

Control of epidemics via social partnership adjustmentBin Wu,^{1,*} Shanjun Mao,² Jiazeng Wang,³ and Da Zhou^{4,†}¹*School of Science, Beijing University of Posts and Telecommunications, Beijing 100876, People's Republic of China*²*School of Mathematical Sciences, Xiamen University, Xiamen 361005, People's Republic of China*³*Department of Mathematics, Beijing Technology and Business University, Beijing 100048, People's Republic of China*⁴*School of Mathematical Sciences and Fujian Provincial Key Laboratory of Mathematical Modeling and High-Performance Scientific Computation, Xiamen University, Xiamen 361005, People's Republic of China*

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Epidemic control is of great importance for human society. Adjusting interacting partners is an effective individualized control strategy. Intuitively, it is done either by shortening the interaction time between susceptible and infected individuals or by increasing the opportunities for contact between susceptible individuals. Here, we provide a comparative study on these two control strategies by establishing an epidemic model with nonuniform stochastic interactions. It seems that the two strategies should be similar, since shortening the interaction time between susceptible and infected individuals somehow increases the chances for contact between susceptible individuals. However, analytical results indicate that the effectiveness of the former strategy sensitively depends on the infectious intensity and the combinations of different interaction rates, whereas the latter one is quite robust and efficient. Simulations are shown to verify our analytical predictions. Our work may shed light on the strategic choice of disease control.

DOI: [10.1103/PhysRevE.94.062314](https://doi.org/10.1103/PhysRevE.94.062314)**I. INTRODUCTION**

Modeling the spreading of infectious diseases has a long history [1–7]. Mathematical models not only deepen the understanding of epidemic dynamics, but also shed light on the control of diseases. In recent years, much attention has been paid to epidemic control via social relationship adjustment [8–10]. As a pioneering work, Gross *et al.* proposed a susceptible-infected-susceptible (SIS) model on an adaptive network. Therein the susceptible individual breaks the link with the infected partner and rewires to another randomly selected susceptible individual [11]. This rewiring rule brings in highly complex dynamics (such as bistability and oscillation) to the classical SIS model. The rewiring dynamics then opens up the avenue on how individualized partnership adjustment alters the epidemic dynamics. On the one hand, besides the SIS model, typical epidemic models have almost been investigated including the susceptible-infected-recovered-susceptible (SIRS) model [12], the susceptible-infected-recovered (SIR) model [13], and the susceptible-infected-vaccinated (SIV) model [14]. On the other hand, more realistic and complex link-rewiring rules are proposed. In particular, generalizations of Gross *et al.*'s rewiring rule are mainly twofold. For one thing, after the disconnection of susceptible-infected (SI) link, the susceptible is assumed to reconnect to a randomly selected member of the population whether it is susceptible or not [15]. For another, the infected is also allowed to switch its partnership from the susceptible to a new randomly selected contact [16]. Besides the rewiring rule which is dependent on the infection process, the rewiring rule that is independent of the infection process was also investigated [17,18]. In spite of different model assumptions, all these models showed that the infection propagation can be

greatly influenced by the dynamical networks. In particular, the infection can be effectively suppressed by reducing the interaction opportunities between susceptible and infected individuals.

Besides the above-mentioned link-rewiring models, another type of adaptive network is the link-activation-deactivation model [19–21]. It assumes that a link can either be broken or recreated on the basis of the infectious states of the two end points of the link. In this model, only local information is required, which could be more realistic [10]. In particular, Guo *et al.* proposed an ASIS model, in which any SI link can be broken (deactivated). After the disconnection of an SI link, the two disconnected nodes can be reconnected again once both of them become susceptible (activated) [21]. Despite seeming differences, the link-activation-deactivation dynamics is similar to the rewiring dynamics. On the one hand, Guo *et al.* showed that the ASIS model (initiated on complete graphs) approximates the link-rewiring model [11]. On the other hand, the quasistationary (metastable) fraction of infected individuals can be reduced by increasing the effective breaking rate (proportional to the ratio of deactivating rate to activating rate). This echoes the results based on the link-rewiring models that the disease can be controlled by reducing the contacts between susceptible and infected individuals. Therefore, both types of linking dynamics in epidemic control can be seen as decreasing the interaction rate between susceptible and infected individuals (called *SI control*). Furthermore, considering that the effective breaking rate in [21] also depends on the activating rate between susceptibles, their work reminds us of the significance of susceptible-susceptible (SS) links in epidemic control.

Intuitively, increasing the interaction time between susceptibles can also be a control strategy (called *SS control*). Yet it is seldom addressed, compared to the SI control that has been intensively studied in previous literatures. It seems that these two control strategies are two sides of the same coin. Actually, this is true in Gross *et al.*'s model [11], since,

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based on their rewiring rule, the decrease of SI links directly leads to the increase of SS links. However, this is no longer valid in Risau-Gusmán and Zanette's model [16], since the disconnection of an SI link does not necessarily result in the reconnection of an SS link. Therefore, the SS and SI control strategies are not equivalent in general.

In this work, we provide a comparative study on the SI control and the SS control by proposing a link-rewiring SIS model. Unlike the models only allowing the breaking of SI links, we allow all three types of links [SS, SI, and infected-infected (II)] to be broken, equipped with three independent parameters to characterize the breaking rates of SS, SI, and II links. Actually, this assumption mimics the intrinsic nature of human mobility [22,23], namely, people moving or changing their social relationships due to a variety of reasons, even without the consideration of avoiding infectious diseases. In this way, SI links should not be the only type that is allowed to be broken; both SS and II links can change. For example, in acquired immune deficiency syndrome (AIDS) not only are the susceptible individuals willing to avoid contact from the infected individuals, but the susceptible-susceptible and infected-infected relationships may also be broken up due to unsatisfactory sexual experiences; i.e., the rewiring processes can happen in SS and II links. Besides, we allow all the individuals to be capable of adjusting any of their partners. This mirrors the freedom of social life. It also excludes the central control of epidemics, for example, via organizations. In this way, we could concentrate on how the social partnership adjustment strategies alone alter the fate of epidemics.

We demonstrate analytically that our model captures the epidemic dynamics with nonuniform interaction rates under fast linking dynamics. It is shown that sometimes the SS control is more effective and robust than the SI control. In particular, strengthening the closeness between susceptibles (SS control) effectively eradicates the disease no matter how infectious the disease is. However, the effectiveness of the SI control sensitively depends on the infectious intensity and the intrinsic mobility rate of the population. In other words, there are cases such that the SI control cannot eliminate the disease as efficiently as the SS control. Simulation results are also shown for validating our theoretical predictions. Our findings suggest that, besides the SI control, it is still of concern that the SS control may serve as a better candidate for epidemic control.

II. MODEL AND ANALYSIS

In this section, we propose the model of epidemic spreading coupled with a simple stochastic link-rewiring dynamics. Then we theoretically analyze the epidemic model with nonuniform interaction rates based on the time scale separation.

A. Epidemic dynamics

We consider a structured population of N individuals. The population is located on a connected network. We assume that the average degree k is much smaller than the population size N , i.e., $k \ll N$. Here nodes refer to individuals and links represent social ties between individuals.

We adopt a standard SIS model to study the epidemic spreading. The SIS model assumes that susceptible individuals get infected with a probability proportional to the number of their infected neighbors; infected individuals recover and become susceptible with no immunity to the disease after a period of recovery time. The SIS model has three features: (i) the whole population size N is constant over time, (ii) the transmission of disease only happens via the SI links, and (iii) the recovery of infected individuals is independent of the status of their neighbors.

Let I_t be the number of infected individuals at time t ; therefore, the mean-field equation of the SIS model on the structured population is given as follows:

$$\frac{dI_t}{dt} = \lambda N_{SI} - \mu I_t. \quad (1)$$

Here λ is the transmission rate and μ is the recovery rate. All through the paper we assume that $\mu = 1$ without loss of generality, and N_{SI} is the number of SI links.

B. Link-rewiring dynamics

The social relationships between individuals are not eternal but are continuously coevolving. As a typical example, susceptible individuals tend to avoid contacts with infected ones by adjusting their local connections. It has served as the most recognized prototype in the study of epidemic control on dynamical networks. However, individuals may receive miscellaneous information when making rewiring decisions; thus, it is possible for all the individuals to adjust all of their current social relationships. Such a rewiring process captures mobilitylike human behavior.

Here we propose a simple link-rewiring dynamics by extending the dynamical nature from SI links to all types of links in the network. Each individual is either susceptible (S) or infected (I). Thus, there are three types of links: susceptible-susceptible (SS), susceptible-infected (SI), and infected-infected (II) links. To characterize the fragilities of different types of links, we define w_{XY} ($XY \in \{SS, SI, II\}$) as the probability with which an XY link breaks off in the process of disconnection. In each rewiring step, a link XY is selected randomly from the network. With probability w_{XY} , the link is broken; otherwise, the link remains connected. If it is broken, X or Y is picked as the active individual, who is entitled to reform a new link. Its new neighbor is randomly selected from the individuals who are not in its current neighborhood. Self-connections and double connections are thus not allowed here.

In this way, the link-rewiring dynamics can be modeled as a Markov chain in the state space of $\{SS, SI, II\}$ [24–26]. Considering the transition probabilities between states, let us take the transition from SI to SS as an example. This happens only when SI is broken off and S is selected to reform a new link to another susceptible individual. Note that the total population size N is much larger than the average degree k , and the transition probability $Q_{SI \rightarrow SS}$ is approximately given by $w_{SI} \times \frac{1}{2} \times s$, where s is the density of susceptibles at the moment. Similarly, we calculate all the other transition probabilities,

yielding the transition probability matrix

$$Q = \begin{matrix} & \text{SS} & \text{SI} & \text{II} \\ \begin{matrix} \text{SS} \\ \text{SI} \\ \text{II} \end{matrix} & \begin{pmatrix} sw_{\text{SS}} + (1 - w_{\text{SS}}) & iw_{\text{SS}} & 0 \\ \frac{sw_{\text{SI}}}{2} & 1 - \frac{w_{\text{SI}}}{2} & \frac{iw_{\text{SI}}}{2} \\ 0 & sw_{\text{II}} & iw_{\text{II}} + (1 - w_{\text{II}}) \end{pmatrix} \end{matrix}, \quad (2)$$

where i is the density of infected individuals. According to the standard Markov chain theory [27], there exists a unique limiting distribution Π satisfying $\Pi Q = \Pi$ provided Q is irreducible and aperiodic. Namely, when $w_{\text{SS}}w_{\text{SI}}w_{\text{II}}is \neq 0$, Q has a unique stationary distribution

$$\Pi = (\pi_{\text{SS}}, \pi_{\text{SI}}, \pi_{\text{II}}) = \frac{1}{A(i)} \left(\frac{s^2}{w_{\text{SS}}}, \frac{2si}{w_{\text{SI}}}, \frac{i^2}{w_{\text{II}}} \right), \quad (3)$$

where $A(i) = \frac{s^2}{w_{\text{SS}}} + \frac{2si}{w_{\text{SI}}} + \frac{i^2}{w_{\text{II}}}$ is the normalization.

C. Time scale separation

It is challenging to capture N_{SI} due to the complexity of real social networks [28–30]. This is already true in static networks, and it becomes even more difficult taking into account the dynamical nature of social networks [8]. Here we overcome this problem by assuming the adiabatic elimination of fast linking dynamics [27] (also called annealed adaptive dynamics [10]); i.e., the adjustment of social ties is much more frequent than the update of infection states. This assumption implies *time scale separation* of the two coupled dynamics. In other words, the disease is unlikely to spread until the social configuration tends to the stationary regime. In this way, N_{SI} is approximated as

$$N_{\text{SI}} \approx \frac{Nk}{2} \pi_{\text{SI}}, \quad (4)$$

where $Nk/2$ is the total number of the links in the network and π_{SI} is the fraction of SI links in the stationary regime. This approximation greatly reduces the complexity of the coupled dynamics. In light of this, the idea of time scale separation has frequently been used in analyzing complex dynamics on adaptive networks (epidemics [18,19,31] and evolutionary games [24,32]).

By substituting Eq. (4) into Eq. (1) we have

$$\frac{dI}{dt} = \frac{\lambda Nk}{2} \pi_{\text{SI}} - \mu I. \quad (5)$$

Noting that $i = I/N$, $s = S/N$, and $\pi_{\text{SI}} = \frac{2is}{A(i)w_{\text{SI}}}$, Eq. (5) can be transformed to

$$\frac{di}{dt} = \frac{k\lambda}{A(i)w_{\text{SI}}} is - \mu i. \quad (6)$$

In particular, when all the interaction rates are uniform and positive ($w_{\text{SS}} = w_{\text{SI}} = w_{\text{II}} > 0$), Eq. (6) reduces to

$$\frac{di}{dt} = i \left(\underbrace{k\lambda}_{\lambda_e} s - \mu \right). \quad (7)$$

Equation (7) is nothing but the classical SIS model [2], provided that $\lambda_e = k\lambda$ is redefined as the effective transmission rate. This implies that the population is as a well-mixed

population, if individuals break their partnerships with no social bias. It should be pointed out that, when $w_{\text{SS}} = w_{\text{SI}} = w_{\text{II}} = 0$, the transition probability matrix Eq. (2) violates the irreducible condition [33] that our analysis relies on. In fact, this case resembles the static network, which has been excluded from our analysis.

When the interactions are violated from above social unbias, on the one hand, it results in nonuniform interactions in the population [34]. Therefore, Eq. (6) extends the classical SIS model from uniform interaction rates to nonuniform interaction rates. Noteworthy is that this nonuniform extension is an emergent property from microscopic stochastic linking dynamics, which is not assumed in prior. On the other hand, if we define $\Lambda(i) = \frac{k\lambda i}{A(i)w_{\text{SI}}}$, our model also extends the classical SIS model from density independent transmission rate to density dependent transmission rate [15,35]. In other words, the dynamical nature of social networks essentially acts as a feedback mechanism on the SIS model. The feedback mechanism, which is taken as the central idea of control, can significantly alter the epidemic dynamics.

Noteworthy is that all the analyses above are based on the time scale separation. Thus it suggests that the link-rewiring event should happen with a sufficiently large probability (close to 1) in each update. Furthermore, we give a more precise lower bound for this probability based on pair approximations: It is found that the time scale separation is at work provided the likelihood of the linking dynamics is greater than (see Appendix A)

$$\frac{k^2}{k^2 + N}. \quad (8)$$

For more general cases where the time scale separation is absent, the approximation based on the moment closure could be applied to provide theoretical insights [36] (see Appendix A).

III. THEORETICAL RESULTS

Our main concern in this comparative study is epidemic control via changing the interaction rates in different ways. Based on Eq. (1), it is N_{SI} that determines the spread of infection. The more SI links there are, the more likely the spread of infection. Generally, there are two ways to control N_{SI} . For one thing, it is natural to increase w_{SI} for reducing the interaction rate ($1/w_{\text{SI}}$) between susceptible and infected individuals (SI control). For another thing, decreasing w_{SS} can also reduce the exposure of susceptibles to infection (SS control). Therefore, we investigate the control of epidemics via these two strategies. More specifically, by taking the uniform interaction rates ($w_{\text{SS}} = w_{\text{SI}} = w_{\text{II}} > 0$) as the reference case, we would like to provide a comparative study on both the SI control ($w_{\text{SI}} > w_{\text{SS}} = w_{\text{II}}$) and the SS control ($w_{\text{SS}} < w_{\text{SI}} = w_{\text{II}}$). In the following, we assume that the effective transmission rate is always larger than the recovery rate, i.e., $\lambda_e > 1$, where the epidemic control is necessary.

A. SI control: Decreasing the interaction rate between susceptible and infected individuals

To decrease the interaction rate between susceptible and infected individuals, it is equivalent to increase the breaking

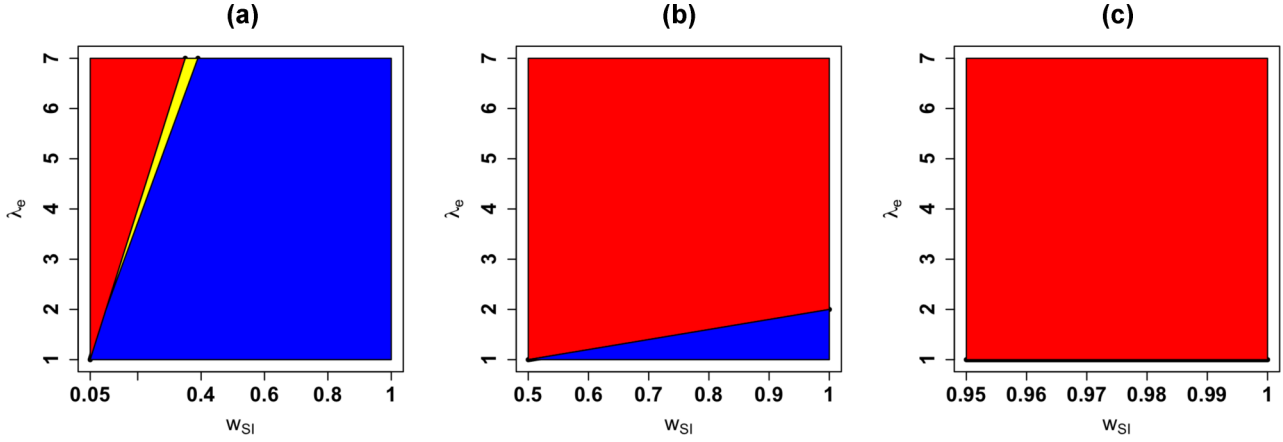


FIG. 1. SI control of epidemics, i.e., increasing the breaking probability between susceptible and infected individuals. For the uniform interaction case ($w_{SS} = w_{SI} = w_{II}$), the model degenerates to the conventional SIS model. There is only one internal equilibrium and it is stable provided $\lambda_e > 1$. Here we solely adjust w_{SI} such that the duration time of SI links is shorter than the other two types of links, i.e., $w_{SI} > w_{II} = w_{SS}$. The three panels show the phase diagrams in the (w_{SI}, λ_e) -plane. The quality of the SI control is sensitively dependent on the reference uniform breaking probabilities. (a) When they are small ($w_{SS} = w_{II} = 0.05$), decreasing the interaction between susceptible and infected individuals makes the phase diagram change from endemic state (red) to bistable state (yellow) and then to final extinct state (blue). (b) When $w_{SS} = w_{II} = 0.5$, there is no bistable state (yellow) any more. It becomes harder to eradicate disease when the population is even more mobile. (c) The right panel shows that the SI control is incapable of eradicating the disease provided the population is intrinsically highly mobile ($w_{SS} = w_{II} = 0.95$).

probability w_{SI} . Based on the uniform interaction as the reference case, we are interested in how the epidemic dynamics is changed by increasing w_{SI} . Here the uniform interaction can mimic the basic migration rate in the population. To illustrate our main results, we consider three typical cases with different initial values of the uniform interaction rates (see Appendix B for technical details):

1. Small initial case [Fig. 1(a)]

In this case, we set initially the breaking probabilities for all types of links to be 0.05. The disease can be controlled by increasing w_{SI} from 0.05 to 1. In particular, for small infectious rate (i.e., $\lambda_e \leq 2$), there is a phase transition with the increase of w_{SI} . That is, the final state of epidemics turns from endemic to extinction. For large λ_e (i.e., $\lambda_e > 2$), there is a small region of bistability where the disease persists or dies out due to the initial infected fraction. Compared to the single continuous phase transition in the conventional (uniform) SIS model, the nonuniform SIS model can give rise to multiple phase transitions. The emergent bistability in adaptive SIS model has already been reported in previous studies [11, 15, 37, 38], but it is quite difficult to approximate the conditions under which bistability is present. For our model, we explicitly provide those analytical conditions under which the bistability emerges based on Eq. (6). In the case of SI control ($w_{SI} > w_{SS} = w_{II}$), it arises if and only if

$$(\lambda_e, w) \in (2, +\infty) \times \left(\lambda_e, \frac{\lambda_e + \sqrt{(\lambda_e^2 + (\lambda_e - 2)^2)}}{2} \right) \quad (9)$$

(see Appendix B), where $w = w_{SI}/w_{SS}$.

2. Intermediate initial case [Fig. 1(b)]

In this case, increasing w_{SI} is not as effective as that in the above small initial case. For small λ_e , even though there still

exists a phase transition from endemic state to extinct state, the marginal value of w_{SI} that needs to cross the transition line is large. More importantly, when λ_e is large enough, increasing w_{SI} is unable to eradicate the disease any more. The disease will persist no matter how large the interaction rates between susceptible and infected individuals are. Moreover, it is shown in Fig. 2 that the endemic level is not sensitive to w_{SI} . In other words, by increasing w_{SI} , the final fraction of infected individuals declines very slowly. That is to say, the endemic of w_{SI} can neither qualitatively change the final state of the endemic nor quantitatively inhibit the final fraction of infected individuals.

3. Large initial case [Fig. 1(c)]

In this case, the endemic state is always the global stable state provided $\lambda_e > 1$. That is, the epidemics cannot be eradicated by the SI control.

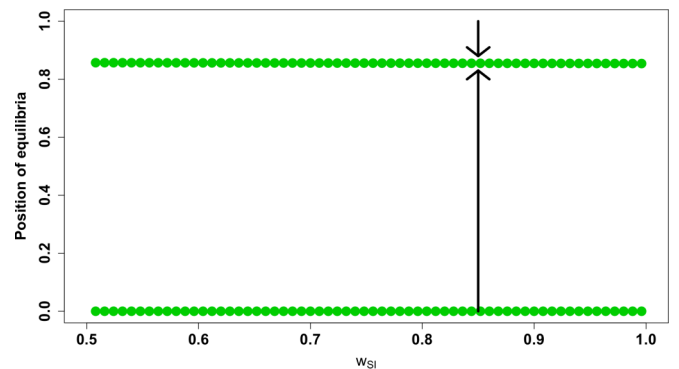


FIG. 2. Equilibrium position of infected fraction as a function of w_{SI} . Here $w_{SS} = w_{II} = 0.5$, and $\lambda_e = 7$. The disease cannot be eradicated by the SI control, and the level of infection in the equilibrium state declines very slowly (from 0.857 to 0.854) by increasing w_{SI} (from 0.5 to 1).

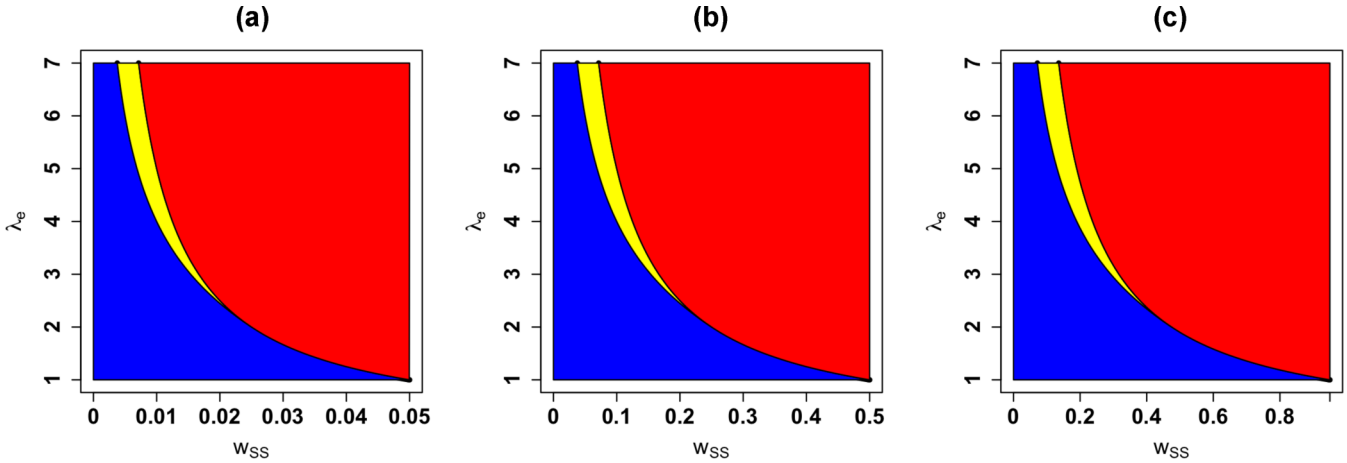


FIG. 3. SS control, i.e., decreasing the breaking probability between susceptibles. We start from the uniform case $w_{SS} = w_{SI} = w_{II}$. Here the disease is solely controlled by increasing the duration time of the social ties between susceptibles, i.e., $w_{SS} < w_{II} = w_{SI}$. These phase diagrams are similar for all the reference uniform interaction rates: *i*) for small λ_e , the SS control makes the disease change from endemic state (red) directly to extinction state (blue); *ii*) for large λ_e , the SS control can still eradicate the disease, but the phase diagram has to pass from endemic (red) to bistable state (yellow) and finally to extinction (blue).

To summarize, the control efficiency via reducing the interaction rate between susceptible and infected individuals strongly depends on the reference breaking probabilities, i.e., the intrinsic population mobility. The more likely the population is mobile, the worse the SI control performs.

B. SS control: Increasing the interaction rate between susceptibles

Unlike the SI control, increasing the interaction rate between susceptibles is shown as an effective and robust strategy for epidemic control. In fact, no matter what the intrinsic mobility rate of the population is, the SS control successfully eradicates the disease. To this end, we study the three typical reference population mobility cases in the above section (see Appendix B for technical details). Figure 3 shows that the phase diagrams for the three cases are quite similar to each other:

- (i) For small λ_e ($1 < \lambda_e \leq 2$), by decreasing w_{SS} , the final state of disease is directly transformed from endemic to extinction.
- (ii) For large λ_e ($\lambda_e > 2$), the bistability arises in all three cases. That is, no matter how large the initial uniform interaction rates are, with the decrease of w_{SS} , there is an intermediate region where the disease persists or dies out depending on the initial fraction of disease. Furthermore, we analytically obtain that the bistable region is given by

$$\left(\lambda_e, \frac{1}{w}\right) \in (2, +\infty) \times \left(\frac{4}{4 + \lambda_e^2}, \frac{1}{\lambda_e}\right). \tag{10}$$

By comparison, the SS control is more effective than the SI control in two ways. On the one hand, the control of w_{SS} is independent of the intrinsic population mobility, i.e., robust control. On the other hand, decreasing w_{SS} can always effectively eradicate the disease regardless of infectious intensity (Fig. 4 illustrates the position of equilibria as a function of w_{SS} in the bistable case).

IV. AGENT-BASED SIMULATIONS

In this section, we present agent-based simulations and further discuss the efficiency of the time scale separation method based on the comparison between the simulation results and theoretical predictions.

A. Simulation procedures

The *contact process* [39] is adopted to model the epidemic spreading on networks. Let $\alpha \in (0,1)$ be the probability of epidemic spreading in each update. The simulation is performed as follows:

- (1) Initially, there are N individuals located on a regular graph with degree k , where each individual has exactly k neighbors. Then N_0 infected individuals and $N - N_0$ susceptible individuals are randomly distributed.

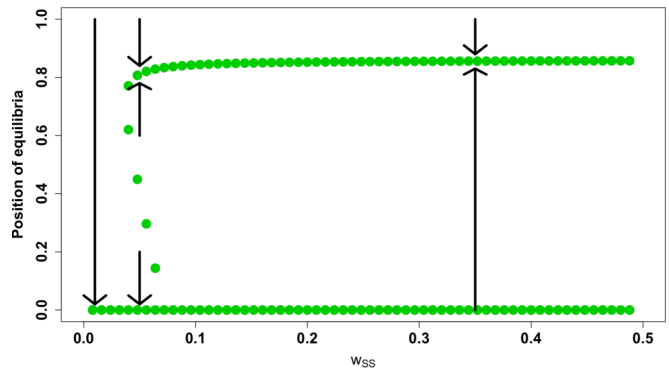


FIG. 4. Equilibrium position of infected fraction as a function of w_{SS} . Here $w_{SI} = w_{II} = 0.5$, and $\lambda_e = 7$. Increasing the interaction time between susceptibles (i.e. decreasing w_{SS}) effectively eradicates the disease. In particular, for $0.033 < w_{SS} < 0.08$ (bistability), the disease dies out provided the initial infection is few in number. Even when the initial number of infection is large, the final level of infection is still lower than the case with $0.08 < w_{SS} < 0.5$. For $w_{SS} < 0.033$, the disease is eradicated no matter what the initial state is.

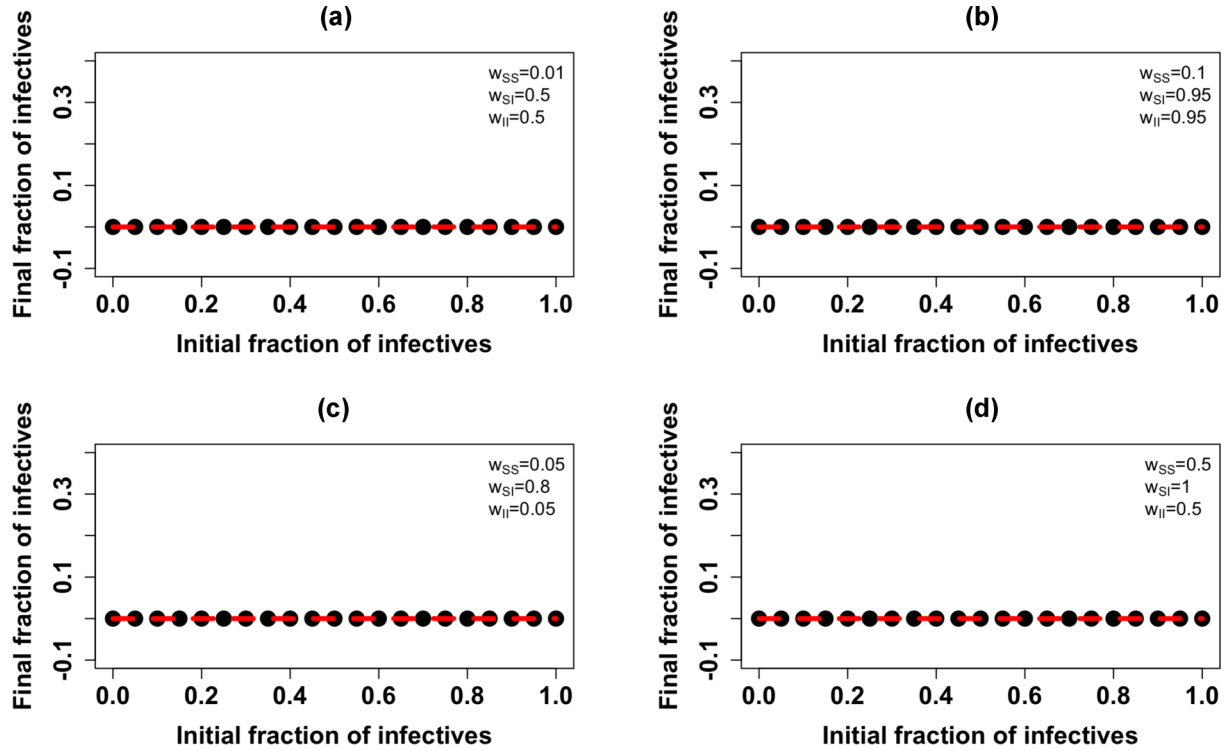


FIG. 5. Extinction. The discrete points are obtained from simulations, and the red lines represent the analytical results based on time scale separation. All of the four panels show that the disease dies out no matter how many infected individuals are present in the beginning. The parameters in all the four panels are from the blue regions in Figs. 1 and 3. Thus they are consistent with the analytical predictions. (Common parameters: $\lambda_e = 1.5$, $N = 100$, $\alpha = 0.01$.)

(2) We generate a random number $r \in (0, 1)$. If $r < \alpha$, we perform the contact process. Otherwise ($r \geq \alpha$), we perform the linking dynamics.

(3) If the contact process occurs, an infected individual (called Bob) is selected randomly. With probability $\frac{\mu}{k_{\text{Bob}}\lambda + \mu}$ Bob becomes susceptible, where k_{Bob} is the degree of Bob. Otherwise a neighbor of Bob is selected at random. This neighbor, namely Jack, is infected with probability $\frac{\lambda}{k_{\text{Bob}}\lambda + \mu}$. It is noteworthy that Jack becomes infected if his status is susceptible. However, this new infection event does not change the state of Jack if Jack has been infected already. Then we return to step 2.

(4) If the linking dynamics occurs, a link is selected randomly. The type of this link is denoted as XY ($XY \in \{\text{SS}, \text{SI}, \text{II}\}$). With probability w_{XY} , the link is broken; otherwise the link remains connected. If it is broken, X or Y is picked as the active individual, who is entitled to reform a new link. The new neighbor is randomly selected from the individuals who are not in its current neighborhood. Then we return to step 2.

Each data point is averaged over 50 independent samples. In each sample, we run a transient time of 10^6 generations, and we set the mean value over time window of the last 10^3 generations to be the final fraction of infected individuals.

It should be pointed out that the simulation results are robust for all initial connected graphs, provided the number of infected individuals, N_0 , population size N , and the average degree k are fixed. The regular graph here only serves as a prototype for simulations. In fact, our linking dynamics

is a Markov chain, which is irreducible and aperiodic. This yields that the limiting behavior is independent of the initial configuration of the network [33]. Furthermore, the assumption of time scale separation allows all the links to converge to the stationary distribution. Therefore, all the links would converge to the stationary distribution no matter what type of graph it is initially.

B. Simulation results

With the coupled linking dynamics, the final fate of the infection can be of three cases: die out no matter what the initial fraction of the infected individuals is (called *extinction*), stabilize at a nonzero fraction of infected individuals no matter what the (positive) initial fraction of infected individuals is (called *endemicity*), or stabilize at a nonzero fraction of infected individuals if the initial fraction of infected individuals exceeds a critical value and die out otherwise (called *bistability*).

For the extinction cases, simulation results are found to be in good agreement with the analytical predictions. This is true for all the parameter regions predicting extinction for both SI and SS controls (see Fig. 5).

For the endemic cases, Fig. 6 shows that the population would end up with a constant fraction of infected individuals, provided there are infected individuals initially. This is exactly in line with the analytical predictions. Furthermore, the inconsistency between the analytical and simulation results is less than 10%, which is acceptable. Considering this

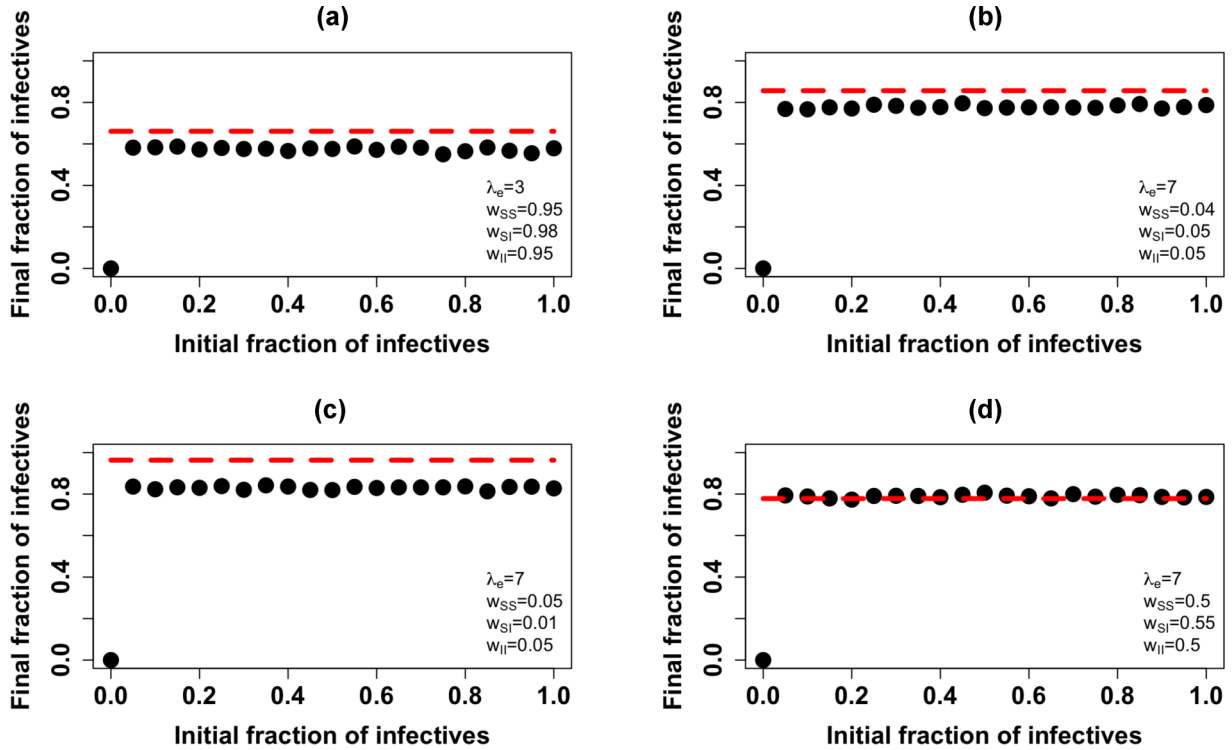


FIG. 6. Endemic. The simulation results (discrete points) show that no matter what the initial fraction of infective individuals is (except for the zero case which is an absorbing state of the agent-based model), the population would end up with a constant positive fraction of infected individuals. However, the analytical predictions would somewhat overestimate the simulation results. This is mainly because the running time for the simulation is beyond the quasistationary time scale of our theoretical analysis. Parameters are from the red regions in Figs. 1 and 3. (Common parameters: $N = 100$, $\alpha = 0.01$.)

10% disagreement, the analytical predictions systematically overestimate the simulation results. In fact, the agent-based contact process is a Markov process with an absorbing state, where no infected individual is present. In other words, the disease would go extinct eventually if the system evolves over a sufficiently long time. Our analytical results, however, are in the quasistationary time scale [40,41]. The inconsistency between the analytical and simulation results suggests that the running time of 10^6 is beyond the quasistationary time scale. Thus the system may evolve to the absorbing state with non-negligible chances.

For the bistability cases, the simulation results show qualitative agreement with the analytical predictions. In particular, the critical initial fraction of infected individuals, ensuring a dramatic outbreak of epidemics, is consistent with the unstable fixed point predicted by the analytical result (see the blue dashed lines in Fig. 7). Disagreements, however, are also present. For example, the theoretical results tend to underestimate the final infection when the infection fraction is rare initially. In fact, this bistable case bears two internal equilibria lying at x_1^* (unstable) and x_2^* (stable) ($x_1^* < x_2^*$). For a small initial fraction of infected individuals, the deterministic part of the system drives the infection to extinction based on the analytical investigation. Yet by its intrinsic stochastic nature of the epidemic spreading, the infection would increase in number and be possibly trapped around the stable equilibria from time to time. Even though it is a type of *rare event*, it takes quite long to escape from this trap. Thus on average it

results in a relatively higher level of final fraction of infected individuals given the running time of simulations (here 10^6 generations). In other words, it is the interplay between the stochastic effect and stable equilibrium at zero that results in such inconsistency. It is noteworthy, despite this quantitative inconsistency, that the salient feature of the bistable dynamics is still captured by the analytical predictions.

In Fig. 8, we investigate how the population size affects the accuracy of the analytical approximation. Theoretically, large population size inhibits the stochasticity arising from the finite population effect, which is closer to the mean-field approximation. Similar discussions can be found in [24]. Figure 8 shows the case with $N = 100$ still captures the bistable dynamics as the case with $N = 500$ does.

V. DISCUSSIONS AND CONCLUSIONS

We have proposed a simple link-rewiring rule to model social partnership adjustment. Therein all the links are about to break, capturing the mobile nature of the population. This simple model paves the way to compare different rewiring-based epidemic control strategies.

We find that, for mild infectious disease, both SI and SS control strategies can eradicate the disease. For strong infectious disease, however, it is more efficient to adopt the SS control than the SI control. This result is counterintuitive. Intuitively, reducing the contacts between susceptible and infected individuals is believed to suppress the disease propagation.

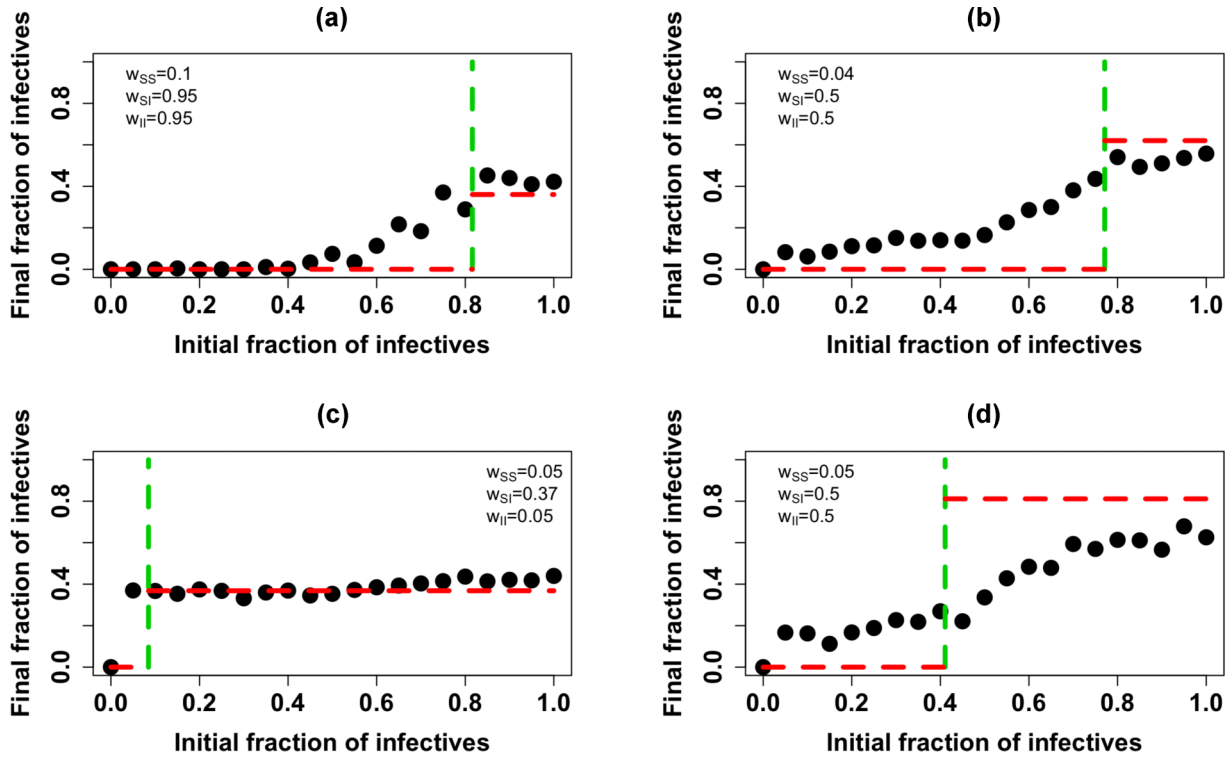


FIG. 7. Bistability. Discrete points represent simulation results. Dashed lines are via the analytical approximations: Red horizontal lines represent the stable fixed points, whereas green vertical lines represent the unstable fixed point (critical value). By comparison, the simulation results show qualitative agreement with the analytical predictions. In particular, the critical initial fraction of infected individuals, ensuring a dramatic outbreak of epidemics, is consistent with the unstable fixed point predicted by the analytical result. However, disagreements are present, where analytical results underestimate the final fraction of infective individuals when the infective individuals are rare initially. It is noteworthy that, despite this quantitative inconsistency, the salient feature of the bistable dynamics is still captured by the analytical predictions. Parameters are from the yellow regions in Figs. 1 and 3. (Common parameters: $\lambda_e = 7$, $N = 100$, $\alpha = 0.01$.)

Moreover, it seems that decreasing the interaction rate of SI links could naturally result in the increase of SS links [37]. How can these two strategies perform so differently? One of the salient features of our model is the variability of II links, which has seldom been addressed previously. Actually, increasing w_{SI} is equivalent to decreasing both w_{SS} and w_{II} . In other words, the SI control is equivalent to simultaneously strengthening SS links and II links. Similarly, the SS control is equivalent to simultaneously reducing the closeness of

SI links and II links. Thus, the relation of the SI and SS control strategies is not as straightforward as expected. To illustrate the impact of II links on the epidemic dynamics, we consider two examples: (1) $w_{SI} = 0.98$, $w_{SS} = 0.2$, and $w_{II} = 0.2$, and (2) $w_{SI} = 0.98$, $w_{SS} = 0.2$, and $w_{II} = 0.98$. The only difference between these two examples is the value of w_{II} . It is easy to show that in example 1 the disease becomes extinct, whereas bistability arises in example 2 [based on Eq. (10)].

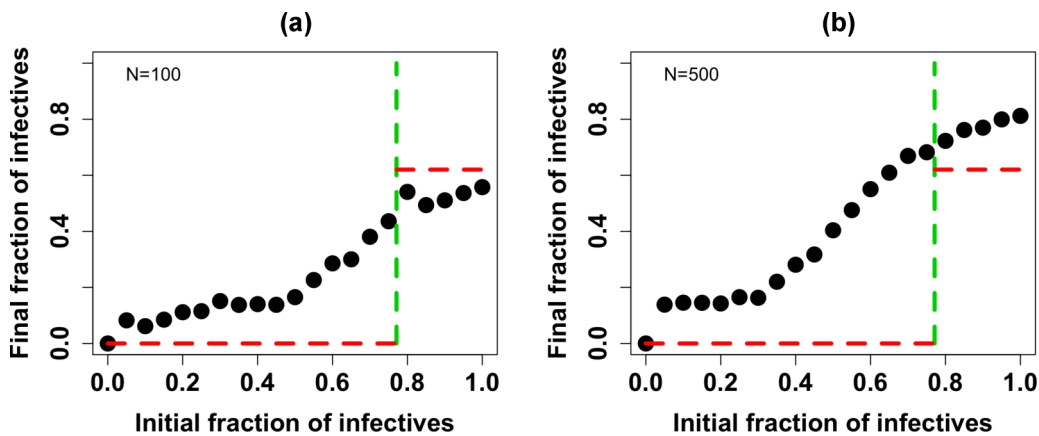


FIG. 8. The effect of the population size on the accuracy of the analytical approximation. Population size with $N = 100$ is enough to capture the salient feature of the bistability, compared with $N = 500$. (Common parameters: $\lambda_e = 7$, $w_{SS} = 0.04$, $w_{SI} = w_{II} = 0.5$ and $\alpha = 0.01$.)

Another feature of our reconnection rule is *nonselectivity*. In other words, individuals are allowed to rewire to a randomly selected member no matter if it is susceptible or not. Compared to the *selective* rule in [11], individuals in our model do not necessarily know who gets infected currently, which is more realistic. Actually, the nonselective rule increases the exposure of the susceptible individuals to the infected individuals. This is very likely in the beginning of an epidemic season, where the information on infection status is unaccessible. In particular, even though the SI control increases the breaking possibility of each SI link, a new SI link may be generated again due to the nonselective rule. By contrast, the SS control makes a straightforward intervention during the process of disconnection. That is, by strengthening the closeness between susceptible individuals, the SS strategy reduces the possibility of SI connection effectively. In this way, the nonselective rule has a relatively small impact on the SS control. Therefore, in the framework of the nonselective rewiring rule, the SS strategy is more efficient than the SI strategy.

Concentrating on the relation between the lifespan of each type of links and epidemic spreading, our model does not account for other features that are also considerable in capturing the epidemic dynamics of real world networks. For example, (1) our linking dynamics does not take into account the social interactions with memory, such as friendship and working partners, in which individuals preserve the contacts that they used to make [20,21], and (2) the link-rewiring process is a strong simplification of real adaptive networked human behavior. It is not necessarily realistic for individuals who break up a relationship to have a new partner immediately. However, it probably mimics the dynamics of networks in AIDS to some extent: The susceptible individuals break up their (mostly sexual) relationships with their infected partners and switch to other perceived healthy individuals. Moreover, the infected individuals may also rewire their links to other infected individuals.

To sum up, our result captures the causation between the link fragility and the disease control. Furthermore, this model might serve as a starting point to compare different rewiring control strategies for more general models closer to reality.

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APPENDIX A: ANOTHER ANALYTICAL APPROXIMATION

Our model couples the linking dynamics and the epidemic dynamics. While the method in the main text is analytically

insightful, it requires the time scales of the two dynamics to be separated. In other words, individuals should adjust their partners much faster than the spread of epidemics to make this method applicable. This is, however, not the case in general. We propose another analytical method to overcome this restriction. The method is based on pair approximation and rate equations [36]. Here we concentrate on how the method helps us estimate the condition under which the time scale separation is valid.

Let $\langle I \rangle$ and $\langle S \rangle$ be the global frequencies of infected and susceptible individuals, i.e., i and s in Eq. (6); and let $\langle XY \rangle = N_{XY}/N$ be the frequencies of XY pairs, where $XY \in \{II, SI, SS\}$. Thus $\langle I \rangle + \langle S \rangle = 1$ and $\langle II \rangle + \langle SI \rangle + \langle SS \rangle = 1$ hold. The system thus is determined by three independent variables: $\langle I \rangle$, $\langle SI \rangle$, and $\langle II \rangle$. The crucial assumption for pair approximation is that a higher order of moments can be captured by moments of pairs. In the following, we write down the rate equations of the three variables under the assumption of pair approximation.

For the evolution of a fraction of the infected, it is only determined by the epidemic dynamics. In this case, the number of infected individuals increases or decreases by one, or stays the same in one time step. By the Kolmogorov forward equation, we have that

$$\Delta \langle I \rangle = \text{Prob}\left(\Delta \langle I \rangle = \frac{1}{N}\right) \frac{1}{N} - \text{Prob}\left(\Delta \langle I \rangle = -\frac{1}{N}\right) \frac{1}{N}. \quad (\text{A1})$$

In particular, the probability that infected individuals increase by one in number happens when (1) the epidemic spreading is ongoing (with probability α), or (2) a susceptible individual is selected (with probability $\langle S \rangle$), and it is infected by one of its infected neighbors. The fraction of the infected individuals around a susceptible individual is $\frac{\langle SI \rangle}{\langle S \rangle}$ based on pair approximation. Thus there are on average $\frac{k \langle SI \rangle}{\langle S \rangle}$ infected neighbors around the selected susceptible individual, where k is the average degree of the entire network. Therefore, the infection probability of the susceptible individual within a small time interval Δt is $\lambda \frac{k \langle SI \rangle}{\langle S \rangle} \Delta t$. Thus $\text{Prob}(\Delta \langle I \rangle = \frac{1}{N}) = \alpha \lambda k \langle SI \rangle \Delta t$. Similarly, we have $\text{Prob}(\Delta \langle I \rangle = -\frac{1}{N}) = \alpha \mu \langle I \rangle \Delta t$.

Let us rescale the time interval $\Delta t = 1/N$. For large population size N , dividing Δt on both sides of Eq. (A1) yields

$$\langle \dot{I} \rangle = \frac{\alpha}{N} (\lambda k \langle SI \rangle - \mu \langle I \rangle). \quad (\text{A2})$$

This equation is identical to the mean-field SIS model, Eq. (1), up to a rescaling factor.

For the evolution of the links, it can be caused both by the linking dynamics and the epidemic spreading. Taking the change of $\langle II \rangle$ as an example, when the linking dynamics happens (with probability $1 - \alpha$), II links would increase by one if an SI link is selected, then broken, and the infected individual of the SI link is selected, and it switches to another infected individual (with probability $\frac{\langle I \rangle \langle SI \rangle w_{SI}}{2}$); II links would decrease by one if an II link is selected, then broken, and the selected infected individual switches to a susceptible individual (with probability $\langle S \rangle \langle II \rangle w_{II}$). When the epidemic spreading happens (with probability α), for the recovery event, an infected individual is selected (with

probability $\langle I \rangle$), and it recovers with probability $\mu \Delta t$. If the selected I individual has q ($0 \leq q \leq k$) infected neighbors [with probability $\binom{k}{q} (\frac{\langle II \rangle}{\langle I \rangle})^q (1 - \frac{\langle II \rangle}{\langle I \rangle})^{k-q}$], the change of II links is $-q$. For the infection event, a susceptible individual is selected (with probability $\langle S \rangle$) if it has h ($0 \leq h \leq k$) infected neighbors [with probability $\binom{k}{h} (\frac{\langle SI \rangle}{\langle S \rangle})^h (1 - \frac{\langle SI \rangle}{\langle S \rangle})^{k-h}$]; the infection happens with probability $h \lambda \Delta t$, and the change of II links in this case is h . Taking into account the formula of the expectation and the variance of the binomial distribution yields

$$\begin{aligned} \langle \dot{II} \rangle = & (1 - \alpha) \left\{ \frac{\langle I \rangle \langle SI \rangle w_{SI}}{2} - (1 - \langle I \rangle) \langle II \rangle w_{II} \right\} \frac{2}{k} \\ & + \frac{2\alpha}{N} \left\{ \mu \langle II \rangle + \lambda \langle SI \rangle + \lambda(k-1) \frac{\langle SI \rangle^2}{1 - \langle I \rangle} \right\}. \end{aligned} \quad (\text{A3})$$

With similar arguments we have

$$\begin{aligned} \langle \dot{IS} \rangle = & \frac{2(1 - \alpha)}{k} \left\{ \langle II \rangle w_{II} (1 - \langle I \rangle) + (1 - \langle II \rangle - \langle IS \rangle) w_{SS} \langle I \rangle \right. \\ & \left. - \frac{1}{2} w_{IS} \langle IS \rangle \right\} + \frac{2\alpha\mu}{N} \left(2 \langle II \rangle - \frac{\langle I \rangle}{k} \right) \\ & + \frac{2\alpha(k-1)\lambda}{N} \langle SI \rangle \left(1 + \frac{\langle SI \rangle}{1 - \langle I \rangle} \right). \end{aligned} \quad (\text{A4})$$

Finally we obtain the equations of moments with closed forms, i.e., Eqs. (A2)–(A4). This method has been used in both evolutionary game theory [42] and epidemic dynamics [10,36] before. These equations can be employed to investigate the coupled dynamics of links and epidemics for any time scales.

Furthermore, the dynamics of $\langle SI \rangle$ and $\langle II \rangle$ can help us figure out the condition under which the time scale separation is valid. The time scale separation requires that the evolution of links is mainly determined by the link-rewiring process. It implies that $\frac{2\alpha(k-1)\lambda}{N} \ll \frac{2(1-\alpha)}{k}$ and $\frac{2\alpha\mu}{N} \ll \frac{2(1-\alpha)}{k}$ based on Eqs. (A3) and (A4). Let us assume that both the infection rate λ and the recovery rate μ are of order 1. Then the two inequalities imply

$$\alpha \ll \left(\frac{k^2}{N} + 1 \right)^{-1}. \quad (\text{A5})$$

This necessary condition is a more precise criterion compared with $\alpha \ll 1$ to ensure the time scale separation. It suggests that the condition for the time scale separation would be more demanding with the increasing of the average degree k . This also supports our assumption in the main text that k should be much smaller than N .

APPENDIX B: DYNAMICAL ANALYSIS

Here we give a rigorous dynamical analysis of Eq. (6), based on which the main results in Sec. III are obtained. Rewriting

Eq. (6) leads to

$$\frac{di}{dt} = \frac{1}{A(i)} f(i), \quad (\text{B1})$$

where the cubic polynomial $f(i)$ is given by

$$\begin{aligned} & (2w_{SS}w_{II} - w_{SI}w_{II} - w_{SS}w_{SI})i^3 + (2w_{SI}w_{II} \\ & - (2 + \lambda_e)w_{SS}w_{II})i^2 + (\lambda_e w_{SS}w_{II} - w_{SI}w_{II})i. \end{aligned} \quad (\text{B2})$$

The asymptotic properties of Eq. (6) are totally determined by $f(i)$, since $A(i)$ is positive. Note that $f(0) = 0$, $i = 0$ is a fixed point.

When $2w_{SS}w_{II} - w_{SI}w_{II} - w_{SS}w_{SI} = 0$,

$$f(i) = iw_{II}[(2w_{SI} - (2 + \lambda_e)w_{SS})i + (\lambda_e w_{SS} - w_{SI})].$$

If $\lambda_e w_{SS} - w_{SI} \leq 0$, $i = 0$ is the only stable fixed point; the infection will finally die out. If $\lambda_e w_{SS} - w_{SI} > 0$, $i = 0$ is an unstable fixed point, and

$$i = \frac{w_{SI} - \lambda_e w_{SS}}{2w_{SI} - (2 + \lambda_e)w_{SS}}$$

becomes the only stable fixed point, corresponding to endemic infection.

It is shown that there exists a phase transition at $\lambda_e = w_{SI}/w_{SS}$, which is quite similar to the conventional SIS model in which the critical point is located at $\lambda = 1$.

When $2w_{SS}w_{II} - w_{SI}w_{II} - w_{SS}w_{SI} \neq 0$, it is possible for the model to give rise to bistability. Letting $w = w_{SI}/w_{SS}$, we have the following:

(a) If $w_{SS} = w_{II}$, bistable $\Leftrightarrow (\lambda_e, w) \in (2, +\infty) \times (\lambda_e, \frac{\lambda_e + \sqrt{\lambda_e^2 + (\lambda_e - 2)^2}}{2})$.

(b) If $w_{SI} = w_{II}$, bistable $\Leftrightarrow (\lambda_e, \frac{1}{w}) \in (2, +\infty) \times (\frac{4}{4 + \lambda_e^2}, \frac{1}{\lambda_e})$.

To show how we get the above results, we take the case $w_{SS} = w_{II}$ as an example. In this case,

$$f(i) = 2w_{SS}^2 i \underbrace{[(1-w)i^2 + (2w - (2 + \lambda_e))i + (\lambda_e - w)]}_{g(i)},$$

and its discriminant is denoted as $\Delta = (2w - (2 + \lambda_e))^2 - 4(1-w)(\lambda_e - w)$; then the sufficient and necessary condition for bistability is given by

$$\begin{aligned} g(0) & < 0, \\ g(1) & < 0, \\ \Delta & > 0, \\ 0 & < -\frac{2w - (2 + \lambda_e)}{2(1-w)} < 1. \end{aligned} \quad (\text{B3})$$

By solving the above set of inequalities, we obtain that $(\lambda_e, w) \in (2, +\infty) \times (\lambda_e, \frac{\lambda_e + \sqrt{\lambda_e^2 + (\lambda_e - 2)^2}}{2})$. Similarly, we get the result for the case $w_{SI} = w_{II}$.

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