Journal of Theoretical Biology 257 (2009) 52–60

Contents lists available at ScienceDirect

Journal of Theoretical Biology

journal homepage: www.elsevier.com/locate/yjtbi

Contact switching as a control strategy for epidemic outbreaks

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ARTICLE INFO

Article history:
Received 22 April 2008
Received in revised form
16 October 2008
Accepted 30 October 2008
Available online 8 November 2008

Keywords:
Epidemiology
SIS models
Network dynamics

ABSTRACT

We study the effects of switching social contacts as a strategy to control epidemic outbreaks. Connections between susceptible and infective individuals can be broken by either individual, and then reconnected to a randomly chosen member of the population. It is assumed that the reconnecting individual has no previous information on the epidemiological condition of the new contact. We show that reconnection can completely suppress the disease, both by continuous and discontinuous transitions between the endemic and the infection-free states. For diseases with an asymptomatic phase, we analyze the conditions for the suppression of the disease, and show that—even when these conditions are not met—the increase of the endemic infection level is usually rather small. We conclude that, within some simple epidemiological models, contact switching is a quite robust and effective control strategy. This suggests that it may also be an efficient method in more complex situations.

1. Introduction

Mathematical models have been applied to the study of infectious diseases since more than a century ago. The last four decades have witnessed a burst of interest in quantitatively understanding the transmission dynamics of a large number of diseases (Anderson and May, 1991). One of the key aims of epidemiological mathematical models, and certainly the most relevant in terms of policy making, is the assessment of the effectiveness of control strategies to curb disease spreading. For many infectious diseases, the most widespread prevention measure is mass vaccination. However, if for a given disease vaccines are not known, or vaccination is not effective, other control measures have to be adopted. These mostly consist in some form of isolation of the infective individuals. Isolation from an individual's social environment, however, can have serious psychological effects such as depression and stress (Hawryluck et al., 2004). An even more drastic measure is quarantine, where individuals *assumed* to have been in contact with infective people are temporarily isolated from the rest of society. Daniel Defoe gives a forceful description of this practice and its tragic effects during the great plague of London (1665–1666) in his *Journal of the Year of the Plague*. A different strategy, that has helped preventing the spread of some sexually transmitted diseases, is contact tracing, where all the partners of an infective individual are located to be informed about the possibility of infection and, eventually, given adequate treatment. It has been argued, however, that this practice violates the right to privacy. In the case of HIV, for instance, it has sometimes met with considerable opposition (Bayer and Toomey, 1992). It must also be mentioned that all these measures require widespread governmental action and, frequently, allocation of substantial resources (Armbruster and Brandeau, 2007).

Recently, it has been suggested that a different kind of strategy, implemented by the individuals themselves, could give surprisingly positive results in controlling disease spreading (Gross et al., 2006; Zanette and Risau-Gusman, 2008). The basic idea is that contact between non-infective and infective acquaintances must be systematically avoided. This can be implemented by either non-infective or infective individuals, or by both. The effects of this simple strategy have been explored by means of network epidemiological models. In these models, each member of a population—hereafter, an *agent*—occupies a node of a network (Keeling and Eames, 2005). Neighboring agents, which are connected by network links, can interact, and their epidemiological states (susceptible, infective, recovered, etc.) thus change. For instance, the interaction of a susceptible agent and an infective agent may lead, with a certain probability, to the infection of the former. The set of neighbors of a given agent represents the individual's acquaintances.

In the framework of adaptive network models (Gross and Blasius, 2008), the mechanism of avoiding contact with infective acquaintances amounts to breaking the links between susceptible and infective agents. If the social connectivity is nevertheless to be preserved, those broken links must be replaced by new connections. In the SIS (susceptible → infective → susceptible) epidemiological model analyzed by Gross et al. (2006), it is susceptible agents who break contacts with their infective

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doi:10.1016/j.jtbi.2008.10.027

neighbors, what necessarily leads to the isolation of infective agents, and new connections are established only with other susceptible agents. Implicitly, this assumes that the epidemiological state of all agents is known to any other agent, irrespectively of whether they are connected or not. More realistically, Zanette and Risau-Gusman (2008) consider links that are broken and reconnected to randomly chosen members of the population. As described in the next section, we adopt here the same viewpoint. Furthermore, in order to avoid straightforward isolation of infective agents, in Section 3 we generalize the model in order to also allow infective individuals to switch connections from their susceptible neighbors to new contacts chosen at random. We show that, even under these less stringent assumptions on the mechanism of link switching, the outcome is still an effective reduction in the probability of infection, which can eventually lead to the complete suppression of the disease. In Section 4, we study the case where the disease has an asymptomatic period, during which infective individuals are not perceived as such but appear as being susceptible, even to themselves (Fraser et al., 2004). In this situation, it may happen that an asymptomatic agent switches contact from an infective neighbor to a susceptible agent, thereby increasing the probability of transmission. We find that, if the asymptomatic period is not very long, large enough switching rates can still suppress the infection. Results are summarized and discussed in the last section.

2. SIS network model with switching links

We consider a population formed by N agents, situated at the nodes of a network. At a given time, each of them can be either susceptible (S) or infective (I). Initially, each agent has exactly k neighbors and, thus, the network has $kN/2$ links. The initial contacts are chosen at random, so that the resulting network is a random regular graph. The corresponding degree distribution, i.e. the distribution of the number of neighbors per agent, is thus a delta-like function centered at k neighbors.

The infection is transmitted from infective agents to their susceptible neighbors, who become immediately infective ($S \to I$), at rate λ. Moreover, infective agents get cured at rate γ. Upon cure, they return to the susceptible state ($I \to S$) and, subsequently, can be infected again. The diseases described by the SIS model are those that do not confer immunity against subsequent infections, and which have very short latent periods. Models of this class have proven particularly useful for the study of chlamydia and gonorrhea (Hethcote and Yorke, 1984; Kretzschmar et al., 1997; Ghani et al., 1996; Turner et al., 2006).

Contact switching, in turn, is modeled as follows. All links joining susceptible agents (or, for short, *susceptibles*) with infective agents (or *infectives*) are broken at rate r, and the corresponding susceptible agent is then connected to a randomly chosen agent in the population. Self-connections and multiple connections between pairs of agents are avoided.

Taking into account the above dynamical rules, and using the pair approximation discussed in the Appendix, it is possible to write down evolution equations for the variables $n_A(t)$ and $m_{AB}(t)$, with $\{A,B\} \equiv \{S,I\}$. They represent, respectively, the fraction of agents in state A and the fraction of links joining agents in states A and B. The equations read

$$\begin{aligned}
\dot{n}_I &= -n_I\gamma + \lambda m_{SI},\\
\dot{m}_{SI} &= 2\gamma m_{II} - \gamma m_{SI} + \lambda k K m_{SI}\frac{2m_{SS}-m_{SI}}{n_S} - \lambda m_{SI} - r n_S m_{SI},\\
\dot{m}_{II} &= -2\gamma m_{II} + \lambda m_{SI} + 2\lambda k K m_{SI}\frac{m_{SI}}{n_S}.
\end{aligned} \tag{1}$$

As the total number of agents and links is preserved, this system is completed by the relations $m_{II}+m_{SI}+m_{SS}=1$ and $n_I+n_S=1$. The constant $K=(k-1)/k$ derives from the pair approximation. The high-connectivity limit, $K=1$, has been considered by Gross et al. (2006).

In the remaining of this article we concentrate on the equilibrium properties of our system, obtained by equating the right-hand side of Eqs. (1) to zero. After some algebra, we find that the equilibrium fraction of infectives n_I satisfies the fourth-degree polynomial equation

$$n_I[-\tilde{r}(1-n_I)^3 - (1-n_I)^2 - K^2 n_I^2 + K(1-n_I)(k\tilde{\lambda} - (2+\tilde{\lambda})n_I)] = 0. \tag{2}$$

The parameters $\tilde{r}=r/\gamma$ and $\tilde{\lambda}=\lambda/\gamma$ result from rescaling time in Eqs. (1), so that the time unit is the average recovery time γ^{-1}. The infection-free state, $n_I=0$, is a trivial solution of Eq. (2) for all values of the parameters. As expected, in fact, the infection cannot spontaneously appear in the absence of infectives. A second solution of the equation is always larger than unity. It therefore has no biological meaning, and will not be further discussed. As for the remaining solutions, it can be shown that for every value of the reconnection parameter $\tilde{r}$, there is a critical value of infectiveness $\tilde{\lambda}_c$ at which a transition from the infection-free ($\tilde{\lambda}<\tilde{\lambda}_c$) to the endemic state ($\tilde{\lambda}>\tilde{\lambda}_c$), with persisting infection, occurs.

To determine the nature of this transition we perform a stability analysis of the solutions of Eq. (2). When the reconnection parameter is below the threshold $\tilde{r}_c=[(2K-1)k+1]/(2k-1)$, the situation is similar to the case without reconnection ($\tilde{r}=0$). For small $\tilde{\lambda}$ the only stable non-negative solution is $n_I=0$. At $\tilde{\lambda}_c$, a transcritical bifurcation takes place. Namely, another real solution becomes simultaneously positive and stable, whereas $n_I=0$ becomes unstable. For larger values of the infectiveness, the equilibrium fraction of infectives grows continuously from zero.

When, on the other hand, $\tilde{r}>\tilde{r}_c$, the only real solution for $\tilde{\lambda}<\tilde{\lambda}_c$ is $n_I=0$. Now, the transition at the critical infectiveness $\tilde{\lambda}_c$ is a tangent bifurcation, and two real and positive solutions appear simultaneously. For $\tilde{\lambda}>\tilde{\lambda}_c$, the larger of these two solutions is stable and the other one is unstable, while the stability of $n_I=0$ does not change. We have therefore a discontinuous transition from the infection-free state to the endemic state. Further increasing $\tilde{\lambda}$, a transcritical bifurcation point is reached, where the unstable solution vanishes and exchanges stability with $n_I=0$. From then on, there is a single non-negative stable solution. Between the two bifurcations, the system has two stable equilibrium states, one endemic and the other infection-free. In this bistable region, the long-time asymptotic state is selected by the initial state of the population. An initial fraction of infectives larger than the unstable solution leads to the endemic state, whereas a smaller fraction leads to the extinction of the infection. The unstable solution thus plays the role of a threshold for the epidemics to spread and, consequently, is sometimes called *breakpoint density* (Anderson and May, 1991).

In Fig. 1 analytical predictions of the equilibrium fraction of infectives, calculated from Eq. (2), are compared with numerical simulations of the epidemic process. As explained below, it makes sense to consider the solutions of Eq. (2) fixing the parameter K at both $K=(k-1)/k$ (see the Appendix) and $K=1$. For a reconnection parameter $\tilde{r}=0$, the analytical curve corresponding to $K=(k-1)/k$ accurately fits the numerical results (Levin and Durrett, 1996). When $\tilde{r}\neq 0$, on the other hand, for all but the smallest values of the reconnection parameter the curves that best fit the data are those with $K=1$. A likely reason for this discrepancy lies in the fact that, depending on $\tilde{r}$, contact switching events generate networks with different degree distributions. It has previously

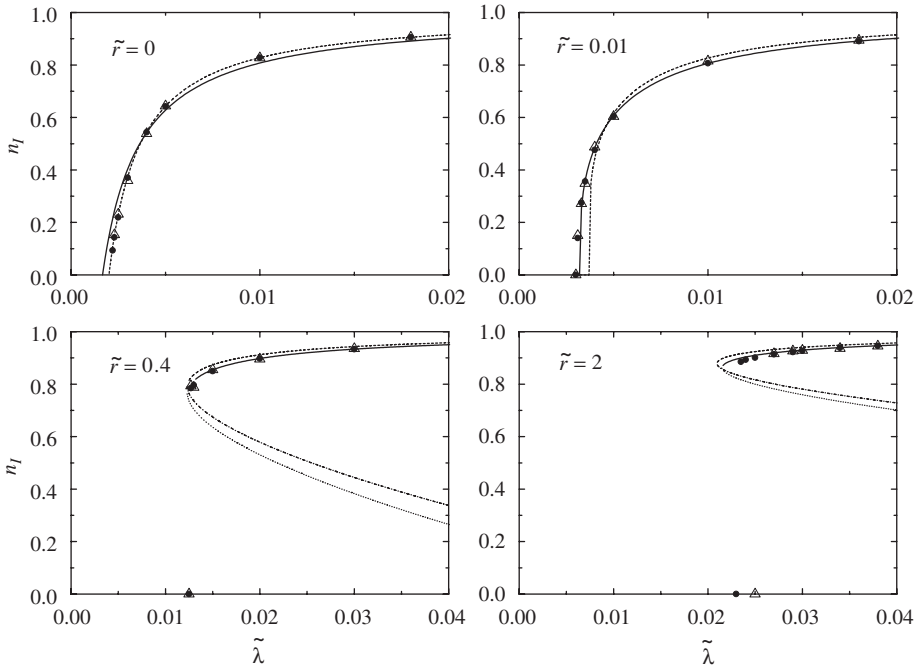

Fig. 1. Equilibrium fraction of infective agents as a function of infectivity, for $k = 3$ and four different values of the reconnection parameter $\tilde{r}$. Full and dashed lines are the analytical values of n_I with $K = (k-1)/k$ and $K = 1$, respectively. In the lower panels, the upper and lower branches of each curve, respectively, correspond to the stable and unstable equilibrium solutions. The lower branch represents the breakpoint density (see main text). Symbols are results of single realizations in numerical simulations for a population of 500 agents (triangles) and 5000 agents (circles), with $\gamma = 1$.

been shown that, not unexpectedly, rewiring can lead to degree distributions that get broader as the reconnection parameter is increased (Gross et al., 2006; Evans, 2007). We have verified that the same happens in our model. It has also been pointed out that the pair approximation is not good for describing contact processes on networks with long-tailed degree distributions (Peyrard and Franc, 2005). The value $K = (k-1)/k$, in fact, results for the pair approximation applied to regular networks with k neighbors per node. A possible reason for the better fit with $K = 1$ could be that, with the progressive broadening of the degree distribution as rewiring proceeds, agents with many connections ($k \gg 1$) appear and become dominant in the epidemiological process. These agents, usually called *superspreaders*, are known to be the main vector of propagation of many diseases (Lloyd-Smith et al., 2005). Taking $K = 1$—which, as mentioned above, corresponds to the limit of $k \gg 1$ for a regular network—is therefore a kind of effective approximation accounting for the role of such agents. In the variants of the model considered below, again, better agreement between analytical and numerical results is found for $K = 1$. Consequently, all the analytical results presented in the following are obtained for this value of K.

The possible asymptotic behaviors of our model can be summarized in a phase diagram on the parameter plane $(\tilde{\lambda}, \tilde{r})$, as shown in Fig. 2 for three values of the average connectivity k, fixing $K = 1$. For each value of k, the plane is divided into three regions. In the left-most region, corresponding to small infectivity, the fraction of infectives asymptotically vanishes and the population reaches the infection-free state. The central V-shaped region is the bistability zone, where the endemic state is stable but the infection-free state is still reached from initial conditions with sufficiently low levels of infectives. Note that, as discussed above, bistability does not occur for small reconnection parameters ($\tilde{r} < \tilde{r}_c$). Finally, for large infectivities, we have the region where the endemic state is the only stable equilibrium of the epidemiological process. To the left, this region is bounded by the transcritical bifurcation line, $\tilde{\lambda}_T = (\tilde{r}+1)/k$.

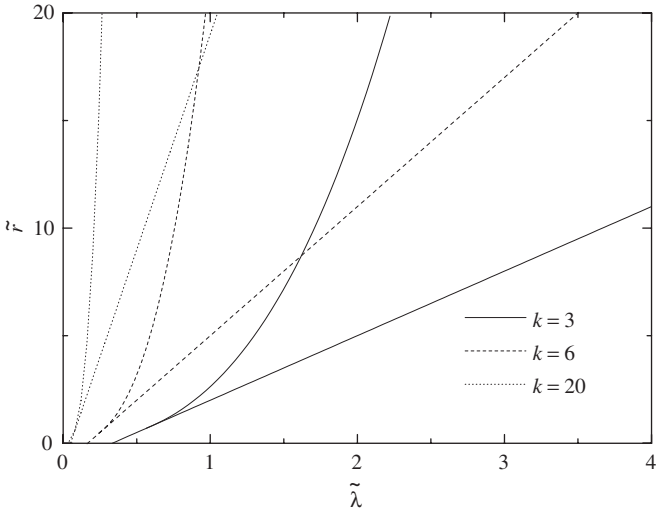

Fig. 2. Phase diagram of the basic model, for three different values of k, and $K = 1$. For each value of k, the curve and the straight line represent, respectively, the tangent and the transcritical bifurcation. The V-shaped region between them is the bistability zone. The left-most region corresponds to the infection-free state, whereas the right-most region corresponds to the endemic state.

It is interesting to notice that results similar to those derived from Eq. (2) can be obtained from the usual approach in mathematical epidemiology, where it is assumed that an epidemic outburst occurs as soon as an infective agent can generate more than one secondary infection (i.e. an infection event between two agents which involves a third, intermediary agent). This is quantified with the basic reproductive number R_0, which is the average number of secondary infections caused by a single infective agent in an otherwise healthy population (Anderson and May, 1991). The epidemic threshold is calculated by setting $R_0 = 1$. In our case, a straightforward calculation of the

basic reproductive number yields

$$R_0 = \frac{k\tilde{\lambda}}{\tilde{r} + \tilde{\lambda} + 1} \tag{3}$$

from which the epidemic threshold turns out to be $\tilde{\lambda}_{R_0} = (\tilde{r}+1)/(k-1)$. This is exactly the value at which the transcritical bifurcation occurs in our model when $K = (k-1)/k$. The slightly smaller result we obtain for $K = 1$, $\tilde{\lambda}_T = (\tilde{r}+1)/k$, is a consequence of the above discussed correction: it is well known that wider degree distributions lead to smaller epidemic thresholds (Anderson and May, 1991; Pastor-Satorras and Vespignani, 2001).

With the aim of making this basic model more realistic, two variants are considered in the following. In the first one, infective agents are allowed to reconnect their contacts with susceptibles. In the second, an asymptomatic stage in the disease is taken into account.

3. Reconnection of infective agents

Our basic model can be generalized to the more realistic situation where not only susceptibles are allowed to change their contacts, but also infectives can break their links with susceptible neighbors and redirect them to other members of the population. This possibility would stand for an altruistic attitude of infectives, in an attempt to inhibit the infection spread. To implement this variation, we introduce a new parameter p. When a link between a susceptible and an infective agent is cut, with probability p it is the infective agent who keeps the connection, whose other end is redirected to a randomly chosen member of the population, avoiding self- and multiple connections. With the complementary probability, $1-p$, the connection is kept by the susceptible agent. Thus, this variation reduces to the basic model for $p = 0$.

The evolution equations for the densities and the number of links—fixing, as advanced above, $K = 1$ in the pair approximation—are now

$$\dot{n}_I = -n_I\gamma + \lambda m_{SI},$$

$$\dot{m}_{SI} = \gamma(2m_{II} - m_{SI}) + \lambda k m_{SI}\frac{2m_{SS} - m_{SI}}{n_S} - \lambda m_{SI} - r m_{SI}[(1-p)n_S + p n_I],$$

$$\dot{m}_{II} = -2\gamma m_{II} + \lambda m_{SI} + 2\lambda k m_{SI}\frac{m_{SI}}{n_S}, \tag{4}$$

and the equation for the equilibrium fraction of infectives reads

$$n_I\{-\tilde{r}[(1-n_I)^3 + p(1-n_I)(2n_I-1)] + \tilde{\lambda}(1-n_I)(k-n_I) - 1\} = 0. \tag{5}$$

As in the case of $p = 0$, this system undergoes a transcritical bifurcation where the infection-free state, with $n_I = 0$, becomes unstable. The threshold infectivity at this bifurcation is $\tilde{\lambda}_T = [\tilde{r}(1-p)+1]/k$. The critical value of the reconnection parameter, above which a tangent bifurcation gives origin to two positive solutions, is $\tilde{r}_c = (k+1)/(1-p)(2k-1)$. Note that, for $\tilde{r} < \tilde{r}_c$, where the epidemic threshold is given by the transcritical bifurcation, λ_T decreases with p. The reconnection of infectives, as expected, favors infection spreading. The disease is better controlled if only susceptibles are allowed to protect themselves against contagion. In the extreme case where only infectives reconnect, the infection threshold does not depend on $\tilde{r}$.

For $\tilde{r} > \tilde{r}_c$, the behavior of the epidemic threshold is more complicated. Fig. 3 shows that the position of the tangent bifurcation is not monotonic with p. This leads to the unusual situation depicted in Fig. 4. In certain regions of parameter space, increasing p first makes the infection disappear, but a further increase of p leads to the reappearance of the endemic state through a second tangent bifurcation. For parameter values for which the infection is not suppressed, on the other hand, the fraction of infectives decreases monotonically.

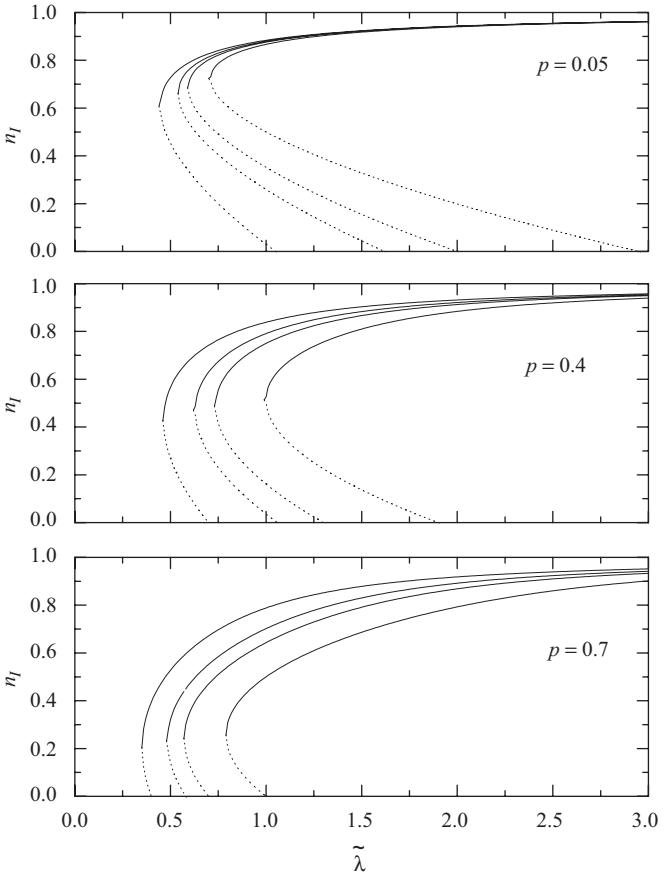

Fig. 3. Bifurcation diagrams for the fraction of infectives, as a function of the infectivity, for $k = 10$ and three different values of p, the probability that, for each susceptible–infective link, it is the infective who reconnects. From left to right the curves in each panel correspond to $\tilde{r} = 10$, 16, 20 and 30. Full and dashed lines represent stable and unstable equilibrium solutions, respectively.

To check that these effects are not an artifact of the mathematical model, we have performed simulations of the corresponding contact process (Levin and Durrett, 1996), whose results are represented by dots in Fig. 4. We verify that simulations display the same qualitative behavior as the analytical model, even though its predictions are not as good as for $p = 0$ (cf. Fig. 1). The coincidence of numerical results for populations of 5000 and 25 000 agents suggests that this discrepancy cannot be ascribed to finite size effects. The difference is due to the fact that the ansatz used in the pair approximation fails when p increases, because the distribution of the number of infectives connected to each susceptible becomes very wide (see Appendix for the details).

The phase diagram in the plane $(\tilde{\lambda}, \tilde{r})$, shown in Fig. 5 for different values of p, reveals the non-monotonic dependence on this parameter in the mutual crossings of the left boundary of the bistability regions. Note that, in any case, large enough reconnection rates always lead to the infection-free state. In fact, reconnection never increases the number of susceptible–infective links. In the next section, on the other hand, we consider a variation of the basic model where susceptible–infective links can be created by contact switching.

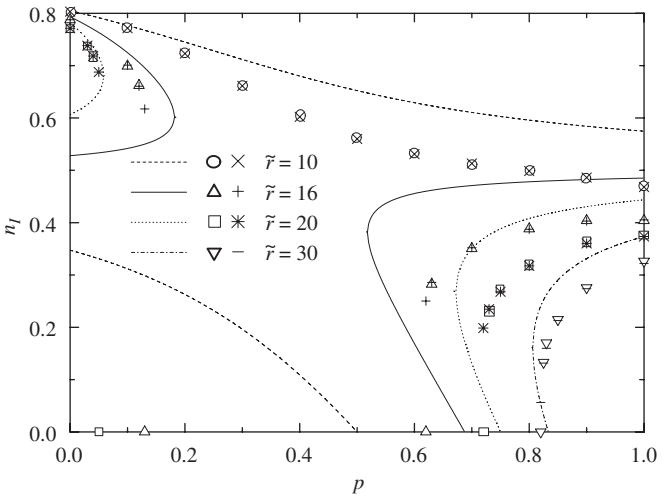

Fig. 4. Bifurcation diagram for the fraction of infectives, as a function of the probability p, for $k = 10$, $\tilde{\lambda} = 0.6$, and several values of the reconnection parameter $\tilde{r}$. The upper and lower branches of each curve, respectively, correspond to the stable and unstable solutions. The symbols ○, △, □, and ▽ correspond to simulations for 5000 agents, whereas ×, +, ∗, and − correspond to 25 000 agents. Simulations were done with $\gamma = 1$.

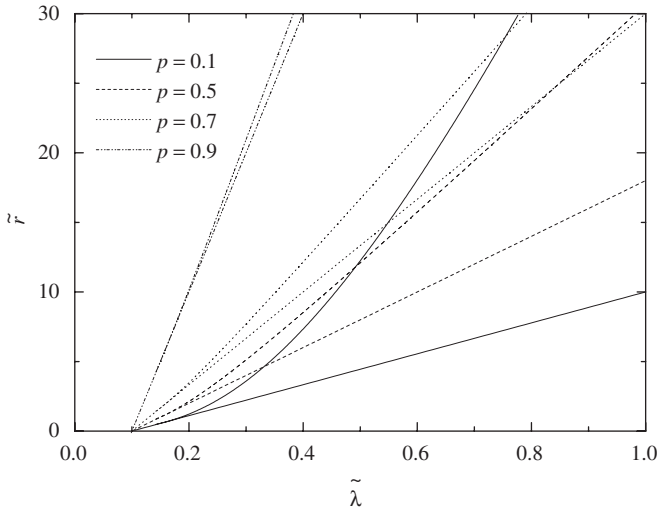

Fig. 5. Phase diagram of the model with infectives who can reconnect, for different values of p. For each value of p, the curve and the straight line represent, respectively, the tangent and transcritical bifurcation. Compare with Fig. 2.

4. Effect of asymptomatic agents

Several common diseases possess an asymptomatic stage, intermediary between the susceptible and the infective phases, during which individuals carrying the infection do not exhibit symptoms, and are therefore seen as non-infective by themselves and by the rest of the population. In the asymptomatic stage, however, an infective person may already transmit the disease by contagion. It is well known that during the several plagues that ravaged Europe, infective people who did not yet have visible symptoms became one of the main vectors for the propagation of the disease, when they fled the cities. It is therefore not unreasonable to fear that, for a disease with an asymptomatic phase, the strategy of contact switching might reinforce the spread of the disease, instead of curbing it.

To address these issues, in this section we analyze a model with three types of agents, namely, S, A (asymptomatic), and I. It is basically an extension of the model studied in Section 2, because the interactions between susceptibles and infectives are the same. We assume that asymptomatic agents do not know that they are infective, and therefore act as susceptible individuals: they reconnect their links with infective neighbors at the same rate r as susceptibles. On the other hand, asymptomatic agents can infect their susceptible neighbors at the same rate λ as infectives. Note that a main difference with the two models analyzed above is that, now, susceptible–asymptomatic links—which allow for the transmission of the infection—can be created by reconnection from asymptomatic–infective links, which join two agents carrying the infection.

To characterize the transitions $A \to I$ and $I \to S$ we introduce a new parameter α. The rate at which asymptomatic agents become (symptomatic) infectives is γ/α, while the rate at which infectives become susceptible is $\gamma/(1-\alpha)$. With these definitions, the asymptomatic and infective phases last, on the average, α/γ and $(1-\alpha)/\gamma$ time units, respectively. The mean total infective period is, as before, γ^{-1}. In the limit $\alpha \to 0$, we recover the model of Section 2, whereas for $\alpha \to 1$, the model is equivalent to the usual SIS model without reconnection.

The evolution equations for this model are

$$\begin{aligned}
\dot{n}_I &= -\frac{\gamma}{1-\alpha} n_I + \frac{\gamma}{\alpha} n_A, \\
\dot{n}_A &= -\frac{\gamma}{\alpha} n_A + \lambda k (m_{SI} + m_{SA})/2, \\
\dot{m}_{SI} &= \frac{\gamma}{1-\alpha}(2m_{II} - m_{SI}) + \frac{\gamma}{\alpha} m_{SA} \\
&\quad - \lambda \frac{k}{2} m_{SI} \frac{m_{SI} + m_{SA}}{n_S} - \lambda m_{SI} - r m_{SI}(n_S + n_A), \\
\dot{m}_{SS} &= \frac{\gamma}{1-\alpha} m_{SI} - \lambda m_{SS} k \frac{m_{SI} + m_{SA}}{n_S} + r n_S m_{SI}, \\
\dot{m}_{II} &= -2\frac{\gamma}{1-\alpha} m_{II} + \frac{\gamma}{\alpha} m_{AI}, \\
\dot{m}_{AI} &= \frac{\gamma}{\alpha}(2m_{AA} - m_{AI}) - \frac{\gamma}{1-\alpha} m_{AI} \\
&\quad + \lambda \frac{k}{2} m_{SI} \frac{m_{SI} + m_{SA}}{n_S} + \lambda m_{SI} - r m_{AI}(n_S + n_E), \\
\dot{m}_{AA} &= -2\frac{\gamma}{\alpha} m_{AA} + \lambda \frac{k}{2} m_{SA} \frac{m_{SI} + m_{SA}}{n_S} + \lambda m_{SA} + r n_A m_{AI}.
\end{aligned} \tag{6}$$

The system is completed by the equations $n_S + n_A + n_I = 1$ and $m_{SS} + m_{SA} + m_{SI} + m_{AA} + m_{AI} + m_{II} = 1$. In spite of the rather imposing look of this mathematical problem, it is still possible to find a single (sixth-order) polynomial equation for the fraction of infectives n_I at equilibrium. The equilibrium fractions of infective and asymptomatic agents are related by the simple equation $(1-\alpha)n_A = \alpha n_I$. Thus, at equilibrium, α exactly coincides with the fraction of agents carrying the infection which do not exhibit symptoms. This, in turn, makes it possible to find the equilibrium fraction of susceptibles, $n_S = 1 - n_I/\alpha$.

As in the models studied in Sections 2 and 3, the infection-free state, $n_I = 0$, is an equilibrium solution for any value of the parameters. The transcritical bifurcation, at which $n_I = 0$ changes its stability, occurs now at a critical infectivity $\tilde{\lambda}_T = [1 + (1-\alpha)\tilde{r}]/k[1 + (1-\alpha)\alpha\tilde{r}]$. As before, $\tilde{\lambda}_T$ grows monotonically with $\tilde{r}$. Here, however, this growth is not unbounded, and the critical infectivity tends to $\tilde{\lambda}_\infty = (k\alpha)^{-1}$ for $\tilde{r} \to \infty$. In other words, for $\tilde{\lambda} > \tilde{\lambda}_\infty$ no reconnection rate is large enough to suppress the disease. This can be understood by inspecting the basic reproductive number corresponding to the present epidemiological process,

$$R_0 = \frac{k\tilde{\lambda}}{\tilde{\lambda} + \alpha^{-1}} + \frac{k\tilde{\lambda}}{\tilde{r} + \tilde{\lambda} + (1-\alpha)^{-1}}. \tag{7}$$

The two terms in the right-hand side of this equation arise from the number of secondary infections, respectively, generated during the asymptomatic and symptomatic phases. As expected, only the latter depends on the reconnection parameter. But, for $\tilde{\lambda} > \tilde{\lambda}_\infty$, the first contribution is already larger than one. In other words, the infectiveness is so high that when symptoms appear—and reconnection is turned on—the infective agent has already caused enough secondary infections to trigger the spreading of the infection.

A numerical analysis of the equilibrium solutions of the system in Eq. (6) reveals that, as before, a tangent bifurcation occurs at a critical infectivity smaller that $\tilde{\lambda}_T$, for sufficiently large reconnection rates. Fig. 6 shows the phase diagrams for three values of α. We see that the critical infectivity corresponding to the tangent bifurcation varies non-monotonically with $\tilde{r}$: it reaches a maximum $\tilde{\lambda}_2$ at an intermediate value of the reconnection rate, and then approaches a limit $\tilde{\lambda}_1$ as $\tilde{r} \to \infty$, with $\tilde{\lambda}_1 < \tilde{\lambda}_2 < \tilde{\lambda}_T$.

Thus, the picture of the effects of reconnection is as follows. For large values of infectivity, $\tilde{\lambda} > \tilde{\lambda}_\infty$, the endemic state is stable for all $\tilde{r}$. When the infectivity is in the interval $(\tilde{\lambda}_2, \tilde{\lambda}_T)$, increasing $\tilde{r}$ takes the system from the endemic state to the bistable region, which here extends to arbitrarily large values of the reconnection rate. Within this region, as discussed in Section 2, there is a threshold initial density of infectives necessary to sustain the epidemics. This breakpoint density grows with the reconnection rate and, for $\tilde{r} \to \infty$, approaches a value smaller than the corresponding equilibrium fraction of infectives. Under these conditions, even an arbitrarily large reconnection rate is unable to suppress the infection, and high values of the initial fraction of infectives asymptotically lead to the endemic state. For infectivities in $(\tilde{\lambda}_1, \tilde{\lambda}_2)$, there is an intermediate interval of reconnection rates where the system reaches the infection-free state. For sufficiently large values of $\tilde{r}$, though, the system returns to the bistable region. Finally, for infectivities smaller than λ_1, the situation is the same as in the previously discussed models: increasing reconnection leads the system from the endemic state to the bistable region and, upon further increase of $\tilde{r}$, the system reaches the infection-free zone.

Coming back to the question raised at the beginning of this section, about the possibility of reconnection having unwanted effects in the spread of the disease, the results shown in Fig. 7 suggest that the answer is not straightforward. As a function of the reconnection rate $\tilde{r}$, the stable equilibrium fraction of infectives can grow or decrease and, even for a given value of $\tilde{r}$ and $\tilde{\lambda}$, the sign of the derivative of n_I with respect to $\tilde{r}$ depends on α. As a partial characterization of this diversity of behavior, we analyze the sign of $\mathrm{d}n_I/\mathrm{d}\tilde{r}$ for $\tilde{r} \to 0$, i.e. the slope of the stable branches depicted in Fig. 7 for small values of the reconnection rate. Fig. 8 shows the zones of the parameter plane $(\tilde{\lambda}, \alpha)$ where the equilibrium fraction of infectives *increases* with the reconnection rate, for $\tilde{r} \to 0$, and for two values of k. It can be shown that, nevertheless, the growth of n_I within this regions is small if the infection level is itself low. In any case, in most of the parameter plane, the fraction of infectives exhibit the desirable decline of the infection level with reconnection.

It is tempting to apply these results to the propagation of real diseases. The parameter α can be associated with the number of infections that occur before the onset of clinical symptoms (Fraser et al., 2004). Using estimates of this number, we can have an idea of how contact switching might work for these infections. For gonorrhea, a rough estimate, using data from Kretzschmar et al. (1997), gives $\alpha \approx 0.2$ and $\tilde{\lambda} \approx 4$. Being a sexually transmitted disease, $k = 3$ is a reasonable assumption. Using these values, we see from the left panel of Fig. 8 that gonorrhea would be in the zone where contact switching is not effective. For influenza, the estimate gives $\alpha \approx 0.4$ (Fraser et al., 2004). Since, in contrast with gonorrhea, influenza is an airborne disease, the number of contacts that may be infected is large. Using $k = 10$, the right panel of Fig. 8 shows that, almost for any infectivity, contact switching might be an effective strategy to curb the epidemic outbreak. Needless to say, any application of these ideas should be based both on more realistic models, taking into account specific features of the disease under study, as well as on more sound estimates of the parameters involved.

5. Discussion and conclusion

We have studied the effects of contact switching as a strategy to prevent the spread of an epidemic disease. Our analysis confirms, and extends in several directions, the results obtained

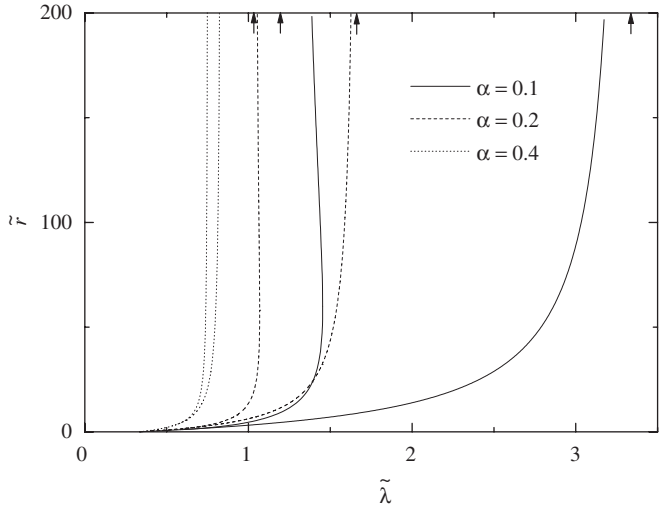

Fig. 6. Phase diagram for a disease with an asymptomatic stage of duration α/γ, for $k = 3$, and three values of α. For each of them, the left-most and right-most curves represent the tangent and transcritical bifurcations, respectively. Compare with Figs. 2 and 5. The arrows indicate the position of the vertical asymptotes of the curves for $\alpha = 0.1$ and 0.2: from right to left, they correspond to $\tilde{\lambda}_1$ ($\alpha = 0.1$), $\tilde{\lambda}_1$ ($\alpha = 0.2$), $\tilde{\lambda}_\infty$ ($\alpha = 0.1$), $\tilde{\lambda}_\infty$ ($\alpha = 0.2$). No arrows have been drawn for $\alpha = 0.4$, because they would fall on top of the corresponding curves.

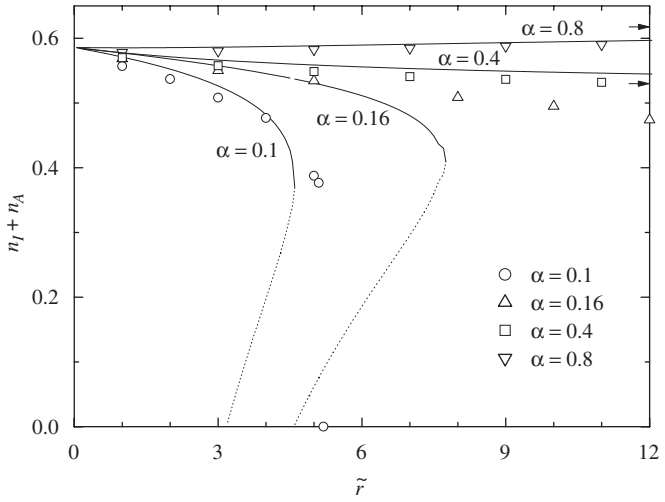

Fig. 7. Bifurcation diagram for the total fraction of agents carrying the infection, $n_I + n_A$, for a disease with an asymptomatic stage of duration α/γ, for $k = 3$, $\tilde{\lambda} = 1$ and several values of α. Full and dashed lines represent stable and unstable equilibrium solutions, respectively. Symbols are simulation results for a system of 5000 agents with $\gamma = 1$. The arrows indicate the asymptotic value of n_I for $\alpha = 0.4$ and 0.8.

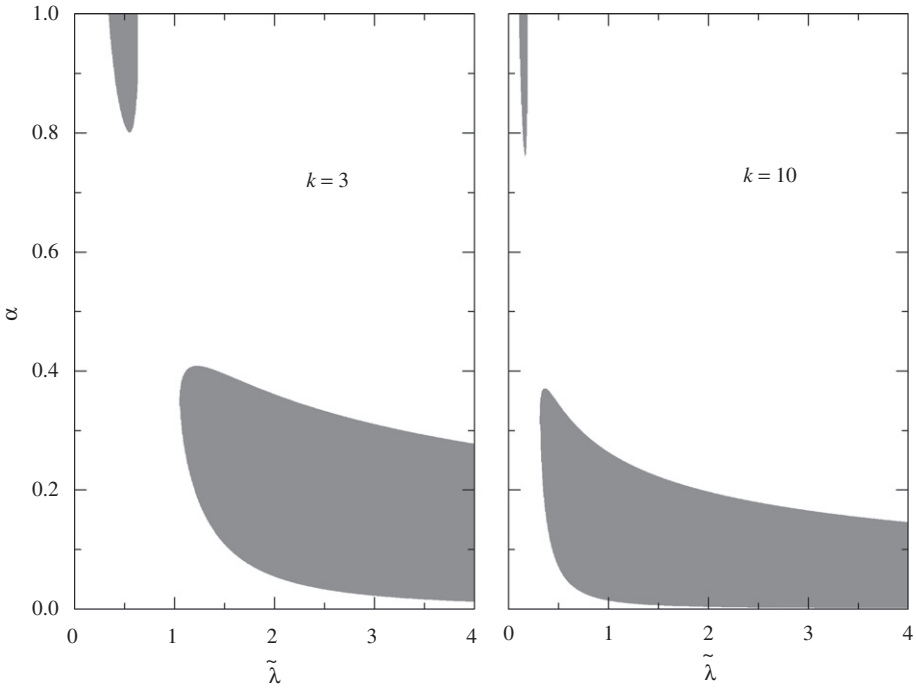

Fig. 8. Phase diagrams showing, for two connectivities, the regions (shaded) in parameter space $(\tilde{\lambda}, \alpha)$ where the derivative of the fraction of agents that have contracted the disease with respect to $\tilde{r}$, at $\tilde{r} = 0$, is positive.

in previous works (Gross et al., 2006; Zanette and Risau-Gusman, 2008). In particular, we have here relaxed some important constraints, for instance, the assumption that every agent knows the state of every other agent (Gross et al., 2006), and the assumption that the mean number of contacts per agent is large (Zanette and Risau-Gusman, 2008). In the basic model that we analyzed first, susceptible agents with infective neighbors are allowed to reconnect these links at rate r. For small values of this reconnection parameter, there is a continuous transition—through a transcritical bifurcation—from an infection-free state to an endemic state, as the infectivity grows. When r is larger than a certain critical value, on the other hand, the transition becomes discontinuous and gives rise to a bistability region where the infection can become asymptotically extinct or endemic. In this situation, the asymptotic state is determined by the initial fraction of infectives. Further increase of the infectivity, however, makes the endemic state globally stable, and the infection-free state becomes unstable.

In their model, Gross et al. (2006) (see also Gross and Kevrekidis, 2008) have observed an oscillatory regime in parameter space, separating the endemic and infection-free regions. We have not found evidence of such behavior in our system, but cannot discard that an exhaustive search in parameter space could disclose regions where oscillations occur.

One crucial feature of our basic model is that only susceptibles are allowed to reconnect. Even though this constraint might seem reasonable, the voluntary nature of contact switching suggests relaxing it. We have analyzed a model where both types of agents can switch contacts, with a new parameter p giving the probability that it is the infective agent who breaks and reconnects the link with a susceptible neighbor. Remarkably, we find that, for certain values of the parameters that characterize the disease, reconnection can completely suppress the infection for intermediate values of p, but not for larger or smaller values. Furthermore, even when the disease is not completely suppressed, equilibrium infection levels can be lower when more infectives are allowed to reconnect. Even though, at first sight, this might seem counterintuitive, it can be readily understood, at least for high infection levels: if the fraction of infectives is large, susceptibles are likely to reconnect their broken links to other infectives, thus not altering their risk of getting infected. On the other hand, when an infective agent reconnects, which with high probability occurs to other infectives, the number of "dangerous" links decreases.

It has been shown that the outcome of many strategies to control epidemic outbreaks depends not only on the reproductive number of the disease, but also on the number of infections caused by an agent before the onset of clinical symptoms (Fraser et al., 2004). In the case of contact switching, it is even possible that the presence of an asymptomatic phase leads to an acceleration of the spread of the disease. In Section 4 we analyzed an extension of the basic model, introducing a parameter α which gives, on average, the fraction of time that the agent spends in an asymptomatic phase during the infection. In this phase, the agent behaves as a susceptible individual, but can transmit the infection through contagion. We find that, in this case, reconnection does lead to higher levels of infection, but this effect is restricted to some limited regions of parameter space. Furthermore, within these regions, the increase of this infection level due to reconnection is rather small, unless its value is already very high in the absence of reconnection.

It should be clear that any significant assessment of the benefits and risks of contact switching must use sound estimates of all the variables involved, applying them to realistic models of the analyzed population. Important features that may be added to the above models are, for instance, the population division into females and males—who often exhibit rather different epidemiological responses—and age groups, as well as more complex degree distributions in the initial network. Such generalizations, however, will hardly be amenable to analytical treatment. In our basic model and its extensions, on the other hand, most epidemiological features can be studied analytically. It is only after these have been fully understood that more complex models should be tackled.

Appendix A

Models of epidemic spreading can be studied as stochastic contact processes on networks (Levin and Durrett, 1996). We illustrate the procedure with the basic SIS model presented in Section 2. For each site x of the network we introduce the probability $P_t(A_x)$ that the agent at x is in state A (either S or I) at time t. The evolution equation for this probability is

$$\dot{P}_t(I_x) = -\dot{P}_t(S_x) = -\gamma P_t(I_x) + \lambda \sum_{y\neq x} P_t(S_x{\sim}I_y), \tag{A.1}$$

where γ is the rate at which an infective agent becomes susceptible, and λ is the rate at which infective agents infect each of their susceptible neighbors. Here, $P_t(S_x{\sim}I_y)$ denotes the probability that the agents in sites x and y are susceptible and infective, respectively, and that they are connected. Generally, we also introduce $P_t(A_x{\sim}B_y)$ as the probability that an agent in state A at site x and an agent in state B at site y are connected, and $P_t(A_x \nsim B_y)$ as the probability that the same agents are *not* connected. The evolution equations for these two-site probabilities are

$$\begin{aligned}
\dot{P}_t(S_x{\sim}I_y) = {} & \gamma P_t(I_x{\sim}I_y) - \gamma P_t(S_x{\sim}I_y) + \lambda \sum_{z\neq x,y} P_t(S_x{\sim}S_y{\sim}I_z) \\
& - \lambda \sum_{z\neq x,y} P_t(I_y{\sim}S_x{\sim}I_z) - \lambda P_t(S_x{\sim}I_y) \\
& + r \sum_{z\neq x,y} \frac{P_t(I_y \nsim S_x{\sim}I_z)}{\sum_{v\neq z} P_t(S_y \nsim O_v)} - r \sum_{z\neq x,y} \frac{P_t(I_y{\sim}S_x \nsim O_z)}{\sum_{v\neq x} P_t(S_y \nsim O_z)},
\end{aligned}$$

$$\begin{aligned}
\dot{P}_t(S_x{\sim}S_y) = {} & \gamma(P_t(I_x{\sim}S_y) + P_t(S_x{\sim}I_y)) \\
& - \lambda\left(\sum_{z\neq x,y} P_t(S_x{\sim}S_y{\sim}I_z) + \sum_{z\neq x,y} P_t(S_y{\sim}S_x{\sim}I_z)\right) \\
& + r\left(\sum_{z\neq x,y} \frac{P_t(S_x \nsim S_y{\sim}I_z)}{\sum_{v\neq z} P_t(S_y \nsim O_v)} + \sum_{z\neq x,y} \frac{P_t(S_y \nsim S_x{\sim}O_z)}{\sum_{v\neq x} P_t(S_y \nsim O_z)}\right),
\end{aligned}$$

$$\begin{aligned}
\dot{P}_t(I_x{\sim}I_y) = {} & -2\gamma P_t(I_x{\sim}I_y) + \lambda(P_t(S_x{\sim}I_y) + (S_y{\sim}I_x)) \\
& + \lambda \sum_{z\neq x,y} (P_t(I_x{\sim}S_y{\sim}I_z) + P_t(I_y{\sim}S_x{\sim}I_z)),
\end{aligned}$$

$$\begin{aligned}
\dot{P}_t(S_x \nsim I_y) = {} & \gamma P_t(I_x \nsim I_y) - \gamma P_t(S_x \nsim I_y) \\
& + \lambda \sum_{z\neq x,y} P_t(S_x \nsim S_y{\sim}I_z) - \lambda \sum_{z\neq x,y} P_t(I_y \nsim S_x{\sim}I_z) \\
& - r \sum_{z\neq x,y} \frac{P_t(I_y{\sim}S_x \nsim I_z)}{\sum_{v\neq z} P_t(S_y \nsim O_v)} - r \sum_{z\neq x,y} \frac{P_t(I_y \nsim S_x{\sim}O_z)}{\sum_{v\neq x} P_t(S_y \nsim O_z)},
\end{aligned}$$

$$\begin{aligned}
\dot{P}_t(I_x \nsim I_y) = {} & -2\gamma P_t(I_x \nsim I_y) \\
& + \lambda \sum_{z\neq x,y} (P_t(I_x \nsim S_y{\sim}I_z) + P_t(I_y \nsim S_x{\sim}I_z)),
\end{aligned} \tag{A.2}$$

where r is the rate at which a susceptible–infective link is broken, and the susceptible agent reconnected to a random agent. The remaining two-site probability $P_t(S_x \nsim S_y)$ is obtained from the fact that the sum of all two-site probabilities equals unity. The interpretation of the different summands in each equation is rather straightforward. In the first equation, for instance, the first two terms account for the probability that a connected susceptible–infective pair is created from an infective–infective pair, or destroyed, by the spontaneous cure of an infective agent. The third and fourth terms account for the probability that a neighbor outside the pair infects agent y (thus creating the SI pair) or agent x (thus destroying the SI pair). The fifth term accounts for the probability that infection is transmitted from the infective agent to the susceptible agent in the same pair. The last two terms account for the probability that the pair is created or destroyed by the reconnection process. In these terms, the denominators take into account the fact that, when the susceptible agent reconnects a link, the new contact must exclude the previous neighbors of the agent in question.

Within the *pair approximation* (Levin and Durrett, 1996), the three-site probabilities in Eq. (A.2) can be approximately given in terms of two-site probabilities, thus closing the equation system. The basic idea of this approximation is that, in a chain of three of connected sites, $x{\sim}y{\sim}z$, the mutual interactions between the central site y of and each the other two neighbors, x and z, can be decoupled. In particular, we have used that

$$\begin{aligned}
P_t(S_x{\sim}S_y{\sim}I_z) &\approx K_1 \frac{P_t(S_x{\sim}S_y)P_t(S_y{\sim}S_z)}{P_t(S_y)}, \\
P_t(I_x{\sim}S_y{\sim}I_z) &\approx K_2 \frac{P_t(I_x{\sim}S_y)P_t(S_y{\sim}S_z)}{P_t(S_y)},
\end{aligned} \tag{A.3}$$

where K_1 and K_2 are numerical constants.

We assume that all the probabilities are homogeneous over the population, i.e. that they do not depend on the site, which is true if the initial probabilities are also homogeneous. By averaging all quantities, it can be shown that Eqs. (A.3) are equivalent to

$$\begin{aligned}
\langle k_S k_{SI}\rangle - \langle k_{SI}^2\rangle &\approx K_1(\langle k_S\rangle\langle k_{SI}\rangle - \langle k_{SI}\rangle^2), \\
\langle k_{SI}^2\rangle - \langle k_{SI}\rangle &\approx K_2\langle k_{SI}\rangle^2,
\end{aligned} \tag{A.4}$$

where k_S denotes the number of neighbors of a susceptible, k_{SI} the number of infected neighbors of a susceptible, and the brackets an average over the population. As is usually done (Levin and Durrett, 1996), we assume the ansatz $K_1 = K_2 \equiv K = (k-1)/k$. Simulations show that, even though this ansatz gives qualitatively good results, in general a much better fit is obtained by using $K = 1$. For the model of Section 3, however, Fig. 4 shows that the choice $K = 1$ does not give a good fit to the simulations. This happens because in this case the dynamics generates a distribution of k_{SI} whose width grows with p, so that K_2 becomes much larger than 1.

We also assume that the epidemiological states at sites that are not connected can be taken as independent, i.e. $P_t(A \nsim B{\sim}C) \approx P_t(A)P_t(B{\sim}C)$ and $P_t(A \nsim B \nsim B) \approx P_t(A)P_t(B{\sim}C)$ (using also the pair approximation). A further approximation that simplifies the equations considerably is to assume that the number of neighbors of each site is much smaller than the total number of agents so that, when an agent reconnects, the probability that another specific agent becomes the new contact is simply $(N-1)^{-1}$, thus simplifying the denominator of the reconnection terms.

To write the equations in terms of the fractions of agents, n_S and n_I, and links, m_{SI}, m_{SS} and m_{II} we take into account the relations

$$\begin{aligned}
n_S(t) &= P_t(S), \\
m_{SS}(t) &= \frac{N-1}{k} P_t(S{\sim}S), \\
m_{SI}(t) &= \frac{2(N-1)}{k} P_t(S{\sim}I),
\end{aligned} \tag{A.5}$$

and $n_I = 1 - n_S$, $m_{II} = 1 - m_{SS} - m_{II}$. Using these equivalences, and the approximations mentioned above, Eqs. (1) are obtained from system (A.2).

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