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Effective Degree Theory on Multiplex Networks for Concurrent Three-State Spreading Dynamics

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ABSTRACT We propose a model of concurrent three-state spreading dynamics on multiplex networks to study the co-evolution process of epidemic spreading and information diffusion. The spreading dynamics and information diffusion process are both described by three-state models which can be used to address a wide range of common spreading behaviors. As accurate prediction is important in understanding the behaviors of complicated spreading dynamics, based on our proposed model, we develop a continuous-time effective degree theory (EDT) to delicately analyze the concurrent dynamics. We show that compared to the Monte Carlo simulations, this developed theory could predict the behavior of the dynamics in high accuracy, outperforming the dominantly adopted heterogeneous mean field theory applied on relevant dynamics on multiplex networks.

INDEX TERMS Effective degree theory, multiplex networks, concurrent spreading dynamics.

I. INTRODUCTION

During the past decades, many efforts have been devoted to the research of epidemic spreading on complex networks by use of computer simulations as well as approximate and exact analytical treatments [1]–[3]. The study of this topic, on one hand contributes to the understanding of behaviors of epidemics on sociology level, and on the other hand provides a simple dynamical framework to demonstrate rich phase diagrams. Several typical models have been proposed to describe common infectious diseases, which include two-state SIS model and three-state SIR model with S standing for susceptible, I for infected, and R for refractory in epidemiological terminology [4]–[10]. A variety of methods have been developed to analyze the epidemic spreading on complex networks, which include generating function [11], [12], pair-approximation [13], heterogeneous mean field theory [14]–[16], probability generating function [17], [18], branching process approximation [19], [20], and Lie algebra methods [21], [22].

Beyond the studies on single layered networks, recently growing attention has been paid to the intertwined effect of concurrent spreading dynamics on multiplex networks [23]–[28]. In multiplex networks, a set of nodes are connected with different types of connections. Each type of connections forms a corresponding configuration which is represented by a layer of the networks. For concurrent spreading dynamics in multiplex networks, each layer may have a specific spreading process running on top of it, and these dynamics in different layers may influence each other and co-evolve. For example, the behaviors of two concurrent epidemic spreading processes have been studied [29] where the characterization that the epidemic threshold of a disease is conditioned by the prevalence of the other disease is revealed. Further, the promotion and suppression effects between two concurrent SIS processes, referred as SIS-SIS model, is systematically studied [30]. In these studies, to describe the concurrent epidemic spreading processes the heterogeneous mean field theory (HMF) is adopted. Further, another kind of concurrent spreading dynamics is considered to study the co-evolution of epidemic spreading process and information diffusion process, where the latter is acted as an auxiliary process to influence the former. In this type of concurrent spreading dynamics, the information diffusion process is defined by a two-state unaware-aware-unaware (UAU) model and the epidemic spreading process is defined by a two-state susceptible-infected-susceptible (SIS) model, referred as UAU-SIS model [31]. As both SIS-SIS model and

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UAU-SIS model possess two-state spreading dynamics in each layer, we denoted them as concurrent two-state spreading dynamics.

The analysis of the UAU-SIS process is based on a microscopic Markov chain approach (MMCA) and a metacritical point is observed in this model [31]. The MMCA is an approach to describe discrete-time processes which has been previously adopted on single-layered quenched networks [32], [33]. Recently, it has been found that the MMCA will return to the HMF when the discrete time step tends to the continuous-time limit [34]. However, these studies have exhibited that the prediction of HMF and MMCA on concurrent epidemic dynamics have noticeable discrepancy comparing to the Monte Carlo simulations, especially when the infection rate is slightly above the epidemic threshold [35]. Since accurate prediction of spreading dynamics is important in understanding the behavior and dynamical features of the process, an effective degree has been introduced to describe the behavior of epidemic spreading on single-layered networks [36]. The key idea of EDT is to compartment the individuals according to the number of neighbors in different states and a set of master equations is derived to approximate the dynamics. This method has been shown to be able to provide high-accuracy approximation of the epidemic dynamics. However, despite the progress of EDT on two-state SIS model [37]–[39] and three-state SIR model [36], an EDT pertinent to concurrent three-state process is still missing. Since three-state dynamics is common in various circumstances, an EDT for concurrent three-state spreading dynamics is in demand.

In this work, we introduce a model of concurrent three-state spreading dynamics. The concurrent dynamics is composed of a three-state epidemic model pertained to the SIR process and a three-state information diffusion process related to the individual awareness of the epidemic. The two three-state dynamics are running on the two layers of multiplex networks, respectively, and co-evolve. In order to provide an accurate description of the concurrent three-state spreading dynamics, we develop an effective degree theory for the purpose. With the comparison to the Monte Carlo simulations, it shows that our proposed theory could successfully predict the concurrent dynamics in high accuracy, outperforming the dominantly used HMF in existing studies. Following we outline the main framework of this work: (i) We first propose a model of concurrent three-state spreading dynamics on multiplex networks in Sec. II; (ii) We propose an effective degree theory to predict the behavior of the concurrent dynamics in Sec. III; (iii) We compare the results of the proposed EDT and Monte Carlo simulation as well as the HMF on synthetic networks and an empirical network in Sec. IV. Results show that the EDT could predict the behavior of the networks in high accuracy, while the results of HMF have obvious deviations; (iv) Finally, we conclude our work in Sec. V with some discussions and outlook of future directions.

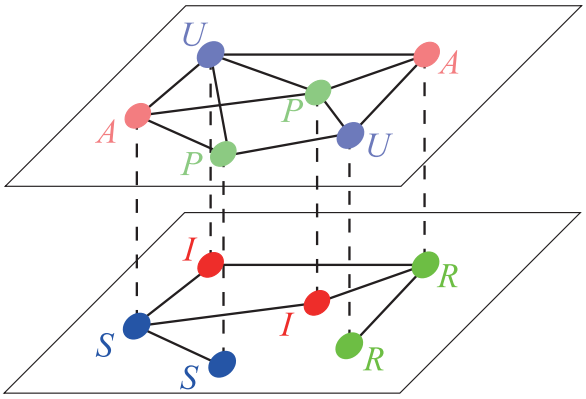

FIGURE 1. The structure of multiplex networks used in our model. The upper layer represents the networks where the news of the epidemic diffuses. Nodes in this layer can be in three kinds of states: Unaware (U), aware (A), and passive (P). The lower layer represents the networks where the epidemic spreads. Nodes in this layer can be in the states: Susceptible (S), infected (I), and refractory (R). Therefore, a node could be in a combination of nine states: Unaware and susceptible (US), unaware and infected (UI), unaware and refractory (UR), aware and susceptible (AS), aware and infected (AI), aware and refractory (AR), passive and susceptible (PS), passive and infected (PI), passive and refractory (PR).

II. UAP-SIR MODEL

We consider a multiplex network composed of two layers, which have the same number of individuals N but different connectivity configurations. In the lower layer (see Fig. 1), an epidemic may spread on it, which is referred as epidemic layer. In this layer, the state of an individual could be in three states, which are susceptible (S), infected (I), and refractory (R). A susceptible individual is susceptible to the epidemic; an infected individual has been infected and is able to spread the epidemic to its neighbors; a refractory individual has recovered from an infection and is immune to the epidemic permanently meanwhile will not spread the epidemic to others. In the upper layer individuals' awareness of an epidemic diffuses, which is referred as awareness layer. In this layer, the state of an individual could be in three states, which are unaware (U), aware (A), and passive (P). An unaware individual is unaware of the epidemic; an aware individual is aware of the epidemic and is willing to spread it to its neighbors; a passive individual has been aware of the epidemic but has lost the interest to spread. Therefore, in each time step each individual in this multiplex networks can be in one of the nine kinds of states: unaware and susceptible (US), unaware and infected (UI), unaware and refractory (UR), aware and susceptible (AS), aware and infected (AI), aware and refractory (AR), passive and susceptible (PS), passive and infected (PI), passive and refractory (PR),

The evolution of the awareness process in the awareness layer is composed of two parts. On one hand, an unaware individual may become aware due to two reasons: First, it may be informed by its aware neighbors, and each aware neighbor could send the news to it with a rate α. Second, if an unaware individual is also an infected one in the epidemic layer, it may be aware of the epidemic with a rate τ because of

self-awareness of the epidemic. On the other hand, an aware individual may turn to be passive at a rate γ because of losing the interest to spread the news.

In the epidemic layer, individuals' dynamics are similar to the SIR epidemic process, but a difference is that it can be influenced by the awareness layer as follows. A susceptible individual can be infected by an infectious neighbor. If this susceptible individual is unaware (aware or passive) of the epidemic, the infection rate coming from an infected neighbor will be β^{U} (β^{A}), where $\beta^{\mathrm{A}} \leq \beta^{\mathrm{U}}$ as an effect of preventive measures taken by the aware or passive ones. For the sake of simplicity, we assume $\beta^{\mathrm{A}} = \theta\beta^{\mathrm{U}}$ with $\theta \in [0, 1]$ and β^{U} is abbreviated as β in the rest part of the paper. Besides, an infected individual can recover to be refractory at a rate μ.

III. EFFECTIVE DEGREE THEORY FOR UAP-SIR MODEL

We now develop the EDT for the concurrent three-state UAP-SIR model on multiplex networks. In the EDT, besides the states of the individuals, the states of its neighbors are also tracked. For example, a US individual will be further classified according to the number of its neighbors in different states. Specifically, we classify an individual into a class of $X_{uap}Y_{sir}$ depending on its states and its neighbors' states, where $X \in \{U, A, P\}$ and $Y \in \{S, I, R\}$ and subscripts u, a, p, s, i, r denote the number of unaware, aware, passive, susceptible, infected, refractory neighbors it has, respectively.

Without causing confusion, we also use $X_{uap}Y_{sir}$ to indicate the fraction of individuals in respective class. Thus, the compartments $X_{uap}Y_{sir}$ satisfy the conservation law as $\sum_{u,a,p,s,i,r}[(U_{uap}S_{sir} + U_{uap}I_{sir} + U_{uap}R_{sir}) + (A_{uap}S_{sir} + A_{uap}I_{sir} + A_{uap}R_{sir}) + (P_{uap}S_{sir} + P_{uap}I_{sir} + P_{uap}R_{sir})] = 1$. For the initial condition, we suppose initially in the awareness layer a fraction a_0 of individuals are aware of the epidemics and the rest are unaware of the epidemics, and in the epidemic layer a fraction i_0 of individuals and the rest are susceptible of the epidemics. These a_0 and i_0 initial fraction of individuals are randomly distributed in the respective layers with no correlation. Thus, we have $X_{uap}Y_{sir}(0) = X_{uap}(0) \cdot Y_{sir}(0)$, where $X_{uap}(Y_{sir})$ denotes the fraction of individuals in the state $X(Y)$ with u unaware (s susceptible), a aware (i infected), and p passive (r refractory) neighbors. Suppose the degree distributions of the awareness layer and epidemic layer are p_i and q_k respectively, one obtains

$$U_{i-j,j,0}(0) = (1-a_0)p_i \binom{i}{j} a_0^j (1-a_0)^{(i-j)}, \quad (1a)$$

$$A_{i-j,j,0}(0) = a_0 p_i \binom{i}{j} a_0^j (1-a_0)^{(i-j)}, \quad (1b)$$

$$S_{k-l,l,0}(0) = (1-i_0)q_k \binom{k}{l} i_0^l (1-i_0)^{(k-l)}, \quad (1c)$$

$$I_{k-l,l,0}(0) = i_0 q_k \binom{k}{l} i_0^l (1-i_0)^{(k-l)}. \quad (1d)$$

We take Eq. (1a) to show the reasoning of the above equations. $U_{i-j,j,0}(0)$ denotes the initial fraction of unaware (U) individuals which has $i-j$ unaware neighbors, j aware neighbors, and 0 passive neighbors (initially there is no passive individuals in the network) in the awareness layer. Therefore, the total number of neighbors of a $U_{i-j,j,0}$ one equals to $(i-j)+j+0 = i$. Thus, the possibility of randomly choosing such an individual in the awareness layer can be separated into three independent events. First, the possibility of choosing an individual with degree i equals to p_i. Secondly, the possibility that this individual is in the unawareness state equals to $(1-a_0)$. Thirdly, the possibility that this i degree individual has $i-j$ unaware neighbors and j aware neighbors follows the binomial distribution $\binom{i}{j} a_0^j (1-a_0)^{(i-j)}$. Hence, multiplying all the three parts gives the Eq. (1a). It is easy to see the conservation law of $\sum_{i,j\leq i}[U_{i-j,j,0}(0)+A_{i-j,j,0}(0)] = 1$ and $\sum_{i,j\leq i}[S_{k-l,l,0}(0) + I_{k-l,l,0}(0)] = 1$. Note that since in the beginning there is no passive ones and refractory ones, we have $P_{i-j,j,0}(0) = 0$ for $\forall i, j$ and $R_{k-l,l,0}(0) = 0$ for $\forall k, l$.

Our method is used for continuous-time process and the evolution are described by a set of ordinary differential equations (ODEs). To simplify the discussion, we separate the whole process into four sub-processes, named as infection process, refractory process, awareness process, and passiveness process, respectively. For continuous-time process, the time interval of each update is very small, during which the mutual influences of these sub-processes could be ignored. Therefore, for each class of individuals, we shall first calculate their variations in the four sub-processes respectively, and then sum them up to obtain the variation of the whole process. Denoting the differential operators of the ODEs for the four sub-processes and the whole process as d^{I}/dt, d^{R}/dt, d^{A}/dt, d^{P}/dt, and d/dt, respectively, we have $d/dt(\cdot) = d^{\mathrm{I}}/dt(\cdot) + d^{\mathrm{R}}/dt(\cdot) + d^{\mathrm{A}}/dt(\cdot) + d^{\mathrm{P}}/dt(\cdot)$. All possible state transitions among different classes of $X_{uap}Y_{sir}$ are presented in Fig. 2 for easy reference, and in the following we will derive the governing equations of these sub-processes accordingly.

We start from the infection process. In the EDT, besides considering the change in the state of an individual itself, the change of its neighbors' states will also be tracked. Since in the continuous-time description, the possibility of two events happened in one time interval can be ignored, the change in the states of an individual's neighbor due to the infection process could only be the case of $X_{uap}Y_{sir} \to X_{uap}Y_{s-1,i+1,r}$, i.e. a susceptible neighbour changes to be infected through infection process. However, since this susceptible neighbour could be in the US, AS, or PS state, we use mean field approximation to estimate the changing rate of this susceptible neighbour. For a $U_{uap}S_{sir}$ individual, one of its susceptible neighbour may be in a class of $U_{u'a'p'}S_{s'i'r'}$, $A_{u'a'p'}S_{s'i'r'}$, or $P_{u'a'p'}S_{s'i'r'}$. Suppose this neighbour is an $A_{u'a'p'}S_{s'i'r'}$ one. This event happens with a possibility of $A_{u'a'p'}S_{s'i'r's'}/\sum_{u'a'p's'i'r'}(U_{u'a'p'}S_{s'i'r'} + A_{u'a'p'}S_{s'i'r'} + P_{u'a'p'}S_{s'i'r'})s'$ under non-correlation assumption, which is proportional to s', and the dominator serves as the normalization condition. Thus, the rate of a $U_{uap}S_{sir}$ individual having an $A_{u'a'p'}S_{s'i'r'}$ neighbour

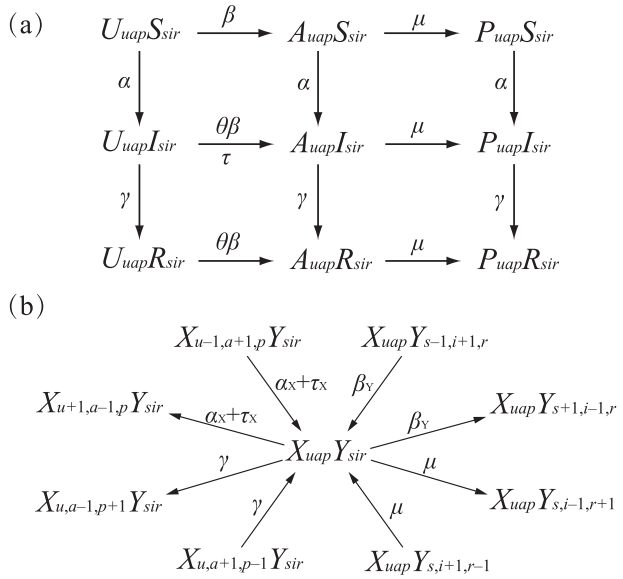

FIGURE 2. State transitions and respective rates among different classes of individuals, where $X_{uap}Y_{sir}$ denotes the fraction of individuals in the XY state, $X \in \{U, A, P\}$ and $Y \in \{S, I, R\}$, with u unaware, a aware, and p passive neighbors in the awareness layer and s susceptible, i infected, and r refractory neighbors in the epidemic layer. (a) Transitions happened on the individuals of respective classes; (b) Transitions happened on the neighbors of the individuals of respective classes. Direction of the arrows indicate the direction of the transitions. The definitions of the rates are provided in the text.

meanwhile this neighbour is infected equals to $\theta\beta i' A_{u'a'p'} S_{s'i'r'} s' / \sum_{u'a'p's'i'r'} (U_{u'a'p'} S_{s'i'r'} + A_{u'a'p'} S_{s'i'r'} + P_{u'a'p'} S_{s'i'r'}) s'$ since this susceptible individual has i' neighbors. Similarly, for an $U_{u'a'p'} S_{s'i'r'}$ neighbour the corresponding rate equals to $\beta i' U_{u'a'p'} S_{s'i'r'} s' / \sum_{u'a's'i'} (U_{u'a'} S_{s'i'} + A_{u'a'} S_{s'i'} + P_{u'a'p'} S_{s'i'r'}) s'$, and for a $P_{u'a'p'} S_{s'i'r'}$ the rate is $\theta\beta i' P_{u'a'p'} S_{s'i'r'} s' / \sum_{u'a'p's'i'r'} (U_{u'a'p'} S_{s'i'r'} + A_{u'a'p'} S_{s'i'r'} + P_{u'a'p'} S_{s'i'r'}) s'$. Summing all the possible classes gives the effective rate of a susceptible neighbour to be infected, defined as β_S, as shown in Eq. (2a), as shown at the bottom of this page. One may further find that a susceptible neighbour of an $A_{uap}S_{sir}$ and an $P_{uap}S_{sir}$ individual has the same effective infection rate β_S. With similar reason, the effective rate of a susceptible neighbour of a $U_{ua}I_{si}$, $A_{ua}I_{si}$, and $P_{ua}I_{si}$ one, defined as β_I, is shown as Eq. (2b), as shown at the bottom of this page; and the effective rate of a susceptible neighbour of a $U_{ua}R_{si}$, $A_{ua}R_{si}$, and $P_{ua}R_{si}$ one, defined as β_R, is shown as Eq. (2c), as shown at the bottom of this page. Hence, one may obtain the evolution equations of the infection process as follows:

$$\frac{d^{\mathrm{I}}(X_{uap}S_{sir})}{dt} = -\beta^* i X_{uap}S_{sir} - \beta_S X_{uap}S_{sir}s + \beta_S X_{uap}S_{s+1,i-1,r}(s+1), \tag{3a}$$

$$\frac{d^{\mathrm{I}}(X_{uap}I_{sir})}{dt} = +\beta^* i X_{uap}I_{sir} - \beta_I X_{uap}I_{sir}s + \beta_I X_{uap}I_{s+1,i-1,r}(s+1), \tag{3b}$$

$$\frac{d^{\mathrm{I}}(X_{uap}R_{sir})}{dt} = -\beta_R X_{uap}R_{sir}s + \beta_R X_{uap}R_{s+1,i-1,r}(s+1), \tag{3c}$$

in which when $X = U$, $\beta^* = \beta$ and when $X = A$ or P, $\beta^* = \theta\beta$.

Thus, in Eq. (3a), when $X = U$, $\beta^* = \beta$, the left hand side of Eq. (3a) is $\frac{d^{\mathrm{I}}(U_{uap}S_{sir})}{dt}$, and the first term on the right hand side is $-\beta i U_{uap}S_{sir}$. This term $-\beta i U_{uap}S_{sir}$ describes the decrement in the fraction of $U_{uap}S_{sir}$ due to the infection process. In fact, a $U_{uap}S_{sir}$ individual has i infected neighbors and each infected neighbor could infect this individual with the rate β, which lead to the decreasing of $U_{uap}S_{sir}$ by the amount of $\beta i U_{uap}S_{sir}$. Note that when $X = A$ and P, the corresponding individuals are $A_{uap}S_{sir}$ and $P_{uap}S_{sir}$, which have been aware of the living epidemic, resulting $\beta^* = \theta\beta$. In this case, the decrement of fraction of these individuals are $-\theta\beta i A_{uap}S_{sir}$ and $-\theta\beta i P_{uap}S_{sir}$, respectively. Now, let us turn to the second term on the right hand side of Eq. (3a) for $X = U$, which is $-\beta_S U_{uap}S_{sir}s$. This term corresponds to the process of $U_{uap}S_{sir} \to U_{uap}S_{s-1,i+1,r}$, which happens when the state of an susceptible neighbor of $U_{uap}S_{sir}$ individuals is changed. Indeed, as introduced above, β_S defined in Eq. (2a) equals to the rate that a susceptible neighbor of a $U_{uap}S_{sir}$ is infected, and further consider the s number susceptible neighbors that a $U_{uap}S_{sir}$ individual has, this term tells the variation in the amount of the $U_{uap}S_{sir}$ according to this process. Similarly, the third term on the right hand side of Eq. (3a), $\beta_S X_{uap}S_{s+1,i-1,r}(s+1)$, gives the incremental amount of the $U_{uap}S_{sir}$ due to the process of $U_{uap}S_{s+1,i-1,r} \to U_{uap}S_{sir}$. Equations (3b) and (3c) show the variation of $X_{uap}I_{sir}$ and $X_{uap}R_{sir}$, respectively. It is worth noting that the rate of a susceptible neighbor of an $X_{uap}S_{sir}$ one and an $X_{uap}I_{sir}$ one to be infected are different, which are β_S and β_I, respectively. This difference is actually come from the fact that $X_{uap}S_{sir}$ and $X_{uap}I_{sir}$ have different possibilities to connect with $U_{u'a'p'}S_{s'i'r'}$, $A_{u'a'p'}S_{s'i'r'}$, and $P_{u'a'p'}S_{s'i'r'}$, embodied in the different weights of s' and i', respectively.

$$\beta_S = \frac{\sum_{u'a'p's'i'r'} (\beta U_{u'a'p'} S_{s'i'r'} s' + \theta\beta A_{u'a'p'} S_{s'i'r'} s' + \theta\beta P_{u'a'p'} S_{s'i'r'} s') i'}{\sum_{u'a'p's'i'r'} (U_{u'a'p'} S_{s'i'r'} s' + A_{u'a'p'} S_{s'i'r'} s' + P_{u'a'p'} S_{s'i'r'} s')} \tag{2a}$$

$$\beta_I = \frac{\sum_{u'a'p's'i'r'} (\beta U_{u'a'p'} S_{s'i'r'} i' + \theta\beta A_{u'a'p'} S_{s'i'r'} i' + \theta\beta P_{u'a'p'} S_{s'i'r'} i') i'}{\sum_{u'a'p's'i'r'} (U_{u'a'p'} S_{s'i'r'} i' + A_{u'a'p'} S_{s'i'r'} i' + P_{u'a'p'} S_{s'i'r'} i')} \tag{2b}$$

$$\beta_R = \frac{\sum_{u'a'p's'i'r'} (\beta U_{u'a'p'} S_{s'i'r'} r' + \theta\beta A_{u'a'p'} S_{s'i'r'} r' + \theta\beta P_{u'a'p'} S_{s'i'r'} r') i'}{\sum_{u'a'p's'i'r'} (U_{u'a'p'} S_{s'i'r'} r' + A_{u'a'p'} S_{s'i'r'} r' + P_{u'a'p'} S_{s'i'r'} r')} \tag{2c}$$

Similar arguments apply to the $X_{uap}R_{sir}$. With the understanding of these points and the reference of Eq. (3a), Eqs. (3b) and (3c) could then be readily understood.

The refractory process is relatively simple and its evolution equations are given as follows:

$$\begin{aligned}\frac{d^{\mathrm{R}}(X_{uap}S_{sir})}{dt} &= -\mu X_{uap}S_{sir}i \\ &\quad + \mu X_{uap}S_{s,i+1,r-1}(i+1), \qquad (4a)\\ \frac{d^{\mathrm{R}}(X_{uap}I_{sir})}{dt} &= -\mu X_{uap}I_{sir} - \mu X_{uap}I_{sir}i \\ &\quad + \mu X_{uap}I_{s,i+1,r-1}(i+1), \qquad (4b)\\ \frac{d^{\mathrm{R}}(X_{uap}R_{sir})}{dt} &= +\mu X_{uap}I_{sir} - \mu X_{uap}R_{sir}i \\ &\quad + \mu X_{uap}R_{s,i+1,r-1}(i+1), \qquad (4c)\end{aligned}$$

with $X \in \{U, A, P\}$ respectively.

In Eq. (4a), the two terms on the right hand side describe the processes of $X_{uap}S_{s,i,r} \to X_{uap}S_{s,i-1,r+1}$ and $X_{uap}S_{s,i+1,r-1} \to X_{uap}S_{sir}$, respectively. These two processes are all caused from the state change on their neighbors of the individuals. Particularly, for the first term, since an $X_{uap}S_{s,i,r}$ individual has i infected neighbors and each of them may become refractory with rate μ, this process contributes to the variation of $X_{uap}S_{s,i,r}$ in an amount of $\mu X_{uap}S_{sir}i$. Since an $X_{uap}S_{s,i,r}$ individual is in the susceptible state, the refractory process will not take place on it. However, as the refractory process could change an $X_{uap}I_{s,i,r}$ one to be an $X_{uap}R_{s,i,r}$ one with a rate μ, this event contributes to the first terms on the right hand side of Eqs. (4b) and (4c), with one for decrement and the other for increment.

Similar to the infection process, the evolution equations of awareness process are as follows:

$$\begin{aligned}\frac{d^{\mathrm{A}}(U_{uap}Y_{sir})}{dt} &= -\alpha U_{uap}Y_{sir}a - (\alpha_{\mathrm{U}}+\tau_{\mathrm{U}})U_{uap}Y_{sir}u \\ &\quad +(\alpha_{\mathrm{U}}+\tau_{\mathrm{U}})U_{u+1,a-1,p}Y_{s,i,r}(u+1), \\ &\quad -\tau U_{uap}I_{sir} \qquad \text{when} \quad Y = I, \qquad (5a)\\ \frac{d^{\mathrm{A}}(A_{uap}Y_{sir})}{dt} &= +\alpha U_{uap}Y_{sir}a - (\alpha_{\mathrm{A}}+\tau_{\mathrm{A}})A_{uap}Y_{sir}u \\ &\quad +(\alpha_{\mathrm{A}}+\tau_{\mathrm{A}})A_{u+1,a-1,p}Y_{s,i,r}(u+1), \\ &\quad +\tau U_{uap}I_{sir} \qquad \text{when} \quad Y = I, \qquad (5b)\\ \frac{d^{\mathrm{A}}(P_{uap}Y_{sir})}{dt} &= -(\alpha_{\mathrm{P}}+\tau_{\mathrm{P}})P_{uap}Y_{sir}u \\ &\quad +(\alpha_{\mathrm{P}}+\tau_{\mathrm{P}})P_{u+1,a-1,p}Y_{s,i,r}(u+1), \qquad (5c)\end{aligned}$$

with $Y \in \{S, I, R\}$. In Eqs. (5a) and (5b), the last terms, i.e. $-\tau U_{uap}I_{sir}$ and $\tau U_{uap}I_{sir}$, account for the self-awareness process which happen when $Y = I$. In Eq. (5a), the first term on the right hand side, $-\alpha U_{uap}Y_{sir}a$, describes the process of $U_{uap}Y_{sir} \to A_{uap}Y_{sir}$, which happens on the individuals $U_{uap}Y_{sir}$ themselves. The second term on the right hand side, $-(\alpha_{\mathrm{U}}+\tau_{\mathrm{U}})U_{uap}Y_{sir}u$, describes the process of $U_{uap}Y_{sir} \to U_{u-1,a+1,p}Y_{sir}$, which happens on the neighbour of $U_{uap}Y_{sir}$ individuals. Obviously, this term is combined with two parts, $\alpha_{\mathrm{U}}U_{uap}Y_{sir}u$ and $\tau_{\mathrm{U}}U_{uap}Y_{sir}u$, where α_{U} (τ_{U}) denotes the possibility that a unaware neighbor of $U_{uap}Y_{sir}$ becomes aware through the awareness process (self-awareness process). The third term in the right hand side could be understood similarly. The interpretation of Eqs. (5b) and (5c) is analogous to the Eq. (5a), in which the definitions of α_{A}, τ_{A}, α_{P}, τ_{P}, are provided in Eq. (6), as shown at the bottom of this page. Moreover, we note that as the awareness process happens between the states U and A, in Eq. (5c) the $P_{uap}Y_{sir}$ individuals themselves will not participate in this process.

Finally, the evolution equations of passiveness process are as follows:

$$\begin{aligned}\frac{d^{\mathrm{P}}(U_{uap}Y_{sir})}{dt} &= -\gamma U_{uap}Y_{sir}a \\ &\quad +\gamma U_{u,a+1,p-1}Y_{s,i,r}(a+1), \qquad (7a)\\ \frac{d^{\mathrm{P}}(A_{uap}Y_{sir})}{dt} &= -\gamma A_{uap}Y_{sir} - \gamma A_{uap}Y_{sir}a \\ &\quad +\gamma A_{u,a+1,p-1}Y_{s,i,r}(a+1), \qquad (7b)\\ \frac{d^{\mathrm{P}}(P_{uap}Y_{sir})}{dt} &= +\gamma P_{uap}Y_{sir} - \gamma P_{uap}Y_{sir}a \\ &\quad +\gamma P_{u,a+1,p-1}Y_{s,i,r}(a+1), \qquad (7c)\end{aligned}$$

with $Y \in \{S, I, R\}$. The passiveness process described in Eq. (7) mirrors the refractory process in Eq. (4), where all the terms in both equations could be understood based on a one-to-one correspondence.

IV. NUMERICAL RESULTS

In this work, we examine the performance of the EDT on multiplex networks in which each layer has a truncated power-law degree distribution with $p_k = q_k \sim k^{-\delta}$ when $0 < k \le k_{\mathrm{T}}$ and $p_k = q_k = 0$ otherwise. As comparison, the results of HMF and the Monte Carlo simulations are also presented. The results of HMF are obtained from the evolution equations in Appendix V. In the Monte Carlo simulations, the results are constructed with configuration model with the same degree distribution, where the number of nodes is 10^4 and the exponent of the degree distribution is $\delta = 2.2$. The results of simulations are averaged from 100 different realizations and the error-bars stand for the standard deviation. Besides, we perform synchronous updating method in the simulations,

$$\alpha_{\mathrm{X}} = \frac{\sum_{u'a'p's'i'r'} \alpha a'(U_{u'a'p'}S_{s'i'r'} + U_{u'a'p'}I_{s'i'r'} + U_{u'a'p'}R_{s'i'r'})x'}{\sum_{u'a'p's'i'r'}(U_{u'a'p'}S_{s'i'r'} + U_{u'a'p'}I_{s'i'r'} + U_{u'a'p'}R_{s'i'r'})x'} \qquad (6a)$$

$$\tau_{\mathrm{X}} = \frac{\sum_{u'a'p's'i'r'} \tau U_{u'a'p'}I_{s'i'r'}x'}{\sum_{u'a'p's'i'r'}(U_{u'a'p'}S_{s'i'r'} + U_{u'a'p'}I_{s'i'r'} + U_{u'a'p'}R_{s'i'r'})x'} \qquad (6b)$$

that is: in each time step, all the individuals will update their states according to their current states and their neighbors' current states. The updated states will be taken as those to be used in the next time step and then the update process repeats until the final stage is reached.

Since our method is for continuous-time spreading process on multiplex networks, in order to fulfill this condition we use small dynamical parameters to realize the process, so that in each time step the increments of the variables are small and high order amounts can be neglected. Thus, in the simulation we set the parameters β, μ, α, γ, and τ in an order of 10^{-3}. Figure 3 shows the results of time evolutions of the fraction of aware individuals $\rho_A(t)$ and the fraction of infected individuals $\rho_I(t)$ for the two kinds of networks. For the convenience of comparison, besides the results of simulation and the EDT, the results of heterogeneous mean field method (HMF) are also presented. One can see that the EDT has an excellent agreement with the simulations, while HMF overestimate the results.

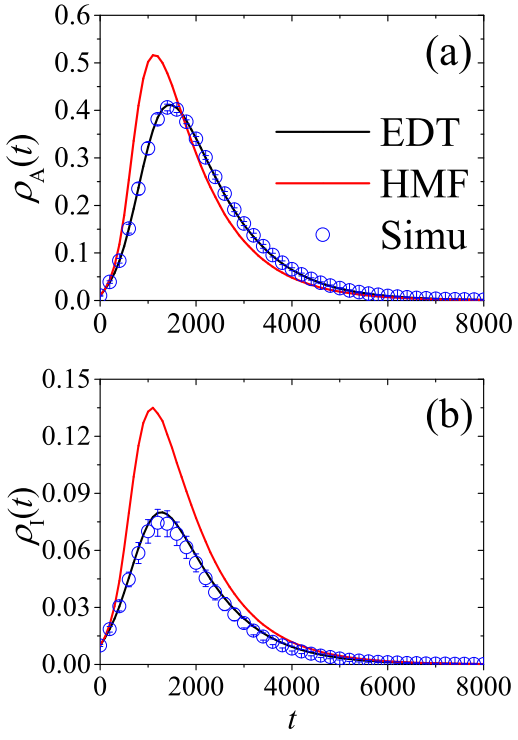

FIGURE 3. (Color online) Time evolution of the fractions of (a) aware individuals $\rho_A(t)$ and (b) infected individuals $\rho_I(t)$, respectively. Results are obtained from numerical simulations (blue open circles), EDT (black curves), and HMF (red curves). The network is constructed with uncorrelated configuration model with power-law degree distribution $p_k \sim k^{-\delta}$, where $\delta = 2.2$ and $k_T = 10$. The network size is $N = 10000$. Results are obtained with 100 different realizations. Other parameter values are $\beta = \mu = \alpha = \gamma = \tau = 10^{-3}$ and $\theta = 0.5$.

We further illustrate the fraction of passive individuals ρ_P and refractory individuals ρ_R in the final stage of the two kinds of networks in Fig. 4. One can also observe the excellent agreements between the Monte Carlo simulations and the EDT, while the results of HMF overestimate

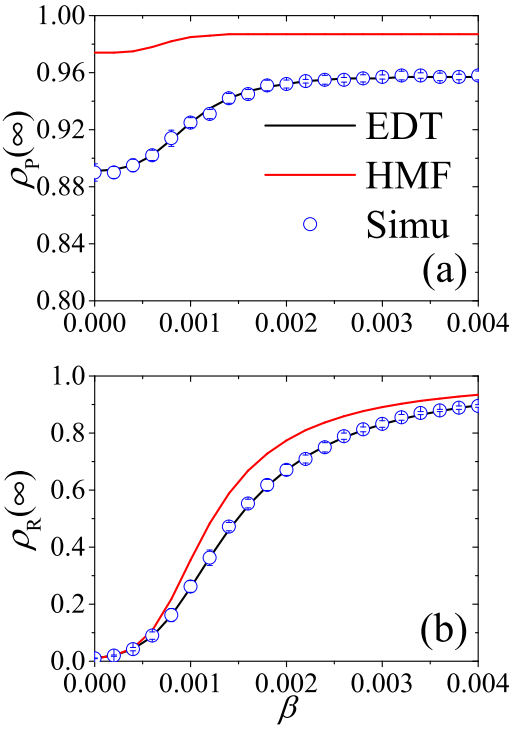

FIGURE 4. (Color online) The fractions of (a) passive individuals $\rho_P(\infty)$ and (b) refractory individuals $\rho_R(\infty)$ at the final stage as functions of infection rate β. The multiplex networks and other parameters are the same as those in Fig. 3.

the dynamics again. We remark that the overestimation of HMF is due to the fact that the dynamical correlation is neglected in the theory. The dynamical correlation in the epidemic layer is that an infected individual is more likely to connect with an infected neighbour since it must be infected by an infected neighbour in previous time. However, the neglect of this dynamical correlation will lead to more susceptible neighbors than expected and therefore cause the overestimation.

In the proposed EDT, each class of individuals are described with six variables to denote the number of different types of neighbors. Thus, the number of different equations is in a scale of $k_{\max}^6$ with $k_{\max}$ being the largest degree, which grows with the sixth power of the largest degree. For the HMF (see Appendix V), the number of different equations is in a scale of $k_{\max}^2$. Therefore, the ratio of the computational amounts between the EDT and the HMF will be in a scale of $k_{\max}^4$. This property makes the computational amount of the proposed theory to be formidable when the $k_{\max}$ is large. However, it could be a preferable approach for the scenario when the $k_{\max}$ is relatively small. In the following, we present the performance of the proposed theory on an empirical network generated with a real dataset. Specifically, we select a period of three years sexual contacts among individuals from the data collected in Ref. [40]. The resulting network is aggregated from the contacts during this period. The giant component of this network is chosen to compose the epidemic layer of the multiplex network, which has 11769 individuals,

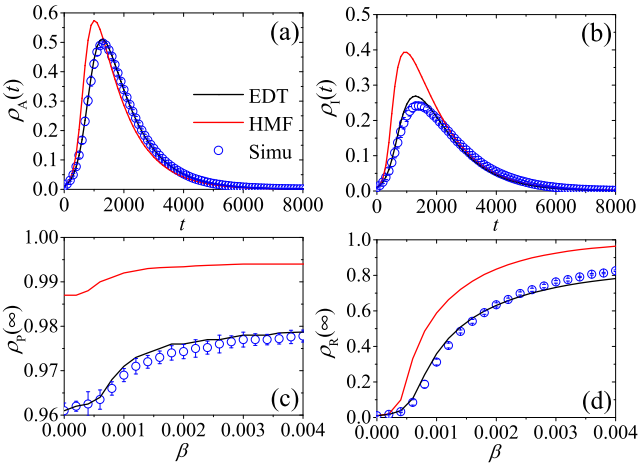

FIGURE 5. (Color online) Comparisons among effective degree theory (EDT), heterogeneous mean field theory (HMF), and numerical simulations (Simu) on an empirical sexual network (details are in the text) for (a) time evolution of aware individuals $\rho_A(t)$, (b) time evolution of infected individuals $\rho_I(t)$, (c) the final fraction of passive individuals $\rho_P(\infty)$, and (d) the final fraction of refractory individuals $\rho_R(\infty)$. The simulation results are obtained from 100 different realizations. The parameter values are $\mu = \alpha = \gamma = \tau = 10^{-3}$ and $\theta = 0.5$ with $\beta = 2 \times 10^{-3}$ in (a) and (b).

34404 connections, and $k_{\max} = 15$. Furthermore, in lack of the network data of the corresponding awareness layer, we construct the awareness layer by randomly adding 2×10^4 edges on the same network of epidemic layer meanwhile avoiding overlapping of existing contacts.

Figure 5 compares the results of proposed EDT, HMF, and Monte Carlo simulations, for the time-evolutions of aware individuals $\rho_A(t)$ and infected individuals $\rho_I(t)$, and the final fractions of passive individuals $\rho_P(\infty)$ and refractory individuals $\rho_R(\infty)$. One may clearly find that the proposed EDT could predict the behavior of the system in high accuracy while the HMF overestimates the results. In Fig. 5(c), it can be seen that $\rho_P(\infty)$ approaches 1, which means almost all of the individuals have been aware of the epidemics in the final stage. However, referring to Fig. 5(a), one may observe that the awareness of the epidemic among the individuals happens gradually. Such a gradual process co-evolves with the epidemic spreading process shown in Fig. 5(b), and the interplay between the two processes may lead to considerable impact to the final results. Indeed, as shown in Fig. 5(d), the predictions of EDT for the $\rho_R(\infty)$ excellently match with the simulation results, while the HMF overestimates the results for about 20%.

V. CONCLUSION

In summary, we proposed a model of concurrent three-state spreading dynamics on multiplex networks. In this model, a multiplex network is composed of two layers, where one is the awareness layer described by the unaware-aware-passive (UAP) process and the other one is the epidemic layer described by the susceptible-infected-refractory (SIR) process. An interesting point of this model is the introduction of three-state UAP awareness process to describe individuals' response to the epidemics. We remark that the effect of individual awareness of epidemics has also been studied in single layered network where different types of awareness processes and degree-dependent infection rates are considered [41], [42]. These studies reflect the wide interest in understanding the effect of awareness in epidemic spreading dynamics. Based on this UAP-SIR model, a continuous-time effective degree theory (EDT) is developed to predict the behavior of the spreading dynamics. For the convenience of discussion, we separate the whole spreading process into four sub-processes, named as infection process, refractory process, awareness process, and passiveness process, respectively. Since the EDT not only considers the dynamical state of individuals but also considers the states of their neighbors, the dynamical correlation is taken into account. An important practice of the EDT for the concurrent spreading dynamics is that in order to address the changing rates of an individual's neighbour from one state to another, several effective rates are defined to account for the changing under mean field approximation which incorporates the mutual influence between the layers. Our results show that the prediction from the EDT provides excellent agreement with Monte Carlo simulation results.

To manifest our theory, we also present the results of the heterogeneous mean field theory (HMF). It is shown that the HMF systematically overestimates the dynamics. The reason of the overestimation comes from the neglecting of dynamical correlation of the dynamics. Specifically, during the spreading process, an infected (aware) individual is more likely to connect with an infected (aware) neighbour because it may be infected (informed) by this neighbour in previous time. If this dynamical correlation is neglected, as performed in HMF, an infected (aware) individual could have more susceptible (unaware) neighbors than expected, which further results in the overestimation of the whole extent of the spreading. We note that the merit of high accuracy in EDT causes greater computational burden than the HMF. To make this approach to be more effective, further improvement of the efficiency of the theory is desired, for example considering the conservation condition to reduce the number of evolution equations or other methods in the network science [43]–[47], which could be our future works.

In this work, the developed EDT captures the transition relations of the dynamical states of individuals and their neighbors, and the governing evolution equations of all the dynamical states are carefully addressed. The excellent match with the simulation results confirms that the EDT could be developed and applied to dynamical processes on complicated multiplex networks. Since the multiplex networks is becoming an even more important topic in complex networks, our work is expected to be useful for the future study of multiplex networks, for example on other spreading dynamics, e. g. opinion spreading dynamics [48] or state-dependent adaptive spreading processes [39].

APPENDIX A
HETEROGENEOUS MEAN FIELD THEORY FOR UAP-SIR MODEL

In the heterogeneous mean field theory, we classify the individuals in to compartments X_jY_k with $X \in \{U, A, P\}$ and $Y \in \{S, I, R\}$, where j and k represent the number of neighbors of XY individuals in the awareness layer and epidemic layer, respectively. The evolution of the whole process could also be divided into four sub-processes, which are infection process, refractory process, awareness process (including the self-awareness process), and passiveness process, respectively, with the corresponding changing rates as β, μ, α (τ), and γ.

Now, we provide the evolution equations of the processes as follows:

$$\frac{dU_jS_k}{dt} = -\bar{\beta}kU_jS_k - \bar{\alpha}jU_jS_k, \tag{8a}$$

$$\frac{dU_jI_k}{dt} = +\bar{\beta}kU_jS_k - \mu U_jI_k - \bar{\alpha}jU_jS_k - \tau U_jI_k, \tag{8b}$$

$$\frac{dU_jR_k}{dt} = +\mu U_jI_k - \bar{\alpha}jU_jR_k, \tag{8c}$$

$$\frac{dA_jS_k}{dt} = -\theta\bar{\beta}kA_jS_k + \bar{\alpha}jU_jS_k - \gamma A_jS_k, \tag{8d}$$

$$\frac{dA_jI_k}{dt} = +\theta\bar{\beta}kA_jS_k - \mu A_jI_k + \bar{\alpha}jU_jI_k + \tau U_jI_k - \gamma A_jI_k, \tag{8e}$$

$$\frac{dA_jR_k}{dt} = +\mu A_jI_k + \bar{\alpha}jU_jR_k - \gamma A_jR_k, \tag{8f}$$

$$\frac{dP_jS_k}{dt} = -\theta\bar{\beta}kP_jS_k + \gamma A_jS_k, \tag{8g}$$

$$\frac{dP_jI_k}{dt} = +\theta\bar{\beta}kP_jS_k - \mu P_jI_k + \gamma A_jI_k, \tag{8h}$$

$$\frac{dP_jR_k}{dt} = +\mu P_jI_k + \gamma A_jR_k, \tag{8i}$$

where $\bar{\alpha}$ and $\bar{\beta}$ are given as

$$\bar{\alpha} = \frac{\sum_{j'k'} \alpha(A_{j'}S_{k'} + A_{j'}I_{k'} + A_{j'}R_{k'})j'}{\sum_{k'} k'p_{k'}}, \tag{9a}$$

$$\bar{\beta} = \frac{\sum_{j'k'} \beta(U_{j'}I_{k'} + A_{j'}I_{k'} + P_{j'}I_{k'})k'}{\sum_{k'} k'q_{k'}}. \tag{9b}$$

For the convenience of representation, an effective awareness (infection) rate $\bar{\alpha}$ ($\bar{\beta}$) is introduced to describe the variation in the awareness (infection) process, which is defined in Eq. (9a) [Eq. (9b)]. In Eq. (9b), the term $\sum_{j'k'}(U_{j'}I_{k'} + A_{j'}I_{k'} + P_{j'}I_{k'})k'$ is proportional to the probability that a randomly chosen link in the epidemic layer connects to an infected individual, and the denominator $\sum_{k'} k'q_{k'}$ severs for the normalization condition. Multiplying this probability by β gives the possibility that an infection event happens through a randomly chosen link in the epidemic layer. Similarly, the effective awareness rate $\bar{\alpha}$ represents the possibility that an awareness event happens through a randomly chosen link in the awareness layer.

Therefore, one may find that the first term on the right hand side of Eq. (8c) describes the incremental rate of $U_jI_k \to U_jR_k$ due to the refractory process, and the second term on the right hand side of Eq. (8c) describes the decremental rate of U_jR_k due to the awareness process. Other terms in Eq. (8) could be read in similar way, and all these processes correspond to the their respective changing rates, i.e. $\bar{\beta}$, μ, $\bar{\alpha}$, γ, and τ.

REFERENCES

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

[2] M. Kivelä, A. Arenas, M. Barthelemy, J. P. Gleeson, Y. Moreno, and M. A. Porter, "Multilayer networks," *J. Complex Netw.*, vol. 2, p. 203, 2014.

[3] S. Boccaletti, G. Bianconi, R. Criado, C. I. del Genio, J. Gómez-Gardeñes, M. Romance, I. Sendiña-Nadal, Z. Wang, and M. Zanin, "The structure and dynamics of multilayer networks," *Phys. Rep.*, vol. 544, no. 1, pp. 1–122, 2014.

[4] N. T. J. Bailey, *The Mathematical Theory of Infectious Diseases*. Berlin, Germany: Springer, 1975.

[5] R. M. May and R. M. Anderson, *Infectious Diseases of Humans: Dynamics and Control*, 1st ed. Oxford, U.K.: Oxford Univ. Press, 1991.

[6] Z. Yin-Zuo, L. Zong-Hua, and Z. Jie, "Periodic wave of epidemic spreading in community networks," *Chin. Phys. Lett.*, vol. 24, no. 2, p. 581, 2007.

[7] Y. Zhou and Y. Xia, "Epidemic spreading on weighted adaptive networks," *Phys. A, Stat. Mech. Appl.*, vol. 399, pp. 16–23, Apr. 2014.

[8] Y. Yao and Y. Zhou, "Epidemic spreading on dual-structure networks with mobile agents," *Phys. A, Stat. Mech. Appl.*, vol. 467, pp. 218–225, Feb. 2017.

[9] Y. Zhou and J. Zhou, "Algorithm for multiplex network generation with shared links," *Phys. A, Stat. Mech. Appl.*, vol. 509, pp. 945–954, Nov. 2018.

[10] Y. Zhou, "A modified algorithm of multiplex networks generation based on overlapped links," *Phys. A, Stat. Mech. Appl.*, vol. 514, pp. 435–442, Jan. 2019.

[11] M. E. J. Newman, S. H. Strogatz, and D. J. Watts, "Random graphs with arbitrary degree distributions and their applications," *Phys. Rev. E, Stat. Phys. Plasmas Fluids Relat. Interdiscip. Top.*, vol. 64, Jul. 2001, Art. no. 026118.

[12] M. E. J. Newman, "Spread of epidemic disease on networks," *Phys. Rev. E, Stat. Phys. Plasmas Fluids Relat. Interdiscip. Top.*, vol. 66, Jul. 2002, Art. no. 016128.

[13] M. J. Keeling, "The effects of local spatial structure on epidemiological invasions," *Proc. Roy. Soc. London. B, Biol. Sci.*, vol. 266, no. 1421, pp. 859–867, 1999.

[14] R. Pastor-Satorras and A. Vespignani, "Epidemic spreading in scale-free networks," *Phys. Rev. Lett.*, vol. 86, p. 3200, Apr. 2001.

[15] R. Pastor-Satorras and A. Vespignani, "Epidemic dynamics and endemic states in complex networks," *Phys. Rev. E, Stat. Phys. Plasmas Fluids Relat. Interdiscip. Top.*, vol. 63, May 2001, Art. no. 066117.

[16] Y. Moreno, R. Pastor-Satorras, and A. Vespignani, "Epidemic outbreaks in complex heterogeneous networks," *Eur. Phys. J. B-Condens. Matter Complex Syst.*, vol. 26, no. 4, pp. 521–529, 2002.

[17] E. Volz and Meyers, "Susceptible–infected–recovered epidemics in dynamic contact networks," *Proc. Roy. Soc. London. B, Biol. Sci.*, vol. 274, no. 1628, pp. 2925–2934, 2007.

[18] E. Volz, "SIR dynamics in random networks with heterogeneous connectivity," *J. Math. Biol.*, vol. 56, no. 3, pp. 293–310, 2008.

[19] P. Neal, "Coupling of two SIR epidemic models with variable susceptibilities and infectivities," *J. Appl. Probab.*, vol. 44, no. 1, pp. 41–57, 2007.

[20] F. Ball and P. Neal, "Network epidemic models with two levels of mixing," *Math. Biosci.*, vol. 212, no. 1, pp. 69–87, 2008.

[21] Y. Shang, "A lie algebra approach tosusceptible-infected-susceptible epidemics," *Electron. J. Differ. Equ.*, vol. 2012, no. 233, pp. 1–7, 2012.

[22] Y. Shang, "Analytical solution for an in-host viral infection model with time-inhomogeneous rates," *Acta Phys. Polonica B*, vol. 46, no. 8, pp. 1567–1577, 2015.

[23] A. Saumell-Mendiola, M. Á. Serrano, and M. Boguñá, "Epidemic spreading on interconnected networks," *Phys. Rev. E, Stat. Phys. Plasmas Fluids Relat. Interdiscip. Top.*, vol. 86, Aug. 2012, Art. no. 026106.

[24] O. Yağan, D. Qian, J. Zhang, and D. Cochran, "Conjoining speeds up information diffusion in overlaying social-physical networks," *IEEE J. Sel. Areas Commun.*, vol. 31, no. 6, pp. 1038–1048, Jun. 2013.

[25] F. D. Sahneh and C. Scoglio, "Competitive epidemic spreading over arbitrary multilayer networks," *Phys. Rev. E, Stat. Phys. Plasmas Fluids Relat. Interdiscip. Top.*, vol. 89, Jun. 2014, Art. no. 062817.
[26] S. Funk and V. A. A. Jansen, "Interacting epidemics on overlay networks," *Phys. Rev. E, Stat. Phys. Plasmas Fluids Relat. Interdiscip. Top.*, vol. 81, Mar. 2010, Art. no. 036118.
[27] V. Marceau, P.-A. Noël, L. Hébert-Dufresne, A. Allard, and L. J. Dubé, "Modeling the dynamical interaction between epidemics on overlay networks," *Phys. Rev. E, Stat. Phys. Plasmas Fluids Relat. Interdiscip. Top.*, vol. 84, Aug. 2011, Art. no. 026105.
[28] H.-J. Li and L. Wang, "Multi-scale asynchronous belief percolation model on multiplex networks," *New J. Phys.*, vol. 21, Jan. 2019, Art. no. 015005.
[29] J. Sanz, C.-Y. Xia, S. Meloni, and Y. Moreno, "Dynamics of interacting diseases," *Phys. Rev. X*, vol. 4, Oct. 2014, Art. no. 041005.
[30] M. M. Danziger, I. Bonamassa, S. Boccaletti, and S. Havlin, "Dynamic interdependence and competition in multilayer networks," *Nature Phys.*, vol. 15, pp. 178–185, Nov. 2019.
[31] C. Granell, S. Gómez, and A. Arenas, "Dynamical interplay between awareness and epidemic spreading in multiplex networks," *Phys. Rev. Lett.*, vol. 111, Sep. 2013, Art. no. 128701.
[32] S. Gómez, A. Arenas, J. Borge-Holthoefer, S. Meloni, and Y. Moreno, "Discrete-time Markov chain approach to contact-based disease spreading in complex networks," *Euro. Phys. Lett.*, vol. 89, no. 3, p. 38009, 2010.
[33] S. Gómez, J. Gómez-Gardeñes, Y. Moreno, and A. Arenas, "Nonperturbative heterogeneous mean-field approach to epidemic spreading in complex networks," *Phys. Rev. E, Stat. Phys. Plasmas Fluids Relat. Interdiscip. Top.*, vol. 84, Sep. 2011, Art. no. 036105.
[34] Y. Zhou, J. Zhou, G. Chen, and H. E. Stanle, "Effective degree theory for awareness and epidemic spreading on multiplex networks," *New J. Phys.*, vol. 21, Mar. 2019, Art. no. 035002.
[35] C.-R. Cai, Z.-X. Wu, and J.-Y. Guan, "Effective degree Markov-chain approach for discrete-time epidemic processes on uncorrelated networks," *Phys. Rev. E, Stat. Phys. Plasmas Fluids Relat. Interdiscip. Top.*, vol. 90, Nov. 2014, Art. no. 052803.
[36] J. Ma, P. van Driessche, and F. Willeboordse, "Effective degree household network disease model," *J. Math. Biol.*, vol. 66, pp. 75–94, Jan. 2013.
[37] J. Lindquist, J. Ma, P. van den Driessche, and F. H. Willeboordse, "Effective degree network disease models," *J. Math. Biol.*, vol. 62, no. 2, pp. 143–164, 2011.
[38] J. P. Gleeson, "High-accuracy approximation of binary-state dynamics on networks," *Phys. Rev. Lett.*, vol. 107, Aug. 2011, Art. no. 068701.
[39] J. Zhou, G. Xiao, and G. Chen, "Link-based formalism for time evolution of adaptive networks," *Phys. Rev. E, Stat. Phys. Plasmas Fluids Relat. Interdiscip. Top.*, vol. 88, Sep. 2013, Art. no. 032808.
[40] L. E. C. Rocha, F. Liljeros, and P. Holme, "Simulated epidemics in an empirical spatiotemporal network of 50,185 sexual contacts," *PLoS Comp. Biol.*, vol. 7, Mar. 2011, Art. no. e1001109.
[41] Y. Shang, "Modeling epidemic spread with awareness and heterogeneous transmission rates in networks," *J. Biol. Phys.*, vol. 39, no. 3, pp. 489–500, 2013.
[42] Y. Shang, "Degree distribution dynamics for disease spreading with individual awareness," *J. Syst. Sci. Complex.*, vol. 28, pp. 96–104, Feb. 2015.
[43] Z. Bu, H.-J. Li, J. Cao, Z. Wang, and G. Gao, "Dynamic cluster formation game for attributed graph clustering," *IEEE Trans. Cybern.*, vol. 49, no. 1, pp. 328–341, Jan. 2019.
[44] Z. Bu, J. Cao, H.-J. Li, G. Gao, and H. Tao, "GLEAM: A graph clustering framework based on potential game optimization for large-scale social networks," *Knowl. Inf. Syst.*, vol. 55, no. 3, pp. 741–770, 2018.
[45] H.-J. Li, Z. Bu, Y. Lia, Z. Zhang, Y. Chu, G. Li, and J. Cao, "Evolving the attribute flow for dynamical clustering in signed networks," *Chaos, Solitons Fractals*, vol. 110, pp. 20–27, May 2018.
[46] H.-J. Li, Z. Bu, Z. Wang, J. Cao, and Y. Shi, "Enhance the performance of network computation by a tunable weighting strategy," *IEEE Trans. Emerg. Topics Comput. Intell.*, vol. 2, no. 3, pp. 214–223, Jun. 2018.
[47] H.-J. Li, Z. Bu, A. Li, Z. Liu, and Y. Shi, "Fast and accurate mining the community structure: Integrating center locating and membership optimization," *IEEE Trans. Knowl. Data Eng.*, vol. 28, no. 9, pp. 2349–2362, Sep. 2016.
[48] Y. Shang, "Deffuant model of opinion formation in one-dimensional multiplex networks," *J. Phys. A, Math. Gen.*, vol. 48, no. 39, 2015, Art. no. 395101.

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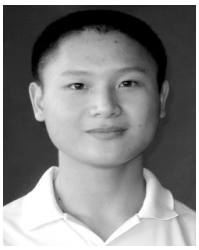

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