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PAPER

Epidemic mitigation via awareness propagation in communication networks: the role of time scales

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Abstract

The participation of individuals in multi-layer networks allows for feedback between network layers, opening new possibilities to mitigate epidemic spreading. For instance, the spread of a biological disease such as Ebola in a physical contact network may trigger the propagation of the information related to this disease in a communication network, e.g. an online social network. The information propagated in the communication network may increase the awareness of some individuals, resulting in them avoiding contact with their infected neighbors in the physical contact network, which might protect the population from the infection. In this work, we aim to understand how the time scale γ of the information propagation (speed that information is spread and forgotten) in the communication network relative to that of the epidemic spread (speed that an epidemic is spread and cured) in the physical contact network influences such mitigation using awareness information. We begin by proposing a model of the interaction between information propagation and epidemic spread, taking into account the relative time scale γ. We analytically derive the average fraction of infected nodes in the meta-stable state for this model (i) by developing an individual-based mean-field approximation (IBMFA) method and (ii) by extending the microscopic Markov chain approach (MMCA). We show that when the time scale γ of the information spread relative to the epidemic spread is large, our IBMFA approximation is better compared to MMCA near the epidemic threshold, whereas MMCA performs better when the prevalence of the epidemic is high. Furthermore, we find that an optimal mitigation exists that leads to a minimal fraction of infected nodes. The optimal mitigation is achieved at a non-trivial relative time scale γ, which depends on the rate at which an infected individual becomes aware. Contrary to our intuition, information spread too fast in the communication network could reduce the mitigation effect. Finally, our finding has been validated in the real-world two-layer network obtained from the location-based social network Brightkite.

1. Introduction

Epidemic spreading models in complex networks have been extensively studied in order to understand the spreading process of epidemics, worms, failures and information [1–3]. Significant efforts have been devoted to understanding the epidemic spread in a single network, especially the influence of the network topology [4–7]. More than one disease could coexist and interact [8, 9]. However, real-world networks are not isolated but instead are interconnected and interdependent on each other [10–12]. The function and behavior of nodes not only depend on the network they locate in, but also rely on other networks. Epidemic spreading as well as other stochastic processes, such as opinion interaction in interconnected and interdependent networks, have been explored extensively since 2010 [13–20].

When the nodes of all networks that depend on each other represent the same set of entities, such an interdependent network is also called a multi-layer network or multiplex, representing different types of relations among the same set of nodes [21–23]. For example, individuals could participate in a physical contact network with friends, family and coworkers, while they may also join a communication network such as the online social networks Facebook or Twitter [24]. The multi-layer network allows interactions or feedback between processes on different network layers. When a flu spreads in the physical contact network, people who are aware they have the flu may post this information online, possibly making some of his/her online friends aware of the epidemic, who then further share this information with their friends. When people are made aware of the flu from the communication network, they could take action to prevent the infection, e.g. decreasing physical contact with others and wearing masks. The awareness information about the epidemic propagated in the communication network could possibly mitigate the epidemic spreading in the physical contact network, as observed in [25, 26] in 2013.

In this work, we aim to further understand how to achieve an optimal mitigation effect when using awareness information. We specifically explore the role of the relative time scale of the information propagation to that of the epidemic spread. People tend to post, read, as well as forget, the news about an epidemic in an online social network everyday, whereas friends call each other to share the news of an epidemic, e.g. once per week(s) via mobile phone contact. Would the relatively fast time scale of an online social network be beneficial for the mitigation compared to that of the mobile phone contact network? We investigate the influence of the time scale γ of the information dynamics relative to the epidemic dynamics on the mitigation effect, quantified by the average fraction of infected nodes in the meta-stable state. We begin by developing a model to describe the interaction between the information and epidemic spread, taking into account the relative time scale of the information propagation γ. Next, we perform simulations and develop two analytical methods to unveil the relation between the mitigation effect, i.e. average fraction of infected nodes and the relative time scale γ. Finally, our findings regarding the effect of the relative time scale are validated on a real-world physical contact-communication network. The influence of other factors, such as the network structure, including size, density and probability that two nodes are linked in both layers and extremal scenarios on the mitigation effect, are discussed in the appendix.

2. Interacting epidemic and awareness spread model

The susceptible–infected–susceptible (SIS) model is one of the most studied epidemic spreading models. In the SIS model, the state of each node at any time t is a Bernoulli random variable, where $X_i(t) = 0$ if node i is susceptible and $X_i(t) = 1$ if it is infected. The recovery (curing) process of each infected node is an independent Poisson process with a recovery rate δ. Each infected agent infects each of its susceptible neighbors with a rate β, which is also an independent Poisson process. The ratio $\tau \triangleq \beta/\delta$ is the effective infection rate. A phase transition has been observed around a critical point τ_c in a single network. When $\tau > \tau_c$, a non-zero fraction of agents will be infected in the meta-stable state, whereas if $\tau < \tau_c$, infection rapidly disappears [27, 28].

We consider a two-layer network which describes two types of relations along the same set of individuals. The bottom layer is a physical contact network, in which an epidemic spreads according to the SIS model. The infection rate along each link is β_2 and the recovery rate of each node is δ_2. The upper layer is a communication network in which the awareness information propagates in the same way as the SIS model. An aware node informs each of its unaware neighbors to become aware with rate β_1 whereas an aware node becomes unaware with rate δ_1. We call this awareness spreading process the unaware–aware–unaware (UAU) process, analogous with the SIS model in real epidemics.

The relative time scale of UAU information propagation with respect to SIS epidemic spread can be controlled by scaling both the spreading rate and the recovery rate of the UAU process as $\gamma * \beta_1$ and $\gamma * \delta_1$ respectively. The relative time scale of UAU is thus characterized by the scaling parameter γ. The larger the time scale γ is, the faster the UAU process is, which represents the case when individuals are more frequently involved in the information propagation in the communication network.

The two spreading processes, SIS and UAU, on the two layers respectively interact with each other. When an individual is infected, he or she becomes aware of the epidemic with rate γ^{ϵ}. An aware individual is alert to the epidemic to the extent that (s)he would like to inform his/her friends via a communication network, such as by posting a message or calling friends. Specifically, we assume that the time for an infected node just to become aware of the epidemic is an exponential random variable with rate γ^{ϵ}. This process of becoming aware of the epidemic due to the node itself getting infected is called the 'injection' of information from a physical contact layer to the communication network layer. The rate of this injection is intuitively related to the relative time scale γ of the information propagation: frequent usage of a communication network allows fast injection of information. For example, if people use an online social network everyday, the delay of the information injection

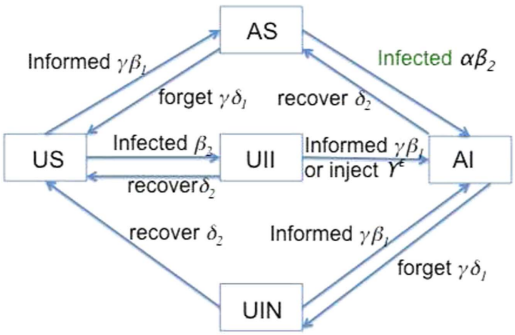

Figure 1. The state transition diagram of an individual in the IEAS model. The self transition to each state is not included. Activities along the links that could trigger the transition of states are: infected (a node gets infected by a neighbor in the physical contact network; the infection rate depends on whether the node is aware of the disease or not; the rate indicated is the infection rate per infected neighbor), recover, informed (a node becomes aware due to the contagion of an aware neighbor in the communication network; the rate is the contagion rate per aware neighbor), forget (an aware node becomes unaware of the disease), inject (a node becomes aware because of its own infection of the disease).

is smaller compared to the case when people use another communication network once per week. Moreover, frequent usage of a communication network implies significant social impact of the network, which motivates possibly even faster injection of information. Without loss of generality, we consider here the injection rate as a polynomial function γ^ϵ of the time scale γ. Note that each time a node gets infected, it may introduce maximally one injection. An infection may happen only if (a) the injection happens before the node recovers from the epidemic, i.e. the injection delay is smaller than the recovery time for the node to become susceptible; (b) the node is unaware of the epidemic at the moment when it gets infected; and (c) the injection happens before the node becomes aware due to any of its aware neighbors.

Conversely, when an individual is aware of the epidemic, (s)he would take precautions which reduces the infection rate of this individual to $\alpha * \beta_2$, where $0 < \alpha < 1$.

The state transition diagram of our Makrovian interacting epidemic and awareness spread (IEAS) model is shown in figure 1. Our model differs from the one proposed in [26]: (i) We introduce the relative time scale γ of the information spread in the communication network with respect to the epidemic spread in the physical contact network. (ii) Our model is more generalized in the sense that it may take time (e.g. injection delay) for an infected node to become aware (post information) in contrast to immediately becoming aware of getting infected, as assumed in [26]. The special case in our model where each node becomes immediately aware after getting infected is studied in section A.4 using two analytical approaches. (iii) In our model, the recovery process for an aware node to become unaware starts immediately once the node becomes aware, which is independent of whether the node is infected or not, whereas [26] assumes that an aware node can start the recovery process to become unaware only after the node becomes susceptible. Our model is driven by the fact that an individual may lose the awareness of the epidemic and stop informing others via the communication network after some time, even though (s)he is still infected, because (s)he might get bored of and/or does not have the energy to continuously inform others.

3. Simulations

3.1. Two-layer network construction

In this paper, we consider two-layer networks where both layers are generated from the same network model—either the Erdős–Rényi random network model or the scale-free network model.

The Erdős–Rényi random network is one of the most studied random network models that allows many problems to be treated analytically [29, 30]. To generate an Erdős–Rényi random network with N nodes and average degree $E[D]$, we start with N nodes and place each link between two nodes that are chosen at random among the N nodes until a total number $L = \frac{N * E[D]}{2}$ of links have been placed. All the links are bidirectional, which means that epidemics could spread in both directions of a link. In this paper, we chose $N = 1000$, $E[D] = 4$ for Erdős–Rényi random networks. Larger networks, e.g. $N = 10000$ and diverse average degree values have also been analyzed with similar results. We focus on networks of size $N = 1000$ to illustrate our results because of the high computational complexity of simulating interacting processes at different time scales, as discussed in section 3.2. Erdős–Rényi random networks are characterized by a Poisson degree distribution, $Pr[D = k] = \frac{(Np)^k e^{-Np}}{k!}$, where D is the degree of a random node in the network, and the link density is $p = \frac{E[D]}{N-1}$.

We use the configuration model to generate scale-free networks having a power-law degree distribution $Pr[D = k] = ck^{-\lambda}$ as observed in many real-world networks [31–34] . Firstly, we generate a degree sequence for N nodes following the power-law degree distribution $Pr[D = k] = ck^{-\lambda}$. Given the degree sequence, we generate a random network according to the configuration model: we assign each node as many 'stubs' as its degree; afterwards, we randomly choose two spare stubs from two different nodes which are not yet connected, and connect them with a link until all stubs are connected. In this paper, we consider $N = 1000$ and $\lambda = 2.5$.

Besides the degree distribution of the two layers, the overlap in links between the two layers may affect the epidemic spreading [35, 36]. Hence, we control the overlap extent when generating the multi-layer networks. In order to generate a two-layer ER network with a fraction ϕ of overlapping links, we first generate $\phi * L$ random links that exist on both layers, then randomly generate the rest of the links on the two layers separately under the constraint that links which exist in one layer do not appear in the other layer. We consider the two extreme cases, i.e. $\phi = 0$ and $\phi = 1$. A two-layer SF network with overlap $\phi = 1$ can be constructed by generating a one-layer scale-free network with the configuration model, and all the links are copied to the other layer. A two-layer SF network with overlap $\phi = 0$ can be obtained by generating the degree sequence for each layer independently and afterwards constructing each layer using the configuration model. Since the scale-free networks are sparse, the two independently generated layers hardly overlap, leading to $\phi = 0$.

3.2. Simulating the IEAS model

For each simulation with a given set of parameters with respect to the two spreading processes and the multi-layer network structure, we generate 200 realizations. Within each realization, we firstly construct a two-layer network with the specified network model ER or SF and the overlap percentage $\phi = 0$ or $\phi = 1$. Initially, 10% of the nodes are randomly selected to get infected. Afterwards, we simulate the two continuous time interacting spreading processes of the epidemic and the awareness in discrete time with time steps of small intervals $\Delta t = 0.01$. Within each time step, the probability that a node gets infected by an infected neighbor is $\beta_2 * \Delta t$ if the node is unaware, $\alpha * \beta_2 * \Delta t$ if it is aware, and the probability that an infected node recovers is $\delta_2 * \Delta t$. A similar situation holds for the UAU process. Once an unaware node gets infected, this node has a probability $\gamma^\epsilon * \Delta t$ in each following step to become aware due to the injection as long as the node has not recovered to be susceptible and has not yet become aware due to its aware neighbors in the UAU process. The two interacting processes continue until both reach their meta-stable states, where the fraction of infected nodes and the fraction of aware nodes remain constant. For each set of parameters, the fraction of infected nodes and aware nodes in the meta-stable state are obtained as the average over all the realizations.

We focus on the following parameters with respect to the IEAS model throughout the paper to illustrate our findings, with $\beta_1 = 0.3$, $\delta_1 = 0.6$, $\delta_2 = 1.0$, $\alpha = 0.2$ and β_2 being the control parameter in the range $[0, 1]$. Note that many other parameter sets have also been tested and lead to similar observations. The relative awareness spreading rate $\frac{\beta_2}{\delta_2} > \tau_c$ is chosen here to be above the critical spreading threshold of the communication network layer such that the epidemic spreading can be possibly reduced via awareness. Various values of the time scales γ will be considered within the range $[0.125, 8]$ and the injection rate scaling ϵ will be varied within $[0.5, 3]$.

The complexity of simulating interacting processes operating at different time scales is significantly higher than that of simulating a single process. Our simulations contain three types of processes at different time scales: the SIS epidemic spread in the communication network layer, the UAU information propagation in the social network layer and the information injection between the two layers. Take the case that $\gamma \ll 1$ as an example, i.e. the information propagation is far slower than the epidemic spread. The sampling time step Δt has to be selected based on the fastest dynamics, i.e. the epidemic spread, such that within each time step no multiple events happen. The time for the simulation to converge to the meta-stable state is long due to the slow dynamics in the information propagation and the possibly even slower information injection between the two layers.

4. Theoretical analysis: individual-based mean field approximation

The probability that each node is infected by the SIS epidemic at any time in a single network as well as in interconnected networks has been derived via the N-intertwined mean-field approximation, which takes into account the network structure [16, 37, 38]. In order to take not only the two-layer network structure into account but also the interacting epidemic and awareness spread, we develop here the individual-based mean field approximation (IBMFA) to derive the probability that each node is infected or aware in our IEAS model. Recall that in the SIS epidemic spread, the rate to infect an unaware and aware node is β_2 and $\alpha * \beta_2$ respectively, and the recovery rate is δ_2. In the UAU information propagation, the spreading rate is β_1 and the recovery rate is δ_1. The rate for an infected node to become aware is γ^ϵ. We thus suggest the following IBMFA equations:

$$\frac{du_i(t)}{dt} = -\gamma\delta_1 u_i(t) + (1 - u_i(t))\gamma\beta_1 \sum_{j=1}^{N} a_{ji} u_j(t) + \gamma^{\epsilon} v_i^{\mathrm{II}}(t) \tag{1a}$$

$$\frac{dv_i^{\mathrm{S}}(t)}{dt} = -v_i^{\mathrm{S}}(t)\left([u_i(t)\alpha + (1 - u_i(t))]\beta_2 \sum_{j=1}^{N} b_{ji}(v_j^{\mathrm{II}}(t) + v_j^{\mathrm{IN}}(t))\right) + \delta_2(v_i^{\mathrm{II}}(t) + v_i^{\mathrm{IN}}(t)) \tag{1b}$$

$$\frac{dv_i^{\mathrm{II}}(t)}{dt} = -v_i^{\mathrm{II}}(t)\left(\delta_2 + \gamma^{\epsilon} + \gamma\beta_1 \sum_{j=1}^{N} a_{ji} u_j(t)\right) + v_i^{\mathrm{S}}(t)(1 - u_i(t))\left(\beta_2 \sum_{j=1}^{N} b_{ji}(v_j^{\mathrm{II}}(t) + v_j^{\mathrm{IN}}(t))\right), \tag{1c}$$

where $u_i(t)$ is the probability that node i is aware of the epidemic at time t, $v_i^{\mathrm{S}}(t)$ is the probability that node i is susceptible at time t, $v_i^{\mathrm{II}}(t)$ is the probability that node i is infected and could inject information to the communication network, $v_i^{\mathrm{IN}}(t)$ is the probability that node i is infected but could not inject the information. Also, $a_{ij} = 1$ if node i and j are connected in the communication network layer, otherwise $a_{ij} = 0$, similar to the physical contact layer, $b_{ij} = 1$ if there is a link between node i and j, otherwise $b_{ij} = 0$. Note that,

$$v_i^{\mathrm{S}}(t) + v_i^{\mathrm{II}}(t) + v_i^{\mathrm{IN}}(t) = 1. \tag{1d}$$

The time derivative of the probability $u_i(t)$ for a node being aware of the epidemic is determined by three processes: (a) when the node is aware of the epidemic, it recovers to unaware with rate $\gamma\delta_1$; (b) when the node is unaware, it gets aware by any of its aware neighbors in the communication network with rate $\gamma\beta_1$; and (c) when the node is in state II and it injects the awareness information with rate γ^{ϵ}.

The derivative of the probability $v_i^{S}(t)$ that a node is susceptible depends on the following two competing processes: (a) when the node is susceptible, it gets infected by any of its infected neighbors in the physical contact network with rate β_2 if the node is unaware of the epidemic and with rate $\alpha * \beta_2$ if this node is aware; and (b) if the node is infected, i.e. either is state II or IN, it recovers to susceptible with rate δ_2.

Similarly, the probability $v_i^{\mathrm{II}}(t)$ that a node is infected and is able to inject the information to the communication network decreases because of the following processes: when the node is in state II, (a) it recovers to susceptible, or (b) injects the awareness information, or (c) the node becomes aware because of the information propagation from its aware neighbors. The probability $v_i^{\mathrm{II}}(t)$ increases when the node is susceptible and unaware and it gets infected by any of its infected neighbors in the physical contact network.

We are interested in the meta-stable state when $\frac{du_i}{dt} = 0$, $\frac{dv_i}{dt} = 0$, $u_{i\infty} = \lim_{t\to\infty} u_i(t)$ and $v_{i\infty} = \lim_{t\to\infty} v_i(t)$. The exact steady state is the susceptible and unaware state for all the nodes, which is the only absorbing state of the Makrovian process IEAS. However, this absorbing state will be reached within an unrealistically long time for realistic sizes of networks [39]. We are thus interested in the meta-stable state in which the system stays for a long time and which will be reached faster, and better characterizes real epidemics. With $\frac{du_i}{dt} = 0$, $\frac{dv_i^{\mathrm{S}}(t)}{dt} = 0$, and $\frac{dv_i^{\mathrm{II}}(t)}{dt} = 0$ we could obtain a trivial solution $u_{i\infty} = 0$, $v_{i\infty}^{\mathrm{S}} = 1, v_{i\infty}^{\mathrm{II}} = 0$ and a possibly positive solution representing the meta-stable state.

In general, mean-field approximations assume the uncorrelation of random variables [40]. The IBMFA is derived based on the assumption that the infection states (infected or susceptible) of two neighboring nodes in the physical contact network are uncorrelated, the awareness states (aware or unaware) of two neighboring nodes in the communication network are uncorrelated, and the infection state and the awareness state of the same node are uncorrelated, although the injection has been taken into account. These types of correlations, especially the correlation between the infection state and the awareness state of the same node, do exist, which explains why IBMFA is not precise.

5. Theoretical analysis: microscopic Markov chain approach

The microscopic Markov chain approach (MMCA) was proposed in [41, 42] and has been applied to the interacting processes on two-layer networks proposed in [26]. Here we derive the MMCA for our IEAS model. Later, we will compare MMCA and IBMFA, the two seemingly most advanced analytical approaches so far, with numerical simulations.

The MMCA examines the discrete time evolution of the probability $p_i^{\mathrm{US}}(t)$, $p_i^{\mathrm{UIN}}(t)$, $p_i^{\mathrm{UII}}(t)$, $p_i^{\mathrm{AS}}(t)$, $p_i^{\mathrm{AI}}(t)$ that each node i is in each of the five possible states respectively: unaware and susceptible (US), unaware, infected and cannot inject the aware information (UIN), unaware, infected and able to inject the aware information (UII), aware and susceptible (AS), and aware and infected (AI) when there is no need to inject the information since the node is already aware of the information.

Within any time step t of interval Δt, we define the probability that node i is NOT informed by any neighbor in the communication network as $r_i(t)$, the probability that an unaware node i is NOT infected by any neighbor in the physical contact network as $q_i^{U}(t)$ and the probability that an aware node i is NOT infected by any neighbor as $q_i^{A}(t)$ and they follow:

$$r_i(t) = \prod_j (1 - a_{ji} p_j^{\mathrm{A}}(t)\beta_1^*)$$
$$q_i^{\mathrm{U}}(t) = \prod_j (1 - b_{ji} p_j^{\mathrm{I}}(t)\beta_2^*)$$
$$q_i^{\mathrm{A}}(t) = \prod_j (1 - b_{ji} p_j^{\mathrm{I}}(t)\alpha\beta_2^*), \quad (2)$$

where $p_j^{\mathrm{A}}(t) = p_j^{\mathrm{AS}}(t) + p_j^{\mathrm{AI}}(t)$ is the probability that node j is aware at time t, $p_j^{\mathrm{I}}(t) = p_j^{\mathrm{UII}}(t) + p_j^{\mathrm{UIN}}(t) + p_j^{\mathrm{AI}}(t)$ is the probability that node j is infected at time t, the probability that a node gets informed by an aware neighbor within a time step of interval Δt follows $\beta_1^* = \gamma\beta_1 \cdot \Delta t$ and similarly, we have $\delta_1^* = \gamma\delta_1 \cdot \Delta t$, $\beta_2^* = \beta_2 \cdot \Delta t$, $\delta_2^* = \delta_2 \cdot \Delta t$. The time interval Δt should be small so that this discrete-time approach well approximates the continuous time IEAS model. For consistency with the simulations, we use the interval time $\Delta t = 0.01$.

The MMCA equations describe the time evolution of the probability that each node is in each of the five possible states[5]:

$$p_i^{\mathrm{US}}(t+1) = (p_i^{\mathrm{UII}}(t) + p_i^{\mathrm{UIN}}(t))r_i(t)\delta_2^* + p_i^{\mathrm{AI}}(t)\delta_1^*\delta_2^* + p_i^{\mathrm{US}}(t)r_i(t)q_i^{\mathrm{U}}(t) + p_i^{\mathrm{AS}}(t)\delta_1^* q_i^{\mathrm{A}}(t) \quad (3a)$$
$$p_i^{\mathrm{UIN}}(t+1) = p_i^{\mathrm{UIN}}(t)r_i(t)(1-\delta_2^*) + p_i^{\mathrm{AI}}(t)\delta_1^*(1-\delta_2^*) + p_i^{\mathrm{AS}}(t)\delta_1^*(1-q_i^{\mathrm{A}}(t)) \quad (3b)$$
$$p_i^{\mathrm{UII}}(t+1) = p_i^{\mathrm{UII}}(t)r_i(t)(1-\delta_2^*)(1-\gamma^{\epsilon *}) + p_i^{\mathrm{US}}(t)r_i(1-q_i^{\mathrm{U}}(t))(1-\gamma^{\epsilon *}) \quad (3c)$$
$$p_i^{\mathrm{AS}}(t+1) = (p_i^{\mathrm{UII}}(t) + p_i^{\mathrm{UIN}}(t))(1-r_i(t))\delta_2^* + p_i^{\mathrm{AI}}(t)(1-\delta_1^*)\delta_2^* + p_i^{\mathrm{US}}(t)(1-r_i(t))q_i^{\mathrm{U}}(t) + p_i^{\mathrm{AS}}(t)(1-\delta_1^*)q_i^{\mathrm{A}}(t) \quad (3d)$$
$$p_i^{\mathrm{AI}}(t+1) = p_i^{\mathrm{UII}}(t)[(1-r_i(t))(1-\delta_2^*) + r_i(t)(1-\delta_2^*)\gamma^{\epsilon *}] + p_i^{\mathrm{UIN}}(t)(1-r_i(t))(1-\delta_2^*) + p_i^{\mathrm{AI}}(t)(1-\delta_1^*)(1-\delta_2^*) + p_i^{\mathrm{US}}(t)[(1-r_i(t))(1-q_i^{\mathrm{U}}(t)) + r_i(t)(1-q_i^{\mathrm{U}}(t))\gamma^{\epsilon *}] + p_i^{\mathrm{AS}}(t)(1-\delta_1^*)(1-q_i^{\mathrm{A}}(t)), \quad (3e)$$

where $\gamma^{\epsilon *} = \gamma^{\epsilon} \cdot \Delta t$ is the probability that the awareness information is injected from the physical contact network to the communication network layer within a time step, i.e. the node becomes aware because it gets infected. The normalization condition should be satisfied at any time t for any node i,

$$p_i^{\mathrm{US}}(t) + p_i^{\mathrm{UIN}}(t) + p_i^{\mathrm{UII}}(t) + p_i^{\mathrm{AS}}(t) + p_i^{\mathrm{AI}}(t) = 1. \quad (4)$$

In the meta-stable state, the probability that each node stays in each state remains the same over time, e.g. $p_i^{\mathrm{US}}(t+1) = p_i^{\mathrm{US}}(t) = p_i^{\mathrm{US}}(\infty)$. The meta-stable state fraction of aware nodes ρ_1 and fraction of infected nodes ρ_2 can be derived as:

$$\rho_1 = \frac{\sum_{i=1}^{N}(p_i^{\mathrm{AS}}(\infty) + p_i^{\mathrm{AI}}(\infty))}{N},$$
$$\rho_2 = \frac{\sum_{i=1}^{N}(p_i^{\mathrm{UIN}}(\infty) + p_i^{\mathrm{UII}}(\infty) + p_i^{\mathrm{AI}}(\infty))}{N}. \quad (5)$$

6. Simulation, IBMFA and MMCA comparison

We compare the average fraction of infected nodes ρ_2 obtained by IBMFA, MMCA and simulations, respectively, to estimate the precision of these two analytical approaches. Figures 2 and 3 show that the two theoretical approaches relatively well approximate the simulation results. Moreover, IBMFA approximates the simulation results slightly better around the epidemic threshold. For the infection rate β_2 above the epidemic threshold, MMCA approximates the simulations better than IBMFA when γ is small and IBMFA approximates the simulations better as γ increases. These observations are more evident in two-layer ER networks and in larger networks, e.g. $N = 10000$, as shown in the appendix (figures A1 and A2). Cai *et al* in [43] already noticed the weak performance of MMCA around the epidemic threshold. Hence, IBMFA and MMCA may serve as complimentary approaches.

In the following sections, we will use the simulation results for discussions instead of the two approximations.

[5] In the MMCA approach, we consider that once a node gets infected in a time step, there is a chance that it injects the information starting from this time step instead of the next time step. In this case, a large injection rate γ^{ϵ} corresponds to the immediate injection scenario as assumed in [25, 26].

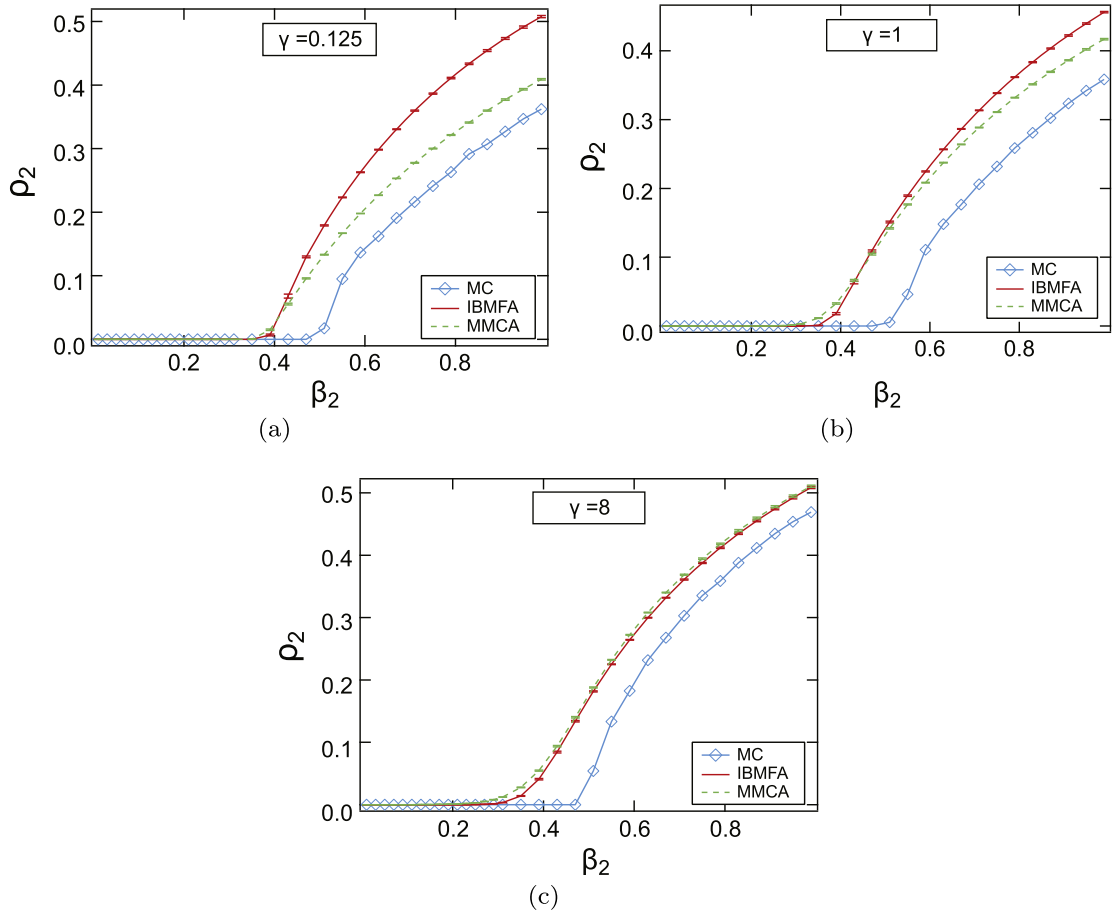

Figure 2. Comparison of the average fraction of infected nodes in the meta-stable state obtained by IBMFA, MMCA and simulations (MC), respectively, in two-layer Erdős–Rényi random networks. The injection rate is γ^{ϵ} with $\epsilon = 2$. The relative time scale in the communication network layer is: (a) $\gamma = 0.125$, (b) $\gamma = 1$ and (c) $\gamma = 8$. The parameters chosen are: $\beta_1 = 0.3$, $\delta_1 = 0.6$, $\delta_2 = 1.0$ and $\alpha = 0.2$. The network size is $N = 1000$. In each Erdős–Rényi random network, the average degree is $E[D] = 4$. The overlap between two network layers is $\phi = 1$. Results are averaged over 100 realizations for the IBMFA and the MMCA, and 200 realizations for the simulations.

7. Effect of time scale γ

In this section, we explore how time scale γ influences the mitigation effect, i.e. the fraction of infected nodes $\rho_2 = \frac{\sum_{i=1}^{N} v_{i\infty}}{N}$ in the meta-stable state.

As seen in figure 4, the average fraction of infected nodes in the meta-stable state can indeed be significantly reduced with the help of awareness information compared to the case when there is no awareness information propagated (see the dotted line, the so-called upper bound). This upper bound corresponds to the case when people do not use the social communication network. Moreover, the effect of γ is non-trivial. When $\epsilon = 1$, the fraction of infected nodes decreases monotonically with decreasing γ for a given β_2, which implies that a smaller γ better mitigates the epidemic. However, this is not the case when $\epsilon = 2$, where there seems to be an optimal γ that mitigates the epidemic spreading, but determining its value is not straightforward.

Hence, we explore further, for a given β_2, which relative time scale γ of information propagation better mitigates the epidemic for various values of ϵ. We consider the specific case of $\beta_2 = 1$ when the effect of γ differs more evidently, as suggested in figure 4. Figure 5 suggests the existence of a non-trivial optimal γ that minimises the average fraction of infected nodes for a given ϵ. For $\epsilon = 2$ it seems that the optimal γ is close to 0.5 while for $\epsilon = 3$, the optimal γ is close to 1. We observe similar results in two-layer ER networks.

We would like to understand how and why the optimal γ changes with ϵ, i.e. with the injection rate γ^{ϵ}. This would provide essential insight to the question: operating at which time scale could the information spread best mitigate the epidemic for a given ϵ that characterizes the relation between the injection rate and the time scale? We find (figure 5) that the optimal γ tends to decrease as ϵ decreases, even though the precision of the optimal γ is limited here due to the complexity of simulating interacting processes operating at different time scales, as discussed in section 3.2. The same trend can be captured by both analytical approximations IBMFA and MMCA. Furthermore, we explain this phenomenon using analytical and physical interpretations. As shown in

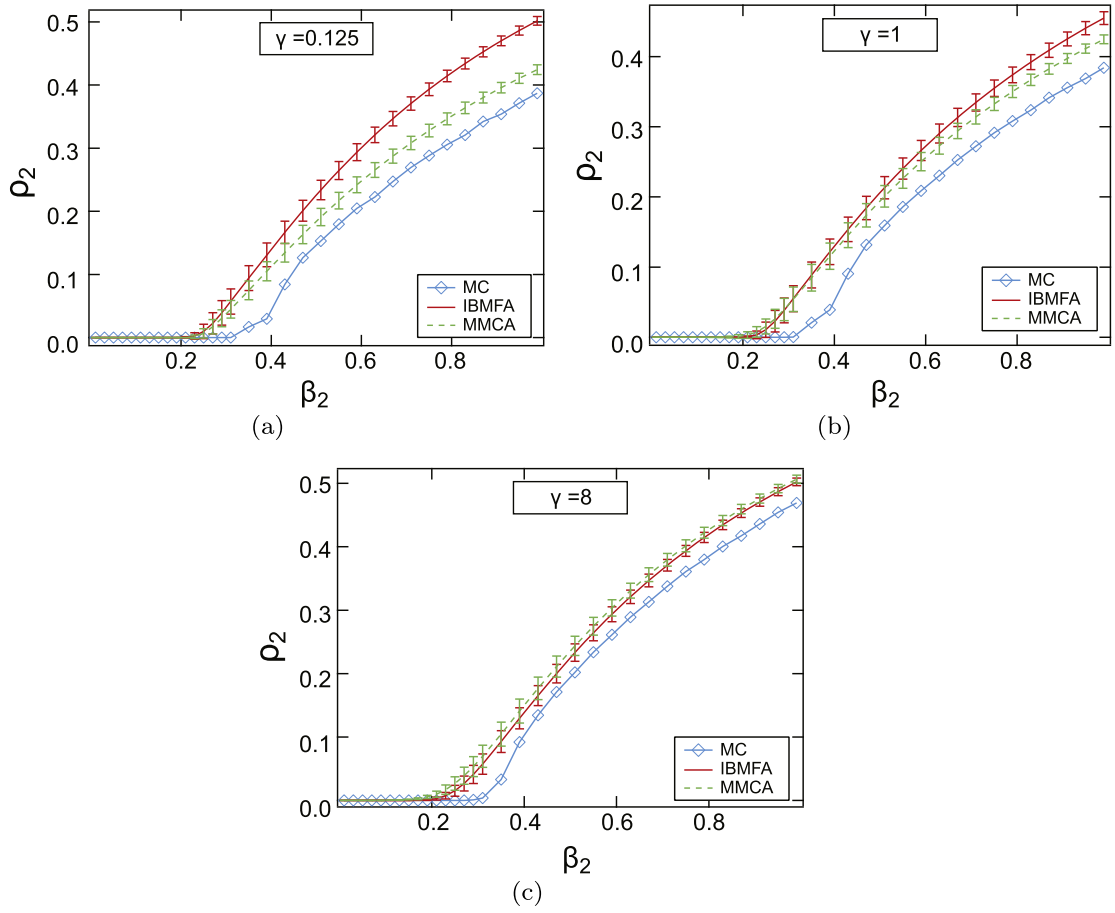

Figure 3. Comparison of the average fraction of infected nodes in the meta-stable state obtained by IBMFA, MMCA and simulations (MC), respectively, in two-layer scale-free networks. The injection rate is γ^{ϵ} with $\epsilon = 2$. The relative time scale in the communication network layer is: (a) $\gamma = 0.125$, (b) $\gamma = 1$ and (c) $\gamma = 8$. The parameters chosen are: $\beta_1 = 0.3$, $\delta_1 = 0.6$, $\delta_2 = 1.0$ and $\alpha = 0.2$. The network size is $N = 1000$. In each scale-free network, the exponent for the power-law degree distribution is $\lambda = 2.5$. The overlap between two network layers is $\phi = 1$. Results are averaged over 100 realizations for the IBMFA and MMCA, and 200 realizations for the simulations.

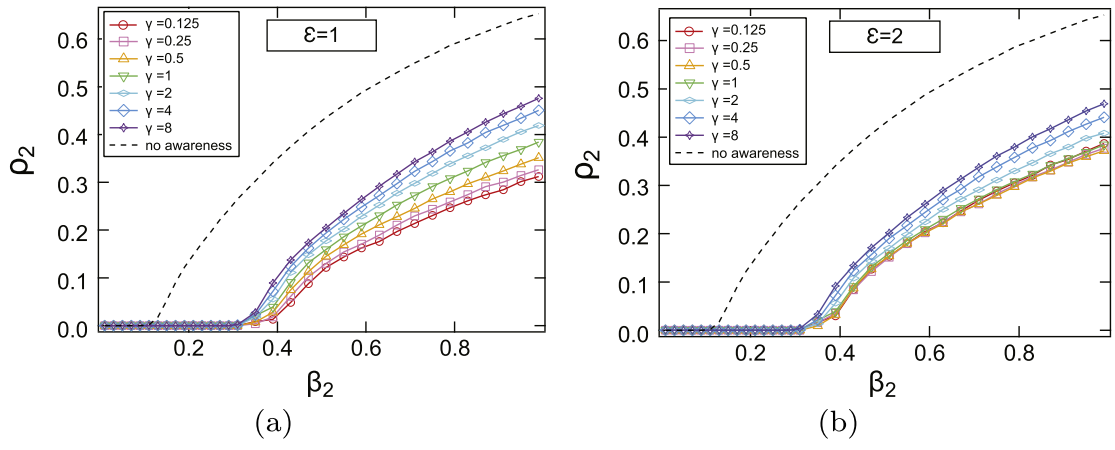

Figure 4. The average fraction of infected nodes ρ_2 as a function of the epidemic spreading rate β_2 for various values of time scale γ in two-layer SF networks with the overlap $\phi = 1$. The injection rate is γ^{ϵ} where $\epsilon = 1$ in (a) and $\epsilon = 2$ in (b). Other parameters used in this figure are: $\beta_1 = 0.3$, $\delta_1 = 0.6$, $\delta_2 = 1.0$ and $\alpha = 0.2$. The network size is $N = 1000$. For a scale-free network, the exponent in the power-law degree distribution is $\lambda = 2.5$. Initially, 10% of the randomly chosen nodes are unaware and infected (UII). The rest of the nodes are unaware and susceptible (US). Results are averaged over 200 realizations.

equation (1*a*), a node's awareness comes from two sources: (1) awareness spread from (being informed by) an aware neighbor with rate $\gamma\beta_1$, and (2) injection, i.e. a node becomes aware because it gets infected in the physical contact network with rate γ^{ϵ}. The information spreading in the communication network without any injection

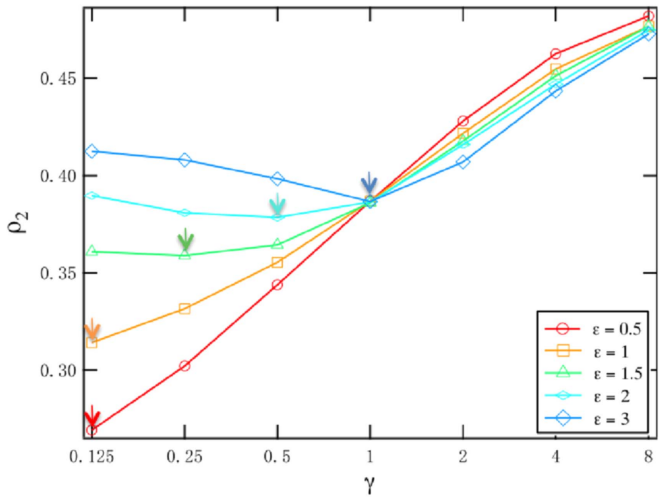

Figure 5. The average fraction of infected nodes ρ_2 as a function of γ for different values of ϵ in two-layer SF networks with the overlap $\phi = 1$. Other parameters used are: $\beta_1 = 0.3$, $\delta_1 = 0.6$, $\beta_2 = 1.0$ and $\delta_2 = 1.0$, $\alpha = 0.2$. The network size is $N = 1000$. For a scale-free network, the exponent in the power-law degree distribution is $\lambda = 2.5$. Initially, 10% of the randomly chosen nodes are unaware and infected (UII). The rest of the nodes are unaware and susceptible (US). Results are averaged over 200 realizations. For each given ϵ, the γ that leads to the minimal ρ_2 is indicated by an arrow.

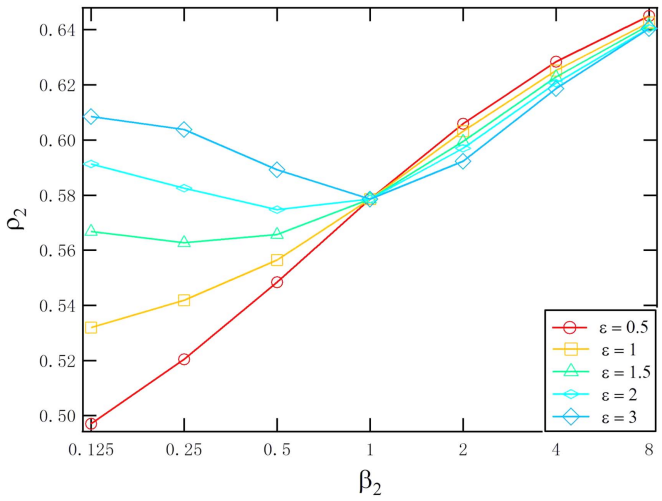

Figure 6. The average fraction of infected nodes ρ_2 as a function of γ for different values of ϵ in the real-world two-layer network. The parameters used are: $\beta_1 = 0.3$, $\delta_1 = 0.6$, $\beta_2 = 1.0$, $\delta_2 = 1.0$, $\alpha = 0.2$. The network size is $N = 1967$. Initially, 10% of the randomly chosen nodes are unaware and infected (UII). The rest of the nodes are unaware and susceptible (US). Results are averaged over 200 realizations of the IEAS process on the real-world network.

from the physical layer corresponds to the SIS model in a single network layer. In this case, the average fraction ρ_1 of aware nodes in the meta-stable is solely determined by β_1/δ_1 for a given network topology. Injections are triggered by the infections of nodes in the physical contact network. Consider the epidemic spreading alone in the physical contact network without the awareness information. In this case, the average fraction of infected nodes depends on β_2/δ_2. The frequency that nodes get infected relative to the information spreading is proportional to $1/\gamma$. An injection occurs if a node is unaware at the moment it gets infected and it gets the injection before it recovers to be susceptible again and before it becomes aware due to its aware neighbors. When an unaware node gets infected, the probability that an injection happens, i.e. before it recovers and before it gets aware via its neighbor, is approximately $\gamma^\epsilon/(\delta_2 + \gamma^\epsilon + c\beta_1\gamma)$, where c is the average number of aware neighbors of a node. Hence, the frequency of injection relative to the information spread is approximately proportional to[6] $\gamma^{\epsilon-1}/(\delta_2 + \gamma^\epsilon + c\beta_1\gamma)$, the maximum of which is obtained at smaller γ as ϵ decreases and is

[6] In our approximation, we have not taken into account the fact that the injection of awareness information may affect the average fraction of infected nodes, i.e. the relative frequency that nodes get infected with respect to the information spread thus slightly influences the injection frequency.

obtained at $\gamma = 0$ when $\epsilon \leqslant 1$. A large injection frequency leads to a higher fraction of aware nodes, which in turn results in a lower fraction of infected nodes. Hence, the optimal γ that best mitigates the epidemic decreases as ϵ decreases. As the time scale γ decreases, the relative frequency that nodes get infected with respect to the information spread increases. However, the probability that an infected node could inject this awareness information, i.e. it gets aware before it recovers and before it gets aware via aware neighbors, becomes smaller. Both effects contribute to the non-trivial optimal time scale when $\epsilon > 1$.

It takes on average 11.4 days (incubation period) for a susceptible individual to become infected by the infectious disease Ebola [44]. Information propagation in online social networks is fast due to the fact that more than 70% of users use, e.g. Facebook, daily [45]. However, the information spread in other communication networks like the mobile phone network could be relatively slower because of the less frequent usage of these networks. Which communication network is the best for epidemic mitigations depends on the speed that information is injected from the physical contact network to the communication network. Our result shows that no matter how the information is propagated at a faster time scale ($\gamma > 1$) or a slower time scale ($\gamma < 1$), a fast information injection from the physical contact network to the communication network (e.g. a small ϵ when $\gamma < 1$ and a large ϵ when $\gamma > 1$) is beneficial for the epidemic mitigation.

8. Validation in real-world network

Finally, we explore the effect of time scale γ on the epidemic spreading on a real-world two-layer network. We consider the two-layer network obtained from the location-based social network Brightkite where users shared their locations by checking-in. One layer is the online friendship network and the other is the physical contact network. We consider the users in the dataset that have been to New York at least once during the observation period April 2008—October 2010 [46]. Two users are assumed to be connected in the physical contact network layer if there is at least one day that their physical distance is less than 200 meters. The largest connected component of the physical contact network with 1967 nodes is considered and the friendship relations among these nodes are considered as the communication network layer where information propagates. Both layers follow a power-law degree distribution. The communication network has 9284 links, the physical contact network contains 11857 links and the two layers overlap in 767 links.

Our IEAS model is deployed upon this real-world network with various parameters. As shown in figure 6, we observe in the real-world two-layer network similar results as in network models: a non-trivial optimal time scale of the information propagation may exist and the optimal γ increases as the information injection control parameter ϵ increases.

9. Conclusions

The participation of individuals in several networks, such as the physical contact network and communication network, allows the dynamic processes deployed on these networks respectively to interact, introducing new possibilities for epidemic mitigation. In this work, we propose a generalised interacting epidemic and awareness spread model where becoming infected may make an individual aware of the epidemic whereas an individual aware of the epidemic reduces its rate of becoming infected by, e.g. avoiding contact with infected friends or wearing masks. We find that the epidemic spreading can indeed be mitigated by using the awareness information propagated in the communication network. Importantly, we discovered how the performance of the mitigation is influenced by the time scale of the awareness propagation relative to the epidemic spreading. Depending on how fast an infected node becomes aware, the optimal mitigation is achieved at a time scale γ that is not necessarily zero nor infinity, which contradicts our intuition that fast information spread better mitigates the epidemic spreading. We developed the IBMFA and MMCA to theoretically analyze such interacting processes on a two-layer network. Our observation is explained using both analytical and physical interpretations and is validated in a real-world physical contact-communication network. Our results imply that an optimal mitigation can be achieved when the time scale of the information spreading is not too fast such that the awareness information injected due to nodes' infection is not diluted, nor too slow such that the awareness information can be fast, thus successfully injected before the infected nodes recover or become aware via aware neighbors. Given a communication network and its corresponding time scale, a somewhat faster information infection from the physical contact network to the communication network, i.e. an infected node becomes aware fast, is in general beneficial for the mitigation.

The effect of the various features of the two-layer network topology on the epidemic spreading is explored in the appendix. We find that the mitigation tends to perform better when the two layers of the network overlap more, i.e. a larger fraction of pairs of nodes are connected in both layers. The optimal time scale decreases as the

density, or equivalently when the average degree of the two-layer networks, increases. This initial work points out the importance of further exploring real-world user behaviors: how long it takes for a user to share the information about an epidemic after (s)he get infected and whether this time delay depends on the social network they use.

Acknowledgments

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Appendix

A.1. Simulation, IBMFA and MMCA comparison in large networks

A.2. Effect of the overlap between the two layers of scale-free networks on epidemic spreading

In this section, we briefly discuss the effect of the overlap between the two layers of scale-free networks on the average fraction ρ_2 of infected nodes. As shown in figure A3, a higher overlap between the two layers better mitigates the epidemic spreading. The same has been observed in two-layer ER networks.

We consider the non-trivial case, $\epsilon > 1$, i.e. when a non-trivial optimal γ exists. When the time scale γ is either small or large, the frequency of injection relative to the information spread $\gamma^{\epsilon-1}/(\delta_2 + \gamma^{\epsilon} + c\beta_1\gamma)$ is close to 0, thus the awareness information injection from the physical contact network to the communication network is negligible. When $\phi = 1$ or equivalently, the same topology for both layers enables more efficient usage of the awareness information than the case when $\phi = 0$: a node with a high (low) risk of infection tends to also have a high (low) chance of becoming aware. When the time scale γ is neither too large nor too small, the complete overlap $\phi = 1$ also allows a newly infected node to inform its neighbors before they get infected, effectively reducing the infection rate of the neighbors. However, the effect of overlap extent would be diminished when β_2 is large (small), i.e. most nodes have a high (low) probability of becoming infected. In general, a large overlap between the two layers facilitates the mitigation.

Note that each node has the same degree in both SF layers when $\phi = 1$, whereas the degrees of a node in the two layers respectively are independent when $\phi = 0$. Hence, the degree–degree correlation between two layers changes as ϕ changes. This can be unavoidable depending on the degree distribution of the two layers. The degree–degree correlation in ER–ER networks is quite stable as ϕ varies, where we have observed the same in SF–SF networks regarding the influence of the overlap ϕ.

A.3. Effect of link density on the optimal time scale

We explore further whether the effect of time scale γ depends on the link density or equivalently the average degree of the two-layer network and whether the IBMFA mean field approximation becomes more precise as the underlying two-layer ER network becomes denser. As shown in figure A4, the IBMFA approaches the exact simulation results, indeed better, as the networks become denser. However, the exact simulation results show that the optimal time scale γ becomes trivial, i.e. zero, when the average degree increases, which cannot be captured by the IBMFA mean field approach. The optimal γ decreases as the network becomes denser. As discussed in section 7, the optimal time scale that leads to the minimal infection corresponds to the time scale that results in the highest frequency of injection $\gamma^{\epsilon-1}/(\delta_2 + \gamma^{\epsilon} + c\beta_1\gamma)$ relative to the information spread, where c is the number of aware neighbors of this node. As the two-layer ER network becomes denser, the average number c of aware neighbors of a node increases, which leads to a smaller optimal time scale γ.

A.4. Special case: immediate injection

Immediate injection, i.e. each node becomes immediately aware once it is infected, was assumed in [25, 26]. This is a special case in our IEAS model and will be discussed in this section. The immediate injection, i.e. zero injection delay, corresponds to our IEAS model when the injection rate is ∞. Still, we consider the case where an aware node could recover to unaware even though the node is still infected, as assumed in IEAS.

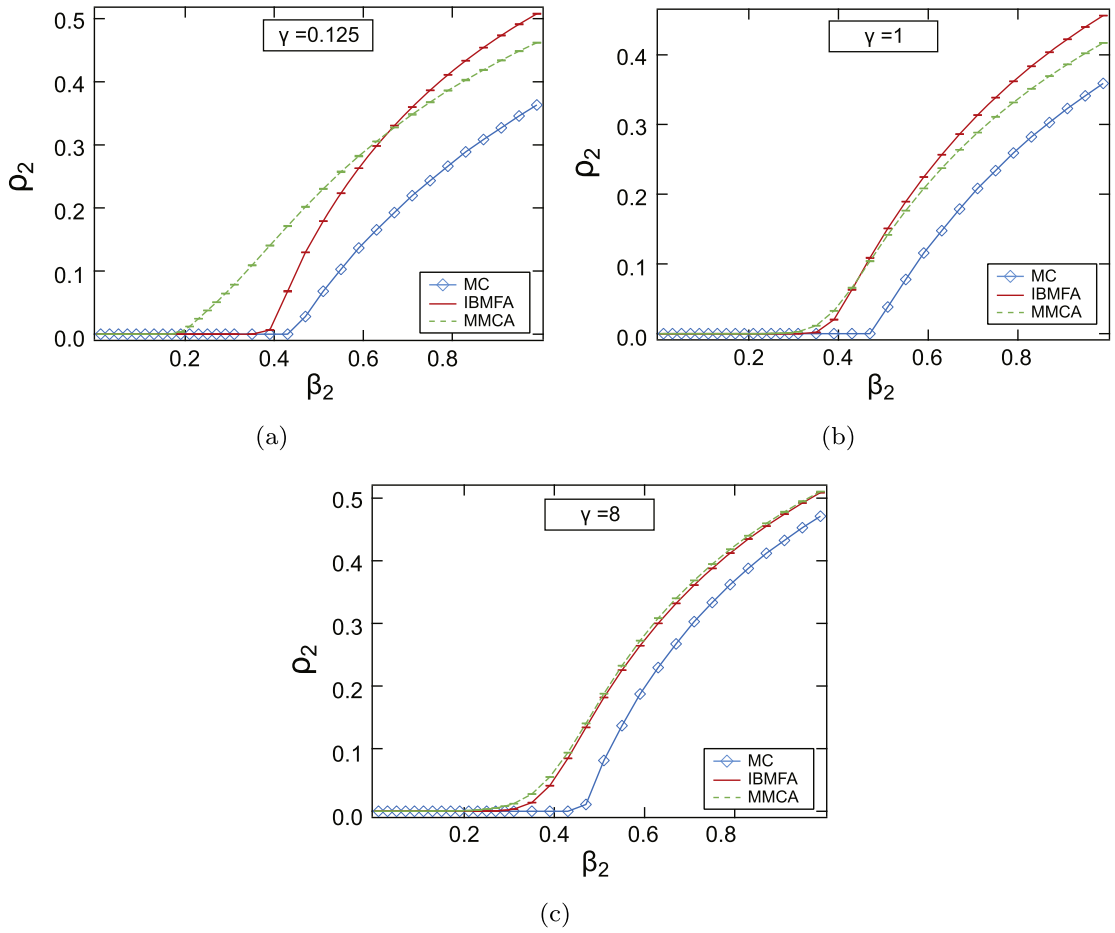

Figure A1. Comparison of the average fraction of infected nodes in the meta-stable state obtained by IBMFA, MMCA and simulations, respectively, in two-layer Erdős–Rényi random networks. The injection rate is γ^{ϵ} with $\epsilon = 2$. The relative time scale in the communication network layer is: (a) $\gamma = 0.125$, (b) $\gamma = 1$, (c) $\gamma = 8$. The parameters chosen are: $\beta_1 = 0.3$, $\delta_1 = 0.6$, $\delta_2 = 1.0$ and $\alpha = 0.2$. The network size is $N = 10000$. In each Erdős–Rényi random network, the average degree is $E[D] = 4$. The overlap between two network layers is $\phi = 1$. Results are averaged over 100 realizations for the IBMFA and MMCA, and 200 realizations for the simulations.

Our analytical approaches can be applied to this special, and actually simpler, case. The IBMFA becomes

$$\begin{aligned}\frac{\mathrm{d}u_i}{\mathrm{d}t} &= -\gamma\delta_1 u_i + (1-u_i)\left(\sum_{j=1}^{N} a_{ji}\gamma\beta_1 u_j + (1-v_i)\sum_{j=1}^{N} b_{ij}\beta_2 v_j\right)\\ \frac{\mathrm{d}v_i}{\mathrm{d}t} &= -\delta_2 v_i + (1-v_i)\left(\sum_{j=1}^{N} b_{ji}(u_i\alpha + (1-u_i))\beta_2 v_j\right),\end{aligned} \tag{6}$$

where $u_i(t)$ is the probability that node i is aware of the epidemic at time t, $v_i(t)$ is the probability that node i is infected at time t. The MMCA can simplified as

$$\begin{cases} p_i^{\mathrm{US}}(t+1) = p_i^{\mathrm{UI}}(t)r_i(t)\delta_2^* + p_i^{\mathrm{AI}}(t)\delta_1^*\delta_2^* + p_i^{\mathrm{US}}(t)r_i(t)q_i^{\mathrm{U}}(t) + p_i^{\mathrm{AS}}(t)\delta_1^* q_i^{\mathrm{A}}(t)\\ p_i^{\mathrm{UI}}(t+1) = p_i^{\mathrm{UI}}(t)r_i(t)(1-\delta_2^*) + p_i^{\mathrm{AI}}(t)\delta_1^*(1-\delta_2^*)\\ p_i^{\mathrm{AS}}(t+1) = p_i^{\mathrm{UI}}(t)(1-r_i(t))\delta_2^* + p_i^{\mathrm{AI}}(t)(1-\delta_1^*)\delta_2^*\\ \quad + p_i^{\mathrm{US}}(t)(1-r_i(t))q_i^{\mathrm{U}}(t) + p_i^{\mathrm{AS}}(t)(1-\delta_1^*)q_i^{\mathrm{A}}(t)\\ p_i^{\mathrm{AI}}(t+1) = p_i^{\mathrm{UI}}(t)(1-r_i(t))(1-\delta_2^*) + p_i^{\mathrm{AI}}(t)(1-\delta_1^*)(1-\delta_2^*)\\ \quad + p_i^{\mathrm{US}}(t)[(1-r_i(t))(1-q_i^{\mathrm{U}}(t)) + r_i(t)(1-q_i^{\mathrm{U}}(t))]\\ \quad + p_i^{\mathrm{AS}}(t)[\delta_1^*(1-q_i^{\mathrm{A}}(t)) + (1-\delta_1^*)(1-q_i^{\mathrm{A}}(t))]. \end{cases} \tag{7}$$

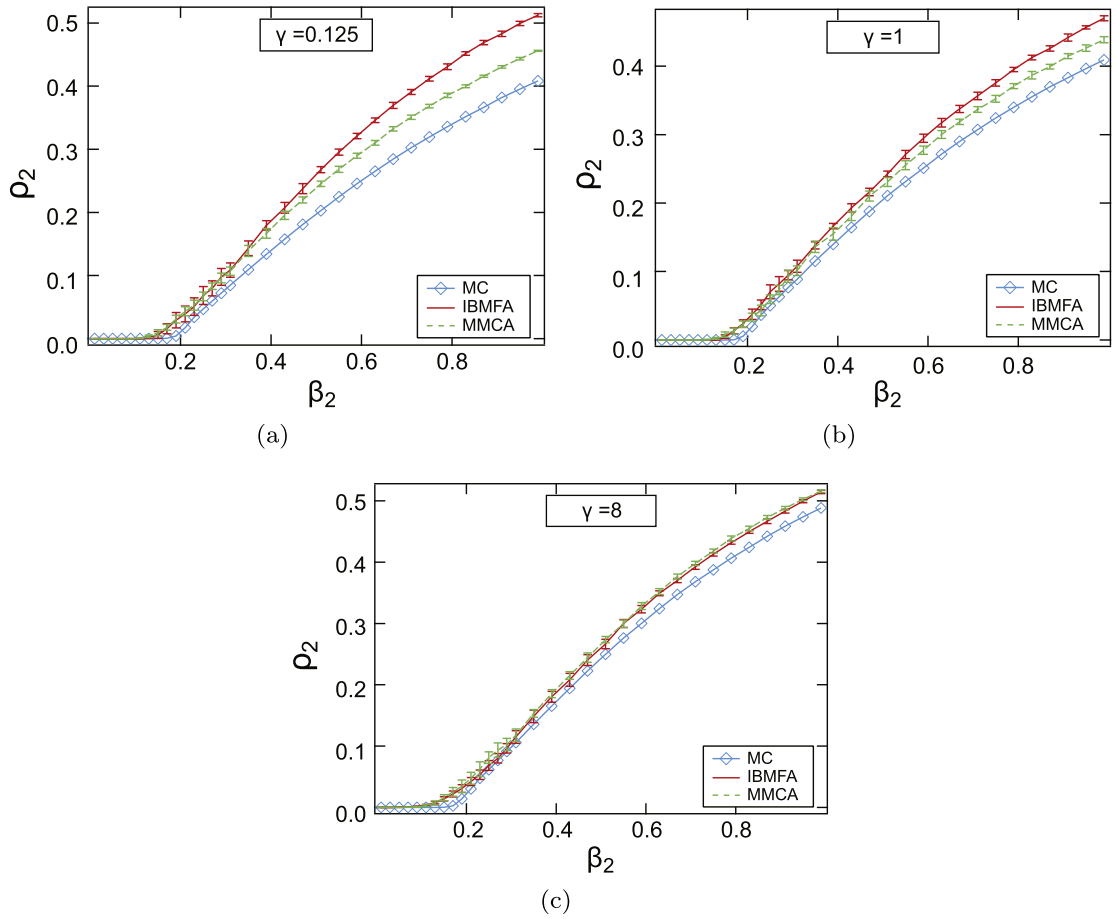

Figure A2. Comparison of the average fraction of infected nodes in the meta-stable state obtained by IBMFA, MMCA and simulations, respectively, in two-layer scale-free networks. The injection rate is γ^{ϵ} with $\epsilon = 2$. The relative time scale in the communication network layer is: (a) $\gamma = 0.125$, (b) $\gamma = 1$, (c) $\gamma = 8$. The parameters chosen are: $\beta_1 = 0.3$, $\delta_1 = 0.6$, $\delta_2 = 1.0$ and $\alpha = 0.2$. The network size is $N = 10000$. In each scale-free network, the exponent for the power-law degree distribution is $\lambda = 2.5$. The overlap between two network layers is $\phi = 1$. Results are averaged over 100 realizations for the IBMFA and MMCA, and 200 realizations for the simulations.

Equation (7) is subject to the normalization condition

$$p_i^{\mathrm{US}}(t) + p_i^{\mathrm{UI}}(t) + p_i^{\mathrm{AS}}(t) + p_i^{\mathrm{AI}}(t) = 1.$$

In this case, the effect of the time scale on the average fraction of infected nodes is relatively straightforward. As seen in figure A5, the fraction of infected nodes, ρ_2, in the meta-stable state can indeed be significantly reduced with the help of awareness information. However, the performance of the epidemic mitigation depends strongly on the relative time scale γ of the information propagation. Counterintuitively, the fraction of infected nodes increases as we increase the relative time scale γ. The epidemic threshold for β_2, above which the epidemic breaks out, tends to decrease when the time scale is increased. These observations hold for all the four types of networks: ER–ER or SF–SF and the overlap parameters $\phi = 0$ or $\phi = 1$. In general, a slower time scale better improves the robustness of the multi-layer network against epidemics. This is due to the fact that a slower time scale leads to a higher frequency of injections relative to the information spreading on the communication network. A higher frequency of injections results in a higher fraction of aware nodes, which better mitigates the epidemic spreading.

The best possible mitigation, i.e. the lower bound of the fraction of infected nodes, is achieved by the slowest time scale. When the time scale γ is infinitely slow, i.e. $\gamma * \beta_1 \approx 0$ and $\gamma * \delta_1 \approx 0$, injection from the physical layer is infinitely fast compared to the recovery rate of the awareness. Therefore, all the nodes are in the aware state. The epidemic spreading in this case is equivalent to the SIS model in a single physical contact network with infection rate $\alpha * \beta_2$.

The worst possible mitigation, i.e. the upper bound of the fraction of infected nodes, can be obtained by the fastest time scale. When time scale γ is infinitely fast, the injection is negligible, since awareness that comes from injection would be recovered instantly. The steady state fraction of aware nodes, as well as the probability for each node to be aware ν_i, is solely determined by the effective spreading rate $\frac{\beta_1}{\delta_1}$ and the topology of the the

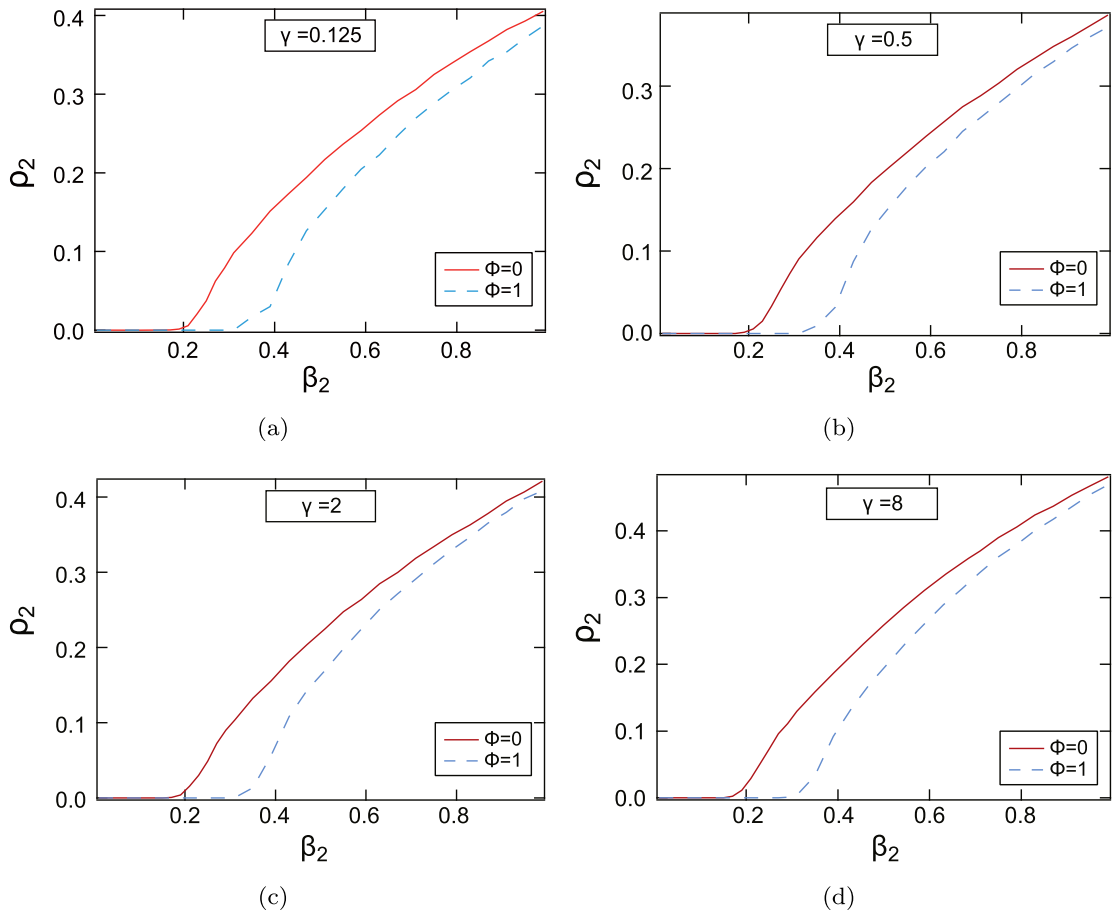

Figure A3. Influence of overlap ϕ between the two layers of scale-free networks on the average fraction ρ_2 of infected nodes in the physical contact network. The injection rate is γ^{ϵ} with $\epsilon = 2$. The relative time scales in the social network layer are: (a) $\gamma = 0.125$, (b) $\gamma = 0.5$, (c) $\gamma = 2$, (d) $\gamma = 8$. Parameters chosen are: $\beta_1 = 0.3$, $\delta_1 = 0.6$, $\delta_2 = 1.0$ and $\alpha = 0.2$. The network size is $N = 1000$. In each scale-free network, the exponent for the power-law degree distribution is $\lambda = 2.5$. Initially 10% of the randomly chosen nodes are infected. Results are averaged over 200 realizations.

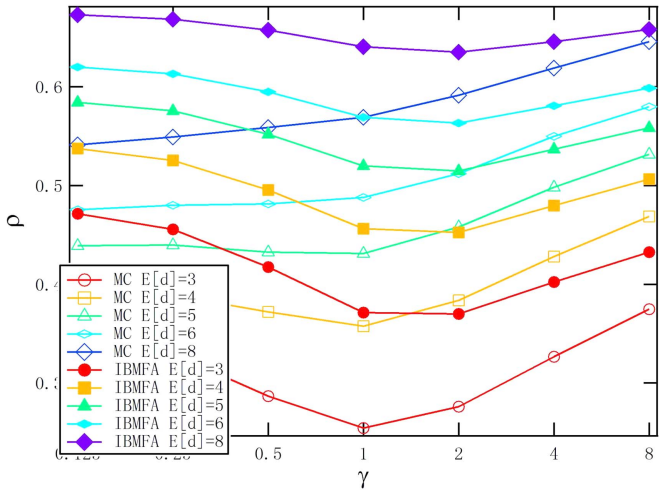

Figure A4. Comparison of the average fraction of infected nodes in the meta-stable state obtained by IBMFA and simulations, respectively, as a function of γ in two-layer Erdős–Rényi networks with various average degree $E[D]$ of each ER layer. The injection rate is γ^{ϵ} with $\epsilon = 3$. The parameters chosen are: $\beta_1 = 0.3$, $\delta_1 = 0.6$, $\beta_2 = 0.99$, $\delta_2 = 1.0$ and $\alpha = 0.2$. The network size is $N = 1000$. The overlap between the two network layers is $\phi = 1$. Results are averaged over 100 realizations for the IBMFA, and 200 realizations for the simulations.

communication network. Since γ is infinitely fast, the UAU process would reach the steady state instantly. At any time t, the rate that a node i gets infected by an infected neighbor is $\alpha * \beta_2$ with probability v_i, and is β_2 with probability $1 - v_i$. In this case, the upper bound, i.e. the worst possible mitigation, can be obtained via

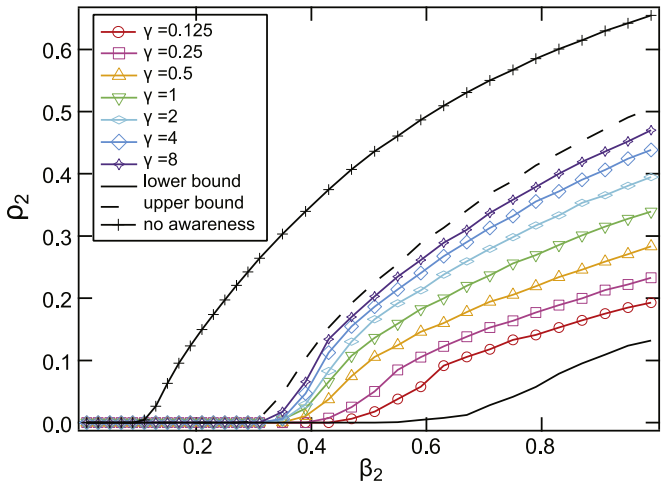

Figure A5. The average fraction of infected nodes ρ_2 as a function of the epidemic spreading rate β_2 for various values of the time scales γ in two-layer SF networks with the overlap $\phi = 1$ and immediate injection. Other parameters used in this figure are: $\beta_1 = 0.3$, $\delta_1 = 0.6$, $\delta_2 = 1.0$ and $\alpha = 0.2$. The network size is $N = 1000$. For the scale-free network, $\lambda = 2.5$. Initially, 10% of the randomly chosen nodes are unaware and infected (UII). The rest of the nodes are unaware and susceptible (US). Results are averaged over 200 realizations.

simulation (results are shown in figure A5) or via the following adapted IBMFA,

$$\frac{\mathrm{d}u_i}{\mathrm{d}t} = -\delta_1 u_i + (1 - u_i)\left(\sum_{j=1}^{N} a_{ji}\beta_1 u_j\right)$$
$$\frac{\mathrm{d}v_i}{\mathrm{d}t} = -\delta_2 v_i + (1 - v_i)\left(\sum_{j=1}^{N} b_{ji}(u_i\alpha + (1 - u_i))\beta_2 v_j\right). \quad (8)$$

where the physical contact layer is influenced by the communication network but not the other way around.

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References

[1] Pastor-Satorras R, Castellano C, Van Mieghem P and Vespignani A 2015 Epidemic processes in complex networks *Rev. Mod. Phys.* **87** 925–79
[2] Anderson R M and May R M 1991 *Infectious Diseases of Humans: Dynamics and Control* (New York: Oxford University Press)
[3] Bailey N T J 1975 The mathematical theory of infectious diseases and its applications (New York: Hafner)
[4] Parshani R, Carmi S and Havlin S 2010 Epidemic threshold for the susceptible-infectious-susceptible model on random networks *Phys. Rev. Lett.* **104** 258701
[5] Newman M E J 2002 Spread of epidemic disease on networks *Phys. Rev.* E **66** 016128
[6] Pastor-Satorras R and Vespignani A 2001 Epidemic spreading in scale-free networks *Phys. Rev. Lett.* **86** 3200–3
[7] Xia C, Wang Z, Sanz J, Meloni S and Moreno Y 2013 Effects of delayed recovery and nonuniform transmission on the spreading of diseases in complex networks *Physica* A **392** 1577–85
[8] Sanz J, Xia C Y, Meloni S and Moreno Y 2014 Dynamics of interacting diseases *Phys. Rev.* X **4** 041005
[9] Guo Q, Lei Y, Xia C, Guo L, Jiang X and Zheng Z 2016 The role of node heterogeneity in the coupled spreading of epidemics and awareness *PloS One* **11** 1–19 08
[10] Buldyrev S V, Parshani R, Paul G, Stanley H E and Havlin S 2010 Catastrophic cascade of failures in interdependent networks *Nature* **464** 1025–8
[11] Quill E 2012 When networks network: once studied solo, systems display surprising behavior when they interact *Science News* **182** 18–25
[12] Vespignani A 2010 Complex networks: the fragility of interdependency *Nature* **464** 984–5
[13] Dickison M, Havlin S and Stanley H E 2012 Epidemics on interconnected networks *Phys. Rev.* E **85** 066109
[14] Gao J, Buldyrev S V, Stanley H E and Havlin S 2012 Networks formed from interdependent networks *Nat. Phys.* **8** 40–8
[15] Liu M, Li D, Qin P, Liu C, Wang H and Wang F 2015 Epidemics in interconnected small-world networks *PloS One* **10** e0120701
[16] Wang H, Li Q, Agostino D G, Havlin S, Stanley H E and Van Mieghem P 2013 Effect of the interconnected network structure on the epidemic threshold *Phys. Rev.* E **88** 022801
[17] Li D, Qin P, Wang H, Liu C and Jiang Y 2014 Epidemics on interconnected lattices *Europhys. Lett.* **105** 68004
[18] Cozzo E, Baños R A, Meloni S and Moreno Y 2013 Contact-based social contagion in multiplex networks *Phys. Rev.* E **88** 050801
[19] Limiao Z *et al* 2016 Reliability analysis of interdependent lattices *Physica* A **452** 120–5

[20] Sahneh F D, Scoglio C and Chowdhury F N 2013 Effect of coupling on the epidemic threshold in interconnected complex networks: a spectral analysis *American Control Conf.* **2013** 2307–12
[21] Baxter G, Dorogovtsev S, Goltsev A and Mendes J 2012 Avalanche collapse of interdependent networks *Phys. Rev. Lett.* **109** 248701
[22] Szell M, Lambiotte R and Thurner S 2010 Multirelational organization of large-scale social networks in an online world *Proc. Natl Acad. Sci.* **107** 13636–41
[23] Bianconi G 2013 Statistical mechanics of multiplex networks: Entropy and overlap *Phys. Rev.* E **87** 062806
[24] Massaro E and Bagnoli F 2014 *Phys. Rev.* E **90** 052817
[25] Granell C, Gómez S and Arenas A 2014 Competing spreading processes on multiplex networks: awareness and epidemics *Phys. Rev.* E **90** 012808
[26] Granell C, Gómez S and Arenas A 2013 Dynamical interplay between awareness and epidemic spreading in multiplex networks *Phys. Rev. Lett.* **111** 128701
[27] Barrat A, Barthelemy M and Vespignani A 2008 *Dynamical Processes on Complex Networks* (Cambridge: Cambridge University Press)
[28] Castellano C and Pastor-Satorras R 2010 Thresholds for epidemic spreading in networks *Phys. Rev. Lett.* **105** 218701
[29] Erdős P and Rényi A 1959 On random graphs I *Publicationes Mathematicae (Debrecen)* **6** 290–7
[30] Bollobás B 1985 *Random Graphs* (Academic: Springer)
[31] Watts D J and Strogatz S H 1998 Collective dynamics of small-world networks *Nature* **393** 440–2
[32] Catanzaro M, Boguñá M and Pastor-Satorras R 2005 Generation of uncorrelated random scale-free networks *Phys. Rev.* E **71** 027103
[33] Cohen R and Havlin S 2003 Scale-free networks are ultrasmall *Phys. Rev. Lett.* **90** 058701
[34] Barabási A L and Albert R 1999 Emergence of scaling in random networks *Science* **286** 509–12
[35] Hu Y, Zhou D, Zhang R, Han Z, Rozenblat C and Havlin S 2013 Percolation of interdependent networks with intersimilarity *Phys. Rev.* E **88** 052805
[36] Cellai D, López E, Zhou J, Gleeson J P and Bianconi G 2013 Percolation in multiplex networks with overlap *Phys. Rev.* E **88** 052811
[37] Van Mieghem P 2011 The N-intertwined SIS epidemic network model *Computing* **93** 147–69
[38] Van Mieghem P, Omic J and Kooij R 2009 Virus spread in networks *IEEE/ACM Trans. Netw.* **17** 1–14
[39] Ganesh A, Massoulié L and Towsley D 2005 The effect of network topology on the spread of epidemics *INFOCOM 2005. IEEE Proc. 24th Annual Joint Conf. of the IEEE Computer and Communications Societies* **2** 1455–66
[40] Sahneh F D, Scoglio C and Mieghem P V 2013 Generalized epidemic mean-field model for spreading processes over multilayer complex networks *IEEE/ACM Trans. Netw.* **21** 1609–20
[41] Chakrabarti D, Wang Y, Wang C, Leskovec J and Faloutsos C 2008 Epidemic thresholds in real networks *ACM Trans. Inf. Syst. Secur.* **10** 1
[42] Gómez S, Gómez-Gardenes J, Moreno Y and Arenas A 2011 Nonperturbative heterogeneous mean-field approach to epidemic spreading in complex networks *Phys. Rev.* E **84** 036105
[43] Cai C R, Wu Z X and Guan J Y 2014 Effective degree Markov-chain approach for discrete-time epidemic processes on uncorrelated networks *Phys. Rev.* E **90** 052803
[44] Team W E R 2014 Ebola virus disease in west Africa-the first 9 months of the epidemic and forward projections *New Engl. J. Med.* **371** 1481–95
[45] Duggan M, Ellison N B, Lampe C, Lenhart A and Madden M 2015 *Social Media Update* 2014 http://www.pewinternet.org/2015/01/09/social-media-update-2014/
[46] Cho E, Myers S A and Leskovec J 2011 Friendship and mobility: user movement in location-based social networks *Proc. 17th ACM SIGKDD Int. Conf. Knowledge Discovery and Data Mining. KDD '11* (New York, NY, USA: ACM) pp 1082–90