Coevolution of agents and networks in an epidemiological model

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Abstract

We study the spreading of an infection within an SIS epidemiological model on a network. Susceptible agents are given the opportunity of breaking their links with infected agents, and reconnecting those links with the rest of the population. Thus, the network coevolves with the population as the infection progresses. We show that a moderate reconnection frequency is enough to completely suppress the infection. A partial, rather weak isolation of infected agents suffices to eliminate the endemic state.

1 Introduction

Among the many potential applications of agent-based models whose pattern of interactions is represented by a network, a broad class is constituted by systems where that pattern is not a static structure, but evolves in response to the changing state of the agents. Generally, the evolution of the interaction network and the dynamics of individual agents occur over different time scales. For instance, in learning processes –such as those implemented in real and artificial neural networks [1]– connections change adaptively over scales that are large as compared with the internal dynamics of agents. At the opposite limit, in models of network growth, the pattern evolves in the absence of any dynamics related to agents at the network nodes [2].

When, on the other hand, the dynamical time scales of a population of agents and its interaction network are comparable, we can speak about their coevolution. Consider, for instance, agents involved in a cooperation-defection game, such as in the prisoner’s dilemma [3], where defected agents are given the opportunity of breaking the connection with their defectors, in such a way that no further interaction is possible between them. Or, in a process of opinion diffusion, that agents which do not succeed at reaching an

1 This is a written version of a talk to be given at the International School on Complexity: Course on Statistical Physics of Social Dynamics: Opinions, Semiotic Dynamics, and Language (Erice, 13-20 July, 2007).
agreement may elude mutual contacts in the future [4, 5, 6, 7]. In such cases, a realistic assumption would be that the breaking of interaction links occurs just after a few, or even one of, such events.

In this talk, we explore a model of agent-network coevolution in a population where an infection is spreading, such that non-infected agents are given the possibility of avoiding contact with their infected partners –perhaps in response to risk perception [8]. The model is based on an SIS epidemiological process, where each agent can be susceptible (S) or infected (I). We recall that in the standard SIS process each infected agent recovers and becomes susceptible at a fixed rate, say, with probability $\gamma$ per unit time. Susceptible agents, in turn, become infected by contagion from infected agents, at a rate proportional to the infection probability per unit time, $\rho$, and to the fraction of infected agents, $n_I$. Within a mean-field description of the standard SIS model, the fraction of infected agents obeys

$$\dot{n}_I = -\gamma n_I + \rho n_I n_S,$$

where $n_S = 1 - n_I$ is the fraction of susceptible agents. In this description, for asymptotically long times, $n_I$ vanishes if $\rho \leq \gamma$. Therefore, the infection is suppressed as time elapses. If, on the other hand, $\rho > \gamma$, the fraction of infected agents approaches a finite value $n_I^* = 1 - \frac{\gamma}{\rho} > 0$, and the infection is endemic. The transition between these two regimes occurs through a transcritical bifurcation.

We introduce in the following an implementation of the SIS model on a network, with agents occupying the nodes and contagion taking place along the links. A susceptible agent can become infected only if it is connected to an infected agent. Moreover, susceptible agents have the opportunity, with a certain probability, of breaking their connection with an infected partner before contagion takes place, and reconnect the broken link with any other agent in the population. We show that these reconnection events are able to control infection spreading, even to the point of completely suppressing the infection as the reconnection probability grows. This suppression is achieved with a moderate decrease in the number of network connections per infected agent.

2 Model and mean-field formulation

The present model is a variation of the model introduced by Gross et al. [9]. Consider a population of $N$ agents at the nodes of a network with $M$ links. The average number of neighbours per agent is $z = 2M/N$. At a given time,
each agent can be in one of two states: susceptible (S-agent) or infected (I-agent). Initially, the $M$ links are distributed at random over the population, and there is a certain fraction of agents in each state.

Each evolution step is divided into two sub-steps. In the first sub-step, an agent is chosen at random from the whole population. If it is infected, it recovers with probability $\gamma$, and becomes susceptible. In the second sub-step, a pair of linked agents is chosen at random. If both agents are susceptible or infected, nothing happens. Otherwise, the S-agent is given the chance to break the link with its infected neighbour and reconnect it with another agent, taken at random from the remaining of the population. This rewiring happens with probability $r$. Finally, if the rewiring has not occurred, the S-agent becomes infected with probability $\lambda$.

A time unit contains $N$ evolution steps. Thus, each I-agent has a probability $\gamma$ per time unit of becoming susceptible, so that the mean duration of the infection period is $\gamma^{-1}$. Moreover, each S-agent in contact with an I-agent becomes in turn infected with probability $2(1-r)\lambda$ per time unit.

### 2.1 Mean-field equations

Although, clearly, the model is defined bearing in mind its implementation as a numerical simulation, a formulation in terms of differential equations, from mean-field arguments, turns out to give an essentially correct description of the system. To define our mean-field formulation, we first introduce a suitable set of variables. We call $N_I$ and $N_S$ the number of infected and susceptible agents, respectively. They satisfy $N_I + N_S = N$. Moreover, we denote by $M_{II}$, $M_{IS}$, and $M_{SS}$, respectively, the number of network links joining two infected agents (II), an infected agent and a susceptible agent (IS), and two susceptible agents (SS). Since the total number of links is preserved by the dynamical rules, we have $M_{II} + M_{IS} + M_{SS} = M$ constant at all times. Differential equations will be formulated for the fractions $n_I = N_I/N$, $m_{II} = M_{II}/M$, and $m_{IS} = M_{IS}/M$. The conservation of the number of agents and links implies $n_S = N_S/N = 1 - n_I$ and $m_{SS} = M_{SS}/M = 1 - m_{II} - m_{IS}$.

The evolution of the above defined quantities is given by the events of infection and recovery, and by the reconnection of links. When, for instance, an I-agent becomes susceptible, there is not only a decay in the fraction of I-agents, but also a change in the fractions $m_{II}$ and $m_{IS}$. In fact, the links joining the recovered agent with I and S-agents pass, respectively, from the II-type to the IS-type, and from the IS-type to the SS-type. A symmetric situation occurs when an S-agent becomes infected. The number of links of each type associated to a given agent is calculated using mean-field-like averages. For instance, the number of II-links associated to an I-agent is
estimated as $2M_{II}/N_I = zm_{II}/n_I$, where $z = 2M/N$ is the overall average number of links per agent. Similarly, the number of IS-links associated to an I-agent is taken to be $M_{IS}/N_I = zm_{IS}/2n_I$. Reconnection events, in turn, can change an IS-link into an SS-link. The probability for this change is proportional to the fraction of S-agents in the whole population and to the reconnection probability $r$. Finally, it has to be taken into account that both infection and reconnection only occur when an IS-link is selected, so that their probabilities are proportional to $m_{IS}$.

Putting all these considerations together, the evolution equations for $n_I$, $m_{II}$, and $m_{IS}$ read

$$\begin{align*}
n_I' &= -n_I + \tilde{\lambda}m_{IS}, \\
m_{II}' &= -2m_{II} + \tilde{\lambda}m_{IS}^2/(1 - n_I), \\
m_{IS}' &= 2m_{II} - m_{IS} - \tilde{r}(1 - n_I)m_{IS} \\
&\quad + \lambda m_{IS}(2 - 2m_{II} - 3m_{IS})/(1 - n_I),
\end{align*}$$

with $\tilde{\lambda} = (1 - r)\lambda/\gamma$ and $\tilde{r} = 2r/z\gamma$. Primes indicate differentiation with respect to the rescaled time $t' = \gamma t$. In this formulation, thus, the inverse recovery probability $\gamma^{-1}$ fixes an overall time scale, and the infection and reconnection probabilities are accordingly redefined as $\tilde{\lambda}$ and $\tilde{r}$, which are the only two parameters left. Note that $\tilde{\lambda}$ depends on both $\lambda$ and $r$, and that $\tilde{r}$ incorporates the only network-specific parameter, namely, the average connectivity per agent $z = 2M/N$.

In the absence of reconnection events, $r = 0$, and provided that we put $m_{II} = n_I^2$ and $m_{IS} = 2n_I n_S = 2n_I(1 - n_I)$, the three lines in Eq. (2) collapse into

$$n_I' = -n_I + 2\tilde{\lambda}n_I(1 - n_I).$$

Rewriting this equation in terms of the non-normalized parameters, we reobtain the mean-field equation (1) for the SIS model, with $\rho = 2\lambda$. The factor of 2 relating the infection frequencies $\rho$ and $\lambda$ originates in the fact that, in our implementation of the SIS model on a network, each agent is (on the average) chosen twice per unit time to possibly become infected.

### 2.2 Infection level at equilibrium

We focus the attention on the equilibrium solutions of Eqs. (2), which are the candidates to represent the infection level and network structure at asymptotically long times. First, we consider the stationary values of the fraction of infected agents. The analysis is restricted to the case of $\tilde{\lambda} > 1/2$ which, in the absence of reconnection events, corresponds an endemic infection where a finite fraction of the population is infected at all times, $n_I > 0$ for $t \to \infty$. 

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At the fixed points of the dynamical equations (2), the equilibrium fraction of links \( m^*_\text{II} \) and \( m^*_\text{IS} \) are related to the equilibrium fraction of infected agents \( n^*_I \) as
\[
m^*_\text{II} = \frac{n^*_I^2}{2\lambda(1-n^*_I)}, \quad m^*_\text{IS} = \frac{n^*_I}{\lambda}.
\] (4)

In turn, \( n^*_I \) satisfies
\[
0 = n^*_I \left[ 2\lambda - 1 - \tilde{r} + (3\tilde{r} - 2\lambda)n^*_I - 3\tilde{r}n^*_I^2 + \tilde{r}n^*_I^3 \right].
\] (5)

This polynomial equation has four solutions. One of them tends to infinity for \( \tilde{r} \to 0 \), and remains real and larger than one for any positive \( \tilde{r} \). Since meaningful solutions to our problem must verify \( n^*_I \leq 1 \), we disregard this solution from now on.

The trivial solution \( n^{(0)}_I = 0 \) exists for any value of the normalized infectivity \( \lambda \) and of the normalized reconnection probability \( \tilde{r} \). For a given \( \lambda \), its stability depends on \( \tilde{r} \). As discussed in more detail below, \( n^{(0)}_I = 0 \) is unstable for small \( \tilde{r} \) and becomes stable as \( \tilde{r} \) grows. The other two solutions read
\[
n^{(1,2)}_I = 1 - \sqrt{\frac{2\lambda}{3\tilde{r}}} \left[ \cos \frac{\alpha}{3} \mp \sqrt{3} \sin \frac{\alpha}{3} \right]
\] (6)
with
\[
\alpha = \arctan \sqrt{\frac{32\lambda^3}{27\tilde{r}} - 1}
\] (7)
(0 \leq \alpha \leq \pi/2). These two solutions are real for \( 32\lambda^3 \geq 27\tilde{r} \). Otherwise, they are complex conjugate numbers. The solution \( n^{(1)}_I \), with the minus sign in the right-hand side of Eq. (6), approaches \( 1 - (2\lambda)^{-1} \) for \( \tilde{r} \to 0 \). Thus, it represents the expected fraction of infected agents in the absence of reconnection. When it is real, it satisfies \( n^{(1)}_I < 1 \), and it is stable as long as it remains positive. Consequently, along with the trivial solution, \( n^{(1)}_I \) is another meaningful equilibrium solution to our problem. Finally, \( n^{(2)}_I \) is negative and stable for small \( \tilde{r} \). Depending on \( \lambda \), it can become positive as \( \tilde{r} \) grows but, at the same time, it becomes unstable. Therefore, it does not represent a meaningful solution.

Figure[1] summarizes, in a bifurcation diagram, the behaviour of \( n^{(0)}_I \), \( n^{(1)}_I \), and \( n^{(2)}_I \) as functions of the normalized reconnection probability \( \tilde{r} \), for three representative values of the normalized infectivity \( \lambda \). In the three cases, we have \( \lambda > 1/2 \), so that –as discussed above– a non-trivial meaningful solution does exist. Full and dotted lines represent, respectively, stable and unstable
Figure 1: Bifurcation diagram for the equilibrium fraction of infected agents $n_1^*$ as a function of the normalized reconnection probability $\tilde{r}$, for three normalized infectivities $\tilde{\lambda}$. Although only positive values of $n_1^*$ are meaningful, an interval in the negative domain is also shown for completeness. Full and dotted lines represent, respectively, stable and unstable branches. For clarity, the solution $n_1^{(0)} = 0$ is plotted in the vicinity of the transcritical bifurcation only.
branches. For small infectivity ($\tilde{\lambda} = 0.6$), the stable solution $n_1^{(1)}$ crosses $n_1^{(0)}$ and becomes negative and unstable, while $n_1^{(0)}$ becomes stable. This transcritical bifurcation takes place at $\tilde{r} = 2\tilde{\lambda} - 1$. As $\tilde{r}$ grows further, $n_1^{(1)}$ and the negative stable solution $n_1^{(2)}$ approach each other, and collide when $n_1^{(1)} = n_1^{(2)} = 1 - 3/4\tilde{\lambda}$. Beyond this tangent bifurcation, which takes place at $\tilde{r} = 32\tilde{\lambda}^3/27$, the two solutions are complex numbers.

The situation is different for larger infection probabilities, as illustrated by Fig. 1 for $\tilde{\lambda} = 1$. Now, for $\tilde{r} = 0$, $n_1^{(1)}$ is large and, as $\tilde{r}$ grows, it is the stable negative solution $n_1^{(2)}$ which first reaches $n_1^{(0)}$. At the transcritical bifurcation at $\tilde{r} = 2\tilde{\lambda} - 1$, $n_1^{(2)}$ becomes positive and unstable, and $n_1^{(0)}$ becomes stable. The tangent bifurcation where $n_1^{(1)}$ and $n_1^{(2)}$ collide and become complex, at $\tilde{r} = 32\tilde{\lambda}^3/27$, takes now place when these two solutions are positive. As a consequence, there is an interval of normalized reconnection probabilities, between the two bifurcations, where the system is bistable: both $n_1^{(0)}$ and $n_1^{(1)}$ are stable meaningful solutions to the problem. The asymptotic state is selected by the initial condition for $n_1$, and $n_1^{(2)}$ stands at the boundary between the two attraction basins.

The regimes of small and large infectivity are separated by the critical value $\tilde{\lambda} = 3/4 = 0.75$, also shown in Fig. 1. At this critical point, the transcritical and the tangent bifurcation collapse into a pitchfork bifurcation at $\tilde{r} = 1/2$. Here, the three equilibria collide simultaneously, and $n_1^{(0)}$ becomes stable, while the other two solutions become complex.

A phase diagram of our system over the parameter plane $(\tilde{r}, \tilde{\lambda})$ is shown in Fig. 2. The zones of endemic infection, where the fraction of infected agents at asymptotically long times is positive ($n_1 \to n_1^{(1)}$), and of infection suppression ($n_1 \to 0$) are separated, for large $\tilde{\lambda}$ and $\tilde{r}$, by the bistability region, where both asymptotic behaviours can be obtained, depending on the initial condition. The three zones are limited by the lines of the transcritical bifurcation [$\tilde{\lambda} = (1 + \tilde{r})/2$, TC], where $n_1^{(0)} = 0$ changes its stability, and of the tangent bifurcation [$\tilde{\lambda} = (27\tilde{r}/32)^{1/3}$, Tg] where $n_1^{(1)}$ and $n_1^{(2)}$ collide and become complex. These two lines are tangent to each other at the “triple point” $(1/2, 3/4)$, where the bistability region disappears, and the system undergoes a pitchfork bifurcation [P]. For smaller $\tilde{\lambda}$ and $\tilde{r}$ bistability is no more possible, and the zones of infection persistence and suppression are separated by the transcritical line. The tangent bifurcation takes now place at negative values of $n_1^{(1)}$ and $n_1^{(2)}$ (dotted line).

The inset of Fig. 2 shows the same phase diagram in terms of the original, non-normalized reconnection and infection probabilities, $r$ and $\lambda$, for a recovery probability $\gamma = 0.01$ over a network with an average of $z = 10$
Figure 2: Phase diagram in the $(\tilde{r}, \tilde{\lambda})$-plane, showing the regions of infection suppression ($n_I \rightarrow 0$) and persistence ($n_I \rightarrow n_I^{(1)}$), and the intermediate bistability zone. Their boundaries are given by the transcritical (TC) and the tangent (Tg) bifurcation lines, which collapse into a pitchfork bifurcation (P) at $(1/2, 3/4)$. The dotted line is the continuation of the tangent bifurcation line in the zone where $n_I^{(1)}$ is negative. The inset shows the same phase diagram in terms of the non-normalized parameters $r$ and $\lambda$, for $\gamma = 0.01$ and $z = 10$. 
neighbours per site. Note that the relation between non-normalized and normalized parameters is not a mere change of scale, because both $r$ and $\lambda$ enter the definition of $\tilde{\lambda}$.

Let us summarize our results on the persistence or suppression of the infection in terms of the non-normalized parameters. First, for small infectivity, $\lambda \leq \gamma/2(1 - r)$, the infection is always suppressed. In this situation, the infectivity is just too small to sustain a finite infected population. For larger infectivities, on the other hand, the infection can become established, depending on the reconnection probability $r$. In the range $\gamma/2(1 - r) < \lambda < 3\gamma/4(1 - r)$, the infection is endemic if reconnections are infrequent, $r < z(\lambda - \gamma/2)/(1 + z\lambda)$. Otherwise, for sufficiently frequent reconnections, the infection dies out. The transition between both situations is continuous in the fraction of infected agents, and occurs through a transcritical bifurcation. For even larger infection probabilities, $\lambda > 3\gamma/4(1 - r)$, the regimes of persistence (low $r$) and suppression (large $r$) are separated by a bistability zone, where the infection persists or dies out depending on the initial fraction of infected agents. The bistability zone is limited by the transcritical bifurcation quoted above and a tangent bifurcation at a reconnection probability given by the solution to $27\gamma^2r = 16(1 - r)^3\lambda^3$. The discontinuous nature of the tangent bifurcation implies that the endemic state present in the bistability zone disappears abruptly at the boundary, with a finite jump in the asymptotic fraction of infected agents, from $n_I = 1 - \sqrt{z(1 - r)\lambda/3r} > 0$ to zero.

2.3 Number of neighbours of infected and susceptible agents

The variables $m_{II}$ and $m_{IS}$ characterize how the structure of the network is related to the state of the agents. Reconnection events favor the growth of the number of SS-links at the expense of IS-links. Thus, for $r > 0$, S-agents should asymptotically possess relatively large numbers of neighbours. The equilibrium values $m_{II}^*$ and $m_{IS}^*$ as functions of the equilibrium fraction $n_I^*$ of I-agents are given by Eqs. (4). These equations show, as expected, that the fraction of links connecting I-agents with any other agent is proportional to the fraction of I-agents itself.

In order to introduce quantities that define the connectivity of I-agents and S-agents independently of their respective fractions, we consider the average number of neighbours per agent of each type. For I-agents, for instance, the average numbers of infected and susceptible neighbours are $2M_{II}/N_I$ and $M_{IS}/N_I$, respectively. The average connectivity of I-agents, $z_I$, is the sum of
Figure 3: Connectivity of infected and susceptible agents, $z_I$ and $z_S$, relative to the overall connectivity $z = 2M/N$, for three values of the normalized infectivity $\tilde{\lambda}$, as functions of the normalized reconnection probability $\tilde{r}$. Only the values corresponding to meaningful stable solutions for the fraction of infected agents are plotted. The connectivity of infected agents is not plotted beyond the threshold of infection suppression. The vertical dashed line represents the finite jump in $z_S$ at the tangent bifurcation where the solution $n_I^{(1)}$ disappears.
these two quantities or, equivalently,

\[
\frac{z_I}{z} = \frac{1}{2\tilde{\lambda}(1 - n^*_I)}, \tag{8}
\]

which gives the ratio between \(z_I\) and the overall average connectivity per agent at equilibrium. With analogous arguments for S-agents, their average connectivity reads

\[
\frac{z_S}{z} = \frac{1}{1 - n^*_I} - \frac{n^*_I}{2\tilde{\lambda}(1 - n^*_I)^2}. \tag{9}
\]

Due to the conservation of the total number of links, \(z_I\) and \(z_S\) are univocally related. This relation can be obtained from Eqs. (8) and (9) by eliminating \(n^*_I\), which yields

\[
z_S = z_I[1 + 2\tilde{\lambda}(1 - z_I/z)]. \tag{10}
\]

In order to describe the correlation between the structure and the state of the population it is however useful to analyze both \(z_I\) and \(z_S\) as functions of the relevant parameters. Figure 3 illustrates the behaviour of \(z_I\) and \(z_S\), as described in the following, for the infection probabilities \(\tilde{\lambda}\) already considered in Fig. 1.

For \(n^*_I = n^{(1)}_I\), which stands for the stable equilibrium solution for low reconnection probabilities, both \(z_I\) and \(z_S\) approach \(z\) as \(\tilde{r} \to 0\). As expected, in the absence of reconnection events, there is no difference in the number of neighbours of infected and susceptible agents. Also, as \(\tilde{r}\) grows from zero, we have \(z_I < z < z_S\). We verify that reconnection tends to increase the connectivity of S-agents at the expense of I-agents.

The other solution relevant to the epidemiological process, \(n^*_I = n^{(0)}_I = 0\), corresponds to a purely susceptible population. Accordingly, we find \(z_S = z\). Note also that Eq. (8) predicts \(z_I = z/2\tilde{\lambda}\), but this value is never realized due to the total absence of I-agents in this state.

For \(\tilde{\lambda} \leq 3/4\), the fraction of I-agents decreases monotonically with \(\tilde{r}\) and vanishes continuously at the transcritical bifurcation –or, for \(\tilde{\lambda} = 3/4\), at the pitchfork bifurcation. The connectivity of S-agents is \(z_S = z\) both at \(\tilde{r} = 0\) and at the bifurcation. For intermediate values of the reconnection probability \(z_S\) is larger than \(z\) and attains a maximum. This maximum, which at first sight may result to be surprising, can be easily explained. In fact, to sustain a value of \(z_S\) larger than the overall average \(z\), it is necessary to have I-agents with a relatively low number of neighbours. As the infection is progressively suppressed by reconnection, the number of I-agents decreases and, accordingly, their contribution to the average number of neighbours per agent becomes less significant. At the bifurcation and beyond, S-agents must
account for the whole average, so that \( z_S \) returns to its value for \( \tilde{r} = 0 \), i.e. \( z_S = z \).

The connectivity of I-agents, in turn, is a monotonically decreasing function of \( \tilde{r} \), and reaches \( z_I = z/2\tilde{\lambda} < z \) at the bifurcation. This implies that, even at the threshold of infection suppression, I-agents maintain a finite number of neighbours within the population.

For \( \tilde{\lambda} > 3/4 \), again, the connectivity \( z_S \) associated with the solution \( n^{(1)}_I \) initially increases with \( \tilde{r} \), and attains a maximum. In the subsequent decay, however, it does not reach \( z_S = z \). In fact, \( n^{(1)}_I \) disappears through a tangent bifurcation when it is still positive, so that the jump in the infection level is discontinuous. At the bifurcation, we find \( z_S = 2z(2\tilde{\lambda} + 3)/9 > z \). The connectivity of I-agents decreases with \( \tilde{r} \) and, at the bifurcation, its value is independent of \( \tilde{\lambda} \): \( z_I = 2/3 \).

From the viewpoint of the interplay of the epidemiological dynamics and the structure of the underlying network, the most interesting result of this section is the fact that the infection dies out even when infected agents keep a substantial connectivity with the rest of the population. In the cases illustrated in Fig. 3 for instance, infected agents preserve more than 60% of their connections at the threshold where the infection level vanishes. In other words, reconnection needs not to completely isolate infected agents to suppress the infection. A moderate, partial isolation of the infected population is enough to asymptotically inhibit the endemic state.

### 3 Discussion and conclusion

What mechanisms are at work when the infection is suppressed by reconnection, even when the connectivity of infected agents remains fairly high? To advance an answer to this question, it helps to consider a simpler dynamical system for the fraction of I-agents:

\[
n'_I = -n_I + [2\tilde{\lambda} - \tilde{r}(1 - n_I)^2]n_I(1 - n_I). \tag{11}
\]

The right-hand side of this equation is just a rearrangement of that of Eq. \( \text{(5)} \). The equilibria of Eq. \( \text{(11)} \) are thus identical to the equilibria for \( n_I \) in Eqs. \( \text{(2)} \). Moreover, their stability properties are also the same as in our original system. It is important to understand, however, that \( \text{(11)} \) and \( \text{(2)} \) are not equivalent: they merely share the same equilibrium behaviour in which regards the fraction of I-agents.

We immediately see that Eq. \( \text{(11)} \) can be put in the form of the standard mean-field equation \( \text{(1)} \) for a SIS process if we introduce the effective infection
In Eq. (1) the threshold of infection suppression, where the trivial equilibrium changes its stability, is given by $\rho = \gamma$. Imposing this same condition to $\rho_{\text{eff}}$, we find $\tilde{r} = 2\tilde{\lambda} - 1$. But this is precisely the suppression threshold in the system with reconnections. Therefore, with respect to the stabilization of the trivial equilibrium, the system (2) is effectively equivalent to the standard SIS model with infectivity $\rho_{\text{eff}}$. The transcritical bifurcation of Eqs. (2), where $n_{I}^{(0)} = 0$ becomes stable, can be interpreted as a kind of continuation for $r \neq 0$ of the transcritical bifurcation of the SIS model without reconnection events.

The interpretation of the tangent bifurcation where the endemic state disappears at a positive value of $n_{I}^{(1)}$, for $\lambda > 3/4$, is less direct. It can however be argued that the presence of such a tangent bifurcation, together with the transcritical bifurcation which stabilizes the trivial equilibrium, constitute the generic critical behaviour expected for a SIS model like Eq. (11), with an infection probability which depends on the density of I-agents:

$$n_{I}' = -n_{I} + \rho(n_{I})n_{I}(1 - n_{I}).$$

Besides the trivial equilibrium, this equation has fixed points at the solutions of

$$\rho(n_{I}^{*}) = (1 - n_{I}^{*})^{-1}.$$

Figure 4 illustrates graphically two representative situations. The dotted curve is the graph of the right-hand side of Eq. (14) as a function of $n_{I}^{*}$. If the graph of $\rho(n_{I}^{*})$ has a single intersection with the dotted curve (A) and if, upon variation of parameters in the infection probability, the graph varies as indicated by the arrow, the intersection crosses $n_{I}^{*} = 0$ and a transcritical bifurcation takes place. The standard SIS model, for which $\rho$ is constant, is a special case within this situation. More generally, the graph of $\rho(n_{I}^{*})$ may have two (B) or more intersections with the dotted curve. When parameters change, it is still possible than one of the intersections becomes involved in a transcritical bifurcation crossing $n_{I}^{*} = 0$, exactly as in situation A. Now, however, it may well be the case that two intersections approach each other, and eventually collapse and disappear, as in B. In this case, Eq. (13) undergoes a tangent bifurcation, as found to happen in our system (2).

In summary, the above discussion shows that the suppression of the endemic state as a result of reconnection events can be reasonably understood in terms of the critical behaviour of a standard SIS model with an effective probability

$$\rho_{\text{eff}} = \gamma[2\tilde{\lambda} - \tilde{r}(1 - n_{I})^2] = 2(1 - r)\lambda - \frac{2r}{z}(1 - n_{I})^2.$$  (12)
Figure 4: Graphical solution of Eq. (14). The dotted curve represents the right-hand side of the equation, and full curves are possible graphs of the left-hand side. Dots stand at their intersections. The arrows illustrate how the graphs may change upon the variation of parameters, in the cases of a transcritical bifurcation (A) and of a tangent bifurcation (B).
infectivity, which depends on both the reconnection probability and on the fraction of infected agents. The transcritical bifurcation which stabilizes the state where the infection is completely inhibited is interpreted as a continuation of a similar transition in the absence of reconnection. In turn, the tangent bifurcation—which, for large infectivities, suppresses the infection as the reconnection probability grows—is a generic phenomenon in SIS models with density-dependent infectivity [10].

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We have studied a model for an epidemiological process in a population of agents on a network, where contagion can occur along the network links. The network coevolves with the population as the infection progresses: a susceptible agent can decide to break a link with an infected partner, and reconnect it with another agent. Our main result is that this reconnection mechanism, implemented with moderate frequency, can completely suppress the endemic state where an infected portion of the population persists at arbitrary long times. Suppression of the endemic state does not require full isolation of the infected population. On the contrary, it can be achieved while each infected agent preserves a substantial part of the links with the rest of the population.

Whether these results are relevant to real epidemiological processes is a question beyond the present study. Our analysis should be regarded as an illustration of the kind of collective effects that may emerge from the coevolution of populations of dynamical elements and their interaction network.

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