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Networks, epidemics and vaccination through contact tracing

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Abstract

We consider a (social) network whose structure can be represented by a simple random graph having a pre-specified degree distribution. A Markovian SIR epidemic model is defined on such a social graph. We then consider two real-time vaccination models for contact tracing during the early stages of an epidemic outbreak. The first model considers vaccination of each friend of an infectious individual (once identified) independently with probability ρ. The second model is related to the first model but also sets a bound on the maximum number an infectious individual can infect before being identified. Expressions are derived for the influence on the reproduction number of these vaccination models. We give some numerical examples and simulation results based on the Poisson and heavy-tail degree distributions where it is shown that the second vaccination model has a bigger advantage compared to the first model for the heavy-tail degree distribution.

Key words: Contact tracing, degree distribution, delay time, epidemic model, reproduction number, networks, vaccination model.

1 Introduction

Networks, for example social networks, are often described by simple undirected random graphs in order to capture social relationships among different individuals (see [1], [2], [3]). Usually the vertices of the graph correspond to individuals and the edges to some social relations (e.g. [4], [5], [6]). On such a (social) graph an epidemic model may be defined, where initially individuals are free from the disease and susceptible. An infectious individual can infect its susceptible friends (those who have not had the disease yet, but can catch it), before it recovers and becomes immune. The identification of individuals that have been in contact with an infectious individual (contact tracing) has attracted attention as a disease control measure that seeks to uncover newly infected cases preferably

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before they become infectious (e.g. see [7], [8], [9], [10]). The traced individuals who are still susceptible can be vaccinated (or immunized in some other way) in order to prevent a major outbreak. How to contain the disease before it takes off is a question that can be addressed by the choice of a vaccination strategy (see e.g. [11], [12], [13], [14]).

In the present paper we study issues arising from such modelling. In particular we consider a simple social graph of a large fixed population where the vertex degree (number of friends) follows a pre-specified distribution F. The social graph is assumed to be otherwise completely random, implying that there will be no clustering (of connected friends) or degree correlation (assortativity or disassortativity). A simple stochastic epidemic model is the Markovian susceptible-infectious-removed (SIR) for the spread of the disease in the social network (see [14], [15]). Initially, one randomly selected individual is infected from outside the population. Any individual who gets infected infects each of his/her friends who are still susceptible at the rate λ during the infectious period having exponential distribution, and then recovers and becomes immune (denoted removed). For this social graph and epidemic model we study two vaccination models implemented during early stages of the epidemic using contact tracing. In both vaccination models infected individuals are detected after some delay and once an individual is detected his/her friends are "vaccinated". We will use the word vaccinated but it could equally well be isolation or some other control measure that stops potential spreading of the individual. In the first vaccination model we introduce some realism in that not all friends are found and vaccinated. In the second model, being more optimistic, it is assumed that all friends are found and vaccinated once the individual is detected, and it is also assumed that there is a maximal value m of the number of friends an individual can infect before being detected.

For a fixed large population of size n we derive the reproduction number R_0 defined as the expected number of secondary cases generated by a typical infectious individual during the early stages of the epidemic, for an epidemic without intervention. The quantity R_0 is of fundamental importance to the dynamics of infections, since a major outbreak is possible if and only if $R_0 > 1$ (e.g. [15]). We also derive the reproduction numbers for the two vaccination models. Using Poisson and heavy-tail degree distributions for the social networks, we illustrate how effective the two vaccination models are and show that the gain in the second model is greater for the heavy-tail degree distribution. The first vaccination model is much less effective for heavy-tail degree distributions mainly because individuals with high degree, which are less uncommon in the heavy-tail case and which have relatively higher chance of getting infected, have the chance to infect many more individuals in the first vaccination model. The numerical results, based on large population approximations, are verified by means of simulations in finite populations.

Much work has been done on vaccination strategies prior to arrival of the disease. For example, targeted vaccination [16], uniform vaccination [17], [18], [19] and acquaintance vaccination ([16], [20]). In the uniform and acquaintance vaccination strategies, individuals are chosen randomly, and targeted vaccination strategy requires the identification of

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individuals with high degrees. However, during the early stages of an epidemic, contact tracing can be used as a control measure of the epidemic in a social network, see e.g. [21], where individuals who are related to an identified infectious person are located and vaccinated (or subjected to some other type of immunization). The present paper contributes to this work on early stage vaccination by proposing two real-time vaccination models applicable during the early stages of an epidemic outbreak, and that require no knowledge of individuals' degree prior to the detection of an infectious person. Another aim of the paper is to derive expressions for the reproduction numbers, without vaccination as well as when each vaccination model is implemented, and to compare the efficiency of the vaccination models in preventing major outbreaks. The rest of the paper is organized as follows. In Section 2 we describe a model to generate a social network with a given degree distribution. We also define a simple epidemic model for a disease spreading on the social network, derive the basic reproduction number and approximate the probability of a minor outbreak. Section 3 treats vaccination as a measure to prevent major outbreaks, and discusses two vaccination models. Section 4 provides numerical examples and simulation results of the model. Finally, Section 5 discusses the results and provides some concluding remarks.

2 The Model

2.1 The network model

Consider a community of size n (assumed to be large) and assume that the degree distribution $F = \{p_k\}_{k=0}^{\infty}$ is given. We define a random network model describing the social structure of the population. The network model is based on the configuration model (see, e.g. [22]) and is defined as follows. Take a set of n vertices and for each vertex i assign a number of stubs D_i independently from the distribution $F = \{p_k\}_{k=0}^{\infty}$. Then pair these stubs completely at random and join the stubs to form edges between the vertices. 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. We then remove all multiple edges and loops thus making the graph simple. This procedure produces a graph with the desired degree distribution as $n \to \infty$, but which in all other aspects is random. That is, we generate a graph which is drawn uniformly at random from the set of graphs with the given distribution. There will hence be no clustering or degree correlation (assortativity or disassortativity). See [23], [24] for details on the mixing patterns of the graph and [25] for details on how to generate the graph and discussion on what degree distributions the algorithm works for.

2.2 A simple epidemic model on the social graph

We now define an epidemic process on the social network described above. As mentioned before, we consider three states: Susceptible, Infectious and Recovered (and immune) that an individual can be in during an epidemic process. The SIR epidemic model is used to describe the dynamic process of infections in the population through friend-

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ships (which are edges in the graph). Assume initially that all individuals are susceptible except one individual who is infected from outside the population. The infected individual remains infectious for a time period according to a random variable I which follows an arbitrary distribution G, and after the period I the individual recovers and becomes immune. During this infectious period I, an infectious individual infects each of his/her susceptible friends according to independent Poisson processes with intensity λ. This implies that the first contact between the infectious individual and a particular neighbour i takes place time T_i (time units) after the infection, where T_i is exponentially distributed with mean $\frac{1}{\lambda}$. We assume that there is no latent period, so any susceptible friend contacted by an infectious individual becomes immediately infectious. Those who become infected behave similarly, and the epidemic process goes on until there are no more infectious individuals, when the epidemic stops. It is worth noting that the time T_i to contact a specific friend i is different from time T_j to contact another friend j, where $i \neq j$, and the contact times $\{T_i\}_{i=1}^{k}$, k being the number of friends, are independent and exponentially distributed, and only contacts occurring in the interval $[0, I]$ lead to infections.

2.3 The basic reproduction number

We now derive the basic reproduction number for the epidemic model, and to do so we start by deriving the probability to infect a given individual before recovery. During the initial stages of an epidemic in a large population, contacted individuals are susceptible with high probability, implying that the number of infectious individuals during early stages may be approximated by a branching process (see e.g. [26], [27]). In a branching process every infectious individual gives birth to (infects) a random number of offspring (infected) independently of each other, but with the same distribution. The process is assumed to start with Z_0 individuals and each individual capable of giving birth has a random lifespan equivalent to her infectious period I. In the following we assume that initially there is only one ($Z_0 = 1$) infectious individual capable of infecting other individuals in its neighbourhood. The approximation of the process can be made precise by coupling arguments as in [26], but this is beyond the scope of the present paper.

Given an infectious individual, transmission of infection to a given friend occurs if and only if the first contact takes place during the infectious period. That is, if the time T_i to contact a specified susceptible friend i is less than the infectious period I having an arbitrary but specified distribution G. We compute the probability p that transmission of infection will occur by conditioning on T_i as follows,

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

To determine the basic reproduction number of the epidemic process, we consider an infectious individual in the second generation since the initial infectious individual is chosen at random without respect to the degree. In the second generation and onwards, an infectious individual with k friends is selected with probability proportional to kp_k (which is size biased), hence individuals with many friends are more likely to be selected.

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If the selected person has degree k, he/she generates on average $(k-1)p$ new cases since the individual he/she was infected by is not susceptible. Hence, it follows that the basic reproduction number becomes;

$$R_0 = p\sum_k (k-1)\frac{kp_k}{\sum_j jp_j} = p\sum_k (k-1)\frac{kp_k}{E(D)}$$

(see [28] for more details). An alternative representation is given by

$$R_0 = p\left(E(D) + \frac{V(D)-E(D)}{E(D)}\right). \tag{2.2}$$

When we derive the probability of a major outbreak below we will restrict ourselves to the case where the infectious period is exponentially distributed (with parameter γ). Suppose hence that $G(t) = 1-e^{-\gamma t}$, i.e. that the infectious period $I \sim$Exp(γ) with mean $1/\gamma$. The probability p (defined in (2.1)) then becomes

$$p = P(T_i < I) = \int_0^\infty e^{-\gamma t}\lambda e^{-\lambda t}dt = \frac{\lambda}{\lambda+\gamma}, \tag{2.3}$$

(see e.g. [29], [30] for related results).
The corresponding basic reproduction number in Equation (2.2) then becomes

$$R_0 = \frac{\lambda}{\lambda+\gamma}\left(E(D) + \frac{V(D)-E(D)}{E(D)}\right). \tag{2.4}$$

If $R_0 > 1$ major outbreaks are possible whereas if $R_0 \leq 1$ then only minor outbreaks can occur.

2.4 The probability of a minor outbreak

In order to determine the probability of a minor outbreak, it is important to consider the distribution of the number of offspring (infected) Z generated by one infectious individual, since given this distribution the extinction probability can be derived (see e.g. [27]). During his/her infectious period, from now on assumed to be exponentially distributed with parameter γ, an individual infects each friend at rate λ, and recovers at rate γ. Conditioning on the degree $D = k$, an individual hence infects at least one of his $k-1$ susceptible friends with probability

$$\begin{aligned} P(Z \geq 1|D=k) &= 1 - P(Z=0|D=k) \\ &= 1 - \int_0^\infty (e^{-\lambda t})^{k-1}\gamma e^{-\gamma t}dt \\ &= 1 - \frac{\gamma}{(k-1)\lambda+\gamma} \\ &= \frac{(k-1)\lambda}{(k-1)\lambda+\gamma}. \end{aligned} \tag{2.5}$$

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Similarly, the probability that an individual infects at least two friends follows the same line of approach as in Equation (2.5), and thus becomes

$$P(Z \geq 2|D = k) = \frac{(k-1)\lambda}{(k-1)\lambda + \gamma} \cdot \frac{(k-2)\lambda}{(k-2)\lambda + \gamma}.$$

In general, the probability that an individual infects to at least z friends hence is

$$P(Z \geq z|D = k) = \prod_{j=1}^{z} \left(\frac{(k-j)\lambda}{(k-j)\lambda + \gamma} \right), \quad z < k. \tag{2.6}$$

Consequently, from (2.6)

$$P(Z = z|D = k) = \left(\prod_{j=1}^{z} \frac{(k-j)\lambda}{(k-j)\lambda + \gamma} \right) \cdot \frac{\gamma}{(k-1-z)\lambda + \gamma}, \quad z < k. \tag{2.7}$$

The degree distribution of the index case is $\{p_k\}$, since the initially infected person was selected at random from the community. However, the degree distribution of infected individuals in the second and later generations of the initial phase of the epidemic will be the size biased version of this distribution: $\{kp_k/E(D)\}$ (see [31] for a proof). This follows because the probability to get infected during the early stages is proportional to the number of friends you have – for example, individuals with no friends clearly will not get infected. The unconditional offspring distribution is thus given by

$$P(Z = z) = \sum_{k=z+1}^{\infty} \left[\left(\prod_{j=1}^{z} \frac{(k-j)\lambda}{(k-j)\lambda + \gamma} \right) \cdot \frac{\gamma}{(k-1-z)\lambda + \gamma} \right] \cdot \frac{kp_k}{E(D)}. \tag{2.8}$$

Hence, all succeeding offspring in the later generations give birth independently according to the distribution in Equation (2.8). If the offspring distribution of the index case was the same as for infected in later generations then, from branching process theory (see e.g [27]), the extinction probability (i.e. the probability of a minor outbreak in the epidemic) q would be the smallest solution to

$$q = \sum_{z=0}^{\infty} q^z P(Z = z). \tag{2.9}$$

It is also known in branching process theory that when the mean number of offspring is less or equal to 1 ($E(Z) = R_0 < 1$) then the only solution is $q = 1$, and if $R_0 > 1$ then there is a second solution $q < 1$ which is equal to the extinction probability. The quantity q is the probability of extinction (i.e. a minor outbreak) if the index case had the same offspring distribution as the infected in later generations. This is however not the case for two reasons: the initially infectious person has degree distribution $\{p_k\}_{k=0}^{\infty}$ (rather than $\{\frac{kp_k}{E(D)}\}$) and may infect all its friends (rather than all except one). So, let

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$\tilde{Z}$ denote the number of individuals that an initial person infects. The unconditional offspring distribution of the index case is hence given by

$$P(\tilde{Z}=z)=\sum_{k=z}^{\infty}\left[\left(\prod_{j=1}^{z}\frac{(k+1-j)\lambda}{(k+1-j)\lambda+\gamma}\right)\cdot\frac{\gamma}{(k-z)\lambda+\gamma}\right]\cdot p_k. \tag{2.10}$$

To obtain the probability of a minor outbreak also taking into account the index case we simply condition on the number of offspring this individual has. If the index case has z offspring (which happens with probability $P(\tilde{Z}=z)$, defined in (2.10)) then the epidemic will go extinct if all the epidemics initiated by the z individuals goes extinct. These z epidemics all go extinct with probability q^z. To conclude, the probability π of a minor outbreak of the epidemic initiated by the index case is hence given by

$$\pi=\sum_{z=0}^{\infty}q^z P(\tilde{Z}=z),$$

where q was defined as the smallest solution to (2.9) This probability is computed numerically in Section 4 for a specific example. It should be noted that $\pi<1$ if and only if $q<1$.

3 Vaccination models

Vaccination is a major tool for protection of individuals in a population against infectious diseases. Similar effects to vaccination may be obtained through isolation and quarantine, in what follows "vaccination" could hence also be interpreted in this more general form. In this section we discuss two hypothetical vaccination models that might be applicable during the early stages of an epidemic upon detection of the diseased cases. In both models, it is assumed that whenever an infectious individual is detected, which happens after some delay time, his or her friends are traced and located, but there is no "multi-step" tracing. Such located individuals are vaccinated, and it is assumed that the vaccine gives full immunity if the person was susceptible, but that the vaccine has no effect if the person was already infected (see e.g. [32] and [33] for more realistic assumption on vaccine efficacy). The delay time between infection and vaccination includes the time to show of symptoms, the time to be identified by authorities, the time to locate and vaccinate friends of the individual and also the time for the vaccine to have its effect. For simplicity it is assumed that all vaccinated friends of an individual become immune at the same time.

The two vaccination models are different in that the first model assumes that not all friends are detected, whereas the second model assumes all friends are detected and also that there is a maximum number an infectious individual can infect before getting detected, irrespective of the number of friends. Below we describe the two vaccination models in more detail and derive expressions for their reproduction numbers and compare their effectiveness.

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3.1 Vaccination of located friends

Let S be the delay time whose distribution is denoted by H. The vaccination model assumes that each friend of an infected and detected individual is located independently with probability ρ, and all located friends who are still susceptible are vaccinated. We also assume that the infectious period I and delay time S are independent. We introduce an indicator random variable X such that $X_i = 1$ (which happens with probability ρ) if a given friend i of an infectious individual is located and $X_i = 0$ otherwise. Taking into account the infectious period I and the time T_i to contact a given friend i, transmission of infection occurs when $T_i < \min(S, I)$, or if $S < T_i < I$ and $\{X_i = 0\}$. Conditioning on $T_i = t$, a given friend gets infected with probability

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

When we consider arbitrary distributions of I and S as before, that is, G and H respectively, we get the following general relation for the probability of infection,

$$\tilde{p} = \int_0^\infty \Big((1 - G(t))(1 - H(t))\Big)\lambda e^{-\lambda t} dt + (1-\rho)\int_0^\infty H(t)(1 - G(t))\lambda e^{-\lambda t} dt. \tag{3.1}$$

As before, during the early stages an infectious individual has degree k with probability $\frac{kp_k}{E(D)}$, and will then on average infect $(k-1)\tilde{p}$ individuals. The corresponding reproduction number R hence equals

$$R = \tilde{p}\sum_k (k-1)\frac{kp_k}{E(D)} = \tilde{p}\left(E(D) + \frac{V(D) - E(D)}{E(D)}\right). \tag{3.2}$$

From now on, we assume that the infectious period I and detection time S are independent and exponentially distributed with mean $1/\gamma$ and $1/\theta$ respectively. We then get from Equation (3.1) that

$$\begin{aligned}\tilde{p} &= \int_0^\infty \lambda e^{-(\lambda+\gamma+\theta)t} dt + (1-\rho)\int_0^\infty (1 - e^{-\theta t})\lambda e^{-(\lambda+\gamma)t} dt \\ &= \frac{\rho\lambda}{\lambda+\gamma+\theta} + \frac{(1-\rho)\lambda}{\lambda+\gamma}.\end{aligned} \tag{3.3}$$

The reproduction number $R_{\theta,\rho}$ in Equation (3.2) becomes

$$R_{\theta,\rho} = \left(\frac{\rho\lambda}{\lambda+\gamma+\theta} + \frac{(1-\rho)\lambda}{\lambda+\gamma}\right)\left(E(D) + \frac{V(D) - E(D)}{E(D)}\right) \tag{3.4}$$

Using Equation (2.4) and some algebra, Equation (3.4) can be simplified to yield the representation of $R_{\theta,\rho}$ in terms of the basic reproduction number R_0 as

$$R_{\theta,\rho} = \left(1 - \frac{\rho\ \theta}{\lambda+\gamma+\theta}\right)R_0. \tag{3.5}$$

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$R_{\theta,\rho}$ hence grows linearly with the basic reproduction number R_0. In order to surely prevent an epidemic outbreak when this vaccination, we must have that $R_{\theta,\rho} \leq 1$, or equivalently that

$$\Big(1 - \frac{\rho\,\theta}{\lambda+\gamma+\theta}\Big) R_0 \leq 1.$$

To be more precise, ρ and θ must satisfy

$$\frac{\rho\theta}{\lambda+\gamma+\theta} \geq 1 - \frac{1}{R_0}.$$

In this vaccination model there are two special cases. First, if all friends of an infectious person are located so that $\rho = 1$ the reproduction number in Equation (3.5) becomes

$$R_{\theta,\rho=1} = \frac{\lambda+\gamma}{\lambda+\gamma+\theta} R_0. \tag{3.6}$$

In this special case, the reproduction number $R_{\theta,\rho=1}$ is assured to be below unity if

$$\theta \geq (\lambda+\gamma)(R_0 - 1),$$

or equivalently if the expected time to detection $\frac{1}{\theta}$ satisfies

$$\frac{1}{\theta} \leq \frac{1}{(\lambda+\gamma)(R_0-1)}$$

implying that the detection intensity θ must be large enough in order to avoid the spread of infection in the social network.

A second special case is when the detection intensity θ is so large that the first term of $\tilde{p}$ in the second equality of Equation (3.3) approaches zero in the limit as $\theta \to \infty$. Then the probability of infection becomes,

$$\tilde{p} = \frac{(1-\rho)\lambda}{\lambda+\gamma}.$$

This is equivalent to detecting an infectious individual immediately when he/she becomes infected and a proportion ρ of all friends are vaccinated. Using the same argument as before, the corresponding reproduction number in Equation (3.5) reduces to

$$R_{\theta=\infty,\rho} = (1-\rho)R_0, \tag{3.7}$$

which is linear in ρ, and surely there will be a minor outbreak if $\rho \geq 1 - \frac{1}{R_0}$. The interpretation of this is that, if R_0 is large, for instance a heavy tailed network, then nearly all friends must be vaccinated. The criterion that $\rho \geq 1 - \frac{1}{R_0}$ is the same as in a general vaccination programme in order to obtain herd immunity. The difference with the approach suggested here is that it is only necessary to vaccinate around those who get infected. If this is possible, the spread can be controlled with considerable fewer vaccinations than if the vaccination took place before an outbreak.

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In principle it is possible to derive the full offspring distribution and thus the extinction probability which we now sketch. We approximate the initial phase of the epidemic by a branching process. Given the infectious period $I = i$ and detection time $S = s$, an individual contacts a particular friend with probability $\eta_{s,i} = \rho(1 - e^{-\lambda \min(s,i)}) + (1 - \rho)(1 - e^{-\lambda i})$. Consequently, $\eta_{s,i} = \rho(1 - e^{-\lambda s}) + (1 - \rho)(1 - e^{-\lambda i})$ if $s < i$ or $\eta_{s,i} = 1 - e^{-\lambda i}$ if $s > i$. Thus, given that degree $D = k$, time to detection $S = s$ and infectious time period $I = i$, the number of infections produced by one individual in the first generation is binomially distributed with parameters $(k-1)$ and $\eta_{s,i}$. The parameter $(k-1)$ follows since the initially infectious individual was infected by one of her friends (who cannot be re-infected). Hence, an individual gives birth to $Z = z$ offspring with probability

$$\begin{aligned} P(Z = z|D = k) = &\int_0^\infty \int_0^i \binom{k-1}{z} \eta_{s,i}^z \Big(1 - \eta_{s,i}\Big)^{k-1-z} f_S(s) f_I(i) \, \mathrm{d}s \, \mathrm{d}i \\ &+ \int_0^\infty \int_i^\infty \binom{k-1}{z} \eta_{s,i}^z \Big(1 - \eta_{s,i}\Big)^{k-1-z} f_S(s) f_I(i) \, \mathrm{d}s \, \mathrm{d}i \end{aligned} \tag{3.8}$$

where $f(s)$ and $f(i)$ are the density functions of the time to detection S and infectious period I respectively.
Unconditionally, the number of individuals infected in a given generation is hence given by

$$P(Z = z) = \sum_{k=z+1}^{\infty} P(Z = z|D = k) \frac{k p_k}{E(D)}. \tag{3.9}$$

As before, we let q be the extinction probability of the branching process if the index case had the same offspring distribution as individuals of later generations, implying that q is the smallest solution to $q = \sum_{z=0}^{\infty} q^z P(Z = z)$. However, the index case has a slightly different offspring distribution $\tilde{Z}$ since its degree distribution is $\{p_k\}_{k=0}^{\infty}$ (and not $kp_k/E(D)$) and all friends can get infected (rather than all but one). Thus the probability π of a minor outbreak is given by $\pi = \sum_{j=0}^{\infty} q^j P(\tilde{Z} = j)$. It is worth pointing out that the offspring distribution and the extinction probability π is numerically quite complicated in most cases. The reproduction number $R_{\theta,\rho}$ given in (3.5), which determines whether major outbreaks may occur or not and which are the focus of the present paper, are of much simpler form.

3.2 Vaccination of friends and bounding the maximum number of infections

In the first vaccination model the reproduction number $R_{\theta,\rho}$ was linear in the basic reproduction number R_0 (see Equation 3.5), implying that if R_0 is large, which is the case with heavy tail degree distributions, so is $R_{\theta,\rho}$. We are then motivated to introduce another more optimistic vaccination model to see if it is possible to perform better. The new model aims at reducing the reproduction number further by controlling individuals who have many friends (super-spreaders) and this is done by assuming that there exists an upper bound m on the number of possible infections from a given infectious individual

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(for example this could be approximately true if infectives are more likely to become detected the more people they infect). The vaccination model is defined as follows. Let S and I be the detection time and infectious period, and assume as before that they are independent and exponentially distributed with mean $1/\theta$ and $1/\gamma$ respectively. While infectious and before being detected an individual having $(k-1)$ susceptible friends, contacts his/her friends independently at rate λ. We denote these contact times $T_i^{(k)}$, $i = 1, \ldots, k-1$ (measured from the time of infection). This implies that the ordered times $T_{(1)}^{(k)} \leq T_{(2)}^{(k)} \leq \ldots \leq T_{(k-1)}^{(k)}$ satisfy $T_{(1)}^{(k)} \sim \text{Exp}((k-1)\lambda)$, $T_{(2)}^{(k)} - T_{(1)}^{(k)} \sim \text{Exp}((k-2)\lambda)$, and so on. Using this notation the individual hence infects at least i friends if $T_{(i)}^{(k)} < \min(S, I)$. However, in the new model we also have a bound m on the number of friends that can be infected before an infectious individual is detected. It follows that an infected individual who has $k-1$ susceptible friends infects Z of them before detection, where

$$Z = \min\left\{ \max\left(i : T_{(i)}^{(k)} < \min(S, I)\right),\ m \right\}.$$

To compute the expected number of infected friends produced by a typical infectious individual during the early stages, we first determine the offspring distribution which is done in the same way as in Section 2.4. The difference between the first and second vaccination models is that, in the second model, all friends are located and also the number of infections caused by one individual is bounded by m. Hence, for $z = 1, \ldots, m \wedge (k-1)$, the probability that at least z friends are infected given $D = k$ is

$$\begin{aligned} P(Z \geq z | D = k) &= \frac{(k-1)\lambda}{(k-1)\lambda + \gamma + \theta} \cdot \frac{(k-2)\lambda}{(k-2)\lambda + \gamma + \theta} \cdots \frac{(k-z)\lambda}{(k-z)\lambda + \gamma + \theta} \\ &= \prod_{j=1}^{z} \left(\frac{(k-j)\lambda}{(k-j)\lambda + \gamma + \theta} \right) \end{aligned} \tag{3.10}$$

Since an individual with k friends cannot infect more than $m \wedge (k-1)$ friends, then $P(Z > z | D = k) = 0$, for $z > m \wedge (k-1)$. The unconditional probability that at least z friends get infected is thus given by

$$P(Z \geq z) = \sum_{k=z+1}^{\infty} P(Z \geq z | D = k) \frac{k p_k}{E(D)},\ z = 1, \ldots, m. \tag{3.11}$$

The sum in (3.11) starts at $k = z+1$ since in order to infect z friends an infectious person needs at least $z+1$ friends, one being the infector. The expected number of individuals infected by the infectious person, which is also the reproduction number, is

$$\begin{aligned} R_{\theta,m} = E(Z) &= \sum_{z=1}^{m} P(Z \geq z) = \sum_{z=1}^{m} \left(\sum_{k=z+1}^{\infty} P(Z \geq z | D = k) \frac{k p_k}{E(D)} \right) \\ &= \sum_{z=1}^{m} \sum_{k=z+1}^{\infty} \left(\prod_{j=1}^{z} \frac{(k-j)\lambda}{(k-j)\lambda + \gamma + \theta} \right) \frac{k p_k}{E(D)}. \end{aligned} \tag{3.12}$$

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The epidemic outbreak is surely avoided if $R_{\theta,m} \leq 1$. We should note here that since $R_{\theta,m}$ depends on the parameters λ and γ, then it also depends on R_0 defined in (2.4), but not as explicitly as $R_{\theta,\rho}$ in the first vaccination model.

As before it is possible to derive the offspring distribution and hence the probability of a minor outbreak which we now sketch. During the early stages the epidemic can be approximated by a branching process. From equation (3.10) the distribution of offspring produced by an individual in the later generations given that it has degree $D = k$ is hence, for $z = 0,\ 1,\ \ldots,\ m \wedge (k-1)$, given by

$$P(Z = z|D = k) = \left(\prod_{j=1}^{z} \frac{(k-j)\lambda}{(k-j)\lambda + \gamma + \theta} \right) \cdot \frac{\gamma}{(k-1-z)\lambda + \gamma + \theta}. \tag{3.13}$$

As a consequence for $z = 0,\ 1,\ \ldots,\ m$, the unconditional offspring distribution is

$$P(Z = z) = \sum_{k=z+1}^{\infty} \left[\left(\prod_{j=1}^{z} \frac{(k-j)\lambda}{(k-j)\lambda + \gamma + \theta} \right) \cdot \frac{\gamma}{(k-1-z)\lambda + \gamma + \theta} \right] \cdot \frac{kp_k}{E(D)}. \tag{3.14}$$

Similar to before, applying theory for branching process [27] the probability q of extinction, assuming all individuals have offspring distribution $P(Z = z)$, is the smallest solution to $q = \sum_{z=0}^{\infty} q^z P(Z = z)$.

However, the initial infectious individual has degree distribution given by $\{p_k\}_{k=0}^{\infty}$ and can infect all friends (rather than all but one). Thus if $\tilde{Z}$ denotes this modified offspring distribution of the index case, then the probability π of a minor epidemic outbreak is given by

$$\pi = \sum_{j=0}^{\infty} q^j P(\tilde{Z} = j).$$

As was mentioned also for the first vaccination model it is for specific cases quite complicated to derive the offspring distribution and the probability π of a minor outbreak. In the examples of the next section we therefore focus on the reproduction numbers which are easier to compute.

4 Examples and simulations

We have compared our two vaccination models by simulations for different parameter values using the same population of size $n = 1000$ individuals. We used two different degree distributions, the Poisson distribution with mean 4 and a heavy-tail (scale-free) distribution, to compare the effectiveness of the vaccination models. For reasonable comparison of the models, the offspring distribution in the heavy tail is modelled as a sum of two independent random variables X_1 and X_2, (that is $Z = X_1 + X_2$), where the distribution of X_1 is $P_{X_1}(k) = C(k+1)^{-2.5},\ \ k = 0,\ 1,\ 2,\ \cdots$, with $C = (\sum_{j=0}^{\infty}(j+1)^{-2.5})^{-1} \approx 1/1.34$ and having mean ≈ 0.9, and where X_2 is Poisson distributed with mean 3.1. It follows that Z is heavy tail with mean 4, i.e. the same as the other (Poisson) degree distribution.

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We have chosen both the contact rate λ and the recovery rate γ to be equal to 1 in all simulations. For the Poisson degree distribution with mean 4, the basic reproduction number is equal to 2 (using Equation (2.4)). In the heavy-tail degree distribution, the theoretical basic reproduction number is infinite ($R_0 = +\infty$), but in a finite population the basic reproduction number is of course finite. For our finite population size, the basic reproduction number of our heavy-tail distribution is approximately 7.2, and it is computed (from Equation (2.4)) with mean degree equal to 4.0 and degree variance 45.6 obtained by using the truncated degree distribution.

In order to compare the effectiveness of the two vaccination models in terms of preventing an outbreak, we have chosen the detection rate θ, the proportion of located friends ρ and the bound on the maximum number m a person can infect to take on the values $\theta = 1, 5, 20$, $\rho = 0.2, 0.5, 1$ and $m = 2, 5, 10$. These parameter values are just a representation of many other values which can be chosen for the same purpose. Based on Equations (3.5) and (3.12) the interest is to observe the behaviour of the reproduction numbers for the two vaccination models as the values of θ, ρ and m are varied. Hence, these parameter values are designated as small ($\theta = 1$, $\rho = 0.2$, $m = 2$), intermediate ($\theta = 5$, $\rho = 0.5$, $m = 5$) and large ($\theta = 20$, $\rho = 1$, $m = 10$) with reference to the simulation results.

We have performed 500 simulations of the epidemic without vaccination and each of the two vaccination models for each parameter set-up. The social graph was generated once in order to have a common social structure, and in each simulation the initial infectious individual was chosen randomly from the graph. We define $\pi^{(1)}$ and $\pi^{(2)}$ as the proportions of the 500 simulations having 50 or less infected individuals (in the first and the second vaccination models respectively), so "minor outbreak" is here interpreted as having 50 or less infected. Admittedly, this number is quite arbitrarily chosen, but as can be seen from the simulations (e.g. Figure 1) a different cut-off would not change results much. We also let $\mu^{(1)}$ and $\mu^{(2)}$ respectively be the average sizes among major outbreaks in the first and the second vaccination models (here the choice with 50 as cut-off plays a slightly bigger role). The summary of the results are shown in Tables 1 and 2.

In Table 1 the numerical results from the Poisson degree distribution are presented. There are nine combinations of parameter values comprised of the pairs (θ, ρ) from the first model, and nine combinations of the pairs (θ, m) from the second model. Each pair of the parameter values is used in the simulations to obtain the proportions $\pi^{(i)}$ of minor outbreaks and the average sizes among major outbreaks $\mu^{(i)}$, for $i = 1, 2$. Using Equations (3.5) and (3.12), the reproduction numbers corresponding to the pairs (θ, ρ) and (θ, m) were computed numerically. The results indicate that the second vaccination model is most effective (as to be expected) in reducing the reproduction numbers below unity, thus preventing an epidemic outbreak. The only cases where the first model is effective is when the detection rate of an infectious individual is intermediate or high (that is $\theta = 5$ or $\theta = 20$) and the proportion of located friends is large ($\rho = 1$). We also note that the first vaccination model with any θ and $\rho = 1$ corresponds to the second vaccination model

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Table 1: Numerical values from a Poisson degree distribution with mean 4 and $\lambda = \gamma = 1$ and $R_0 = 2$. $R_{\theta,\rho}$ and $R_{\theta,m}$ are computed numerically whereas π and μ are obtained from the simulations. See text for further details.

Model 1	$R_{\theta,\rho}$	$\pi^{(1)}$	$\mu^{(1)}$	Model 2	$R_{\theta,m}$	$\pi^{(2)}$	$\mu^{(2)}$
$\theta = 1,\ \rho = 0.2$	1.87	0.414	260	$\theta = 1,\ m = 10$	1.33	0.732	231
$\theta = 1,\ \rho = 0.5$	1.67	0.490	238	$\theta = 1,\ m = 5$	1.30	0.710	218
$\theta = 1,\ \rho = 1$	1.33	0.698	224	$\theta = 1,\ m = 2$	1.01	0.908	82
$\theta = 5,\ \rho = 0.2$	1.71	0.472	252	$\theta = 5,\ m = 10$	0.57	1	–
$\theta = 5,\ \rho = 0.5$	1.28	0.706	187	$\theta = 5,\ m = 5$	0.56	1	–
$\theta = 5,\ \rho = 1$	0.57	1	–	$\theta = 5,\ m = 2$	0.51	1	–
$\theta = 20,\ \rho = 0.2$	1.64	0.496	215	$\theta = 20,\ m = 10$	0.18	1	–
$\theta = 20,\ \rho = 0.5$	1.09	0.874	100	$\theta = 20,\ m = 5$	0.18	1	–
$\theta = 20,\ \rho = 1$	0.18	1	–	$\theta = 20,\ m = 2$	0.17	1	–

with the same θ and $m = +\infty$. This is reflected in Table 1 that $R_{\theta,\rho=1}$ equals $R_{\theta,m=10}$ up to rounding errors for $\theta = 1,\ 5,\ 20$. The second model performs well for all values of our choice for the maximum bound of infections ($m = 2, 5, 10$) when the detection rate is intermediate ($\theta = 5$) or high ($\theta = 20$).

Table 2 shows the corresponding results, but for the heavy-tail degree distribution. When the basic reproduction number $R_0 = 7.2$ (in our case) the first model is effective when the detection rate is large ($\theta = 20$) and requires the vaccination of all friends of the infectious individuals ($\rho = 1$). The second model works well for the intermediate or high detection rate (that is $\theta = 5$ or $\theta = 20$) and for all our choices of the maximum bound of infections is small (that is $m = 2,\ 5,\ 10$). Hence, model 2 is more efficient in reducing the reproduction number as well as preventing outbreaks. More importantly, the difference between the two vaccination models is greater when considering the heavy-tail degree distribution.

Figures 1 and 2 illustrate the outbreak sizes of the epidemic for the 500 simulations in the heavy-tail degree distribution. Figure 1 presents the outbreak size from 500 simulations of the epidemic without vaccination and the basic reproduction number R_0 equals 7.2. The proportion of simulations resulting in minor outbreaks is 0.402 which is in agreement with the computed extinction probability $\pi = 0.413$ obtained using methods presented in Section 2.4, and the average size among major outbreaks is 265. Figure 2*A* shows the outbreak size after the implementation of the first vaccination model with detection rate $\theta = 5$ and the proportion of located friends $\rho = 1$. It is seen that there are

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Table 2: Numerical values from a heavy-tail degree distribution with mean 4 and $\lambda = \gamma = 1$ and $R_0 = 7.2$. $R_{\theta,\rho}$ and $R_{\theta,m}$ are computed numerically whereas π and μ are obtained from the simulations. See text for further details.

Model 1	$R_{\theta,\rho}$	$\pi^{(1)}$	$\mu^{(1)}$	Model 2	$R_{\theta,m}$	$\pi^{(2)}$	$\mu^{(2)}$
$\theta = 1,\ \rho = 0.2$	6.7	0.430	256	$\theta = 1,\ m = 10$	3.5	0.694	172
$\theta = 1,\ \rho = 0.5$	6.0	0.468	219	$\theta = 1,\ m = 5$	2.5	0.742	147
$\theta = 1,\ \rho = 1$	4.8	0.702	234	$\theta = 1,\ m = 2$	1.1	0.968	68
$\theta = 5,\ \rho = 0.2$	6.2	0.470	230	$\theta = 5,\ m = 10$	0.92	1	–
$\theta = 5,\ \rho = 0.5$	4.6	0.654	197	$\theta = 5,\ m = 5$	0.95	1	–
$\theta = 5,\ \rho = 1$	2.1	0.990	73	$\theta = 5,\ m = 2$	0.85	1	–
$\theta = 20,\ \rho = 0.2$	5.9	0.478	193	$\theta = 20,\ m = 10$	0.98	1	–
$\theta = 20,\ \rho = 0.5$	3.9	0.778	126	$\theta = 20,\ m = 5$	0.89	1	–
$\theta = 20,\ \rho = 1$	0.7	1	–	$\theta = 20,\ m = 2$	0.5	1	–

never as many infected as in the major outbreaks when no vaccination was in place. Still, several of the outbreaks have quite a number of infected. For example, the proportion of major outbreaks is $1 - \pi^{(1)} = 0.010$, indicating that there still is a small risk of getting many infected for these particular parameter values. This is in agreement with the computed reproduction number $R_{\theta,\rho} = 2.1$, which is above threshold. Figure $2B$ shows the corresponding results for the second vaccination model with the same detection rate $\theta = 5$ and having $m = 10$ as bound on the maximum number of individuals one person can infect (so the only difference between the two vaccination models is that in model 2 individuals can at most infect 10 friends). All outbreaks are now "minor" ($\pi^{(2)} = 1$), in fact no outbreak had more than 5 infected people, which shows that the second vaccination model is efficient. This too agrees with the computed reproduction number $R_{\theta,m} = 0.92$, below threshold. Hence, we note from Figure 2 that the second vaccination model is more effective when compared with the first vaccination model. However, both vaccination models have big impact in stopping the disease from spreading when compared with Figure 1 for epidemics without vaccination.

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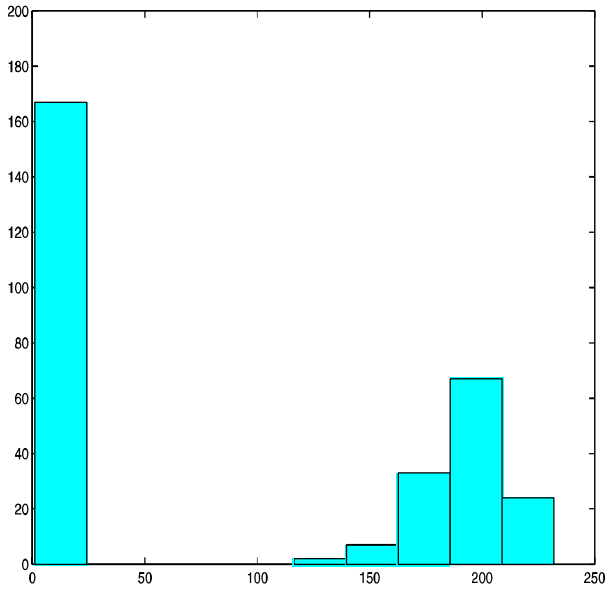


Figure 1: Histogram of final sizes for 500 simulations of an epidemic without vaccination in a heavy-tail degree distribution with $n = 1000$ individuals and $R_0 = 7.2 > 1$, i.e above threshold, indicating that there is a major outbreak.

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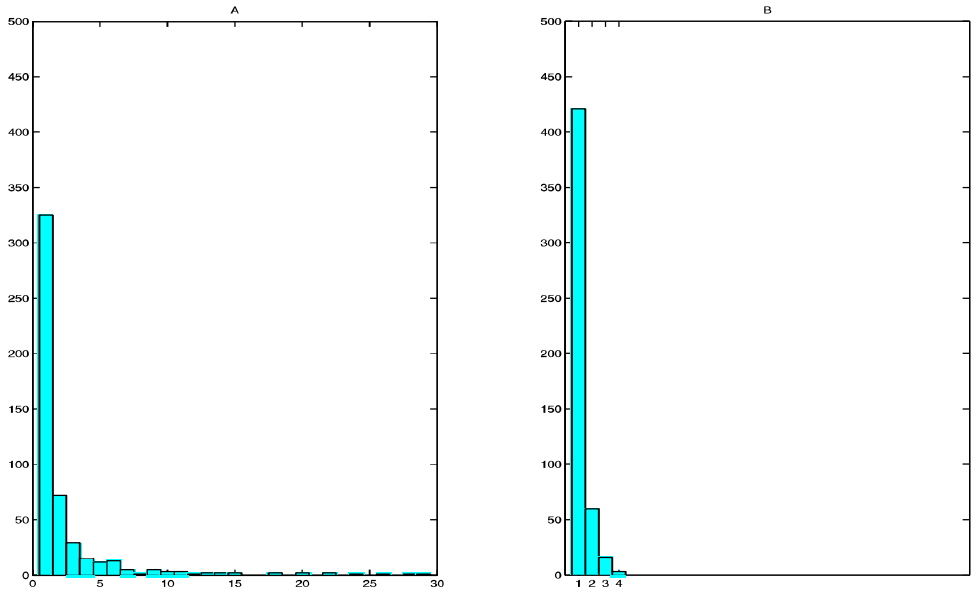


Figure 2: Histograms of outbreak sizes for 500 simulations with $n = 1000$ individuals in the heavy-tail degree distribution. In (A) (left) we implemented the first vaccination model with $\theta = 5$ and $\rho = 1$, whereas in (B) (right) is the second vaccination model with $\theta = 5$ and $m = 10$.

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5 Discussion

In the present paper we have studied two vaccination models implemented during the early stages of an epidemic outbreak. We used contact tracing as part of epidemic control in the social networks. The aim was to incorporate and determine the role of delay time, from the point at which an individual becomes infectious until he is detected and his friends are vaccinated. The reproduction numbers for the two vaccination models were derived and compared through simulation and some numerical examples when having Poisson and a heavy-tail degree distribution in the social network.

It was shown that the first vaccination model was less effective in preventing disease spread as compared to the second model where also a maximal bound on the number of people one person can infect was assumed. The second vaccination model is more effective since the effect of individuals with many friends (super-spreaders) is reduced. Since super-spreaders are more frequent when having heavy-tail degree distributions, the difference between the models is greater for such degree distributions.

In general, if two different control strategies are compared, the first having a larger reproduction number than the second, one would typically expect the first strategy to have a smaller extinction probability and larger mean number of among major outbreaks. We note the two vaccination models treated in the present paper illustrate that this is not true in general. For instance, when $\theta = 20$ and $\rho = 0.5$ model 1 in Table 2 yields the reproduction number $R_{\theta,\rho} = 3.9$, and when $\theta = 1$ and $m = 10$ yield the reproduction number $R_{\theta,m} = 3.5$. Still, the proportion of minor outbreak is $\pi^{(1)} = 0.778$ for model 1 and $\pi^{(2)} = 0.694$ for model 2, and the average size among major outbreaks is $\mu^{(1)} = 126$ for model 1 and $\mu^{(2)} = 172$ for model 2. This illustrates that there is no direct relation between reproduction numbers and probabilities of major outbreaks or of their sizes, but only that their relation to the critical value of 1 determines whether or not major outbreaks are at all possible.

The model for the social network can be made more realistic in several ways. One underlying assumption is that an individual chooses his/her friends independently of each other. In real life there might be some assortative mixing (see [34]), meaning that individuals with many (few) friends are connected to individuals with many (few) friends. Also many real world networks show strong clustering, implying that there is positive probability that two individuals with a common friend are also friends. However, to include these features in the social network would make the analysis of the epidemic and comparison of the vaccination models much harder. Another assumption is that the social network is considered fixed over time. This is appropriate for diseases with short infectious periods but for diseases with long infectious period, a dynamic social network would be preferred. The advantage with a fixed network is that the expression of the reproduction number can be derived and the performance of the vaccination models can be analyzed and compared in more detail.

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An important question not addressed in this paper is to study the effects of different distributions for the infectious period and delay time, and to introduce a latency period. To derive expressions for the reproduction number in such situations after the implementation of our vaccination models are important problems. Other interesting problems could be to numerically determine the probability of major outbreaks and the final size when these vaccination models are implemented and to compare the theoretical results with the corresponding simulation results in Table 1 and Table 2. We still believe the findings of the present paper give some insight into possible effects of different vaccination strategies and that the qualitative conclusions may be valid in more complex and realistic settings.

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