Classification of link-breaking and link-creation updating rules in susceptible-infected-susceptible epidemics on adaptive networks

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In the classical susceptible-infected-susceptible (SIS) model, a disease or infection spreads over a given, mostly fixed graph. However, in many real complex networks, the topology of the underlying graph can change due to the influence of the dynamical process. In this paper, besides the spreading process, the network adaptively changes its topology based on the states of the nodes in the network. An entire class of link-breaking and link-creation mechanisms, which we name Generalized Adaptive SIS (G-ASIS), is presented and analyzed. For each instance of G-ASIS using the complete graph as initial network, the relation between the epidemic threshold and the effective link-breaking rate is determined to be linear, constant, or unknown. Additionally, we show that there exist link-breaking and link-creation mechanisms for which the metastable state does not exist. We confirm our theoretical results with several numerical simulations.

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I. INTRODUCTION

Complex networks have been studied in many fields varying from biology, ecology, and infrastructure to social networks, in which information spreads. One of the simplest, nontrivial dynamical processes on networks is the susceptible-infected-susceptible (SIS) model [1]. Epidemic models like the SIS model describe a wide variety of diffusive processes, including epidemics [2,3], opinion spreading [4], computer viruses [5], brain data transfers [6], fake news spreading [7], failure propagation [8], and internet packet routing [9]. Most studies have either addressed the dynamics of the network or the dynamics on the network. However, in many networks the dynamics (function/process) and the structure (graph/topology) coevolve. These networks are referred to as coevolutionary or adaptive networks [10]. In an adaptive spreading network, the graph adapts to the spreading process (e.g., contact with infected neighbors is avoided) and, in turn, the spreading process is constrained by the modified graph.

Many networks can be modeled as adaptive networks. For example, the brain connectome is a highly adaptive network [11]. Opinion networks, in which opinions are transferred between people, also adapt over time as people commonly prefer to contact people with similar opinions [12].

Even though adaptive networks are ubiquitous, their analysis has proven difficult. Nevertheless, disease spreading processes have been successfully modeled using adaptive networks. The seminal work by Gross *et al.* [13] introduced an adaptive susceptible-infected-susceptible (SIS) model, where a rewiring mechanism was introduced. In Gross's model, a susceptible node can be infected by neighboring infectious nodes with probability p. Infected nodes recover independently of the contagion process with probability r. In addition to this classical contagion process, the link between susceptible and infected nodes can be rewired with probability w. When a link is rewired, the link between the susceptible node and infected node is broken, and the susceptible node connects to a randomly chosen susceptible node in the network. The rewiring process is based on social distancing, a concept from social studies, indicating the tendency of healthy people to avoid infected people [14]. An extensive analysis of Gross's model was performed by Marceau *et al.* [15]. Several extensions of Gross's model have been investigated with different link-adaption rules [16,17], and Gross's model was applied to an SIR model [18] and on growing networks [19].

Although Gross *et al.* [13] have had a large impact on the field, their approach is based on a mean-field average and ignores higher-order correlations. Guo *et al.* [20] introduced a similar but slightly different approach, called the Adaptive SIS (ASIS) model, where links between susceptible and infected nodes are not rewired but temporarily broken. Independently, the link can be restored between two susceptible nodes. Hence, the network evolves according to two processes: a link-breaking and a link-recreation mechanism. For a complete initial network, an exact, implicit relationship for the number of infected nodes and the network structure was obtained. It was shown that the epidemic threshold scales linearly in the effective link-breaking rate. Aside from epidemiology, the methodology was successfully applied to model the spread of information propagation in the Adaptive Information Diffusion (AID) model [21].

To gain understanding of how the link dynamics affect the overall dynamics of adaptive networks, we propose a Generalized Adaptive SIS model (G-ASIS for short). The novel, versatile G-ASIS model comprises the Adaptive SIS (ASIS) and Adaptive Information Diffusion (AID) models by incorporating all possible link-breaking and link-creation mechanisms. Each mechanism adapts the topology of the network

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based on the infection state of the end nodes of a link. We show that six unique updating rules are available for each mechanism, leading to 36 instances in the G-ASIS model. All 36 instances are parametrized in G-ASIS and we derive a general expression for the lower bound of the epidemic threshold. Numerical simulations corroborate to our analytical estimates. For each of the 36 instances, the epidemic threshold is shown to (a) depend linearly on the effective link-breaking rate, (b) be independent of the effective link-breaking rate, or (c) the relation between the epidemic threshold and the effective link-breaking rate cannot be determined.

This paper is structured as follows. In Sec. II, we derive and explain the G-ASIS model and discuss the possible updating rules for the link dynamics. In Sec. III, we demonstrate that each of the instances of G-ASIS has an explicit epidemic threshold that is bounded from below. An implicit relation for the epidemic threshold is also derived. Next, we present simulation results in Sec. IV, and finally, we summarize and discuss our findings in Sec. V.

II. GENERALIZED ADAPTIVE SIS MODEL

A. Model description

We consider the spreading of diseases over a graph $\mathcal{G}(\mathcal{N}, \mathcal{L})$, where $\mathcal{N}$ is the set of N nodes and $\mathcal{L}$ is the set of L links. Every node i represents an individual which can be in two states: infected or healthy. The state of node i is modeled using a Bernoulli random variable, where $X_i(t) = 1$ indicates that node i is infected at time t and $X_i(t) = 0$ indicates that node i is healthy but susceptible to the disease. Infected nodes can infect a neighboring susceptible node, which is modeled as a Poisson process with rate β. Independently, an infected node cures with Poisson rate δ. The adjacency matrix $a_{ij}(t)$ indicates whether nodes i and j are linked $[a_{ij}(t) = 1]$ in the network at time t or not $[a_{ij}(t) = 0]$. The state $X_i(t)$ of node i changes as follows:

$$\frac{d\,\mathbb{E}[X_i(t)]}{dt} = \mathbb{E}\left[-\delta X_i(t) + \beta[1 - X_i(t)]\sum_{j=1}^{N} X_j(t)a_{ij}(t)\right]. \tag{1}$$

The right-hand side of Eq. (1) consists of two parts: an infected node i cures with rate δ and a susceptible node i can be infected by each of its neighboring infected nodes with rate β.

Besides the dynamic spreading process, the graph evolves over time as well. The link a_{ij} between node i and j is modeled as a Bernoulli random variable. A link between two nodes can be broken and re-created based on the state of the end nodes of the link. These link-breaking and link-creation mechanisms make the network adaptive. Here we present the Generalized Adaptive SIS model (G-ASIS for short) which includes all possible updating rules for the link-breaking and link-creation mechanisms. The following assumptions have been made. The topology only changes based on two independent processes: (a) a link-creation process f_{cr} with Poisson rate ξ and (b) a link-breaking process f_{br} with Poisson rate ζ. For each of these mechanisms, the link changes based on the state of the end nodes of the link. The interaction of a link $a_{ij}(t)$ depending on node i and j is assumed to be symmetric. Hence, the adjacency matrix $A(t)$ with elements $a_{ij}(t)$ is symmetric at all times. These assumptions determine the governing equations of the link a_{ij} as

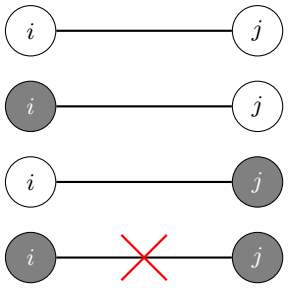


FIG. 1. Schematic overview of two connected nodes. Gray nodes are infected nodes, white nodes are healthy nodes. The decision to break or create the link between node i and j depends on the viral states X_i and X_j. In this example, the link is broken when $X_i = X_j = 1$ which corresponds to link-breaking rule $f_{\text{br}} = X_iX_j$.

$$\frac{d\,\mathbb{E}[a_{ij}(t)]}{dt} = a_{ij}(0)\mathbb{E}\big[-\zeta a_{ij}(t)f_{\text{br}}\{X_i(t), X_j(t)\} + \xi(1 - a_{ij}(t))f_{\text{cr}}\{X_i(t), X_j(t)\}\big], \tag{2}$$

where f_{br} and f_{cr} are specific choices for the link-breaking and link-creation mechanism, respectively. The initial link existence $a_{ij}(0)$ indicates that only links in the initial network can be broken and recreated, whereas links in the complement of the initial graph are never created (nor broken). We assume that the initial network $A(0)$ is connected, however, the connectivity constraint can be circumvented by investigating each connected component separately.

B. Derivation of the updating rules

The link-breaking mechanism f_{br} and link-creation mechanism f_{cr} in G-ASIS depend on the state of the nodes $X_i(t)$ and $X_j(t)$, but not on $a_{ij}(t)$ nor explicitly on the time t. We next determine all possible updating rules for f_{br} and f_{cr}. For convenience, a rule is denoted by f and applies to f_{br} as well as to f_{cr}. Each rule f of a link between node i and j has Bernoulli random variables X_i and X_j as input. Each rule f is a linear or quadratic function of X_i and X_j that evaluates to zero or one, similar to a logical gate.

We classify the updating rules according to the number of possible inputs that give $f = 1$. Consider for example the rule $f = X_iX_j$. Then, $f = 1$ only for $X_i = X_j = 1$. Any other input for X_i and X_j yields $f = 0$. This rule is visualized in Fig. 1. In the same way, three other rules can be derived where $f = 1$ for one combination of X_i and X_j. The number of permutations of this type can be computed as follows. There are four possible inputs (combinations of X_i and X_j) and one positive outcome: $\binom{4}{1} = 4$. All complying rules are:

$$X_iX_j, \quad (1 - X_i)X_j,$$
$$(1 - X_i)(1 - X_j), \quad X_i(1 - X_j).$$

There are rules for which two combinations of X_i and X_j yield $f = 1$. As an example, consider the rule $f = (X_i - X_j)^2$. Then $f = 1$ if X_i is not equal to X_j. There are six rules of this type, because there are four inputs and two combinations; $\binom{4}{2} = 6$.

These six rules are:

$$\begin{array}{ccc}(X_i - X_j)^2, & X_i, & X_j,\\ 1-(X_i - X_j)^2, & (1-X_i), & (1-X_j).\end{array}$$

Third, there are rules for which three combinations of X_i and X_j yield $f = 1$. For example, consider $f = 1 - X_iX_j$. The function's result is one if $X_i = 0$ or $X_j = 0$. The only situation to find $f = 0$ occurs when $X_i = X_j = 1$. All four rules of this type, namely, $\binom{4}{3} = 4$, are:

$$\begin{array}{cc}1-X_iX_j, & 1-(1-X_i)X_j,\\ 1-(1-X_i)(1-X_j), & 1-X_i(1-X_j).\end{array}$$

Two trivial rules have not yet been specified. The trivial rules $f = 1$ (which occurs in $\binom{4}{4} = 1$ case) and $f = 0$ ($\binom{4}{0} = 1$) are independent of the state of nodes X_i and X_j. Including the trivial rules, the total number of possible rules is $\sum_{k=0}^{4}\binom{4}{k} = 2^4 = 16$.

Each of the 16 possibilities for the function f can be rewritten, using the binomial property that $\mathbb{E}[X_i^2] = \mathbb{E}[X_i]$, in the following parametrized form:

$$f(X_i, X_j) = a + bX_i + \tilde{b}X_j + cX_iX_j, \tag{3}$$

where the parameters $a, b, \tilde{b}, c \in \mathbb{Z}$. Since the assumed network is undirected, the function f is symmetric in X_i and X_j, which implies that $\tilde{b} = b$ in Eq. (3). This removes eight asymmetric updating rules from the original derivation and simplifies Eq. (3) to

$$f(X_i, X_j) = a + b(X_i + X_j) + cX_iX_j, \tag{4}$$

where the parameters $a, b, c \in \mathbb{Z}$.

The trivial updating rules $f = 0$ and $f = 1$ are not particularly relevant. Choosing the updating rule $f = 0$ for either the link-breaking or link-creation mechanism removes the mechanism entirely from the governing equation (2). Hence, there is an exponentially fast convergence to the steady-state topology, without any dependence on the SIS process. The updating rule $f = 1$ is also a nonadaptive rule which is independent of the infection state of X_i and X_j. Moreover, for our analysis of the epidemic threshold in the G-ASIS model, the nonadaptive rules are not incorporated since they barely provide any further insight.

After the removal of the nonadaptive and nonsymmetric rules for the function f, only six updating rules remain. Therefore, the link-breaking mechanism f_{br} and link-creation mechanism f_{cr} each have six updating rules in the G-ASIS model. Since the link-breaking mechanism f_{br} and the link-creation mechanism f_{cr} can be chosen independently, and for each of them six updating rules are available, in total 36 Markov processes for topology updating are contained in G-ASIS. Each instance of G-ASIS contains two mechanisms: a link-breaking mechanism f_{br} and a link-creation mechanism f_{cr}, which are given in general form by Eq. (4). An overview of all updating rules is presented in Table I [22].

TABLE I. All updating rules for the link-breaking and the link-creation mechanism in the G-ASIS model. The rules for the link-breaking and link-creation mechanisms are structured. The inverse of any rule f is $1 - f$. Also, taking the multiplication of two rules is equivalent to taking the intersection between the number of times a positive result for the rules is found.

Rule f	a	b	c	Gate
X_iX_j	0	0	1	AND
$1 - X_iX_j$	1	0	−1	NAND
$(1 - X_i)(1 - X_j)$	1	−1	1	NOR
$1 - (1 - X_i)(1 - X_j)$	0	1	−1	OR
$(X_i - X_j)^2$	0	1	−2	XOR
$1 - (X_i - X_j)^2$	1	−1	2	XNOR

As an example, we consider the Adaptive SIS model, where the link between a susceptible node and an infected node is broken to prevent the spreading of the disease. Hence, the link-breaking mechanism f_{br} is equal to the updating rule $f_{\mathrm{br}} = (X_i - X_j)^2$ and the corresponding parameters in Eq. (4) are $(a_{\mathrm{br}}, b_{\mathrm{br}}, c_{\mathrm{br}}) = (0, 1, -2)$. When both end nodes of a link are susceptible, the link between the nodes is restored. The link-creation mechanism is therefore $f_{\mathrm{cr}} = (1 - X_i)(1 - X_j)$ with parameters $(a_{\mathrm{cr}}, b_{\mathrm{cr}}, c_{\mathrm{cr}}) = (1, -1, 1)$.

III. THEORETICAL RESULTS

For analytic feasibility only, we mainly confine ourselves in this paper to the complete initial graph, but we expect that the conclusions and insights also hold for any other graph. One of the main concepts in epidemiology is the epidemic threshold τ_c. The epidemic threshold τ_c in a finite graph specifies a small interval for the effective infection rate $\tau = \beta/\delta$ in which the process quickly changes from the disease-free phase to the endemic phase [1]. The epidemic threshold τ_c can be defined as the largest value of the effective infection rate τ for which the prevalence y exponentially decays to zero over sufficiently large time [23]. Finding an analytical expression for the epidemic threshold is generally infeasible due to the complexity of the process. It is, however, possible to derive lower and upper bounds for the epidemic threshold for the complete initial network.

A. Lower bound on the epidemic threshold

Following Ogura and Preciado [24], the epidemic threshold τ_c can be bounded from below. This methodology was also successfully applied to the static SIS model by Van Mieghem [25, Theorem 17.3.1]. The static SIS model is obtained from the G-ASIS model by setting $\zeta = \xi = 0$. We state one of our main results in the following theorem.

Theorem III.1. The epidemic threshold τ_c for the G-ASIS model is bounded from below by

$$\tau_c \geqslant \frac{1}{\rho}\left[1 + \frac{\omega(\mathbf{1}_{\{a_{\mathrm{br}}=0, b_{\mathrm{br}}=1, c_{\mathrm{br}}=-1\}}) - (\mathbf{1}_{\{a_{\mathrm{cr}}=1, b_{\mathrm{cr}}=0, c_{\mathrm{cr}}=-1\}\cup\{a_{\mathrm{cr}}=0, b_{\mathrm{cr}}=1, c_{\mathrm{cr}}=-2\}})}{(1 - \mathbf{1}_{\{a_{\mathrm{cr}}=1, b_{\mathrm{cr}}=-1, c_{\mathrm{cr}}=1\}}) + \delta/\xi}\right], \tag{5}$$

where ρ is the spectral radius (the largest eigenvalue of the adjacency matrix) of the initial graph, $\omega = \zeta/\xi$ is the effective link-breaking rate and $\mathbf{1}_x$ is the indicator function which is one if condition x is satisfied, and zero otherwise.

Proof. See Appendix B. ■

Theorem III.1 states that the epidemic threshold τ_c can be reduced by introducing adaptive link-breaking and link-creation mechanisms because the second term in Eq. (5) can be negative and $\tau_c \geqslant \frac{1}{\rho}$ holds for the SIS model in a static graph [26]. If the link-breaking coefficients satisfy $a_{\mathrm{br}} = 0, b_{\mathrm{br}} = 1, c_{\mathrm{br}} = -1$ [corresponding to link-breaking rule $f_{\mathrm{br}} = 1 - (1 - X_i)(1 - X_j)$], then the epidemic threshold τ_c in Eq. (5) has a nonzero dependence on the effective link-breaking rate ω. The link-creation rule can be chosen freely. Hence, for $1 \times 6 = 6$ out of the 36 instances of G-ASIS, the epidemic threshold τ_c increases at least linearly with the effective link-breaking rate ω. For the remaining 30 instances in G-ASIS, satisfying $f_{\mathrm{br}} \neq 1 - (1 - X_i)(1 - X_j)$, the lower bound in Eq. (5) is independent of the effective link-breaking rate ω and is similar to the lower bound of the classical SIS epidemic threshold [26,27]. In epidemiology, a high epidemic threshold is preferable, because the disease only develops into an endemic for higher infection rates. Other areas of application, such as information spreading and human brain interactions, benefit from a low epidemic threshold as fast communication is advantageous for these phenomena.

B. Upper bound on the epidemic threshold

We denote the fraction of infected nodes by $Z = \frac{1}{N}\sum_{i=1}^{N} X_i$. Above the epidemic threshold τ_c, the process is in the *metastable state*, where stochastic variables are denoted by an asterisk (*) [28]. We denote by $y = \mathbb{E}[Z^*]$ the average metastable fraction of infected nodes, commonly known as the prevalence, and by d_i the degree of node i. Combining Eqs. (1) and (2), an analytic, implicit quadratic relationship for the prevalence y can be obtained, similarly as in Refs. [20,21].

Theorem III.2. The metastable prevalence y for a complete initial network satisfies the quadratic equation

$$y^2 + \left[\frac{2b_{\mathrm{cr}}N\tau - (2b_{\mathrm{cr}} + c_{\mathrm{cr}})\tau + c_{\mathrm{br}}\omega + c_{\mathrm{cr}}}{c_{\mathrm{cr}}N\tau}\right]y + \left\{\frac{(N-1)a_{\mathrm{cr}}}{c_{\mathrm{cr}}N} - \frac{a_{\mathrm{br}}\omega + a_{\mathrm{cr}}}{c_{\mathrm{cr}}N^2}\mathbb{E}\left[\sum_{i=1}^{N} d_i^*\right] + \mathrm{Var}(Z^*) - \frac{(2b_{\mathrm{br}} + c_{\mathrm{br}})\omega + 2b_{\mathrm{cr}} + c_{\mathrm{cr}}}{c_{\mathrm{cr}}N^2}\mathbb{E}\left[\sum_{i=1}^{N} d_i^* X_i^*\right]\right\} = 0. \quad (6)$$

Proof. See Appendix C. ■

The quadratic formula Eq. (6) for the prevalence y leads to an exact, implicit expression for the epidemic threshold τ_c:

Theorem III.3. The epidemic threshold τ_c in the G-ASIS model for a complete initial network is implicitly given by

$$\tau_c = \frac{\frac{c_{\mathrm{br}}}{c_{\mathrm{cr}}}\omega + 1}{2\frac{b_{\mathrm{cr}}}{c_{\mathrm{cr}}}(1 - N) + 1 - Nh(\omega, \xi)}, \quad (7)$$

where $h(\omega, \xi)$ is defined in Eq. (D8) in Appendix D. Moreover, for 27 instances of G-ASIS, the epidemic threshold τ_c is bounded by a linear function in ω and for 9 instances, the epidemic threshold is bounded by a constant.

Proof. See Appendix D. ■

Theorems III.1 and III.3 enable us to classify the instances of G-ASIS: one type has a linear relation between the epidemic threshold τ_c and the effective link-breaking rate $\omega = \zeta/\xi$ and for the other type, the epidemic threshold τ_c is independent of the effective link-breaking rate ω. The Adaptive Information Diffusion (AID) model is one example of a model with a constant τ_c and the ASIS model is an example where τ_c is linear in ω. Comparing the results of Theorems III.1 and III.3, two striking differences appear. First, the lower bound for the epidemic threshold τ_c in Eq. (5) is explicit, whereas Eq. (7) depends *implicitly* on the function $h(\omega, \xi)$. Second, the lower bound in Eq. (5) concludes that six instances have a linear relation between the epidemic threshold τ_c and the effective link-breaking rate ω, which contrasts the upper bound in Theorem III.3, which has 27 linear-scaling instances. Subsequently, $27 - 6 = 21$ instances have an undetermined relation: their lower bound is constant in ω, whereas their upper bound scales linearly in ω. In Sec. IV, simulation results indicate that undetermined relations can exhibit both linear and constant behavior. So far, the actual relation between the epidemic threshold τ_c and the effective link-breaking rate ω is still unknown. The relation between the epidemic threshold τ_c and the effective link-breaking rate ω can be summarized as follows:

6 instances:	linear in ω,
9 instances:	constant in ω,
21 instances:	undetermined.

C. Nonexistent metastable states

The quadratic relationship Eq. (6) for the prevalence y always has the all-healthy state $y = 0$ as a solution. Above the epidemic threshold τ_c, we conjecture that some instances of G-ASIS do not possess a metastable state. Then Eq. (6) does **not** possess a unique, real-valued, nonzero solution for the prevalence y.

Conjecture III.1. The metastable state in the G-ASIS model for a complete initial network does *not* exist when all of the following three conditions hold:

$$c_{\mathrm{cr}} < 0, \quad c_{\mathrm{br}} > 0, \quad \omega > -\frac{c_{\mathrm{cr}}}{c_{\mathrm{br}}}. \quad (8)$$

Sketch of the proof. See Ref. [29]. ■

If the metastable state does not exist, then simulations show a nonempty region of τ-values above the epidemic threshold τ_c in which oscillatory behavior is observed for the prevalence y. One example of an instance of G-ASIS showing oscillatory behavior is the AID model in Fig. 2(c) in Sec. IV.

IV. NUMERICAL SIMULATIONS

The time in the governing equations (1) and (2) of the G-ASIS Markov process can be rescaled by curing rate δ; hence, we always take $\delta = 1$ and thus measure the time in units of the average curing time. All figures in this section are simulations of the G-ASIS Markov process. As initial condition, we used a complete graph as initial network and all nodes are initially infected. The continuous-time Markov process

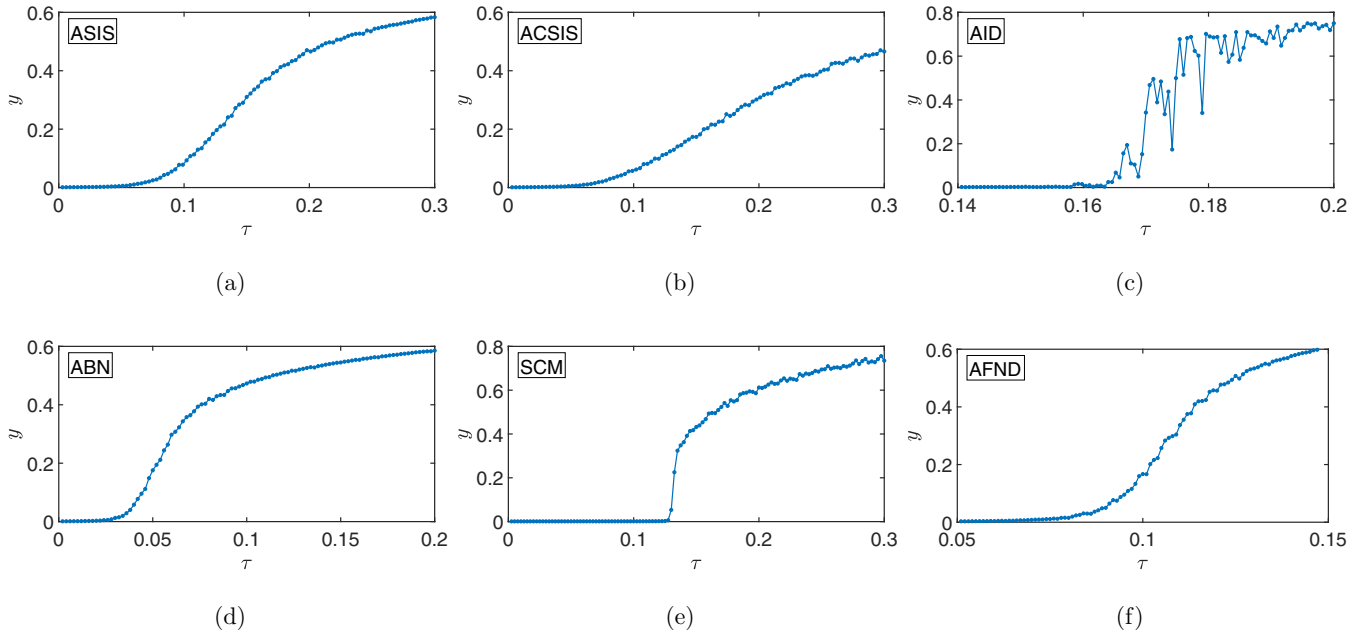


FIG. 2. The relation between the effective infection rate τ and prevalence y for various instances of the G-ASIS model. We have taken $N = 40$, $\delta = 1$, $\varepsilon = 0.001$ and a complete initial network for all models. For (a), (b), (d), (f), we have taken $\zeta = \xi = 1$, for (c) $\zeta = 0.5$, $\xi = 0.1$ and for (e) $\zeta = \xi = 0.1$.

has been approximated by a sampled-time Markov chain [25] with a sufficiently small time step Δt, which is taken to be $\Delta t = 0.05$. Each simulation has been performed using 10^6 time units, measured in units of the average curing time.

A. Phase transitions

The relation between the prevalence y and the effective infection rate τ shows a phase transition from the all-healthy state to the endemic state. Such phase transitions are shown for various instances of the G-ASIS model in Fig. 2. The simulations have been performed using the ε-SIS approach described by Van Mieghem and Cator [30], where a small nodal self-infection rate $\varepsilon < \frac{\delta}{N}$ is introduced to exclude an absorbing state. The ε-SIS model allows for the numerical estimation of the metastable state.

The numerical results for the prevalence y in the ASIS model are presented in Fig. 2(a). Below the epidemic threshold $\tau_c \approx 0.05$, the prevalence is zero. For effective infection rates $\tau > \tau_c$ the prevalence y increases rapidly. The growth saturates as the effective infection rate τ increases and the prevalence y asymptotically increases to 1 as $\tau \to \infty$.

The Adaptive Contagious SIS (ACSIS) model is a variation on the ASIS model, where links are not only broken between susceptible and infected nodes, but also between two infected nodes. Two people suffering from the same disease are more likely to stay at home, effectively breaking links with each other. The phase transition of ASIS [Fig. 2(a)] and ACSIS [Fig. 2(b)] and the epidemic threshold are nearly equivalent, although the prevalence y is generally lower in the ACSIS model. Due to the extra link-breaking rule in the ACSIS model, the disease is able to spread less quickly, causing the prevalence to decrease.

In contrast to the ASIS and ACSIS model, the Adaptive Information Diffusion (AID) model describes the spreading of information. In the AID model, nodes represent people and links their social interactions. The link between two susceptible nodes is broken, because the nodes have no interest in one another as both do not have the information. The link between susceptible and infected nodes is created to enhance the propagation of information. Conjecture III.1 states that the metastable state does not exist in the AID model, which is illustrated in Fig. 2(c). Just above the epidemic threshold τ_c, the process highly fluctuates, indicating that the metastable state does not exist. The process does not collapse to the all-healthy state either, but instead oscillates for effective infection rates $\tau \in [0.16, 0.18]$. Under these conditions, the AID model has no prevalence y and the model can only predict the average effectiveness of information spreading throughout the network for small or large τ.

The Adaptive Brain Network (ABN) model is also contained in the G-ASIS model. In the ABN model, nodes are parts of the human brain and links connect the different parts. Nodes can be active (infected) or inactive (healthy) at any time. Active nodes cure and infect their inactive neighbors as usual. The ABN topology updating rules are derived from the homeostatic structural plasticity in the brain, where a new link is created between two inactive nodes and existing links are removed when both nodes are active. When one node is infected and one is healthy, the link between the nodes is preserved. The phase transition in Fig. 2(d) is comparable to the ASIS model, although the epidemic threshold is smaller and the ascent of the prevalence y is steeper around the epidemic threshold τ_c.

Another model in the G-ASIS model is the Scientific Collaboration Model (SCM), where nodes represent researchers who either have interest or not in a particular research area. Links represent collaborations between researchers. Researchers can spread their interest to collaborating, connected researchers. Independently, researchers can

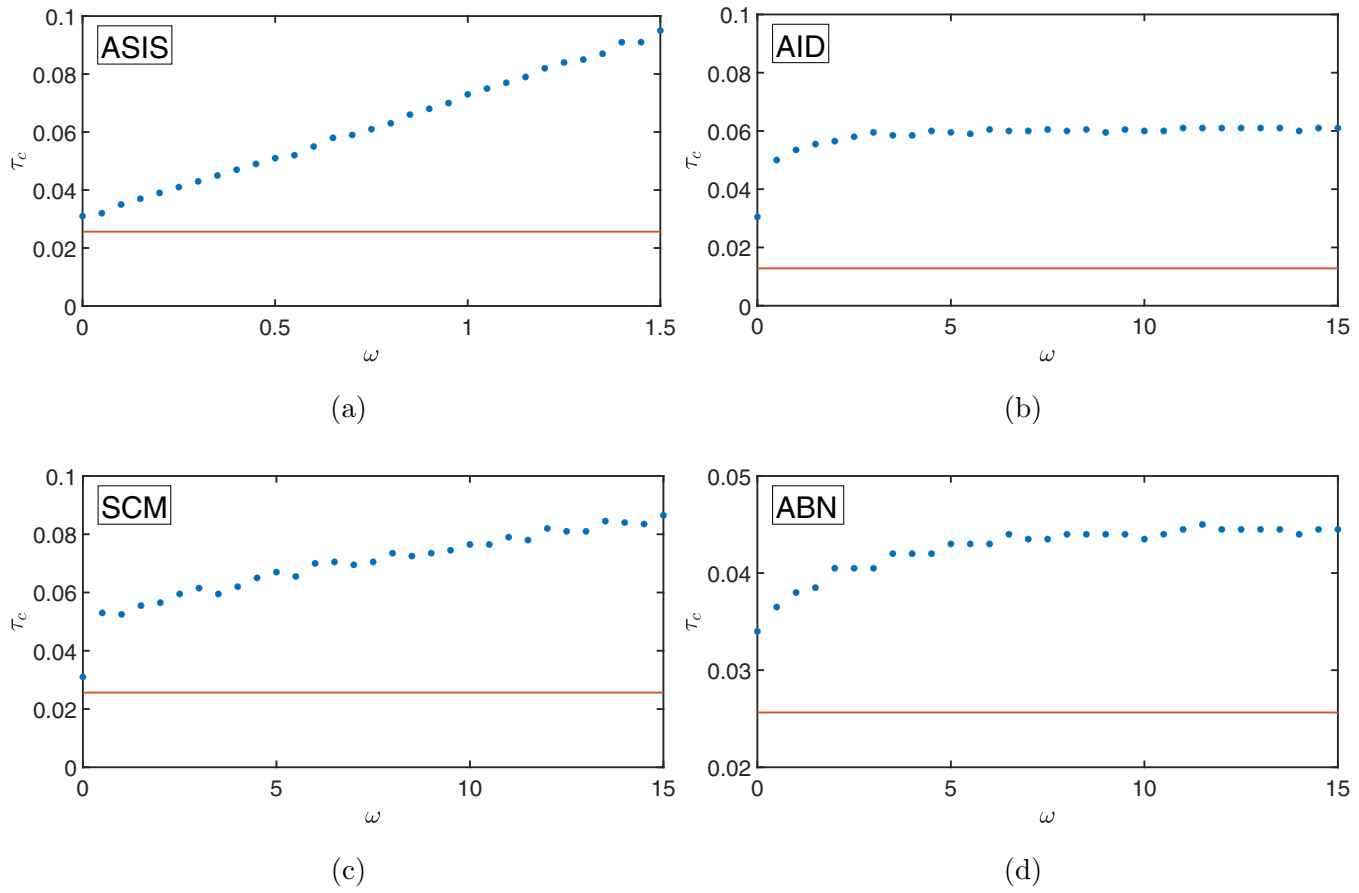


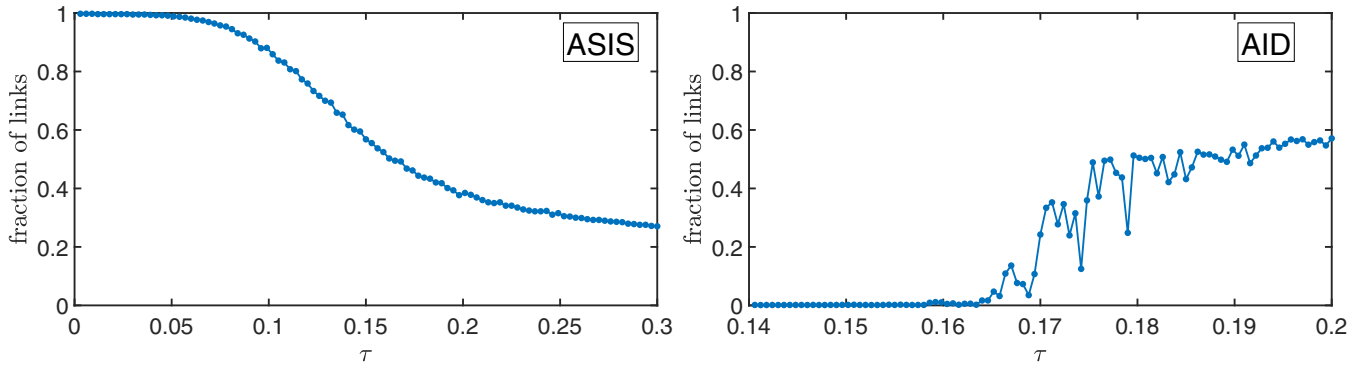
FIG. 3. The epidemic threshold τ_c as a function of the effective link-breaking rate ω for four instances of the G-ASIS model. The data points are obtained from simulations and the solid line represents the lower bound from Theorem III.1. The theory predicts a constant relationship (b) or it is undetermined (a), (c), (d). We have taken $N = 40$, $\delta = \xi = 1$, $\varepsilon = 0.001$ and a complete initial network.

lose, forget or do not pay attention to the research area. Besides these processes, the network is adaptive in the following way. Researchers break their link if both are not interested in the research area. Since there is a potential collaboration between susceptible and infected researchers, their link persists. Finally, the link is created between two researchers who share interests. Different from most instances of G-ASIS, the numerical estimation of the prevalence y for the SCM model sensitively depends on the initial conditions. The phase diagram in Fig. 2(e) is comparable to the ABN model in Fig. 2(d); however, the ascent of the prevalence y is very steep around the epidemic threshold τ_c.

The Adaptive Fake News Diffusion (AFND) model was introduced in Ref. [31] to model the spread of fake news in a healthy network. The nodes in the AFND model represent people who are either under the influence of a fake news item, or do not believe the item. People are connected to other people (and possible other sources) over adaptive links. Infected nodes try to persuade healthy, neighboring nodes to believe the fake news item. At the same time, infected nodes 'cure' from the fake news as well. Links in the AFND model can be broken between susceptible and infected nodes based on social awareness against fake news. Simultaneously, two healthy nodes have no interest in keeping in touch and their link can be broken. Additionally, links are created between healthy and susceptible nodes since fake news items are mostly sensational: the fake news directly appeals to the human's emotions. Hence, the spreading of fake news causes links between susceptible and infected nodes to be created and broken simultaneously. The behavior for the AFND model shown in Fig. 2(f) is similar to that of the ASIS and ACSIS model in Figs. 2(a) and 2(b), respectively.

B. Relation between epidemic threshold and effective link-breaking rate

Although the epidemic threshold was shown for various models in Fig. 2, the effect of the link-updating mechanisms on the spreading of the disease remains unclear. Therefore, the dependence of the epidemic threshold τ_c on the effective link-breaking rate ω is shown for various models in Fig. 3. The dots represent numerical simulations whereas the solid line represents the lower bound from Theorem III.1. The result from Theorem III.3 is not shown in Fig. 3 because Eq. (7) is merely an implicit relation for the epidemic threshold τ_c. The AID model in Fig. 3(b) shows nearly constant behavior, which is in agreement with Theorem III.3. The ASIS, SCM and ABN models are considered in Figs. 3(a), 3(c), and 3(d), respectively. The theory was not conclusive about the relation between the epidemic threshold τ_c and the effective link-breaking rate ω. Figure 3(a) shows a clear linear relationship and Fig. 3(d) depicts a nearly constant relation-



(a) (b)

FIG. 4. The relation between the effective infection rate τ and the average fraction of links $\mathbb{E}[L]/[\frac{1}{2}N(N-1)]$ for two instances of the G-ASIS model. We have taken $N = 40$, $\delta = 1$, $\varepsilon = 0.001$ and a complete initial network for all models. Subfigure (a) shows the ASIS model with $\zeta = \xi = 1$ and (b) illustrates the AID model with $\zeta = 0.5, \xi = 0.1$.

ship. In contrast, the slope in Fig. 3(c) appears to be constant, but the relatively small slope indicates a weak relationship between the effective link-breaking rate ω and the epidemic threshold τ_c.

C. The metastable topology

In the G-ASIS model, the topology of the underlying graph is constantly changing over time. The metastable graph, though still changing around a fixed number of links, allows for the estimation of any graph metric in the metastable state. When the effective infection rate τ is smaller than the epidemic threshold τ_c, the prevalence y is zero and the average number of links is equal to

$$\mathbb{E}[L] = \frac{a_{\mathrm{cr}}}{a_{\mathrm{br}}\omega + a_{\mathrm{cr}}}\frac{1}{2}N(N-1), \quad \text{for } \tau < \tau_c, \tag{9}$$

which directly follows from (6) in Theorem III.2 by substituting an all-healthy population $y = 0$. However, the number of metastable links in the endemic state $\tau > \tau_c$ can be computed from neither Eq. (6) nor Eq. (7) in closed form.

Figure 4 illustrates the behavior for two instances of G-ASIS below and above the epidemic threshold τ_c. The ASIS model in Fig. 4(a) starts with a completely connected graph at $\tau = 0$, because the prevalence is zero and the link-breaking mechanism has not been activated yet. If the effective infection rate τ is larger than the epidemic threshold τ_c, then the prevalence y is nonzero [see Fig. 2(a)] and the link-breaking mechanism reduces the fraction of links. As the effective infection rate τ increases up to infinity, the prevalence y increases to 1 and the link-creation mechanism between two susceptible nodes is rarely activated. Hence, the fraction of links decreases to zero. For the AID model in Fig. 4(b), we observe opposite behavior. If the effective infection rate τ is

TABLE II. A selected set of instances from the G-ASIS model and their properties. The table assumes $\delta = 1$.

Model name and appearance in literature	Updating rules		Metastable state always exists (conjecture)	Lower bound on epidemic threshold τ_c	Upper bound on epidemic threshold τ_c
	link-breaking	link-creation			
ASIS model [4,20,21]	$(X_i - X_j)^2$	$(1-X_i)(1-X_j)$	Yes	$\frac{1}{\rho}$	Linear
ACSIS model [31]	$1-(1-X_i)(1-X_j)$	$(1-X_i)(1-X_j)$	Yes	$\frac{1}{\rho}(1+\omega\xi)$	Linear
AID model [21]	$(1-X_i)(1-X_j)$	$(X_i - X_j)^2$	No	$\frac{1}{\rho}\left(\frac{1}{1+\xi}\right)$	Constant
ABN model	X_iX_j	$(1-X_i)(1-X_j)$	Yes	$\frac{1}{\rho}$	Linear
SCM model	$(1-X_i)(1-X_j)$	X_iX_j	Yes	$\frac{1}{\rho}$	Linear
AFND model [31]	$1-X_iX_j$	$(X_i - X_j)^2$	Yes	$\frac{1}{\rho}\left(\frac{1}{1+\xi}\right)$	Linear

smaller than the epidemic threshold τ_c, then the prevalence y is zero. In the AID model, links are broken between susceptible nodes and created between susceptible-infected pairs. Therefore, the fraction of links is zero as well. In the endemic state $\tau > \tau_c$, the prevalence y increases, which enables the creation of links in the network. As the effective infection rate τ approaches infinity, the prevalence increases to 1. Then the fraction of links also converges to 1 because the link-breaking rule between susceptible nodes is rarely used as there are hardly any susceptible nodes.

D. Summary

We summarize all results in Table II. For the ACSIS model the epidemic threshold τ_c is a linear function of the effective link-breaking rate ω and the metastable state always exists. The AID model, which has a constant relation between the epidemic threshold τ_c and the effective link-breaking rate ω, does not possess a metastable state for sufficiently large ω. Unfortunately, the relation between τ_c and ω in the ASIS, AFND, ABN, and SCM models could not be determined. The simulations support the hypothesis that the lower bound is strict for the ABN model and the linear bound is correct for the ASIS, AFND, and SCM models, indicating that undetermined models may show different behavior. All 36 instances of G-ASIS are listed in Table III in Appendix E.

V. CONCLUSION

In this paper, the Generalized Adaptive SIS model is introduced. The G-ASIS model consists of two adaptive mechanisms: links between nodes can be broken and created. We have shown that for each mechanism, six updating rules are available. Hence, the G-ASIS model contains 36 adaptive processes. Out of these 36 instances, 9 are likely to have a nonexistent metastable state for sufficiently large effective link-breaking rates ω. If the metastable state is nonexistent and the effective infection rate τ is larger than the epidemic threshold τ_c, then the Markov process shows large fluctuations, indicating that the process is unstable. We have also shown that the relation between the epidemic threshold τ_c and the effective link-breaking rate ω is linear for 6 instances, constant for 9 instances and undetermined for 21 instances.

The G-ASIS model can be extended by allowing for heterogeneous curing, infection, link-breaking and link-creation rates. Heterogeneous parameters are required for modeling processes with nonuniform nodes or links. For example, in epidemics, people from different age groups are likely to react differently to a disease, and for information propagation, some people are more influential (have a larger infection parameter) than others.

In the G-ASIS model, the link-breaking and link-creation mechanisms depend on the viral state of the nodes i and j. The general formulation of any rule f, which is determined in Eq. (4), can be generalized by allowing the parameters a, b and c to have any nonnegative value. Using this approach, more focus can be laid on a particular link-creation or link-breaking process. For example, suppose that the link between two susceptible nodes can be broken with rate a_{br} and the link can be broken between two infected nodes with rate $3a_{\mathrm{br}}$, then link-breaking is more likely to happen between two infected nodes. By allowing a, b and c to have any nonnegative value, the G-ASIS model includes an infinitely large class of adaptive processes and is capable of modeling more real-world spreading phenomena.

Another promising area of research is the investigation of the mean-field approximation of the governing equations (1) and (2) and the subsequent derivation of constraints on the epidemic threshold. Achterberg [31] has derived a cubic equation for the steady state of the first-order mean-field equations for the G-ASIS model using Eq. (4). The steady state of the mean-field equations appears to show poor agreement with the metastable state from the G-ASIS model, which is in contrast to the static SIS model [32]. Nevertheless, the mean-field estimate can be relevant to determine the epidemic threshold. In particular, in the classical SIS model the correlation between two adjacent nodes is always positive [33]. Then the mean-field estimate for the epidemic threshold is always a lower bound of the true epidemic threshold. As far as we know, nodal correlations have not been analyzed for adaptive processes.

Finally, the G-ASIS model can be extended to "simplicial contagion" [34]. Besides infection spreading over 1-simplices (links), simplicial contagion considers higher-order simplices to enhance the spreading of the disease. For example, the 2-simplex considers the interaction between three nodes (a full triangle). In addition to extending the governing equation (1), the topology updating rule (4) can also be generalized to link-breaking and link-creation mechanisms between D-dimensional simplices.

APPENDIX A: GENERAL REMARKS

Throughout the Appendices, the explicit time-dependence of each stochastic variable $X_i(t)$ is omitted for readability: We write X_i instead of $X_i(t)$.

APPENDIX B: PROOF OF THEOREM III.1

We follow the method of Ogura and Preciado [24]. The change over time of the term $\mathbb{E}[a_{ij}X_i]$ can be computed analogous to Eqs. (1) and (2), and it is found that

$$\frac{d\,\mathbb{E}[a_{ij}X_i]}{dt} = a_{ij}(0)\Big\{(a_{\mathrm{cr}}+b_{\mathrm{cr}})\xi\,\mathbb{E}[X_i] - (a_{\mathrm{br}}\zeta + b_{\mathrm{br}}\zeta + a_{\mathrm{cr}}\xi + b_{\mathrm{cr}}\xi + \delta)\mathbb{E}[a_{ij}X_i] + (b_{\mathrm{cr}}+c_{\mathrm{cr}})\xi\,\mathbb{E}[(1-a_{ij})X_iX_j] - (b_{\mathrm{br}}+c_{\mathrm{br}})\zeta\,\mathbb{E}[a_{ij}X_iX_j] + \beta\mathbb{E}\Big[(1-X_i)a_{ij}\sum_{k=1}^{N}a_{ik}X_k\Big]\Big\}. \tag{B1}$$

In this proof, the governing equations of $\mathbb{E}[X_i]$ from Eq. (1) and $\mathbb{E}[a_{ij}X_i]$ from Eq. (B1) are used. The governing equations (1) and (B1) are rewritten in terms of $\mathbb{E}[X_i]$ and $\mathbb{E}[a_{ij}X_i]$, and the remaining terms are denoted by W. Our goal is to define W such that it is negative. For Eq. (B1), we rewrite the infection term with coefficient β as

$$\beta\mathbb{E}\left[(1-X_i)a_{ij}\sum_{k=1}^{N}a_{ik}X_k\right]=\beta\sum_{k=1}^{N}\mathbb{E}[a_{ik}X_k-X_ia_{ik}X_ka_{ij}-(1-a_{ij})a_{ik}X_k].$$

Then Eq. (B1) can be rewritten as

$$\frac{d\,\mathbb{E}[a_{ij}X_i]}{dt}=a_{ij}(0)\left\{(a_{\rm cr}+b_{\rm cr})\xi\mathbb{E}[X_i]-(a_{\rm br}\zeta+b_{\rm br}\zeta+a_{\rm cr}\xi+b_{\rm cr}\xi+\delta)\mathbb{E}[a_{ij}X_i]+\beta\mathbb{E}\left[\sum_{k=1}^{N}a_{ik}X_k\right]+W_A\right\},\tag{B2}$$

where the remaining terms of the network W_A are

$$W_A=(b_{\rm cr}+c_{\rm cr})\xi\mathbb{E}[(1-a_{ij})X_iX_j]-(b_{\rm br}+c_{\rm br})\zeta\mathbb{E}[a_{ij}X_iX_j]-\beta\sum_{k=1}^{N}\mathbb{E}[X_iX_ka_{ik}a_{ij}+(1-a_{ij})a_{ik}X_k].$$

Similarly for $\mathbb{E}[X_i]$,

$$\frac{d\,\mathbb{E}[X_i]}{dt}=-\delta\mathbb{E}[X_i]+\beta\sum_{k=1}^{N}\mathbb{E}[a_{ik}X_k]+W_X,\tag{B3}$$

where the remaining terms for the nodes W_X are

$$W_X=-\beta\sum_{k=1}^{N}\mathbb{E}[X_ia_{ik}X_j].$$

The remaining term W_X is always negative, whereas W_A is only negative in some cases. Each positive term in W_A is merged with other terms to ensure that W_A is negative. The term W_A is surely negative when each of the individual components is negative. The term with infection rate β is negative. For the link-breaking rate ζ, the case $b_{\rm br}+c_{\rm br}=-1$ is a potential problem. By applying $b_{\rm br}+c_{\rm br}=-1$, Table I illustrates that $a_{\rm br}+b_{\rm br}=1$. Then we combine terms from Eq. (B2) with W_A in the following way:

$$-(a_{\rm br}+b_{\rm br})\zeta\mathbb{E}[a_{ij}X_i]-(b_{\rm br}+c_{\rm br})\zeta\mathbb{E}[a_{ij}X_iX_j]=-\zeta\mathbb{E}[a_{ij}X_i(1-X_j)],\quad\text{if } b_{\rm br}+c_{\rm br}=-1.$$

Therefore, we propose the following changes to ensure that W_A is negative for the link-breaking coefficients ζ:

$$\begin{aligned}&\text{In Eq. (B2): }-(a_{\rm br}+b_{\rm br})\zeta\mathbb{E}[a_{ij}X_i]\to-\zeta\mathbf{1}_{\{a_{\rm br}=0,b_{\rm br}=1,c_{\rm br}=-1\}}\mathbb{E}[a_{ij}X_i]\\&\text{In } W_A:\quad-(b_{\rm br}+c_{\rm br})\zeta\mathbb{E}[a_{ij}X_iX_j]\to-\zeta(\mathbf{1}_{\{a_{\rm br}=0,b_{\rm br}=0,c_{\rm br}=1\}}+\mathbf{1}_{\{a_{\rm br}=1,b_{\rm br}=-1,c_{\rm br}=2\}})\mathbb{E}[a_{ij}X_iX_j]\\&\quad-\zeta(\mathbf{1}_{\{a_{\rm br}=1,b_{\rm br}=0,c_{\rm br}=-1\}}+\mathbf{1}_{\{a_{\rm br}=0,b_{\rm br}=1,c_{\rm br}=-2\}})\mathbb{E}[a_{ij}X_i(1-X_j)],\end{aligned}$$

where $\mathbf{1}_f$ is the indicator function, which is 1 if the link-breaking rule $f_{\rm br}$ satisfies f and is zero otherwise. We repeat the procedure for the link-creation term ξ, except that we apply an extra trick: we add zero to Eq. (B2), where $\varepsilon>0$ is small:

$$-\varepsilon\mathbb{E}[X_i(1-a_{ij})]+\varepsilon\mathbb{E}[X_i]-\varepsilon\mathbb{E}[a_{ij}X_i]=0,$$

such that

$$\begin{aligned}&\text{In Eq. (B2):}\qquad(a_{\rm cr}+b_{\rm cr})\xi\mathbb{E}[X_i]\to\{\xi(1-\mathbf{1}_{\{a_{\rm cr}=1,b_{\rm cr}=-1,c_{\rm cr}=1\}})+\varepsilon\}\mathbb{E}[X_i]\\&\text{In } W_A:(b_{\rm cr}+c_{\rm cr})\xi\mathbb{E}[(1-a_{ij})X_iX_j]\to-\xi(\mathbf{1}_{\{a_{\rm cr}=1,b_{\rm cr}=0,c_{\rm cr}=-1\}}+\mathbf{1}_{\{a_{\rm cr}=0,b_{\rm cr}=1,c_{\rm cr}=-2\}})\mathbb{E}[(1-a_{ij})X_iX_j]\\&\quad-\xi(\mathbf{1}_{\{a_{\rm cr}=0,b_{\rm cr}=0,c_{\rm cr}=1\}}+\mathbf{1}_{\{a_{\rm cr}=1,b_{\rm cr}=-1,c_{\rm cr}=2\}})\mathbb{E}[a_{ij}X_iX_j]-\xi(\mathbf{1}_{\{a_{\rm cr}=0,b_{\rm cr}=0,c_{\rm cr}=1\}}+\mathbf{1}_{\{a_{\rm cr}=1,b_{\rm cr}=-1,c_{\rm cr}=2\}})\mathbb{E}[X_i(1-X_j)]\\&\quad-\varepsilon\mathbb{E}[X_i(1-a_{ij})]\\&\text{In } W_A:\qquad0\cdot\mathbb{E}[a_{ij}X_i]\to-\varepsilon\mathbb{E}[a_{ij}X_i].\end{aligned}$$

The differential equations (B2) and (B3) can be written in matrix notation. Given a sequence of matrices $A_1,\ldots,A_n$, define $A=\oplus_{i=1}^{n}A_i$ to be the block diagonal matrix with A_i on its block diagonals; A_i does not necessarily have to be square. The matrix A can be visualized as

$$A=\begin{pmatrix}A_1&0&0&\ldots\\0&A_2&0&\ldots\\\vdots&\ddots&\ddots&\ddots\\\ldots&0&0&A_n\end{pmatrix}.$$

Define the vectors $q_i=\mathrm{col}_{j:a_{ij}(0)=1}(\mathbb{E}[a_{ij}X_i])$ and $q=\mathrm{col}_{1\leqslant i\leqslant N}(q_i)$. Moreover, we define T_i as the row vector satisfying

$$T_iq=\sum_{k:a_{ik}(0)=1}\mathbb{E}[a_{ki}X_k].$$

Here T_i is a Boolean row vector containing ones when an initial link is present between node i and node j (where j is the jth element of T_i) and zero otherwise. The dimension of T_i is therefore $1\times 2L_0$ where L_0 is the number of links in the initial network. Then define the matrix $T = \mathrm{col}_{1\leqslant i\leqslant N}(T_i)$. Also define the matrix $J = \oplus_{i=1}^{n}\mathbf{1}_{d_i}$ where d_i is the number of degrees of node i in the initial network. Finally, define the matrix $S = \mathrm{col}_{1\leqslant i\leqslant N}(\mathbf{1}_{d_i}\otimes T_i)$ where $\otimes$ is the Kronecker product. To summarize, the following parameters have been defined:

$$\begin{aligned} q_i &= \underset{j\,:\,a_{ij}(0)=1}{\mathrm{col}}(\mathbb{E}[a_{ij}X_i]),\\ q &= \underset{1\leqslant i\leqslant N}{\mathrm{col}}(q_i),\\ T_i q &= \sum_{k\,:\,a_{ik}(0)=1}\mathbb{E}[a_{ki}X_k],\\ T &= \underset{1\leqslant i\leqslant N}{\mathrm{col}}(T_i),\\ J &= \oplus_{i=1}^{n}\mathbf{1}_{d_i},\\ S &= \underset{1\leqslant i\leqslant N}{\mathrm{col}}(\mathbf{1}_{d_i}\otimes T_i). \end{aligned}$$

The differential equations (B2) and (B3) can be formulated as a system in the following way:

$$\frac{d}{dt}\begin{pmatrix}\mathbb{E}[X_i]\\ \mathbb{E}[a_{ij}X_i]\end{pmatrix} = M\begin{pmatrix}\mathbb{E}[X_i]\\ \mathbb{E}[a_{ij}X_i]\end{pmatrix} + \begin{pmatrix}W_X\\ W_A\end{pmatrix}, \tag{B4}$$

where

$$M = \begin{pmatrix} -\delta I & \beta T\\ \{\xi(1-\mathbf{1}_{\{a_{\mathrm{cr}}=1,b_{\mathrm{cr}}=-1,c_{\mathrm{cr}}=1\}})+\varepsilon\}J & \beta S-(\zeta\mathbf{1}_{\{a_{\mathrm{br}}=0,b_{\mathrm{br}}=1,c_{\mathrm{br}}=-1\}}+a_{\mathrm{cr}}\xi+b_{\mathrm{cr}}\xi+\delta+\varepsilon)I\end{pmatrix}. \tag{B5}$$

Since the remaining terms W_X and W_A are negative by construction, it follows that

$$\frac{d}{dt}\begin{pmatrix}\mathbb{E}[X_i]\\ \mathbb{E}[a_{ij}X_i]\end{pmatrix} \leqslant M\begin{pmatrix}\mathbb{E}[X_i]\\ \mathbb{E}[a_{ij}X_i]\end{pmatrix}. \tag{B6}$$

In case the eigenvalues of the matrix M are smaller than zero, the solution is bounded by an exponentially decaying function. This implies the solution dies out over sufficiently large time. The point where one of the eigenvalues becomes zero, changes the solution from an exponentially decaying function to an exponentially growing function. This bifurcation point is commonly known as the epidemic threshold. To derive a bound for the epidemic threshold, the eigenvalues of M are investigated. Specifically, the largest (real) eigenvalue is of interest and can be determined by using the Perron-Fröbenius theory.

Lemma B.1. Given a positive eigenvector $\boldsymbol{x}$ of M, its corresponding eigenvalue is the largest eigenvalue of M.

Proof. The initial network was taken to be connected. Since the network is undirected, it is also strongly connected. Ogura and Preciado (2016) proved that the matrix M is irreducible when the initial network is strongly connected [24, Appendix A]. Then, by Perron-Fröbenius theory for irreducible matrices, the statement follows [35, Theorem 8.4.4]. ■

Based on Lemma B.1, our approach is to construct a positive eigenvector for the matrix M. Using the positive eigenvector, a lower bound for the epidemic threshold is computed.

Proof of Theorem III.1. First a positive eigenvector is constructed for the matrix M. Since the initial network is strongly connected, there exists a positive eigenvector $\boldsymbol{v}$ corresponding to eigenvalue ρ (the spectral radius) [24]. We define the vector $\boldsymbol{w} = \mathrm{col}_{1\leqslant i\leqslant N}(v_i\mathbf{1}_{d_i})$. Using the definition of T_i, it follows that

$$T_i\boldsymbol{w} = \sum_{k\,:\,a_{ik}(0)=1} w_{ki} = \sum_{k\,:\,a_{ik}(0)=1} v_k = (Av)_i = \rho v_i. \tag{B7}$$

So $T\boldsymbol{w} = \rho\boldsymbol{v}$. Equivalently, it follows that $S\boldsymbol{w} = \rho\boldsymbol{w}$ and $J\boldsymbol{v} = \boldsymbol{w}$.

Define the vector $\boldsymbol{x} = \binom{z\boldsymbol{v}}{\boldsymbol{w}}$ where $z\in\mathbb{R}$, which is an eigenvector of M. Indeed,

$$\begin{aligned} M\begin{pmatrix}z\boldsymbol{v}\\ \boldsymbol{w}\end{pmatrix} &= \begin{pmatrix} -\delta I & \beta T\\ \{\xi(1-\mathbf{1}_{\{a_{\mathrm{cr}}=1,b_{\mathrm{cr}}=-1,c_{\mathrm{cr}}=1\}})+\varepsilon\}J & \beta S-(\mathbf{1}_{\{a_{\mathrm{br}}=0,b_{\mathrm{br}}=1,c_{\mathrm{br}}=-1\}}\zeta+a_{\mathrm{cr}}\xi+b_{\mathrm{cr}}\xi+\delta)I\end{pmatrix}\begin{pmatrix}z\boldsymbol{v}\\ \boldsymbol{w}\end{pmatrix}\\ &= \begin{pmatrix}(\beta\rho-z\delta)\boldsymbol{v}\\ \{\xi(1-\mathbf{1}_{\{a_{\mathrm{cr}}=1,b_{\mathrm{cr}}=-1,c_{\mathrm{cr}}=1\}})+\varepsilon\}z+\beta\rho-(\mathbf{1}_{\{a_{\mathrm{br}}=0,b_{\mathrm{br}}=1,c_{\mathrm{br}}=-1\}}\zeta+a_{\mathrm{cr}}\xi+b_{\mathrm{cr}}\xi+\delta))\boldsymbol{w}\end{pmatrix}\\ &= \lambda\begin{pmatrix}z\boldsymbol{v}\\ \boldsymbol{w}\end{pmatrix}, \end{aligned}$$

where the eigenvalue λ corresponds to the eigenvector $\boldsymbol{x}$. Since $\boldsymbol{v}$ and $\boldsymbol{w}$ are positive, the eigenvector $\boldsymbol{x}$ is positive if and only if $z > 0$. To conclude $z > 0$, a system of equations for z and λ is obtained:

$$z\lambda = \beta\rho - z\delta, \tag{B8a}$$

$$\lambda = \{\xi(1 - \mathbf{1}_{\{a_{\rm cr}=1,b_{\rm cr}=-1,c_{\rm cr}=1\}}) + \varepsilon\}z + \beta\rho - (\mathbf{1}_{\{a_{\rm br}=0,b_{\rm br}=1,c_{\rm br}=-1\}}\zeta + a_{\rm cr}\xi + b_{\rm cr}\xi + \delta + \varepsilon). \tag{B8b}$$

Define $X = \xi(1 - \mathbf{1}_{\{a_{\rm cr}=1,b_{\rm cr}=-1,c_{\rm cr}=1\}}) + \varepsilon$ and $Y = -\beta\rho + \mathbf{1}_{\{a_{\rm br}=0,b_{\rm br}=1,c_{\rm br}=-1\}}\zeta + (a_{\rm cr} + b_{\rm cr})\xi + \delta + \varepsilon$ and notice that $X > 0$. Then Eqs. (B8a) and (B8b) simplify to

$$z(\lambda + \delta) = \beta\rho, \tag{B9a}$$

$$\lambda = zX - Y. \tag{B9b}$$

Inserting Eqs. (B9a) into (B9b), we find a quadratic equation for z:

$$Xz^2 + (\delta - Y)z - \beta\rho = 0. \tag{B10}$$

Based on Eq. (B10), we find that $z_1 < 0, z_2 > 0$. The corresponding values for λ can be obtained using Eq. (B9a), which can be rewritten as

$$\lambda = \frac{\beta\rho}{z} - \delta.$$

Since $\beta, \delta, \rho > 0$, for $z_1 < 0$ it follows that $\lambda_1 < 0$. For $z_2 > 0$, the sign of λ cannot be determined. However, we require $z_2 > 0$ to have a positive eigenvector and we require $\lambda_2 < 0$ for stability. From the system given by Eqs. (B9a) and (B9b), the quadratic equation for λ can be derived:

$$\lambda^2 + (\delta + Y)\lambda + \underbrace{(\delta Y - \beta\rho X)}_{\text{constant term}} = 0.$$

We have concluded earlier that $\lambda_1 < 0$. The eigenvalues of M are required to be negative, hence $\lambda_2 < 0$. When λ_1, λ_2 are negative, the constant term of the quadratic equation is positive. This leads to the condition

$$\delta Y - \beta\rho X > 0.$$

Substitution of the definition of X and Y and rewriting yields

$$\frac{\beta}{\delta} < \frac{\mathbf{1}_{\{a_{\rm br}=0,b_{\rm br}=1,c_{\rm br}=-1\}}\zeta + a_{\rm cr}\xi + b_{\rm cr}\xi + \delta + \varepsilon}{\rho[\xi(1 - \mathbf{1}_{\{a_{\rm cr}=1,b_{\rm cr}=-1,c_{\rm cr}=1\}}) + \delta + \varepsilon]},$$

such that the final form becomes

$$\tau < \frac{1}{\rho}\left[1 + \frac{\mathbf{1}_{\{a_{\rm br}=0,b_{\rm br}=1,c_{\rm br}=-1\}}\omega - (\mathbf{1}_{\{a_{\rm cr}=1,b_{\rm cr}=0,c_{\rm cr}=-1\}\cup\{a_{\rm cr}=0,b_{\rm cr}=1,c_{\rm cr}=-2\}})}{(1 - \mathbf{1}_{\{a_{\rm cr}=1,b_{\rm cr}=-1,c_{\rm cr}=1\}}) + \delta/\xi + \varepsilon/\xi}\right]. \tag{B11}$$

Since we did not assume any value for ε, we take $\lim_{\varepsilon\to 0}$. Eq. (B11) is a required condition for the process to exponentially decay to zero over sufficiently large time. Therefore, the epidemic threshold τ_c needs to be larger than those τ-values, which proves Theorem III.1. ■

APPENDIX C: PROOF OF THEOREM III.2

We follow the method of Guo *et al.* [20]. Using Eq. (2) and the general formulation of any updating rule of the G-ASIS model in Eq. (4), we find

$$\frac{d\,\mathbb{E}[a_{ij}]}{dt} = a_{ij}(0)\mathbb{E}[-\zeta a_{ij}(a_{\rm br} + b_{\rm br}(X_i + X_j) + c_{\rm br}X_iX_j) + \xi(1 - a_{ij})(a_{\rm cr} + b_{\rm cr}(X_i + X_j) + c_{\rm cr}X_iX_j)].$$

By using that we have a complete initial network, i.e., $a_{ij}(0) = 1$ for all $i \neq j$, we obtain

$$\begin{aligned}\frac{d\,\mathbb{E}[a_{ij}]}{dt} = {} & a_{\rm cr}\xi + b_{\rm cr}\xi\mathbb{E}[X_i] + b_{\rm cr}\xi\mathbb{E}[X_j] - (a_{\rm br}\zeta + a_{\rm cr}\xi)\mathbb{E}[a_{ij}] + c_{\rm cr}\xi\mathbb{E}[X_iX_j] \\ & - (b_{\rm br}\zeta + b_{\rm cr}\xi)\mathbb{E}[a_{ij}X_i] - (b_{\rm br}\zeta + b_{\rm cr}\xi)\mathbb{E}[a_{ij}X_j] - (c_{\rm br}\zeta + c_{\rm cr}\xi)\mathbb{E}[a_{ij}X_iX_j].\end{aligned}$$

Taking the sum over all $j \neq i$ and using the degree $d_i = \sum_{j=1, j\neq i}^{N} a_{ij}$ and $a_{ii} = 0$, we obtain

$$\frac{d\,\mathbb{E}[d_i]}{dt} = a_{\mathrm{cr}}\xi(N-1) + b_{\mathrm{cr}}\xi(N-1)\mathbb{E}[X_i] + b_{\mathrm{cr}}\xi \sum_{j=1, j\neq i}^{N} \mathbb{E}[X_j] - (a_{\mathrm{br}}\zeta + a_{\mathrm{cr}}\xi)\mathbb{E}[d_i]$$
$$+ c_{\mathrm{cr}}\xi\,\mathbb{E}\left[X_i \sum_{j=1, j\neq i}^{N} X_j\right] - (b_{\mathrm{br}}\zeta + b_{\mathrm{cr}}\xi)\mathbb{E}[d_i X_i] - (b_{\mathrm{br}}\zeta + b_{\mathrm{cr}}\xi)\mathbb{E}\left[\sum_{j=1}^{N} a_{ij} X_j\right] - (c_{\mathrm{br}}\zeta + c_{\mathrm{cr}}\xi)\mathbb{E}\left[\sum_{j=1}^{N} a_{ij} X_i X_j\right].$$

Two terms need to be investigated in more detail. The following relations hold:

$$(N-1)\mathbb{E}[X_i] + \sum_{j=1, j\neq i}^{N} \mathbb{E}[X_j] = ((N-2))\mathbb{E}[X_i] + \sum_{j=1}^{N} \mathbb{E}[X_j],$$
$$\mathbb{E}\left[X_i \sum_{j=1, j\neq i}^{N} X_j\right] = \mathbb{E}\left[X_i\left(\sum_{j=1}^{N} X_j - X_i\right)\right] = \mathbb{E}\left[X_i \sum_{j=1}^{N} X_j\right] - \mathbb{E}[X_i],$$

where in the last equation, for the last equality, we used the Bernoulli property. Reinserting these yields

$$\frac{d\,\mathbb{E}[d_i]}{dt} = a_{\mathrm{cr}}\xi(N-1) + (b_{\mathrm{cr}}\xi(N-2) - c_{\mathrm{cr}}\xi)\mathbb{E}[X_i] + b_{\mathrm{cr}}\xi \sum_{j=1}^{N} \mathbb{E}[X_j] - (a_{\mathrm{br}}\zeta + a_{\mathrm{cr}}\xi)\mathbb{E}[d_i]$$
$$+ c_{\mathrm{cr}}\xi\,\mathbb{E}\left[X_i \sum_{j=1}^{N} X_j\right] - (b_{\mathrm{br}}\zeta + b_{\mathrm{cr}}\xi)\mathbb{E}[d_i X_i] - (b_{\mathrm{br}}\zeta + b_{\mathrm{cr}}\xi)\mathbb{E}\left[\sum_{j=1}^{N} a_{ij} X_j\right] - (c_{\mathrm{br}}\zeta + c_{\mathrm{cr}}\xi)\mathbb{E}\left[\sum_{j=1}^{N} a_{ij} X_i X_j\right].$$

Up to now only the network equations from Eq. (2) have been used. We intend to use the epidemic equations in Eq. (1) to remove the largest correlation term. Hence, we rewrite Eq. (1) as

$$\mathbb{E}\left[\sum_{j=1}^{N} a_{ij} X_i X_j\right] = -\frac{1}{\beta}\frac{d\,\mathbb{E}[X_i]}{dt} - \frac{1}{\tau}\mathbb{E}[X_i] + \mathbb{E}\left[\sum_{j=1}^{N} a_{ij} X_j\right],$$

where $\tau = \beta/\delta$ is the effective infection rate. Inserting this back into the previous result gives

$$\frac{d\,\mathbb{E}[d_i]}{dt} = a_{\mathrm{cr}}\xi(N-1) + (b_{\mathrm{cr}}\xi(N-2) - c_{\mathrm{cr}}\xi)\mathbb{E}[X_i] + b_{\mathrm{cr}}\xi \sum_{j=1}^{N} \mathbb{E}[X_j] - (a_{\mathrm{br}}\zeta + a_{\mathrm{cr}}\xi)\mathbb{E}[d_i]$$
$$+ c_{\mathrm{cr}}\xi\,\mathbb{E}\left[X_i \sum_{j=1}^{N} X_j\right] - (b_{\mathrm{br}}\zeta + b_{\mathrm{cr}}\xi)\mathbb{E}[d_i X_i] - (b_{\mathrm{br}}\zeta + b_{\mathrm{cr}}\xi)\mathbb{E}\left[\sum_{j=1}^{N} a_{ij} X_j\right]$$
$$- (c_{\mathrm{br}}\zeta + c_{\mathrm{cr}}\xi)\left\{-\frac{1}{\beta}\frac{d\,\mathbb{E}[X_i]}{dt} - \frac{1}{\tau}\mathbb{E}[X_i] + \mathbb{E}\left[\sum_{j=1}^{N} a_{ij} X_j\right]\right\}.$$

Taking all time-derivatives to the left and dividing every term by ζ, we obtain

$$\frac{d}{dt}\mathbb{E}\left[\frac{d_i}{\zeta} - \frac{c_{\mathrm{br}} + c_{\mathrm{cr}}\omega^{-1}}{\beta} X_i\right] = a_{\mathrm{cr}}\omega^{-1}(N-1) + \left[b_{\mathrm{cr}}\omega^{-1}(N-2) - c_{\mathrm{cr}}\omega^{-1} + \frac{c_{\mathrm{br}} + c_{\mathrm{cr}}\omega^{-1}}{\tau}\right]\mathbb{E}[X_i]$$
$$+ b_{\mathrm{cr}}\omega^{-1}\sum_{j=1}^{N} \mathbb{E}[X_j] - (a_{\mathrm{br}} + a_{\mathrm{cr}}\omega^{-1})\mathbb{E}[d_i] + c_{\mathrm{cr}}\omega^{-1}\mathbb{E}\left[X_i \sum_{j=1}^{N} X_j\right]$$
$$- (b_{\mathrm{br}} + b_{\mathrm{cr}}\omega^{-1})\mathbb{E}[d_i X_i] - (b_{\mathrm{br}} + b_{\mathrm{cr}}\omega^{-1} + c_{\mathrm{br}} + c_{\mathrm{cr}}\omega^{-1})\mathbb{E}\left[\sum_{j=1}^{N} a_{ij} X_j\right].$$

Using $2L = \sum_{i=1}^{N} d_i$ where L is the number of links, we sum over all $1 \leqslant i \leqslant N$ to find

$$\frac{d}{dt}\mathbb{E}\left[\frac{2L}{\zeta} - \frac{c_{\rm br} + c_{\rm cr}\omega^{-1}}{\beta}\sum_{i=1}^{N} X_i\right] = a_{\rm cr}\omega^{-1}N(N-1) + \left(b_{\rm cr}\omega^{-1}(N-2) - c_{\rm cr}\omega^{-1} + \frac{c_{\rm br} + c_{\rm cr}\omega^{-1}}{\tau}\right)\sum_{i=1}^{N}\mathbb{E}[X_i]$$
$$+ b_{\rm cr}\omega^{-1}N\sum_{j=1}^{N}\mathbb{E}[X_j] - (a_{\rm br} + a_{\rm cr}\omega^{-1})\mathbb{E}\left[\sum_{i=1}^{N} d_i\right] + c_{\rm cr}\omega^{-1}\mathbb{E}\left[\sum_{i=1}^{N} X_i\sum_{j=1}^{N} X_j\right]$$
$$- (b_{\rm br} + b_{\rm cr}\omega^{-1})\sum_{i=1}^{N}\mathbb{E}[d_i X_i] - (b_{\rm br} + b_{\rm cr}\omega^{-1} + c_{\rm br} + c_{\rm cr}\omega^{-1})\mathbb{E}\left[\sum_{j=1}^{N} d_j X_j\right].$$

Using the fraction of infected nodes $Z = \frac{1}{N}\sum_{i=1}^{N} X_i$, we can simplify this to

$$\frac{d}{dt}\mathbb{E}\left[\frac{2L}{\zeta} - \frac{c_{\rm br}N + c_{\rm cr}\omega^{-1}N}{\beta}Z\right] = a_{\rm cr}\omega^{-1}N(N-1) + \left(b_{\rm cr}\omega^{-1}N(N-2) - c_{\rm cr}N\omega^{-1} + \frac{c_{\rm br}N + c_{\rm cr}\omega^{-1}N}{\tau}\right)\mathbb{E}[Z]$$
$$+ b_{\rm cr}\omega^{-1}N^2\mathbb{E}[Z] - (a_{\rm br} + a_{\rm cr}\omega^{-1})\mathbb{E}\left[\sum_{i=1}^{N} d_i\right] + c_{\rm cr}\omega^{-1}N^2\mathbb{E}[Z^2]$$
$$- (b_{\rm br} + b_{\rm cr}\omega^{-1} + b_{\rm br} + b_{\rm cr}\omega^{-1} + c_{\rm br} + c_{\rm cr}\omega^{-1})\mathbb{E}\left[\sum_{j=1}^{N} d_j X_j\right].$$

When the derivative on the left-hand side vanishes (in the metastable state, which we denote by as asterisk $*$) we have

$$a_{\rm cr}\omega^{-1}N(N-1) + \left[b_{\rm cr}\omega^{-1}N(N-1) - b_{\rm cr}N\omega^{-1} + b_{\rm cr}\omega^{-1}N^2 - c_{\rm cr}N\omega^{-1} + \frac{c_{\rm br}N + c_{\rm cr}\omega^{-1}N}{\tau}\right]\mathbb{E}[Z^*]$$
$$- (a_{\rm br} + a_{\rm cr}\omega^{-1})\mathbb{E}\left[\sum_{i=1}^{N} d_i^*\right] + c_{\rm cr}\omega^{-1}N^2\mathbb{E}[(Z^*)^2] - (2b_{\rm br} + 2b_{\rm cr}\omega^{-1} + c_{\rm br} + c_{\rm cr}\omega^{-1})\mathbb{E}\left[\sum_{j=1}^{N} d_j^* X_j^*\right] = 0.$$

Using $\mathrm{Var}[Z^*] = \mathbb{E}[(Z^*)^2] - \mathbb{E}[Z^*]^2$ and the prevalence $y = \mathbb{E}[Z^*]$, we finally find

$$c_{\rm cr}\omega^{-1}N^2y^2 + \left[2b_{\rm cr}\omega^{-1}N(N-1) - c_{\rm cr}N\omega^{-1} + \frac{c_{\rm br}N + c_{\rm cr}\omega^{-1}N}{\tau}\right]y + a_{\rm cr}\omega^{-1}N(N-1) - (a_{\rm br} + a_{\rm cr}\omega^{-1})\mathbb{E}\left[\sum_{i=1}^{N} d_i^*\right]$$
$$+ c_{\rm cr}\omega^{-1}N^2\mathrm{Var}[Z^*] - (2b_{\rm br} + 2b_{\rm cr}\omega^{-1} + c_{\rm br} + c_{\rm cr}\omega^{-1})\mathbb{E}\left[\sum_{i=1}^{N} d_i^* X_i^*\right] = 0,$$

which is a quadratic equation in y. Since $c_{\rm cr}$ is never zero, every term can be multiplied by $\frac{\omega}{c_{\rm cr}N^2}$, which proves Theorem III.2. ■

APPENDIX D: PROOF OF THEOREM III.3

The quadratic equation for the prevalence y from Eq. (6) can be rewritten in more compact form by defining

$$V = -\left(\frac{2b_{\rm cr}N\tau - (2b_{\rm cr} + c_{\rm cr})\tau + c_{\rm br}\omega + c_{\rm cr}}{2c_{\rm cr}N\tau}\right), \tag{D1}$$

$$H = \left\{\frac{(N-1)a_{\rm cr}}{c_{\rm cr}N} - \frac{a_{\rm br}\omega + a_{\rm cr}}{c_{\rm cr}N^2}\mathbb{E}\left[\sum_{i=1}^{N} d_i^*\right] + \mathrm{Var}[Z^*] - \frac{(2b_{\rm br} + c_{\rm br})\omega + 2b_{\rm cr} + c_{\rm cr}}{c_{\rm cr}N^2}\mathbb{E}\left[\sum_{i=1}^{N} d_i^* X_i^*\right]\right\}, \tag{D2}$$

such that Eq. (6) can be written as

$$y^2 - 2Vy + H = 0. \tag{D3}$$

The two possible solutions are

$$y = V \pm \sqrt{V^2 - H}. \tag{D4}$$

The quadratic equation (D3) for the prevalence y can be rewritten as

$$V = \frac{1}{2}\left(\frac{H}{y} + y\right). \tag{D5}$$

Using the definition of V from Eq. (D1), Eq. (D5) can be rewritten as

$$-\frac{b_{\rm cr}}{c_{\rm cr}} + \frac{(2b_{\rm cr} + c_{\rm cr})}{2c_{\rm cr}N} - \frac{c_{\rm br}\omega + c_{\rm cr}}{2c_{\rm cr}N\tau} = \frac{1}{2}\left(\frac{H}{y} + y\right),$$

which can be rearranged to

$$\tau = \frac{c_{\rm br}\omega + c_{\rm cr}}{2c_{\rm cr}N\left[-\frac{b_{\rm cr}}{c_{\rm cr}} + \frac{(2b_{\rm cr}+c_{\rm cr})}{2c_{\rm cr}N} - \frac{1}{2}\left(\frac{H}{y} + y\right)\right]}. \tag{D6}$$

Taking the limit $y \to 0$ [36], we find an implicit relationship for the epidemic threshold;

$$\tau_c = \frac{\frac{c_{\rm br}}{c_{\rm cr}}\omega + 1}{2\frac{b_{\rm cr}}{c_{\rm cr}}(1-N) + 1 - N\lim_{y\downarrow 0}\frac{H}{y}}. \tag{D7}$$

Since $c_{\rm br}, c_{\rm cr} \neq 0$, Eq. (D7) is an explicit relation between the epidemic threshold τ_c and the effective link-breaking rate ω. The function H defined in Eq. (D2) depends on ω, ξ and τ and $H = 0$ zero if $y = 0$. Since we have taken $\lim_{y\downarrow 0}$, we have $H(\omega, \tau_c, \xi)$. This makes Eq. (D7) an implicit relation for the epidemic threshold τ_c. Our main effort will be to show the dependence of the epidemic threshold τ_c on the effective link-breaking rate ω by bounding $H(\omega, \tau_c, \xi)$. Due to the continuity of H, we may define

$$\lim_{y\downarrow 0}\frac{H}{y} = h(\omega, \xi), \tag{D8}$$

such that the epidemic threshold τ_c becomes

$$\tau_c = \frac{\frac{c_{\rm br}}{c_{\rm cr}}\omega + 1}{2\frac{b_{\rm cr}}{c_{\rm cr}}(1-N) + 1 - Nh(\omega, \xi)}, \tag{D9}$$

which proves the first part of Theorem III.3. Next the function $h(\omega, \xi)$ must be bounded. In Ref. [29, Lemma S1.1], it is shown that all but the first term of H, given in Eq. (D2), are positive and the first term is nonnegative whenever $\frac{a_{\rm cr}}{c_{\rm cr}} \geqslant 0$. Therefore, the proof of Theorem III.3 is split up into two parts. The first part is covered by Lemma D.1.

Lemma D.1. Let τ_c be the epidemic threshold from Eq. (D9) and assume $H \geqslant 0$. Then τ_c is bounded by a linear function in ω or by a constant.

Proof. The only instances of G-ASIS which do not satisfy $H \geqslant 0$, are instances satisfying $\frac{a_{\rm cr}}{c_{\rm cr}} < 0$ and correspond to the link-creation rule $f_{\rm cr} = 1 - X_i X_j$. These instances are not included in this lemma, but are taken care of by Lemma D.2. This means 30 out of 36 instances of G-ASIS are treated in this lemma. We follow the approach of Ref. [21].

Step 1. The prevalence y is real.

The solutions of the quadratic equation (D3) for the prevalence y need to have a positive discriminant to be real solutions. From Eq. (D4), it is required that $H \leqslant V^2$. Since $H \geqslant 0$, it is sufficient to show that $\sqrt{H} \leqslant V$. Inserting the definition of V from Eq. (D1) brings

$$-\sqrt{H} \geqslant \frac{2b_{\rm cr}N\tau - (2b_{\rm cr} + c_{\rm cr})\tau + c_{\rm br}\omega + c_{\rm cr}}{2c_{\rm cr}N\tau},$$

which can be rearranged as

$$\frac{c_{\rm br}\omega + c_{\rm cr}}{2c_{\rm cr}\tau N} \leqslant \frac{2\frac{b_{\rm cr}}{c_{\rm cr}} + 1}{2N} - \frac{b_{\rm cr}}{c_{\rm cr}} - \sqrt{H}. \tag{D10}$$

In the metastable state, the right-hand side of Eq. (D10) is positive, such that

$$\tau \geqslant \frac{\frac{c_{\rm br}}{c_{\rm cr}}\omega + 1}{2\frac{b_{\rm cr}}{c_{\rm cr}}(1-N) + 1 - 2N\sqrt{H}}.$$

This holds for the metastable state, i.e., for all $\tau \geqslant \tau_c$. Hence,

$$\frac{\frac{c_{\rm br}}{c_{\rm cr}}\omega + 1}{2\frac{b_{\rm cr}}{c_{\rm cr}}(1-N) + 1 - Nh(\omega,\xi)} = \tau_c \geqslant \tau^* = \frac{\frac{c_{\rm br}}{c_{\rm cr}}\omega + 1}{2\frac{b_{\rm cr}}{c_{\rm cr}}(1-N) + 1 - 2N\sqrt{H}}. \tag{D11}$$

We conclude that

$$0 \leqslant 2\sqrt{H} \leqslant h(\omega,\xi).$$

Furthermore, since τ_c is bounded for $\frac{c_{\rm br}}{c_{\rm cr}}\omega + 1 > 0$, the denominator of Eq. (D9) should be nonzero. In other words,

$$0 \leqslant 2\sqrt{H} \leqslant h(\omega,\xi) < \frac{b_{\rm cr}}{c_{\rm cr}}\frac{1-N}{N} + \frac{1}{2N} \quad \text{for } \frac{c_{\rm br}}{c_{\rm cr}}\omega + 1 > 0. \tag{D12}$$

In Eq. (D12), the function $h(\omega,\xi)$ is bounded for some ω values, but not all. The remaining ω values are taken care of by Step 2.

Step 2. Bounding $h(\omega,\xi)$ for the other ω values.

Step 2A. Case $\frac{c_{\rm br}}{c_{\rm cr}} < 0$. (ASIS, AID)

Out of the 30 instances considered in this lemma, 15 are part of this case.

For the limit of $\omega \uparrow -\frac{c_{\rm cr}}{c_{\rm br}}$, the epidemic threshold τ_c given in Eq. (D9) should still be nonnegative, or at least not suddenly become zero. This can only be assured when the denominator in Eq. (D9) becomes zero as well. This continuity argument shows that equality holds for Eq. (D12), which is

$$\lim_{\omega\uparrow -\frac{c_{\rm cr}}{c_{\rm br}}} h(\omega,\xi) = \frac{b_{\rm cr}}{c_{\rm cr}}\frac{1-N}{N} + \frac{1}{2N}.$$

For $\omega > -\frac{c_{\rm cr}}{c_{\rm br}}$, the epidemic threshold τ_c in Eq. (D9) should be positive as well, so using Eq. (D11) one finds

$$h(\omega,\xi) > \frac{b_{\rm cr}}{c_{\rm cr}}\frac{1-N}{N} + \frac{1}{2N} \quad \text{for } \omega > -\frac{c_{\rm cr}}{c_{\rm br}}.$$

We now consider the situation where the effective link-breaking rate ω increases up to infinity. Suppose a node i is infected. The link between node i and its neighbors j is removed (as ω is high) when the link-breaking rule allows for that. The link can be recreated only when (I) the link-creation rule $f_{\rm cr}$ creates the link between node i and j when either i or j is infected [these updating rules are $f_{\rm cr} = 1 - X_iX_j$, $f_{\rm cr} = 1 - (1-X_i)(1-X_j)$ and $f_{\rm cr} = (X_i - X_j)^2$] and (II) the link-breaking rule $f_{\rm br}$ does not break the link between susceptible and infected nodes [these updating rules are $f_{\rm br} = X_iX_j$, $f_{\rm br} = (1-X_i)(1-X_j)$ and $f_{\rm br} = 1 - (X_i - X_j)^2$]. Only when (I) and (II) are satisfied, spreading in the network continues despite the link-breaking rate ω increasing up to infinity. This allows for a split-up into two classes: Class A and B.

(Class A) (AID). The epidemic threshold remains constant.

The only eligible instances for this class have been listed above. Some of these are still invalid, because they do not obey $H \geqslant 0$ (e.g., the link-creation rule $f_{\rm cr} = 1 - X_iX_j$) or do not obey $\frac{c_{\rm br}}{c_{\rm cr}} \geqslant 0$ (which is Step 2B). These constraints yield six instances having any combination of the following link-breaking rules: $f_{\rm br} = X_iX_j$, $f_{\rm br} = (1-X_i)(1-X_j)$ and $f_{\rm br} = 1 - (X_i - X_j)^2$ and for the link-creation rules: $f_{\rm cr} = 1 - (1-X_i)(1-X_j)$ and $f_{\rm cr} = (X_i - X_j)^2$. These instances have in common that, while increasing ω, the epidemic threshold τ_c barely increases. In other words, the limit of $\omega \to \infty$ of τ_c is finite. So define

$$\lim_{\omega\to\infty} \tau_c(\omega,\xi) = C_1 > 0.$$

We continue to prove that $h(\omega,\xi)$ is linear in ω for large ω. The epidemic threshold can be rewritten in terms of h:

$$h(\omega,\xi) = 2\frac{b_{\rm cr}}{c_{\rm cr}}\frac{1-N}{N} + \frac{1}{N} - \frac{\frac{c_{\rm br}}{c_{\rm cr}}\omega + 1}{N\tau_c(\omega,\xi)}. \tag{D13}$$

Then we may compute the following:

$$\frac{1}{NC_1} = \frac{1}{N\lim_{\omega\to\infty}\tau_c(\omega,\xi)} = \lim_{\omega\to\infty}\frac{1}{N\tau_c(\omega,\xi)} \overset{\text{def.}}{=} \lim_{\omega\to\infty}\frac{-h(\omega,\xi) + \frac{1}{N} + 2\frac{b_{\rm cr}}{c_{\rm cr}}\frac{1-N}{N}}{\frac{c_{\rm br}}{c_{\rm cr}}\omega + 1}$$
$$\overset{\text{l'hôpital}}{=} -\frac{c_{\rm cr}}{c_{\rm br}}\left.\frac{\partial h}{\partial\omega}\right|_{\omega\to\infty} = C_2.$$

Since $C_1 > 0$, we conclude $C_2 > 0$. Hence, $h(\omega,\xi)$ is a linear function in ω for all instances in Class A.

(Class B) (ASIS). The epidemic threshold scales linearly in ω.

The remaining $15 - 6 = 9$ instances not belonging to Class A are part of this class. For each instance in this class, the link-breaking rule is dominant in the sense that spreading between susceptible and infected nodes cannot take place (for $\omega \to \infty$

and fixed τ, ξ) because the link between susceptible and infected nodes is removed immediately. Hence, the epidemic threshold τ_c must increase along ω to keep spreading the disease (in the limit of $\omega \to \infty$). This proves that the epidemic threshold scales linearly in ω.

Step 2B. Case $\frac{c_{\rm br}}{c_{\rm cr}} \geqslant 0$.

(Class C) (ABN). The remaining $30 - 15 = 15$ instances of G-ASIS follow this constraint. Table I shows that the ratio $\frac{c_{\rm br}}{c_{\rm cr}}$ is strictly positive. Therefore the relation in Eq. (D12) holds for all $\omega \geqslant 0$. So $h(\omega, \xi)$ is strictly bounded by a constant, also in the limit of $\omega \to \infty$. Then the epidemic threshold τ_c scales linearly in ω, which proves the lemma. ■

All instances of G-ASIS have been classified except for instances having the link-creation rule $f_{\rm cr} = 1 - X_i X_j$. These instances are covered by the next lemma.

Lemma D.2. Let τ_c be the epidemic threshold from Eq. (D9) and assume $H < 0$. Then $h(\omega, \xi)$ is bounded by a linear function in ω or by a constant.

Proof. The only instances of G-ASIS satisfying $H < 0$ are instances which have link-creation rule $f_{\rm cr} = 1 - X_i X_j$. Therefore we substitute $a_{\rm cr} = 1, b_{\rm cr} = 0, c_{\rm cr} = -1$ in Eq. (D2) to find

$$H = \frac{1}{N} - 1 + \frac{a_{\rm br}\omega + 1}{N^2}\mathbb{E}\left[\sum_{i=1}^{N} d_i^*\right] + \mathrm{Var}[Z^*] + \frac{(2b_{\rm br} + c_{\rm br})\omega - 1}{N^2}\mathbb{E}\left[\sum_{i=1}^{N} d_i^* X_i^*\right].$$

We have derived that $y = V \pm \sqrt{V^2 - H}$. Since $H < 0$, the prevalence y has two solutions: $y_1 > 0$ and $y_2 < 0$. Our focus lies on the physical solution y_1.

Step 1. The prevalence y_1 is bounded by 1.

This provides the constraint

$$V + \sqrt{V^2 - H} \leqslant 1. \tag{D14}$$

The sign of V is not determined. The equation above can be rewritten, so

$$V^2 - H \leqslant 1 - 2V + V^2.$$

Removing V^2 and inserting the definition for V from Eq. (D1), we find

$$-H \leqslant 1 - 2\left(\frac{1}{2N} + \frac{c_{\rm br}\omega - 1}{2N\tau}\right),$$

where the values of $a_{\rm cr}$, $b_{\rm cr}$ and $c_{\rm cr}$ have been substituted already. The last equation can be rewritten as

$$\frac{1 - c_{\rm br}\omega}{1 - Nh(\omega, \xi)} = \tau_c \geqslant \tau^* = \frac{1 - c_{\rm br}\omega}{1 - N - NH}. \tag{D15}$$

The denominator is positive, so this constraint is confining for $c_{\rm br} < 0$ and for ($c_{\rm br} > 0$ and $\omega < \frac{1}{c_{\rm br}}$). In either case, we conclude that

$$0 \leqslant 1 + H \leqslant h(\omega, \xi) < \frac{1}{N}. \tag{D16}$$

implying that $h(\omega, \xi)$ is always positive.

Step 2. Bounding $h(\omega, \xi)$ for all other ω-values. This step is analogous to step 2 from Lemma D.1.

Step 2A. Case $c_{\rm br} > 0$. Since τ_c is positive for $\omega < \frac{1}{c_{\rm br}}$, by taking $\lim_{\omega \uparrow \frac{1}{c_{\rm br}}}$, the limit must be finite and nonzero. This implies

$$\lim_{\omega \uparrow \frac{1}{c_{\rm br}}} h(\omega, \xi) = \frac{1}{N}.$$

For the relation in Eq. (D15) to be meaningful (τ_c should be nonnegative) for $\omega > \frac{1}{c_{\rm br}}$, it is required that

$$h(\omega, \xi) > \frac{1}{N}, \quad \text{for } \omega > \frac{1}{c_{\rm br}}.$$

It remains to analyze the behavior of $h(\omega, \xi)$ when the effective link-breaking rate ω approaches infinity. For increasing ω and fixed τ, the link-breaking process occurs almost immediately. Nodes become isolated and cure without having any links. There is, however, another possibility. The link-creation mechanism is here $f_{\rm cr} = 1 - X_i X_j$, which implies a link is created between a susceptible and an infected node. In case the link-breaking process does not include the updating rule where the link is broken between a susceptible and an infected node, the spreading continues despite $\omega \to \infty$. There are two possibilities.

(Class D). The epidemic threshold remains constant.

The link-creation process is $f_{\rm cr} = 1 - X_i X_j$ and as link-breaking rule, we require either $f_{\rm br} = X_i X_j$, $f_{\rm br} = (1 - X_i)(1 - X_j)$ or $f_{\rm br} = 1 - (X_i - X_j)^2$. These updating rules have in common that, whilst increasing ω, the epidemic threshold barely increases. In other words, the following limit holds:

$$\lim_{\omega \to \infty} \tau_c(\omega, \xi) = C_1 > 0.$$

Then $h(\omega, \xi)$ scales linearly with ω in a way analogous to Lemma D.1, Step 2A, Class A.

(Class E). The epidemic threshold is constant.

No updating rules belong to this class, as the only eligible do not comply with $c_{\mathrm{br}} > 0$.

Step 2B. Case $c_{\mathrm{br}} < 0$.

(Class F). In this case the relationship Eq. (D16) holds for all ω. So $h(\omega, \xi)$ is bounded by a constant for all ω. This implies τ_c scales linearly in ω, which proves the lemma. ■

Combining the result of Lemma D.1 and D.2 proves Theorem III.3. ■

APPENDIX E: ALL INSTANCES OF G-ASIS

All instances of G-ASIS and their properties are shown in Table III.

TABLE III. All instances from the G-ASIS model and their properties. The existence of the metastable state is merely a conjecture. The table assumes $\delta = 1$.

Updating rules		Model name and appearance in in literature	Metastable state always exists (conjecture)	Lower bound on epidemic threshold τ_c	Upper bound on epidemic threshold τ_c
link-breaking	link-creation				
X_iX_j	X_iX_j		Yes	$\frac{1}{\rho}$	Linear
X_iX_j	$1 - X_iX_j$		No	$\frac{1}{\rho}\left(\frac{1}{1+\xi}\right)$	Constant
X_iX_j	$(1 - X_i)(1 - X_j)$	ABN model	Yes	$\frac{1}{\rho}$	Linear
X_iX_j	$1 - (1 - X_i)(1 - X_j)$		No	$\frac{1}{\rho}$	Constant
X_iX_j	$(X_i - X_j)^2$		No	$\frac{1}{\rho}\left(\frac{1}{1+\xi}\right)$	Constant
X_iX_j	$1 - (X_i - X_j)^2$		Yes	$\frac{1}{\rho}$	Linear
$1 - X_iX_j$	X_iX_j		Yes	$\frac{1}{\rho}$	Linear
$1 - X_iX_j$	$1 - X_iX_j$		Yes	$\frac{1}{\rho}\left(\frac{1}{1+\xi}\right)$	Linear
$1 - X_iX_j$	$(1 - X_i)(1 - X_j)$		Yes	$\frac{1}{\rho}$	Linear
$1 - X_iX_j$	$1 - (1 - X_i)(1 - X_j)$		Yes	$\frac{1}{\rho}$	Linear
$1 - X_iX_j$	$(X_i - X_j)^2$	AFND model [31]	Yes	$\frac{1}{\rho}\left(\frac{1}{1+\xi}\right)$	Linear
$1 - X_iX_j$	$1 - (X_i - X_j)^2$		Yes	$\frac{1}{\rho}$	Linear
$(1 - X_i)(1 - X_j)$	X_iX_j	SCM model	Yes	$\frac{1}{\rho}$	Linear
$(1 - X_i)(1 - X_j)$	$1 - X_iX_j$		No	$\frac{1}{\rho}\left(\frac{1}{1+\xi}\right)$	Constant
$(1 - X_i)(1 - X_j)$	$(1 - X_i)(1 - X_j)$		Yes	$\frac{1}{\rho}$	Linear
$(1 - X_i)(1 - X_j)$	$1 - (1 - X_i)(1 - X_j)$		No	$\frac{1}{\rho}$	Constant
$(1 - X_i)(1 - X_j)$	$(X_i - X_j)^2$	AID model [21]	No	$\frac{1}{\rho}\left(\frac{1}{1+\xi}\right)$	Constant
$(1 - X_i)(1 - X_j)$	$1 - (X_i - X_j)^2$		Yes	$\frac{1}{\rho}$	Linear

TABLE III. (*Continued.*)

Updating rules		Model name and appearance in in literature	Metastable state always exists (conjecture)	Lower bound on epidemic threshold τ_c	Upper bound on epidemic threshold τ_c
link-breaking	link-creation				
$1-(1-X_i)(1-X_j)$	X_iX_j		Yes	$\frac{1}{\rho}\left(1+\frac{\omega}{1+1/\xi}\right)$	Linear
$1-(1-X_i)(1-X_j)$	$1-X_iX_j$		Yes	$\frac{1}{\rho}\left(1+\frac{\omega-1}{1+1/\xi}\right)$	Linear
$1-(1-X_i)(1-X_j)$	$(1-X_i)(1-X_j)$	ACSIS model [31]	Yes	$\frac{1}{\rho}(1+\omega\xi)$	Linear
$1-(1-X_i)(1-X_j)$	$1-(1-X_i)(1-X_j)$		Yes	$\frac{1}{\rho}\left(1+\frac{\omega}{1+1/\xi}\right)$	Linear
$1-(1-X_i)(1-X_j)$	$(X_i-X_j)^2$		Yes	$\frac{1}{\rho}\left(1+\frac{\omega-1}{1+1/\xi}\right)$	Linear
$1-(1-X_i)(1-X_j)$	$1-(X_i-X_j)^2$		Yes	$\frac{1}{\rho}\left(1+\frac{\omega}{1+1/\xi}\right)$	Linear
$(X_i-X_j)^2$	X_iX_j		Yes	$\frac{1}{\rho}$	Linear
$(X_i-X_j)^2$	$1-X_iX_j$		Yes	$\frac{1}{\rho}\left(\frac{1}{1+\xi}\right)$	Linear
$(X_i-X_j)^2$	$(1-X_i)(1-X_j)$	ASIS model [4,20,21]	Yes	$\frac{1}{\rho}$	Linear
$(X_i-X_j)^2$	$1-(1-X_i)(1-X_j)$		Yes	$\frac{1}{\rho}$	Linear
$(X_i-X_j)^2$	$(X_i-X_j)^2$		Yes	$\frac{1}{\rho}\left(\frac{1}{1+\xi}\right)$	Linear
$(X_i-X_j)^2$	$1-(X_i-X_j)^2$		Yes	$\frac{1}{\rho}$	Linear
$1-(X_i-X_j)^2$	X_iX_j		Yes	$\frac{1}{\rho}$	Linear
$1-(X_i-X_j)^2$	$1-X_iX_j$		No	$\frac{1}{\rho}\left(\frac{1}{1+\xi}\right)$	Constant
$1-(X_i-X_j)^2$	$(1-X_i)(1-X_j)$		Yes	$\frac{1}{\rho}$	Linear
$1-(X_i-X_j)^2$	$1-(1-X_i)(1-X_j)$		No	$\frac{1}{\rho}$	Constant
$1-(X_i-X_j)^2$	$(X_i-X_j)^2$		No	$\frac{1}{\rho}\left(\frac{1}{1+\xi}\right)$	Constant
$1-(X_i-X_j)^2$	$1-(X_i-X_j)^2$		Yes	$\frac{1}{\rho}$	Linear

[1] R. Pastor-Satorras, C. Castellano, P. Van Mieghem, and A. Vespignani, Epidemic processes in complex networks, Rev. Mod. Phys. **87**, 925 (2015).

[2] P. Van Mieghem, The N-intertwined SIS epidemic network model, Computing **93**, 147 (2011).

[3] F. D. Sahneh, A. Vajdi, J. Melander, and C. M. Scoglio, Contact adaption during epidemics: A multilayer network formulation approach, IEEE Trans. Netw. Sci. Eng. **6**, 16 (2019).

[4] I. Tunc, M. S. Shkarayev, and L. B. Shaw, Epidemics in adaptive social networks with temporary link deactivation, J. Stat. Phys. **151**, 355 (2013).

[5] R. Pastor-Satorras and A. Vespignani, Epidemic Spreading in Scale-Free Networks, Phys. Rev. Lett. **86**, 3200 (2001).

[6] J. Meier, X. Zhou, A. Hillebrand, P. Tewarie, C. J. Stam, and P. Van Mieghem, The epidemic spreading model and the direction of information flow in brain networks, NeuroImage **152**, 639 (2017).

[7] F. Jin, E. Dougherty, P. Saraf, Y. Cao, and N. Ramakrishnan, Epidemiological modeling of news and rumors on Twitter, in *Proceedings of the 7th Workshop on Social Network Mining and Analysis*, SNAKDD 13 (Association for Computing Machinery, New York, NY, 2013).

[8] I. Seoane, E. Calle, J. A. Hernández, J. Segovia, R. Romeral, P. Vilà, M. Urueña, and M. Manzano, Failure propagation in GMPLS optical rings: CTMC model and performance analysis, Optical Switch. Netw. **9**, 39 (2012).

[9] A. Vespignani, Modelling dynamical processes in complex socio-technical systems, Nat. Phys. **8**, 32 (2011).
[10] T. Gross and B. Blasius, Adaptive coevolutionary networks: A review, J. R. Soc. Interface **5**, 259 (2008).
[11] F. G. Hillary and J. H. Grafman, Injured brains and adaptive networks: The benefits and costs of hyperconnectivity, Trends Cognit. Sci. **21**, 385 (2017).
[12] I. J. Benczik, S. Z. Benczik, B. Schmittmann, and R. K. P. Zia, Opinion dynamics on an adaptive random network, Phys. Rev. E **79**, 046104 (2009).
[13] T. Gross, C. J. Dommar D'Lima, and B. Blasius, Epidemic Dynamics on an Adaptive Network, Phys. Rev. Lett. **96**, 208701 (2006).
[14] World Health Organisation, Nonpharmaceutical interventions for pandemic influenza, international measures, Emerg. Infect. Dis. **12**, 81 (2006).
[15] V. Marceau, P. A. Noël, L. Hébert-Dufresne, A. Allard, and L. J. Dubé, Adaptive networks: Coevolution of disease and topology, Phys. Rev. E **82**, 036116 (2010).
[16] S. Van Segbroeck, F. C. Santos, and J. M. Pacheco, Adaptive contact networks change effective disease infectiousness and dynamics, PLoS Comput. Biol. **6**, 1 (2010).
[17] S. Risau-Gusman and D. H. Zanette, Contact switching as a control strategy for epidemic outbreaks, J. Theor. Biol. **257**, 52 (2009).
[18] C. Lagorio, M. Dickison, F. Vazquez, L. A. Braunstein, P. A. Macri, M. V. Migueles, S. Havlin, and H. E. Stanley, Quarantine-generated phase transition in epidemic spreading, Phys. Rev. E **83**, 026102 (2011).
[19] G. Demirel, E. Barter, and T. Gross, Dynamics of epidemic diseases on a growing adaptive network, Sci. Rep. **7**, 42352 (2017).
[20] D. Guo, S. Trajanovski, R. van de Bovenkamp, H. Wang, and P. Van Mieghem, Epidemic threshold and topological structure of susceptible-infectious-susceptible epidemics in adaptive networks, Phys. Rev. E **88**, 042802 (2013).
[21] S. Trajanovski, D. Guo, and P. Van Mieghem, From epidemics to information propagation: Striking differences in structurally similar adaptive network models, Phys. Rev. E **92**, 030801(R) (2015).
[22] The number of updating rules can also be derived by regarding power sets. For any updating rule f for the link a_{ij}, we write the symmetric nodal input as $\{X_i, X_j\}$. All possible symmetric input combinations for the rule f are $\mathcal{X} = \{\{0,0\},\{0,1\},\{1,1\}\}$. Each element in $\mathcal{X}$ can be zero or one, depending on whether the link can be changed. The total number of combinations is then given by the power set of $\mathcal{X}$, denoted as $2^{\mathcal{X}}$, which contains $2^3 = 8$ elements. Two elements from $2^{\mathcal{X}}$ correspond to the trivial rules $f = 0$ and $f = 1$. After removal of the trivial rules, we find six updating rules for each link-updating mechanism.
[23] P. Van Mieghem, Approximate formula and bounds for the time-varying susceptible-infected-susceptible prevalence in networks, Phys. Rev. E **93**, 052312 (2016).
[24] M. Ogura and V. M. Preciado, Epidemic processes over adaptive state-dependent networks, Phys. Rev. E **93**, 062316 (2016).
[25] P. Van Mieghem, *Performance Analysis of Complex Networks and Systems* (Cambridge University Press, Cambridge, UK, 2014).
[26] P. Van Mieghem and R. van de Bovenkamp, Non-Markovian Infection Spread Dramatically Alters the Susceptible-Infected-Susceptible Epidemic Threshold in Networks, Phys. Rev. Lett. **110**, 108701 (2013).
[27] P. Van Mieghem, J. Omic, and R. Kooij, Virus spread in networks, IEEE/ACM Trans. Netw. **17**, 1 (2009).
[28] E. Cator and P. Van Mieghem, Susceptible-infected-susceptible epidemics on the complete graph and the star graph: Exact analysis, Phys. Rev. E **87**, 012811 (2013).
[29] See Supplemental Material at http://link.aps.org/supplemental/10.1103/PhysRevE.101.052302 for the sketch of the proof of Conjecture III.1.
[30] P. Van Mieghem and E. Cator, Epidemics in networks with nodal self-infection and the epidemic threshold, Phys. Rev. E **86**, 016116 (2012).
[31] M. A. Achterberg, Modelling Markovian epidemic and information diffusion over adaptive networks, MSc. Thesis, Delft, University of Technology, 2019.
[32] C. Li, R. van de Bovenkamp, and P. Van Mieghem, Susceptible-infected-susceptible model: A comparison of n-intertwined and heterogeneous mean-field approximations, Phys. Rev. E **86**, 026116 (2012).
[33] E. Cator and P. Van Mieghem, Nodal infection in markovian susceptible-infected-susceptible and susceptible-infected-removed epidemics on networks are non-negatively correlated, Phys. Rev. E **89**, 052802 (2014).
[34] I. Iacopini, G. Petri, A. Barrat, and V. Latora, Simplicial models of social contagion, Nat. Commun. **10**, 2485 (2019).
[35] R. Horn and C. Johnson, *Matrix Analysis*, 2nd ed. (Cambridge University Press, Cambridge, UK, 2012).
[36] We implicitly assume here that the metastable state exists, because we consider the limit of $y \to 0$ from above. During numerical simulations, we have observed that the quadratic equation for the prevalence Eq. (6) actually has two physical solutions. Therefore, despite Conjecture III.1 about nonexisting metastable states, taking the limit $y \to 0$ is allowed, because there always exist nonzero solutions for the prevalence y in Eq. (6).