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Nyimvua Shaban, Mikael Andersson, Åke Svensson and Tom Britton

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Postal address:

Mathematical Statistics
Dept. of Mathematics
Stockholm University
SE-106 91 Stockholm
Sweden

Internet:

http://www.math.su.se/matstat

Network epidemics and early stage vaccination: the effects of infectious and vaccination delay periods and their randomness

Nyimvua Shaban[†] *, Mikael Andersson [†], Åke Svensson[†] , Tom Britton[†]

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Abstract

It is known that the distributions of the latent and infectious periods affect the dynamics of the spread of an infectious disease. Here we consider the SEIR epidemic model describing the spread of an infectious disease giving life-long immunity in a community whose social structure can be represented by a simple random graph having a pre-specified degree distribution. Two real time vaccination strategies, based on tracing and vaccinating the friends of infectious individuals during the early stages of an epidemic, are proposed. The first strategy considers vaccination of each friend of a detected infectious individual independently with probability ρ. The second strategy sets an upper bound on the number of friends an individual can infect before being detected. We derive both the basic reproduction number and the strategy-specific reproduction numbers and show that these reproduction numbers decrease when the variances of the infectious period and the time to detection increase. Under the assumption that detection may only occur after the latent period, the reproduction numbers are independent of the distribution of the latent period.

Key words: branching approximation, coefficient of variation, degree distribution, epidemic models, social networks, vaccination strategies.

1 Introduction

In network theory, the contact structure among individuals in a social community can be represented by a simple undirected random graph (see e.g. Scott 2000, Newman 2003), where vertices correspond to individuals and edges to some type of social relations, here referred to as friendships. A model for the spread of an infectious disease may be defined

*Department of Mathematics, University of Dar es Salaam, Postal address: Box 35062, Dar es Salaam, Tanzania. *E-mail*: shabanmbare@yahoo.com

[†]Department of Mathematics, Stockholm University, SE-106 91 Stockholm, Sweden.

on such a social graph, where individuals are at first susceptible but may be infected by a friend. In our model, the infection is started by one randomly chosen individual who is infected from outside the community. The final outbreak, both its size and who gets infected, depends on the properties of the social graph as well as on the properties of the disease transmission. For instance, the dynamics of the disease is affected by the distributions of the latent and infectious periods (see e.g. Wearing *et al.* 2005, Lloyd 2001). It is possible to prevent an outbreak by vaccinating friends of infected individuals during the early stages of the epidemic. Vaccination may be delayed for some time (here referred to as delay time), because we assume that an infectious individual can only be detected after showing symptoms.

The present paper investigates what effects the distributions of the latent, infectious and delay periods have on the potential of a large disease outbreak. In particular, we consider a random social graph of a closed community, where the degree distribution (number of friends) follows some pre-specified distribution F, typically having heavy tails, but where the random graph is otherwise chosen randomly. The epidemic model considered here is a model for the susceptible-exposed-infectious-removed (SEIR) disease. Initially, one randomly selected individual is externally infected. This individual is first latent, after which she becomes infectious and may infect her susceptible friends before she recovers and become immune, a state called removed. Friends of such a person or any other individual who become infected behave similarly.

SEIR epidemic in a homogeneously mixing community have been extensively studied (e.g. Bailey 1975, Diekmann and Heesterbeek 2000, Hethcote 2000) under the assumption that the latent (when considered) and infectious periods are exponentially distributed. The exponential distribution is mathematically convenient, but it corresponds to the assumption that the chance of being removed in a given time interval is independent of the time since infection. It is argued (e.g. Gough 1977, Lloyd 2001), that in reality the chance of recovery (in a given time interval) is initially small but increases overtime. This indicates that the infectious period distribution is less dispersed and more closely centred around its mean than the exponential distribution. This can be interpreted that the exponential distribution overestimates the number of individuals whose duration of infection is much shorter or much longer than the mean. Below we study how the distributional effects (other than exponential distribution) of the infectious period and vaccination delay time influence the reproduction numbers.

For the above social graph and epidemic model we study two vaccination strategies: vaccination of located friends after delay (the first strategy) and vaccination of friends after delay with a bound on the maximum number of possible infections (the second strategy). Both vaccination strategies are implemented during the early stages of the epidemic after tracing the friends of infectious individuals in order to uncover possible chains of infection. Shaban *et al.,* (2007) studied these vaccination schemes, but then assuming no latent period and exponentially distributed infectious periods and detection times. Here we

model the latent period with an arbitrary distribution, and infectious period and delay time with independent gamma distributions. We investigate how the random properties of latent and infectious periods as well as the properties of the vaccination schemes affect the reproduction numbers.

The transmission potential of an infectious disease can be quantified by the basic reproduction number often denoted R_0, which determines whether a major outbreak can occur or not: if $R_0 \leq 1$, only minor outbreaks can occur, whereas if $R_0 > 1$ then there is a positive probability for a major outbreak, (e.g see p.6 in Andersson and Britton, 2000). For a closed large population of size n we derive R_0 for the network epidemic model without vaccination and show that, for our model, the latent period does not affect R_0 and hence plays no role on the distribution of the final size. We also derive the strategy-specific reproduction numbers for the two vaccination schemes, and show that the distributions of the infectious period and delay time affect the dynamics of the disease transmission (and hence the reproduction numbers). The reproduction numbers are independent of the distribution of the latent period, since we assume that an infected individual can only be detected while infectious and not during latent period. All this is done while assuming independent gamma distributions for infectious periods and delay times.

The rest of the paper is organized as follows. In Section 2 we define the models for the social graph, the epidemic on the social graph and we derive the basic reproduction number. In Section 3 we present our two vaccination strategies and derive the corresponding strategy-specific reproduction numbers. The effects of the random properties of the infectious periods and delay time on the reproduction numbers are also investigated. Discussion of the results as well as some concluding remarks are treated in Section 4.

2 Models

2.1 Social structure

Let n denote the number of vertices (i.e. the population size) of a random graph, not allowing for multiple edges and loops (i.e. a simple random graph). We describe the social network by a simple undirected random graph where the degree distribution follows some pre-specified distribution $F = \{p_k\}_{k=0}^{\infty}$. That is, we are given the probabilities p_k that a randomly chosen vertex from the network has degree $D = k$. We define a simple random graph with a given degree sequence $\{D_i\}_{i=1}^{n}$ by the configuration model (see e.g. Bollobás 2001): assign independent degrees $D_1, \ldots, D_n$ from F to the vertices and give a vertex with degree k, k stubs. Then join the stubs randomly pairwise to form edges between them. That is, first pick two stubs randomly among all stubs in the graph and join them. Then pick two stubs at random from the remaining stubs and join them, and so on (see, e.g. Molloy and Reed, 1995). Vertices i and j are neighbours (friends) if there is an edge between them. For details on how to generate simple undirected random graphs we refer the reader to Britton *et al.* (2006) and references therein. This procedure produces a

graph with the desired degree distribution as $n \to \infty$, but which in all other respects is completely random. That is, we generate a graph which is drawn uniformly at random from the set of graphs with the given distribution.

2.2 Epidemic model

Consider an infectious disease that spreads along the edges of a graph defined above. The vertices of a graph are the individuals in the community and the edges represent friendships through which the disease may spread. We now define the course of the disease in the following way. Initially, one randomly selected individual (vertex) is infected from the outside. This individual then is first latent for a random time with arbitrary distribution, after which she becomes infectious and remains so for a period I being $\Gamma(n_I, n_I\gamma)$-distributed. During the infectious period an individual infects each of her friends independently according to independent Poisson processes with the rate λ. Those who get infected make out the first generation infected in the epidemic. These individuals then behave similarly and may infect their not yet infected friends thus making a second infected generation, and so on. An individual cannot be re-infected after her infectious period, so she is considered recovered and immune from the disease. This epidemic continues until there are no more latent or infectious individuals present when it stops, because then all individuals are either still susceptible or immune and no one is infectious or latent. It follows from the above distribution of L that the average length of latent period is $1/\delta$, the standard deviation is $1/(\delta\sqrt{n_L})$ and the coefficient of variation is $1/\sqrt{n_L}$. Similarly, the mean length of the infectious period is $1/\gamma$ with standard deviation $1/(\gamma\sqrt{n_I})$ and the coefficient of variation $1/\sqrt{n_I}$. As usual, we assume that all Poisson processes describing infectious contacts, as well as latent and infectious periods, are mutually independent.

2.3 Basic reproduction number

We now derive the basic reproduction number for the epidemic model defined above. To do this, we begin by deriving the probability for an individual to infect her friends before recovery. If initially the number of infectious individual is small, the growth of an epidemic can be approximated by a suitable branching process. This approximation can be made more precise by coupling arguments (see e.g. Ball 1996), but this will not be treated in the present paper.

Let T_i denote the time an individual makes the first contact with a given friend i during her infectious period, and let I be the the length of the infectious period. Note that contacts made by an individual while latent have no effect in the progress of infections, while contacts made when infectious result into infection. It follows that the probability of transmission of infection is given by

$$P(T_i < I) = \int_0^\infty P(T_i < I | T_i = t)\lambda e^{-\lambda t} dt = \int_0^\infty (1 - P(I \leq t))\lambda e^{-\lambda t} dt. \tag{2.1}$$

Since $I \sim \Gamma(n_I, n_I\gamma)$ with n_I integer, the distribution function of the infectious period I is

$$F_I(t) = 1 - \sum_{j=0}^{n_I-1} \frac{(n_I\gamma t)^j}{j!}\, e^{-n_I\gamma t}. \tag{2.2}$$

A related expression to Equation (2.2) is obtained in Bain and Engelhardt (1991) (see page 113). As a consequence, Equation (2.1) becomes

$$\begin{aligned} P(T_i < I) &= \lambda \sum_{j=0}^{n_I-1} \frac{(n_I\gamma)^j}{j!} \int_0^\infty t^j e^{-(n_I\gamma+\lambda)t} dt \\ &= 1 - \left(\frac{n_I\gamma}{\lambda + n_I\gamma}\right)^{n_I}, \end{aligned} \tag{2.3}$$

since

$$\int_0^\infty t^j e^{-(n_I\gamma+\lambda)t} dt = \frac{j!}{(\lambda + n_I\gamma)^{j+1}}.$$

The initial infectious individual has the degree distribution $\{p_k\}_{k=0}^\infty$. The friends of this individual (or friends of any other individual) have the size biased degree distribution $\{\tilde{p}_k\}_{k=0}^\infty$, where $\tilde{p}_k = \frac{kp_k}{E(D)}$, and $E(D) = \sum_j jp_j$ is the average degree of an individual. During the early stages and conditioning on the degree k, an individual in the second generation will on average infect $(k-1)P(T_i < I)$ new cases, because all individuals except the one she was infected by are susceptible. As a consequence, the basic reproduction number R_0 becomes

$$\begin{aligned} R_0 &= \Big[1 - \Big(\frac{n_I\gamma}{\lambda + n_I\gamma}\Big)^{n_I}\Big] \sum_k (k-1)\frac{kp_k}{E(D)} \\ &= \Big[1 - \Big(\frac{n_I\gamma}{\lambda + n_I\gamma}\Big)^{n_I}\Big] \Big(E(D) + \frac{V(D) - E(D)}{E(D)}\Big), \end{aligned} \tag{2.4}$$

where $V(D) = \sum_j j^2 p_j - (E(D))^2$ is the variance in the degree distribution. If $n_I = 1$ (see e.g. Andersson 1999) Equation (2.4) becomes

$$R_0 = \frac{\lambda}{\lambda + \gamma}\Big(E(D) + \frac{V(D) - E(D)}{E(D)}\Big).$$

We should comment here that the extinction probability (probability of minor outbreak in epidemic) can be calculated by using a suitable branching process approximation (see e.g. Jagers 1975). Note that the branching process is subcritical, critical or supercritical depending on whether $R_0 < 1$, $R_0 = 1$ or $R_0 > 1$. In terms of the epidemic, this means that a major outbreak is possible if and only if $R_0 > 1$.

We conclude from (2.4) that the latent period L plays no role on R_0, following the assumption that the detection of an infected person may only occur during her infectious period. We also see in (2.4) that R_0 is increasing in $V(D)$, so the more variance in the

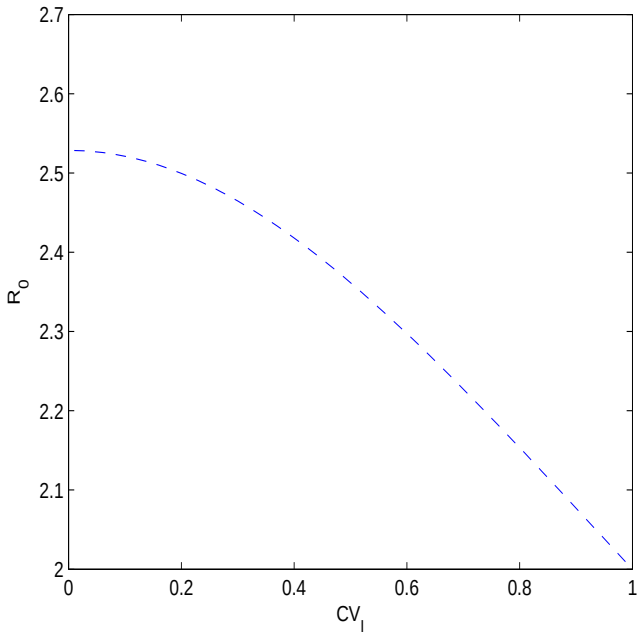

Figure 1: Basic reproduction number R_0 as a function of the coefficient of variation of the infectious period CV_I.

degree distribution, the higher R_0, and if the degree distribution has infinite variance then $R_0 = +\infty$, a case which we do not pursue in this paper. We now look into the effects of the variation in the distribution of the infectious period on the basic reproduction number (i.e. how n_I affects R_0). In Figure 1 we plot R_0, as a function of $CV_I (= 1/\sqrt{n_I})$, the coefficient of variation of the infectious period. It is seen that R_0 is decreasing with increase in CV_I, showing that the variation in the distribution of the infectious periods affect the dynamics of the disease.

Furthermore, if $n_I = +\infty$ corresponding to having a constant infectious period, then (2.4) becomes

$$R_0(n_I = +\infty) = (1 - e^{-\frac{\lambda}{\gamma}})\Big(E(D) + \frac{V(D) - E(D)}{E(D)}\Big).$$

Likewise, for the case when $n_I = 1$, corresponding to infectious period being exponential distributed with parameter γ, (2.4) becomes

$$R_0(n_I = 1) = \frac{\lambda}{\lambda + \gamma}\Big(E(D) + \frac{V(D) - E(D)}{E(D)}\Big).$$

Taking the ratio of $R_0(n_I = +\infty)$ to $R_0(n_I = 1)$ (i.e. the extreme cases) yields a function of $\frac{\lambda}{\gamma}$ (the mean number of infectious contacts with a given individual during infectious period) given by

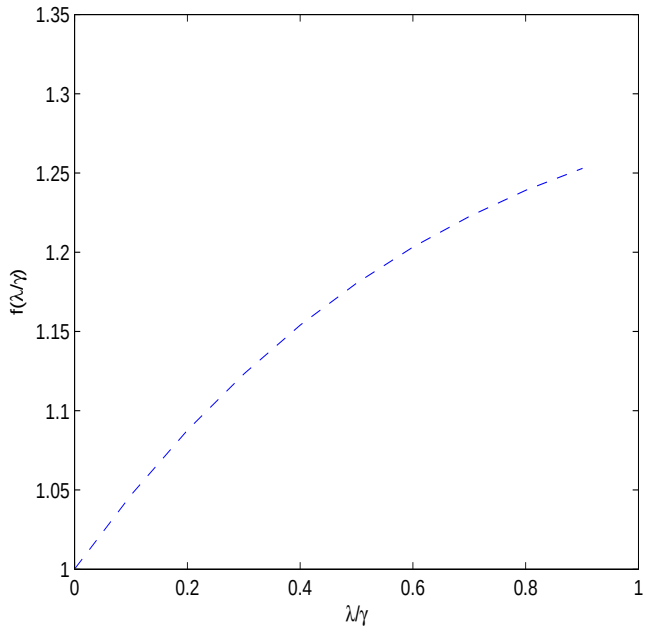

Figure 2: The ratio of R_0 at $n_I = 1$ and $n_I = +\infty$ (i.e. $f\left(\frac{\lambda}{\gamma}\right)$ as a function of $\frac{\lambda}{\gamma}$), the mean of infectious contacts during infectious period.

$$\frac{R_0(n_I = \infty)}{R_0(n_I = 1)} = f\left(\frac{\lambda}{\gamma}\right) = \left(1 + \frac{\gamma}{\lambda}\right)\left(1 - e^{-\frac{\lambda}{\gamma}}\right).$$

In Figure 2, this function f is illustrated and it is seen that $f(\frac{\lambda}{\gamma})$ is increasing in $\frac{\lambda}{\gamma}$. The interpretation is that there is biggest difference between constant and exponentially distributed infectious periods when R_0 is large. It is also observed that $f(\frac{\lambda}{\gamma}) \geq 1$, implying that constant infectious period always make R_0 larger.

3 Vaccination strategies

Assume now that a perfect vaccine is available. By this we mean that a susceptible individual who is vaccinated is completely protected (that is immune to) from the disease and is unable to spread the disease. Below we present two vaccination strategies which are implemented during the early stages of the epidemic after detecting infectious individuals. That is, an infected individual can only be detected while infectious (after latency period) and we assume that all located friends of a detected infectious individual are vaccinated, but that vaccination has no effect on already infected individuals. Below we derive the strategy-specific reproduction numbers for the two strategies.

3.1 Vaccination of located friends after delay

Assume that an infected individual is never detected before showing symptoms. The time between the point of infection and the point at which an individual shows symptoms is called incubation period. We assume that the incubation period is approximately equal to the latent period. Once the person starts showing symptoms (hence also becomes infectious), she may be detected by the authorities, and then friends of this individual are located and become vaccinated. The time starting from when the person becomes infectious until she is detected and friends are located and vaccinated is referred to as the delay time. For simplicity, we assume that all located friends are vaccinated at the same time.

More precisely, this vaccination strategy goes as follows. An infectious individual is detected and her friends get located after some delay time S say, having $\Gamma(n_S, n_S\theta)$-distribution. Each friend of this individual is located independently with probability ρ and all located friends are vaccinated without further delay. It follows from the distribution of S that the mean length of delay time is $1/\theta$, the standard deviation is $1/(\theta\sqrt{n_S})$ and the coefficient of variation is $1/\sqrt{n_S}$.

We now derive the reproduction number in order to determine the performance of this vaccination strategy. Let X_i be an indicator variable such that $X_i = 1$ if a given friend i of an infectious individual is located (which happens with probability ρ), and $X_i = 0$ otherwise. Suppose T_i is the time an infectious individual first contacts friend i. Then transmission of infection occurs if $T_i < \min(S, I)$, or if $S < T_i < I$ and $\{X_i = 0\}$. Let p denote the probability that an infected person infects a given susceptible friend i. By conditioning on T_i, p can be derived as

$$\begin{aligned} p &= P(T_i < \min(S, I)) + P(S < T_i < I \cap \{X_i = 0\}) \\ &= \int_0^\infty \Big(P\big(T_i < \min(S, I) | T_i = t\big) + (1-\rho) P\big(S < T_i < I | T_i = t\big) \Big) \lambda e^{-\lambda t} dt. \end{aligned} \tag{3.1}$$

Since I and S are independent, and T_i is Exp(λ), Equation (3.1) hence becomes

$$p = \int_0^\infty P(I > t) P(S > t) \lambda e^{-\lambda t} dt + (1-\rho) \int_0^\infty P(S \leq t) P(I > t) \lambda e^{-\lambda t} dt. \tag{3.2}$$

Using the gamma distributions for S and I in Equation (3.1) we get that (see e.g. Bain and Engelhardit 1991, page 113),

$$\begin{aligned} p = &\int_0^\infty \sum_{j=0}^{n_I - 1} \frac{(n_I \gamma t)^j}{j!} \, e^{-n_I \gamma t} \sum_{i=0}^{n_S - 1} \frac{(n_S \theta t)^i}{i!} \, e^{-n_S \theta t} \, \lambda e^{-\lambda t} dt \\ &+ (1-\rho) \int_0^\infty \sum_{j=0}^{n_I - 1} \frac{(n_I \gamma t)^j}{j!} \, e^{-n_I \gamma t} \left(1 - \sum_{i=0}^{n_S - 1} \frac{(n_S \theta t)^i}{i!} \, e^{-n_S \theta t} \right) \lambda e^{-\lambda t} dt. \end{aligned} \tag{3.3}$$

Interchanging integration and summation and performing some algebra, (3.3) simplifies to the following expression

$$p = (1-\rho)\left[1 - \Big(\frac{n_I\gamma}{\lambda + n_I\gamma}\Big)^{n_I}\right] + \frac{\rho\lambda}{\lambda + n_I\gamma + n_S\theta}\ \xi, \tag{3.4}$$

where

$$\xi = \sum_{i=0}^{n_S-1}\sum_{j=0}^{n_I-1}\binom{i+j}{i}\Big(\frac{n_S\theta}{\lambda + n_I\gamma + n_S\theta}\Big)^{i}\Big(\frac{n_I\gamma}{\lambda + n_I\gamma + n_S\theta}\Big)^{j}. \tag{3.5}$$

Applying some combinatorial techniques in (3.5), then (3.4) becomes

$$p = (1-\rho)\left[1 - \Big(\frac{n_I\gamma}{\lambda + n_I\gamma}\Big)^{n_I}\right] + \rho\left[1 - \Big(\frac{n_I\gamma + n_S\theta}{\lambda + n_I\gamma + n_S\theta}\Big)^{n_I+n_s-1}\right]. \tag{3.6}$$

During the early stages of the epidemic, individuals in the second and later generation have degree k with probability $\tilde{p_k} = \frac{kp_k}{E(D)}$, and of these $(k-1)$ are susceptible. The reproduction number hence becomes (using equation (2.4))

$$\begin{aligned} R_{\theta,\rho} &= p\sum_k (k-1)\frac{kp_k}{E(D)} \\ &= \left[(1-\rho) + \rho\left(1 - \Big(\frac{n_I\gamma}{\lambda + n_I\gamma}\Big)^{n_I}\right)^{-1} \cdot \left(1 - \Big(\frac{n_I\gamma + n_S\theta}{\lambda + n_I\gamma + n_S\theta}\Big)^{n_I+n_s-1}\right)\right] R_0. \end{aligned} \tag{3.7}$$

We note that $R_{\theta,\rho}$ in (3.7) is linear in R_0, thus if the basic reproduction number is large so is $R_{\theta,\rho}$. The disease will surely be contained if and only if $R_{\theta,\rho} \leq 1$. In Figure 3 we see that $R_{\theta,\rho}$ is decreasing with θ, though it does not necessarily prevent the disease from taking off.

We now investigate how $R_{\theta,\rho}$ is influenced by the variation in the distributions of the infectious period and delay time (i.e. how n_I and n_S affect $R_{\theta,\rho}$). Of particular interest is when $n_I = n_S = 1$ and when both n_I and n_S are tending to infinity, corresponding to exponential distributed and constant periods respectively. Using the pair of values $(n_I = 1, n_S)$ and $(n_I = +\infty, n_S)$ in (3.7) we obtain respectively

$$R_{\theta,\rho}(n_I = 1, n_S) = \left[(1-\rho) + \rho(1 + \frac{\gamma}{\lambda})\left(1 - \Big(\frac{\gamma + n_S\theta}{\lambda + \gamma + n_S\theta}\Big)^{n_S}\right)\right] R_0 \tag{3.8}$$

and

$$R_{\theta,\rho}(n_I = +\infty, n_S) = \left[(1-\rho) + \rho\Big(1 - e^{-\frac{\lambda}{\gamma}}\Big)^{-1}\Big(1 - e^{-(\frac{\lambda}{\gamma} + n_S\frac{\theta}{\gamma})}\Big)\right] R_0. \tag{3.9}$$

As an example, we choose $\lambda = \gamma = 1$, $\rho = 0.5$, $\theta = 1$, $R_0 = 2$ and using Equations (3.8) and (3.9), it is seen that both $R_{\theta,\rho}(n_I = 1, n_S)$ and $R_{\theta,\rho}(n_I = +\infty, n_S)$ are increasing

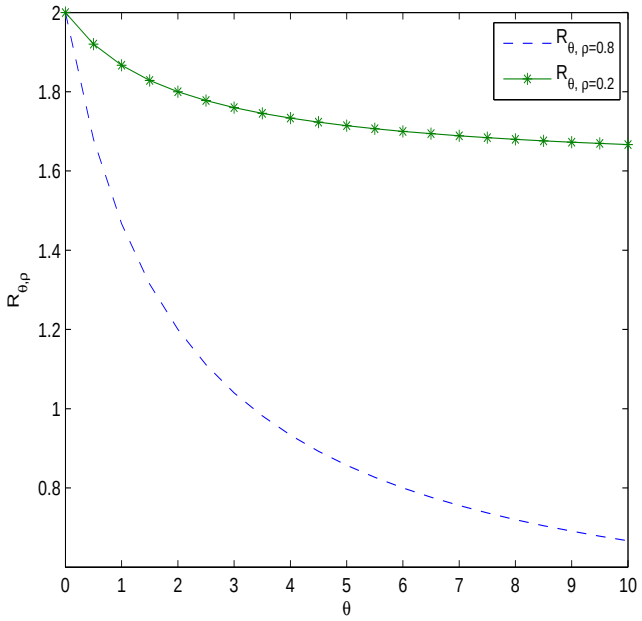

Figure 3: Reproduction number $R_{\theta,\rho}$, as a function of θ, the detection rate when $R_0 = 2$, and $\lambda = \gamma = 1$, for $\rho = 0.2$ and $\rho = 0.8$.

with n_S (see Figure 4). The interpretation of this is that the reproduction number is decreasing with $CV_S = 1/\sqrt{n_S}$, the coefficient of variation of the delay time. However, $R_{\theta,\rho}$ grows faster for the case $n_I = \infty$ (I is constant) than when $n_I = 1$ (I is exponentially distributed).

Applying the same line of argument for the pairs $(n_I, n_S = 1)$ and $(n_I, n_S = +\infty)$ in Equation (3.7) we obtain respectively

$$R_{\theta,\rho}(n_I, n_S = 1) = \left[(1-\rho)+\rho\left(1-\left(\frac{n_I\gamma}{\lambda+n_I\gamma}\right)^{n_I}\right)^{-1}\left(1-\left(\frac{n_I\gamma+\theta}{\lambda+n_I\gamma+\theta}\right)^{n_I}\right)\right]R_0 \quad (3.10)$$

and

$$R_{\theta,\rho}(n_I, n_S = +\infty) = \left[(1-\rho)+\rho\left(1-\left(\frac{n_I\gamma}{\lambda+n_I\gamma}\right)^{n_I}\right)^{-1}\left(1-e^{-(\frac{\lambda}{\gamma}+n_I\frac{\theta}{\gamma})}\right)\right]R_0. \quad (3.11)$$

Using the above set of parameter values in (3.10) and (3.11), we plot $R_{\theta,\rho}(n_I, n_S = 1)$ and $R_{\theta,\rho}(n_I, n_S = +\infty)$ as functions of n_I (see Figure 5). It is observed that $R_{\theta,\rho}$ is increasing with n_I for the case $n_S = 1$, implying that $R_{\theta,\rho}$ is decreasing with CV_I the coefficient of variation of the infectious period. However, $R_{\theta,\rho}$ is decreasing with n_I if $n_S = +\infty$ (i.e. S is constant period), showing that the reproduction number is increasing in CV_I if

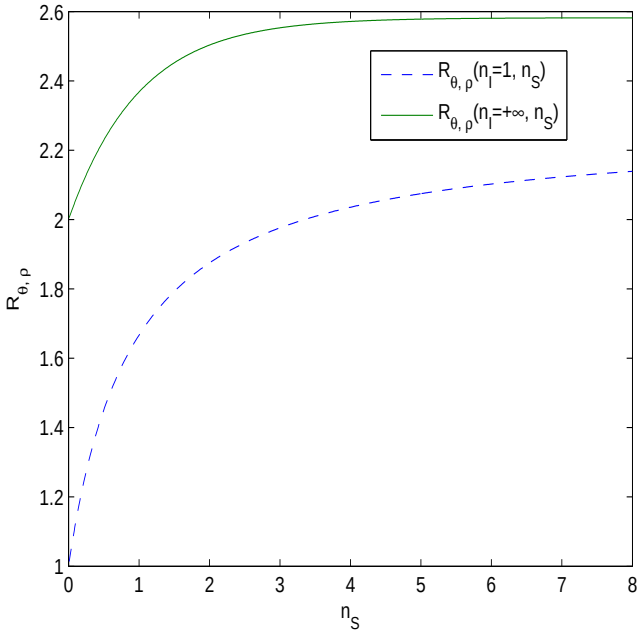

Figure 4: Reproduction number $R_{\theta,\rho}$, as a function of n_S, for two extreme cases of n_I (i.e. $n_I = 1$ and n_S).

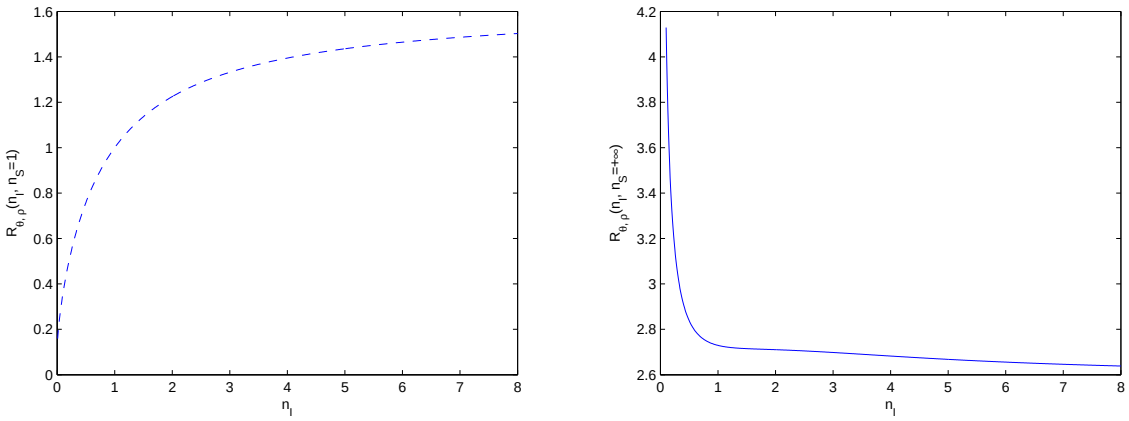

Figure 5: The reproduction number $R_{\theta,\rho}$ as a function of n_I for $n_S = 1$ (left) and $n_S = +\infty$ (right).

the vaccination delay time is constant. To conclude, we have shown that $R_{\theta,\rho}$ is always decreasing with the coefficient of variation of S, but $R_{\theta,\rho}$ can go either way as a function of CV_I.

3.2 Vaccination after delay with bounds on the maximum number of infections

The reproduction number $R_{\theta,\rho}$ in the first strategy is proportional to the basic reproduction number R_0. As a consequence, if R_0 is very large, for example due to a heavy tailed degree distribution, so will $R_{\theta,\rho}$ be. The second strategy aims at reducing the reproduction number more than the first strategy. To achieve this we assume that it is possible to put an upper bound, m say, on the number of friends that can be infected by one infectious individual. By this we mean that an individual can at most infect m friends. A heuristic motivation as to why this could be the case is that an individual is more likely to be detected the more friends she has infected, so if the number is as large as some value m she will always be detected. We assume that, if an infectious person is detected then all friends are located (so $\rho = 1$) and vaccinated after the same delay time S counted from the time at which the person becomes infectious.

So given that the degree of an individual is $D = k$, we want to compute the expected number of friends say Z, who ultimately get infected (i.e. $E(Z|D = k)$) and then the unconditional expected value of Z, given by $E(Z) = \sum_{k=1}^{\infty} E(Z|D = k)\frac{kp_k}{E(D)}$. As before, a typical infectious individual has k friends with probability $\frac{kp_k}{E(D)}$. Let m be the maximum number of friends an infectious person can infect among the $k-1$ initial susceptible friends. We now consider two possible cases. The first case is, when $k-1 \leq m$, implying that m plays no role in the transmission of infection. Consequently, the number of newly infected individuals can be written as the sum of indicators, showing whether each of the $k-1$ friends is infected or not, here denoted Z. Thus, the conditional mean of Z is given by

$$E(Z|D = k) = \sum_{i=1}^{k-1} P(\text{friend } i \text{ gets infected}) = (k-1)P(\text{friend } i \text{ gets infected}). \quad (3.12)$$

The probability of infecting individual i, P(friend i gets infected), here denoted π can be computed from Equation (3.6), but now with $\rho = 1$, since all susceptible friends are vaccinated after the detection of an infectious individual. Thus, π equals

$$\pi = 1 - \Big(\frac{n_I\gamma + n_S\theta}{\lambda + n_I\gamma + n_S\theta}\Big)^{n_I+n_s-1},$$

and as a result Equation (3.12) becomes

$$E(Z|D = k) = (k-1)\ \pi.$$

The second case is when $k-1 > m$. So, given that the individual has degree $D = k$, the delay time $S = s$ and infectious period $I = i$, each individual out of the $k-1$ susceptible friends is contacted independently with the same probability $1 - e^{-\lambda \min(s,i)}$. However, not more than m friends can be infected. As a consequence, the conditional expected number of infected friends, denoted Z is given by

$$\begin{aligned} E(Z|D=k, S=s, I=i) = &\sum_{j=0}^{m} j \binom{k-1}{j} \left(1 - e^{-\lambda \min(s,i)}\right)^j \left(e^{-\lambda \min(s,i)}\right)^{k-1-j} \\ &+ \sum_{j=m+1}^{k-1} m \binom{k-1}{j} \left(1 - e^{-\lambda \min(s,i)}\right)^j \left(e^{-\lambda \min(s,i)}\right)^{k-1-j}. \end{aligned} \tag{3.13}$$

The unconditional expected number of friends infected by a single person during her infectious period (i.e. the reproduction number), denoted R, is hence given by

$$\begin{aligned} R &= \sum_{k=1}^{\infty} E(Z|D=k) \frac{k p_k}{E(D)} \\ &= \sum_{k=1}^{m+1} (k-1)\pi \frac{k p_k}{E(D)} + \sum_{m+2}^{\infty} \left[\sum_{j=0}^{m} j \binom{k-1}{j} \left(1 - e^{-\lambda \min(s,i)}\right)^j \left(e^{-\lambda \min(s,i)}\right)^{k-1-j} \right. \\ &\left. + \sum_{j=m+1}^{k-1} m \binom{k-1}{j} \left(1 - e^{-\lambda \min(s,i)}\right)^j \left(e^{-\lambda \min(s,i)}\right)^{k-1-j} \right] \frac{k p_k}{E(D)}. \end{aligned} \tag{3.14}$$

As usual only small outbreaks of the disease may be observed if $R \leq 1$. The expression of the reproduction number R in this strategy is somehow complicated to enable us perform some theoretical analysis of the effects of the random behaviour in the distributions of infectious period and delay times. However, though tedious it can be shown numerically that R is decreasing with the coefficient of variations of both the infectious period and detection time.

4 Discussions

In the present paper we have studied how the random properties of the infectious period and delay time affect the reproduction numbers when the disease propagates in a random social network. The epidemic model is an SEIR, and we assumed that the latent period has an arbitrary distribution, and infectious period and delay time have independent gamma distributions. Under the assumption that the detection of an infected individual may only occur while the person is infectious (after latent period), it is seen that the reproduction numbers are independent of the latent period.

In particular we have shown that the stochasticity of the distributions of the infectious period and delay time have significant impacts on the reproduction numbers, and hence on the final size of the epidemic. The basic reproduction number R_0 and the reproduction

number $R_{\theta,\rho}$ in the first strategy are both decreasing with the coefficient of variation of the infectious period. Furthermore, $R_{\theta,\rho}$ is decreasing with the coefficient of variation of the delay time, implying that the variations in the distributions of both infectious periods and delay times affect the dynamic of an infectious disease. Similar effects of the distributions of I and S can be observed on the reproduction number R of the second strategy, though it is hard to get a simple expression of R for theoretical analysis.

The models which have been developed are not fully realistic, but we believe that they may capture some of the relevant behaviour which would appear in complex models. For example, a more realistic social structure would consider some network properties such as clustering, associativity and preferential attachment (see e.g. Newman 2003). A possible generalization of the model would be to consider different types of individuals, and to assume that both network properties as well as transmission probabilities depend on the type of an individual; see e.g. Ball Clancy (1993). For example, it would be interesting to consider the varying susceptibility of individuals to the disease,(see e.g. O'Neill 2001).

The most obvious continuation of the present paper is however, to partition the community into small groups such as households, schools, workplaces and so on. It is well known that in a community partitioned into small groups, the contact rates among individuals are different for within the group and between the groups. The main interest would be to study the effect of some vaccination strategies in reducing the spread of the disease in such the community with heterogeneties between individuals.

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