ELSEVIER

Mathematical Biosciences 164 (2000) 39–64

Mathematical Biosciences

www.elsevier.com/locate/mbs

Contact tracing in stochastic and deterministic epidemic models

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Received 21 May 1999; received in revised form 9 November 1999; accepted 18 November 1999

Abstract

We consider a simple unstructured individual based stochastic epidemic model with contact tracing. Even in the onset of the epidemic, contact tracing implies that infected individuals do not act independent of each other. Nevertheless, it is possible to analyze the embedded non-stationary Galton–Watson process. Based upon this analysis, threshold theorems and also the probability for major outbreaks can be derived. Furthermore, it is possible to obtain a deterministic model that approximates the stochastic process, and in this way, to determine the prevalence of disease in the quasi-stationary state and to investigate the dynamics of the epidemic.

Keywords: Contact tracing; Epidemic models; Galton–Watson process; Threshold theorem; Quasi-stationary state

1. Introduction

A large part of mathematical epidemiology is concerned with the investigation of mechanisms and efficacy of control strategies against infectious diseases. Many types of control measures – such as vaccination or screening – are implemented at the population level and take little account of the impact of contact structure on the individual level. Often, only core groups are taken into consideration, e.g. commercial sex workers are intensely screened for sexually transmitted diseases (STDs). These kinds of control strategies are meanwhile quite well understood [1].

However, it is also possible to implement control measures at the individual level under consideration of the current contact structure and the history of previous contacts. For example,

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0025-5564/00/$ - see front matter
PII: S0025-5564(99)00061-9

vaccination is applied in the form of ring vaccination [2,3]: If an infectious individual is discovered, all individuals in a certain neighborhood are vaccinated. The idea of this procedure is that the prevalence of a disease is likely to be relatively high in the neighborhood of an index case. Hence, all individuals in the vicinity of the index case who are not yet infected are in danger of acquiring the infection and thus should be protected. Moreover, vaccinated individuals form a barrier for the infection and inhibit the spread into the remaining part of the susceptible population.

Also, screening has an individual based counterpart: Similar to ring vaccination, one tries to identify infected contacts of a known infected index case. Persons, who have been in contact with the index case, are notified and examined and in case they are found infected, also their contacts are traced. This procedure is common practice in the case of STDs [4], but also for tuberculosis [5,6] and infections that are spread by needle sharing [7].

In contrast to models that describe strategies at the population level, there are not very many tools to analyze models for individual based strategies. Simulation studies show the efficacy of contact tracing, especially for STDs [8–10], but in general it is not always clear whether to use contact tracing or not [5]. All in all, it is necessary to develop mathematical tools that can contribute to a better understanding of the effect of contact tracing.

The present work presents an approach for the analysis of a simple model. It is based on a stochastic model for a disease of SIRS-type. The population is assumed to be homogeneous, i.e. there is no core group or the like. With a certain rate infected individuals are identified and form the index cases. Once an index case is discovered, all persons who are known to have had a possibly infectious contact with the index case are also examined. With 'infectious contact' we mean a contact during which transmission of the infection has actually taken place. As usually not all persons who have had infectious contacts with the index case are known, only a fraction of all secondary cases caused by the index will be found by this procedure. In this article, we assume that a secondary case is discovered with a certain probability p_c. The parameter p_c describes the fraction of secondary cases caused by an index case that is discovered by contact tracing. This parameter will be central in the analysis.

The paper is organized as follows. In Section 2 we introduce the stochastic model. In Section 3, the stochastic process at the onset of an epidemic is analyzed and the reproduction number is determined. Note, that even at the onset of the epidemic individuals do not act independently, because of the contact tracing. Let $\tilde{R}_0$ be the reproduction number without contact tracing. One of the central results of our analysis is the fact that under certain conditions the critical value of p_c that reduces the effective reproduction number to 1 is $1 - 1/\tilde{R}_0$. If the fraction of secondary cases p_c that are found by contact tracing is above this threshold, the epidemic dies out, while if it is below this threshold major outbreaks are possible. In Section 4, the full epidemic process is investigated and the prevalence of infection in the quasi-stationary state is determined. Furthermore, an approximation of the stochastic process by ordinary differential equations is obtained on the basis of heuristic arguments; simulations show a good agreement with the stochastic process.

2. Basic model

We consider a stochastic SIRS-model without demographic processes (birth and death). Let the population size be N, S the number of susceptible, I the number of infected and R the number of

removed individuals. Hence, $S + I + R = N$. The model describes five different possible transitions between states. The first three – infection, recovery and loss of immunity – are rather standard; the remaining two are screening and contact tracing. In order to incorporate contact tracing, some infected persons must be recognized as being infected. This can be the case, when an infected individual consults a doctor who diagnoses the infection, or, more systematically, if there a screening program designed to identify infected persons. Once an infected individual is found, he/she is questioned about his/her contacts within a certain critical time period. This time interval should cover the infectious period of the person under consideration. However, this period will vary widely, depending not only on the kind of infection and the kind of contacts that should be traced, but also on the effort one wants to spend on contact tracing. A person who has been at risk of getting infected will be notified and asked to come for examination and treatment.

In the following we summarize the model assumptions.

Infection: We assume random mixing. A susceptible individual becomes infected at rate $\beta I/N$. We assume that an infected individual is infectious and vice versa.

Recovery: An infected person looses his/her infectivity at rate α and becomes removed (immune).

Loss of immunity: An immune person looses the protection at rate γ and is susceptible again. We assume that immune persons cannot be distinguished from susceptible persons without an expensive test. Hence, with respect to contact tracing, susceptibles and immunes are not distinguished, i.e. we assume that contacts of immunes are not traced. We will see later that this assumption can be changed easily.

Screening: The population is screened at rate σ. If an infected individual is found, he/she is treated and becomes immune. In general, the rates of loss of immunity are different for treated and spontaneously recovered individuals. However, for the sake of simplicity we assume those rates to be equal.

Contact tracing: The implementation of contact tracing is the interesting part of the model. If an infected individual is detected, he/she is asked to identify persons with whom he/she has had possibly infectious contacts. Those persons are then notified and offered a medical examination and treatment. In order to describe this procedure mathematically, the individuals of the population are numbered by $1, \ldots, N$. We call these numbers the id-number of an individual. An infected individual corresponds to a tuple (x_1, x_2) with $x_1, x_2 \in \{1, \ldots, N\}, x_1 \neq x_2$. The first number is the id of the infected individual itself, the second the id of the person from whom the individual has been infected. In this way, infected persons form a directed graph. The nodes correspond to infected individuals, an edge goes from individual i to individual j, if j has been infected by i (Fig. 1).

In general, this graph is not simply connected, because removed persons are not members of the graph any more and destroy in this way its connectedness. Now we assume that an individual i_0 is observed. This person is called the index case. The index case will remember only a certain fraction of his/her contacts. Thus an individual infected by the index case has a certain probability p_c to be traced as does the person who infected the index case. If one of these neighboring individuals are actually discovered, then he/she forms again an index case, and recursively the tracing process starts anew. As an example, lets consider the connected component of the graph of infected individuals shown in Fig. 1. Here, person 7 infected individual 5, who in turn infected individuals 8, 33 and 11 a.s.o. We assume that individual 8 is discovered and forms the first index case. At this point of time, all individuals shown in Fig. 1 are assumed to be still infectious. Then

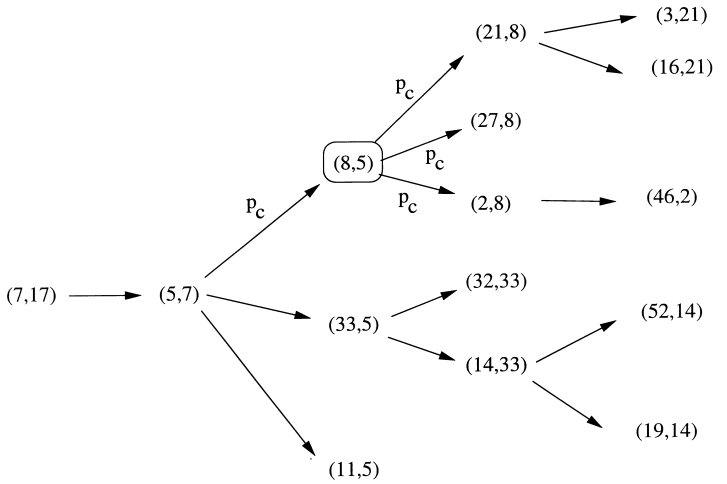

Fig. 1. The directed graph of infected individuals. The pairs of numbers (x_1, x_2) attached to the nodes of the graph denote the id of the individual corresponding to that node (x_1) and the id of the individual by whom he/she has been infected (x_2). An edge pointing from node x_2 to node x_1 then describes the direction of transmission. If an infected individual is discovered (say the one in the box), then every individual connected to this index individual by an edge (regardless of the direction of this edge) will be discovered with probability p_c. If an infected contact is found, he/she will be the starting point for a next step of contact tracing, thus creating a a snowball effect.

each of individuals 21, 27, 2 and 5 have the probability p_c to be discovered. In a concrete realisation of the stochastic process one finds, say, individuals 27 and 5. These two persons are new index cases. Since individual 27 has no further neighbors, here the tracing process stops. This is different in case of individual 5: Now, each of the individuals 33, 11 and 7 have the probability p_c to be discovered. In this way, the tracing process runs recursively over the connected component of the graph of infected persons.

Of course, it is possible that contacts between the index case and another individual took place without transmission, but that this individual was infected by another source. When this infected individual is found by contact tracing another branch of the infectious graph is discovered by chance. This event happens especially if the prevalence of disease is very high. For the sake of simplicity we exclude this effect. Furthermore, it is assumed that contact tracing happens instantaneously, i.e. that the time scale of infection is much slower than that for contact tracing.

In the stochastic model, there are two kinds of dependencies between individuals: The first is infection, the second the contact tracing. If there is no screening, $\sigma = 0$, or the probability for contact tracing is zero, $p_c = 0$, then the above model coincides with the usual stochastic SIRS-model [11].

An interesting generalization is the case of variable coefficients. In general, the infectivity of an individual will depend on the time since infection; also, recovery or the success of screening might not be well described by a Poisson process with a constant rate. Furthermore, the probability to find a person who has had contact with an index case may depend on the time between this contact and the discovery of the index case. In the following, we first consider constant rates. However, it will be possible to generalize the results to variable rates. Let a be the age of infection for an individual, i.e. the time since the individual was infected. We may replace the constant rate

β by a function $\beta(a)$. In this way it is possible to include a latent period or relatively complex infectivity functions like the one that is assumed reasonable for HIV infection. Also the recovery rate may depend on a, allowing for the typical time the immune system needs to fight the infection successfully. A dependence of the screening rate σ on a may reflect the fact that after a certain time symptoms may appear that make it easier to recognize a person as being infected. The tracing probability should not directly depend on the age of infection but rather on the time passed since a contact took place. If one questions an index case only about the contacts of the last month, then p_c becomes zero for all contacts that occurred earlier. Furthermore, a person may not remember all contacts that happened a longer time ago. Also this fact can be taken into account by a tracing probability p_c that depends on the time since a contact occurred.

3. Onset of the epidemic

At the onset of the epidemic there are only few infected individuals. Since we assume random mixing, it is not likely that a contact occurs between two infected individuals. Therefore, we can assume that the transmission process is well described by an independent branching process [12]. An infected individual infects others with a rate $\beta S/N$ which approximately equals β at the onset of the epidemic. However, if there is contact tracing, infected individuals do not act independently from each other even at the onset of the epidemic. In this section, the resulting branching process will be analyzed.

It is not convenient to treat the time dependent stochastic process, rather we will investigate the embedded Galton–Watson process. We adopt the following notations introduced by Hethcote and Yorke [13]: A person who infects another person will be called an 'infector', while the one, who gets infected is called an 'infectee', or, an 'infectee of the first generation'. The 'infectees of the second generation' are all those who are infected by an infectee of the first generation, and so on. The Galton–Watson process here is not a stationary Markov process, because the distribution of the number of infectees an infected individual produces depends on his/her generation: The primary infected individual can be traced only via his/her infectees, while infectees of the first generation can be also traced via their infector (i.e. the primary infected). With increasing generation, the size of the component of the graph of infected persons via which an infected individual may be traced increases, and eventually converges to a maximum size.

Let $f_i(x)$ be the probability generating function for generation i, i.e.

$$f_i(x) = \sum_{j=0}^{\infty} x^j P \text{ (to have } j \text{ individuals in generation } i).$$

There are functions $g_i(x)$, such that

$$f_i(x) = g_{i-1}(f_{i-1}(x)).$$

For a stationary Galton–Watson process $g_i(x) = g(x)$ for all $i \in \mathbb{N}$. In our case, the process will be asymptotically stationary,

$$g_i(x) \to g_\infty(x) \quad \text{for } i \to \infty.$$

We define the reproduction number for generation i as

$$R_i := \frac{\mathrm{d}}{\mathrm{d}x} g_i(x)|_{x=1}.$$

Then $R_i \to R_\infty$ for $i \to \infty$. There are three generic cases.

1. $R_\infty > 1$. The branching process is supercritical, the epidemic takes off with a certain probability and then grows without bounds.
2. $R_0 > 1 > R_\infty$. The number of infected individuals increases for the first few generations, but the branching process is asymptotically subcritical, and thus the epidemic breaks down and dies out in the long run.
3. $R_0 < 1$. The branching process is subcritical, there are no major outbreaks.

In the following we will determine the reproduction numbers R_i. By inspecting R_∞ one can decide whether the branching process is asymptotically sub- or supercritical, i.e. whether a major outbreak can occur and whether the epidemic growth is persistent over many generations. Therefore R_∞ is also called effective reproduction number.

For technical reasons, we distinguish three cases with respect to tracing. In the first case ('backward tracing') it is assumed that infectors are traced via their infectees, but not vice versa. The second case ('forward tracing') is just the inverse: infectees are traced via their infectors, but no infectors via their infectees . The third case ('full tracing') is a combination of the first two: infectors may be traced via infectees and vice versa. In reality, forward and backward tracing are usually hard to distinguish, because one does not know who in a pair of infected persons has infected whom. To increase the clarity of the analysis, however, the two cases are better handled separately.

3.1. Backward tracing

In this section we consider only backward tracing, i.e. an infected individual can only be traced via his/her infectees, but not via his/her infector. We will use the following formulation: we say an infected individual is directly observed, when he/she is found due to the screening program and not by contact tracing; we say that an infected individual is observed, if he/she is found either due to the screening program or by contact tracing. If an infected individual is observed, he/she is assumed to receive treatment and move into the removed class.

Definition 3.1. Let $\kappa_i^-(a)$ be the probability for an individual of generation i to be infectious at age a of infection.

Proposition 3.2. *The probability to be infectious at age a of infection does not depend on the generation. Furthermore,* $\kappa_i^-(0) = 1$.

Proof. Since no infectee can be found via the infector, the probability to be traced is not affected by the number of generations. Hence, $\kappa_i^-(a) = \kappa_0^-(a)$ for $i = 1, 2, 3, \ldots$ □

Proposition 3.3. *The probabilities* $\kappa_i^-(a)$ *satisfy the integro-differential equation*

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa_i^-(a) = -\kappa_i^-(a)\left[\sigma + \alpha + \beta p_c(1-\kappa_i^-(a)) - \beta p_c \alpha \int_0^a \kappa_i^-(c)\,\mathrm{d}c\right], \tag{1}$$

$$\kappa_i^-(0) = 1.$$

Proof. The starting point for deriving an equation for $\kappa_0^-(a)$ is

$$\begin{aligned}\kappa_0^-(a+\Delta a) &= \kappa_0^-(a) - P\ (\text{Removal in the age classes } [a, a+\Delta a))\\ &= \kappa_0^-(a) - \kappa_0^-(a)P\ (\text{Removal in } [a, a+\Delta a)\,|\,\text{Individual infective at age } a).\end{aligned}$$

The aim is to determine the probability for a transition to the removed class in the interval $[a, a+\Delta a)$ for an infected individual with age of infection a. Three types of events may result in removal:
1. The individual recovers spontaneously with probability $\alpha\Delta a + o(\Delta a)$.
2. The individual is directly observed with probability $\sigma\Delta a + o(\Delta a)$.
3. The individual is traced, because one of his/her infectees has been observed.

The probability for point 3 above can be obtained as follows:

Step 1: We assumed that the infected individual under consideration (the infector) has age of infection a and is still infectious. How big is the probability for an infectee with age of infection in the interval $[c, c+\Delta a)$ to exist? Concerning this interval, two things may have happened: The infector may have produced an infection or not. If the infector produced an infection, this infectee can be still infectious or can be removed. We determine the probabilities for these events.

- The probability that the infector produced an infection in the time interval $[a-c-\Delta a, a-c]$ is $\beta\Delta a + o(\Delta a)$ and thus the probability not to produce an infection amounts to $1-\beta\Delta a$ (of course we assume $c+\Delta a < a$).
- If there is an infectee, the probability for her/him to be still infectious is $\kappa_0^-(c)$.
- If the infectee is removed (i.e. spontaneously recovered or discovered either by direct observation or by contact tracing), then the infector is *not* traced with some probability $\tilde{p}(a,c)$.

Hence, the probability for an infected individual with age of infection a to have an infectious infectee with age of infection between c and $c+\Delta a$ is

$$\frac{(\beta\Delta a)\kappa_0^-(c)}{(\beta\Delta a)\kappa_0^-(c) + (\beta\Delta a)\tilde{p}(a,c)(1-\kappa_0^-(a)) + (1-\beta\Delta a)} + o(\Delta a) = (\beta\Delta a)\kappa_0^-(c) + o(\Delta a).$$

Step 2: An infectious infectee with age of infection c has the probability

$$\frac{-\kappa_0^{-\prime}(c)}{\kappa_0^-(c)}\Delta a + o(\Delta a)$$

to move to the removed class in the age interval $[c, c+\Delta a)$ (prime denotes the derivative with respect to the age of infection). Reasons for removal are spontaneous recovery or observation. Only if the infectee is observed, his/her infector may be traced. The probability of spontaneous recovery is $\alpha\Delta a + o(\Delta a)$, hence

$$\begin{aligned}&P\ (\text{Observation of an individual in } [c, c+\Delta a)\,|\,\text{Individual is infective at age } c)\\ &\quad= \left(\frac{-\kappa_0^{-\prime}(c)}{\kappa_0^-(c)} - \alpha\right)\Delta a + o(\Delta a).\end{aligned}$$

The probability for the infector to be traced via an infectee who is in the age interval $[c, c+\Delta a)$ is therefore

$$p_c\Big[-(\Delta a)^2\beta\kappa_0^{-\prime}(c) - (\Delta a)^2\beta\alpha\kappa_0(c)\Big] + o(\Delta a^2).$$

Step 3: By now we know the probability to be traced via an infectee with a specific age. The probability to be traced via any infectee yields

$$p_c\Delta a\int_0^a \left[-\beta\kappa_0^{-\prime}(c)-\beta\alpha\kappa_0^-(c)\right]\mathrm{d}c+\mathrm{o}(\Delta a)=\Delta a\left[\beta p_c(1-\kappa_0^-(a))-\beta p_c\alpha\int_0^a \kappa_0^-(c)\,\mathrm{d}c\right]+\mathrm{o}(\Delta a).$$

Therefore we conclude that $\kappa_0^-(s)$ satisfies (1). The assertion follows with $\kappa_i^-(a)=\kappa_0^-(a)$. □

Remark 3.4. In general, the differential equation for $\kappa_0^-(a)$ cannot be solved analytically. In Fig. 2 we compare numerical solutions of $\kappa_0^-(a)$ with simulations of the stochastic process and find agreement. A trivial case, where it is possible to solve (1) explicitly, is $p_c=0$, i.e. if there is no contact tracing, and individuals either recover spontaneously at rate α or are observed at rate σ. Then

$$\kappa_0^-(a)=\mathrm{e}^{-(\alpha+\sigma)a}.$$

Since there is no dependency between different individuals, the probability to be infectious decreases exponentially with age. The reproduction numbers R_i^- (which do not depend on i because the probabilities κ_i^- do not depend on i) read in this case

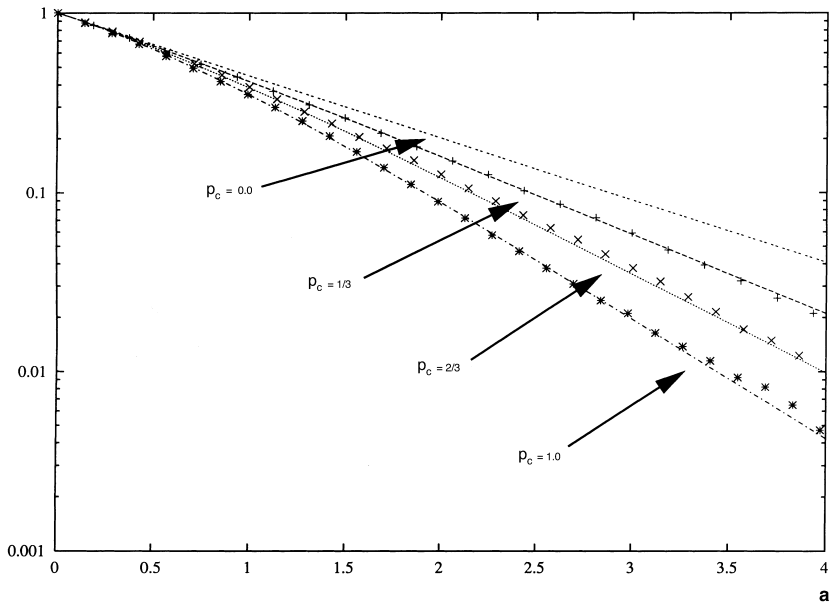

Fig. 2. Probability to be infectious by age of infection. The lines are computed by solving Eq. (1), the points are obtained by simulation of the stochastic process. The four different curves correspond to $p_c=0.0$, $p_c=1/3$, $p_c=2/3$ and $p_c=1.0$. The other parameter values are $\beta=1.0$/time unit, $\sigma=0.5$/time unit, $\alpha=0.3$/time unit. For the simulation of the stochastic process 10000 runs have been performed.

$$R_i^- = \frac{\beta}{\alpha+\sigma}.$$

3.1.1. No natural recovery

In this section we assume that there is no spontaneous recovery, i.e. $\alpha = 0$. An individual is removed only after observation.

Proposition 3.5. *Let* $\alpha = 0$. *Then*,

$$\kappa_0^-(a) = \frac{1 + p_c\tilde{R}_0}{p_c\tilde{R}_0 + e^{\sigma(1+p_c\tilde{R}_0)a}}$$

with $\tilde{R}_0 := \beta/\sigma$. *Furthermore, the reproduction number of generation i is*

$$R_i = \int_0^\infty \beta\kappa_0(a)\,da = \frac{1}{p_c}\log(1 + p_c\tilde{R}_0).$$

Proof. If $\alpha = 0$, then the integro-differential equation (1) becomes an ordinary differential equation

$$\frac{d}{da}\kappa_0^-(a) = -\kappa_0^-(a)\left[\sigma + \beta p_c(1 - \kappa_0^-(a))\right], \quad \kappa_0^-(0) = 1.$$

This is a logistic equation that can be solved explicitly

$$\kappa_0^-(a) = \frac{1 + p_c\tilde{R}_0}{p_c\tilde{R}_0 + e^{\sigma(1+p_c\tilde{R}_0)a}}.$$

The reproduction number is defined by

$$R_i^- = \int_0^\infty \beta\kappa_0^-(a)\,da = \frac{1}{p_c}\log(1 + p_c\tilde{R}_0) \quad \text{for } i = 0, 1, 2, \ldots \qquad \square$$

Remark 3.6. Since $p_c \in [0, 1]$, we obtain

$$\tilde{R}_0 \geqslant R_i^- \geqslant \log(1 + \tilde{R}_0).$$

It is in general not possible to reduce the effective reproduction number below one with backward tracing only: On the one hand, a supercritical branching process grows exponentially per generation. On the other hand, if an individual of the ith generation is observed, at most i infected individuals can be found by backward tracing. This number grows only linearly and is, compared with the exponential growth of the branching process, in general not sufficient to stop the branching process, even if we choose $p_c = 1$.

3.1.2. Variable parameters

If the parameter α, β, σ depend on age of infection and p_c on the time since contact, it is still possible to find an equation describing the probability to be infective at age a, $\kappa_i^-(a)$. With similar arguments as above we arrive at the following proposition.

Proposition 3.7. *For variable parameters we obtain*

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa_0^-(a) = -\kappa_0^-(a)\left\{\sigma(a)+\alpha(a)-\int_0^a p_c(a-c)\left[\alpha(c)\beta(c)\kappa_0^-(c)+\beta(c)\frac{d}{dc}\kappa_0^-(c)\right]\mathrm{d}c\right\}, \qquad (2)$$
$$\kappa_0^-(0) = 1.$$

3.2. Forward tracing

We now concentrate on the second case: Infectees are found via their infectors, but not vice versa. Again, we investigate the probability for an individual to be infectious at age a of infection.

Definition 3.8. Let $\kappa_i^+(a)$ be the probability for an individual of generation i to be infectious at age a of infection.

The zeroth generation, i.e. the primary infected case, cannot be traced at all. The only way he/she looses the infectivity is to recover spontaneously or to be observed directly. Hence the following proposition holds.

Proposition 3.9. *For the zero*th *generation we obtain*

$$\kappa_0^+(a) = \mathrm{e}^{-(\sigma+\alpha)a}. \qquad (3)$$

In order to derive a recursion formula for higher generations, we investigate the probability that an infected individual is infectious at age of infection a under the condition that his/her infector had age of infection b at the time transmission took place. This approach is convenient, since the probability for the infectee to be traced depends on the age of infection b of the infector.

Proposition 3.10. *Let* $i > 0$ *and*

$$\kappa_i^+(a\,|\,b) := P\,(\textit{Individual is infectious with age of infection } a \mid \textit{Infector has age of infection } a+b).$$

Then $\kappa_i^+(a\,|\,b)$ *satisfies*

$$\kappa_i^+(a\,|\,b) = (1-p_c)\kappa_0^+(a) + p_c\frac{\kappa_{i-1}^+(a+b)}{\kappa_{i-1}^+(b)}\kappa_0^+(a) + \alpha p_c\frac{\int_0^a \kappa_{i-1}^+(b+c)\,\mathrm{d}c}{\kappa_{i-1}^+(b)}\kappa_0^+(a).$$

Proof. If the individual under consideration has not been traced so far, his/her probability to be infectious is the same as that for the zeroth generation $\kappa_0^+(a)$. This probability is decreased by tracing via the infector. Hence, to obtain $\kappa_i^+(a\,|\,b)$, $\kappa_0^+(a)$ is multiplied by the probability not to be traced via the infector

$$\kappa_i^+(a\,|\,b) = [P(\text{Infector infective}) + P(\text{Infector spontaneously recovered}) + (1-p_c)P(\text{Infector observed})]\kappa_0^+(a). \tag{4}$$

We now determine the probability for the infector to be observed (either by screening or by contact tracing) in the age interval $[b, b+a)$.

- The probability for the infector to be infectious at a given age $b+c \in [b, b+a)$ is $\kappa_{i-1}^+(b+c)/\kappa_{i-1}^+(b)$.
- The probability to be observed in the interval $[b+c, b+c+\Delta a)$ is

$$\frac{\kappa_{i-1}^+(b+c)}{\kappa_{i-1}^+(b)}\left(\frac{-\kappa_{i-1}^{+\prime}(b+c)}{\kappa_{i-1}^+(b+c)} - \alpha\right)\Delta a + o(\Delta a) = \left(\frac{-\kappa_{i-1}^{+\prime}(b+c)}{\kappa_{i-1}^+(b)} - \alpha\frac{\kappa_{i-1}^+(b+c)}{\kappa_{i-1}^+(b)}\right)\Delta a + o(\Delta a).$$

- Therefore the probability for the infector to be observed in the age interval $[b, b+a)$ reads

$$\int_0^a \left(\frac{-\kappa_{i-1}^{+\prime}(b+c)}{\kappa_{i-1}^+(b)} - \alpha\frac{\kappa_{i-1}^+(b+c)}{\kappa_{i-1}^+(b)}\right)\mathrm{d}c = 1 - \frac{\kappa_{i-1}^+(a+b)}{\kappa_{i-1}^+(b)} - \alpha\frac{\int_0^a \kappa_{i-1}^+(b+c)\,\mathrm{d}c}{\kappa_{i-1}^+(b)}$$

and the probability to be spontaneously recovered in the age interval $[b, a+b)$

$$\alpha\frac{\int_0^a \kappa_{i-1}^+(b+c)\,\mathrm{d}c}{\kappa_{i-1}^+(b)}.$$

The formula for $\kappa_i^+(a\,|\,b)$ can be obtained by substituting these probabilities into (4). □

Proposition 3.11. *For the probabilities $\kappa_i^+(a)$, the recursion formula*

$$\kappa_i^+(a) = \kappa_0^+(a)\left\{1 - p_c\left[1 - (1+\alpha a)\frac{\int_a^\infty \kappa_{i-1}^+(b)\,\mathrm{d}b}{\int_0^\infty \kappa_{i-1}^+(b)\,\mathrm{d}b} - \alpha\frac{\int_0^a b\kappa_{i-1}^+(b)\,\mathrm{d}b}{\int_0^\infty \kappa_{i-1}^+(b)\,\mathrm{d}b}\right]\right\} \tag{5}$$

holds.

Proof. The probability density for an individual of the ith generation to be infected by a person who has age of infection b at the time of transmission is

$$\beta\kappa_{i-1}^+(b)\Big/\int_0^\infty \beta\kappa_{i-1}^+(b)\,\mathrm{d}b.$$

Hence we obtain

$$\begin{aligned}\kappa_i^+(a) &= \frac{\int_0^\infty \kappa_i^+(a\,|\,b)\,\kappa_{i-1}^+(b)\,\mathrm{d}b}{\int_0^\infty \kappa_{i-1}^+(b)\,\mathrm{d}b}\\ &= (1-p_c)\kappa_0^+(a) + p_c\kappa_0^+(a)\frac{\int_0^\infty \kappa_{i-1}^+(a+b)\,\mathrm{d}b}{\int_0^\infty \kappa_{i-1}^+(b)\,\mathrm{d}b} + \alpha p_c\kappa_0^+(a)\frac{\int_0^\infty \int_0^a \kappa_{i-1}^+(b+c)\;\mathrm{d}c\,\mathrm{d}b}{\int_0^\infty \kappa_{i-1}^+(b)\,\mathrm{d}b}.\end{aligned}$$

The assertion follows with

$$\int_0^\infty \kappa_{i-1}^+(a+b)\,\mathrm{d}b = \int_a^\infty \kappa_{i-1}^+(b)\,\mathrm{d}b$$

and

$$\int_0^\infty \int_0^a \kappa_{i-1}^+(b+c)\,\mathrm{d}c\,\mathrm{d}b = \int_0^a b\kappa_{i-1}^+(b)\,\mathrm{d}b + a\int_a^\infty \kappa_{i-1}^+(b)\,\mathrm{d}b. \qquad \square$$

Remark 3.12. In Fig. 3 we show the graphs of $\kappa_i^+(a)$ for $i = 0,\ldots,3$ and $p_c = 0$, $p_c = 1/3$, $p_c = 2/3$ and $p_c = 1.0$. The bigger the p_c, slower is the convergence to $\kappa_\infty^+(a)$. For $p_c = 0.0$, all $\kappa_i^+(a)$ are the same. For $p_c = 1/3$, $\kappa_i^+(a)$ practically does not change any more for $i \geqslant 1$, and in the case of $p_c = 2/3$ for $i \geqslant 2$.

3.2.1. No natural recovery

Also for forward tracing, the case without spontaneous recovery ($\alpha = 0$) is considerably easier to handle than the case $\alpha > 0$. The reason is that without spontaneous recovery the tree of infected individuals remains connected, and in principle every infected individual can be traced.

The probabilities to be infectious at age of infection a for the zeroth and first generation are easy to compute.

Generation 0: The only possibility to lose the infection for the primary infected is detection by screening, i.e.

$$\kappa_0^+(a) = \mathrm{e}^{-\sigma a}.$$

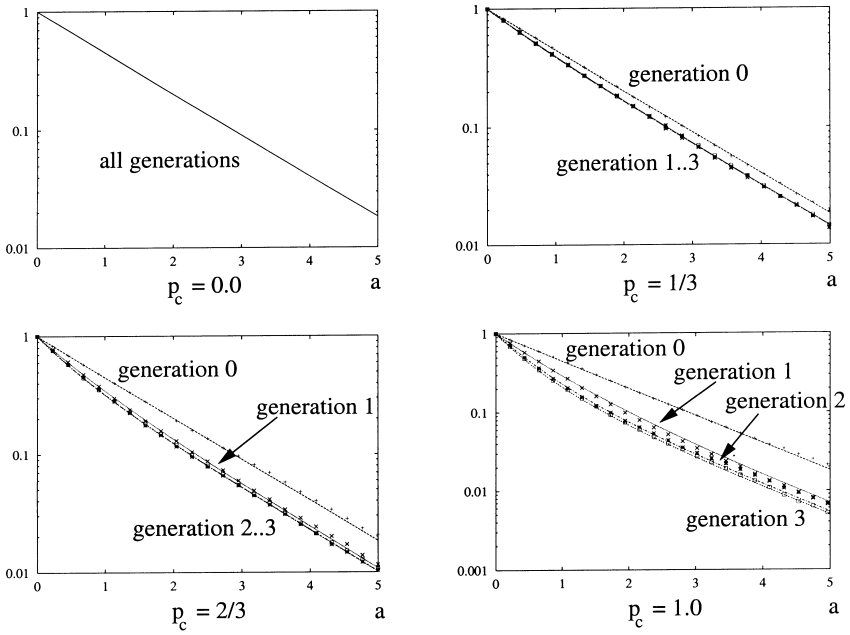

Fig. 3. Probability to be infectious by age of infection for generations 0–3. The lines are obtained by solving Eq. (5), the points by simulation of the stochastic process (for $p_c = 0$, no simulations of the stochastic process have been performed). The four panels of the figure correspond to $p_c = 0.0$, $p_c = 1/3$, $p_c = 2/3$ and $p_c = 1.0$. The other parameter values are $\beta = 1.0$/time unit, $\sigma = 0.5$/time unit, $\alpha = 0.3$/time unit. For the stochastic process we performed 10 000 runs.

Generation 1: Plugging κ_0^+ into the recursion formula (5) leads to

$$\kappa_1^+(a) = \mathrm{e}^{-\sigma a}(1 - p_c(1 - \mathrm{e}^{-\sigma a})).$$

The first term on the right-hand side of this formula describes the probability to be infectious subject to screening only. This probability is reduced by the factor given in the second term, which describes the probability *not* to be found by tracing via the infector.

The form of $\kappa_0^+(a)$ and $\kappa_1^+(a)$ suggests that $\kappa_i^+(a)$ is a finite power series of $\exp(-\sigma a)$. With induction over i one obtains the following proposition.

Proposition 3.13. *The probability for an infected individual of generation i to be infectious at age of infection a can be expressed as*

$$\kappa_i^+(a) = \sum_{j=1}^{i+1} \theta_j^{(i)} \mathrm{e}^{-j\sigma a},$$

where the coefficient $\theta_1^{(0)}$ *is given by*

$$\theta_1^{(0)} = 1$$

and the coefficients of the following generations satisfy the recursion formula

$$\theta_1^{(i)} = 1 - p_c, \tag{6}$$

$$\theta_j^{(i)} = p_c \frac{\theta_{j-1}^{(i-1)}}{(j-1)\sum_{k=1}^{i} \frac{1}{k}\theta_k^{(i-1)}} \quad \text{for } j = 2, \ldots, i+1. \tag{7}$$

Whether contact tracing can be sufficient to stop the growth of the epidemic can be determined by inspecting the limit $i \to \infty$ of κ_i^+.

Proposition 3.14. *Let* $\kappa_\infty^+(a)$ *be the pointwise limit of* $\kappa_i^+(a)$,

$$\kappa_\infty^+(a) := \lim_{i\to\infty} \kappa_i(a) \quad \text{for } a \geqslant 0$$

Then,

$$\kappa_\infty^+(a) = \mathrm{e}^{-\sigma a}(1 - p_c)^{(1-\mathrm{e}^{-\sigma a})} \tag{8}$$

and the corresponding limit of the reproduction numbers R_i^+ *reads* (*with* $\tilde{R}_0 := \beta/\sigma$)

$$R_\infty^+ := \lim_{i\to\infty} R_i^+ = \frac{-p_c}{\log(1 - p_c)} \tilde{R}_0.$$

Proof. The function κ_∞^+ is a fixed point of the recursion formula (5), i.e.

$$\kappa_\infty^+(a) = \left[(1 - p_c) + p_c \frac{\int_a^\infty \kappa_\infty^+(b)\,\mathrm{d}b}{\int_0^\infty \kappa_\infty^+(b)\,\mathrm{d}b}\right] \kappa_0^+(a).$$

Let $\zeta := \int_0^\infty \kappa_\infty^+(b)\,\mathrm{d}b$. Dividing by κ_0^+ and differentiating with respect to a yields

$$\frac{\mathrm{d}}{\mathrm{d}a}\left(\frac{\kappa_\infty^+(a)}{\kappa_0^+(a)}\right) = -\frac{p_c}{\zeta}\kappa_0^+(a)\left(\frac{\kappa_\infty^+(a)}{\kappa_0^+(a)}\right), \quad \frac{\kappa_\infty^+(0)}{\kappa_0^+(0)} = 1.$$

Hence

$$\kappa_\infty^+(a) = \kappa_0^+(a)\,\exp\left\{-\frac{p_c}{\zeta}\int_0^a \kappa_0^+(\tau)\,\mathrm{d}\tau\right\} = \mathrm{e}^{-\sigma a}\,\exp\left\{-\frac{p_c}{\zeta\sigma}(1-\mathrm{e}^{-\sigma a})\right\}. \tag{9}$$

For ζ we obtain a fixed point equation,

$$\zeta = \int_0^\infty \kappa_\infty^+(a)\,\mathrm{d}a = \frac{\zeta}{p_c}\,\mathrm{e}^{-(p_c/\zeta\sigma)}\left(\mathrm{e}^{(p_c/\zeta\sigma)}-1\right)$$

and thus

$$\zeta = \frac{-p_c}{\sigma\,\log(1-p_c)}.$$

Using this expression for ζ in (9), we obtain (8). Furthermore,

$$R_\infty^+ = \beta\int_0^\infty \kappa_\infty^+(a)\,\mathrm{d}a = \beta\zeta = \frac{-p_c}{\log(1-p_c)}\frac{\beta}{\sigma} = \frac{-p_c}{\log(1-p_c)}\tilde{R}_0. \qquad \square$$

Remark 3.15. The factor $-p_c/\log(1-p_c)$ reduces the effective reproduction number to an arbitrary small value (Fig. 4). This factor decreases approximately linearly with increasing p_c until p_c is relatively large. For large p_c the decrease becomes steeper, until the value of this factor tends to 0 for $p_c \to 1$. Hence, by an increase of p_c one gains more for large p_c than if p_c is small.

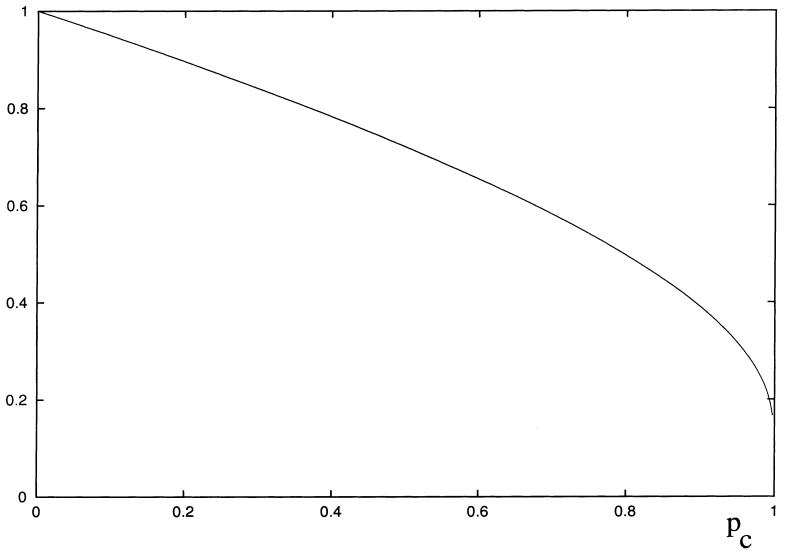

Fig. 4. This figure shows the factor by which contact tracing reduces the effective reproduction number.

3.2.2. Variable parameters

Now the case of variable parameters is considered. The same arguments as above lead to a recursion formula for $\kappa_0^+(a)$

$$\kappa_0^+(a) = \exp\left\{ -\int_0^a [\sigma(c) + \alpha(c)]\,\mathrm{d}c \right\}. \tag{10}$$

The equation for $\kappa_i^+(a\,|\,b)$ changes to

$$\begin{aligned}\kappa_i^+(a\,|\,b) &= \left\{ \frac{\kappa_{i-1}^+(a+b)}{\kappa_{i-1}^+(b)} + \int_0^a \alpha(b+c)\frac{\kappa_{i-1}^+(b+c)}{\kappa_{i-1}^+(b)}\,\mathrm{d}c \right. \\ &\quad \left. + \int_0^a (1 - p_c(a-c))\left[\frac{-(\mathrm{d}/\mathrm{d}c)\kappa_{i-1}^+(b+c)}{\kappa_{i-1}^+(b)} - \alpha(b+c)\frac{\kappa_{i-1}^+(b+c)}{\kappa_{i-1}^+(b)} \right] \mathrm{d}c \right\} \kappa_0^+(a) \\ &= \left\{ 1 - \int_0^a p_c(a-c)\left[\frac{-(\mathrm{d}/\mathrm{d}c)\kappa_{i-1}^+(b+c)}{\kappa_{i-1}^+(b)} - \alpha(b+c)\frac{\kappa_{i-1}^+(b+c)}{\kappa_{i-1}^+(b)} \right] \mathrm{d}c \right\} \kappa_0^+(a) \end{aligned} \tag{11}$$

and

$$\kappa_i^+(a) = \frac{\int_0^\infty \kappa_i^+(a\,|\,b)\beta(b)\kappa_{i-1}^+(b)\,\mathrm{d}b}{\int_0^\infty \beta(b)\kappa_{i-1}^+(b)\,\mathrm{d}b}. \tag{12}$$

3.3. Full tracing

Full tracing is a combination of forward and backward tracing. The primary infected individual only experiences backward tracing, because he/she is not infected by someone else within the population. The following generations fulfill the recursion formula for forward tracing.

Definition 3.16. Let $\kappa_i(a)$ be the probability for an individual of generation i to be infectious at age a of infection. Let furthermore

$$\kappa_\infty(a) := \lim_{i\to\infty} \kappa_i(a)$$

and the corresponding reproduction numbers

$$R_i = \int_0^\infty \beta\kappa_i(a)\,\mathrm{d}a \quad \text{and} \quad R_\infty = \lim_{i\to\infty} R_i.$$

Theorem 3.17. *$\kappa_0(a)$ satisfies the integro-differential equation*

$$\kappa_0'(a) = -\kappa_0(a)\left[\sigma + \alpha + \beta p_c(1 - \kappa_0(a)) - \beta p_c \alpha \int_0^a \kappa_0(c)\,\mathrm{d}c \right], \tag{13}$$

$$\kappa_0(0) = 1. \tag{14}$$

The generations i, $i \geqslant 1$, fulfill the recursion formula

$$\kappa_i(a) = \kappa_0(a)\left\{(1-p_c) + p_c(1+\alpha a)\frac{\int_a^\infty \kappa_{i-1}(b)\,db}{\int_0^\infty \kappa_{i-1}(b)\,db} + \alpha p_c \frac{\int_0^a b\kappa_{i-1}(b)\,db}{\int_0^\infty \kappa_{i-1}(b)\,db}\right\}. \tag{15}$$

Proof. The zeroth generation cannot be traced via their infectors since they are assumed not to be a member of the population. Hence, $\kappa_0(a) = \kappa_0^-(a)$, which yields (13) and (14). In order to derive an equation for $\kappa_i(a)$, $i > 0$, we introduce $\kappa_i(a\,|\,b)$ similar to $\kappa_i^+(a\,|\,b)$,

$$\kappa_i(a\,|\,b) := P\ (\text{Individual is infectious with age of infection } a \mid \text{Infector has age of infection } a+b).$$

We now use the argumentation of Proposition 3.10

$$\begin{aligned}\kappa_i(a\,|\,b) &= [P(\text{Infector infective}) + P(\text{Infector spontaneously recovered}) \\ &\quad + (1-p_c)P(\text{Infector observed})] \\ &\quad \times P(\text{Individual infectious at age a} \mid \text{no tracing via the infector}) \\ &= \left[1 - p_c + p_c\frac{\kappa_{i-1}(a+b)}{\kappa_{i-1}(b)} + \alpha p_c \frac{\int_0^a \kappa_{i-1}(b+c)\,dc}{\kappa_{i-1}(b)}\right]\kappa_0(a).\end{aligned}$$

Furthermore, with

$$\kappa_i(a) = \frac{\int_0^\infty \kappa_i(a\,|\,b)\,\beta\kappa_{i-1}(b)\,db}{\int_0^\infty \beta\kappa_{i-1}(b)\,db}$$

we obtain (15). □

Remark 3.18. In Fig. 5 we show simulations and solutions of these equations for $i = 0, \ldots, 3$ and $p_c = 0$, $p_c = 1/3$, $p_c = 2/3$ and $p_c = 1.0$. The graphs look like those for forward tracing, only that the slopes of the curves are somewhat steeper. Comparison of Figs. 2, 3 and 5 suggests that forward tracing contributes most to the effectiveness of contact tracing. In other words, finding the individual by whom the index case was infected is not as important as finding the infectees of the index case.

3.3.1. No natural recovery

As before, the special case of constant coefficients and $\alpha = 0$ can be handled more easily and leads to explicit results for the reproduction number and the critical value of p_c.

Theorem 3.19. *Let $\kappa_\infty(a) = \lim \kappa_i(a)$ for fixed a and $i \to \infty$. Let furthermore $R_\infty = \lim R_i$ for $i \to \infty$ and $\tilde{R}_0 = \beta/\sigma$. Then*

$$\kappa_\infty(a) = (1-p_c)\left(\frac{1-p_c\tilde{R}_0}{p_c\tilde{R}_0 + e^{\sigma(1+p_c\tilde{R}_0)a}}\right)\left(1 + p_c\tilde{R}_0 + e^{-\sigma(1+p_c\tilde{R}_0)a}\right)^{-\frac{\log(1-p_c)}{\log(1+p_c\tilde{R}_0)}} \tag{16}$$

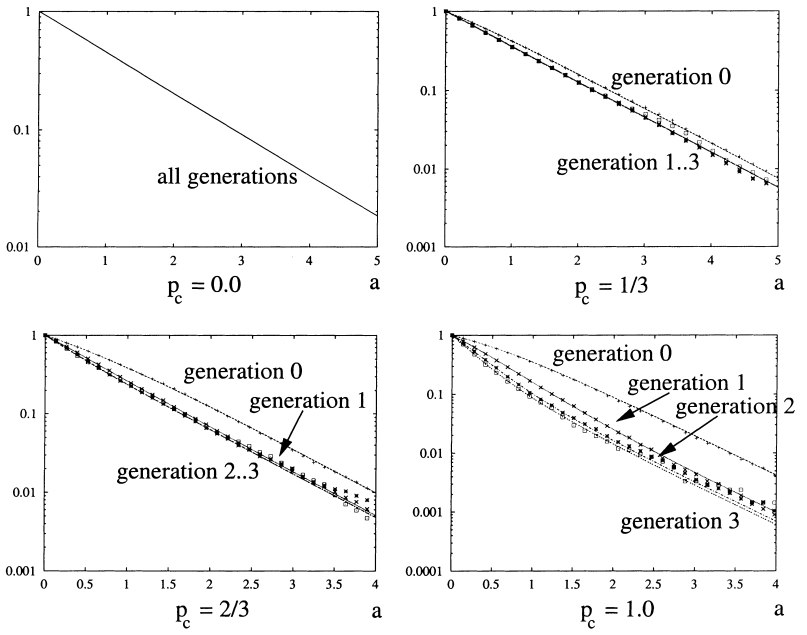

Fig. 5. Probability to be infective over age for generations 0–3. The lines are the theoretical curves, the points obtained by simulation of the stochastic process (for $p_c = 0$, no simulations of the stochastic process have been performed). The four different graphs correspond to $p_c = 0.0$, $p_c = 1/3$, $p_c = 2/3$ and $p_c = 1.0$. The other parametervalues are $\beta = 1.0$/time unit, $\sigma = 0.5$/time unit, $\alpha = 0.3$/time unit. For the stochastic process we performed 10 000 runs.

and

$$R_\infty = \frac{\log(1 + p_c\tilde{R}_0)}{-\log(1 - p_c)}. \tag{17}$$

Proof. From Proposition 3.5 we know that

$$\kappa_0(a) = \frac{1 + p_c\tilde{R}_0}{p_c\tilde{R}_0 + e^{\sigma(1+p_c\tilde{R}_0)a}}$$

and with conclusions similar to those in the proof of Proposition 3.14 we obtain

$$\kappa_\infty(a) = \kappa_0(a) \exp\left\{ -\frac{p_c}{\zeta} \int_0^a \kappa_0(\tau)\,d\tau \right\}, \tag{18}$$

where again $\zeta = \int_0^\infty \kappa_\infty(a)\,da$. With

$$\int_0^a \kappa_0(\tau)\,d\tau = \frac{1}{p_c\tilde{R}_0\sigma}\left[\log(1 + p_c\tilde{R}_0) - \log\left(1 + p_c\tilde{R}_0\, e^{-\sigma(1+p_c\tilde{R}_0)a}\right)\right]$$

it follows that

$$\kappa_\infty(a) = \frac{1 + p_c\tilde{R}_0}{p_c\tilde{R}_0 + e^{\sigma(1+p_c\tilde{R}_0)a}} \left(1 + p_c\tilde{R}_0\right)^{-1/(\zeta\tilde{R}_0\sigma)} \left(1 + p_c\tilde{R}_0\, e^{\sigma(1+p_c\tilde{R}_0)a}\right)^{1/(\zeta\tilde{R}_0\sigma)}.$$

Now we get the fixed point equation

$$\zeta = \int_0^\infty \kappa_\infty(a)\,\mathrm{d}a = \frac{\zeta}{p_c}\left(1 + p_c\tilde{R}_0\right)^{-1/(\tilde{R}_0\sigma\zeta)}\left[\left(1 + p_c\tilde{R}_0\right)^{1/(\tilde{R}_0\sigma\zeta)} - 1\right]$$

and hence $\zeta = -\log(1 + p_c\tilde{R}_0)/(\sigma\tilde{R}_0\log(1 - p_c))$. Thus, $R_\infty = \beta\zeta$ yields (17) and with

$$\mathrm{e}^{-\frac{p_c}{\zeta}\int_0^a \kappa_0(\tau)\,\mathrm{d}\tau} = (1 - p_c)\left(1 + p_c\tilde{R}_0\,\mathrm{e}^{-\sigma(1+p_c\tilde{R}_0)a}\right)^{-\frac{\log(1-p_c)}{\log(1+p_c\tilde{R}_0)}}$$

the representation for $\kappa_\infty(a)$ follows. □

Remark 3.20. The reduction of the effective reproduction number due to contact tracing is visualized by plotting $R_\infty/\tilde{R}_0$ as a function of p_c in Fig. 6. One finds, that especially for large $\tilde{R}_0$, even small tracing probabilities p_c have a considerable effect.

From Theorem 3.19 we immediately conclude the following result.

Lemma 3.21. *The critical tracing probability for achieving $R_\infty = 1$ is given by*

$$p_c = 1 - 1/\tilde{R}_0. \tag{19}$$

Remark 3.22. This formula resembles that for the critical vaccination coverage in simple epidemic models [1]. In both cases the number of infectious contacts of an infected individual during his infectious period has to be decreased by the factor $1 - 1/\tilde{R}_0$ to ensure that the average number of secondary cases does not exceed 1. In the case of vaccination this reduction is reached by moving susceptible individuals to the removed class and thus preventing transmission during contact with

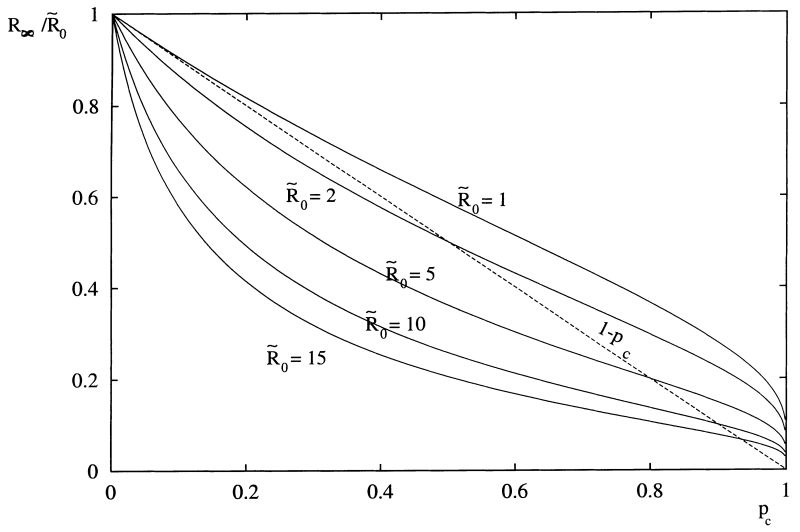

Fig. 6. The ratio of the reproduction numbers with and without contact tracing as a function of p_c. The intersection of the line $1 - p_c$ and the reduction factor gives the value of p_c, at which the threshold condition $R_\infty = 1$ is fulfilled.

those individuals. In the case of contact tracing the reduction is reached by effectively reducing the average duration of the infectious period by moving individuals who have been found by tracing to the removed class.

The result for the critical vaccination coverage is strictly spoken valid only for very simple, unstructured models. The success of this rule of thumb relies on the stability of the conclusion against perturbations of the model structure. Further investigation is needed to obtain an impression about the stability of the threshold condition for p_c with changes in model structure. If we allow for $\alpha > 0$, then $p_c = 1 - \sigma/\beta$ will be in general too large, since the reproduction number is diminished due to natural recovery and thus also the critical effort for contact tracing needed to eliminate the infection. However, $p_c = 1 - (\alpha + \sigma)/\beta$ will be too small, since natural recovery decreases the efficacy of contact tracing (some links between infected individuals will stay unknown). One may conjecture that the critical threshold lies in the interval $[1 - \sigma/\beta, 1 - (\alpha + \sigma)/\beta]$.

3.3.2. Variable coefficients

Again, from a combination of forward and backward tracing we obtain the following theorem.

Theorem 3.23. *For variable parameters we obtain*

$$\frac{\mathrm{d}}{\mathrm{d}a}\kappa_0(a) = -\kappa_0(a)\left\{\sigma(a) + \alpha(a) - \int_0^a p_c(a-c)\left[\alpha(c)\beta(c)\kappa_0(c) + \beta(c)\frac{\mathrm{d}}{\mathrm{d}c}\kappa(c)\right]\mathrm{d}c\right\}, \quad \kappa_0(0) = 1, \tag{20}$$

$$\kappa_i(a\,|\,b) = \left\{1 - \int_0^a p_c(a-c)\left[\frac{-(\mathrm{d}/\mathrm{d}c)\kappa_{i-1}(b+c)}{\kappa_{i-1}(b)} - \alpha(b+c)\frac{\kappa_{i-1}(b+c)}{\kappa_{i-1}(b)}\right]\mathrm{d}c\right\}\kappa_0(a), \tag{21}$$

$$\kappa_i(a) = \frac{\int_0^\infty \kappa_i(a\,|\,b)\beta(b)\kappa_{i-1}(b)\,\mathrm{d}b}{\int_0^\infty \beta(b)\kappa_{i-1}(b)\,\mathrm{d}b}. \tag{22}$$

Remark 3.24. It is easy to see that in general the effective reproduction number does not depend in a monotonous way on $\alpha(a)$. If we consider two scenarios, in which all parameters are the same except α, one expects the effective reproduction number for the scenario with the bigger α to be smaller since on average the infectious period is shorter. Let $\alpha_1(a) \leqslant \alpha_2(a)$. We refer to the scenario with the recovery rate $\alpha_1(a)$ as scenario 1 and to that with rate α_2 as scenario 2. Under certain circumstances there are rates α_1 and α_2 such that the effective reproduction number R_∞^1 in scenario 1 is smaller than R_∞^2 (the effective reproduction number in scenario 2).

The example can be constructed as follows. We assume $p_c > 0$ and $\sigma > 0$. Furthermore, $\beta(a) > 0$ for $a \leqslant \underline{a}$ and $\beta(a) = 0$ for $a > \underline{a}$, i.e. individuals are infectious only up to the time $\underline{a}$. After this time, they are not infectious anymore, but are still within the class of infected persons, and thus can still be recognized as persons who have had the infection. This means that they can identify other persons to whom they may have spread the infection. They only leave the class of infected individuals by recovery. If $\alpha_1(a) = \alpha_2(a)$ for $a \leqslant \underline{a}$, then the number of secondary cases

produced by an infected individual can only differ for the two scenarios as a result of contact tracing after the infectious period; infectivity and recovery rate during the infectious period are the same for both scenarios. Now we assume $\alpha_2(a) > \alpha_1(a)$ for $a > \underline{a}$. This means, that in scenario 2 individuals tend to leave the class of infected individuals earlier than in scenario 1. Hence, contact tracing in scenario 2 is less effective than in scenario 1, and hence $R_\infty^2 > R_\infty^1$ even though $\alpha_1 \leqslant \alpha_2$.

For the case of constant parameters, numerical simulations suggest that the effective reproduction number is decreasing in α.

4. The full epidemic process with contact tracing

4.1. Threshold theorem

Results about the probability for a major outbreak can be obtained from the analysis of the branching process describing the onset of the epidemic.

Theorem 4.1. *Let $R_\infty < 1$ and N be the population size. For $N \to \infty$, the branching process without nonlinear effects due to the infection and the full epidemic process agree P-almost. If $R_\infty > 1$, the two processes agree only up to a time which grows with $\log(N)$ as $N \to \infty$.*

Proof. The proof is a direct generalization of the proof of Theorem 2.1 in [12]. □

Hence we obtain the following threshold theorem.

Theorem 4.2. *If $R_\infty < 1$ the infection dies out. If $R_\infty > 1$, there is a positive probability for a major outbreak. This probability is that of the embedded asymptotically stationary Galton–Watson process at the onset of the epidemic.*

Proof. If $R_\infty < 1$, then it follows directly from Theorem 4.1 that the number of infected individuals tends to 0 with probability 1.

The second part of the theorem can be seen by the fact that the embedded Galton–Watson process becomes stationary: Let $f_i(x)$ be the probability generating function for generation i, i.e.

$$f_i(x) = \sum_{j=0}^{\infty} x^j P \text{ (to have } j \text{ individuals in generation } i).$$

There are functions $g_i(x)$, such that $f_i(x) = g_{i-1}(f_{i-1}(x))$. Since we assume $R_\infty > 1$, it follows that $p_c < 1$ and therefore $\kappa_i(a) \to \kappa_\infty(a)$ in $C^0(\mathbb{R}_+)$. Furthermore, we conclude

$$g_i(x) \to g_\infty(x) \quad \text{in } C^0[0,1] \text{ for } i \to \infty.$$

Hence it is possible to consider the stationary Galton–Watson process with the generating function $g_\infty(x)$ in order to estimate the probability of a major outbreak. □

4.2. Prevalence for the quasi-stationary state

We now consider the situation that the stochastic process is in the quasi-stationary state, i.e. in an equilibrium where the infection is present. Let u be the probability that a randomly chosen individual of the population is susceptible, v that he/she is infectious and w that he/she is immune. We assume that these probabilities are approximately independent of time (i.e. change only on a large time scale). In the quasi-stationary state, the effective reproduction number is one: In average, every infected individual is replaced by exactly one successor. An infectious person has potentially infectious contacts at rate β, but only in a fraction u of these contacts can transmission actually take place. Hence, the effective reproduction number is given by R_∞, where we replace β by βu,

$$R_\infty = R_\infty(u).$$

If $\alpha = 0$, this expression is explicitly known, $R_\infty(u) = -\log(1 + p_c\tilde{R}_0 u)/\log(1 - p_c)$. For $\alpha > 0$, $R_\infty(u)$ is implicitly determined as

$$R_\infty(u) = \beta u \int_0^\infty \kappa_\infty(a;u)\,\mathrm{d}u,$$

where $\kappa_\infty(a;u)$ is fixed point of Eq. (15), where again β is replaced by βu. In the endemic equilibrium, the magnitude u can be obtained by

$$R_\infty(u) = 1, \tag{23}$$

i.e. for $\alpha = 0$, this leads to

$$u = \left(\frac{1}{1 - p_c}\right)\frac{1}{\tilde{R}_0}. \tag{24}$$

Note that in the absence of contact tracing, $p_c = 0$, the density of the susceptibles is just one over the reproduction number, since in this case there is no dependency of the removal rate on u.

Perhaps more interesting than the relative prevalence of susceptibles is that of infectious persons. Since the probability to be susceptible is assumed to be constant in time, it follows that

$$0 = \frac{\mathrm{d}}{\mathrm{d}t}u = \frac{1}{N}\left\{ -\beta\frac{S}{N}I + \gamma R \right\} = -\beta uv + \gamma(1 - u - v)$$

and hence

$$v = \frac{\gamma}{\beta u + \gamma}(1 - u). \tag{25}$$

Furthermore, w is given by $u + v + w = 1$. In Fig. 7 we show a simulation of the stochastic process and find a good agreement of observed and predicted prevalence after an initial transient period.

4.3. Deterministic approximation

The aim of this section is to approximate the full stochastic process by a system of ordinary differential equations, that incorporate the most important effects. Hethcote and Yorke [13] describe a purely phenomenological approach to this task. The authors argue that the incidence is

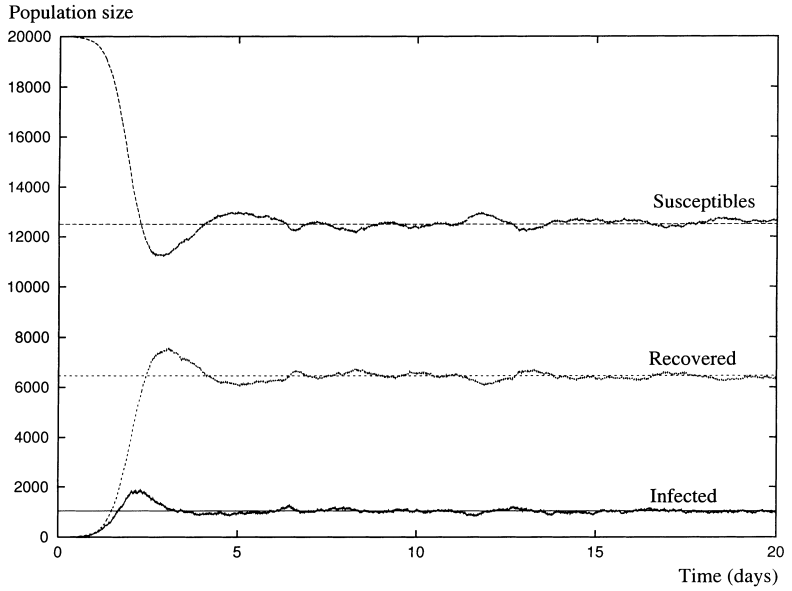

Fig. 7. Quasi-stationary state of the stochastic process. The lines mark the predicted prevalence. Parameter values: $\beta = 10.0$/time unit, $\sigma = 5.0$/time unit, $p_c = 0.2$, $\alpha = 0.0$/time unit and $N = 20000$.

diminished by a certain factor, since in average for each observed infectious individual more than one infectious individual is removed. However, the considerations above suggest that not the incidence function but the removal rate of the infectious individuals should be modified.

We denote the effective removal rate by α_{eff}, which is defined by the requirement that for a fixed state of the population the effective reproduction number of the stochastic process and that of the ODE-model should be the same. Let $u(t)$ be the density of susceptibles, $v(t)$ that of infective persons and $w(t)$ that for immunes. If $u(t) = u$ is fixed, then α_{eff} is given by

$$\frac{\beta u}{\alpha_{\text{eff}}} = R_\infty(u).$$

Of course, u will change over time. The idea is that $\alpha_{\text{eff}} = \alpha_{\text{eff}}(u)$ is a good approximation if the time course of the epidemic changes rather slowly in comparison to the infectious period of one individual. In other words, the fraction of susceptibles in the environment of an infectious individual will not change very much during his/her infectious period, and also the density of the network of infectious contacts is more or less constant. If $\alpha = 0$, it is possible to determine α_{eff} explicitly

$$\alpha_{\text{eff}}(u) = -\beta u \log(1 - p_c)/\log(1 + p_c \tilde{R}_0 u).$$

Hence, altogether we obtain the set of equations:

$$\dot{u} = -\beta uv + \gamma w, \tag{26}$$

$$\dot{v} = \beta uv - \alpha_{\text{eff}}(u)v, \tag{27}$$

$$\dot{w} = \alpha_{\text{eff}}(u)v - \gamma w. \tag{28}$$

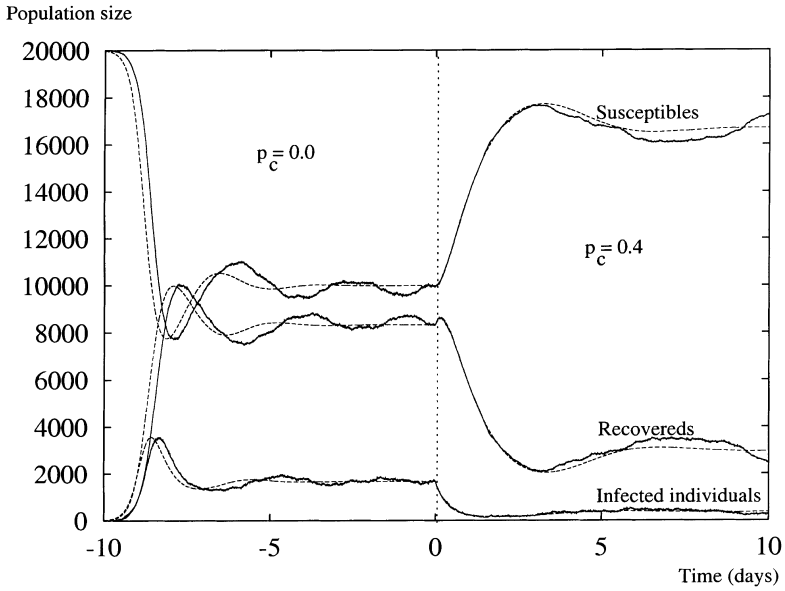

Fig. 8. Comparison of the deterministic and stochastic processes. For $t < 0$ there is no contact tracing, i.e. $p_c = 0$. At $t = 0$, contact tracing is introduced with $p_c = 0.4$. Parameter values: $\beta = 10.0$/time unit, $\sigma = 5.0$/time unit, $\alpha = 0.0$/time unit and $N = 20000$.

We show a comparison between deterministic and stochastic models in Fig. 8. As initial conditions, we assumed that there was one infected individual at $t = 0$ for the stochastic model, and for the deterministic model $u = 1 - 1/20000$, $v = 1/20000$ and $w = 0$ for $t = 0$. While in the deterministic model the prevalence of infection starts increasing immediately, in the stochastic model it takes some time until the epidemic takes off. Nonetheless, once a certain level of prevalence is reached, both models behave similar.

The following theorem shows that this system cannot exhibit complex dynamics.

Theorem 4.3. *Consider the system* (26)–(28). *If* $R_\infty(u)$ *is strictly monotonously decreasing in* u, *then for* $R_\infty(1) < 1$ *there is only the disease-free equilibrium, which is globally stable. For* $R_\infty(1) > 0$, *there is also a unique endemic equilibrium. In this case, the disease-free equilibrium is unstable, and the endemic equilibrium attracts all trajectories with* $v(0) > 0$.

Proof. First, using $u + v + w = 1$, the system can be reduced to a two-dimensional differential equation in u and v. Since $R_\infty(u)$ is assumed to be monotone, there is for $v \neq 0$ at most one equilibrium. This equilibrium is non-negative for $R_\infty(1) > 1$. Hence we obtain for $R_\infty(1) < 1$ that there is only the disease-free equilibrium, while for $R_\infty(1) > 1$ there is additionally the endemic equilibrium (which is explicitly computed in Section 4.2). The local stability results can easily be obtained by inspecting the eigenvalues of the linearization of the system at the stationary points. In order to prove the global stability results, we note that the set $\{(u, v) \mid u + v \leqslant 1,\ 0 \leqslant u, v\}$ is invariant. Furthermore, with the negative criterion of Bendixon–Dulac we exclude periodic orbits: Scaling time by $1/(uv)$ and inspecting the divergence of the vector field for u and v, we obtain for $u, v > 0$

$$\frac{\partial}{\partial u}\left\{\left(\frac{1}{uv}\right)(-\beta uv+\gamma(1-u-v))\right\}+\frac{\partial}{\partial v}\left\{\left(\frac{1}{uv}\right)(\beta uv+\alpha_{\text{eff}}(u)v)\right\}$$
$$=\gamma\left(-\frac{1}{u^2v}+\frac{1}{u^2}\right)=-\gamma\frac{1-v}{u^2v}<0.$$

Since there are no periodic orbits touching $u=0$ or $v=0$, periodic orbits can be excluded and thus the global stability result follows. □

Remark 4.4. The required monotonicity of $R_\infty(u)$ holds for $\alpha=0$. Also for $\alpha>0$ one expects that R_∞ depends in a monotonous way on u, although this is not shown here.

5. Example

In order to give an impression about results the described methods may provide, we assign values to the rates that are in the magnitude of those of gonorrhea. It must be emphasized that the model in the present form is not suited as a realistic description of the dynamics of this infection, since it assumes homogeneous mixing (which is not appropriate for the most STDs), there is no distinction between men and women, and since there is no explicit distinction between symptomatic and unsymptomatic cases. The latter may be incorporated in the rates α and σ: Even if there is no screening program at all, a certain part of the symptomatic cases consult the doctor who confirms the disease. This part is given by $\sigma/(\alpha+\sigma)$.

However, we assume that without screening there are no index cases. The reproduction number of gonorrhea is about 1.4 and the typical infectious period for symptomatic infections is about 1 month [13], leading to rates

$$\beta=16.8\ \text{yr}^{-1},\quad \alpha=12.0\ \text{yr}^{-1},\quad \sigma=0.0\ \text{yr}^{-1}\quad\text{and}\quad p_c=0.$$

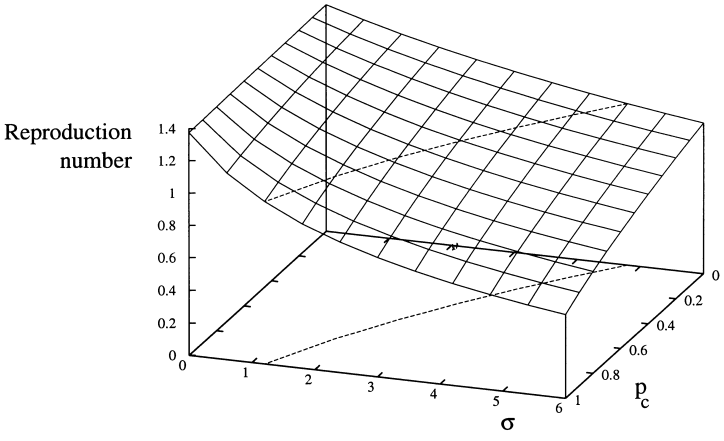

Fig. 9. The effect of screening with rate σ and contact tracing with probability p_c on the effective reproduction number. The lines on the surface of the graph and in the σ–p_c plane show the values of σ and p_c that imply $R_\infty=1$.

Now we start a screening program with rate σ and at the same time an index case, discovered by screening, is interviewed about his/her contacts leading to a tracing probability p_c. Fig. 9 shows the resulting effective reproduction number.

Without contact tracing ($p_c = 0$), the screening rate has to be $\sigma = 4.8\ \mathrm{yr}^{-1}$ in order to ensure $R_\infty = 1$. In the other extreme, $p_c = 1$, this rate has to be approximately $\sigma = 1.2\ \mathrm{yr}^{-1}$. Of course, it is extremely expensive (if possible at all) to achieve $p_c = 1$. On the other hand, also a high screening rate implies a very expensive screening program. If it is possible to assign costs to a screening program with a certain screening rate, and also to a certain tracing probability, it is possible to compute the optimum, i.e. the most inexpensive strategy that brings R_∞ down to 1.

6. Discussion

The model under consideration, especially its contact structure, is very simple. The assumption of homogeneity of the population may be more or less appropriate for tuberculosis, but is definitely not justified for STDs, where core groups play a major role [14]. Our model predicts that screening for infectors is less effective than screening for infectees. This seems to be a contradiction to the conclusions of Hethcote and Yorke [13]. This apparent contradiction is a consequence of the simplicity of the contact structure considered here. Hethcote and Yorke investigate a core group model. Hence, the infector is likely to belong to the core group and thus should be removed with priority. In our model, the contact rate of infector and infectee do not differ. Furthermore, all cases infected by an infectee are still unknown, while at least the index case already is known as one infectee of the infector. Hence, one expects to find more cases per observed infectee of an index case than per observed infector. It is not clear how these two effects balance in a core group model. However, this discussion is somewhat theoretic, since in practice one usually cannot distinguish between infectors and infectees.

It is easily possible to extend the method presented here to a model for STDs in a purely heterosexual population. In that case one knows that the infectee is a man if the infector is a woman and vice versa. Hence, one can define parameters (e.g. transmission rate, fraction of reported cases etc.) that are specific for women in the even generations and for men in the odd generations. This yields a two-level iterative equation instead of Eq. (15), but does not change the structure of the analysis. Furthermore, it is also easily possible to consider other types of models (SIS etc.) rather than SIRS-models.

Our central result is Theorem 4.2. Crucial for this theorem is the parameter p_c. Since for estimates of this parameter not only the number of identified infectious contacts, but also the number of all infectious contacts is necessary, this parameter is difficult to determine in practice. Some estimates have been obtained for the fraction of secondary infections found by conventional contact tracing for tuberculosis [15,16]. Those estimates (5–10%) were obtained by comparing clusters of recent infections identified by methods of molecular epidemiology with the network of contacts found by conventional contact tracing. For STDs there have been studies to estimate the fraction of identified sexual partners of patients, that after notification seek examination and treatment [17,18]. This gives an idea of the fraction of partners that can be reached for treatment, but the number of partners named by patients might be an underestimate of the total number of partners.

The main conclusions drawn from this article for practical applications are:

(a) for diseases, where the removed can be traced or no natural recovery exists, there is a critical value for the fraction of infectious contacts that should be traced in order to bring the effective reproduction number below 1; and

(b) tracing for a very small number of steps from the index case may already practically maximize the effect that can be reached by tracing, thus it might not be useful to put much effort into tracing longer chains of contacts.

A task for future research will be to make those conclusions more concrete and to quantify them for specific infectious diseases and populations.

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