Coupled adaptive complex networks

S. Shai and S. Dobson
School of Computer Science, University of St Andrews, St Andrews, Fife KY16 9SX, Scotland, United Kingdom
(Received 7 December 2012; published 18 April 2013)

Adaptive networks, which combine topological evolution of the network with dynamics on the network, are ubiquitous across disciplines. Examples include technical distribution networks such as road networks and the internet, natural and biological networks, and social science networks. These networks often interact with or depend upon other networks, resulting in *coupled* adaptive networks. In this paper we study susceptible-infected-susceptible (SIS) epidemic dynamics on coupled adaptive networks, where susceptible nodes are able to avoid contact with infected nodes by rewiring their intranetwork connections. However, infected nodes can pass the disease through internetwork connections, which do not change with time: The dependencies between the coupled networks remain constant. We develop an analytical formalism for these systems and validate it using extensive numerical simulation. We find that stability is increased by increasing the number of internetwork links, in the sense that the range of parameters over which both endemic and healthy states coexist (both states are reachable depending on the initial conditions) becomes smaller. Finally, we find a new stable state that does not appear in the case of a single adaptive network but only in the case of weakly coupled networks, in which the infection is endemic in one network but neither becomes endemic nor dies out in the other. Instead, it persists only at the nodes that are coupled to nodes in the other network through internetwork links. We speculate on the implications of these findings.

DOI: 10.1103/PhysRevE.87.042812 PACS number(s): 89.75.Fb, 89.75.Hc

I. INTRODUCTION

Inspired by the availability of huge network data resources, the structure and dynamics of complex networks have been widely investigated in the past decade, and used as a framework for studying complex systems where nodes are the entities and links are the relations between them. As most real-world networks have the ability to adapt their topology dynamically in response to the dynamic states of nodes, recent studies suggest a new class of networks called *adaptive networks* [1]. These are characterized by the existence of a feedback loop between the network's topology and dynamics (nodes' states) which has been shown to give rise to different behaviors such as self-organizing criticality [2,3], spontaneous division of labor (the emergence of distinct classes of nodes from an initially homogeneous population) [4,5] and cooperation [6–8].

However, real-world networks often interact with or depend on other networks, resulting in *coupled* adaptive networks in which there are links between otherwise independent networks. For example, brain networks consisting of synaptic connections linking neural units can be divided into separate cortical areas coupled together through inter-regional pathways [9]. Technical distribution networks are often divided into subsystems where each subsystem is adaptive and interconnected to other subsystems (e.g., data centers). Finally, distant populations are linked via people traveling (e.g., by flights) between the networks, creating interacting networks [10]. An important characteristic is that, while the individual networks are independently adaptive, their dependencies—the links between them—are often permanent (nonadaptive). This limits an individual network's ability to adapt in the face of challenges, since it can change its own topology but not its dependence on other networks. In geographically distant social networks, for example, long-distance links are often family links which are not subject to rewiring compared to short-distance friend and acquaintance links.

Motivated by these examples, we study a model of two random adaptive networks with internetwork connections between them. In particular, we extend Gross *et al.*'s model of SIS epidemic dynamics on adaptive networks [11]. In this model, similarly to SIS epidemic spreading in static networks [12], nodes can be in either one of two states—susceptible or infected—where susceptible nodes can get infected from their infected neighbors and infected nodes can recover and become susceptible again. In addition, the network is adaptive in the sense that susceptible nodes can rewire their links from infected neighbors to randomly selected susceptible ones. In a human-contact network, for example, this corresponds to not meeting a friend one knows to be ill. This dynamics has been shown [11] to lead to the emergence of new epidemic thresholds and the coexistence of two stable equilibria where both endemic and disease-free states are attractive while in static networks only one stable equilibrium is observed. Recent studies consider more realistic scenarios extending Gross *et al.*'s model such as susceptible-infected-recovered-susceptible (SIRS) epidemic dynamics where infected nodes are recovered (and temporarily immune to the disease) before they become susceptible again [13], susceptible-infected-vaccinated (SIV) dynamics where vaccination occurs in Poisson-distributed pulses [14], and the effect of different rewiring rules [15]. These models result in similar dynamics that is not observed in static network model.

In our model some of the nodes are also connected to nodes in the other network through permanent (nonrewireable) internetwork links, which can also pass the disease. The additional internetwork links between the networks create an interesting situation where nodes in each network change their links according to the states of their neighbors in the same network, but are also affected by the states of the nodes in the coupled network. Using the human-contact example again,

there are some people one cannot avoid even if one knows them to be ill.

We provide an analytical formalism able to account for the complete time evolution of the disease spreading in each network. Our formalism enables each network to have different intranetwork infection rates as well as different starting configurations. Moreover, it allows different infection rates between (inter-) and within (intra-) the networks, accounting for limited cases of time-scale separation between the intra- and internetwork disease-spreading processes. We present a bifurcation analysis of the stationary disease prevalence when changing the dynamics parameters: infection rate, rewiring rate, and number of internetwork links. We verify the result obtained analytically by explicit simulation of the networks.

We discover that the internetwork links give rise to nontrivial behaviors. Similar to Gross *et al.*'s model, two stable equilibria are also observed in our model. We find that by increasing the number internetwork links (higher coupling), the width of the bistability region (the range of parameters where both endemic and disease-free states are reachable depending on the initial conditions) becomes smaller. In other words, higher coupling provides more stability, where the disease prevalence depends less on the initial number of infected nodes. Secondly, we discover a new stable state that appears only in the case of weakly coupled networks and not at all in the case of a single adaptive network. In this state, intermediate disease prevalence values are observed in one of the networks describing the case where the disease persists only at the coupled nodes (the ones that are connected to nodes in the other network through internetwork links) and does not break out into the wider network. This is equivalent to the case, in a transport network, where people who travel a lot are becoming infected by a disease that does not exist in their home population—but since the infection rate is too low (or the defense mechanisms are too efficient) for the disease to persist, these individuals are the *only* ones who remain infected and the disease does not break out into the rest of the population.

II. MODEL

We consider two networks A and B with the same number of nodes $N_A = N_B = N$, where a randomly chosen fraction of nodes p_{coup} (the *coupling probability*) from each network are connected through internetwork connections to nodes in the other network. This is a similar model to the one of interdependent networks studied by Buldyrev *et al.* [16]: However, in our model the internetwork links are regular connectivity links instead of dependency links. While dependency links are used to model a situation where a failure of a node in one network leads to the failure of its dependent node in the other network, internetwork connectivity links model interaction between networks, such that an epidemic or rumor that is spreading in one network, for example, can spread to the other. Though simple, this model already gives rise to nontrivial behaviors and can be extended to account for more general cases, for example, by using any internetwork degree distribution [17,18].

The dynamics we consider is adaptive SIS epidemic spreading [11], where nodes in each network are either susceptible (S) or infected (I). Starting with two networks of susceptible nodes, we introduce a seed proportion of infected nodes in each network $0 \leqslant \epsilon_A, \epsilon_B \leqslant 1$. Then at every time step infected nodes can pass the disease to their intra- and internetwork neighbors at some rate (the *infection rate*), while they recover and become susceptible again at some other rate α (the *recovery rate*). Let β_A, β_B, β_{inter} be the infection rates within network A, B and between the networks, respectively. For every intranetwork link connecting an infected with a susceptible in network A (respectively, network B), the susceptible node becomes infected with infection rate β_A (β_B). In addition, for every internetwork link connecting an infected node from network A (network B) with a susceptible from network B (network A), the susceptible node becomes infected with infection rate β_{inter}. In addition, we allow susceptible nodes to protect themselves by rewiring their intranetwork links. For every intranetwork link connecting a susceptible node with an infected node in network A (network B), the susceptible node rewires the link with rewiring probability γ: It breaks the link to the infected node and forms a new intranetwork link to another randomly selected susceptible node, where double- and self-connections are prohibited. The dynamic rules described above guarantee that the number of nodes in both networks as well as the number of intra- and internetwork links remain constant over time.

III. FORMALISM

Let $[X_\sigma]$,$[X_\sigma Y_{\sigma'}]$, and $[X_\sigma Y_{\sigma'} Z_{\sigma''}]$, where $X,Y,Z \in \{I,S\}$ and $\sigma,\sigma',\sigma'' \in \{A,B\}$ represent the zeroth-, first-, and second-order moments of the system, so $[X_\sigma]$ corresponds to the fraction of X nodes in network σ, $[X_\sigma Y_{\sigma'}]$ is the density of XY intra- or internetwork links per node, and $[X_\sigma Y_{\sigma'} Z_{\sigma''}]$ is the density of XYZ triplets per node. By this notation, $[I_A]$ ($[I_B]$) and $[S_A]$ ($[S_B]$) is the fraction of infected and susceptible nodes in network A (network B), respectively. By the conservation of the number of nodes we obtain

$$[I_A] + [S_A] = 1, \quad [I_B] + [S_B] = 1.$$

$[S_A S_A]$,$[I_A I_A]$,$[I_A S_A]$ are the densities per node of intranetwork links in network A connecting susceptible to susceptible, infected to infected, and infected with susceptible, respectively. Note that $[I_A S_A]$ accounts for all the links connecting either susceptible to infected or infected to susceptible in network A. In other words, we do not distinguish between $[I_A S_A]$ and $[S_A I_A]$, or between $[I_B S_B]$ and $[S_B I_B]$. By the conservation of the number of intranetwork links we obtain

$$[S_A S_A] + [I_A I_A] + [I_A S_A] = \frac{\langle k_A \rangle}{2},$$
$$[S_B S_B] + [I_B I_B] + [I_B S_B] = \frac{\langle k_B \rangle}{2},$$

where $\langle k_A \rangle$, $\langle k_B \rangle$ are the mean degree of nodes in networks A and B, respectively. Note that the mean degrees only account for intranetwork links while ignoring internetwork links. The latter are considered in the coupling probability p_{coup}. Finally $[S_A S_B]$,$[I_A I_B]$,$[I_A S_B]$,$[I_B S_A]$ are the densities per node of internetwork links connecting susceptible A node to susceptible B node, infected A node to infected B node, infected A node to susceptible B node, and infected B node to susceptible A node, respectively. By the conservation

of the number of inter-network links we obtain

$$[S_A S_B] + [I_A I_B] + [I_A S_B] + [I_B S_A] = p_{\text{coup}},$$

where p_{coup}, the coupling probability, is the fraction of nodes that are connected to nodes in the other network through internetwork links.

We may now derive a system of ordinary differential equations (ODEs) describing the time evolution of the model. The time evolution of the fraction of infected nodes in each network is given by

$$\frac{d[I_A]}{dt} = \beta_A[I_A S_A] + \beta_{\text{inter}}[I_B S_A] - \alpha[I_A], \quad (1)$$

$$\frac{d[I_B]}{dt} = \beta_B[I_B S_B] + \beta_{\text{inter}}[I_A S_B] - \alpha[I_B]. \quad (2)$$

The first and second terms in Eq. (1) describe the infection of susceptible nodes in network A due to intra- and inter-network links, respectively, while the third term describes recovery, and similarly for network B.

The time evolution of the densities of intranetwork links per node is given by

$$\frac{d[I_A I_A]}{dt} = \beta_A[I_A S_A] + 2\beta_A[I_A S_A I_A] + \beta_{\text{inter}}[I_A S_A I_B] - 2\alpha[I_A I_A], \quad (3)$$

$$\frac{d[I_B I_B]}{dt} = \beta_B[I_B S_B] + 2\beta_B[I_B S_B I_B] + \beta_{\text{inter}}[I_B S_B I_A] - 2\alpha[I_B I_B]. \quad (4)$$

The first and second terms in Eq. (3) correspond to the conversion of $S_A I_A$ links into $I_A I_A$ links as a result of new infections through intranetwork connections, while the third describes the conversion of these links as a result of infections through internetwork links. The fourth term represents the conversion of $I_A I_A$ links into $S_A I_A$ links as a result of recovery. Similarly, we can write the evolution of links connecting susceptible to susceptible as

$$\frac{d[S_A S_A]}{dt} = (\alpha + \gamma)[I_A S_A] - \beta_A[S_A S_A I_A] - \beta_{\text{inter}}[S_A S_A I_B], \quad (5)$$

$$\frac{d[S_B S_B]}{dt} = (\alpha + \gamma)[I_B S_B] - \beta_B[S_B S_B I_B] - \beta_{\text{inter}}[S_B S_B I_A]. \quad (6)$$

The first and second terms in Eq. (5) correspond to the conversion of $I_A S_A$ links into $S_A S_A$ links as a result of recovery and rewiring, and the third and fourth terms describe the conversion of $S_A S_A$ links into $I_A S_A$ links as a result of infection through intra- and internetworks links, respectively.

Finally, we describe the time evolution of the densities of internetwork links per node,

$$\frac{d[I_A I_B]}{dt} = \beta_{\text{inter}}[I_A S_B] + \beta_{\text{inter}}[I_B S_A] + \beta_A[I_A S_A I_B] + \beta_B[I_B S_B I_A] - 2\alpha[I_A I_B], \quad (7)$$

$$\frac{d[S_A S_B]}{dt} = \alpha[I_A S_B] + \alpha[I_B S_A] - \beta_A[I_A S_A S_B] - \beta_B[I_B S_B S_A], \quad (8)$$

$$\frac{d[I_A S_B]}{dt} = \alpha[I_A I_B] + \beta_A[I_A S_A S_B] - \alpha[I_A S_B] - \beta_{\text{inter}}[I_A S_B] - \beta_B[I_B S_B I_A]. \quad (9)$$

The first and second terms in Eq. (7) correspond to the conversion of interlinks connecting susceptible to infected into $I_A I_B$ links as a result of infection through internetwork connections, while the third and fourth describe the same conversion due to infection through intranetwork connection. The last term corresponds to the recovery of either an A or B node. Similarly, the first and second terms in Eq. (8) correspond to recovery while the last two terms correspond to infection through intranetwork links. Note that infection due to internetwork links is not possible here since both nodes are susceptible. Finally, the first and second terms in Eq. (9) describe the gain of internetwork links connecting susceptible A node with infected B node due to recovery of a B node and the infection of an A node through the intranetwork neighbor. The last three terms describe the loss of $I_A S_B$ links due to recovery of an A node and the infection of a B node through inter- and intranetwork links, respectively.

Equations (1)–(9) together with the conservation rules of links and nodes still do not constitute a closed model since the second-order variables describing the evolution of triplets in the networks are unknown. In the following we are using the pair approximation method [11,19], where the triplet moments $[X_\sigma Y_{\sigma'} Z_{\sigma''}]$ are expressed in terms of links and nodes:

$$[I_A S_A I_A] = \frac{[I_A S_A][I_A S_A]}{2[S_A]}; \quad [I_B S_B I_B] = \frac{[I_B S_B][I_B S_B]}{2[S_B]},$$

$$[S_A S_A I_A] = \frac{2[S_A S_A][I_A S_A]}{[S_A]}; \quad [S_B S_B I_B] = \frac{2[S_B S_B][I_B S_B]}{[S_B]},$$

$$[I_A S_A I_B] = \frac{[I_A S_A][I_B S_A]}{[S_A]}; \quad [I_B S_B I_A] = \frac{[I_B S_B][I_A S_B]}{[S_B]},$$

$$[S_A S_A I_B] = \frac{2[S_A S_A][I_B S_A]}{[S_A]}; \quad [S_B S_B I_A] = \frac{2[S_B S_B][I_A S_B]}{[S_B]},$$

$$[I_A S_A S_B] = \frac{[I_A S_A][S_A S_B]}{[S_A]}; \quad [I_B S_B S_A] = \frac{[I_B S_B][S_B S_A]}{[S_B]}.$$

The first-order approximation assumes—similarly to the mean-field approximation, where S and I nodes are assumed to be homogeneously distributed in the network—the homogeneous distribution of links $X_\sigma Y_{\sigma'}$ in the network. This assumption might not be valid at the beginning of the spreading process where only a seed of infected nodes exists in the network, but gives a good approximation for the situation at equilibrium.

We are aware that the approximation above might only provide an accurate prediction for the behavior of homogenous networks such as Erdös-Rényi (ER) random graphs [20], and might fail in the case of nonhomogenous networks like scale-free graphs [21]. We test the analytical solution derived from Eqs. (1)–(9) together with the pair approximation and the conservation rules only against a simulation of ER networks. Extending the model to give a better prediction for other types of networks is possible, however. One way to do this is by computing the densities of triplets in the networks numerically on-demand from short bursts of appropriately initialized numerical simulations [21]. Another approach is to write the ODEs describing the time evolution of higher-order

variables, truncating at the maximum degree of the networks [22]. Finally, a recent approach inspired by the computation of epidemic thresholds, assumes a low density of active links (connecting nodes in different states), which allows more careful handling of long-range correlations [23]. We chose the pair approximation method for its simplicity in order to show the potential of our analytical formalism. More complicated approaches can, however, be applied easily within the framework of our model. Indeed, one of the main results of this paper is that the failure of the approximation explains the emergence of a new stable dynamics whose existence is masked unless the higher-order moments are dealt with properly.

IV. RESULTS

We confirm results computed analytically by explicitly simulating the dynamics over a system of two coupled adaptive networks using the LARGENET2 C++ library [25]. We use two ER networks with $N_A = N_B = 100000$ nodes and average connectivity $\langle k_A \rangle = \langle k_B \rangle = 20$ (as in [21]) and recovery rate $\alpha = 0.002$, (as in [21,22]). As stated previously, we start the epidemic spreading by initially infecting a fraction ϵ_A of nodes in network A and ϵ_B in network B. Thus, the initial conditions for Eqs. (1)–(9) are given by

$$
\begin{aligned}
[I_A](0) &= \epsilon_A; \quad [I_B](0) = \epsilon_B, \\
[I_A I_A](0) &= {\epsilon_A}^2 \frac{\langle k_A \rangle}{2}; \quad [I_B I_B](0) = {\epsilon_B}^2 \frac{\langle k_B \rangle}{2}, \\
[S_A S_A](0) &= (1-\epsilon_A)^2 \frac{\langle k_A \rangle}{2}; \quad [S_B S_B](0) = (1-\epsilon_B)^2 \frac{\langle k_B \rangle}{2}, \\
[I_A I_B](0) &= p_{\text{coup}}\, \epsilon_A \epsilon_B, \\
[S_A S_B](0) &= p_{\text{coup}} (1-\epsilon_A)(1-\epsilon_B), \\
[I_A S_B](0) &= p_{\text{coup}}\, \epsilon_A (1-\epsilon_B).
\end{aligned}
$$

Figure 1 shows bifurcation diagrams of the stationary disease prevalence I_B^* in network B as a function of the infection rate $\beta = \beta_A = \beta_B = \beta_{\text{inter}}$ for different values of the coupling probability p_{coup} when no rewiring is taking place ($\gamma = 0$). In this case, all coupling probabilities result in a single stable branch corresponding to continuous dynamical transition from the healthy to endemic state which occurs at the epidemic threshold. The coupling has almost no effect in this case as the epidemic threshold is already very small in the case of a single network.

Figure 2 shows the bifurcation diagrams obtained for rewiring rate $\gamma = 0.04$. In the case where $p_{\text{coup}} = 0$, our model recovers the result obtained in [11]: The two networks are separated and the resulting bifurcation diagram has two stable branches corresponding to lower and upper transition depending on the initial number of infected nodes. Numerical simulation is in good agreement with the results computed analytically.

In the cases of coupling probability $p_{\text{coup}} = 0.01, 0.1$, both the results obtained analytically and numerically (shown more obviously in the inset) show a new stable state of intermediate I_B^* values, where the epidemic does not spread to all nodes in network B, but does not die out either. This state is obtained by starting the numerical simulation with no infected nodes in network B, $\epsilon_B = 0$, and almost all the nodes infected in

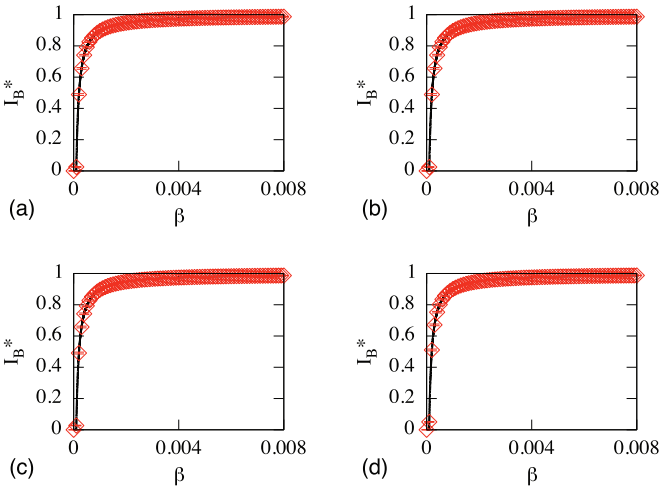

FIG. 1. (Color online) Bifurcation diagram of the stationary disease prevalence in network B, I_B^* as a function of the intra- and internetwork infection rate, β when no rewiring is taking place for coupling probabilities (a) $p_{\text{coup}} = 0$, (b) $p_{\text{coup}} = 0.01$, (c) $p_{\text{coup}} = 0.1$, (d) $p_{\text{coup}} = 0.9$. Stable (thick black) branches have been computed analytically from Eqs. (1)–(9) using the dynamical systems analysis software XPPAUT [24]. Analytical results were confirmed using numerical simulation of two ER networks of 10^5 nodes and 10^6 edges (red diamonds). Points and error bars (if larger than marker size) were obtained by averaging at least 25 000 values corresponding to the prevalence at equilibrium (at least 5000 time steps) of 50 simulation runs for each of the initial values: $\epsilon_A = 0.001; \epsilon_B = 0.001$, $\epsilon_A = 0.999; \epsilon_B = 0$, $\epsilon_A = 0.999; \epsilon_B = 0.999$.

network A, $\epsilon_A = 0.999$. In the following we show that this state describes the case where the epidemic does not spread equally to all the nodes in network B, but mostly to the coupled ones.

We define $I^*_{\text{B}_{\text{coupled}}}$, the fraction of coupled nodes in network B that remain infected at the end of the epidemic outbreak, as

$$
I^*_{\text{B}_{\text{coupled}}} = \begin{cases} \frac{N^*_{\text{B}_{\text{coupled infected}}}}{N^*_{\text{B}_{\text{infected}}}}, & \text{if } N^*_{\text{B}_{\text{infected}}} > 0 \\ 0, & \text{otherwise,} \end{cases} \tag{10}
$$

where $N^*_{\text{B}_{\text{infected}}}$ and $N^*_{\text{B}_{\text{coupled infected}}}$ are, respectively, the number of infected and coupled infected nodes in network B at the end of the epidemic outbreak.

Figure 3 shows $I^*_{\text{B}_{\text{coupled}}}$ and I_B^* as a function of the infection rate β when starting with initial conditions $\epsilon_A = 0.999$ and $\epsilon_B = 0$. In a network where the infected nodes are equally distributed, we would expect the fraction of infected coupled nodes $I^*_{\text{B}_{\text{coupled}}}$ to constitute the same part of the infected nodes as the fraction of coupled nodes of all of the nodes. In other words, we would expect $I^*_{\text{B}_{\text{coupled}}}$ to be close to p_{coup} (unless there are no infected nodes, where it would be 0). Indeed, the graph starts with y values close to 0, indicating that there are no infected nodes. But, in the range of β where intermediate I_B^* values are obtained ($0.0008 < \beta < 0.002$ for $p_{\text{coup}} = 0.01$ and $0.0008 < \beta < 0.0014$ for $p_{\text{coup}} = 0.1$), the fraction of infected coupled nodes $I^*_{\text{B}_{\text{coupled}}}$ is close to one, indicating that almost all infected nodes are coupled. For larger

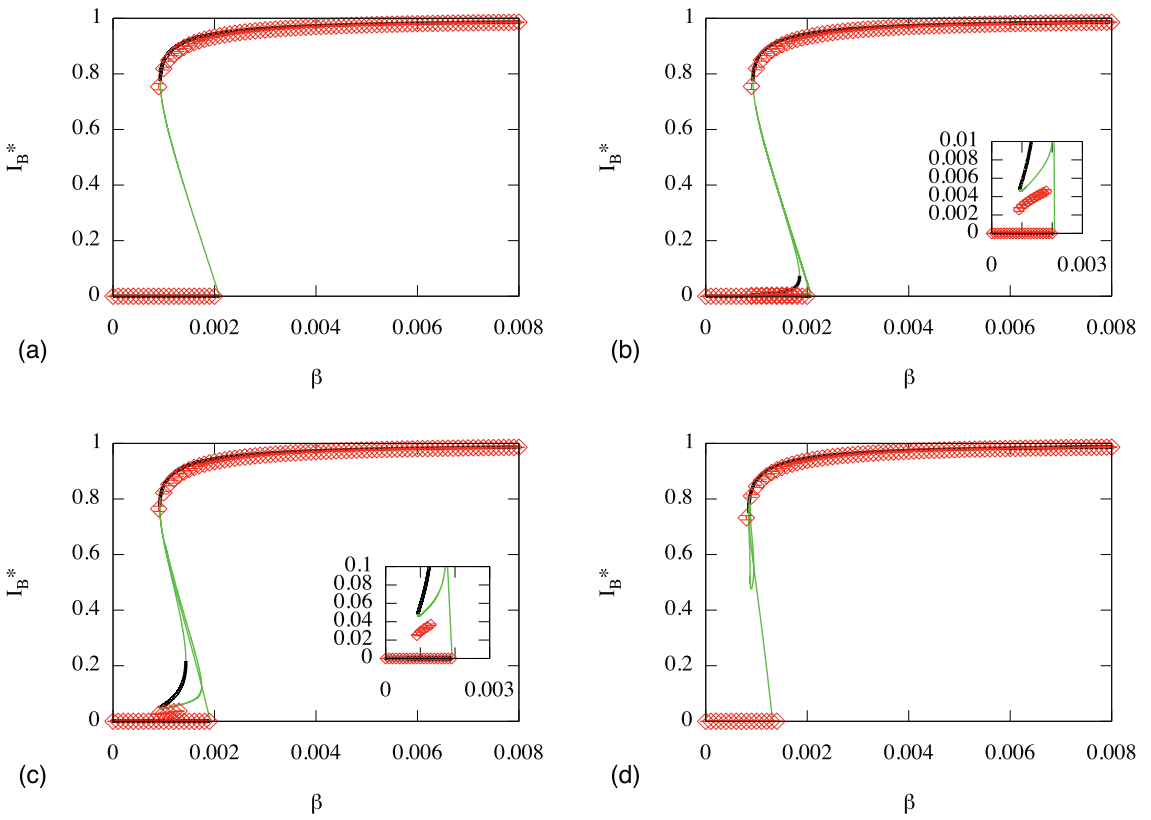

FIG. 2. (Color online) Bifurcation diagram of the stationary disease prevalence in network B, I_B^* as a function of the intra- and internetwork infection rate, β for rewiring probability $\gamma = 0.04$ and coupling probabilities (a) $p_{coup} = 0$, (b) $p_{coup} = 0.01$, (c) $p_{coup} = 0.1$, (d) $p_{coup} = 0.9$. Analytical computations of stable (thick black) and unstable (thin green) branches as well as numerical values (red diamonds) were obtained in the same way as in Fig. 1. The insets show a zoom of a new stable state in the cases of $p_{coup} = 0.01$ and $p_{coup} = 0.1$.

β values, the expected result is obtained and $I^*_{B_{coupled}}$ is close to the coupling probability p_{coup}.

This explains the quantitative disagreement between the analytical solution and the numerical simulation. Recall that the pair approximation assumes homogeneous distribution of links in the network. Therefore, just as the approximation does not hold during the initial phases of the dynamics, in a partially invaded network (as shown in Fig. 3) correlations exist that are not captured by the approximation. The emergence of this new stable state, observed both numerically and analytically for the case of coupling probability $p_{coup} = 0.01,0.1$, is a consequence of the fact that the coupled nodes cannot rewire their internetwork links. Therefore, for low infection rates, the disease can be established in one network while not invading to the other but instead reaching only to the coupled nodes, since the nodes that are not coupled can protect themselves by rewiring their links. This is not possible for larger infection rates where the new stable state is no longer reachable and only the two other states of endemic and disease-free remain stable.

In the following we confirm the deviation of the simulation from the analytical solution (shown in Fig. 2) originates from the pair approximation of the triplets $[I_A S_A I_B], [S_A S_A I_B], [I_A S_A S_B]$. We do this by substituting the pair approximation of these triplets with the actual densities obtained from the simulation, and leave everything else in Eqs. (1)–(9) the same.

Figure 4 shows both numerical and analytical results for $\gamma = 0.04$ and $p_{coup} = 0.01,0.1$ starting with initial conditions $\epsilon_A = 0.999$ and $\epsilon_B = 0$. Following the observation above that the assumption of homogeneous distribution of links in the network does not hold in this case (since the epidemic persists only in the coupled nodes) we substitute the pair approximation of all the triplets involving nodes from both networks with the values obtained numerically. The graph shows a very good agreement between results obtained numerically and analytically, indicating that the deviation in Fig. 2 was indeed as a result of the pair approximation for these triplets. As mentioned before, there are other methods to overcome the inadequacy of the pair approximation in the presence of long-range correlations, such as higher order approximation [22] or using epidemic threshold calculation [11,23]. This is not in the scope of the current paper but can be addressed in future more accurate models.

The third stable state shown in Fig. 2 for the cases of coupling probability $p_{coup} = 0.01,0.1$, becomes unstable again for strongly coupled networks ($p_{coup} = 0.9$). This result is expected since in tightly coupled networks, there exist a larger number of coupled nodes that cannot protect themselves from infection due to internetwork links even at small infection rates. Therefore, any epidemic persisting in one network, will eventually spread and persist in the other network as well.

Finally, we see in Fig. 2 that the width of the multistability area becomes smaller as the coupling probability increases. In

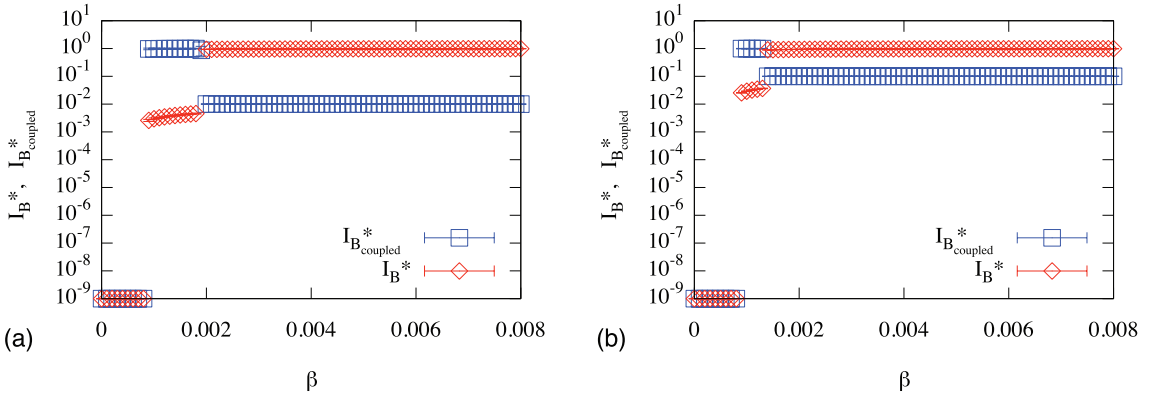

FIG. 3. (Color online) Fraction of infected nodes (red diamonds) and coupled infected nodes (blue squares) in network B at the end of the epidemic outbreak as a function of the intra- and internetwork infection rate β for rewiring rate $\gamma = 0.04$ and coupling probabilities (a) $p_{coup} = 0.01$ and (b) $p_{coup} = 0.1$. Red diamonds show the fraction of infected nodes at the end of the epidemic outbreak, and the blue ones show how many of these nodes are coupled [see Eq. (10)]. Results obtained from numerical simulation of two ER networks of 10^5 nodes and 10^6 edges starting with initial values $\epsilon_A = 0.999; \epsilon_B = 0$. Mean and error bars (if larger than marker size) were obtained by averaging at least 25 000 values corresponding to the prevalence at equilibrium (at least 5000 time steps) of 50 simulation runs. The figures show that in the range where intermediate values of the disease prevalence are obtained, the fraction of coupled infected nodes is close to 1 and not to the coupling probability as expected. In other words, in this range mostly the coupled nodes are the ones who remain infected.

other words, the range of β values for which more than one stable state is reachable depending on the initial conditions becomes smaller with more internetwork links meaning that a system of tightly coupled networks is more stable and depends less on the initial conditions.

V. CONCLUSION

We have considered a model of two coupled adaptive networks, where intranetwork links are rewired based on the states of the nodes. We have presented an analytical formalism and verified it using extensive numerical simulation of two random networks. Our model shows that a system of coupled adaptive networks is more stable (less dependent on initial conditions) with the existence of more internetwork links. In addition, we find a new stable state (that does not exist in the case of one adaptive network) where in one network the epidemic persists throughout the network and in the other it only persists in the coupled nodes and does not break out into the rest of the network. In order to obtain the exact quantitative behavior for this state from the analytical model, one must account for the actual second-order moments of the system—even for ER and other homogenous networks, where such higher-order terms may generally be treated by a uniform approximation.

In the future, we would like to examine the effect of rewiring of internetworks links. In the example mentioned earlier of coupled nodes representing people who travel a lot, rewiring of internetwork links corresponds to the imposition of travel restrictions. In this case, we expect the new stable

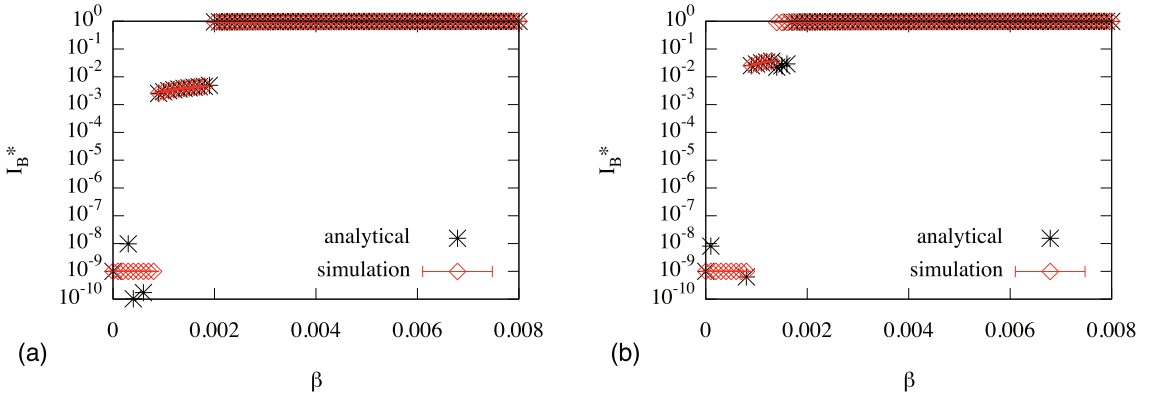

FIG. 4. (Color online) Fraction of infected nodes in network B at the end of the epidemic outbreak as a function of the intra- and internetwork infection rate β for rewiring rate $\gamma = 0.04$ and coupling probabilities (a) $p_{coup} = 0.01$ and (b) $p_{coup} = 0.1$ starting with initial conditions $\epsilon_A = 0.999; \epsilon_B = 0$. Red diamonds correspond to results obtained from numerical simulation of two ER networks of 10^5 nodes and 10^6 edges (same as in Fig. 2). Mean and error bars (if larger than marker size) were obtained by averaging at least 25 000 values corresponding to the prevalence at equilibrium (at least 5000 time steps) of 50 simulation runs. Black points correspond to results obtained analytically from Eqs. (1)–(9), where pair approximation of the triplets $[I_A S_A I_B], [S_A S_A I_B], [I_A S_A S_B]$ is substituted with the mean values obtained from the simulation.

state discovered to disappear for sufficiently high rewiring rates of the internetwork links. In addition, we would like to investigate the effect of time-scale separation between the epidemic spreading processes taking place within and between the networks. Finally, it would be very interesting to study a system consisting of any number of coupled adaptive networks within this model, and not just two as in the current paper, and see if more networks will lead to more stable states, as happened with the extension from one to two networks. We suspect that there might be more stable states, for example, in the case where some nodes are coupled with more than one network (i.e., there is an overlapping between the groups of nodes that are chosen to be coupled with each network). In this case, there might be a stable state for each group of nodes that are coupled with more than a certain number of networks.

ACKNOWLEDGMENTS

This work is supported by a scholarship from the Scottish Informatics and Computer Science Alliance (SICSA). The authors would like to acknowledge the help of Professor Jane Hillston and the anonymous reviewer, whose comments greatly improved the presentation of the work.

[1] T. Gross and B. Blasius, J. R. Soc. Interface **5**, 259 (2008).
[2] S. Bornholdt and T. Rohlf, Phys. Rev. Lett. **84**, 6114 (2000).
[3] C. Meisel, A. Storch, S. Hallmeyer-Elgner, E. Bullmore, and T. Gross, PLoS Comput Biol **8**, e1002312 (2012).
[4] A.-L. Do, L. Rudolf, and T. Gross, New J. Phys. **12**, 063023 (2010).
[5] J. Ito and K. Kaneko, Phys. Rev. E **67**, 046226 (2003).
[6] H. Ebel and S. Bornholdt, Phys. Rev. E **66**, 056118 (2002).
[7] A. Szolnoki and M. Perc, Europhys. Lett. **86**, 30007 (2009).
[8] M. G. Zimmermann, V. M. Eguiluz, and M. SanMiguel, Phys. Rev. E **69**, 065102 (2004).
[9] O. Sporns, D. R. Chialvo, M. Kaiser, and C. C. Hilgetag, Trends in Cognitive Sciences **8**, 418 (2004).
[10] E. A. Leicht and R. M. D'Souza, arXiv:0907.0894.
[11] T. Gross, Carlos J.Dommar D'Lima, and B. Blasius, Phys. Rev. Lett. **96**, 208701 (2006).
[12] R. Pastor-Satorras and A. Vespignani, Phys. Rev. E **63**, 066117 (2001).
[13] L. B. Shaw and I. B. Schwartz, Phys. Rev. E **77**, 066101 (2008).
[14] L. B. Shaw and I. B. Schwartz, Phys. Rev. E **81**, 046120 (2010).
[15] S. Wieland, T. Aquino, and A. Nunes, Europhys. Lett. **100**, 69901 (2012).
[16] S. V. Buldyrev, R. Parshani, G. Paul, H. E. Stanley, and S. Havlin, Nature (London) **464**, 1025 (2010).
[17] M. Dickison, S. Havlin, and H. E. Stanley, Phys. Rev. E **85**, 066109 (2012).
[18] A. Saumell-Mendiola, M. A. Serrano, and M. Boguñá, Phys. Rev. E **86**, 026106 (2012).
[19] M. J. Keeling and K. T. Eames, Journal of The Royal Society Interface **2**, 295 (2005).
[20] P. Erdös and A. Rényi, Publ. Math. Inst. Hung. Acad. Sci. **5**, 17 (1960).
[21] T. Gross and I. G. Kevrekidis, Europhys. Lett. **82**, 380041 (2008).
[22] V. Marceau, P.-A. Noël, L. Hébert-Dufresne, A. Allard, and L. J. Dubé, Phys. Rev. E **82**, 036116 (2010).
[23] G. A. Böhme and T. Gross, Phys. Rev. E **83**, 035101 (2011).
[24] B. Ermentrout, *Simulating, Analyzing, and Animating Dynamical Systems: A Guide Simulating, Analyzing, and Animating Dynamical Systems: A Guide to XPPAUT for Researchers and Students* (SIAM, Philadelphia, 2002).
[25] G. Zschaler and T. Gross, Bioinformatics **29**, 277 (2012).