Epidemics on interconnected networks

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Populations are seldom completely isolated from their environment. Individuals in a particular geographic or social region may be considered a distinct network due to strong local ties but will also interact with individuals in other networks. We study the susceptible-infected-recovered process on interconnected network systems and find two distinct regimes. In strongly coupled network systems, epidemics occur simultaneously across the entire system at a critical infection strength β_c, below which the disease does not spread. In contrast, in weakly coupled network systems, a mixed phase exists below β_c of the coupled network system, where an epidemic occurs in one network but does not spread to the coupled network. We derive an expression for the network and disease parameters that allow this mixed phase and verify it numerically. Public health implications of communities comprising these two classes of network systems are also mentioned.

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I. INTRODUCTION

Complex network models of the interactions in human society have been used to understand many problems in epidemiology [1–8]. These models have generally assumed that all of the nodes interact on a single network with a single degree distribution. Even when these degree distributions allow for large heterogeneities—as in the case of scale-free networks [9], where hubs with large numbers of connections can arise—the assumption remains that every node is part of a single network and is represented by a single underlying topology. In reality, however, societies are composed of many interconnected networks, as in Fig. 1, which may be communities within a larger population or separate systems entirely. A disease can spread through the network of direct personal contacts, via the water utilities network, and travel from city to city over highway or airline networks. These interconnected network systems may be composed of different types of nodes, which may have degrees drawn from distinct degree distributions and may have different connectivities between them. Real-world examples of these systems can be seen in a 2009 study by Stehlè *et al*. which found a threefold difference in interaction time between students inside and outside of their own class [10]. Other studies have shown similar patterns [11,12]. Human-animal interacting network systems are also of great importance. The H5N1 variant of influenza spreads through the network of birds and from them to individuals in the network of humans that work or live closely with them. While no current mechanism exists for efficient spreading from human to human, there is substantial concern that such a mechanism will evolve [13]. Human-mosquito-human transmission for the *Plasmodium knowlesi* malaria strain is also a worrying concern [14].

Interconnected network systems have been of interest to researchers in numerous different ways [15–21]. Interconnected dependency networks, where failure in nodes in one network causes failures of dependent nodes in the other network, exhibit failure cascades, where the cross network dependencies result in a network much more easily fragmented than single networks of the same degree distribution [22]. Interconnected power networks, where transport capacity and failure vulnerability are competing properties, were examined and an optimal level of interconnection was found [23]. Networks without dependencies, such as interconnected social networks, where populations exist at city, state, and national levels, have also been examined. In these networks, the level of movement between cities (the interconnections between them) have been shown to affect the epidemic transition on the metapopulation level [24,25], although, in this case, the low-level networks were treated in a mean-field fashion, classified only by rate equations and infection numbers, with no internal network features. In addition, the percolation threshold in interacting networks was found to be lower than in single networks, with a giant cluster appearing for a smaller total number of links [26].

In this work we consider two interconnected networks (or, alternately, interconnected communities within a single, larger network). We pose the following question: Under what conditions will an epidemic spread only on the one network, with minimal isolated infections on other network components, and under what conditions will it spread across the entire interconnected network system? Depending on the parameters of the individual networks and their interconnections, connecting one network to another can have a profound or a small effect on the spread of an epidemic. Identifying the conditions in which these cases occur is vital to our understanding and management of epidemic processes.

We define two different interconnected network regimes, strongly and weakly coupled, and find the interaction strength value separating these two regimes. Our primary result is to show that, in the strongly coupled case, we find that all networks are simultaneously either disease free or part of an epidemic, while in the weakly coupled case a new "mixed" phase can exist. In this mixed phase, the disease is epidemic on only one network, and not in other networks, despite the interconnections. The applications to public health are straightforward. If two neighboring communities comprise a strongly coupled network system, then an outbreak in any community is cause for immediate concern in the other. Due to this, in the strongly coupled case it becomes important to pursue a strategy of communication and joint action between

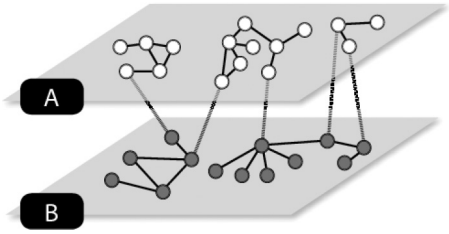

FIG. 1. An interconnected network system with two networks: A and B. Nodes have intranetwork links within their own network but also internetwork links connecting them to the other network.

public health agencies and perhaps even intervention from a single agency with higher authority.

II. MODEL

In this section, we consider the case of only two interconnected networks of equal size, but it is easily possible to extend the model to an arbitrary number of networks of any size.

We form our interconnected network systems in the following way:

(1) Generate two networks, A and B, with their own intranetwork ($A \leftrightarrow A$ and $B \leftrightarrow B$) degree distributions $P_A(k)$ and $P_B(k)$ according to the standard Molloy-Reed configuration model [27].

(2) Draw a degree from the internetwork ($A \leftrightarrow B$) degree distribution, $P_{AB}(k)$, for all the nodes in both networks.

(3) If the total degree assigned to nodes in network A is not equal to the total degree assigned to nodes in network B, randomly reassign a node in B until the total numbers in each network are equal.

(4) Randomly connect nodes in network A to nodes in network B to form the interconnected network system.

This method generates random, uncorrelated, interconnected network systems with specified inter- and intranetwork degree distributions. While this method works for any arbitrary degree distribution, $P_x(k)$, we present results only for random Poissonian degree distributions.

The susceptible-infected-recovered (SIR) epidemic model is used here to study the effects of interconnected network structure on epidemic threshold. The SIR model is well established and describes diseases such as HPV, seasonal influenza, or H1N1 [24,28]. In this model, each node has three possible states: susceptible (s), infected (i), or recovered (r). Each node begins in state s, except for a single node in one network chosen to be in state i. Nodes in state i infect their neighbors in state s with probability β at each time step, changing them to i. Nodes enter state r after spending a recovery time t_r in state i.

In order to find the threshold for an epidemic, we can think of epidemic spreading as a bond percolation process [3,29,30] on a network. In bond percolation, links between nodes are activated with a certain probability p. If this probability is greater than a certain critical value, p_c, then a giant cluster emerges, where the existence of a path between any two nodes is almost certain. In a disease-spreading model, nodes infect their neighbors, "activating" the links between them with a certain probability, and a disease reaches nodes through this entire network above a certain critical value, β_c, just as in the case for percolation.

In complex networks, this critical threshold for percolation if all potential links are activated is $\kappa = 2$. Here κ is the expected number of nearest neighbors that a node chosen by following an arbitrary link will have and is calculated from the ratio between the second and the first moments of the degree distribution: $\kappa = \langle k^2 \rangle / \langle k \rangle$. For $\kappa \geqslant 2$, a giant cluster exists, while for $\kappa \leqslant 2$ only small isolated clusters exist. If some subset of bonds is activated at random with probability p, a giant cluster appears at a critical value of $p_c = 1/(\kappa - 1)$ [31].

The SIR model likewise has an epidemic phase transition at a critical $\beta = \beta_c$, below which the disease remains confined to the local neighborhood of the initial infection and above which the disease spreads throughout the network. This transition from the disease-free phase to the epidemic phase depends on the average number of secondary infections per infected node becoming larger than 1. This allows the long-term survival of the disease, as the infection density will grow over time on average, and, thus, ensures that the epidemic spreads to a large fraction of the population. In our problem, the expected number of susceptible neighbors that a node has when it just becomes infected is given by $\kappa - 1$, since the total expected number of neighbors is κ, and one of them must be excluded as the infected parent from which the current node descended. The transmissibility $T_\beta = 1 - (1 - \beta)^{t_r}$ is the probability to infect a neighbor before recovery. The mean number of secondary infections per infected node is, thus, $N_I = (\kappa - 1)T_\beta$. The infection will die out if each infected node does not infect on average at least one replacement so, for a very large network, the critical point is given by the relation $(\kappa - 1)T_\beta = 1$. The single network model exhibits only a single transition at β_c given by [3]

$$\beta_c(\kappa) = 1 - [1 - (\kappa - 1)^{-1}]^{1/t_r}. \quad (1)$$

In the interconnected network model, the behavior is more complicated, as the disease can potentially cause an epidemic in different combinations of the networks. The disease can be in the epidemic phase in both networks, in the disease-free phase in both networks, or active in one network while the other remains disease free, called here the mixed phase. The boundaries of these phases are controlled by κ_A, κ_B, and κ_T, where κ_A and κ_B are calculated over the individual A and B networks, disregarding internetwork connections, and κ_T is calculated over the entire coupled network system, including intra- and internetwork links.

III. STRONGLY COUPLED NETWORK SYSTEMS

We consider an interconnected network system to be strongly coupled if κ_T is larger than κ_A and κ_B. For random networks, $\kappa = \langle k \rangle + 1$, and, thus, we may write κ_T in terms of the average degrees $\langle k_A \rangle$, $\langle k_B \rangle$, and $\langle k_{AB} \rangle$ as follows:

$$\begin{aligned}\kappa_T = {} & [\langle k_A \rangle^2 + \langle k_A \rangle + \langle k_B \rangle^2 + \langle k_B \rangle + 2\langle k_{AB} \rangle^2 + 2\langle k_{AB} \rangle \\ & + 2\langle k_A \rangle \langle k_{AB} \rangle + 2\langle k_B \rangle \langle k_{AB} \rangle] \\ & \times [\langle k_A \rangle + \langle k_B \rangle + 2\langle k_{AB} \rangle]^{-1}. \quad (2)\end{aligned}$$

Without loss of generality, we define network B as the more intraconnected network ($\langle k_B\rangle > \langle k_A\rangle$). For fixed network parameters $\langle k_A\rangle$ and $\langle k_B\rangle$, we can then derive the critical interaction strength $\langle k_{AB}\rangle_c$ that separates strongly coupled ($\kappa_T > \kappa_B$) from weakly coupled ($\kappa_T < \kappa_B$) networks,

$$\langle k_{AB}\rangle_c = \frac{\sqrt{2\langle k_A\rangle\langle k_B\rangle - \langle k_A\rangle^2} - \langle k_A\rangle}{2}. \quad (3)$$

In strongly coupled network systems, we expect any epidemic to emerge simultaneously on networks A and B. Using Eq. (1) for each of the three κ, it can be seen that, for the strongly coupled case $\beta_c(\kappa_T)$, the critical value of β for the disease to emerge on the giant component formed by the entire interconnected network is smaller than both $\beta_c(\kappa_A)$ and $\beta_c(\kappa_B)$, the critical values of β for epidemics to spread on networks A or B ignoring internetwork links. As such, any pathogen virulent enough to spread in network A or B alone will have already caused an epidemic occurring across the interconnected network system. For this case, the disease spreads across the interconnected network system as a single network, with the internetwork connections bringing an epidemic into existence before any intranetwork connections can do so independently; the mixed phase will not be seen. To support this, we plot the ratio of the largest connected infected cluster formed solely from nodes connected with intranetwork links, compared to the size of the largest connected cluster formed by nodes connected with all links, in Fig. 2. For a strongly coupled network system, the relative size of the largest connected infected component contained entirely in a single network decreases initially, showing that the epidemic is occurring across the interconnected network system, not locally in one of the networks. Thus, in the strongly coupled case, epidemic spreading is enhanced due to internetwork connections, with epidemics occurring for less virulent diseases than would spread on either network alone (lower β_c.)

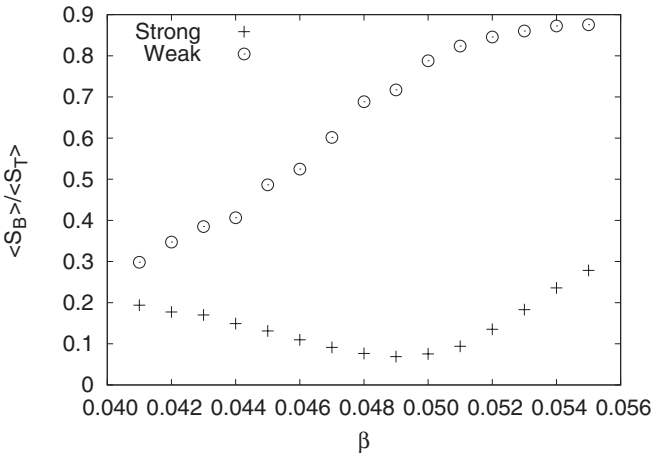

FIG. 2. Epidemics in strongly coupled network systems spread across all networks, remaining confined to one network in weakly coupled network systems. We plot S_B, the size of the largest connected cluster solely in network B, divided by S_T, the size of the infected cluster across the interconnected network system, for both strongly and weakly coupled network systems. The B-only cluster decreases in relative size until criticality [$\beta_c(\kappa_B) = 0.048$], showing that the epidemic spreads throughout both networks rather than remaining confined in B in the strongly coupled system. By contrast, in the weakly coupled system, the relative size of the B-only cluster grows until $\beta = \beta_c(\kappa_T) = 0.054$, showing that growth is localized in the more strongly coupled network. For the strongly coupled network, $\langle k_A\rangle = 1.5$, $\langle k_B\rangle = 2.5$, and $\langle k_{AB}\rangle = 2.5$. For the weakly coupled network, $\langle k_A\rangle = 1.5$, $\langle k_B\rangle = 4.55$, and $\langle k_{AB}\rangle = 0.3$. In all cases, $N_A = N_B = 10^4$ and $t_r = 5$.

One note is that for networks of identical intranetwork degree ($\langle k_A\rangle = \langle k_B\rangle$) $\langle k_{AB}\rangle_c = 0$. That is to say, identical networks always form strongly coupled network systems. This is in agreement with our findings that the phase diagram of strongly coupled network systems is similar to that of single networks. An interacting network system formed by attaching the labels "A" and "B" to different halves of a single network would be such an example system, and one should not expect that this relabeling could have any effect on the physical properties, such as phase transitions, of that network.

IV. WEAKLY COUPLED NETWORK SYSTEMS

If two networks are connected with $\langle k_{AB}\rangle$ below the threshold value from Eq. (3), i.e., $\kappa_B > \kappa_T$, we define the interconnected network system to be weakly coupled. From Eq. (2) this also gives $\kappa_T > \kappa_A$. Turning again to Eq. (1) we see that $\beta_c(\kappa_B) < \beta_c(\kappa_T) < \beta_c(\kappa_A)$.

Epidemic spreading is a noncompetitive process. Adding more links to a network can only increase the spread of an epidemic, never decrease it, as the chance of a node infecting its neighbors is constant regardless of degree. Thus, a disease with β above the individual epidemic threshold of network B [$\beta_c(\kappa_B)$] will enter the epidemic phase on that network, regardless of the other network and the values of κ_T and κ_A. If β is below $\beta_c(\kappa_T)$, however, the disease cannot spread to more than isolated small clusters of network A. Thus, in the weakly coupled case, we expect to see a mixed phase, with the boundaries dependent on the values of β and $\langle k_{AB}\rangle$. A mixed phase indicates that the addition of the interconnections between the two networks is only affecting epidemic spreading on the network with weaker intranetwork connections, with the epidemic on the network with stronger intranetwork connections unchanged by the internetwork links. The weakly coupled case in Fig. 2 shows this, with the largest connected cluster contained entirely in B becoming larger compared to the size of the giant component with increasing β until $\beta_c(\kappa_T)$ is reached, indicating that the disease does not spread through connected regions of network A.

If β is increased to above $\beta_c(\kappa_T)$, network B becomes capable of spreading the disease to network A, which now enters the epidemic phase, even for $\beta < \beta_c(\kappa_A)$. This matches the work done by Leicht and D'Souza [23], where a giant cluster forms consisting of nodes in both networks, even when the less intraconnected network is below its own percolation threshold. We plot the full phase diagram for both weakly and strongly coupled networks in Fig. 3, showing the disease-free phase, the mixed phase, the epidemic phase, and the transition between weakly and strongly coupled networks. The existence of this mixed phase is important in the real-world context of interacting networks, as the communities or systems that comprise the components are likely to be governed by different bodies. If two cities, for example, together form a weakly

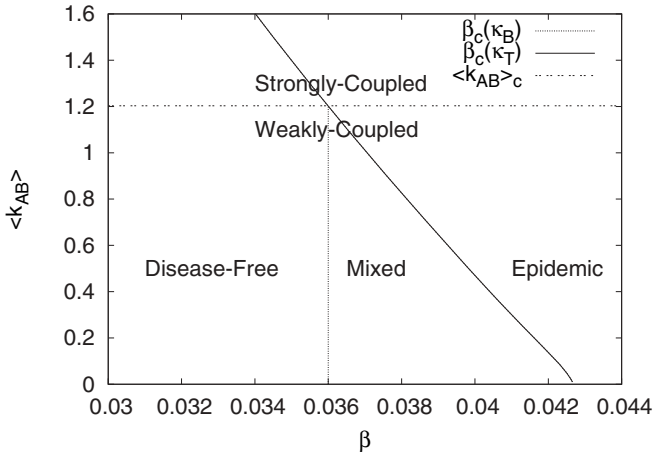

FIG. 3. The mixed phase disappears in the transition from weakly to strongly coupled network systems. Sample phase diagram for interacting network systems with $\langle k_A \rangle = 1.5$ and $\langle k_B \rangle = 6.0$ as a function of infection strength β and internetwork degree $\langle k_{AB} \rangle$, showing the two critical β and $\langle k_{AB} \rangle_c$. In the weakly coupled case, below $\beta_c(\kappa_B)$ no epidemic occurs. For $\beta_c(\kappa_B) < \beta < \beta_c(\kappa_T)$, there exists a mixed phase, where a finite fraction of network B becomes infected, but network A has only small infected clusters. Above $\beta_c(\kappa_T)$, an epidemic occurs across the entire network in both the weakly and strongly coupled cases. For this diagram, $N_A = N_B = 10^4$ and $t_r = 5$.

coupled network system, the more highly connected city can more safely disregard the links to, and response of, the less highly connected city, as the spread of the epidemic will depend on local parameters only.

We performed Monte Carlo simulations to verify this result. First, Fig. 4 shows infection densities at different β,

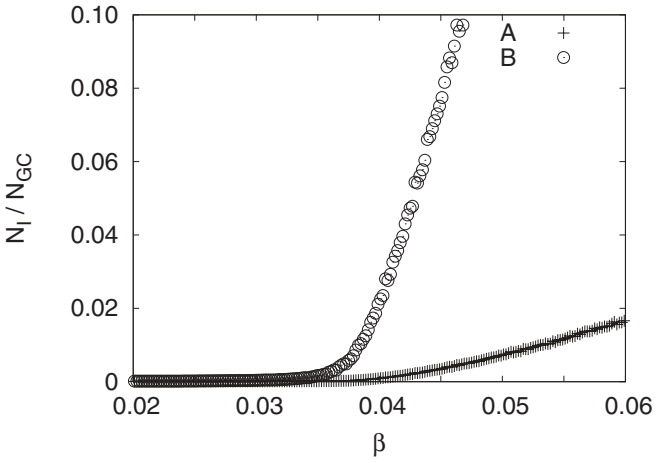

FIG. 4. In the mixed phase, the two networks have separate transition values. Ratio of total number of infected N_I in each network to the size of the giant cluster N_{GC} for two weakly coupled networks with $\langle k_A \rangle = 1.5$, $\langle k_B \rangle = 6.0$, and $\langle k_{AB} \rangle = 0.1$. The respective epidemic thresholds calculated from Eq. (1) are $\beta_c(\kappa_B) \approx 0.035$, $\beta_c(\kappa_T) \approx 0.0425$. The infection can be seen to become epidemic in network B well before it does in network A. The network of networks has $N_A = N_B = 10^4$ and $t_r = 5$.

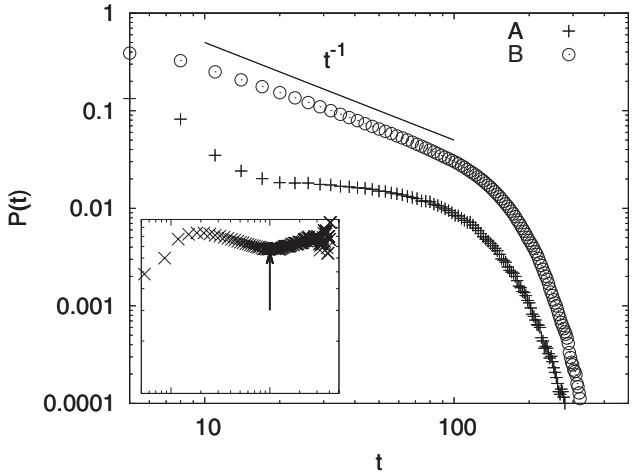

FIG. 5. Epidemics exhibit critical survival only in one network for weakly coupled networks. Infection survival probabilities $P(t)$ on the individual networks with internetwork connectivity $\langle k_{AB} \rangle = 0.1$ at $\beta = \beta_c(\kappa_B)$. The survival probability in network A (lower curve, with $+$), the less connected network, is a small fraction of that in network B (upper curve with ◯), the more connected network. The survival probability in network B falls off as t^{-1}, as expected of a system at criticality. Network A does not show a smooth decrease toward 0 typical of a network much below criticality. The inset shows the relative difference in the survival probabilities, $[P_B(t) - P_A(t)]/P(t)$. The arrow indicates the minimum difference between the two curves, after the initial increase. Network parameters are $\langle k_A \rangle = 1.5, \langle k_B \rangle = 6.0$, $N_A = N_B = 10^4$, and $t_r = 5$.

corresponding to a horizontal sweep across the phase diagram seen in Fig. 3 at $\langle k_{AB} \rangle = 0.1$. The epidemic spreading first occurs at $\beta_c(\kappa_B)$, where the disease enters the epidemic phase and spreads through network B, while the infection density in network A remains negligible. This mixed region, in agreement with our predictions, occurs in the region $\beta_c(\kappa_B) < \beta < \beta_c(\kappa_T)$. In this regime, network A plays no role in the spreading of the infection on network B. Above $\beta_c(\kappa_T)$, we see that the infection density in network A begins to rise, showing that the entire interconnected network system is now in the epidemic phase, as predicted.

For networks approaching the strongly coupled regime from below, the mixed phase is expected to be small and, thus, is difficult to identify from graphs such as the one in Fig. 4. We thus examine not only the infection densities but also the survival probability $P(t)$, which is the probability of an infection started from a single infected site being active at a time t. Equivalently and more accessible from public health records, the distribution of time spans of reported outbreaks can be used. At criticality, the probability of an infection started from a single infected site remaining active at a later time t is expected to scale as $P(t) \sim t^{-1}$ [32]. Figure 5 shows the survival probabilities of the networks comprising an interconnected with $\beta = \beta_c(\kappa_B)$ for both the strongly and weakly coupled cases. In both cases, network B exhibits the expected t^{-1} falloff in survivability with time that is expected of a system at criticality, indicating that network B is actually undergoing a phase transition at the disease-free/mixed phase line. In the weakly coupled case, however, the survival

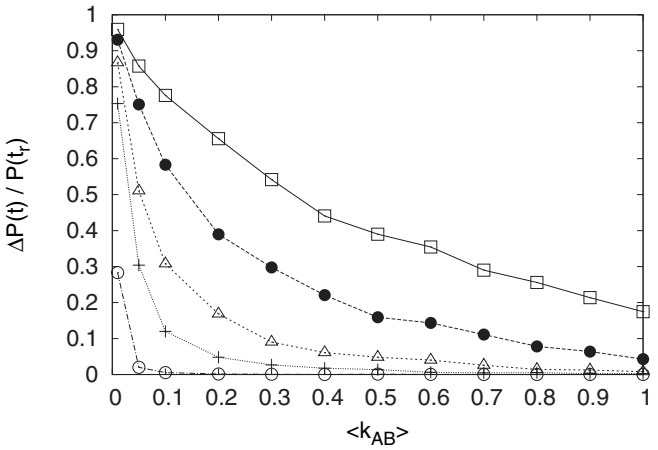

FIG. 6. Survival probability gap shows mixed phase boundaries. Fractional size of the minimum survival probability gap, $\Delta P(t)/P(t_r)$ (minimum distance between the two curves in Fig. 5 after the time t_r has passed) between two interacting ER networks with $\langle k_A\rangle = 1.5$ and $\langle k_B\rangle = 6.0$ at various infection strengths. From top to bottom, $\beta = \beta_c(\kappa_B) = 0.0358, 0.038, 0.04, 0.042$ and $\beta_c(\kappa_T) = 0.044$. The gap shrinks with increasing interaction and with increasing β, matching Fig. 3. At $\beta_c(\kappa_T)$, the survival probability is the same in both networks for all but $\langle k_{AB}\rangle = 0.01$, where it remains distinct due to finite-size effects. For all systems, $N_A = N_B = 10^4$ and $t_r = 5$.

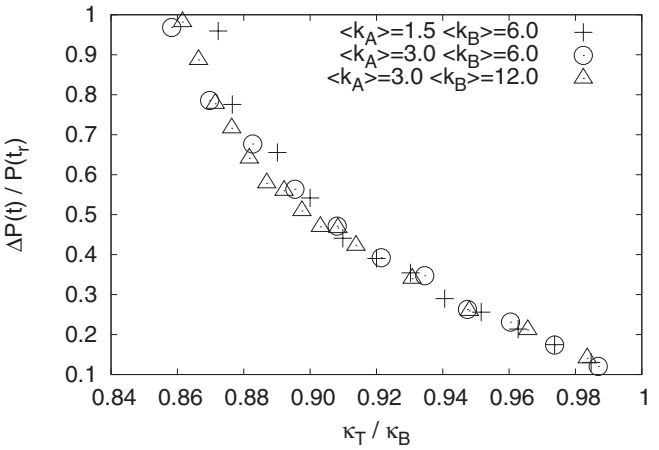

FIG. 7. Survival probability gap scaling is universal when rescaled by κ_B for different networks. The data collapse onto a single curve, showing this universal behavior. The ratio of κ_T to κ_B determines $\Delta P(t)$ along the disease-free/mixed phase border. For the network systems with $\langle k_B\rangle = 6.0$, $\beta = 0.0358$; for $\langle k_B\rangle = 12.0$, $\beta = 0.01725$. Both are $\beta_c(\kappa_B)$ of the respective systems. Again, for all systems, $N_A = N_B = 10^4$ and $t_r = 5$.

probability in network A does not fall off as expected, due to infrequent and nonepidemic instances of infections from network B. The slope of the survival probability for network A thus cannot be used directly to confirm when it enters the epidemic phase. However, if both networks are participating in an epidemic, the disease should be active in both networks at each time step. We thus introduce the survival probability gap (inset of Fig. 5), $\Delta P(t) = \min\left[\left[P_B(t) - P_A(t)\right]/P(t)\right]$, or the minimum relative difference in the likelihoods that each network will have any infected members (nodes in class i) present at time t. We use this quantity to measure the deviations of the survival probability in network A from the value that is obtained from a network at criticality (network B) and, thus, of how far away network A is from its own critical point.

In Fig. 6 we plot this survival gap at different β, equivalent to vertical slices across the phase diagram seen in Fig. 3. We see that when $\langle k_{AB}\rangle$ or β is increased to move outside the expected mixed phase region and into the epidemic phase, $\Delta P(t)$ goes to zero. In other words, at the mixed/epidemic phase line, network A is behaving identically to a network known to be at criticality and, thus, is itself critical along that line. This confirms the assertion that there can be a gap in survival probability only when one network is in the epidemic phase and the other is not, i.e., in the mixed phase. Thus, a nonzero survival probability gap can serve as a good predictor for the presence of the mixed phase.

Last, we addressed the question of universality under different values of inter- and intranetwork degree, finding that along the disease-free/mixed phase transition line, the behavior of networks with different κ is universal under appropriate scaling. Figure 7 shows three different networks, all with $\langle k_B\rangle > \langle k_A\rangle$ and $\beta = \beta_c(\kappa_B)$. Rescaling the survival probability gaps by $P(t_r)$ and plotting versus κ_T/κ_B instead of κ_T directly, the curves collapse, showing $\Delta P(t)$, and, thus, the mixed phase, disappears uniformly as the network approaches the strongly coupled regime ($\kappa_T > \kappa_B$ and $\langle k_{AB}\rangle > \langle k_{AB}\rangle_c$.) Near the critical point, $\Delta P(t) \sim [(\kappa_T - \kappa_B)/\kappa_B]^{-1}$ (fit not shown). This identical behavior implies survival probabilities in networks could be used as as a measure of network connectivities near criticality, as the latter may be difficult to obtain for social and biological networks, whereas information on the duration of an epidemic outbreak in various communities [from which $P(t)$ can be estimated] is likely to be recorded. In addition, the survival probability gap persists well beyond $\langle k_{AB}\rangle = 1$ for appropriate $\langle k_A\rangle$ and $\langle k_B\rangle$. For the system with $\langle k_A\rangle = 3.0$ and $\langle k_B\rangle = 12.0$, $\langle k_{AB}\rangle_c \approx 2.47$. Even when every node in network A is connected to two or more nodes in network B, there can still be an order-of-magnitude difference at minimum in the likelihood of finding the disease active in the two networks; the mixed phase region is not confined to small $\langle k_{AB}\rangle$ only.

V. CONCLUSIONS

In summary, we introduced two classes for interconnected network systems, strongly coupled and weakly coupled, and studied the behavior of epidemics on them. In strongly coupled network systems, epidemics occur always across the entire interacting network system, with the presence of interconnections enhancing epidemic spreading. In weakly coupled network systems, a mixed phase exists where epidemics do not always occur across the full interconnected network system, and interconnections affect only epidemic spreading across the less intraconnected network. We demonstrated the boundaries and behavior of the mixed phase numerically as well as analytically. Proper analysis of which groups of communities

comprise strongly or weakly coupled systems could inform public policy and highlight the necessity of cooperation between different governing bodies or provide information about the epidemic danger of increasing interaction between human and animal populations.

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