) &= \frac{\lambda(1-p)}{\theta^2} + \frac{\lambda p\xi\left(\phi_L(\theta) - \phi_L(\xi)\right)}{\theta(\xi-\theta)}\left(\frac{\lambda}{(\theta+\gamma)^2} + \frac{\gamma}{\theta}L_{k-1}(\theta+\gamma)\right) \\
&\quad + \frac{\lambda p\xi\phi_L(\xi)}{\theta(\xi-\theta)}\left\{\frac{\lambda}{(\theta+\gamma)^2} - \frac{\lambda}{(\xi+\gamma)^2} + \frac{\gamma}{\theta}L_{k-1}(\theta+\gamma) - \frac{\gamma}{\xi}L_{k-1}(\xi+\gamma)\right\} \\
&= \frac{\lambda(1-p)}{\theta^2} + \frac{\lambda p\xi}{\theta(\xi-\theta)}\left\{\frac{\lambda\phi_L(\theta)}{(\theta+\gamma)^2} - \frac{\lambda\phi_L(\xi)}{(\xi+\gamma)^2} + \frac{\gamma\phi_L(\theta)}{\theta}L_{k-1}(\theta+\gamma)\right. \\
&\qquad \left. - \frac{\gamma\phi_L(\xi)}{\xi}L_{k-1}(\xi+\gamma)\right\},
\end{aligned}$$

while

$$\begin{aligned}
L_k(\xi) &= \frac{\lambda(1-p)}{\xi^2} - \lambda p\phi_L'(\xi)\left(\frac{\lambda}{(\xi+\gamma)^2} + \frac{\gamma}{\xi}L_{k-1}(\xi+\gamma)\right) + \lambda p\phi_L(\xi)\left\{\frac{2\lambda}{(\xi+\gamma)^3}\right. \\
&\qquad \left. - \frac{\gamma}{\xi}L_{k-1}'(\xi+\gamma)\right\} \\
&= \frac{\lambda(1-p)}{\xi^2} + \lambda p\lim_{\theta\to\xi}\frac{\phi_L(\theta)-\phi_L(\xi)}{\xi-\theta}\left(\frac{\lambda}{(\xi+\gamma)^2} + \frac{\gamma}{\xi}L_{k-1}(\xi+\gamma)\right) \\
&\quad + \lambda p\phi_L(\xi)\left\{\lim_{\theta\to\xi}\left(\frac{\lambda}{(\xi-\theta)(\theta+\gamma)^2} - \frac{\lambda}{(\xi-\theta)(\xi+\gamma)^2}\right)\right. \\
&\qquad \left. + \frac{\gamma}{\xi}\lim_{\theta\to\xi}\frac{L_{k-1}(\theta+\gamma) - L_{k-1}(\xi+\gamma)}{\xi-\theta}\right\} \\
&= \lim_{\theta\to\xi} L_k(\theta),
\end{aligned}$$

thus we can obtain $L_k(\theta)$ when $\theta = \xi$ by taking the limit $\theta \to \xi$. From now on we assume that $\theta \neq \xi$, and that ξ is non-integer. If ξ is an integer, we can obtain R_U by considering the limit as ξ approaches this integer value.

Setting $\gamma = 1$, without loss of generality, yields

$$\begin{aligned}
L_k(\theta) = \frac{\lambda(1-p)}{\theta^2} + \frac{\lambda p\xi}{\theta(\xi-\theta)}\left\{\frac{\lambda\phi_L(\theta)}{(\theta+1)^2} - \frac{\lambda\phi_L(\xi)}{(\xi+1)^2}\right. \\
\left. + \frac{\phi_L(\theta)}{\theta}L_{k-1}(\theta+1) - \frac{\phi_L(\xi)}{\xi}L_{k-1}(\xi+1)\right\}.
\end{aligned} \tag{3.5.11}$$

If $y_{k,j} = L_k(\xi+j)$ ($k = 0, 1, \ldots$, $j = 1, 2, \ldots$), then $y_{0,j} = \frac{\lambda(1-p)}{(\xi+j)^2}$ and, for $k = 1, 2, \ldots$ and

$j = 1, 2, \ldots,$

$$y_{k,j} = \alpha_j + \hat{\rho}_j y_{k-1,1} - \beta_j y_{k-1,j+1}, \tag{3.5.12}$$

where

$$\begin{aligned}
\alpha_j &= \frac{\lambda(1-p)}{(\xi+j)^2} + \frac{\lambda^2 p\xi}{j(\xi+j)}\left(\frac{\phi_L(\xi)}{(\xi+1)^2} - \frac{\phi_L(\xi+j)}{(\xi+j+1)^2}\right), \\
\hat{\rho}_j &= \frac{\lambda p \phi_L(\xi)}{j(\xi+j)}, \\
\beta_j &= \frac{\lambda p \xi \phi_L(\xi+j)}{j(\xi+j)^2}.
\end{aligned}$$

Let $\delta_0 = 1$ and $\delta_j = \prod_{i=1}^{j} \beta_i$ $(j = 1, 2, \ldots)$.

Suppose that, for $1 \leq \kappa \leq k-1$,

$$\begin{aligned}
y_{\kappa,k-\kappa+1} = &\sum_{i=k-\kappa}^{k-1} (-1)^{i-k+\kappa} \frac{\delta_i}{\delta_{k-\kappa}} \left(\alpha_{i+1} + \hat{\rho}_{i+1} y_{k-i-1,1}\right) \\
&+ (-1)^{\kappa} \frac{\delta_k}{\delta_{k-\kappa}} y_{0,k+1},
\end{aligned} \tag{3.5.13}$$

then using Eqn. (3.5.12),

$$\begin{aligned}
y_{\kappa+1,k-\kappa} &= \alpha_{k-\kappa} + \hat{\rho}_{k-\kappa} y_{\kappa,1} - \beta_{k-\kappa} y_{\kappa,k-\kappa+1} \\
&= \alpha_{k-\kappa} + \hat{\rho}_{k-\kappa} y_{\kappa,1} \\
&\quad - \beta_{k-\kappa}\left(\sum_{i=k-\kappa}^{k-1} (-1)^{i-k+\kappa} \frac{\delta_i}{\delta_{k-\kappa}} \left(\alpha_{i+1} + \hat{\rho}_{i+1} y_{k-i-1,1}\right) + (-1)^{\kappa} \frac{\delta_k}{\delta_{k-\kappa}} y_{0,k+1}\right) \\
&= \alpha_{k-\kappa} + \hat{\rho}_{k-\kappa} y_{\kappa,1} + \sum_{i=k-\kappa}^{k-1} (-1)^{i-k+\kappa+1} \frac{\delta_i}{\delta_{k-\kappa-1}} \left(\alpha_{i+1} + \hat{\rho}_{i+1} y_{k-i-1,1}\right) \\
&\qquad + (-1)^{\kappa+1} \frac{\delta_k}{\delta_{k-\kappa-1}} y_{0,k+1} \\
&= \sum_{i=k-\kappa-1}^{k-1} (-1)^{i-k+\kappa+1} \frac{\delta_i}{\delta_{k-\kappa-1}} \left(\alpha_{i+1} + \hat{\rho}_{i+1} y_{k-i-1,1}\right) + (-1)^{\kappa+1} \frac{\delta_k}{\delta_{k-\kappa-1}} y_{0,k+1},
\end{aligned}$$

hence Eqn. (3.5.13) is true for $1 \leq \kappa \leq k$, and setting $\kappa = k$ yields

$$y_{k,1} = \sum_{i=0}^{k-1} (-1)^i \delta_i \left(\alpha_{i+1} + \hat{\rho}_{i+1} y_{k-i-1,1}\right) + (-1)^k \delta_k y_{0,k+1}.$$

Now, let $\pi_j = y_{j,1} - y_{j-1,1}$ for $j = 1, 2, \ldots$, then

$$\pi_j = \begin{cases} \alpha_1 + \hat{\rho}_1 y_{0,1} - \delta_1 y_{0,2} - y_{0,1} & \text{for } j = 1 \\ (-1)^{j-1}\delta_{j-1}\alpha_j + (-1)^j \delta_j y_{0,j+1} - (-1)^{j-1}\delta_{j-1} y_{0,j} & \\ \quad +(-1)^{j-1}\delta_{j-1}\hat{\rho}_j y_{0,1} + \sum_{i=0}^{j-2}(-1)^i \delta_i \hat{\rho}_{i+1}\pi_{j-i-1} & \text{for } j = 2, 3, \ldots, \end{cases}$$

and we have that $y_{k,1} = y_{0,1} + \sum_{j=1}^{k} \pi_j$ for $k = 1, 2, \ldots$. Now we can examine $\sum_{j=1}^{\infty} \pi_j$ to consider the limiting behaviour of $y_{k,1}$ (since $y_{k,1}$ is increasing in k, its limit exists, whether finite or infinite):

$$\begin{aligned} \sum_{j=1}^{\infty} \pi_j &= \sum_{j=1}^{\infty}(-1)^{j-1}\left(\delta_{j-1}\alpha_j - \delta_j y_{0,j+1} - \delta_{j-1} y_{0,j} + \delta_{j-1}\hat{\rho}_j y_{0,1}\right) \\ &\quad + \sum_{j=2}^{\infty}\sum_{i=0}^{j-2}(-1)^i \delta_i \hat{\rho}_{i+1}\pi_{j-i-1} \\ &= \sum_{j=1}^{\infty}(-1)^{j-1}\left(\delta_{j-1}\alpha_j - \delta_j y_{0,j+1} - \delta_{j-1} y_{0,j} + \delta_{j-1}\hat{\rho}_j y_{0,1}\right) \\ &\quad + \sum_{i=0}^{\infty}(-1)^i \delta_i \hat{\rho}_{i+1} \sum_{j=i+2}^{\infty} \pi_{j-i-1} \\ &= \sum_{j=0}^{\infty}(-1)^{j}\left(\delta_{j}\alpha_{j+1} - \delta_{j+1} y_{0,j+2} - \delta_j y_{0,j+1} + \delta_j\hat{\rho}_{j+1} y_{0,1}\right) \\ &\quad + \sum_{i=0}^{\infty}(-1)^i \delta_i \hat{\rho}_{i+1} \sum_{j=1}^{\infty} \pi_j. \end{aligned}$$

Letting $y_1 = \lim_{k\to\infty} y_{k,1}$,

$$\begin{aligned} y_1 &= y_{0,1} + \sum_{j=1}^{\infty} \pi_j \\ &= y_{0,1} + \sum_{j=0}^{\infty}(-1)^{j}\left(\delta_{j}\alpha_{j+1} - \delta_{j+1} y_{0,j+2} - \delta_j y_{0,j+1} + \delta_j\hat{\rho}_{j+1} y_{0,1}\right) \\ &\quad + (y_1 - y_{0,1})\sum_{i=0}^{\infty}(-1)^i \delta_i \hat{\rho}_{i+1} \\ &= y_{0,1} + \sum_{j=0}^{\infty}(-1)^j \delta_j \alpha_{j+1} - \sum_{j=0}^{\infty}(-1)^j \delta_{j+1} y_{0,j+2} - \sum_{j=0}^{\infty}(-1)^j \delta_j y_{0,j+1} \\ &\quad + y_1 \sum_{i=0}^{\infty}(-1)^i \delta_i \hat{\rho}_{i+1} \end{aligned}$$

$$
\begin{aligned}
&= y_{0,1} + \sum_{j=0}^{\infty}(-1)^j\delta_j\alpha_{j+1} + \sum_{j=1}^{\infty}(-1)^j\delta_j y_{0,j+1} - \sum_{j=0}^{\infty}(-1)^j\delta_j y_{0,j+1} \\
&\quad + y_1\sum_{i=0}^{\infty}(-1)^i\delta_i\hat{\rho}_{i+1} \\
&= \sum_{j=0}^{\infty}(-1)^j\delta_j\alpha_{j+1} + y_1\sum_{i=0}^{\infty}(-1)^i\delta_i\hat{\rho}_{i+1},
\end{aligned}
$$

and so either y_1 is infinite or

$$
y_1 = \frac{\sum_{j=0}^{\infty}(-1)^j\delta_j\alpha_{j+1}}{1-\sum_{i=0}^{\infty}(-1)^i\delta_i\hat{\rho}_{i+1}}. \tag{3.5.14}
$$

Now, for $k, j = 1, 2, \ldots$, setting $\theta = j$ in Eqn. (3.5.11) yields

$$
\begin{aligned}
L_k(j) = \frac{\lambda(1-p)}{j^2} + \frac{\lambda^2 p}{j(\xi-j)}\left\{\frac{\phi_L(j)}{(j+1)^2} - \frac{\phi_L(\xi)}{(\xi+1)^2}\right\} \\
- \frac{\lambda p\phi_L(\xi)}{j(\xi-j)}L_{k-1}(\xi+1) + \frac{\lambda p\xi\phi_L(j)}{j^2(\xi-j)}L_{k-1}(j+1).
\end{aligned}
$$

If $x_{k,j} = L_k(j)$ (for $k = 0, 1, \ldots$, $j = 1, 2, \ldots$) then $x_{0,j} = \frac{\lambda(1-p)}{j^2}$, and, for $j, k = 1, 2, \ldots$,

$$
x_{k,j} = a_j - \rho_j y_{k-1,1} + b_j x_{k-1,j+1}, \tag{3.5.15}
$$

where

$$
\begin{aligned}
a_j &= \frac{\lambda(1-p)}{j^2} + \frac{\lambda^2 p\xi}{j(\xi-j)}\left(\frac{\phi_L(j)}{(j+1)^2} - \frac{\phi_L(\xi)}{(\xi+1)^2}\right) \\
\rho_j &= \frac{\lambda p\phi_L(\xi)}{j(\xi-j)} \\
b_j &= \frac{\lambda p\xi\phi_L(j)}{j^2(\xi-j)}.
\end{aligned}
$$

Let $c_0 = 1$ and $c_j = \prod_{i=1}^{j} b_i$ $(j = 1, 2, \ldots)$.

By iterating Eqn. (3.5.15), we obtain

$$
\begin{aligned}
R_U^{(k)} &= x_{k,1} \\
&= \sum_{i=0}^{k-1} c_i\left(a_{i+1} - \rho_{i+1}y_{k-i-1,1}\right) + c_k x_{0,k+1} \\
&= \sum_{i=0}^{k-1} c_i\left(a_{i+1} - \rho_{i+1}y_{k-i-1,1}\right) + c_k\frac{\lambda(1-p)}{(k+1)^2}.
\end{aligned}
$$

Note that for all (k, j), $y_{k,j} \leq x_{k,j}$. Hence if $y_{k,1}$ has infinite limit, then $x_{k,1}$ must do too, i.e. if $y_1 = \infty$ then $R_U = \infty$.

Suppose then that $y_{k,1}$ has a finite limit. Note that as $k \to \infty$, $c_k x_{0,k+1} \to 0$, and that $\sum_{i=0}^{\infty} c_i a_{i+1}$ is absolutely convergent, since $|c_i| \leq \frac{1}{i!}\left(\frac{\lambda p\xi}{|\xi-[\xi]|}\right)^i$ and $|a_i| \leq \lambda(1-p) + \frac{\lambda^2 p\xi}{|\xi-[\xi]|}$ which implies

$$\begin{aligned}\sum_{i=0}^{\infty} |c_i a_{i+1}| &\leq \left(\lambda(1-p) + \frac{\lambda^2 p\xi}{|\xi-[\xi]|}\right) \sum_{i=0}^{\infty} \frac{1}{i!}\left(\frac{\lambda p\xi}{|\xi-[\xi]|}\right)^i \\ &= \left(\lambda(1-p) + \frac{\lambda^2 p\xi}{|\xi-[\xi]|}\right) e^{\frac{\lambda p\xi}{|\xi-[\xi]|}},\end{aligned}$$

and hence is convergent. Further, given that $0 \leq y_{k-i-1,1} \leq \lim_{k\to\infty} y_{k,1} = y_1$ and $|\rho_i| \leq \frac{\lambda p}{|\xi-[\xi]|}$,

$$\begin{aligned}\sum_{i=0}^{\infty} |c_i \rho_{i+1} y_{k-i-1,1}| &\leq \frac{\lambda p}{|\xi-[\xi]|} y_1 \sum_{i=0}^{\infty} \frac{1}{i!}\left(\frac{\lambda p\xi}{|\xi-[\xi]|}\right)^i \\ &= \frac{\lambda p}{|\xi-[\xi]|} y_1 e^{\frac{\lambda p\xi}{|\xi-[\xi]|}},\end{aligned}$$

and hence $\sum_{i=0}^{\infty} c_i \rho_{i+1} y_{k-i-1,1}$ is convergent. Therefore, $R_U^{(k)}$ has finite limit. Hence, when $\sum_{i=0}^{\infty} \delta_i \hat{\rho}_{i+1} < 1$,

$$R_U = \lim_{k\to\infty} \sum_{i=0}^{k-1} c_i \left(a_{i+1} - \rho_{i+1} y_{k-i-1,1}\right)$$

otherwise $R_U = \infty$.

Letting $x_j = \lim_{k\to\infty} x_{k,j}$, this limit exists for all j if $\sum_{i=0}^{\infty}(-1)^i \delta_i \hat{\rho}_{i+1} < 1$, since x_1 exists and $x_{k,j}$ is decreasing in j for fixed k. Hence, if $\sum_{i=0}^{\infty} \delta_i \hat{\rho}_{i+1} < 1$, letting $k \to \infty$ in Eqn. (3.5.15) gives us

$$x_j = a_j - \rho_j y_1 + b_j x_{j+1}, \tag{3.5.16}$$

which by iterating gives, when $y_1 < \infty$,

$$\begin{aligned}R_U &= x_1 \\ &= \sum_{i=0}^{\infty} c_i \left(a_{i+1} - \rho_{i+1} y_1\right). \end{aligned} \tag{3.5.17}$$

Conditions for finiteness of R_U

We have already ascertained that R_U is finite if and only if y_1 is finite. Here we establish stronger conditions.

First we consider a lower bound for R_U in order to show that it is infinite for λ sufficiently large. Recall that in the MBDP, R is the total number of unnamed immediate offspring of (a) an unnamed individual and (b) of all named descendants of this unnamed individual, for whom the unnamed individual is their nearest unnamed ancestor. Consider R^-, a lower bound for R in which instead of counting the unnamed offspring of *all* named descendants as described, we count only (a) the immediate unnamed offspring of the unnamed individual and (b) unnamed individuals who are separated in the family tree from the unnamed individual by only named ancestors with a natural lifetime in the region $(\frac{1}{2}, 1)$ and an associated delay 1 time unit greater than their latent period. Clearly then, $R^- \leq R$, and if $R_U^- = E[R^-]$, then $R_U^- \leq R_U$.

Named individuals who have a natural lifetime in the interval $(\frac{1}{2}, 1)$ and who experience a delay 1 time unit greater than their latent period, must end their lifetime before the delay ends, and hence are necessarily untraced. Therefore they produce named offspring at rate λp over the length of their lifetime, and the probability that a typical named offspring has a natural lifetime in the interval $(\frac{1}{2}, 1)$ and experience a delay 1 time unit greater than their latent period is $\left(e^{-\frac{1}{2}} - e^{-1}\right) P(T_D > T_L + 1)$, and so these particular named offspring are produced at rate $\lambda p \left(e^{-\frac{1}{2}} - e^{-1}\right) P(T_D > T_L + 1)$. Such named individuals behave independently from one another, and hence are described by a branching process. The expected offspring in this branching process is

$$\lambda p \left(\frac{3}{2} e^{-\frac{1}{2}} - e^{-1}\right) \left(e^{-\frac{1}{2}} - e^{-1}\right) P(T_D > T_L + 1)$$

and so the total progeny is infinite with positive probability if

$$\lambda > \left[p\left(\frac{3}{2}e^{-\frac{1}{2}} - e^{-1}\right)\left(e^{-\frac{1}{2}} - e^{-1}\right)P\left(T_D > T_L + 1\right)\right]^{-1}.$$

So (and since these individuals have positive lifetimes and in the MBDP give birth to unnamed individuals at rate $\lambda(1-p)$), for large enough λ, $R_U^- = \infty$ and so also $R_U = \infty$.

Now, R_U is non-decreasing in λ, so if we define $\lambda^* = \inf\{\lambda : R_U = \infty\}$, then for all $\lambda \geq \lambda^*$, R_U is infinite. However, if $\lambda \in [0, \lambda^*)$ then R_U is finite and is given by Eqn. (3.5.17), while y_1 must also be finite and so is given by Eqn. (3.5.14).

3.6 Iterative tracing, no latent period or delay

We now assume that traced individuals can also name contacts, that is that all individuals name contacts when they are removed, and we begin by treating the case where $T_L = T_D = 0$, for which we show that the type-reproduction number R_U depends on the infectious period distribution only through its mean (which we assume to be finite). We do this by first assuming T_I has finite support, then countable support and finally assume that it has an arbitrary distribution.

3.6.1 Infectious period with finite support

Suppose that T_I has finite support $\{t_1, t_2, \ldots, t_n\}$, where $0 < t_1 < t_2 \ldots < t_n$, such that

$$\mathrm{P}(T_I = t_i) = p_i > 0 \quad (i = 1, 2, \ldots, n)$$

and

$$\sum_{i=1}^{n} p_i = 1.$$

To examine the offspring random variable of the GWP, we return to our typical unnamed individual in the MDBP, A, with lifetime T (so $T \stackrel{D}{=} T_I$). We need to find $R_U = \sum_{i=1}^{n} p_i h(t_i)$, where $h(t) = \mathrm{E}\left[R|T=t\right]$ ($0 \leq t \leq t_n$; while T_I has finite support we will require $h(t)$ to have continuous support for the purposes of a renewal argument).

Recall that $R = R^{(0)} + \sum_{i=1}^{\infty} R_i$, where $R^{(0)}$ is the number of unnamed immediate descendants and $\sum_{i=1}^{\infty} R_i$ is the remaining number of offspring in the GWP. Now, $R^{(0)}|T=t \sim$ Poisson$(\lambda(1-p)t)$ and so,

$$E\left[R^{(0)}\middle|\, T=t\right] = \lambda(1-p)t.$$

Let N_1 denote the number of named immediate (i.e. type-1) offspring of A and, for $i = 1, 2, \ldots N_1$ let Z_i be the total number of descendants from the ith (arbitrarily ordered) such individual that contribute to R. Thus

$$\sum_{i=1}^{\infty} R_i = \sum_{i=1}^{N_1} Z_i,$$

where the sum on the right is zero if $N_1 = 0$. Now $N_1|\, T = t \sim$ Poisson$(\lambda p t)$, and conditional upon $N_1 = k$, $T = t$, the birth times of these k individuals can be obtained by sampling x independent U$(0,t)$ random variables, and hence the Z_i are independent and identically distributed, with common distribution, Z, say. Thus

$$\begin{aligned} E\left[\sum_{i=1}^{\infty} R_i \middle|\, T=t\right] &= \sum_{x=0}^{\infty} \mathrm{P}\left(N_1 = k|\, T=t\right) xE\left[Z|T=t\right] \\ &= \lambda p t g(t), \end{aligned}$$

where $g(t) = E\left[Z|T=t\right]$, and so

$$h(t) = \lambda(1-p)t + \lambda p t g(t).$$

To obtain $g(t)$, we return to our typical immediate named offspring of A, B, who is born V time units before A dies (i.e. $V \sim$ U$(0,t)$ and has natural lifetime T_B. Suppose that

$t_{i-1} < t \leq t_i$, $V = v$ and $j = \arg\max_k \{t_{k-1} : v \geq t_{k-1}\}$ i.e. j is such that $v \in [t_{j-1}, t_j)$ for $j = 1, \ldots, i-1$ or $v \in [t_{i-1}, t]$ for $j = i$ (letting $t_0 = 0$). If $T_B \geq v$, then B dies when A dies B's actual lifetime is v, while if $T_B < v$, B's actual lifetime is T_B. Hence

$$Z|\, T_B = t_k \stackrel{D}{=} \begin{cases} R|T = t_k & (k < j) \\ R|T = v & (k \geq j), \end{cases}$$

and so

$$\begin{aligned} g(t) = \sum_{j=1}^{i-1} \int_{t_{j-1}}^{t_j} \frac{1}{t} \left(\sum_{k=j}^{n} p_k h(v) + \sum_{k=1}^{j-1} p_k h(t_k) \right) dv \\ + \int_{t_{i-1}}^{t} \frac{1}{t} \left(\sum_{k=i}^{n} p_k h(v) + \sum_{k=1}^{i-1} p_k h(t_k) \right) dv, \end{aligned}$$

where sums are zero if vacuous.

Therefore, for $t_{i-1} < t \leq t_i$,

$$\begin{aligned} h(t) = \lambda(1-p)t + \lambda p \sum_{j=1}^{i-1} \int_{t_{j-1}}^{t_j} \left(\sum_{k=j}^{n} p_k h(v) + \sum_{k=1}^{j-1} p_k h(t_k) \right) dv \\ + \lambda p \int_{t_{i-1}}^{t} \left(\sum_{k=i}^{n} p_k h(v) + \sum_{k=1}^{i-1} p_k h(t_k) \right) dv, \end{aligned}$$

and so,

$$h'(t) = \lambda(1-p) + \lambda p \sum_{k=i}^{n} p_k h(t) + \lambda p \sum_{k=1}^{i-1} p_k h(t_k),$$

which can be solved to give

$$h(t) \sum_{k=i}^{n} p_k = \frac{A_i}{\lambda p} e^{\lambda p t \sum_{k=i}^{n} p_k} - \frac{(1-p)}{p} - \sum_{k=0}^{i-1} p_k h(t_k).$$

From this we get

$$h(t_n) p_n = \frac{A_n}{\lambda p} e^{\lambda p t_n p_n} - \frac{(1-p)}{p} - \sum_{k=0}^{n-1} p_k h(t_k),$$

and so

$$R_U = \frac{A_n}{\lambda p} e^{\lambda p t_n p_n} - \frac{(1-p)}{p}.$$

Now we shall show that $h(t)$ is continuous. Note that $h(t+\Delta t)-h(t)$ is bounded above by the mean number of births in the untraced process (i.e $p=0$) during $[t,t+\Delta t]$, since (a) not all births count towards h and (b) the tracing has the effect of reducing lifetimes, and therefore the number of births. Since the mean number of births in the untraced process during $[t,t+\Delta t]$ tends to 0 as $\Delta t \downarrow 0$, then $h(t+\Delta t)-h(t)\to 0$ as $\Delta t\downarrow 0$, and so $h(t)$ is continuous.

Since $h(t)$ is continuous, the values of the constants A_i ($i=1,\ldots,n$) can be determined by matching boundary values, and also by noting that $h(0)=0$. Solving this, we get

$$A_i = \lambda(1-p)e^{\lambda p\sum_{j=1}^{i-1} t_j p_j},$$

and hence

$$\begin{aligned} R_U &= \frac{1-p}{p}\left(e^{\lambda p\sum_{i=1}^{n} t_i p_i}-1\right) \\ &= \frac{1-p}{p}\left(e^{\lambda p E[T_I]}-1\right). \end{aligned}$$

3.6.2 Infectious period with countable support

Suppose now that T_I has countable support $\{t_1,t_2,t_3,\ldots\}$, where $0<t_1<t_2<t_3\ldots$, such that

$$\mathrm{P}(T_I=t_i)=p_i>0 \quad (i=1,2,\ldots)$$

and

$$\sum_{i=1}^{\infty} p_i = 1 \text{ and } \sum_{i=1}^{\infty} p_i t_i < \infty.$$

Similar to the finite support, we get, for $t_{i-1}<t\le t_i$,

$$h(t)\sum_{k=i}^{\infty} p_k = \frac{(1-p)}{p}\left(e^{\lambda p\left(\sum_{j=1}^{i-1} t_j p_j + t\sum_{k=i}^{\infty} p_k\right)}-1\right) - \sum_{k=0}^{i-1} p_k h(t_k),$$

and hence

$$\sum_{k=0}^{i} p_k h(t_k) + h(t_i) \sum_{k=i+1}^{\infty} p_k = \frac{(1-p)}{p} \left(e^{\lambda p \left(\sum_{j=1}^{i} t_j p_j + t_i \sum_{k=i+1}^{\infty} p_k \right)} - 1 \right). \tag{3.6.1}$$

Now let X be the random variable with mass function $P(X = t_k) = p_k$ ($k = 1, 2, \ldots$) and for $i = 1, 2, \ldots$, let $X_i = \min(X, t_i)$. Then, we can rewrite Eqn. (3.6.1) as

$$E[h(X_i)] = \frac{(1-p)}{p} \left(e^{\lambda p E[X_i]} - 1 \right).$$

Note that $X_i \uparrow X$ almost surely as $i \to \infty$, so the Monotone Convergence Theorem tells us that $E[X_i] \to E[X] = E[T_I]$ as $i \to \infty$. Also, $h(X_i) \uparrow h(X)$ as $i \to \infty$, so the Monotone Convergence Theorem implies that $R_U = E[h(X)] = \lim_{i \to \infty} E[h(X_i)]$. Putting this together, we get

$$R_U = \frac{1-p}{p} \left(e^{\lambda p \mathrm{E}[T_I]} - 1 \right).$$

3.6.3 Arbitrarily distributed infectious period

Suppose that T_I has an arbitrary distribution, but with the requirement that $\mathrm{E}[T_I] < \infty$, i.e. T_I has finite mean.

For $h > 0$, let $T_h^- = h\lfloor T_I/h \rfloor$ and $T_h^+ = T_h^- + h$. Then $T_h^- \leq T_I \leq T_h^+$, and so

$$\mathrm{E}\left[T_h^-\right] \leq \mathrm{E}[T_I] \leq \mathrm{E}\left[T_h^+\right] = \mathrm{E}\left[T_h^-\right] + h,$$

which gives us

$$\mathrm{E}[T_I] - h \leq \mathrm{E}\left[T_h^-\right] \leq \mathrm{E}[T_I],$$

so as $h \downarrow 0$, $\mathrm{E}\left[T_h^-\right] \to \mathrm{E}[T_I]$ and $\mathrm{E}\left[T_h^+\right] \to \mathrm{E}[T_I]$.

We let R_h^- and R_h^+ be the type-reproduction numbers when the natural lifetime has distribution T_h^- and T_h^+, respectively. By sampling shorter natural lifetimes for R_h^-

it is clear that less unnamed offspring are produced, and by sampling longer natural lifetimes for R_h^+ it is clear that more unnamed offspring are produced. Hence,

$$R_h^- \leq R_U \leq R_h^+.$$

However, T_h^- and T_h^+ both have countable support, and hence

$$R_h^- = \frac{1-p}{p}\left(e^{\lambda p \mathrm{E}[T_h^-]} - 1\right)$$
$$R_h^+ = \frac{1-p}{p}\left(e^{\lambda p \mathrm{E}[T_h^+]} - 1\right).$$

As $h \downarrow 0$,

$$R_h^- \to \frac{1-p}{p}\left(e^{\lambda p \mathrm{E}[T_I]} - 1\right)$$
$$R_h^+ \to \frac{1-p}{p}\left(e^{\lambda p \mathrm{E}[T_I]} - 1\right).$$

Therefore,

$$R_U = \frac{1-p}{p}\left(e^{\lambda p \mathrm{E}[T_I]} - 1\right).$$

If we let $\mathrm{E}[T_I] = 1$ without loss of generality, then

$$R_U = \frac{1-p}{p}\left(e^{\lambda p} - 1\right). \tag{3.6.2}$$

3.6.4 A simpler derivation

Given that this model provides such a tidy expression for R_U, we feel there should be a tidier method of deriving it in a general case than the method which we have presented here. To gain some insight into this we present a derivation for the constant infectious period case.

3.6.5 Constant infectious period

In the constant infectious period case ($T_I \equiv \iota$), we present an alternative derivation which involves analysing the tree length of a pure birth process over $[0, \iota]$, enabling us to obtain not only the correct expression for R_U, but also the probability-generating function of the offspring random variable GWP.

Consider again our typical unnamed individual in the MBDP, A, who has an active lifetime of length ι. Recall that each individual gives birth to unnamed individuals over their active lifetime at the points of a Poisson process (independently of all other individuals) with rate $\lambda(1-p)$. A's number of offspring in the MBDP therefore has a Poisson$(\lambda(1-p)D)$ distribution, where D is the length of A's family tree of named individuals in the MBDP (i.e. the sum of the active lifetimes of A and all descendants of A who are separated from A by only named individuals), and so $R_U = \lambda(1-p)E[D]$. Further, since all of the individuals who contribute to D live until the end of A's lifetime, D is equivalent in distribution to the tree length of a pure-birth process (PBP) with one initial individual and birth rate λp over $[0, \iota]$.

Consider the initial individual in the PBP, $\hat{A}$, say. We shall now analyse D by conditioning back from time-ι to $\hat{A}'s$ last birth. First note that since there are no births over $[0, \iota]$ with probability $e^{-\lambda p \iota}$, in which case $D = \iota$.

Conditioning on $\hat{A}$ having a birth (which happens with probability $1 - e^{-\lambda p \iota}$), then the amount of time this takes place before ι has an Exp(λp) distribution restricted to $[0, \iota]$. Suppose that this last birth happens t units before ι (i.e. at time $\iota - t$), then we know that over $[\iota - t, \iota]$ (i.e. a part of the tree of length t), $\hat{A}$ has no births. The remainder of the tree length is determined by the first $\iota - t$ time units of the $\hat{A}$'s lifetime, and the t time units of the offspring's lifetime (which begins at time $\iota - t$). Since individuals give birth

uniformly over their lifetimes, this is equivalent to just one individual with lifetime ι, and hence the remaining tree length is independent of and identically distributed to D. For an example of how this method works, see Figure 3.2.

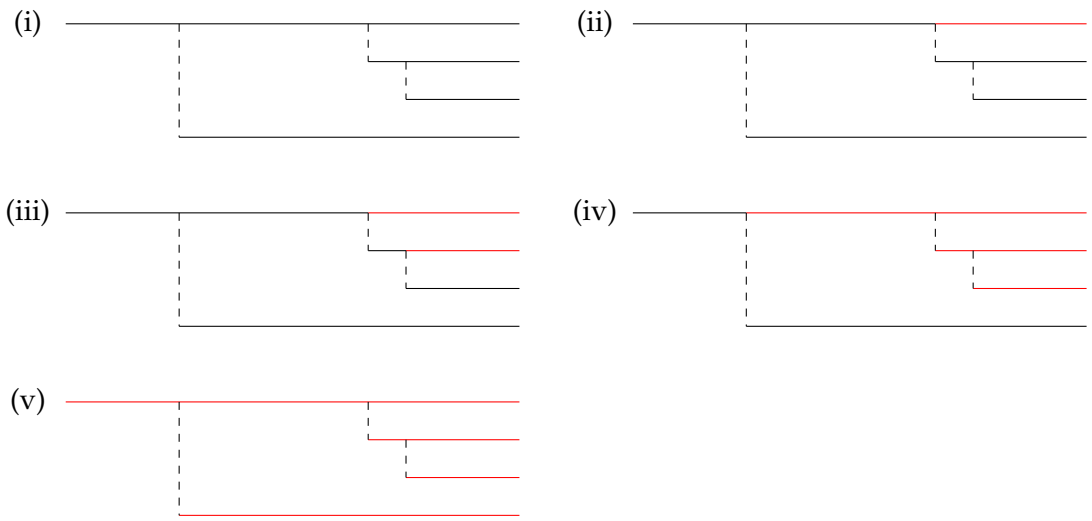

Figure 3.2: An example of the 'conditioning-back' method used for the constant infectious period. In the tree, active lifetimes are represented by solid lines with dashed lines indicating births. Each time we condition back to a birth from (i) to (v), we add a length of time to the total counted (in red), but until there are no more births, it is always possible to trace a full lifetime back through the tree in black.

Putting this together we get

$$\begin{aligned}\mathrm{E}[D] &= \iota e^{-\lambda p \iota} + \int_0^{\iota} \lambda p e^{-\lambda p t}(t + \mathrm{E}[D])dt \\ &= \iota e^{-\lambda p \iota} + \frac{1}{\lambda p} - \frac{1}{\lambda p} e^{-\lambda p \iota} - \iota e^{-\lambda p \iota} + \left(1 - e^{-\lambda p \iota}\right) \mathrm{E}[D]\end{aligned}$$

which on rearranging gives

$$\mathrm{E}[D] = \frac{1}{\lambda p}\left(e^{\lambda p \iota} - 1\right),$$

and thus (setting $\iota = 1$ without loss of generality)

$$R_U = \frac{1-p}{p}\left(e^{\lambda p} - 1\right),$$

which is the same expression as Eqn. (3.6.2).

Further, we have that since $R \sim \text{Poisson}(\lambda(1-p)D)$, $H(s) = \mathrm{E}\left[s^R\right] = \psi(\lambda(1-p)(1-s))$, where $\psi(\theta) = \mathrm{E}\left[e^{-\theta D}\right]$ and conditioning on the last birth as above, we have, for $\theta \geq 0$,

$$\begin{aligned}\psi(\theta) &= e^{-\lambda p\iota}e^{-\theta\iota} + \int_0^\iota \lambda p e^{-\lambda p t}e^{-\theta t}\psi(\theta)dt \\ &= e^{-(\lambda p+\theta)\iota} + \frac{\lambda p}{\lambda p+\theta}\left(1 - e^{-(\lambda p+\theta)\iota}\right)\psi(\theta),\end{aligned}$$

whence

$$\psi(\theta) = \frac{e^{-(\lambda p+\theta)\iota}}{1 - \frac{\lambda p}{\lambda p+\theta}\left(1 - e^{-(\lambda p+\theta)\iota}\right)},$$

and

$$H(s) = \frac{e^{-\lambda(1-s+ps)\iota}}{1 - \frac{p}{1-s+ps}\left(1 - e^{-\lambda(1-s+ps)\iota}\right)}.$$

The method of looking back along the tree in this manner has been used by, for example, Lambert [35], who describes a *contour process* for a splitting tree, which moves backwards along the tree until it hits a birth (of a different individual), at which point it jumps to the death of the individual corresponding to that birth. It is also shown that the *coalescence levels* (time back to the point of common ancestry) between consecutive (in a *linear order*, which is defined, and which we are implicitly using here) individuals alive at a certain time, are independent and identically distributed. This corresponds here to the time lengths we are conditioning back to a birth being independent and identically distributed.

Note further that we can also obtain the distribution of the number of descendants of $\hat{A}$ born in the PBP over $[0, \iota]$: this is the number of times we have to condition back

before we find no offspring, and hence this has a Geometric distribution with success probability $e^{-\lambda p \iota}$. The Geometric nature of the size of a birth-death process at a specific time has previously been noted by Kendall [32]. In an epidemics context, Trapman and Bootsma [43] showed that the size of the appropriate approximating branching process at the time of the first detection of an SIR epidemic with detections has a Geometric distribution.

3.7 Iterative tracing, with latent period and delay

Here we see how the iterative tracing can extend to incorporate a latent period and delay, in the case of Exponential infectious periods.

3.7.1 Exponential infectious period

Suppose that the infectious periods and delays are exponentially distributed (i.e. $T_I \sim$ Exp(γ) and $T_D \sim$ Exp(ξ), and that the latent periods have an arbitrary distribution T_L, with distribution measure μ_L and known moment-generating function $\phi_L(\theta)$ ($\theta \geq 0$).

Recall that

$$\begin{aligned} h_k(t) &= \mathrm{E}\left[R^{(k)} \middle| T = t \right] \\ &= \lambda(1-p)t + \lambda p t g_k(t), \end{aligned}$$

where $g_k(t) = \mathrm{E}\left[Z^{(k)} \middle| T = t \right]$, and $Z^{(k)}$ is the total number of descendants that contribute to $R^{(k)}$ from a typical type-1 individual. As in the analogous single-step case (see Section 3.5.4) we let $T_{B,D}$ and $T_{B,L}$ be the tracing delay and latent period of B, respectively, and we let $W = \max\{0, V + T_{B,D} - T_{B,L}\}$. Now, since both traced and

untraced individuals can name offspring, we have that

$$(Z^{(k)}|W=w,T_B=t_B) =^D \begin{cases} R^{(k-1)}|T=t_B & (t_B<w) \\ R^{(k-1)}|T=w & (t_B\geq w). \end{cases}$$

Putting this all together yields

$$\begin{aligned} g_k(t) = &\int_{t_D=0}^{\infty} \xi e^{-\xi t_D} \int_{t_L=t_D}^{t_D+t} \int_{w=0}^{t+t_D-t_L} \frac{1}{t}\chi(w)\, dw\, d\mu_L(t_L)\, dt_D \\ &+ \int_{t_D=0}^{\infty} \xi e^{-\xi t_D} \int_{t_L=0}^{t_D} \int_{w=t_D-t_L}^{t+t_D-t_L} \frac{1}{t}\chi(w)\, dw d\mu_L(t_L)\, dt_D, \end{aligned} \tag{3.7.1}$$

where

$$\chi(w) = \left\{ e^{-\gamma w} h_{k-1}(w) + \int_{u=0}^{w} \gamma e^{-\gamma u} h_{k-1}(u) du \right\},$$

and multiplying Equation(3.7.1) by t gives

$$\begin{aligned} tg_k(t) = &\int_{t_D=0}^{\infty} \xi e^{-\xi t_D} \int_{t_L=t_D}^{t_D+t} \int_{w=0}^{t+t_D-t_L} \chi(w)\, dw\, d\mu_L(t_L)\, dt_D \\ &+ \int_{t_D=0}^{\infty} \xi e^{-\xi t_D} \int_{t_L=0}^{t_D} \int_{w=t_D-t_L}^{t+t_D-t_L} \chi(w)\, dw d\mu_L(t_L)\, dt_D. \end{aligned}$$

We let $L_k(\theta) = \int_0^\infty e^{-\theta t} h_k(t) dt$, and noting that

$$\begin{aligned} &\int_{t=0}^{\infty} e^{-\theta t} \int_{t_D=0}^{\infty} \xi e^{-\xi t_D} \int_{t_L=t_D}^{t_D+t} \int_{w=0}^{t+t_D-t_L} \chi(w)\, dw\, d\mu_L(t_L)\, dt_D\, dt \\ &= \int_{t_D=0}^{\infty} \xi e^{-\xi t_D} \int_{t_L=t_D}^{\infty} \int_{t_1=0}^{\infty} e^{-\theta(t_1-t_D+t_L)} \int_{w=0}^{t_1} \chi(w)\, dw\, dt_1\, d\mu_L(t_L)\, dt_D \\ &= \int_{t_D=0}^{\infty} \xi e^{-\xi t_D} \int_{t_L=t_D}^{\infty} \int_{w=0}^{\infty} \chi(w) \int_{t_1=w}^{\infty} e^{-\theta(t_1-t_D+t_L)}\, dt_1\, dw\, d\mu_L(t_L)\, dt_D \\ &= \frac{1}{\theta} \int_{t_L=0}^{\infty} e^{-\theta t_L} \int_{t_D=0}^{t_L} \xi e^{-(\xi-\theta)t_D} \int_{w=0}^{\infty} e^{-\theta w} \chi(w)\, dw\, dt_D\, d\mu_L(t_L) \\ &= \frac{1}{\theta} \int_{t_L=0}^{\infty} e^{-\theta t_L} \int_{t_D=0}^{t_L} \xi e^{-(\xi-\theta)t_D} \left(L_{k-1}(\theta+\gamma) + \frac{\gamma}{\theta} L_{k-1}(\theta+\gamma) \right) dt_D\, d\mu_L(t_L) \\ &= \begin{cases} \frac{\xi(\theta+\gamma)}{\theta^2(\xi-\theta)} \int_{t_L=0}^{\infty} \left(e^{\theta t_L} - e^{-\xi t_L} \right) L_{k-1}(\theta+\gamma)\, d\mu_L(t_L) & \text{if } \theta \neq \xi \\ \frac{\xi+\gamma}{\xi} \int_{t_L=0}^{\infty} t_L e^{-\xi t_L} L_{k-1}(\xi+\gamma)\, d\mu_L(t_L) & \text{if } \theta = \xi \end{cases} \\ &= \begin{cases} \frac{\xi(\theta+\gamma)(\phi_L(\theta)-\phi_L(\xi))}{\theta^2(\xi-\theta)} L_{k-1}(\theta+\gamma) & \text{if } \theta \neq \xi \\ -\frac{xi+\gamma}{\xi} \phi_L'(\xi) L_{k-1}(\xi+\gamma) & \text{if } \theta = \xi, \end{cases} \end{aligned}$$

and further that

$$
\begin{aligned}
&\int_{t=0}^{\infty} e^{-\theta t} \int_{t_D=0}^{\infty} \xi e^{-\xi t_D} \int_{t_L=0}^{t_D} \int_{w=t_D-t_L}^{t+t_D-t_L} \chi(w)\, dw\, d\mu_L(t_L)\, dt_D\, dt \\
&= \int_{t=0}^{\infty} e^{-\theta t} \int_{t_L=0}^{\infty} \int_{t_D=t_L}^{\infty} \xi e^{-\xi t_D} \int_{w=t_D-t_L}^{t+t_D-t_L} \chi(w)\, dw\, dt_D\, d\mu_L(t_L)\, dt \\
&= \int_{t=0}^{\infty} e^{-\theta t} \int_{t_L=0}^{\infty} e^{-\xi t_L} \int_{t'_D=0}^{\infty} \xi e^{-\xi t'_D} \int_{w=t'_D}^{t+t'_D} \chi(w)\, dw\, dt'_D\, d\mu_L(t_L)\, dt \\
&= \phi_L(\xi) \int_{t=0}^{\infty} e^{-\theta t} \int_{t'_D=0}^{\infty} \xi e^{-\xi t'_D} \int_{w=t'_D}^{t+t'_D} \chi(w)\, dw\, dt'_D\, dt \\
&= \phi_L(\xi) \int_{t'_D=0}^{\infty} \xi e^{-\xi t'_D} \int_{w=t'_D}^{\infty} \chi(w) \int_{t=w-t'_D}^{\infty} e^{-\theta t}\, dt\, dw\, dt'_D \\
&= \frac{\phi_L(\xi)}{\theta} \int_{w=0}^{\infty} e^{-\theta w} \chi(w) \int_{t'_D=0}^{w} \xi e^{-(\xi-\theta)t'_D}\, dt'_D\, dw \\
&= \begin{cases} \frac{\xi\phi_L(\xi)}{\theta(\xi-\theta)} \int_{w=0}^{\infty} \left(e^{-\theta w} - e^{-\xi w}\right) \left\{e^{-\gamma w} h_{k-1}(w) + \int_{u=0}^{w} \gamma e^{-\gamma u} h_{k-1}(u) du\right\} dw & \text{if } \theta \neq \xi \\ \frac{\phi_L(\xi)}{\xi} \int_{w=0}^{\infty} w e^{-\xi w} \left\{e^{-\gamma w} h_{k-1}(w) + \int_{u=0}^{w} \gamma e^{-\gamma u} h_{k-1}(u) du\right\} dw & \text{if } \theta = \xi \end{cases} \\
&= \begin{cases} \frac{\xi\phi_L(\xi)}{\theta(\xi-\theta)} \left\{\frac{\theta+\gamma}{\theta} L_{k-1}(\theta+\gamma) - \frac{\xi+\gamma}{\xi} L_{k-1}(\xi+\gamma)\right\} & \text{if } \theta \neq \xi \\ -\frac{\xi+\gamma}{\xi} \phi_L(\xi) L'_{k-1}(\xi+\gamma) & \text{if } \theta = \xi \end{cases},
\end{aligned}
$$

it follows that, for $\theta \neq \xi$

$$
\begin{aligned}
L_k(\theta) &= \frac{\lambda(1-p)}{\theta^2} + \frac{\lambda p \xi(\theta+\gamma)\left(\phi_L(\theta) - \phi_L(\xi)\right)}{\theta^2(\xi-\theta)} L_{k-1}(\theta+\gamma) \\
&\quad + \frac{\lambda p \xi \phi_L(\xi)}{\theta(\xi-\theta)} \left\{\frac{\theta+\gamma}{\theta} L_{k-1}(\theta+\gamma) - \frac{\xi+\gamma}{\xi} L_{k-1}(\xi+\gamma)\right\} \\
&= \frac{\lambda(1-p)}{\theta^2} + \frac{\lambda p \xi}{\theta(\xi-\theta)} \left\{\frac{(\theta+\gamma)\phi_L(\theta)}{\theta} L_{k-1}(\theta+\gamma) - \frac{(\xi+\gamma)\phi_L(\xi)}{\xi} L_{k-1}(\xi+\gamma)\right\}.
\end{aligned}
$$

Note that it can be shown in a similar way to Section 3.5.4, that in the case where $\theta = \xi$, we can obtain $L_k(\xi)$ by taking the limit $\theta \to \xi$. We assume now that $\theta \neq \xi$ and that ξ is non-integer, while integer values of ξ can be considered by taking the limit as ξ approaches the integer.

Setting $\gamma = 1$ without loss of generality,

$$
L_k(\theta) = \frac{\lambda(1-p)}{\theta^2} + \frac{\lambda p \xi}{\theta(\xi-\theta)} \left\{\frac{(\theta+1)\phi_L(\theta)}{\theta} L_{k-1}(\theta+1) - \frac{(\xi+1)\phi_L(\xi)}{\xi} L_{k-1}(\xi+1)\right\}.
$$

If $y_{k,j} = L_k(\xi + j)$ ($k = 0, 1, \ldots$, $j = 1, 2, \ldots$), then $y_{0,j} = \frac{\lambda(1-p)}{(\xi+j)^2}$, and for $k = 1, 2, \ldots$,

$$y_{k,j} = \alpha_j + \hat{\rho}_j y_{k-1,1} - \beta_j y_{k-1,j+1}, \tag{3.7.2}$$

where

$$\begin{aligned}
\alpha_j &= \frac{\lambda(1-p)}{(\xi+j)^2} \\
\hat{\rho}_j &= \frac{\lambda p(\xi+1)\phi_L(\xi)}{j(\xi+j)} \\
\beta_j &= \frac{\lambda p\xi(\xi+j+1)\phi_L(\xi+j)}{j(\xi+j)^2}
\end{aligned}$$

and we shall let $\delta_0 = 1$, $\delta_j = \prod_{i=1}^{j} \beta_i$ ($j = 1, 2, \ldots$).

This is of the same form as Eqn. (3.5.12), and hence, either $y_1 = \infty$ or $y_1 = y_1^*$, where,

$$y_1^* = \frac{\sum_{j=0}^{\infty}(-1)^j \delta_j \alpha_{j+1}}{1 - \sum_{i=0}^{\infty}(-1)^i \delta_i \hat{\rho}_{i+1}}.$$

Conditions for y_1 to be finite can be obtained in the same fashion as in Section 3.5.4.

If $x_{k,j} = L_k(j)$ (for $k = 0, 1, \ldots$, $j = 1, 2, \ldots$) then $x_{0,j} = \frac{\lambda(1-p)}{j^2}$, and, for $k = 1, 2, \ldots$,

$$x_{k,j} = a_j - \rho_j y_{k-1,1} + b_j x_{k-1,j+1}, \tag{3.7.3}$$

where

$$\begin{aligned}
a_j &= \frac{\lambda(1-p)}{j^2} \\
\rho_j &= \frac{\lambda p(\xi+1)\phi_L(\xi)}{j(\xi-j)} \\
b_j &= \frac{\lambda p\xi(j+1)\phi_L(j)}{j^2(\xi-j)}.
\end{aligned}$$

This is of the same form as Eqn. 3.5.15, and so, when $y_1 < \infty$,

$$\begin{aligned}
R_U &= x_1 \\
&= \sum_{i=0}^{\infty} c_i \left(a_{i+1} - \rho_{i+1} y_1\right),
\end{aligned}$$

where $c_0 = 1$ and $c_j = \prod_{i=1}^{j} b_i$ ($j = 1, 2, \ldots$), otherwise R_U is infinite.

3.8 Numerical illustrations

In this section we simulate finite-size epidemics to examine the reliability of the branching process approximation used in this chapter, and then use numerical results to illustrate the theory of this chapter and examine the effect of dependencies within the model.

3.8.1 Comparison with simulations

While we consider the population size, n, to be large to enable us to use branching process approximations to analyse our epidemic model, in real life n is always finite, and often not 'large' in a mathematical sense. Hence, it is of interest to examine how quickly the approximation becomes a valid description of the true epidemic model. Figures 3.3 (single-step case) and 3.4 (iterative case) show the final size (i.e. total number of removals) distributions from $100,000$ simulations for population sizes 20, 50, 100 and 200 for the constant infectious period case (no latent period or delay), with $\lambda = 2$ and $p = 0.5$. For the final size of an epidemic, we expect to see a bimodal distribution, with one mode corresponding to *minor outbreaks* (i.e. a small proportion of the population is infected) and a second corresponding to *major outbreaks* (i.e. a significant proportion is infected). We see this behaviour even for $n = 20$, but there is not a clear distinction between outbreaks minor and major until about $n = 200$.

As $n \to \infty$, the proportion of minor outbreaks should tend to the theoretical extinction probability for the branching process approximation, p_E. Thus, for n large enough we estimate extinction probability as the proportion of outbreaks that are minor. In Figure 3.5 we plot these estimate extinction probabilities ($\hat{p}_E$) with confidence intervals given as $\hat{p}_E \pm 2SE$ where the standard error is $SE = \left(\frac{(1-\hat{p}_E)\hat{p}_E}{n_0}\right)^{\frac{1}{2}}$ ($n_0 = 100,000$ is the num-

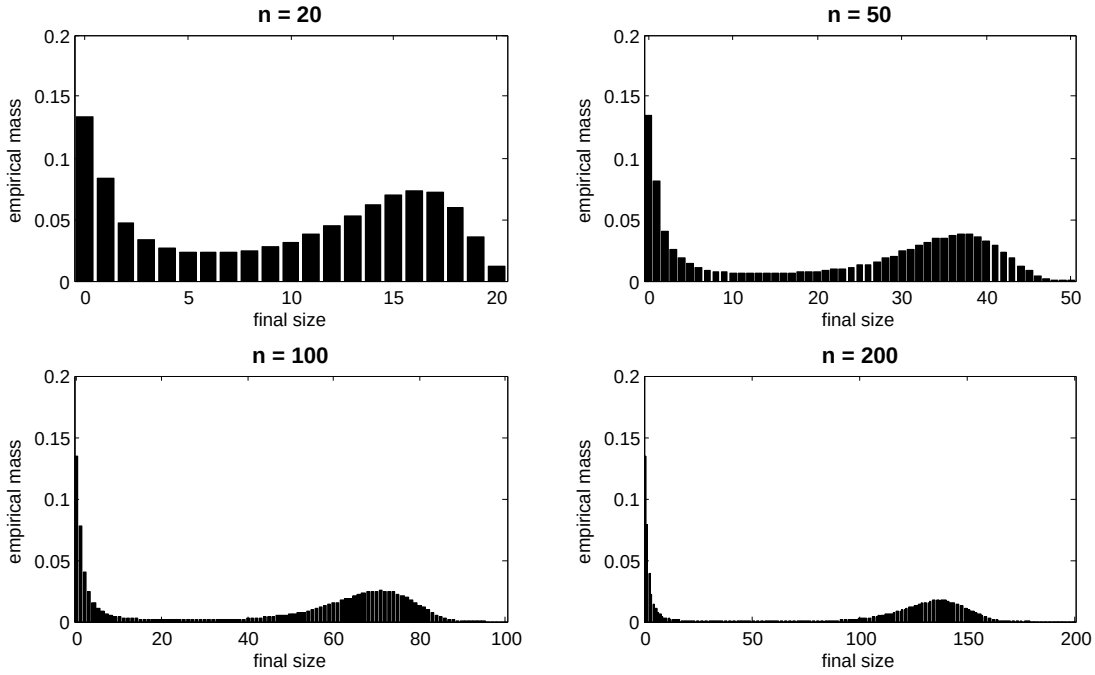

Figure 3.3: Final size distributions from 100,000 simulations, when $T_I \equiv 1$, $\lambda = 2$ and $p = 0.5$ (single-step tracing)

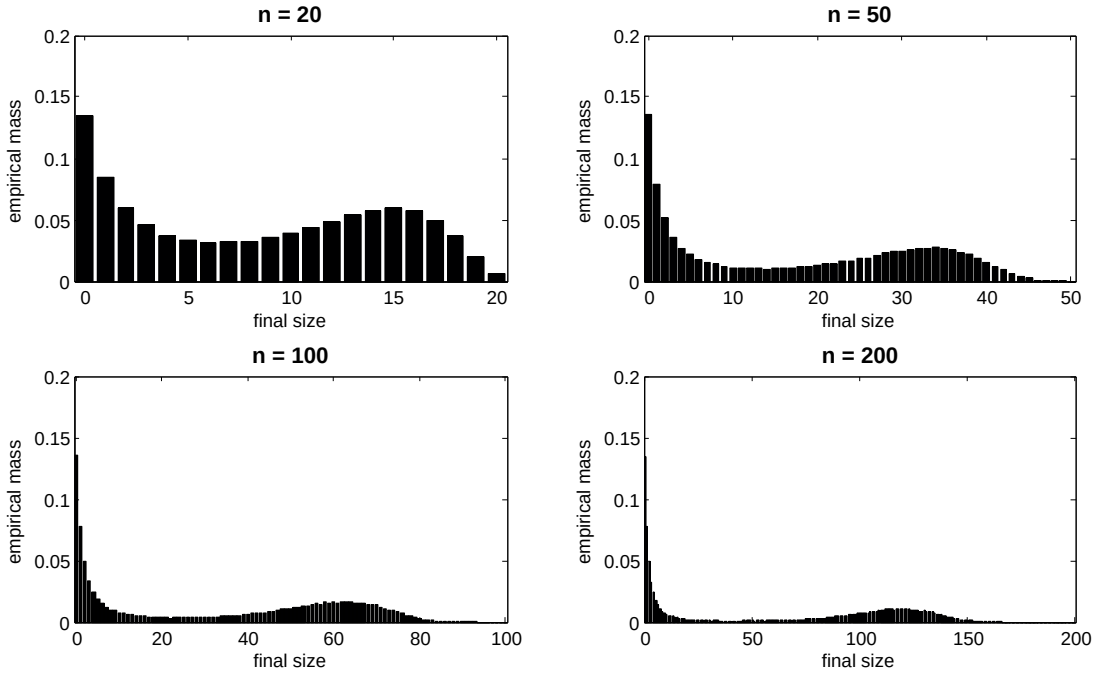

Figure 3.4: Final size distributions from 100,000 simulations, when $T_I \equiv 1$, $\lambda = 2$ and $p = 0.5$ (iterative tracing)

ber of simulations). These estimates are obtained by plotting the final size distributions of the simulations, determining a cut-off between major and minor outbreaks by sight, and letting the proportion of outbreaks that are minor be our estimate. The extinction probabilities in the limit $n \to \infty$ are known to be 0.3236 in the single-step case and 0.4064 in the iterative case (which are represented on the plots by solid lines). In the single-step case, 0.3236 is consistently in the confidence interval above $n = 800$, while in the iterative case, 0.4064 is above $n = 1600$. This slower convergence to the limiting behaviour in the iterative case may be less to do with the model and more to do with a higher extinction probability here (λ was reduced in the single-step case to achieve a similar extinction probability to the iterative case here and it was seen that convergence was indeed slower). However, in both cases, even for $n = 200$ the estimates are reasonably close to the asymptotic value. In the limit $n \to \infty$, there is always infinitely many susceptibles, and thus infections do not affect a reduction in the number of susceptibles. For small n, infections noticeably reduce the number of susceptibles, and the probability of new infections, and so there will be more minor outbreaks. This is why we see the extinction probability being overestimated for small n.

3.8.2 Convergence of R_U with delays

In the case where $T_I \sim \text{Exp}(1)$, $T_D \sim \text{Exp}(\xi)$ and T_L has an arbitrary distribution (Section 3.5.4), we showed how conditions for the finiteness of R_U can be obtained. Here we illustrate how this works in practice.

Figure 3.6 shows a plot of y_1^* varying with λ for $T_L \equiv 0$ (note that no latent period gives an upper bound for R_U for arbitrary latent period distribution), $p = 1$ and $\xi = 0.7$. We see that y_1^* increases monotonically before 'blowing-up' for the first time (this occurs at

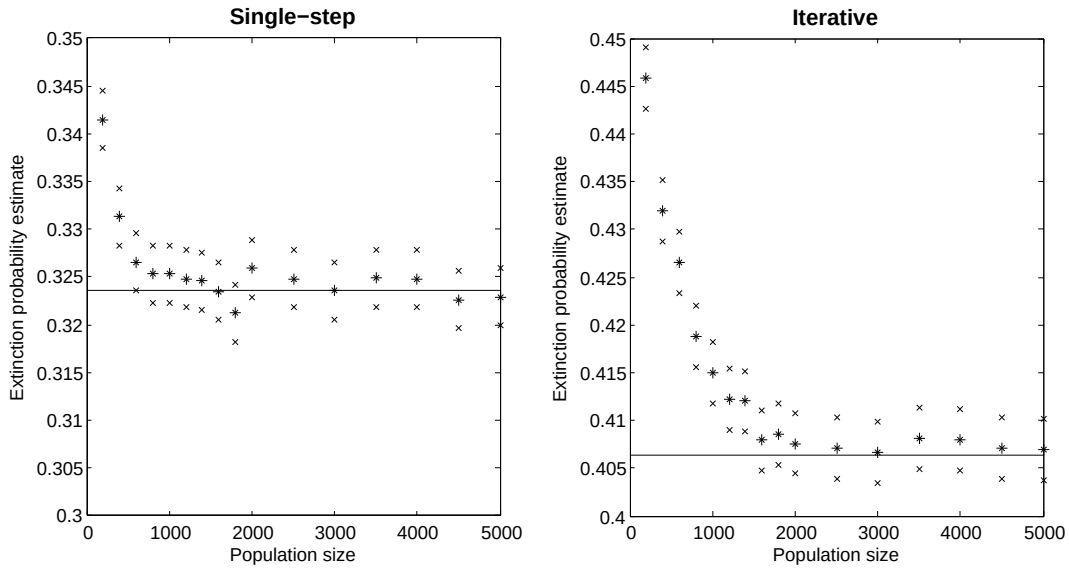

Figure 3.5: Estimated extinction probabilities (represented by asterisks) from $100,000$ simulations, when $T_I \equiv 1$, $\lambda = 2$ and $p = 0.5$. Crosses represent two standard errors above and below the estimate and solid lines represent the true asymptotic extinction probabilities.

around $\lambda = 1.9876$). It then becomes negative, but y_1 is non-negative and so must then be ∞ (and so also is R_U). Since y_1 is non-decreasing in λ, y_1 must be infinite always after this, even if y_1^* is positive (which it would appear is possible). So in this case, y_1 and R_U are finite if and only if $\lambda \in [0, \lambda_\infty)$, where $\lambda_\infty \approx 1.9876$.

Figure 3.6 also shows a plot of R_U varying with λ (note that we are assuming R_U is indeed finite for $\lambda \in [0, 1.9876)$). We see that R_U blows up as it gets closer to 1.9876, i.e. as $y_1^* \to \infty$ for the first time. We get a similar occurrence with other parameter values/latent period distributions.

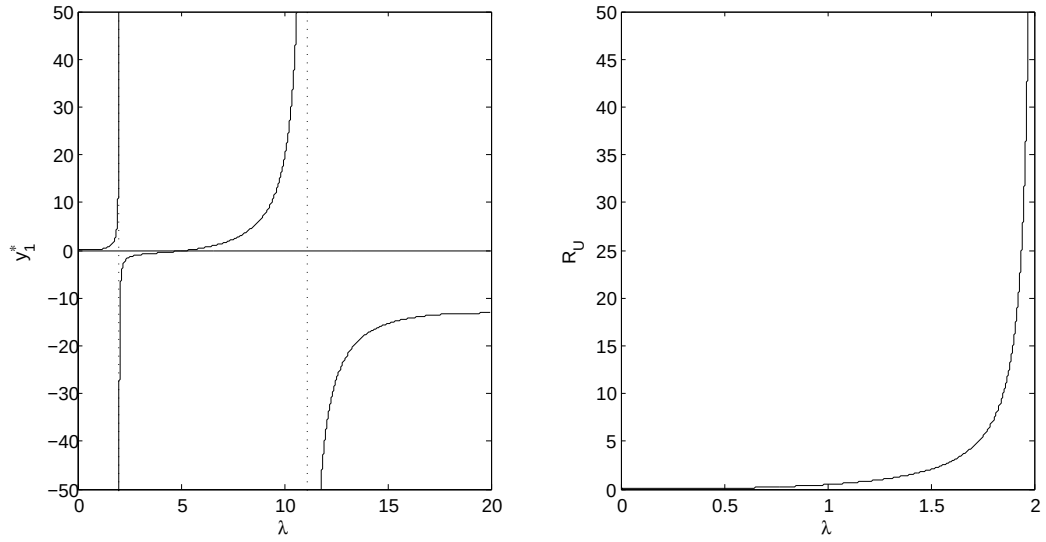

Figure 3.6: y_1^* and R_U varying with λ, when $T_I \sim \text{Exp}(1)$, $T_D \sim \text{Exp}(0.7)$, $T_L \equiv 0$ and $p = 1$.

3.8.3 Analysis of model assumptions

We have derived expressions for a type-reproduction number, R_U, which differs from the more traditional reproduction number, R_0, in that the counting of offspring is only of those in an embedded process - many individuals are not directly being counted. As such, it is less obvious how to interpret differing values of R_U - if R_U is twice as large, does that necessarily mean the epidemic is twice as bad? However, recall that the epidemic will die out with probability 1 if and only if $R_U < 1$. Hence, to study the model numerically, we do so by examining λ_{crit}, the critical contact rate, i.e. the contact rate which gives $R_U = 1$.

How λ_{crit} varies with the naming probability, p, is shown in Figure 3.7 for the no latent period, no delay cases: single-step tracing with exponential and constant infectious periods, and iterative tracing (recall that R_U depends on the infectious period distribution only through its mean, and hence so does λ_{crit}). As we would expect, λ_{crit} is higher

when the infectious period distribution is Exponential, and the range of values of λ, for which the Exponential case is supercritical while the constant case is subcritical, increases with p.

As $p \to 1$, $\lambda_{crit} \to \infty$ in the iterative case, since everyone is being named and so there are no unnamed offspring. Note however, that we do still have extinction with probability 1 as the epidemic ends when the initial individual's infectious period ends. We can see that the effectiveness of iterative tracing over single-step tracing may be minor for small p, but increases greatly as p does.

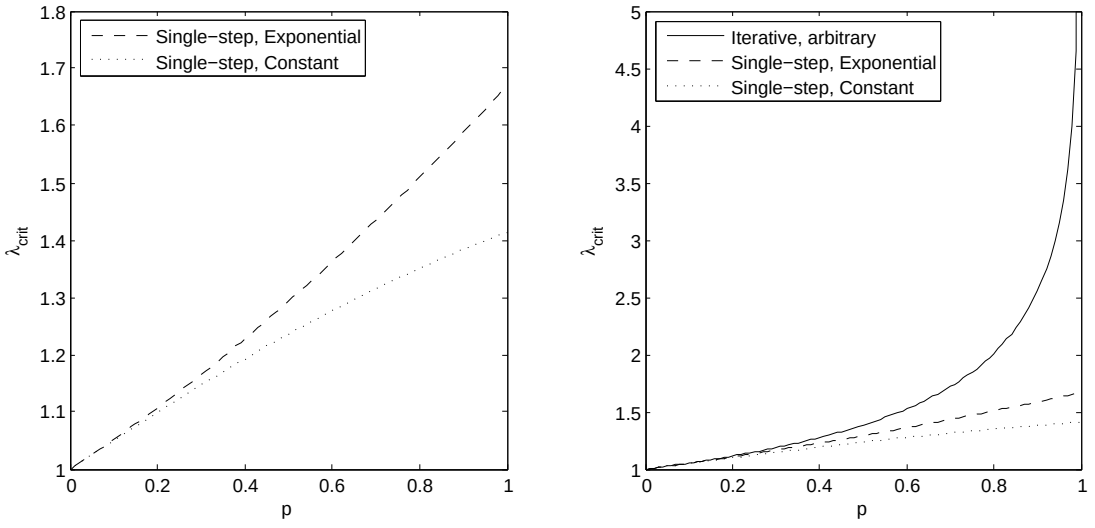

Figure 3.7: λ_{crit} varying with p, for single-step tracing (Exponential and Constant infectious periods) and iterative tracing (arbitrary infectious period) when $\mathrm{E}[T_I] = 1$.

Figure 3.8 compares the single-step cases (constant and exponentially-distributed infectious periods), by evaluating the extinction probability, p_E, in the constant case at the critical contact rate in the exponential case for the corresponding value of p (the critical contact rate being the extreme-most value for which we know the extinction probability, i.e. it is 1). We see that while for low values of p the extinction probabil-

ity is close to 1, it drops down to 0.7008 when $p = 1$. This difference becomes even more pronounced when there are $k > 1$ initial unnamed infectives, with an extinction probability of 0.7008^k.

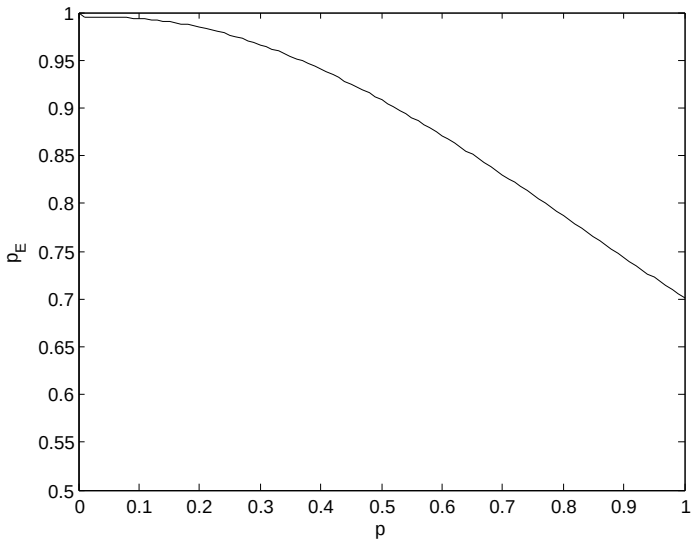

Figure 3.8: p_E varying with p in the single-step constant infectious period case, evaluated at λ_{crit} for the single-step exponential infectious period case.

While we do not have an analytical expression for obtaining p_E in subcritical exponential cases, we can estimate it by simulating the offspring random variable in the GWP (though it is always more preferable to have analytical results since they are more accurate and generally involve significantly less computation time). In Figure 3.9, we examine how p_E varies with λ, using the analytical values in the constant infectious period cases and estimated values from simulations in the exponential infectious period cases (any 'wiggles' exhibited in the curve arise from randomness, we would expect the true curve to be smoother). These estimates are obtained by simulating the offspring random variable 100,000 times to obtain an empirical distribution for it, then our estimate is given as the solution of $\bar{H}(s) = s$ in $(0, 1)$ where $\bar{H}(s)$ is the empirical pgf of the offspring random variable. There is a clear difference between the iterative

and single-step cases when $p = 1$ (and $p_E = 1$ in the iterative case), but this becomes very small when $p = 0.5$. However, both when $p = 0.5$ and when $p = 1$, the difference between the exponential and constant cases is pronounced.

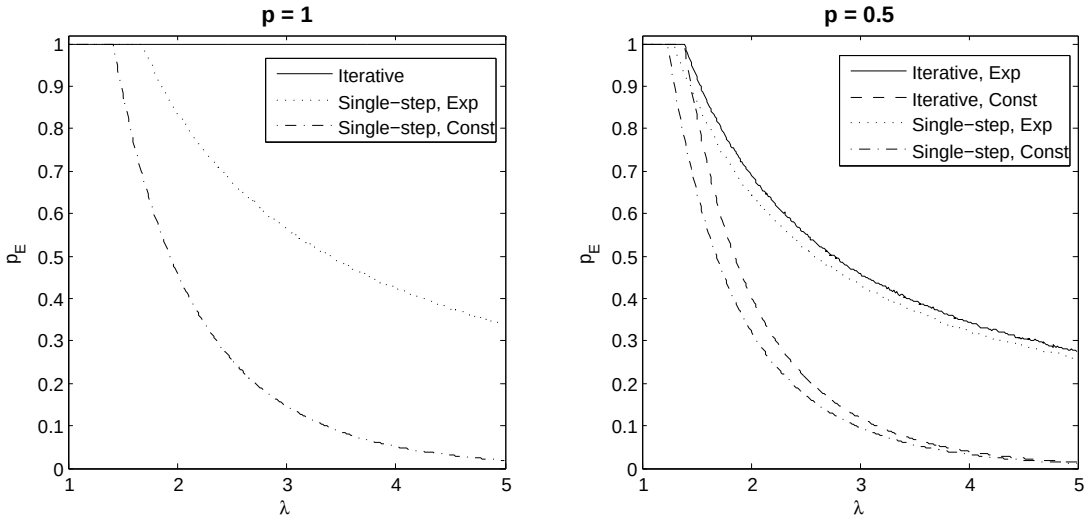

Figure 3.9: p_E varying with λ, when $\mathrm{E}[T_I] = 1$ and $\mathrm{E}[T_D] = \mathrm{E}[T_L] = 0$.

Figure 3.10 shows the effect of latent period distribution choice in the model, plotting λ_{crit} against the latent period mean for the latent period distributions: Exponential, Gamma (with shape parameter $\kappa = 2, 3, 5, 10$) and Constant. Note that here we use the definition for a Gamma distribution with shape parameter κ and mean μ here as a distribution having moment-generating function $\left(1 + \frac{\mu}{\kappa}\theta\right)^{\kappa}$ for $\theta \geq 0$. The infectious period has an Exp(1) distribution and the delay has an Exp(ξ) distribution. We can see that the effects of choosing a different latent period distribution increase as the latent period mean increases, as the delay mean decreases and as the naming probability increases. The difference between Exponential and Constant latent periods (two extremes of the Gamma distribution, $\kappa = 1$ and $\kappa = \infty$, respectively) is clearly distinct for given values of the other parameters. As the latent period mean increases, λ_{crit} increases. We would expect this behaviour as a longer latent period increases the likelihood of an

individual being traced, and further they serve less of their natural infectious period if they are traced.

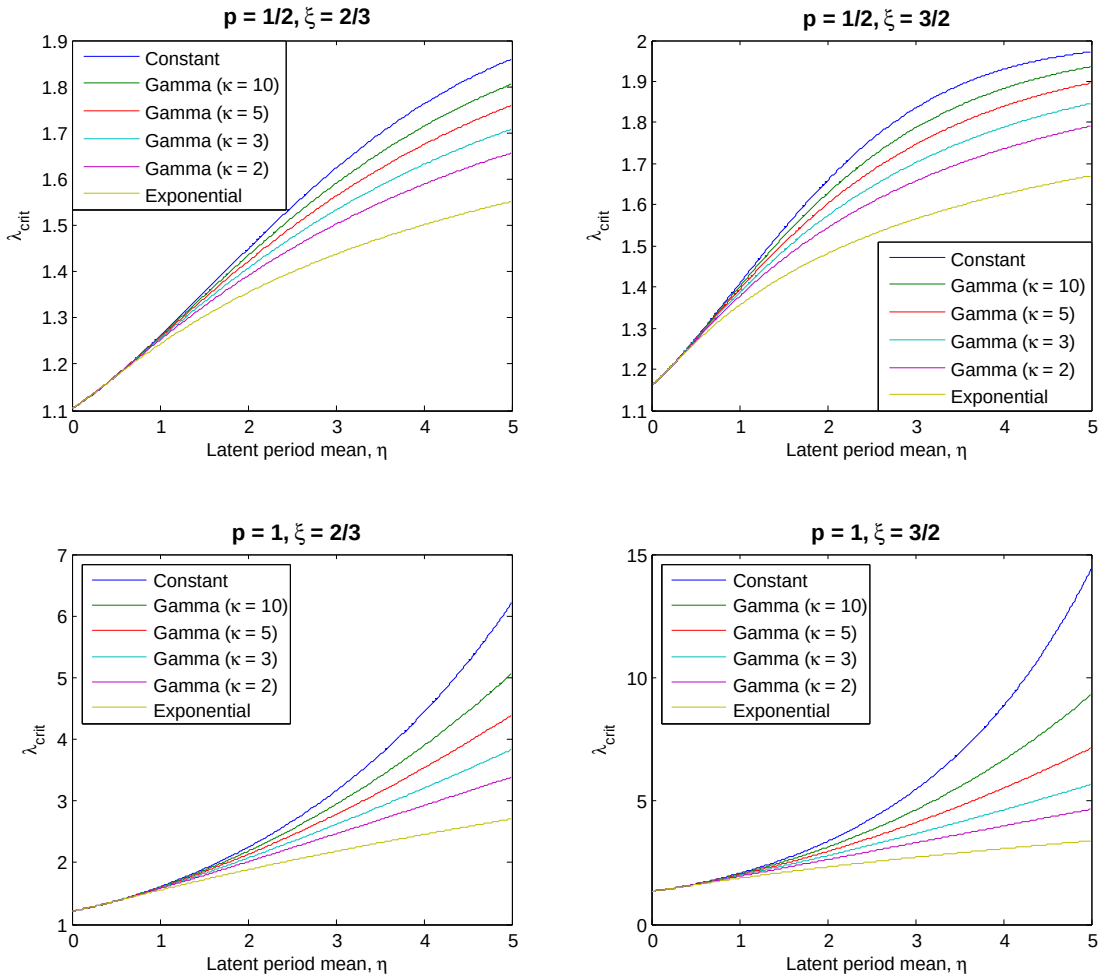

Figure 3.10: λ_{crit} varying with latent period mean for different latent period distributions, when $T_I \sim \text{Exp}(1)$ and $T_D \sim \text{Exp}(\xi)$.

Figure 3.11 shows the effect of delay distribution choice in the model, plotting λ_{crit} against the delay mean for the delay distributions: Exponential, Gamma (with shape parameter $\kappa = 2, 3, 5, 10$) and Constant. The infectious period is constant ($T_I \equiv 1$) and the latent period has an Exp(μ) distribution. We can see that the effects of choosing a different delay distribution increase as the delay mean increases from zero (though as the delay mean tends to infinity, $\lambda_{crit} \to 1$, irrespective of the exact distribution), and as the naming probability increases. It would appear that as the shape parameter κ of the

Gamma distribution increases, there is much slower convergence of λ_{crit} to that in the constant case for smaller μ (i.e. longer latent periods). As delay mean increases, λ_{crit} decreases. We would expect this as a longer delay means an individual is less likely to be traced, while if they are they serve more of their natural infectious period.

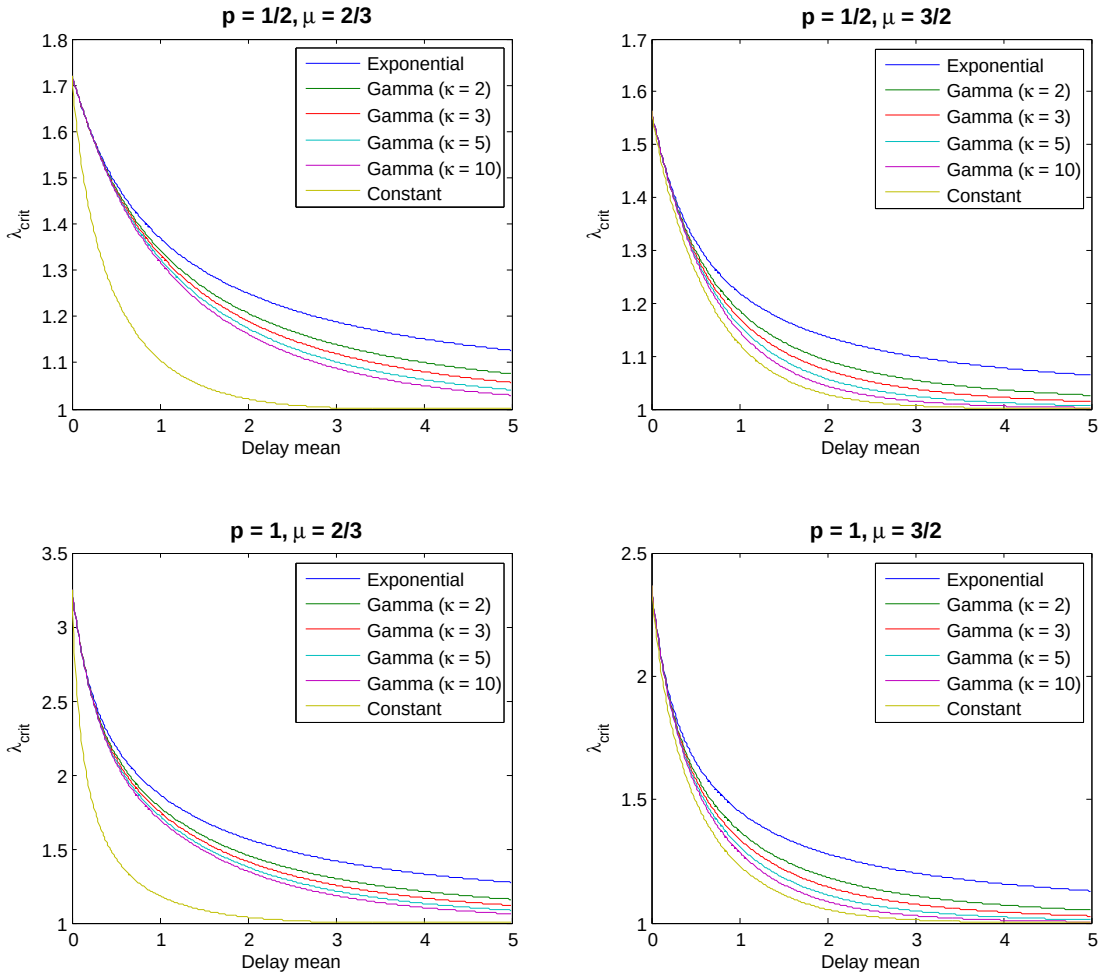

Figure 3.11: λ_{crit} varying with delay mean for different delay distributions, when $T_I \equiv 1$ and $T_L \sim \text{Exp}(\mu)$.

3.8.4 Effect of dependencies within the tracing model

In Figure 3.12 we compare the true tracing model with the two approximations discussed in Sections 3.5.3 (a 2-type approximation) and 3.5.3 (a multitype approximation), when $T_I \sim \text{Exp}(1)$. The multitype approximation has the same critical contact

rate as the true model, while the 2-type approximation is fairly close.

We can obtain the extinction probability numerically in the 2-type approximation by letting $\left(q_U^{(0)}, q_N^{(0)}\right) = (0,0)$, and letting, for $k \geq 1$,

$$q_U^{(k)} = \frac{1}{1+\lambda(1-p)\left(1-q_U^{(k-1)}\right)+\lambda p\left(1-q_N^{(k-1)}\right)},$$
$$q_N^{(k)} = \frac{2}{2+\frac{\lambda}{2}(2-p)\left(1-q_U^{(k)}\right)+\frac{\lambda p}{2}\left(1-q_N^{(k-1)}\right)},$$

then the extinction probability is given by $\lim_{k\to\infty} q_U^{(k)}$.

In the multitype approximation, note that from Eqn. (3.5.9), we can see that as $k \to \infty$, $q_k \to 1$, and so we are able to solve for q_0 numerically using a truncation method. We let, for $i = 1, 2, \ldots$, $q_0^{(i,0)} = 0$ and $q_i^{(i,j)} = 1$ (for $j = 0, 1, \ldots$), and then, for $i = 1, 2, \ldots$, $j = 1, 2, \ldots$ and $k = 0, 1, \ldots, i-1$,

$$q_k^{(i,j)} = \frac{(k+1)\gamma}{(k+1)\gamma+\frac{\lambda(1-p+k)}{k+1}\left(1-q_0^{(i,j-1)}\right)+\frac{\lambda p}{k+1}\left(1-q_{k+1}^{(i,j-1)}\right)} \qquad (k = 0, 1, 2, \ldots).$$

and our extinction probability is given by $\lim_{i\to\infty}\lim_{j\to\infty} q_0^{(i,j)}$.

For smaller supercritical values of λ the more sophisticated multitype approximation is close to giving the true extinction probability (we have used an estimate obtained from simulations as in Section 3.8.3), however it is not significantly better than the 2-type approximation for larger λ. Generally the approximations are fairly reasonable, though the extinction probabilities can be about 0.1 smaller.

With the 2-type approximation, named individuals have the same active lifetime distribution, no matter how far removed they are from an unnamed ancestor, so this is the likely reason that it overestimates the spread of the true model, in which active lifetimes are reduced as you go down the naming tree. We also see that the multitype approximation overestimates the spread of the true model, so it seems the inter-sibling

dependencies have an effect of reducing spread. This may be because if an individual has a short active lifetime, then their siblings are more likely to, and, furthermore all their named offspring are then more likely to have much shorter lifetimes and so on.

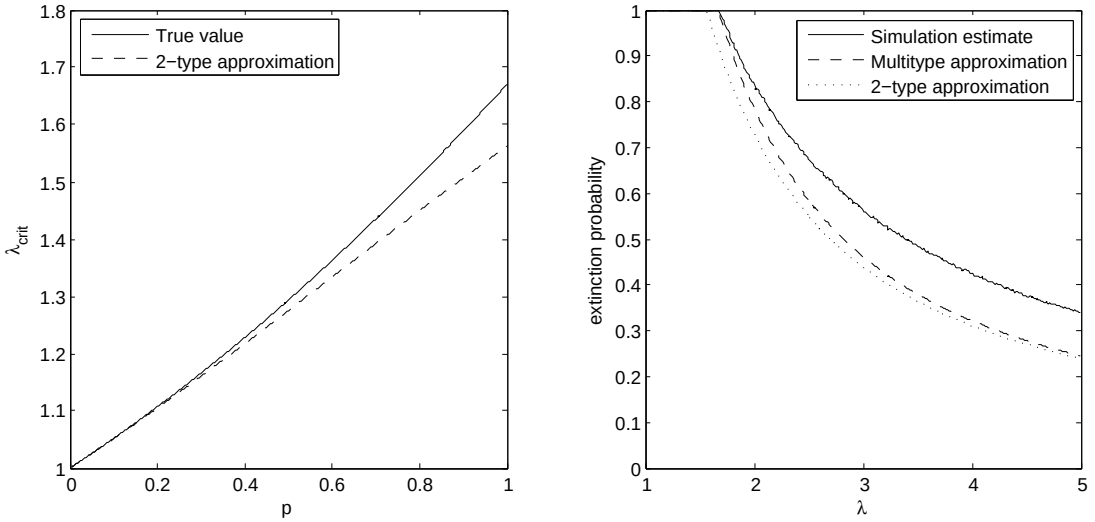

Figure 3.12: Comparing the 2-type and multitype approximations with the full model, with $T_I \sim \text{Exp}(\gamma)$. Extinction probabilities are evaluated for $p = 1$.

3.8.5 Independent or mutual delays

While we have assumed that sibling units experience independent delays, it has been seen that R_U was unchanged by assuming instead that sibling units experience the same delay. However, the probability of extinction would not be unchanged, so it would be of interest to see how much it differs between the two delay assumptions.

Figure 3.13 shows how the extinction probabilities compare for independent and mutual delays in the single-step case when $p = 1$ and $T_I \sim \text{Exp}(1)$. This is done from simulations of the offspring random variable of the GWP in a similar manner as for 3.11 in Section 3.8.3, with one slight difference: for longer delays, R is infinite with non-zero probability; to circumvent this we assume that if the number of offspring is

sufficiently large (here we consider this to mean at least 100), then it is infinite. The results are shown for $\lambda = 1.5$ and $T_L \equiv 0$, $\lambda = 2.5$ and $T_L \equiv 0$, and $\lambda = 1.5$ and $T_L \sim \text{Exp}(1)$.

We see that there is no dramatic difference between the two delay assumptions, even as we increase λ or introduce a latent period. In the latter case we can see at least that the extinction probability is slightly higher for mutual delays, which concurs with the findings of Section 3.8.4, that dependencies between sibling units increase the extinction probability.

Similar results were obtained when different assumptions were tested, such as constant infectious periods, constant latent periods or iterative tracing.

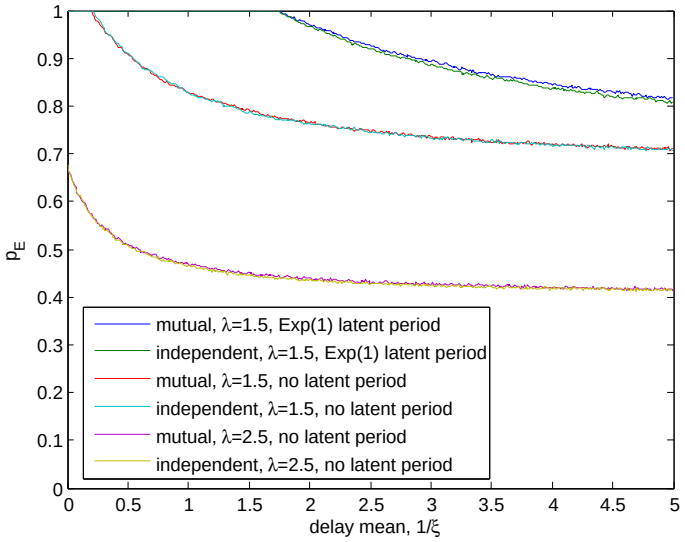

Figure 3.13: p_E varying with delay mean ($\frac{1}{\xi}$) for both mutual and independent delays in single-step case when $T_I \sim \text{Exp}(1)$, $T_D \sim \text{Exp}(\xi)$ and $p = 1$.

3.9 Concluding comments

In this chapter, a threshold parameter, R_U, has been defined for the contact tracing model, such that a major epidemic can occur if and only if $R_U \geq 1$. Expressions for this parameter were obtained for varying assumptions about the tracing and epidemic models, and then analysed numerically to gain insight in to their effects. It is clear that choice of infectious period, latent period and tracing delay distributions has an effect, increasingly so as it becomes more likely that individuals are traced. This also increases the material difference between the single-step tracing (traced individuals are not allowed to name their contacts) and iterative tracing (traced individuals are allowed to name their contacts). In some cases, explicit expressions for determining the probability the epidemic becomes extinct are obtained, and while generally it is seen that we can use simulations to obtain an estimate, it is felt that explicit analysis is better as it gives us more accurate values in less computation time. Analysis required the approximating assumption that the at-risk population is large, and it is seen that this approximation quickly becomes adequate as true population size increases.

We saw that whether an individual's traced contacts experience the same or independent (and identically distributed) delays does not have any impact on R_U, nor does it have a huge impact on the extinction probability. Generally then it would seem that it is preferable to assume the delays are independent to reduce the dependencies of sibling units, without fear that making this assumption to increase tractability will dramatically alter results.

Here we have only considered forward tracing, that is, that infectees may be traced by being named by their infector, but not vice versa. Backward tracing assumes the tracing goes in the other direction, while full tracing allows for an individual to name

anyone they have been in contact with. These are generally more difficult to analyse than forward tracing, but likely more effective at controlling an epidemic (certainly in the full tracing case). Hence it would be of interest to see what progress, if any, can be made considering either of these. We have not assumed any population structure in this chapter beyond homogeneous mixing. We consider a model in Chapter 4 that incorporates both the household-structured population of Chapter 2 and the contact tracing of this chapter. We have also assumed that naming probability is fixed for all individuals, it may be of interest to consider differing models for the naming probability for a given individual, wherein it may be random, and it may depend on the individual's infectious period length and number of contacts.

CHAPTER 4

Household contact tracing with local tracing

4.1 Introduction

In Chapter 2 an intervention strategy was modelled for an epidemic spreading amongst a population partitioned into households, in which, upon diagnosis of an infected individual, intervention is directed towards that individual's household, by vaccinating their housemates or isolating them. It was seen that, although this intervention can reduce spread of the disease, it can not always guarantee to prevent a major outbreak, particularly if a diagnosed individual is expected to have made at least one infectious contact outside their household. Thus it is of interest to consider further intervention to see if it can make major outbreaks an impossibility.

So, in this chapter we consider an intervention model which not only has the local tracing element of Chapter 2, in that, upon diagnosis of an infected individual their housemates are vaccinated or isolated, but also a contact tracing element, as in Chapter 3. We assume that diagnosed individuals can name the infectious contacts they have made

outside their household, and then a named contact and their household are vaccinated after some tracing delay. The effect of this contact tracing is that an infected household may be vaccinated before anyone has been diagnosed. Further, it can prevent the disease spreading within the infected household, or better still prevent anyone in an infected household even becoming infectious. This is possible because we assume that infected individuals experience two latent periods - during the first a vaccination will prevent them from becoming infectious, while during the second it will not (this differs from Chapter 2, in which it is assumed there is only one latent period, and whether or not the vaccine had an effect on latent individuals was specified).

Kaplan et al. [30] suggested that a contact-traced vaccination scheme is not particularly effective in a homogeneously-mixing population, under the two latent period assumption we make here. Their reasoning was that there is a 'race to trace' - the infected individual must be traced before they become vaccine-insensitive. In this chapter, we are assuming that when an individual is traced, not only they, but also their entire household, is vaccinated, and so the effect of tracing an individual is increased, particularly because the other household members are more likely to be vaccine-sensitive or even, more specifically, susceptible.

Becker et al. [18] and Pike [40] also modelled contact tracing for epidemics spreading amongst a community of households. Becker et al. [18] assumed that on diagnosis an individual's housemates are isolated and a fraction of infections outside the household are traced and isolated. The work in this chapter differs from theirs on four main counts: (i) they made a simplifying assumption by assuming that infectious contacts between households occur at the beginning of the infector's infectious period, while here results are more exact; (ii) here a household may be vaccinated instead of isolated,

vaccination being possibly a less intrusive form of intervention; (iii) in their contact tracing model, only the traced individual is treated, while here it is assumed that the traced individual and all their housemates are treated, increasing the effect of the tracing; and (iv) we incorporate a tracing delay. Pike [40] assumed that on diagnosis an individual's housemates are vaccinated and isolated and a fraction of infections outside the household are traced and the traced individual and their household is vaccinated. The work in this chapter differs from his on two main counts: (i) he makes a simplifying assumption to remove the interdependencies of sibling units, here by assuming infectious periods are of fixed length the results are more exact; and (ii) in our model the assumption of households being isolated is relaxed to assume there is a probability of isolation of a household at the first removal within that household, which may be considered a less intrusive intervention policy, but does allow infectious contacts emanating from a household which has at least one diagnosed individual.

This chapter is structured as follows. In Section 4.2, the epidemic, vaccine and contact tracing models are defined. The two-type branching process of named and unnamed households and single household epidemic are introduced, and how these are used to determine the threshold behaviour is described. Possible outcomes of the single household epidemic are listed, and their contributions to the threshold parameter and probability of a global epidemic are described. Expressions for these contributions are derived for constant latent periods in Section 4.3 and for exponentially-distributed latent periods in Section 4.4, with constant and exponentially-distributed delays considered in both sections. Section 4.5 contains some numerical illustrations of the theory, and Section 4.6 provides some concluding comments.

4.2 Background and general theory

4.2.1 Model

Consider the following modified SEIR (susceptible → exposed → infective → removed) epidemic model among a closed population of size N. At any time, each individual in the population is in one of five states: susceptible, vaccine-sensitive latent, vaccine-insensitive latent, infective or removed. Initially a small number of individuals are infectives and the rest are susceptible. A susceptible individual becomes a vaccine-sensitive latent individual if he/she makes contact with an infective in a manner described below. A vaccine-sensitive latent individual remains vaccine-sensitive for a period of time distributed according to a random variable $T_{L,1}$, having an arbitrary but specified distribution, at the end of which he/she becomes vaccine-insensitive latent. A vaccine-insensitive latent individual remains latent for a period of time distributed according to a random variable $T_{L,2}$, having an arbitrary but specified distribution, at the end of which he/she becomes infective. An infective individual remains infectious for a period of time distributed according to a random variable T_I, having an arbitrary but specified distribution with finite moment-generating function, and then becomes removed. Once removed, an individual no longer plays a part in the epidemic process. The epidemic ends when there are no more latent or infective individuals left in the population.

The population of N individuals is partitioned into m households of size n. During his/her infectious period, a given infective makes *global* contacts with any given susceptible in the population at times given by the points of a homogeneous Poisson process with rate λ_G/N and, additionally, *local* contacts with any given susceptible in its

household at times given by the points of a homogeneous Poisson process with rate λ_L. All of the Poisson processes, and the random variables describing latent and infectious periods, are assumed to be mutually independent. Note that, for ease of exposition, households are assumed to be of the *same* size, but the theory may be easily extended to consider households of unequal size.

There are local and global contact tracing policies incorporated in the model. The local tracing works at the within-household level. Upon the first removal within a household, all remaining members of the household are vaccinated, unless they have already been vaccinated as a result of the global tracing described below. We assume that the vaccine is perfect, that is that if an individual is in either the susceptible or vaccine-sensitive latent state when they receive the vaccine, then they are rendered entirely immune (i.e. they will never become infectious), otherwise the vaccine has no effect. Additionally, at the first removal (and only at the first removal) in a household, the household is isolated (or quarantined) with probability p_I. Once a household has been isolated, members of that household can no longer make global contacts. While a household is not isolated we call it *active*, and *inactive* while it is isolated.

The global tracing works at the between-household level. Additionally, when an infectious individual becomes removed in an active household, they name each of their global contacts, independently, with probability p_c, and after a (independent) delay, distributed according to a random variable T_D (with an arbitrary but specified distribution), a given named contact and their household are vaccinated, unless they have already been vaccinated as a result of the local tracing described above, with the vaccine action being the same as that of the local tracing. Note that if a household is never isolated, then all infectious individuals will be able to name global contacts when they

are removed, while if the household *is* isolated, only the first removed individual will be able to. Further, note that each infected household is vaccinated once, and only once.

4.2.2 Threshold behaviour

If the number of households m is large and the number of initial infectives is small, then during the early stages of an epidemic, there is only a small probability that a global contact is made with an individual from a household containing at least one non-susceptible individual. Thus, we can approximate the initial stages of the epidemic by a process in which all global contacts are made with individuals residing in completely susceptible households.

A problem with making analytical progress in contact tracing models, is that sibling units (i.e. households who are infected by the same individual) are not necessarily independent, and thus the process of infected households in the approximation does not necessarily follow a branching process. Consider an infectious individual who is asked to name global contacts. The time until vaccination from infection for these contacts depends on (a) their delays (which are independent of one another) and (b) the time that they are named in relation to when they were infected. The latter depends upon the length of their infector's infectious period, and thus these sibling units are co-dependent.

From now on we will assume that the infectious periods all have fixed value ι (i.e. $T_I \equiv \iota$). In this case, if an infector (who can name global contacts) has k global contacts, then since global contacts occur uniformly and at random over the length of the infectious period, the time periods from infectious contact to the infector's removal (and hence the naming of contacts) can be sampled as k independent and identically distributed

$\mathrm{U}(0, \iota)$ random variables. Since the infectious period length is fixed, their is no co-dependence for the sibling units, and hence the process of infected households in the approximation does follow a branching process.

Consider a single household epidemic: a completely susceptible, typical household into which a global contact introduces infection, and suppose that no further global contact into the household occurs subsequently (thus initially there are $n-1$ susceptibles and 1 vaccine-sensitive latent). We call the number of global contacts emanating from this single household epidemic R. Then $R_* = \mathrm{E}[R]$ is a threshold parameter, since if $R_* \leq 1$ then a global epidemic cannot occur (a *global epidemic* occurs if, in the limit $m \to \infty$, the epidemic infects infinitely many households in the branching process approximation). When $R_* > 1$, the probability of a global epidemic, p_G say, is non-zero.

4.2.3 Two-type process and active severity

There are two types of households in this branching process: unnamed and named, which we will label type-0 and 1, respectively. For $i, j = 0, 1$, let R_{ij} be the number of type-j contacts emanating from a typical type-i household, and let $M_{ij} = \mathrm{E}\left[R_{ij}\right]$. Then, from standard branching process theory (e.g. Chapters 2 and 5 of Haccou et al. [23]), R_* is given by the largest eigenvalue of the matrix of mean offspring $[M_{ij}]$, i.e.

$$R_* = \frac{M_{00} + M_{11} + \sqrt{\left(M_{00} - M_{11}\right)^2 + 4M_{01}M_{10}}}{2},$$

and recall that when $R_* > 1$, there is a positive probability of a global epidemic. So, when $R_* > 1$, let p_{Gi} $(i = 0, 1)$ be the probability of a global epidemic given that the epidemic is started by one infective in a type-i household, then $(p_{G0}, p_{G1}) = (1 - \tau_0, 1 - \tau_1)$

where (τ_0, τ_1) is the solution for (s_0, s_1) in $(0,1) \times (0,1)$ of the system of equations

$$s_i = f_i(s_0, s_1), \qquad (i = 0, 1)$$

where, for $i = 0, 1$, $f_i(s_0, s_1) = \mathrm{E}\left[s_0^{R_{i0}} s_1^{R_{i1}}\right]$.

Consider an infective in the single household epidemic. They will experience part (or all) of their infectious period while their household is active, which we refer to as their active infectious period. We call the sum of all active infectious periods in a household, the active severity of that household. We label the active severity of a type-i ($i = 0, 1$) household as X_i. We can write this in the form, for $i = 0, 1$

$$X_i = X_i^+ + X_i^-,$$

where X_i^+ and X_i^- are the sums of the active infectious periods of individuals who can name contacts and who cannot name contacts, respectively (recall that the latter can only occur in isolated households). Since global contacts are made by an infective at rate λ_G during his/her active infectious period, the numbers of unnamed and named global contacts emanating from a type-i ($i = 0, 1$) household (i.e. R_{i0} and R_{i1}, respectively) have Poisson distributions with random means $\lambda_G\left((1-p_c)\, X_i^+ + X_i^-\right)$ and $\lambda_G p_c X_i^+$, respectively. Therefore, for $i = 0, 1$,

$$M_{i0} = \lambda_G\left((1-p_c)\,\mathrm{E}\left[X_i^+\right] + \mathrm{E}\left[X_i^-\right]\right), \tag{4.2.1}$$

$$M_{i1} = \lambda_G p_c \mathrm{E}\left[X_i^+\right], \tag{4.2.2}$$

while, for $i = 0, 1$,

$$f_i\left(s_0, s_1\right) = \psi_i\left(\lambda_G\left\{(1-p_c)\left(1-s_0\right) + p_c\left(1-s_1\right)\right\}, \lambda_G\left(1-s_0\right)\right), \tag{4.2.3}$$

where, for $i = 0, 1$ and $\theta_1, \theta_2 \geq 0$, $\psi_i\left(\theta_1, \theta_2\right) = \mathrm{E}\left[e^{-\left(\theta_1 X_i^+ + \theta_2 X_i^-\right)}\right]$.

4.2.4 Subcases of the single household epidemic

Consider a global contact between an infector, A say, and an infectee, B say. Let the remaining infectious period of A when B is infected be U. Then $U \sim \mathrm{U}(0, \iota)$. Let the delay until vaccination for B's household be W, so if B is named, B's household is vaccinated at time $U + W$ after B is infected. Now, let B have vaccine-sensitive and -insensitive latent periods $T_{B,1}$ and $T_{B,2}$ respectively.

We will set time-zero for B's household to be the moment at which B begins his/her infectious period, i.e. B is at time $-(T_{B,1} + T_{B,2})$, becomes vaccine-insensitive at time $-T_{B,2}$ and is removed at time ι. We let $S(t)$, $L_1(t)$, $L_2(t)$, $I(t)$ and $R(t)$ be the number of susceptible, vaccine-sensitive latent, vaccine-insensitive latent, infective and removed individuals, respectively, in B's household at time t. Further, we let $Y(t) = \max\{0, L_2(t) + I(t) + R(t) - 1\}$ be the number of B's housemates (excluding B themselves) who are vaccine-insensitive at time t.

Let V be the time of the vaccination if B were named (note that if B is not named the vaccination time is ι), then

$$V = \begin{cases} U + W - T_{B,1} - T_{B,2} & \text{if } U + W - T_{B,1} - T_{B,2} < \iota \\ \iota & \text{otherwise} \end{cases}$$

We now break this down into 6 possible cases, represented in Table 4.1.

Note that in the first two cases it does not matter whether or not the household is isolated as there will be no infectives after B's removal.

For $j = 1, 2, \ldots, 6$, let q_j be the probability of C_j given that B is named. From the

Table 4.1: Subcases of the single household epidemic

Event	Vaccination time if B named	Household isolated
C_1	$-T_{B,1} - T_{B,2} \leq V < -T_{B,2}$	yes or no
C_2	$-T_{B,2} \leq V < 0$	yes or no
C_3	$0 \leq V < \iota$	no
C_4	$0 \leq V < \iota$	yes
C_5	$V = \iota$	no
C_6	$V = \iota$	yes

information above,

$$q_1 = \mathrm{P}\left(U + T_D < T_{L,1}\right),$$

$$q_2 = \mathrm{P}\left(T_{L,1} \leq U + T_D < T_{L,1} + T_{L,2}\right),$$

$$q_3 = (1 - p_I)\,\mathrm{P}\left(T_{L,1} + T_{L,2} \leq U + T_D < T_{L,1} + T_{L,2} + \iota\right),$$

$$q_4 = p_I \mathrm{P}\left(T_{L,1} + T_{L,2} \leq U + T_D < T_{L,1} + T_{L,2} + \iota\right),$$

$$q_5 = (1 - p_I)\,\mathrm{P}\left(U + T_D \geq T_{L,1} + T_{L,2} + \iota\right),$$

$$q_6 = p_I \mathrm{P}\left(U + T_D \geq T_{L,1} + T_{L,2} + \iota\right).$$

Define, for $j = 1, 2, \ldots, 6$ and $\theta_1, \theta_2 \geq 0$,

$$G_j^+ = \mathrm{E}\left[X_1^+ \middle| C_j\right],$$

$$G_j^- = \mathrm{E}\left[X_1^- \middle| C_j\right],$$

$$H_j\left(\theta_1, \theta_2\right) = \mathrm{E}\left[e^{-\left(\theta_1 X_1^+ + \theta_2 X_1^-\right)} \middle| C_j\right].$$

We have (noting that an unnamed household is effectively the same as a named house-

hold with $V = \iota$)

$$\begin{aligned}
\mathrm{E}\left[X_1^+\right] &= \sum_{j=1}^{6} q_j G_j^+, \\
\mathrm{E}\left[X_1^-\right] &= \sum_{j=1}^{6} q_j G_j^-, \\
\mathrm{E}\left[X_0^+\right] &= (1-p_I)\, G_5^+ + p_I G_6^+, \\
\mathrm{E}\left[X_0^-\right] &= (1-p_I)\, G_5^- + p_I G_6^-, \\
\psi_1(\theta_1,\theta_2) &= \sum_{j=1}^{6} q_j H_j(\theta_1,\theta_2), \\
\psi_0(\theta_1,\theta_2) &= (1-p_I)\, H_5(\theta_1,\theta_2) + p_I H_6(\theta_1,\theta_2).
\end{aligned}$$

Now, we consider the outcomes in each event. Note that in all cases except C_1, B will experience a full active infectious period (i.e. of length ι).

4.2.5 Outcomes in the subcases of the single household epidemic

In event C_1, the household is vaccinated before B becomes vaccine-insensitive latent and hence no-one in B's household ever becomes infectious, and so

$$G_1^+ = G_1^- = 0,$$

while, for $\theta_1, \theta_2 \geq 0$,

$$H_1(\theta_1,\theta_2) = 1.$$

In event C_2, the household is vaccinated while B is vaccine-insensitive latent, and hence only B will become infectious (and experience their full lifetime), and so

$$G_2^+ = \iota,$$

$$G_2^- = 0,$$

while, for $\theta_1, \theta_2 \geq 0$,

$$H_2(\theta_1, \theta_2) = e^{-\theta_1 \iota}.$$

In event C_3, the household is not isolated and the household is vaccinated while B is infectious. In this case, if the number of vaccine-insensitive individuals when the household is vaccinated is k ($k = 1, 2, \ldots, n$), then $X_1^+ = k\iota$ and $X_1^- = 0$. This will depend on the vaccination time in this case, i.e. V restricted to $[0, \iota)$. If $U + T_D$ and $T_{L,1} + T_{L,2}$ have probability measures μ_{U+T_D} and $\mu_{T_{L,1}+T_{L,2}}$, then V restricted to $[0, \iota)$ has probability measure, for $t \in [0, \iota)$

$$\frac{\mu(t)}{\mathrm{P}(0 \leq V < \iota)} = (1 - p_I) \frac{\mu(t)}{q_3},$$

noting that $q_3 = (1 - p_I)\,\mathrm{P}(0 \leq V < \iota)$, and where, for $t \in (0, \iota)$,

$$\mu(t) = \int_0^\infty \mu_{U+T_D}(t + \tau)\, d\mu_{T_{L,1}+T_{L,2}}(\tau). \tag{4.2.4}$$

So, if we let

$$A_3 = \int_0^\iota \mathrm{E}\left[Y(t) | V = t\right]\, d\mu(t),$$
$$B_3(\theta) = \int_0^\iota \mathrm{E}\left[e^{-\theta\iota Y(t)} \middle| V = t\right]\, d\mu(t)$$

then

$$G_3^+ = \iota + \frac{1 - p_I}{q_3} A_3 \iota,$$
$$G_3^- = 0,$$

while, for $\theta_1, \theta_2 \geq 0$,

$$H_3(\theta_1, \theta_2) = \frac{1 - p_I}{q_3} e^{-\theta_1 \iota} B_3(\theta_1).$$

In event C_4 the household is isolated and the household is vaccinated while B is infectious. In the isolation cases (C_4 and C_6) only B is allowed to name contacts, and so $X_1^+ = \iota$, while X_1^- is the rest of the household's active severity. Let $Z(t)$ be the total severity of the household excluding B themselves from time-zero up until time t, that is, for $t > 0$

$$Z(t) = \int_0^t (I(\tau) - 1)\ d\tau.$$

In this case V restricted to $[0, \iota)$ has probability measure

$$\frac{\mu(t)}{\mathrm{P}\left(0 \leq V < \iota\right)} = p_I \frac{\mu(t)}{q_4},$$

noting that $q_4 = p_I \mathrm{P}\left(0 \leq V < \iota\right)$, and where $\mu(t)$ is the same as in Eqn. (4.2.4). Let

$$A_4 = \int_0^\iota \mathrm{E}\left[Z(\iota)|V = t\right]\ d\mu(t),$$
$$B_4(\theta) = \int_0^\iota \mathrm{E}\left[e^{-\theta Z(\iota)}\middle| V = t\right]\ d\mu(t)$$

then

$$G_4^+ = \iota,$$
$$G_4^- = \frac{p_I}{q_4} A_4,$$

while, for $\theta_1, \theta_2 \geq 0$,

$$H_4\left(\theta_1, \theta_2\right) = \frac{p_I}{q_4} e^{-\iota\theta_1} B_4\left(\theta_2\right).$$

In event C_5 the household is not isolated and the household is vaccinated when B has been removed. In this case, if there are k vaccine-insensitive individuals immediately when B is removed (including B), then $X_1^+ = k\iota$ and $X_1^- = 0$, and so if we let

$$A_5 = \mathrm{E}\left[Y(\iota)|V = \iota\right],$$
$$B_5(\theta) = \mathrm{E}\left[e^{-\theta\iota Y(\iota)}\middle| V = \iota\right] \qquad (\theta > 0),$$

then

$$G_5^+ = (A_5 + 1)\,\iota,$$

$$G_5^- = 0,$$

while, for $\theta_1, \theta_2 \geq 0$,

$$H_5\left(\theta_1, \theta_2\right) = e^{-\theta_1 \iota} B_5\left(\theta_1\right).$$

Finally, in event C_6, the household is isolated and is vaccinated at the first removal (though the vaccination actually has no effect in this case). If we let

$$A_6 = \mathrm{E}\left[Z(\iota) | V = \iota\right],$$

$$B_6(\theta) = \mathrm{E}\left[e^{-\theta Z(\iota)} \middle| V = \iota\right],$$

then

$$G_6^+ = \iota,$$

$$G_6^- = A_6,$$

while, for $\theta_1, \theta_2 \geq 0$,

$$H_6\left(\theta_1, \theta_2\right) = e^{-\iota\theta_1} B_6\left(\theta_2\right).$$

Putting this all together

$$\mathrm{E}\left[X_0^+\right] = \iota + (1 - p_I)\, A_5 \iota,$$

$$\mathrm{E}\left[X_0^-\right] = p_I A_6,$$

$$\mathrm{E}\left[X_1^+\right] = \iota\left[1 - q_1 + (1 - p_I)\, A_3 + q_5 A_5\right],$$

$$\mathrm{E}\left[X_1^-\right] = p_I A_4 + q_6 A_6,$$

while for $\theta_1, \theta_2 \geq 0$,

$$\psi_0(\theta_1, \theta_2) = e^{-\theta_1 \iota} \left[(1 - p_I) B_5(\theta_1) + p_I B_6(\theta_2) \right],$$

$$\psi_1(\theta_1, \theta_2) = q_1 + e^{-\theta_1 \iota} \left[q_2 + (1 - p_I) B_3(\theta_1) + q_5 B_5(\theta_1) + p_I B_4(\theta_2) + q_6 B_6(\theta_2) \right].$$

Note that we do not explicitly need to determine q_3 or q_4. What remains then is to determine q_i for $i = 1, 2, 5, 6$, and A_i and $B_i(\theta)$ for $i = 3, 4, 5, 6$. We obtain these for different latent period and delay distributions in the sequel, but first we derive distributions for $U + T_D$ for different delay period distributions.

4.2.6 Different delay period distributions

Suppose that the $T_D \equiv \eta$, i.e. constant with value η, then $U + T_D$ has probability density function

$$f_{U+T_D}(t) = \begin{cases} \frac{1}{\iota} & \text{if } \eta \leq t \leq \eta + \iota \\ 0 & \text{otherwise} \end{cases}$$

and distribution function

$$F_{U+T_D}(t) = \begin{cases} 0 & \text{if } t < \eta \\ \frac{t-\eta}{\iota} & \text{if } \eta \leq t \leq \eta + \iota \\ 1 & \text{if } t > \eta + \iota \end{cases}$$

Suppose instead that $T_D \sim \text{Exp}(\xi)$, i.e. exponentially distributed with mean $\frac{1}{\xi}$, then $U + T_D$ has probability density function

$$\begin{aligned} f_{U+T_D}(t) &= \begin{cases} \int_0^t \frac{1}{\iota} \xi e^{-\xi(t-u)} du & \text{if } 0 \leq t \leq \iota \\ \int_0^\iota \frac{1}{\iota} \xi e^{-\xi(t-u)} du & \text{if } t > \iota \end{cases} \\ &= \begin{cases} \frac{1}{\iota} \left(1 - e^{-\xi t}\right) & \text{if } 0 \leq t \leq \iota \\ \frac{1}{\iota} e^{-\xi t} \left(e^{\xi \iota} - 1\right) & \text{if } t > \iota \end{cases} \end{aligned}$$

and distribution function

$$F_{U+T_D}(t) = \begin{cases} \frac{1}{\iota}\left(t - \frac{1}{\xi}\left(1 - e^{-\xi t}\right)\right) & \text{if } 0 \leq t \leq \iota \\ 1 - \frac{1}{\xi\iota}e^{-\xi t}\left(e^{\xi\iota} - 1\right) & \text{if } t > \iota \end{cases}.$$

4.2.7 Notation

The most important parameters appearing throughout this chapter are listed in Table 4.2, along with brief definitions.

4.3 Constant latent periods

Let $T_{L_1} \equiv \nu_1$ and $T_{L_2} \equiv \nu_2$, where $\nu_1 + \nu_2 \geq \iota$ (it is difficult to make analytical progress when $\nu_1 + \nu_2 < \iota$ as this means that it is possible that multiple individuals may be infectious before the first removal, and therefore the single household epidemic is non-Markovian before the first removal, which is fairly problematic given that we are concerned with the state of the single household epidemic up until the first removal).

Consider the isolation cases C_4 and C_6. Besides the original infected individual, B, no other members of B's household will be infectious before B's removal (and the household's isolation). Thus,

$$A_4 = A_6 = 0,$$

while for $\theta \geq 0$,

$$B_4(\theta) = \int_0^\iota d\mu(t), B_6(\theta) = 1.$$

For the non-isolation cases C_3 and C_5, we need to obtain the distribution of $Y(t)$, the number of B's housemates who are vaccine-insensitive at time t. Housemates of B

Table 4.2: List of important parameters for Chapter 4.

parameter	description
N	number of individuals in the population
n	number of individuals in each household
λ_L	local (i.e. within-household) individual-to-individual contact rate
λ_G/N	global (i.e. between-household) individual-to-individual contact rate
p_I	probability that a household is isolated at the first removal
p_c	probability that a global contact is named
ι	length of infectious period (which is constant)
μ_1	rate parameter for exponentially distributed vaccine-sensitive latent period (i.e. mean=$\frac{1}{\mu_1}$)
μ_2	rate parameter for exponentially distributed vaccine-insensitive latent period (i.e. mean=$\frac{1}{\mu_2}$)
ν_1	length of constant vaccine-sensitive latent period
ν_2	length of constant vaccine-insensitive latent period
η	length of constant tracing delay
ξ	rate parameter for exponentially distributed tracing delay (i.e. mean=$\frac{1}{\xi}$)
R_*	expected number of global contacts emanating from a typical single household epidemic
p_G	probability of a global epidemic
M_{ij}	expected number of type-j global contacts emanating from a typical type-i household (type-0=unnamed, type-1=named)
X_i	active severity of a type-i household
X_i^+, X_i^-	sum of active infectious periods in type-i household of individuals who can name contacts ($^+$)/can not name contacts ($^-$)

will be vaccine-insensitive at time t if they have been infected and ended their vaccine-sensitive latent period before t, i.e. they have been infected before time $t-\nu_1$, provided $\nu_1 \leq t$ (if $\nu_1 > t$, then none of B's housemates will be vaccine-insensitive at time t). Each housemate of B is infected before time $t-\nu_1$ ($\nu_1 \leq t$) with probability $1-e^{-\lambda_L(t-\nu_1)}$, independently of one another. Thus, for $t \geq \nu_1$, $Y(t)|V=t \sim \text{Bin}\left(n-1, 1-e^{-\lambda_L(t-\nu_1)}\right)$, and so

$$\mathrm{E}[Y(t)|V=t] = \begin{cases} 0 & \text{if } t < \nu_1 \\ (n-1)\left(1-e^{-\lambda_L(t-\nu_1)}\right) & \text{if } t \geq \nu_1, \end{cases}$$

and thus

$$A_3 = \begin{cases} 0 & \text{if } \iota < \nu_1 \\ (n-1)\int_{\nu_1}^{\iota}\left(1-e^{-\lambda_L(t-\nu_1)}\right)\, d\mu(t) & \text{if } \iota \geq \nu_1, \end{cases}$$

while

$$A_5 = \mathrm{E}[Y(\iota)|V=\iota] = \begin{cases} 0 & \text{if } \iota < \nu_1 \\ (n-1)\left(1-e^{-\lambda_L(\iota-\nu_1)}\right) & \text{if } \iota \geq \nu_1. \end{cases}$$

Further, for $\theta \geq 0$,

$$\mathrm{E}\left[e^{-\theta\iota Y(t)}\middle| V=t\right] = \begin{cases} 1 & \text{if } t < \nu_1 \\ \left(e^{-\lambda_L(t-\nu_1)} + \left(1-e^{-\lambda_L(t-\nu_1)}\right)e^{-\iota\theta}\right)^{n-1} & \text{if } t \geq \nu_1, \end{cases}$$

and thus

$$B_3(\theta) = \begin{cases} \int_0^{\iota} d\mu(t) & \text{if } \iota < \nu_1 \\ \int_0^{\nu_1} d\mu(t) + \int_{\nu_1}^{\iota}\left(e^{-\lambda_L(t-\nu_1)} + \left(1-e^{-\lambda_L(t-\nu_1)}\right)e^{-\iota\theta}\right)^{n-1}\, d\mu(t) & \text{if } \iota \geq \nu_1, \end{cases}$$

while

$$B_5(\theta) = \begin{cases} 1 & \text{if } \iota < \nu_1 \\ \left(e^{-\lambda_L(\iota-\nu_1)} + \left(1-e^{-\lambda_L(\iota-\nu_1)}\right)e^{-\iota\theta}\right)^{n-1} & \text{if } \iota \geq \nu_1. \end{cases}$$

In the remainder of this section we determine q_i ($i = 1, 2, 5, 6$), A_3 and $B_3(\theta)$ for different delay distributions.

Notation

For $\theta \geq 0$, $\gamma \geq 0$ and $a \leq b$, let

$$\begin{aligned}\chi(\theta,\gamma,a,b) &= \int_a^b e^{-\gamma t}\left(e^{-\lambda_L(t-\nu_1)} + \left(1-e^{-\lambda_L(t-\nu_1)}\right)e^{-\iota\theta}\right)^{n-1} dt \\ &= e^{-\gamma\nu_1}\sum_{k=0}^{n-1}\binom{n-1}{k}e^{-k\iota\theta}\sum_{j=0}^{k}\binom{k}{j}(-1)^j \\ &\qquad \times \frac{e^{-(\gamma+\lambda_L(n+j-k-1))(a-\nu_1)} - e^{-(\gamma+\lambda_L(n+j-k-1))(b-\nu_1)}}{\gamma+\lambda_L(n+j-k-1)}.\end{aligned}$$

4.3.1 Exponential delay

Suppose that $T_D \sim \text{Exp}(\xi)$, i.e. exponentially distributed with mean $\frac{1}{\xi}$.

The probability of event C_1 given B's is named is

$$\begin{aligned}q_1 &= \text{P}\left(U + T_D < T_{L,1}\right) \\ &= F_{U+T_D}(\nu_1) \\ &= \begin{cases} \frac{1}{\iota}\left(\nu_1 - \frac{1}{\xi}\left(1-e^{-\xi\nu_1}\right)\right) & \text{if } 0 \leq \nu_1 \leq \iota \\ 1 - \frac{1}{\xi\iota}e^{-\xi\nu_1}\left(e^{\xi\iota}-1\right) & \text{if } \nu_1 > \iota, \end{cases}\end{aligned}$$

while the probability of event C_2 given B is named is

$$\begin{aligned}q_2 &= \text{P}\left(T_{L,1} \leq U + T_D < T_{L,1} + T_{L,2}\right) \\ &= F_{U+T_D}(\nu_1+\nu_2) - F_{U+T_D}(\nu_1) \\ &= \begin{cases} 1 - \frac{1}{\xi\iota}e^{-\xi(\nu_1+\nu_2)}\left(e^{\xi\iota}-1\right) - \frac{1}{\iota}\left(\nu_1 - \frac{1}{\xi}\left(1-e^{-\xi\nu_1}\right)\right) & \text{if } 0 \leq \nu_1 \leq \iota \\ \frac{1}{\xi\iota}e^{-\xi\nu_1}\left(1-e^{-\xi\nu_2}\right)\left(e^{\xi\iota}-1\right) & \text{if } \nu_1 > \iota. \end{cases}\end{aligned}$$

To obtain A_3 and $B_3(\theta)$ we need to find the measure of V restricted to $(0, \iota)$, which is, for $t \in (0, \iota)$

$$\begin{aligned}\mu(t) &= f_{U+T_D}\left(t + \nu_1 + \nu_2\right) \\ &= \frac{1}{\iota} e^{-\xi(t+\nu_1+\nu_2)} \left(e^{\xi\iota} - 1\right),\end{aligned}$$

thus, for $\iota \geq \nu_1$,

$$\begin{aligned}A_3 = \frac{(n-1)}{\iota} e^{-\xi(\nu_1+\nu_2)} &\left(e^{\xi\iota} - 1\right) \\ &\times \left(\frac{1}{\xi}\left(e^{-\xi\nu_1} - e^{-\xi\iota}\right) - \frac{1}{\xi + \lambda_L}\left(e^{-\xi\nu_1} - e^{-(\xi+\lambda_L)\iota + \lambda_L\nu_1}\right)\right)\end{aligned}$$

while, for $\theta \geq 0$,

$$B_3(\theta) = \begin{cases} \frac{1}{\xi\iota} e^{-\xi(\nu_1+\nu_2-\iota)} \left(1 - e^{-\xi\iota}\right)^2 & \text{if } \iota < \nu_1 \\ \frac{1}{\iota} e^{-\xi(\nu_1+\nu_2)} \left(e^{\xi\iota} - 1\right)\left(1 - e^{-\xi\nu_1}\right) \chi\left(\theta, \xi, \nu_1, \iota\right) & \text{if } \iota \geq \nu_1 \end{cases}$$

and

$$B_4(\theta) = \frac{1}{\xi\iota} e^{-\xi(\nu_1+\nu_2-\iota)} \left(1 - e^{-\xi\iota}\right)^2.$$

The probability of event C_5 given that B is named is

$$\begin{aligned}q_5 &= (1 - p_I)\, \mathrm{P}\left(U + T_D \geq T_{L,1} + T_{L,2} + \iota\right) \\ &= (1 - p_I)\left(1 - F_{U+T_D}\left(\nu_1 + \nu_2 + \iota\right)\right) \\ &= \frac{1 - p_I}{\xi\iota} e^{-\xi(\nu_1+\nu_2)} \left(1 - e^{-\xi\iota}\right)\end{aligned}$$

while the probability of event C_6 given that B is named is

$$q_6 = \frac{p_I}{\xi\iota} e^{-\xi(\nu_1+\nu_2)} \left(1 - e^{-\xi\iota}\right).$$

4.3.2 Constant delay

Suppose that $T_D \equiv \eta$, i.e. constant with value η.

The probability of event C_1 given that B is named is

$$\begin{aligned} q_1 &= \mathrm{P}\left(U + T_D < T_{L,1}\right) \\ &= F_{U+T_D}\left(\nu_1\right) \\ &= \begin{cases} 0 & \text{if } \nu_1 < \eta \\ \frac{\nu_1 - \eta}{\iota} & \text{if } \eta \leq \nu_1 \leq \eta + \iota \\ 1 & \text{if } \nu_1 > \eta + \iota, \end{cases} \end{aligned}$$

while the probability of event C_5 given that B is named is

$$\begin{aligned} q_2 &= \mathrm{P}\left(T_{L,1} \leq U + T_D < T_{L,1} + T_{L,2}\right) \\ &= F_{U+T_D}\left(\nu_1 + \nu_2\right) - F_{U+T_D}\left(\nu_1\right) \\ &= \begin{cases} 0 & \text{if } \nu_1 + \nu_2 < \eta \\ 0 & \text{if } \nu_1 > \eta + \iota \\ 1 & \text{if } \nu_1 < \eta, \nu_1 + \nu_2 > \eta + \iota \\ \frac{\nu_1 + \nu_2 - \eta}{\iota} & \text{if } \nu_1 < \eta \leq \nu_1 + \nu_2 \leq \eta + \iota \\ \frac{\nu_2}{\iota} & \text{if } \eta \leq \nu_1, \nu_1 + \nu_2 \leq \eta + \iota \\ \frac{\eta + \iota - \nu_1}{\iota} & \text{if } \eta \leq \nu_1 \leq \eta + \iota < \nu_1 + \nu_2. \end{cases} \end{aligned}$$

The measure of V restricted to $(0, \iota)$ is

$$\mu(t) = f_{U+T_D}\left(t+\nu_1+\nu_2\right)$$
$$= \begin{cases} 0 & \text{if } \nu_1+\nu_2 > \eta+\iota \\ 0 & \text{if } \nu_1+\nu_2+\iota < \eta \\ \frac{1}{\iota} & \text{for } \eta-\nu_1-\nu_2 < t \leq \iota \text{ if } \nu_1+\nu_2 < \eta \leq \nu_1+\nu_2+\iota \\ \frac{1}{\iota} & \text{for } 0 < t \leq \eta+\iota-\nu_1-\nu_2 \text{ if } \eta \leq \nu_1+\nu_2 \leq \eta+\iota \end{cases}$$

thus, for $\iota \geq \nu_1$,

$$A_3 = \begin{cases} 0 & \text{if } \nu_1+\nu_2 > \eta+\iota \\ 0 & \text{if } \nu_1+\nu_2+\iota < \eta \\ 0 & \text{if } \eta \leq \nu_1+\nu_2 \leq \eta+\iota < 2\nu_1+\nu_2 \\ \frac{n-1}{\iota}\left(\iota+\nu_1+\nu_2-\eta\right. & \\ \quad \left.-\frac{1}{\lambda_L}\left(e^{-\lambda_L(\eta-2\nu_1-\nu_2)}-e^{-\lambda_L(\iota-\nu_1)}\right)\right) & \text{if } 2\nu_1+\nu_2 < \eta \leq \nu_1+\nu_2+\iota \\ \frac{n-1}{\iota}\left(\iota-\nu_1-\frac{1}{\lambda_L}\left(1-e^{-\lambda_L(\iota-\nu_1)}\right)\right) & \text{if } \nu_1+\nu_2 < \eta \leq 2\nu_1+\nu_2 \\ \frac{n-1}{\iota}\left(\iota+\eta-2\nu_1-\nu_2\right. & \\ \quad \left.-\frac{1}{\lambda_L}\left(1-e^{-\lambda_L(\eta+\iota-2\nu_1-\nu_2)}\right)\right) & \text{if } 2\nu_1+\nu_2-\iota \leq \eta \leq \nu_1+\nu_2 \end{cases}$$

while, for $\theta \geq 0$,

$$B_3(\theta) = \begin{cases} 0 & \text{if } \nu_1+\nu_2 > \eta+\iota \\ 0 & \text{if } \nu_1+\nu_2+\iota < \eta \\ \frac{\eta+\iota-\nu_1-\nu_2}{\iota} & \text{if } \eta \leq \nu_1+\nu_2 \leq \eta+\iota < 2\nu_1+\nu_2 \\ \frac{1}{\iota}\chi\left(\theta,0,\eta-\nu_1-\nu_2,\iota\right) & \text{if } 2\nu_1+\nu_2 < \eta \leq \nu_1+\nu_2+\iota \\ \frac{1}{\iota}\left(2\nu_1+\nu_2-\eta+\chi\left(\theta,0,\nu_1,\iota\right)\right) & \text{if } \nu_1+\nu_2 < \eta \leq 2\nu_1+\nu_2 \\ \frac{1}{\iota}\left(\nu_1+\chi\left(\theta,0,\nu_1,\eta+\iota-\nu_1-\nu_2\right)\right) & \text{if } \eta \leq \nu_1+\nu_2 \leq \eta+\iota. \end{cases}$$

and

$$B_4(\theta) = \begin{cases} 0 & \text{if } \nu_1 + \nu_2 > \eta + \iota \\ 0 & \text{if } \nu_1 + \nu_2 + \iota < \eta \\ \frac{\iota + \nu_1 + \nu_2 - \eta}{\iota} & \text{if } \nu_1 + \nu_2 < \eta \leq \nu_1 + \nu_2 + \iota \\ \frac{\iota + \eta - \nu_1 - \nu_2}{\iota} & \text{if } 2\nu_1 + \nu_2 - \iota \leq \eta \leq \nu_1 + \nu_2, \end{cases}$$

The probability of event C_5 given that B is named is

$$\begin{aligned} q_5 &= (1 - p_I)\,\mathrm{P}\left(U + T_D \geq T_{L,1} + T_{L,2} + \iota\right) \\ &= (1 - p_I)\left(1 - F_{U+T_D}(\nu_1 + \nu_2 + \iota)\right) \\ &= \begin{cases} 1 - p_I & \text{if } \nu_1 + \nu_2 + \iota < \eta \\ (1 - p_I)\,\frac{\eta - \nu_1 - \nu_2}{\iota} & \text{if } \eta \leq \nu_1 + \nu_2 + \iota \leq \eta + \iota \\ 0 & \text{if } \nu_1 + \nu_2 > \eta \end{cases} \end{aligned}$$

while the probability of event C_6 given that B is named is

$$q_6 = \begin{cases} p_I & \text{if } \nu_1 + \nu_2 + \iota < \eta \\ p_I\,\frac{\eta - \nu_1 - \nu_2}{\iota} & \text{if } \eta \leq \nu_1 + \nu_2 + \iota \leq \eta + \iota \\ 0 & \text{if } \nu_1 + \nu_2 > \eta. \end{cases}$$

4.4 Exponential latent periods

Suppose that $T_{L,1} \sim \mathrm{Exp}\,(\mu_1)$ and $T_{L,2} \sim \mathrm{Exp}\,(\mu_2)$, then $T_{L,1} + T_{L,2}$ has probability density function

$$\begin{aligned} f_{T_{L,1}+T_{L,2}}(t) &= \int_0^t \mu_1 e^{-\mu_1 u} \mu_2 e^{-\mu_2 (t-u)} du \\ &= \begin{cases} \frac{\mu_1 \mu_2}{\mu_1 - \mu_2}\left(e^{-\mu_2 t} - e^{-\mu_1 t}\right) & \text{if } \mu_1 \neq \mu_2 \\ \mu_1^2 t e^{-\mu_1 t} & \text{if } \mu_1 = \mu_2 \end{cases}, \end{aligned}$$

and cumulative distribution function

$$F_{T_{L,1}+T_{L,2}}(t) = \int_0^t \mu_1 e^{-\mu_1 u} \mu_2 e^{-\mu_2 (t-u)} du$$
$$= \begin{cases} 1 - \frac{\mu_2}{\mu_2 - \mu_1} e^{-\mu_1 t} - \frac{\mu_1}{\mu_1 - \mu_2} e^{-\mu_2 t} & \text{if } \mu_1 \neq \mu_2 \\ 1 - e^{-\mu_1 t} - \mu_1 t e^{-\mu_1 t} & \text{if } \mu_1 = \mu_2 \end{cases}.$$

We now look at the cases where $V \geq 0$.

Let $\Delta = \left\{(i, j_1, j_2, k) \in \mathbb{Z}_+^4 : i + j_1 + j_2 + k = n, k \geq 1\right\}$ be the set of possible states of $\{(S(t), L_1(t), L_2(t), I(t)) : 0 \leq t \leq \iota\}$, n' be the cardinality of Δ and $h : \Delta \to 1, 2, \ldots, n'$ be bijective. Then the household epidemic process between B becoming infectious and vaccination can be represented by a process $\{X(t) : 0 \leq t \leq V\}$, where $X(t) = h\left(S(t), L_1(t), L_2(t), I(t)\right)$ and $X(0) = x_0 = h(n-1, 0, 0, 1)$. Let $\{X(t) : 0 \leq t \leq V\}$ have transition-rate matrix, Q, obtained by the following transition table for $\{(S(t), L_1(t), L_2(t), I(t))\}$

from	to	rate
$(i, j_1, j_2, n-i-j_1-j_2)$	$(i-1, j_1+1, j_2, n-i-j_1-j_2)$	$\lambda_L i\,(n-i-j_1-j_2)$
	$(i, j_1-1, j_2+1, n-i-j_1-j_2)$	$\mu_1 j_1$
	$(i, j_1, j_2-1, n-i-j_1-j_2+1)$	$\mu_2 j_2$.

In case C_3 ($0 \leq V < \iota$) and C_5 ($V = \iota$) we need only determine the expected number of B's housemates who are vaccine-insensitive at time V. If we let $\boldsymbol{\omega} = (\omega_1, \omega_2, \ldots, \omega_{n'})^\top$, where $\omega_{h(i,j_1,j_2,k)} = j_2 + k - 1$, and let $\boldsymbol{x}_0$ be a 1-by-n' vector with the x_0th entry being 1 and all other entries zero, then

$$A_3 = \left(\int_0^\iota e^{Qt}\, d\mu(t) \right)_{x_0} \boldsymbol{\omega}$$
$$= \boldsymbol{x}_0 \hat{A}_3 \boldsymbol{\omega}$$
$$A_5 = \boldsymbol{x}_0 e^{Q\iota} \boldsymbol{\omega},$$

letting $\hat{A}_3 = \int_0^\iota e^{Qt}\, d\mu(t)$ while, if we let $e^{-\iota\theta\boldsymbol{\omega}} = \left(e^{-\iota\theta\omega_1}, e^{-\iota\theta\omega_2}, \ldots, e^{-\iota\theta\omega_{n'}}\right)^\top$, then, for $\theta \geq 0$,

$$B_3(\theta) = \boldsymbol{x}_0 \hat{A}_3 e^{-\iota\theta\boldsymbol{\omega}}$$

$$B_5(\theta) = \boldsymbol{x}_0 e^{Q\iota} e^{-\iota\theta\boldsymbol{\omega}}.$$

In cases C_4 ($0 \leq V < \iota$) and C_6 ($V = \iota$) we are interested in the total active severity of the household up until the first removal. Noting that in $\{X(t) : 0 \leq t \leq V\}$, the state which corresponds to all individuals being infective is absorbing (without loss of generality we shall assume that this is state n'), so Q is singular. States $\{1, 2, \ldots, n'-1\}$ form a transient class, so Q has the form

$$Q = \begin{bmatrix} Q_T & -Q_T\mathbf{1} \\ \mathbf{0} & 0 \end{bmatrix},$$

where Q_T is non-singular.

Note that $\{Z(t)|0 \leq t \leq V\}$ is a reward process, similar to that used for the isolation at the first removal case in Chapter 2, since $Z(t) = \int_0^t I(u) - 1\, du$ is the total active severity of B's housemates up until time t. Thus, for $0 \leq t < V$, if $X(t)$ is in state $h(i, j_1, j_2, k)$, so there are k infectives, reward is earned at rate $\rho_{h(i,j_1,j_2,k)} = k - 1$ per unit time. Let $\boldsymbol{\rho}_T = (\rho_1, \rho_2, \ldots, \rho_{n'-1})^\top$, $D = \text{diag}(\rho_1, \rho_2, \ldots, \rho_{n'})$ and $\boldsymbol{x}_{0T}$ be a 1-by-$n'-1$ vector with the x_0th entry being 1 and all other entries zero. Then for $0 \leq t \leq V$, adapting Eqn. (2.4.2)

$$\mathrm{E}\left[Z(t)\right] = \boldsymbol{x}_{0T}\left(Q_T^{-1}(e^{Q_T t} - I)\boldsymbol{\rho}_T + \left(t\mathbf{1} - Q_T^{-1}\left(e^{Q_T t} - I\right)\mathbf{1}\right)(n-1)\right),$$

and, for $\theta \geq 0$,

$$\mathrm{E}\left[e^{-\theta Z(t)}\right] = \boldsymbol{x}_0 e^{(Q-\theta D)t}\mathbf{1}.$$

Therefore,

$$\begin{aligned} A_6 &= \mathrm{E}\left[Z(\iota)|V=\iota\right] \\ &= \boldsymbol{x}_{0T}\left(Q_T^{-1}(e^{Q_T\iota}-I)\boldsymbol{\rho}_T + \left(\iota\mathbf{1} - Q_T^{-2}\left(e^{Q_T\iota}-I\right)Q_T\mathbf{1}\right)(n-1)\right) \end{aligned}$$

and, for $\theta \geq 0$,

$$\begin{aligned} B_6(\theta) &= \mathrm{E}\left[e^{-\theta Z(\iota)}\middle| V=\iota\right] \\ &= \boldsymbol{x}_0\left(e^{(Q-\theta D)\iota}\mathbf{1}\right)_{x_0}. \end{aligned}$$

Suppose now that $0 \leq V < \iota$ (i.e. case C_4). Then we let Q_V be the transition-rate matrix of $\{X(t) : V \leq t \leq \iota\}$, obtained by the following transition table for $\{(S(t), L_1(t), L_2(t), I(t))\}$,

from	to	rate
(i, j_1, j_2, k)	$(i, j_1, j_2-1, k+1)$	$\mu_2 j_2$.

Now we have a set of absorbing states (which we assume to have cardinality $\tilde{n}$) corresponding to $j_2 = 0$, i.e. no vaccine-insensitive latents, while the remaining states form a transient class. We assume without loss of generality that the set of absorbing states is $\Delta_A = \{n' - \tilde{n} + 1, n' - \tilde{n} + 2, \ldots, n'\}$. So, Q_V has the form

$$Q_V = \begin{bmatrix} Q_{V,T} & Q_{V,A} \\ 0_{\tilde{n},n'-\tilde{n}} & 0_{\tilde{n}} \end{bmatrix},$$

where $Q_{V,T}$ is non-singular, and $Q_{V,A}$ is the matrix of transition rates from the transient states to the absorbing states.

For $0 \leq \tau \leq \iota - V$, let $\tilde{Z}(\tau) = \int_V^{V+\tau} I(u) - 1\, du$ (so $Z(\iota) = Z(V) + \tilde{Z}(\iota - V)$). Then $\left\{\tilde{Z}(\tau) : 0 \leq \tau \leq \iota - V\right\}$ is a reward process where, for $0 \leq \tau \leq \iota - V$, if $X(V+\tau)$ is in state $\tilde{h}\,(i, j_1, j_2, k)$, then reward is earned at rate $\rho_{h(i,j_1,j_2,k)} = k - 1$ per unit of time.

The outcome in this case will depend on the state of the household at vaccination. We have three contributions to $\tilde{Z}(\tau)$ consider: (i) $\tilde{Z}_1(\tau)$, from the case of the household epidemic process being absorbed before or at vaccination; (ii) $\tilde{Z}_2(\tau)$, from the household epidemic process being in the transient class post-vaccination; and (iii) $\tilde{Z}_3(\tau)$, from the household epidemic process being in the absorbing states, having been absorbed post-vaccination. So $\tilde{Z}(\tau) = \tilde{Z}_1(\tau) + \tilde{Z}_2(\tau) + \tilde{Z}_3(\tau)$.

If the single household epidemic has already been absorbed into state $h(i, j_1, 0, k)$, then reward will thereafter been earned at rate k per unit time. If we let W_1 be an n'-by-n' diagonal matrix with the form

$$W_1 = \begin{bmatrix} 0_{n'-\tilde{n}} & 0_{n'-\tilde{n},\tilde{n}} \\ 0_{\tilde{n},n'-\tilde{n}} & I_{\tilde{n}} \end{bmatrix},$$

then

$$\mathrm{E}\left[\tilde{Z}_1(\tau)\middle| V = t\right] = \boldsymbol{x}_0 e^{Qt} W_1 \tau \boldsymbol{\rho}.$$

Let W_2 be an n'-by-$n' - \tilde{n}$ matrix with the form

$$W_2 = \begin{bmatrix} I_{n'-\tilde{n}} \\ 0 \end{bmatrix}.$$

Letting $\boldsymbol{\rho}_{V,T} = (\rho_1, \rho_2, \ldots, \rho_{n'-\tilde{n}})^\top$ and $\boldsymbol{\rho}_{V,A} = (\rho_{n'-\tilde{n}+1}, \rho_{n'-\tilde{n}+2}, \ldots, \rho_{n'})^\top$, then

$$\begin{aligned} \mathrm{E}\left[\tilde{Z}_2(\tau)\middle| V = t\right] &= \boldsymbol{x}_0 e^{Qt} W_2 \int_0^\tau e^{Q_{V,T}u} \boldsymbol{\rho}_{V,T}\, du \\ &= \boldsymbol{x}_0 e^{Qt} W_2 Q_{V,T}^{-1} \left(e^{Q_{V,T}\tau} - I\right) \boldsymbol{\rho}_{V,T} \end{aligned}$$

To obtain $\mathrm{E}\left[\tilde{Z}_3(\tau)\middle| V = t\right]$, we condition on $\tau_A = \min\{\tau : X(V+\tau) \in \Delta_A | \, X(V) \notin \Delta_A\}$:

$$\begin{aligned} \mathrm{E}\left[\tilde{Z}_3(\tau)\middle| V = t\right] &= \mathrm{E}\left[\mathrm{E}\left[\tilde{Z}_3(\tau)\middle| V = t, \tau_A\right]\right] \\ &= \boldsymbol{x}_0 e^{Qt} W_2 \int_0^\tau e^{Q_{V,T}u} Q_{V,A}(\tau - u) \boldsymbol{\rho}_{V,A}\, du \\ &= \boldsymbol{x}_0 e^{Qt} W_2 \left(-\tau Q_{V,T}^{-1} + Q_{V,T}^{-2}\left(e^{Q_{V,T}\tau} - I\right)\right) Q_{V,A} \boldsymbol{\rho}_{V,A}, \end{aligned}$$

so

$$\mathrm{E}\left[\tilde{Z}(\tau)\middle| V=t\right] = \boldsymbol{x}_0\left(e^{Qt}W_1\tau\boldsymbol{\rho} + e^{Qt}W_2Q_{V,T}^{-1}\left(e^{Q_{V,T}\tau}-I\right)\boldsymbol{\rho}_{V,T} \right.$$
$$\left. + e^{Qt}W_2\left(-\tau Q_{V,T}^{-1} + Q_{V,T}^{-2}\left(e^{Q_{V,T}\tau}-I\right)\right)Q_{V,A}\boldsymbol{\rho}_{V,A}\right).$$

Putting this all together, we get

$$\begin{aligned}\mathrm{E}\left[Z(\iota)|V=t\right] &= \mathrm{E}\left[Z(t)|V=t\right] + \mathrm{E}\left[\tilde{Z}(\iota-t)\middle| V=t\right] \\ &= \boldsymbol{x}_{0T}\left(Q_T^{-1}(e^{Q_Tt}-I)\boldsymbol{\rho}_T + \left(t\mathbf{1} - Q_T^{-2}\left(e^{Q_Tt}-I\right)Q_T\mathbf{1}\right)(n-1)\right) \\ &\quad + \boldsymbol{x}_0\left(e^{Qt}W_1(\iota-t)\boldsymbol{\rho} + e^{Qt}W_2Q_{V,T}^{-1}\left(e^{Q_{V,T}(\iota-t)}-I\right)\boldsymbol{\rho}_{V,T}\right. \\ &\quad \left. + e^{Qt}W_2\left(-(\iota-t)Q_{V,T}^{-1} + Q_{V,T}^{-2}\left(e^{Q_{V,T}(\iota-t)}-I\right)\right)Q_{V,A}\boldsymbol{\rho}_{V,A}\right).\end{aligned}$$

Thus

$$\begin{aligned}A_4 &= \int_0^\iota \mathrm{E}\left[Z(\iota)|V=t\right]\, d\mu(t) \\ &= \boldsymbol{x}_{0T}\left(\hat{A}_{4,1}\boldsymbol{\rho}_T + \hat{A}_{4,2}(n-1)\right) + \boldsymbol{x}_0\left(\hat{A}_{4,3}W_1\boldsymbol{\rho} + \hat{A}_{4,4}\boldsymbol{\rho}_{V,T} + \hat{A}_{4,5}\boldsymbol{\rho}_{V,A}\right),\end{aligned}$$

where

$$\begin{aligned}\hat{A}_{4,1} &= \int_0^\iota Q_T^{-1}(e^{Q_Tt}-I)\, d\mu(t) \\ \hat{A}_{4,2} &= \int_0^\iota t\mathbf{1} - Q_T^{-2}\left(e^{Q_Tt}-I\right)Q_T\mathbf{1}\, d\mu(t) \\ &= \int_0^\iota t\mathbf{1}\, d\mu(t) - \hat{A}_{4,1}\mathbf{1} \\ \hat{A}_{4,3} &= \int_0^\iota e^{Qt}(\iota-t)\, d\mu(t) \\ \hat{A}_{4,4} &= \int_0^\iota e^{Qt}W_2Q_{V,T}^{-1}\left(e^{Q_{V,T}(\iota-t)}-I\right)\, d\mu(t) \\ &= \int_0^\iota e^{Qt}W_2Q_{V,T}^{-1}e^{Q_{V,T}(\iota-t)}\, d\mu(t) - \hat{A}_3W_2Q_{V,T}^{-1} \\ \hat{A}_{4,5} &= \int_0^\iota e^{Qt}W_2\left(-(\iota-t)Q_{V,T}^{-1} + Q_{V,T}^{-2}\left(e^{Q_{V,T}(\iota-t)}-I\right)\right)Q_{V,A}\, d\mu(t) \\ &= -\left(\hat{A}_{4,3}W_2 + \hat{A}_{4,4}\right)Q_{V,T}^{-1}Q_{V,A},\end{aligned}$$

while, for $\theta \geq 0$,

$$\begin{aligned} B_4(\theta) &= \left(\int_0^\iota e^{(Q-\theta D)t} e^{(Q_V-\theta D)(\iota-t)} \, d\mu(t) \mathbf{1} \right)_{x_0} \\ &= \left(\hat{B}_4(\theta) \mathbf{1} \right)_{x_0}, \end{aligned}$$

where $\hat{B}_4(\theta) = \int_0^\iota e^{(Q-\theta D)t} e^{(Q_V-\theta D)(\iota-t)} \, d\mu(t)$.

Notation

For square matrix Ω, $k \geq 0$ and $a < b$, let

$$g_k(\Omega, a, b) = \int_a^b t^k e^{\Omega t} \, dt,$$

then,

$$g_k(\Omega, a, b) = \sum_{j=0}^{\infty} \frac{1}{j!(j+k+1)} \Omega^j \left(b^{j+k+1} - a^{j+k+1} \right).$$

Note that if Ω is non-singular,

$$g_k(\Omega, a, b) = \sum_{i=0}^{k} \frac{k!}{i!} (-1)^{k-i} \Omega^{-(k-i+1)} \left(b^i e^{\Omega b} - a^i e^{\Omega a} \right).$$

For square matrices Ω_1 (size m_1-by-m_1) and Ω_2 (size m_2-by-m_2), and matrix W (size m_1-by-m_2), $k \geq 0$ and $a < b$, let

$$h_k\left(\Omega_1, W, \Omega_2, a, b\right) = \int_a^b t^k e^{\Omega_1 t} W e^{\Omega_2 t} \, dt,$$

then,

$$h_k\left(\Omega_1, W, \Omega_2, a, b\right) = \sum_{i=0}^{\infty} \sum_{j=0}^{\infty} \frac{1}{i!j!(i+j+k+1)} \Omega_1^i W \Omega_2^j \left(b^{i+j+k+1} - a^{i+j+k+1} \right).$$

4.4.1 Exponential delay

Suppose that $T_D \sim \text{Exp}(\xi)$.

The probability of event C_1 given that B is named is

$$\begin{aligned} q_1 &= \mathrm{P}\left(U + T_D < T_{L,1}\right) \\ &= \int_0^\infty f_{U+T_D}(t) e^{-\mu_1 t} dt \\ &= \frac{\xi}{\mu_1 \iota(\mu_1 + \xi)} \left(1 - e^{-\mu_1 \iota}\right) \end{aligned}$$

while probability of event C_5 given that B is named is

$$\begin{aligned} q_5 &= (1 - p_I)\, \mathrm{P}\left(U + T_D \geq T_{L,1} + T_{L,2} + \iota\right) \\ &= (1 - p_I) \int_\iota^\infty f_{U+T_D}(t) F_{T_{L,1}+T_{L,2}}(t - \iota)\, dt \\ &= \frac{(1 - p_I)\, \mu_1 \mu_2}{\xi \iota\, (\mu_1 + \xi)\, (\mu_2 + \xi)} \left(1 - e^{-\xi \iota}\right), \end{aligned}$$

and the probability of event C_6 given that B is named is

$$\begin{aligned} q_6 &= p_I \mathrm{P}\left(U + T_D \geq T_{L,1} + T_{L,2} + \iota\right) \\ &= \frac{p_I \mu_1 \mu_2}{\xi \iota\, (\mu_1 + \xi)\, (\mu_2 + \xi)} \left(1 - e^{-\xi \iota}\right). \end{aligned}$$

We shall suppose now that $\mu_1 \neq \mu_2$. The probability of event C_2 given that B is named is

$$\begin{aligned} q_2 &= \mathrm{P}\left(T_{L,1} \leq U + T_D < T_{L,1} + T_{L,2}\right) \\ &= \int_0^\infty f_{U+T_D}(t) \left(1 - F_{T_{L,1}+T_{L,2}}(t)\right)\, dt - q_1 \\ &= \int_0^\iota \frac{1}{\iota} \left(1 - e^{-\xi t}\right) \left(1 - F_{T_{L,1}+T_{L,2}}(t)\right)\, dt \\ &\quad + \int_\iota^\infty \frac{1}{\iota} e^{-\xi t} \left(e^{\xi \iota} - 1\right) \left(1 - F_{T_{L,1}+T_{L,2}}(t)\right)\, dt - q_1 \\ &= \frac{\xi}{\iota\, (\mu_1 - \mu_2)} \left(\frac{\mu_1}{\mu_2\, (\mu_2 + \xi)} \left(1 - e^{-\mu_2 \iota}\right) - \frac{\mu_2}{\mu_1\, (\mu_1 + \xi)} \left(1 - e^{-\mu_1 \iota}\right) \right) - q_1 \\ &= \frac{\xi}{\iota\, (\mu_1 - \mu_2)} \left(\frac{\mu_1}{\mu_2\, (\mu_2 + \xi)} \left(1 - e^{-\mu_2 \iota}\right) - \frac{1}{(\mu_1 + \xi)} \left(1 - e^{-\mu_1 \iota}\right) \right). \end{aligned}$$

The measure of V restricted to $(0, \iota)$ is

$$\begin{aligned}
\mu(t) &= \int_0^\infty f_{T_{L,1}+T_{L,2}}(\tau) f_{U+T_D}(\tau + t)\, d\tau \\
&= \int_0^{\iota - t} f_{T_{L,1}+T_{L,2}}(\tau) \frac{1}{\iota} \left(1 - e^{-\xi(t+\tau)}\right) d\tau \\
&\quad + \int_{\iota - t}^\infty f_{T_{L,1}+T_{L,2}}(\tau) \frac{1}{\iota} e^{-\xi(t+\tau)} \left(e^{\xi\iota} - 1\right) d\tau \\
&= \frac{1}{\iota} \left[1 - \frac{\mu_1\mu_2}{(\mu_1 + \xi)(\mu_2 + \xi)} e^{-\xi t} \right. \\
&\qquad \left. - \frac{\xi\mu_1}{(\mu_1 - \mu_2)(\mu_2 + \xi)} e^{-\mu_2(\iota - t)} + \frac{\xi\mu_2}{(\mu_1 - \mu_2)(\mu_1 + \xi)} e^{-\mu_1(\iota - t)}\right],
\end{aligned}$$

and so,

$$\begin{aligned}
\hat{A}_3 &= \int_0^\infty e^{Qt} \mu(t)\, dt \\
&= \frac{1}{\iota} g_0(Q, 0, \iota) - \frac{\mu_1\mu_2}{\iota(\mu_1 + \xi)(\mu_2 + \xi)} g_0(Q - \xi I, 0, \iota) \\
&\quad - \frac{\xi\mu_1}{\iota(\mu_1 - \mu_2)(\mu_2 + \xi)} e^{-\mu_2\iota} g_0(Q + \mu_2 I, 0, \iota) \\
&\quad + \frac{\xi\mu_2}{\iota(\mu_1 - \mu_2)(\mu_1 + \xi)} e^{-\mu_1\iota} g_0(Q + \mu_1 I, 0, \iota)
\end{aligned}$$

while

$$\begin{aligned}
\hat{A}_{4,1} &= \frac{1}{\iota} Q_T^{-1} \left[g_0(Q_T, 0, \iota) - \iota I \right. \\
&\quad - \frac{\mu_1\mu_2}{(\mu_1 + \xi)(\mu_2 + \xi)} \left(g_0(Q_T - \xi I, 0, \iota) - \frac{1}{\xi}\left(1 - e^{-\xi\iota}\right) I \right) \\
&\quad - \frac{\xi\mu_1}{(\mu_1 - \mu_2)(\mu_2 + \xi)} e^{-\mu_2\iota} \left(\frac{1}{\mu_2}\left(1 - e^{\mu_2\iota}\right) I + g_0(Q_T + \mu_2 I, 0, \iota) \right) \\
&\quad \left. + \frac{\xi\mu_2}{(\mu_1 - \mu_2)(\mu_1 + \xi)} e^{-\mu_1\iota} \left(\frac{1}{\mu_1}\left(1 - e^{\mu_1\iota}\right) I + g_0(Q_T + \mu_1 I, 0, \iota) \right) \right], \\
\hat{A}_{4,2} &= \frac{1}{\iota} \left[\frac{\iota^2}{2} - \frac{\mu_1\mu_2}{\xi^2(\mu_1 + \xi)(\mu_2 + \xi)} \left(1 - e^{-\xi\iota}(\xi\iota + 1)\right) \right. \\
&\quad - \frac{\xi\mu_1}{\mu_2^2(\mu_1 - \mu_2)(\mu_2 + \xi)} \left(e^{-\mu_2\iota} + \mu_2\iota - 1\right) \\
&\quad \left. + \frac{\xi\mu_2}{\mu_1^2(\mu_1 - \mu_2)(\mu_1 + \xi)} \left(e^{-\mu_1\iota} + \mu_1\iota - 1\right) \right] \mathbf{1} \\
&\quad - \hat{A}_{4,1}\mathbf{1},
\end{aligned}$$

$$\begin{aligned}\hat{A}_{4,3} = \frac{e^{Q\iota}}{\iota}\Big[&g_1(-Q,0,\iota)\\ &-\frac{\mu_1\mu_2 e^{-\xi\iota}}{(\mu_1+\xi)(\mu_2+\xi)}g_1(\xi I-Q,0,\iota)\\ &-\frac{\xi\mu_1}{(\mu_1-\mu_2)(\mu_2+\xi)}g_1(-Q-\mu_2 I,0,\iota)\\ &+\frac{\xi\mu_2}{(\mu_1-\mu_2)(\mu_1+\xi)}g_1(-Q-\mu_1 I,0,\iota)\Big]W_1,\\ \hat{A}_{4,4} = \frac{e^{Q\iota}}{\iota}\Big[&h_0(-Q,W_2,Q_{V,T},0,\iota)\\ &-\frac{\mu_1\mu_2 e^{-\xi\iota}}{(\mu_1+\xi)(\mu_2+\xi)}h_0(\xi I-Q,W_2,Q_{V,T},0,\iota)\\ &-\frac{\xi\mu_1}{(\mu_1-\mu_2)(\mu_2+\xi)}h_0(-Q-\mu_2 I,W_2,Q_{V,T},0,\iota)\\ &+\frac{\xi\mu_2}{(\mu_1-\mu_2)(\mu_1+\xi)}h_0(-Q-\mu_1 I,W_2,Q_{V,T},0,\iota)\Big]Q_{V,T}^{-1}\\ &-\hat{A}_3 W_2 Q_{V,T}^{-1},\end{aligned}$$

and, for $\theta \geq 0$,

$$\begin{aligned}\hat{B}_4(\theta) = \frac{e^{(Q-\theta D)\iota}}{\iota}\Big[&h_0(\theta D-Q,I,Q_V-\theta D,0,\iota)\\ &-\frac{\mu_1\mu_2 e^{-\xi\iota}}{(\mu_1+\xi)(\mu_2+\xi)}h_0(\theta D-Q+\xi I,I,Q_V-\theta D,0,\iota)\\ &-\frac{\xi\mu_1}{(\mu_1-\mu_2)(\mu_2+\xi)}h_0(\theta D-Q-\mu_2 I,I,Q_V-\theta D,0,\iota)\\ &+\frac{\xi\mu_2}{(\mu_1-\mu_2)(\mu_1+\xi)}h_0(\theta D-Q-\mu_1 I,I,Q_V-\theta D,0,\iota)\Big].\end{aligned}$$

Suppose now that $\mu_1 = \mu_2$, then the probability of event C_2 given that B is named is

$$\begin{aligned}q_2 &= \frac{\xi}{\iota(\mu_1+\xi)}\left[\frac{3\mu_1+2\xi}{\mu_1(\mu_1+\xi)}\left(1-e^{-\mu_1\iota}\right)-\iota e^{-\mu_1\iota}\right]-q_1\\ &= \frac{\xi}{\iota(\mu_1+\xi)}\left[\frac{2\mu_1+\xi}{\mu_1(\mu_1+\xi)}\left(1-e^{-\mu_1\iota}\right)-\iota e^{-\mu_1\iota}\right].\end{aligned}$$

The measure of V restricted to $(0,\iota)$ is

$$\mu(t) = \frac{1}{\iota}\left[1-\frac{\mu_1^2}{(\mu_1+\xi)^2}e^{-\xi t}-\frac{\xi(\xi+2\mu_1)}{(\mu_1+\xi)^2}e^{-\mu_1(\iota-t)}-\frac{\xi\mu_1(\iota-t)}{\mu_1+\xi}e^{-\mu_1(\iota-t)}\right],$$

and so,

$$\begin{aligned}\hat{A}_3 = &\frac{1}{\iota} g_0(Q,0,\iota) - \frac{\mu_1^2}{\iota\left(\mu_1+\xi\right)^2} g_0(Q-\xi I,0,\iota) \\ &- \frac{\xi\left(\xi+2\mu_1\right)}{\iota\left(\mu_1+\xi\right)^2} e^{-\mu_1\iota} g_0\left(Q+\mu_1 I,0,\iota\right) - \frac{\xi\mu_1}{\iota\left(\mu_1+\xi\right)} e^{Q\iota} g_1\left(-Q-\mu_1 I,0,\iota\right),\end{aligned}$$

while

$$\begin{aligned}\hat{A}_{4,1} = &\frac{1}{\iota} Q_T^{-1}\left[g_0\left(Q_T,0,\iota\right) - \iota I\right. \\ &- \frac{\mu_1^2}{\left(\mu_1+\xi\right)^2}\left(g_0\left(Q_T+\xi I,0,\iota\right) - \frac{1}{\xi}\left(1-e^{-\xi\iota}\right) I\right) \\ &- \frac{\xi\left(\xi+2\mu_1\right)}{\left(\mu_1+\xi\right)^2} e^{-\mu_1\iota}\left(\frac{1}{\mu_1}\left(1-e^{\mu_1\iota}\right) I + g_0\left(Q_T+\mu_1 I,0,\iota\right)\right) \\ &\left.- \frac{\xi\mu_1}{\mu_1+\xi}\left(e^{Q_T\iota} g_1\left(-Q_T-\mu_1 I,0,\iota\right) - \frac{1}{\mu_1^2}\left(1-e^{-\mu_1\iota}\left(1+\mu_1\iota\right)\right)\right)\right], \\ \hat{A}_{4,2} = &\frac{1}{\iota}\left[\frac{\iota^2}{2} - \frac{\mu_1^2}{\xi^2\left(\mu_1+\xi\right)^2}\left(1-e^{-\xi_\iota}(\xi\iota+1)\right)\right. \\ &- \frac{\xi\left(\xi+2\mu_1+\mu_1\iota\left(\mu_1+\xi\right)\right)}{\mu_1^2\left(\mu_1+\xi\right)^2}\left(e^{-\mu_1\iota}+\mu_1\iota-1\right) \\ &\left.+ \frac{\xi}{\mu_1+\xi}\left(\iota^2 - \frac{2\iota}{\mu_1} + \frac{2}{\mu_1^2} - \frac{2}{\mu_1^2} e^{-\mu_1\iota}\right)\right] \\ &- \hat{A}_{4,1}\mathbf{1}, \\ \hat{A}_{4,3} = &\frac{e^{Q\iota}}{\iota}\left[g_1\left(-Q,0,\iota\right)\right. \\ &- \frac{\mu_1^2 e^{-\xi\iota}}{\left(\mu_1+\xi\right)^2} g_1\left(\xi I-Q,0,\iota\right) \\ &- \frac{\xi\left(\xi+2\mu_1\right)}{\left(\mu_1+\xi\right)^2} g_1\left(-Q-\mu_1 I,0,\iota\right) \\ &\left.- \frac{\xi\mu_1}{\mu_1+\xi} g_2\left(-Q-\mu_1 I,0,\iota\right)\right] W_1,\end{aligned}$$

$$\begin{aligned}\hat{A}_{4,4} = \frac{e^{Q\iota}}{\iota} \Big[& h_0\left(-Q, W_2, Q_{V,T}, 0, \iota\right) \\ & - \frac{\mu_1^2 e^{-\xi\iota}}{\left(\mu_1+\xi\right)^2} h_0\left(\xi I - Q, W_2, Q_{V,T}, 0, \iota\right) \\ & - \frac{\xi\left(\xi+2\mu_1\right)}{\left(\mu_1+\xi\right)^2} h_0\left(-Q-\mu_1 I, W_2, Q_{V,T}, 0, \iota\right) \\ & - \frac{\xi\mu_1}{\mu_1+\xi} h_1\left(-Q-\mu_1 I, W_2, Q_{V,T}, 0, \iota\right) \Big] Q_{V,T}^{-1} \\ & - \hat{A}_3 W_2 Q_{V,T}^{-1},\end{aligned}$$

and, for $\theta \geq 0$,

$$\begin{aligned}\hat{B}_4(\theta) = \frac{e^{(Q-\theta D)\iota}}{\iota} \Big[& h_0\left(\theta D - Q, I, Q_V - \theta D, 0, \iota\right) \\ & - \frac{\mu_1^2 e^{-\xi\iota}}{\left(\mu_1+\xi\right)^2} h_0\left(\theta D - Q + \xi I, I, Q_V - \theta D, 0, \iota\right) \\ & - \frac{\xi\left(\xi+2\mu_1\right)}{\left(\mu_1+\xi\right)^2} h_0\left(\theta D - Q - \mu_1 I, I, Q_V - \theta D, 0, \iota\right) \\ & - \frac{\xi\mu_1}{\mu_1+\xi} h_1\left(\theta D - Q - \mu_1 I, I, Q_V - \theta D, 0, \iota\right) \Big].\end{aligned}$$

4.4.2 Constant delay

Suppose that $T_D \equiv \eta$.

The probability of event C_1 given that B is named is

$$\begin{aligned} q_1 &= \mathrm{P}\left(U + T_D < T_{L,1}\right) \\ &= \int_0^\infty f_{U+T_D}(t) e^{-\mu_1 t} dt \\ &= \frac{1}{\mu_1 \iota} e^{-\mu_1 \eta}\left(1 - e^{-\mu_1 \iota}\right), \end{aligned}$$

We shall suppose now that $\mu_1 \neq \mu_2$. Then the probability of event C_2 given that B is

named is

$$\begin{aligned}
q_2 &= \mathrm{P}\left(T_{L,1} \leq U + T_D < T_{L,1} + T_{L,2}\right) \\
&= \int_0^\infty f_{U+T_D}(t)\left(1 - F_{T_{L,1}+T_{L,2}}(t)\right)\, dt - q_1 \\
&= \frac{1}{\iota\left(\mu_1 - \mu_2\right)}\left(\frac{\mu_1}{\mu_2}e^{-\mu_2\eta}\left(1 - e^{-\mu_2\iota}\right) - \frac{\mu_2}{\mu_1}e^{-\mu_1\eta}\left(1 - e^{-\mu_1\iota}\right)\right) - q_1 \\
&= \frac{1}{\iota\left(\mu_1 - \mu_2\right)}\left(\frac{\mu_1}{\mu_2}e^{-\mu_2\eta}\left(1 - e^{-\mu_2\iota}\right) - e^{-\mu_1\eta}\left(1 - e^{-\mu_1\iota}\right)\right).
\end{aligned}$$

The measure of V restricted to $(0, \iota)$ is

$$\begin{aligned}
\mu(t) &= \int_0^\infty f_{T_{L,1}+T_{L,2}}(\tau) f_{U+T_D}(\tau + t)\, d\tau \\
&= \begin{cases} \int_{\eta-t}^{\eta+\iota-t} f_{T_{L,1}+T_{L,2}}(\tau) f_{U+T_D}(\tau + t)\, d\tau & \text{if } t \leq \eta \\ \int_0^{\eta+\iota-t} f_{T_{L,1}+T_{L,2}}(\tau) f_{U+T_D}(\tau + t)\, d\tau & \text{if } t > \eta \end{cases} \\
&= \frac{1}{\iota}\Bigg[1_{\{t>\eta\}} + \frac{1}{\mu_1 - \mu_2}\Big(\mu_1 e^{-\mu_2(\eta-t)}\left(1_{\{t\leq\eta\}} - e^{-\mu_2\iota}\right) \\
&\qquad\qquad -\mu_2 e^{-\mu_1(\eta-t)}\left(1_{\{t\leq\eta\}} - e^{-\mu_1\iota}\right)\Big)\Bigg],
\end{aligned}$$

and so,

$$\begin{aligned}
\hat{A}_3 &= \int_0^\iota e^{Qt}\mu(t)\, dt \\
&= \frac{1}{\iota}\Big[1_{\{\iota>\eta\}} g_0(Q, \eta, \iota) \\
&\quad + \frac{1}{\mu_1 - \mu_2}\Big(\mu_1 e^{-\mu_2\eta}\left(g_0\left(Q + \mu_2 I, 0, \min(\eta, \iota)\right) - e^{-\mu_2\iota} g_0\left(Q + \mu_2 I, 0, \iota\right)\right) \\
&\qquad -\mu_2 e^{-\mu_1\eta}\left(g_0\left(Q + \mu_1 I, 0, \min(\eta, \iota)\right) - e^{-\mu_1\iota} g_0\left(Q + \mu_1 I, 0, \iota\right)\right)\Big)\Big],
\end{aligned}$$

while

$$\begin{aligned}
\hat{A}_{4,1} = \frac{Q_T^{-1}}{\iota} \Big[& 1_{\{\iota>\eta\}} \left(g_0 \left(Q_T, \eta, \iota \right) - \left(\iota - \eta \right) I \right) \\
& + \frac{1}{\mu_1 - \mu_2} \Bigg(\mu_1 e^{-\mu_2 \eta} \Bigg(g_0 \left(Q_T + \mu_2 I, 0, \min(\eta, \iota) \right) - \frac{1}{\mu_2} \left(e^{\mu_2 \min(\eta,\iota)} - 1 \right) I \\
& \qquad - e^{-\mu_2 \iota} \left(g_0 \left(Q_T + \mu_2 I, 0, \iota \right) - \frac{1}{\mu_2} \left(e^{\mu_2 \iota} - 1 \right) I \right) \Bigg) \\
& \qquad - \mu_2 e^{-\mu_1 \eta} \Bigg(g_0 \left(Q_T + \mu_1 I, 0, \min(\eta, \iota) \right) - \frac{1}{\mu_1} \left(e^{\mu_1 \min(\eta,\iota)} - 1 \right) I \\
& \qquad - e^{-\mu_1 \iota} \left(g_0 \left(Q_T + \mu_1 I, 0, \iota \right) - \frac{1}{\mu_1} \left(e^{\mu_1 \iota} - 1 \right) I \right) \Bigg) \Bigg) \Bigg], \\
\hat{A}_{4,2} = \frac{1}{\iota} \Bigg[& 1_{\{\iota>\eta\}} \frac{\iota^2 - \eta^2}{2} \\
& + \frac{1}{\mu_1 - \mu_2} \Bigg(\frac{\mu_1}{\mu_2^2} e^{-\mu_2 \eta} \left(2 + e^{\mu_2 \min(\eta,\iota)} \left(\mu_2 \min(\eta, \iota) - 1 \right) - \mu_2 \iota - e^{-\mu_2 \iota} \right) \\
& \qquad - \frac{\mu_2}{\mu_1^2} e^{-\mu_1 \eta} \left(2 + e^{\mu_1 \min(\eta,\iota)} \left(\mu_1 \min(\eta, \iota) - 1 \right) - \mu_1 \iota - e^{-\mu_1 \iota} \right) \Bigg) \Bigg] \\
& - \hat{A}_{4,1} \mathbf{1}, \\
\hat{A}_{4,3} = \frac{e^{Q\iota}}{\iota} \Big[& 1_{\{\iota>\eta\}} g_1 \left(-Q, 0, \iota - \eta \right) \\
& + \frac{1}{\mu_1 - \mu_2} \big(\mu_1 e^{-\mu_2 \eta} \left(e^{\mu_2 \iota} g_1 \left(-Q - \mu_2 I, \max(0, \iota - \eta), \iota \right) \right. \\
& \qquad \left. - g_1 \left(-Q - \mu_2 I, 0, \iota \right) \right) \\
& \qquad - \mu_2 e^{-\mu_1 \eta} \left(e^{\mu_1 \iota} g_1 \left(-Q - \mu_1 I, \max(0, \iota - \eta), \iota \right) \right. \\
& \qquad \left. - g_1 \left(-Q - \mu_1 I, 0, \iota \right) \right) \big] W_1, \\
\hat{A}_{4,4} = \frac{e^{Q\iota}}{\iota} \Big[& 1_{\{\iota>\eta\}} h_0 \left(-Q, W_2, Q_{V,T}, 0, \iota - \eta \right) \\
& + \frac{1}{\mu_1 - \mu_2} \big(\mu_1 e^{-\mu_2 \eta} \left(e^{\mu_2 \iota} h_0 \left(-Q - \mu_2 I, W_2, Q_{V,T}, \max(0, \iota - \eta), \iota \right) \right. \\
& \qquad \left. - h_0 \left(-Q - \mu_2 I, W_2, Q_{V,T}, 0, \iota \right) \right) \\
& \qquad - \mu_2 e^{-\mu_1 \eta} \left(e^{\mu_1 \iota} h_0 \left(-Q - \mu_1 I, W_2, Q_{V,T}, \max(0, \iota - \eta), \iota \right) \right. \\
& \qquad \left. - h_0 \left(-Q - \mu_1 I, W_2, Q_{V,T}, 0, \iota \right) \right) \big] Q_{V,T}^{-1} \\
& - \hat{A}_3 W_2 Q_{V,T}^{-1},
\end{aligned}$$

and, for $\theta \geq 0$,

$$\begin{aligned}\hat{B}_4(\theta) &= \int_0^{\iota} e^{(Q-\theta D)t} e^{(Q_V-\theta D)(\iota-t)}\, d\mu(t) \\ &= \frac{e^{(Q-\theta D)\iota}}{\iota}\left[1_{\{\iota>\eta\}} h_0\left(\theta D - Q, I, Q_V - \theta D, 0, \iota-\eta\right)\right. \\ &\quad + \frac{1}{\mu_1-\mu_2}\left(\mu_1 e^{-\mu_2\eta}\left(e^{\mu_2\iota} h_0\left(\theta D - Q - \mu_2 I, I, Q_V - \theta D, \max(0,\iota-\eta), \iota\right)\right.\right. \\ &\qquad\qquad \left. - h_0\left(\theta D - Q - \mu_2 I, I, Q_V - \theta D, 0, \iota\right)\right) \\ &\qquad - \mu_2 e^{-\mu_1\eta}\left(e^{\mu_1\iota} h_0\left(\theta D - Q - \mu_1 I, I, Q_V - \theta D, \max(0,\iota-\eta), \iota\right)\right. \\ &\qquad\qquad \left.\left. - h_0\left(\theta D - Q - \mu_1 I, I, Q_V - \theta D, 0, \iota\right)\right)\right)\Bigg].\end{aligned}$$

The probability of event C_5 given that B is named is

$$\begin{aligned}q_5 &= (1-p_I)\,\mathrm{P}\left(U + T_D \geq T_{L,1} + T_{L,2} + \iota\right) \\ &= (1-p_I)\int_{\eta}^{\eta+\iota} f_{U+T_D}(t) F_{T_{L,1}+T_{L,2}}(t-\iota)\, dt \\ &= \frac{(1-p_I)}{\iota}\left[\min\left(\eta,\iota\right) - \frac{1}{(\mu_1-\mu_2)}\left(\frac{\mu_1}{\mu_2}\left(\min\left(1, e^{-\mu_2(\eta-\iota)}\right) - e^{-\mu_2\eta}\right)\right.\right. \\ &\qquad\qquad \left.\left. - \frac{\mu_2}{\mu_1}\left(\min\left(1, e^{-\mu_1(\eta-\iota)}\right) - e^{-\mu_1\eta}\right)\right)\right],\end{aligned}$$

while the probability of event C_6 given that B is named is

$$\begin{aligned}q_6 &= p_I \mathrm{P}\left(U + T_D \geq T_{L,1} + T_{L,2} + \iota\right) \\ &= p_I \int_{\iota}^{\eta+\iota} f_{U+T_D}(t) F_{T_{L,1}+T_{L,2}}(t-\iota)\, dt \\ &= \frac{p_I}{\iota}\left[\min\left(\eta,\iota\right) - \frac{1}{(\mu_1-\mu_2)}\left(\frac{\mu_1}{\mu_2}\left(\min\left(1, e^{-\mu_2(\eta-\iota)}\right) - e^{-\mu_2\eta}\right)\right.\right. \\ &\qquad\qquad \left.\left. - \frac{\mu_2}{\mu_1}\left(\min\left(1, e^{-\mu_1(\eta-\iota)}\right) - e^{-\mu_1\eta}\right)\right)\right],\end{aligned}$$

Now suppose that $\mu_1 = \mu_2$. Then the probability of event C_2 given that B is named is

$$\begin{aligned}q_2 &= \frac{1}{\iota} e^{-\mu_1\eta}\left(\frac{2}{\mu_1} + \eta - \left(\frac{2}{\mu_1} + \eta + \iota\right) e^{-\mu_1\iota}\right) - q_1 \\ &= \frac{1}{\iota} e^{-\mu_1\eta}\left(\frac{1}{\mu_1} + \eta - \left(\frac{1}{\mu_1} + \eta + \iota\right) e^{-\mu_1\iota}\right).\end{aligned}$$

The measure of V restricted to $(0, \iota)$ is

$$\mu(t) = \frac{1}{\iota}\left[1_{\{t>\eta\}} + 1_{\{t\leq\eta\}} e^{-\mu_1(\eta-t)}\left(1+\mu_1(\eta-t)\right) - e^{-\mu_1(\eta+\iota-t)}\left(1+\mu_1(\eta+\iota-t)\right)\right],$$

and so,

$$\begin{aligned}\hat{A}_3 = \frac{1}{\iota}\big[&1_{\{\iota>\eta\}} g_0(Q,\eta,\iota)\\ &+ e^{-\mu_1\eta}\left(\left(1+\mu_1\eta\right) g_0\left(Q+\mu_1 I, 0, \min(\eta,\iota)\right) - \mu_1 g_1\left(Q+\mu_1 I, 0, \min(\eta,\iota)\right)\right.\\ &\left.-e^{-\mu_1\iota}\left(\left(1+\mu_1(\eta+\iota)\right) g_0\left(Q+\mu_1 I, 0, \iota\right) - \mu_1 g_1\left(Q+\mu_1 I, 0, \iota\right)\right)\right)\big],\end{aligned}$$

while

$$\begin{aligned}\hat{A}_{4,1} = \frac{Q_T^{-1}}{\iota}\big[&1_{\{\iota>\eta\}}\left(g_0\left(Q_T,\eta,\iota\right) - \left(\iota-\eta\right) I\right)\\ &+ e^{-\mu_1\eta}\left(\left(1+\mu_1\eta\right)\left(g_0\left(Q_T+\mu_1 I, 0, \min(\eta,\iota)\right) - \frac{1}{\mu_1}\left(e^{\mu_1\min(\eta,\iota)}-1\right) I\right)\right.\\ &\qquad - \mu_1 g_1\left(Q_T+\mu_1 I, 0, \min(\eta,\iota)\right)\\ &\qquad \left.+\frac{1}{\mu_1}\left(e^{\mu_1\min(\eta,\iota)}\left(\mu_1\min(\eta,\iota)-1\right)+1\right) I\right)\\ &- e^{-\mu_1(\eta+\iota)}\left(\left(1+\mu_1\eta\right)\left(g_0\left(Q_T+\mu 1_I, 0, \iota\right) - \frac{1}{\mu_1}\left(e^{\mu_1\iota}-1\right) I\right)\right.\\ &\qquad \left.-\mu_1 g_1\left(Q_T+\mu_1 I, 0, \iota\right) + \frac{1}{\mu_1}\left(e^{\mu_1\iota}\left(\mu_1\iota-1\right)+1\right) I\right)\Big],\end{aligned}$$

$$\begin{aligned}\hat{A}_{4,2} = \frac{1}{\iota}\Bigg[&1_{\{\iota>\eta\}}\frac{\iota^2-\eta^2}{2}\\ &+ e^{-\mu_1\eta}\left(\left(\frac{1}{\mu_1}+\eta\right)\left(e^{\mu_1\min(\eta,\iota)}\left(\min(\eta,\iota)-\frac{1}{\mu_1}\right) - \iota + \frac{1}{\mu_1}\left(2-e^{-\mu_1\iota}\right)\right)\right.\\ &\left.-e^{\mu_1\min(\eta,\iota)}\left(\min(\eta,\iota)^2 - \frac{2\min(\eta,\iota)}{\mu_1} + \frac{2}{\mu_1^2}\right) + \frac{2}{\mu_1^2}\left(2-e^{-\mu_1\iota}\right) + \iota^2 - \frac{2\iota}{\mu_1}\right]\mathbf{1}\\ &- \hat{A}_{4,1}\mathbf{1},\end{aligned}$$

$$\begin{aligned}\hat{A}_{4,3} = \frac{e^{Q\iota}}{\iota}\big[&1_{\{\iota>\eta\}} g_1\left(-Q, 0, \iota-\eta\right)\\ &+ e^{-\mu_1(\eta-\iota)}\left(\left(1+\mu_1(\eta-\iota)\right) g_1\left(-Q-\mu_1 I, \max(0,\iota-\eta), \iota\right)\right.\\ &\qquad \left.+\mu_1 g_2\left(-Q-\mu_1 I, \max(0,\iota-\eta), \iota\right)\right)\\ &-e^{\mu_1\eta}\left(\left(1+\mu_1\eta\right) g_1\left(-Q-\mu_1 I, 0, \iota\right) + \mu_1 g_2\left(-Q-\mu_1 I, 0, \iota\right)\right)\big] W_1,\end{aligned}$$

$$\hat{A}_{4,4} = \frac{e^{Q\iota}}{\iota}\left[1_{\{\iota>\eta\}}h_0\left(-Q, W_2, Q_{V,T}, 0, \iota-\eta\right)\right.$$
$$+ e^{-\mu_1(\eta-\iota)}\left(\left(1+\mu_1(\eta-\iota)\right)h_0\left(-Q-\mu_1 I, W_2, Q_{V,T}, \max(0,\iota-\eta), \iota\right)\right.$$
$$\left.+\mu_1 h_1\left(-Q-\mu_1 I, W_2, Q_{V,T}, \max(0,\iota-\eta), \iota\right)\right)$$
$$- e^{-\mu_1\eta}\left(\left(1+\mu_1\eta\right)h_0\left(-Q-\mu_1 I, W_2, Q_{V,T}, 0, \iota\right)\right.$$
$$\left.\left.+\mu_1 h_1\left(-Q-\mu_1 I, W_2, Q_{V,T}, 0, \iota\right)\right)\right] Q_{V,T}^{-1}$$
$$- \hat{A}_3 W_2 Q_{V,T}^{-1},$$

and, for $\theta \geq 0$,

$$\hat{B}_4(\theta) = \frac{e^{(Q-\theta D)\iota}}{\iota}\left[1_{\{\iota>\eta\}}h_0\left(\theta D - Q, I, Q_V - \theta D, 0, \iota-\eta\right)\right.$$
$$+ e^{-\mu_1(\eta-\iota)}\left(\left(1+\mu_1(\eta-\iota)\right)h_0\left(\theta D - Q - \mu_1 I, I, Q_V - \theta D, \max(0,\iota-\eta), \iota\right)\right.$$
$$+h_1\left(\theta D - Q - \mu_1 I, I, Q_V - \theta D, \max(0,\iota-\eta), \iota\right)\Big)$$
$$- e^{-\mu_1\eta}\left(\left(1+\mu_1\eta\right)h_0\left(\theta D - Q - \mu_1 I, I, Q_V - \theta D, 0, \iota\right)\right.$$
$$\left.\left.+h_1\left(\theta D - Q - \mu_1 I, I, Q_V - \theta D, 0, \iota\right)\right)\right].$$

The probability of event C_5 given that B is named is

$$q_5 = \frac{(1-p_I)}{\iota}\left[\min\left(\eta,\iota\right) - \left(\min\left(1, e^{-\mu_1(\eta-\iota)}\right)\left(\frac{2}{\mu_1} + 1_{\{\eta>\iota\}(\eta-\iota)}\right) - \eta e^{-\mu_1\eta}\right)\right],$$

while the probability of event C_6 given that B is named is

$$q_6 = \frac{p_I}{\iota}\left[\min\left(\eta,\iota\right) - \left(\min\left(1, e^{-\mu_1(\eta-\iota)}\right)\left(\frac{2}{\mu_1} + 1_{\{\eta>\iota\}(\eta-\iota)}\right) - \eta e^{-\mu_1\eta}\right)\right].$$

4.5 Numerical illustrations

In this section we simulate finite-size epidemics to examine the reliability of the branching process approximation used in this chapter, and then use numerical results to illustrate the theory of the chapter.

4.5.1 Comparison with simulations

In order to make analytical progress with this model, we assume that the number of households, m, is large. This assumption enables us to approximate by assuming that, in the early stages of the epidemic, infectives only make global contacts with entirely susceptible households. In real life, m may not be so large, so it is useful to see how quickly the branching process approximation describes the true behaviour of the model, in which we can expect (i) infectives to make global contacts with already infected individuals, and (ii) multiple members of a household being infected as a result of (separate) global contacts. On the one hand we can expect the approximation to have an underestimation effect on the spread of the epidemic as less global infectious contacts will emanate from an infected household (as only one global infectious contact can come into the household) but on the other hand there will be an overestimation effect as more of these contacts will be with susceptible households.

To examine how quickly the approximation becomes reasonable we simulate the full epidemic $n_0 = 10,000$ times for different values of m for three different intervention models (all without isolation, i.e. $p_I = 0$): (i) local tracing only, i.e. $p_c = 0$, (ii) local and global tracing ($p_c = 1$), and (iii) global tracing only ($p_c = 1$), i.e. vaccination is not triggered by the first removal in a household. We have not derived analytical results for model (iii) in this chapter, but to obtain results for the corresponding branching process approximation, we use 100,000 simulations to estimate the final size distribution of a named single household epidemic under this intervention model (analytic results for an unnamed single household epidemic can be used from Ball [5]). We use $n = 4$, $T_D \sim \text{Exp}\left(\frac{1}{3}\right)$ and $T_I \equiv 1$ (so time is relative to a unit-length infectious period). We also use $T_{L_1} \sim \text{Exp}(1)$ and $T_{L_2} \sim \text{Exp}\left(\frac{3}{8}\right)$ (so, respectively, the latent period means are 1 and

$\frac{8}{3}$ times the infectious period mean, as used for smallpox by Kaplan et al. [30]), while $\lambda_L = 0.3821$ and $\lambda_G = 1.4159$ (as calculated by Ball and Lyne [7] for variola minor, a virus which causes a mild form of smallpox). The theoretical asymptotic extinction probabilities for the cases are (i) 0.7162, (ii) 0.5106 and (iii) 0.7444 (from simulations), while the values of R_* in the cases are (i) 1.9403, (ii) 1.0353 and (iii) 1.0740.

Figure 4.1 shows the final size outcomes for the local and global tracing model for $m = 100, 200, 300, 400, 500$. There are several different final size interpretations of the epidemic, we restrict attention to (i) the number of individuals who become infectious in the epidemic, (ii)the number of individuals infected in the epidemic (recall that an individual may be infected but not become infectious), and (iii) the number of households that are infected in the epidemic. We see the typical behaviour expected of final size outcomes, with a bimodal shape. Further, we can see the convergence of the distributions (to the theoretical approximating distribution), and that the different final size interpretations result in similarly shaped distributions.

From these simulations we may obtain estimates for p_G. In the branching process approximation, p_G is the probability of a global epidemic, i.e. the probability that infinitely many households are infected. With the simulations we see that the true behaviour of the epidemic is that generally either only a few households are infected, or a relatively large proportion is infected. The former case represents a *minor* epidemic, while the latter case represents a *major* epidemic (which is analogous to the global epidemic). So our estimate, $\hat{p}_G$, is the proportion of simulations that result in a major epidemic, and is obtained by examining the histogram of the number of households infected and determining the cut-off between minor and major epidemics by sight. In Figure 4.2 we plot these estimates (in relation to the asymptotic values) with confidence

intervals given as $\hat{p}_G \pm 2SE$ where the standard error is $SE = \left(\frac{(1-\hat{p}_G)\hat{p}_G}{n_0}\right)^{\frac{1}{2}}$.

It should be noted that there is a large degree of subjectivity in calculating the proportion of major epidemics in the local tracing only case for $m = 100$, in the local and global tracing case for m up to about 500 and the global tracing only case for m up to about 300. We see that the asymptotic probability is within the confidence interval consistently above $m = 500$ in the local tracing case and $m = 700$ in the global tracing case, but only above $m = 1000$ in the local and global tracing case. However, the slower convergence in the local and global tracing case may be partly due to the asymptotic value being 0.5106, i.e. an epidemic is almost as likely to go extinct as it is to blow up. Thus, Figure 4.2 also shows what happens when λ_G is increased to 1.8 for this case (and as a result the asymptotic probability of a global epidemic is 0.7205): the convergence is much quicker, with the asymptotic value being consistently within the confidence interval above $m = 400$. Overall though, even when convergence is relatively slow, the asymptotic value is still fairly close to the empirical value for smaller values of m.

4.5.2 Analysis of model assumptions

Figures 4.3-4.6 show R_* and p_G varying with p_c for differing intervention models. These include the subcases $p_I = 0$ and $p_I = 1$ of the model considered in this chapter, and for comparison, (i) isolation at the first removal in a household, without any vaccine (which is the best local intervention method whereby intervention is taken upon diagnoses); details for this can be found in Section 2.4.4, and (ii) the global tracing-only, vaccine-only policy as mentioned in Section 4.5.1, in which vaccination of a household is not triggered by the first removal in that household; here we again use single household epidemic simulations to obtain results, but we let p_c vary. Again, we use $n = 4$,

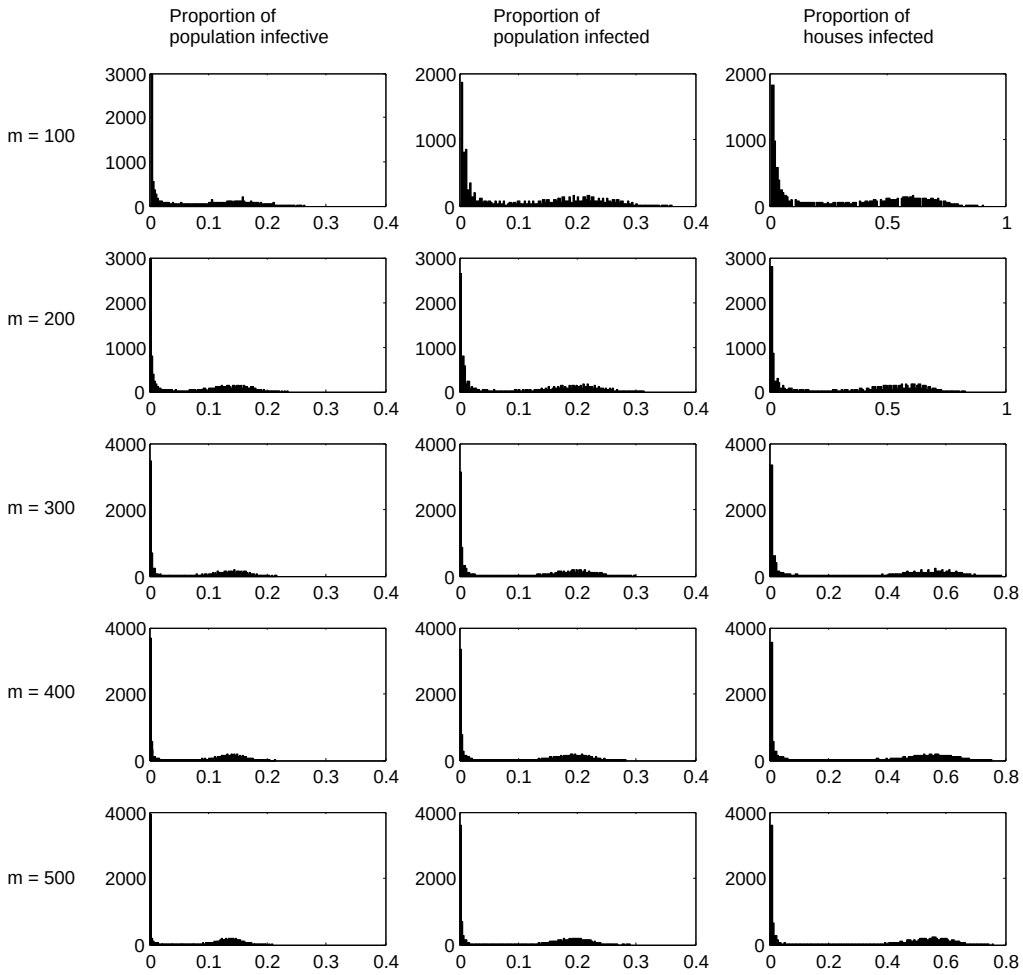

Figure 4.1: Final size outcome histograms for the local and global tracing model ($p_c = 1$, $p_I = 0$) for 10,000 simulations, when $n = 4$, $T_I \equiv \iota$, $T_D \sim \text{Exp}\left(\frac{1}{3}\right)$, $T_{L_1} \sim \text{Exp}(1)$, $T_{L_2} \sim \text{Exp}\left(\frac{3}{8}\right)$, $\lambda_L = 0.3821$ and $\lambda_G = 1.4159$. These give $R_* = 1.0353$.

$T_I \equiv 1$, $E[T_{L_1}] = 1$, $E[T_{L_2}] = \frac{8}{3}$, $\lambda_L = 0.3821$ and $\lambda_G = 1.4159$. In Figures 4.3 and 4.5 the latent periods are exponentially distributed, while in Figures 4.4 and 4.6 they are constant. In Figures 4.3 and 4.4 $E[T_D] = \frac{1}{3}$, while in Figures 4.5 and 4.6 $E[T_D] = 3$.

One thing to note, is that with constant latent periods of the lengths here, isolation has no effect on top of vaccination since, at the first removal, all other household members will be vaccine-sensitive (with probability 1). Further, if the delay is constant and

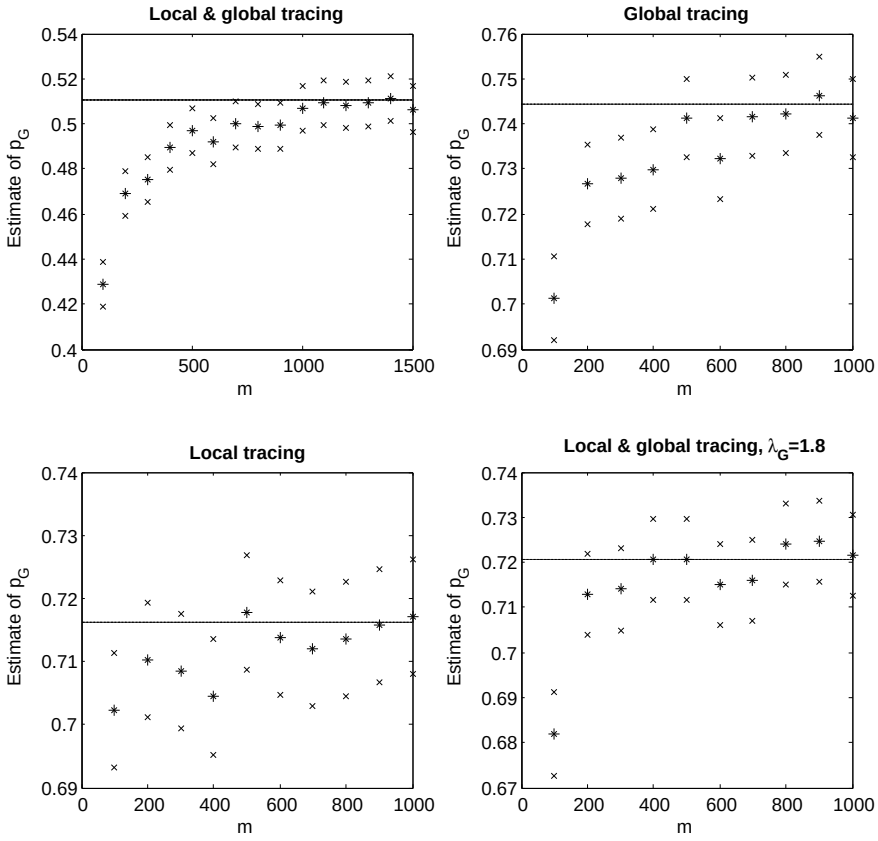

Figure 4.2: Estimates of global epidemic probabilities (represented by asterisks) from 10,000 simulations, when $n = 4$, $T_I \equiv \iota$, $T_D \sim \text{Exp}\left(\frac{1}{3}\right)$, $T_{L_1} \sim \text{Exp}(1)$, $T_{L_2} \sim \text{Exp}\left(\frac{3}{8}\right)$, $\lambda_L = 0.3821$ and $\lambda_G = 1.4159$ (unless stated otherwise). Crosses represent two standard errors above and below the estimate and solid lines represent the true asymptotic global epidemic probabilities.

sufficiently long (as in Figure 4.6), the global tracing has no effect on top of the local tracing, because the named individual will be vaccine-insensitive when the delay ends. These phenomena are not observed with exponentially distributed latent periods. Adding that the outbreak is reduced for constant latent periods (as to be expected; longer latent periods mean intervention is more likely to occur when more individuals are in vaccine-sensitive states), we can see that there is a clear qualitative difference for

different latent period distributions.

We can see that for the smaller delay mean, the difference between exponentially-distributed and constant delays is very slight, but it does become more noticeable for the larger delay mean, especially with constant latent periods as discussed above. The epidemic is 'worse' for a constant delay, as is expected.

The local and global tracing policies offer a decent improvement upon the global tracing-only policy, and, as p_c increases, quite quickly better the isolation at first removal without global tracing case for small delay mean but for a larger delay mean they cannot reduce R_* to below 1. With exponentially-distributed latent periods, isolation combines well with the vaccination to reduce R_* as much as is possible within this model.

In Figure 4.6 (i.e. constant latent periods with a delay mean of 3), with the global tracing-only policy R_* is higher for an exponentially-distributed delay than a constant one, even thought p_G is lower in the exponential case. This differs from the other cases in Figures 4.3-4.5, and is counterintuitive since usually we would expect the exponential distributed delay to result in lower R_* (an exponential distribution has a median below the mean, i.e. most of the distribution is below the mean). This model is not the focus of this chapter, but it would be remiss not to explain this phenomenon. Consider again our typical named household, with named contact B being the initial infective within the household, and time-zero being the time at which they become infectious. Consider first the constant delay case, in which case the vaccination will take place during B's vaccine-insensitive latent period, or during B's infectious period (before any other individual can be vaccine-insensitive), thus the final size of the named single household epidemic (number of individuals in the household who become infectious) must always be 1. However, if the delay is exponentially-distributed, the vaccination

can occur at any time - during B's vaccine-sensitive latent period, vaccine-insensitive latent period or infectious period, or long after these - and so the final size can be any value in $\{0, 1, 2, 3, 4\}$, and so for the value of λ_L, this must result in a mean final size of the named single household epidemic being greater than 1. However, the probability that the final size of the named single household epidemic is 0 must be sufficient that the full epidemic is more likely to die out quickly than for the constant delay case. (We are seeing that increased variance in offspring distribution results in a higher extinction probability for the branching process, a previously noted phenomenon, see for example Becker and Marschner [15] and Lloyd-Smith et al. [36].) It should be noted that this behaviour is parameter-dependent, we do not observe it when the delay mean is $\frac{1}{3}$, nor would we observe it when it is quite large (e.g. 20 here, so that a constant delay is long enough so as to render the vaccine inert). When we introduce the local tracing into the policy as well, the vaccination will occur in the exponential case at the latest at the first removal, when (since the vaccine-insensitive latent period equals the infectious period) B's housemates are all almost surely vaccine-insensitive. Thus, the final size is either 0 or 1, while in the constant delay case it is still always 1, and hence the epidemic with exponentially-distributed delays is statistically smaller than the epidemic with constant delays when we have the combined local and global tracing policy.

4.5.3 Controlling the epidemic: critical delay length

The model of this chapter is informed by a need to control epidemics, so it is of interest to know what efforts are needed to control under this model. In Figure 4.7 we again compare the local and global tracing policy of this chapter (two cases: $p_I = 0$ and $p_I = 1$) with the global tracing only, by showing how the critical delay mean (the delay mean which achieves $R_* = 1$) varies with p_c. Again $n = 4$, $\lambda_L = 0.3821$, $\lambda_G = 1.4159$,

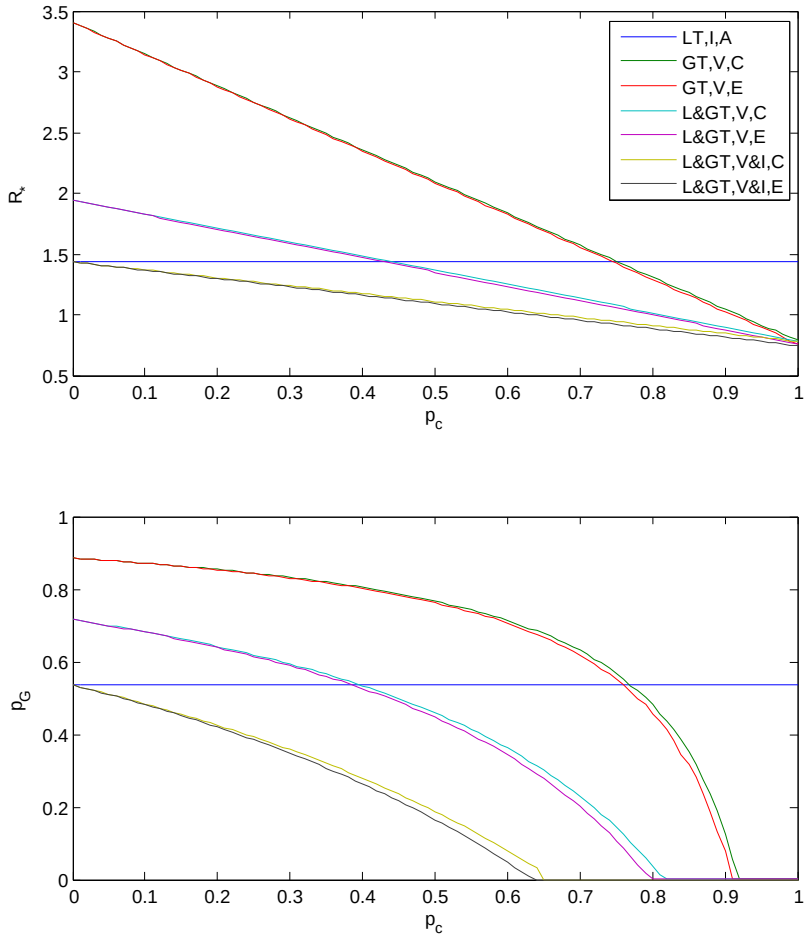

Figure 4.3: R_* and p_G varying with p_c when $n = 4$, $T_I \equiv 1$, $T_{L_1} \sim \text{Exp}(1)$, $T_{L_2} \sim \text{Exp}\left(\frac{3}{8}\right)$, $E[T_D] = \frac{1}{3}$, $\lambda_L = 0.3821$ and $\lambda_G = 1.4159$. Key to legend: LT=local tracing only, GT=global tracing only, L&GT=local and global tracing, V=vaccination only ($p_I = 0$), I=isolation only ($p_I = 1$), V&I=vaccination and isolation ($p_I = 1$), A=arbitrarily distributed delay, C=constant delay, E=exponentially-distributed delay.

$E[T_{L,1}] = 1$, $E[T_{L,2}] = \frac{8}{3}$ and $T_I \equiv 1$. Note that where the critical delay mean may appear to be zero, it is actually undefined because it is impossible to reduce R_* to or below 1.

We again see qualitative differences between latent period distributions: for constant latent periods, you can control the epidemic more easily. We also see that the difference

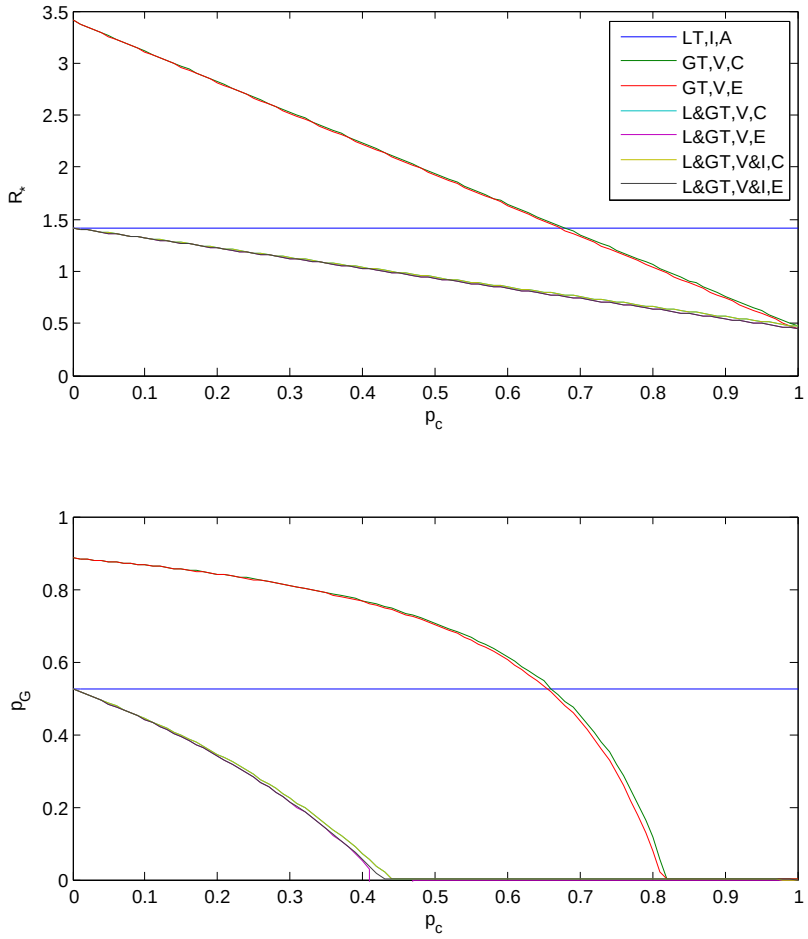

Figure 4.4: R_* and p_G varying with p_c when $n = 4$, $T_I \equiv 1$, $T_{L_1} \equiv 1$, $T_{L_2} \equiv \frac{8}{3}$, $E[T_D] = \frac{1}{3}$, $\lambda_L = 0.3821$ and $\lambda_G = 1.4159$. For key to legend see Fig. 4.3. Isolation has no effect on top of the vaccine.

between delay distributions is more pronounced in the constant latent period case.

It is clear that combining local tracing with global tracing can work to good effect. In this example, $\lambda_G = 1.4159$, and so local tracing alone would not be sufficient to reduce R_* below 1. With global tracing only we can control the epidemic but only for larger values of p_c, while a global and local tracing policy can control the epidemic even for values of p_c less than 0.5. Note though that the longest critical delay mean is around 1.35, which (noting the total latent period mean is $\frac{11}{3}$) suggests the crucial time

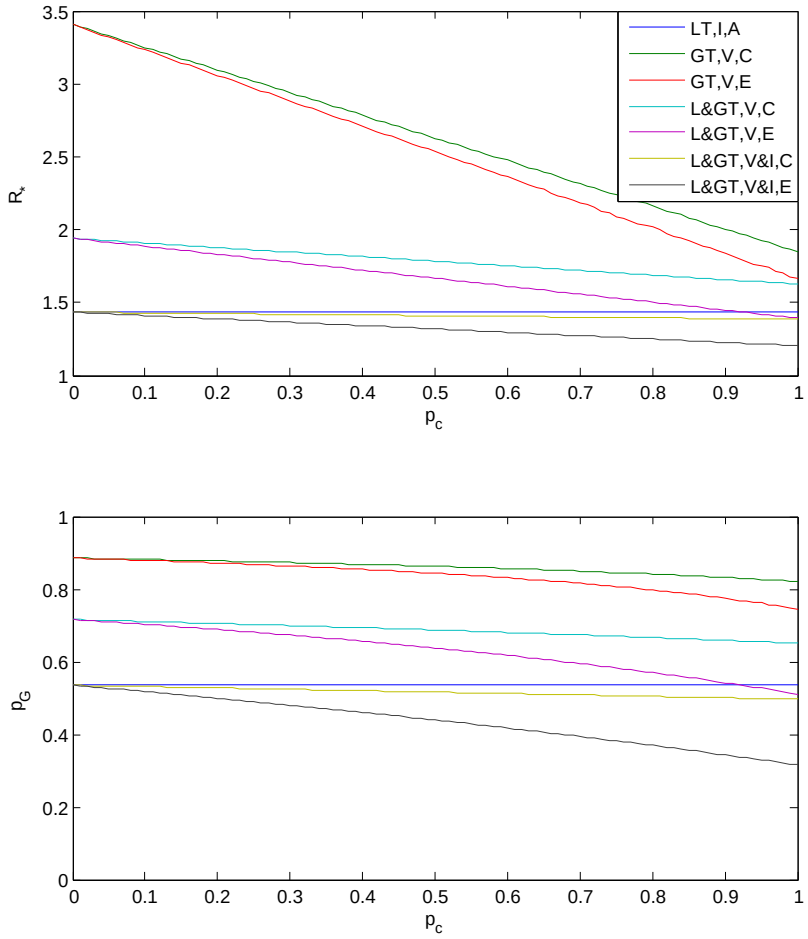

Figure 4.5: R_* and p_G varying with p_c when $n = 4$, $T_I \equiv 1$, $T_{L_1} \sim \text{Exp}(1)$, $T_{L_2} \sim \text{Exp}\left(\frac{3}{8}\right)$, $E[T_D] = 3$, $\lambda_L = 0.3821$ and $\lambda_G = 1.4159$. For key to legend see Fig. 4.3.

to vaccinate is before infection spreads within the household, as then at most only one individual becomes infectious in the household.

4.6 Concluding comments

In this chapter, expressions for R_* and p_G have been obtained for a model with intervention that works at both a local (vaccination/isolation at first removal in a household) and a global (contact tracing leading to vaccination of households) level. It has

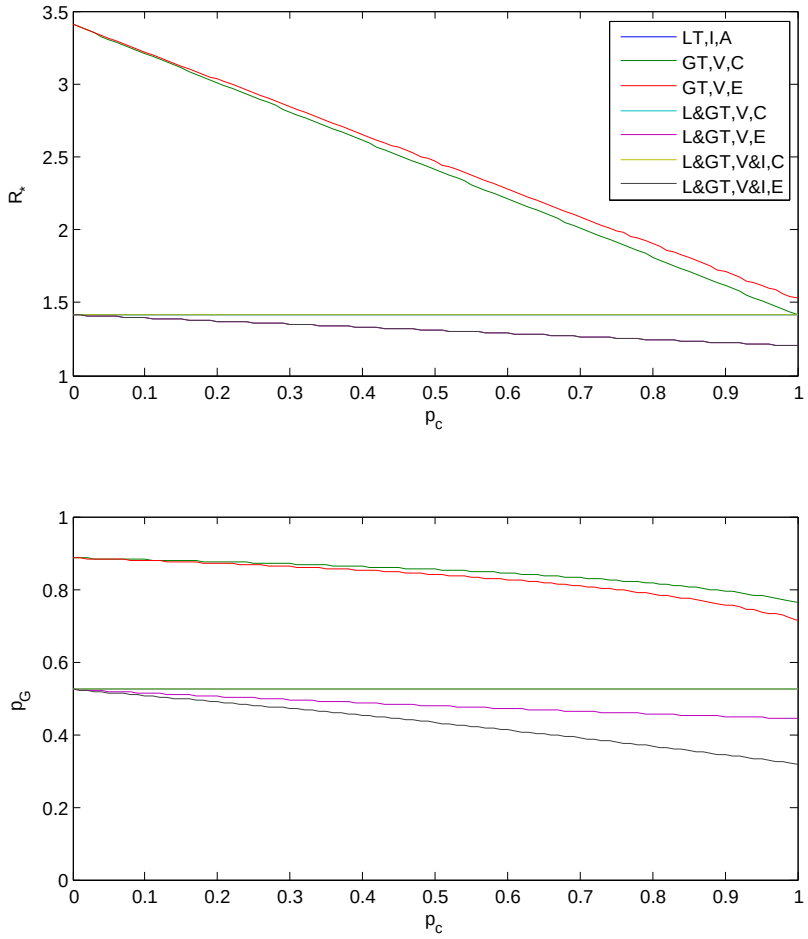

Figure 4.6: R_* and p_G varying with p_c when $n = 4$, $T_I \equiv 1$, $T_{L_1} \equiv 1$, $T_{L_2} \equiv \frac{8}{3}$, $E[T_D] = 3$, $\lambda_L = 0.3821$ and $\lambda_G = 1.4159$. For key to legend see Fig. 4.3. The cases with constant delay and both local and global tracing match up with the local tracing-only isolation case here. Isolation has no effect on top of the vaccine.

been shown that this combined policy can have a great effect in reducing these such values, when compared to related local tracing- or global tracing-only policies. Different distribution choices for the latent periods and the delays were considered. The material differences between constant and exponential choices for these vary under other assumptions, for instance as delay mean and the naming probability increase, the effect of the choice of delay distribution becomes greater. Simulations of finite-size

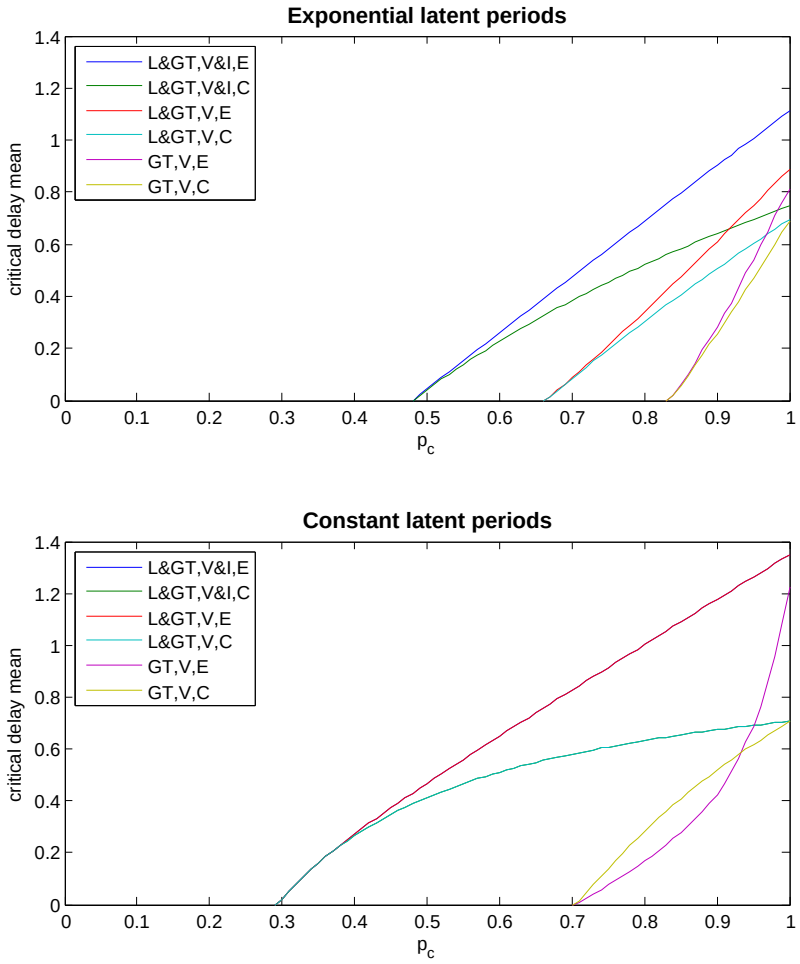

Figure 4.7: Critical delay means (i.e. that result in $R_* = 1$) varying with p_c when $n = 4$, $\lambda_L = 0.3821$, $\lambda_G = 1.4159$, $E[T_{L,1}] = 1$, $E[T_{L,2}] = \frac{8}{3}$ and $T_I \equiv 1$. For key to legend see Fig. 4.3. In the constant latent periods case isolation has no effect on top of the vaccine.

epidemics showed that the approximating assumptions made to obtain R_* and p_G do not lead to unreliable results.

It may be possible to extend the model from a constant infectious period to one with finite support i.e. $\mathrm{P}(T_I = \iota_k) = p_k$ for $k = 1, 2, \ldots, K$ such that $\sum_{k=1}^{K} p_k = 1$. In this case, to preserve a multitype branching process framework, one would have to replace the solitary named households type with K types, where the kth type represents a

household into which infection was introduced by global contact with an individual with infectious period of length ι_k. However, the single household epidemic becomes more complicated, since now the time of the first removal within the household is not fixed. To preserve some of the analytical properties we have used, one could assume that all housemates have the same infectious period length (or at least that no-one in a household has an infectious period shorter than the initially-infected individual within that household), though in practice this would be a fairly unrealistic restriction.

In this model only a fixed naming probability has been considered. The theory of this chapter can be extended to some random naming probability models. For instance, suppose the probability of an individual (who can name) naming each of their global contacts (with the same probability) is distributed according to a random variable P_c, with finite support, i.e. $P\left(P_c = p_{c,j}\right) = \pi_j$ for $i = j, 2, \ldots, J$ such that $0 \leq p_{c,1} < p_{c,2} < \ldots < p_{c,J} \leq 1$ and $\sum_{j=1}^{J} \pi_j = 1$, then Eqns. (4.2.1) and (4.2.2) become

$$M_{i0} = \lambda_G \left(\left(1 - \mathrm{E}\left[P_c\right]\right) \mathrm{E}\left[X_i^+\right] + \mathrm{E}\left[X_i^-\right] \right),$$

$$M_{i1} = \lambda_G \mathrm{E}\left[P_c\right] \mathrm{E}\left[X_i^+\right],$$

while Eqn. (4.2.3) becomes

$$f_i\left(s_0, s_1\right) = \sum_{j=1}^{J} \pi_j \psi_i \left(\lambda_G \left\{\left(1 - p_{c,j}\right)\left(1 - s_0\right) + p_{c,j}\left(1 - s_1\right)\right\}, \lambda_G\left(1 - s_0\right)\right).$$

It may also be beneficial to study other, more complicated naming probability models, in which the naming probability may be time-dependent or depend upon the number of contacts made.

The contact tracing in this model is 'forward' in that infectees are named by infectors (but not vice versa). If we assumed that 'backward' tracing were also possible (i.e. infectors may be named by infectees), then all results in this chapter would be unchanged

because a constant infectious period results in an infectee's infectious period always ending after their infector's, by which time the infector's household will have already been vaccinated. However, including backwards tracing would have an effect if the infectious period were not fixed.

The model in this chapter has been restricted to households of equal size, but the theory can be extended to households of unequal size in a similar manner to that mentioned in Section 2.8.

Attention has also been restricted to a perfect vaccine, i.e the vaccine renders all vaccine-sensitive individuals immune. Other vaccine models could be considered: vaccines that reduce susceptibility and infectivity of individuals, vaccines with random effects, second applications of vaccines, the effects of vaccination being different for susceptible and vaccine-sensitive latent individuals, or a combination of these and other assumptions.

CHAPTER 5

Conclusions and extensions

5.1 Conclusions

Three models for real-time, responsive intervention applied after the start of an epidemic have been considered.

In Chapter 2 an intervention scheme (incorporating vaccination and isolation) targeting housemates of diagnosed individuals was modelled, and it was seen how different assumptions about the vaccine action and distributions for latent and infectious periods may or may not affect conclusions. It was seen that while this type of intervention can be effective in reducing the spread of an epidemic, the reduction may be insufficient to prevent a major outbreak if the between-household contact rate is large enough.

Chapter 3 looked at a contact tracing model for a homogeneously-mixing population wherein traced individuals are isolated, considering that traced individuals may (iterative tracing) or may not (single-step tracing) be asked to name their own contacts. As the naming probability increased their was a more pronounced difference between the two subcases, there was also a greater difference in results between distribution choices for the tracing delay and latent and infectious periods. It was seen that whether

an individual's traced contacts experienced the same or independent (and identically distributed) delays did not have much of an impact on conclusions.

The model of Chapter 4 combined a local-tracing intervention scheme (as in Chapter 2) with an additional vaccination triggered by tracing between-household contacts, and vaccinating the traced individual's household. This combination of contact tracing with local-level intervention was seen to be effective, in concurrence with findings by Becker et al. [18] and Eichner [22]. However, in their models, intervention is directed towards only the traced individual themselves, and not their housemates or local contacts. By vaccinating the household of a traced individual in the model of Chapter 4, the effect of tracing is amplified. For tractability, it was assumed that the infectious period is of a fixed length. However, different distributions for the tracing delay and latent periods were considered.

Kaplan et al. [30] modelled a traced vaccination scheme and noted there was a 'race to trace' - if a traced individual is vaccinated after they have become vaccine-insensitive, the act of tracing and vaccinating that individual has been a waste. However, in the models of Chapters 3 and 4, the urgency of the 'race-to-trace' is reduced: in the former, we isolate traced individuals, and so tracing an infectious individual reduces their number of contacts; in the latter, we vaccinate the traced individual's household, and so while tracing a vaccine-insensitive individual may result in the waste of a vaccination, their housemates may be vaccine-sensitive, and so the act of tracing this individual is not a waste.

5.2 Extensions

The targeted intervention schemes of Chapters 2 and 4 exploited the household structure of the model. It may be interesting to extend this into a model with more levels of mixing, by assuming that intervention is directed towards not only the housemates of a diagnosed or traced individual, but also their neighbours, schoolmates or workmates. However, given that contact tracing and more sophisticated population structures tend to reduce mathematical tractability, it may be difficult to make analytical progress.

For tractability, it was assumed in Chapter 4 that the infectious period is of a fixed length. In real life the validity of this assumption will depend upon the disease. Further, it was also assumed that only an infected individual who experiences their full infectious period in the at-risk population may be allowed to name their contacts. To relax these assumptions, it may be possible to apply some of the methodology of Chapter 3 to the household environment, by considering the embedded process of unnamed households. However, this would be much more complicated analytically, due to the increased variability of the outcome of a named household compared to that of a named individual, but it might also enable us to consider whether there is little difference in results between assuming sibling units experience the same or independent delays (as was the case in Chapter 3).

Throughout, the intervention models have implicitly assumed that all infected individuals become symptomatic, since treatment is given to contacts or housemates of an infected individual after this individual has shown symptoms. It may be useful to incorporate asymptomatic carriers, who spread the disease without being detected, and thus never trigger intervention. It would be interesting to see how this affects the effectiveness (in reducing spread) of the intervention methods, and, particularly in the

household cases, how this affects the quality of the branching process approximations used in this thesis.

It is also assumed that individuals are removed from the at-risk population when their infectious period ends, and since this triggers intervention towards their contacts or housemates, the end of their infectious period is when they are detected. It may be of interest to consider a separate detection time beginning at infection. This would allow display of symptoms to be independent of infectivity. It may also be a way of incorporating asymptomatic carriers by assuming that 'detecting' an individual after their infectious period ends does not result in targeting intervention towards their contacts or housemates.

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