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Structural diversity effects of multilayer networks on the threshold of interacting epidemics

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HIGHLIGHTS

- We introduce the "top–bottom" framework to define multilayer networks.
- We use the framework to solve collaboration–competition coexisting epidemic model.
- We introduce three diversity indicators, i.e. richness, evenness, and likeness.
- Both level and type of network diversity affect the epidemic dynamics.
- Transmission and collaboration are trade-off in diverse multilayer networks.

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ABSTRACT

Foodborne diseases always spread through multiple vectors (e.g. fresh vegetables and fruits) and reveal that multilayer network could spread fatal pathogen with complex interactions. In this paper, first, we use a "top-down analysis framework that depends on only two distributions to describe a random multilayer network with any number of layers. These two distributions are the overlaid degree distribution and the edge-type distribution of the multilayer network. Second, based on the two distributions, we adopt three indicators of multilayer network diversity to measure the correlation between network layers, including network richness, likeness, and evenness. The network richness is the number of layers forming the multilayer network. The network likeness is the degree of different layers sharing the same edge. The network evenness is the variance of the number of edges in every layer. Third, based on a simple epidemic model, we analyze the influence of network diversity on the threshold of interacting epidemics with the coexistence of collaboration and competition. Our work extends the "top-down" analysis framework to deal with the more complex epidemic situation and more diversity indicators and quantifies the trade-off between thresholds of inter-layer collaboration and intra-layer transmission.

1. Introduction

Over the last decade, the use of the network has been proved to be a powerful approach to model the structural complexity of complex systems [1–5]. A large body of theoretical literature discusses how network structures may shape the spread of infectious diseases and influence the design of optimal control strategies [6–8]. Such works usually focus on the single network and isolated epidemics. In most of the real-world complex systems, however, nodes in the system

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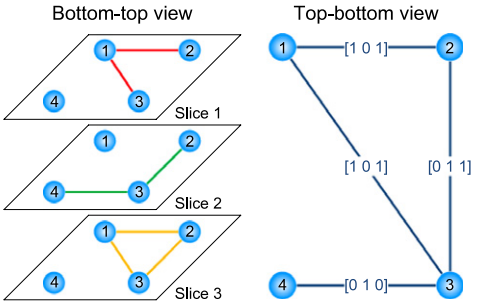

Fig. 1. Comparison of "bottom-up" and "top-down" frameworks, and illustration of network diversity. In the bottom-up framework, the multilayer network is described by the degree distribution of each layer. The degree distribution of layer 1 is $p_1(0) = 0.25$; $p_1(1) = 0.5$; $p_1(2) = 0.25$, that of layer 2 is $p_1(0) = 0.25$; $p_1(1) = 0.5$; $p_1(2) = 0.25$, and that of layer 3 is $p_3(0) = 0.25$; $p_3(2) = 0.75$. In the top-down framework, the multilayer network is described by an overlaid degree distribution $[p(1) = 0.25; p(2) = 0.5; p(3) = 0.25]$ and an edge-type distribution $[q([010]) = 0.25; q([011]) = 0.25; q([101]) = 0.5]$. Furthermore, the richness of the multilayer network is 3; the 'likeness' is 1.75 (because there are three overlaid edges constructed from two intra-layer edges and one overlaid edge constructed from one individual edge), and the evenness is 0.98 (because there are in total seven intra-layer edges, with layers 1 and 2 containing two edges each and layer 3 containing three edges).

can engage in multiple interactions or edges. For example, in the global trade system, countries interact via various trade channels ranging from agricultural products to electronic products [1]. Additionally, in society people interact via their friendships, family relationships and/or more formal work-related links [9].

The recent infection of enterohemorrhagic Escherichia coli (EHEC) in Europe and Listeria monocytogenes in the United States poses a new challenge to the single network approach. The unique network for several reasons is not an appropriate one to explore such diseases mediated by trade networks. First, the trade-mediated epidemics involve multiple vectors to spread single viral agent, and each vector could form a particular network layer to link the same set of countries and territories [6,7,10,11]. Second, the viral agent involves complex interactions between multiple vectors, including both collaborating interaction [12–14] and competitive interaction [6,15,16]. With collaborating interaction, infection in one network layer can cause infecting processes on other layers within a node. Contrarily, competitive interaction (e.g. leaky partial immunity) leads to infection from one network layer reducing transmitting probability from or to other layers. These new features and uncertainties affect our ability to assess the global behavior of trade-mediated diseases and control their diffusion.

Many studies have focused on diffusion dynamics on multilayer networks with two layers, because of an initial interest in interdependence or overlay of two network layers [6,7,10]. However, it is notable that only a few previous studies of diffusion dynamics in multilayer networks study more than two types of edge [8] and understanding of the generic effects of diversity on diffusion behavior remains unclear. Recently, Paczuski et al. [8] considered percolation on interdependent locally treelike networks and the method can be conveniently extended to any number of interdependent networks. A serial of papers has studied multiple types of diseases competitively spread over one or more networks [17]. These studies have provided pioneering insights into multilayer networks, showing that the presence of more than one type of edge, or channel of interactions, in a network can lead to nontrivial effects.

In previous studies, although the specific contexts are various, we can regard each type of edge as a network layer [18], and then these studies can be described by a uniform framework at the mathematical level. A multilayer network can be represented by the overlay of multiple network layers (Fig. 1), with a set of r network layers. Each layer connects the same number N of nodes with their set of edges. Each infectious agent is represented by one node in all r network layers while its neighbors are independent for each of the layers. Researchers have tended to adopt the "bottom-up" framework for multilayer networks [18]. Here, "bottom" means the properties of each network layer, and "up" means the integration of these properties to describe and analyze the characteristics of entire multilayer networks. Usually, a multilayer network is defined by the degree distribution of each network layer and the correlation between all pairs of degree distributions. In our previous work [19], we present a different framework to study multilayer networks, i.e. the "top-down" analysis framework. The framework directly uses a few overall properties to define multilayer networks, and we can deduce the properties of each network layers from the definition if necessary. In this work, we have used the framework to reveal the effect of collaborating patterns on simple interaction epidemics on multilayer networks. However, the ability of the "top-down" should be evaluated in the more complex situation, especially with more interaction types.

One of the most key structural properties of multilayer networks is the relationship between layers, and degree distribution correlation is the major metric to the relationship. However, the degree correlation is restricted to deal with two network layers. Therefore, a general model and solution of epidemic dynamics in multilayer networks with more than two layers remain rare. Additionally, structural diversity has been recognized as another critical factor in control network dynamics [20–24]. However, the study of the diversity of multiple network layers is still in their initial stage. In our previous work [19],we use evenness and likeness of two network layers to evaluate the effect of diversity to collaborating epidemics. However, the paper still do not consider the effect of richness (i.e. the number of network layers) to interacting epidemics.

In this paper, we extend the "top-down" analysis framework to epidemic model with the coexistence of collaborating and competitive interactions. We also consider three indicators to measure the different types of "network diversity" including

richness, likeness, and evenness. Based on this framework, we compare the influence of network diversity on the thresholds of two diffusion parameters.

2. Method

2.1. Top-down view of multilayer networks

Let us first describe the top-down view of the multilayer backbone in which viral agents diffuse. We define the adjacency matrix A of the multilayer network to describe the overlay of all r network layers. In the matrix, $A_{ij} = 1$ when nodes v_i and v_j connect within at least one network layer and $A_{ij} = 0$ otherwise. $A_{ij} = 1$ indicates the existence of a multilayer edge between two nodes (Fig. 1). A given node v_i is connected to $k_i = \sum_j A_{ij}$ other nodes; k_i is the overlaid degree of the multilayer network. Thus, the overlaid degree distribution of the multilayer network, $p(k)$, indicates the ratio of nodes with degree k. Based on probability generating function, the overlaid degree is:

$$P(x) = \sum_k p(k)x^k, \tag{1}$$

where $P(1) = 1$. This function encapsulates all about the overlaid degree distribution of the multilayer network.

We also need to consider the type of multilayer edges [25]. Here, the type of a multilayer edge $\langle v_i, v_j\rangle$ is defined as an r-length vector $[t_1, t_2, \ldots, t_r]$, where $t_\tau = 1$ indicates that layer τ has a edge connecting v_i and v_j, otherwise $t_\tau = 0$ indicates that the edge $\langle v_i, v_j\rangle$ does not exist in layer τ. The probability of finding an edge with given type $T = [t_1, t_2, \ldots, t_r]$ is $q(T)$, and we know that $q([0, 0, \ldots, 0]) = 0$ because there is at least one layer involved in each multilayer edge. Therefore, a multivariate probability generating function can be used to describe the multivariate distribution $q(T)$:

$$\begin{aligned} Q(X) &= Q(x_1, x_2, \ldots, x_r) \\ &= \sum_{t_1=0}^{1}\sum_{t_2=0}^{1}\cdots\sum_{t_r=0}^{1} q([t_1, t_2, \ldots, t_r])x_1^{t_1}x_2^{t_2}\cdots x_r^{t_r}, \end{aligned} \tag{2}$$

where $Q(\mathbf{1}) = 1$. It is a top-down view, which focuses on the overall degree and edge type of multilayer networks.

We can use the overlaid degree distribution and the edge-type distribution to deduce the degree distribution of a single layer $P_\tau(x_\tau)$:

$$P_\tau(x_\tau) = P\left(\frac{\partial Q}{\partial x_\tau}(\mathbf{1})x_\tau + 1 - \frac{\partial Q}{\partial x_\tau}(\mathbf{1})\right), \tag{3}$$

where $\frac{\partial Q}{\partial x_\tau}(\mathbf{1})$ is the probability of layer τ involved in a multilayer edge.

By setting $x_1 = x_2 = \cdots = x_r = x$, we can obtain a univariate probability generating function for the number of layers involved in a multilayer edge, and we denote this operation by U:

$$\begin{aligned} U(Q(X)) &= S(x) \\ &= \sum_{h=1}^{r}\left(x^h \sum_{t_1+t_2+\cdots+t_r=h} q([t_1, t_2, \ldots, t_r])\right), \end{aligned} \tag{4}$$

where $S(1) = 1$.

Note that we will use some language from bottom-up view (e.g. network layer) in the following text for describing models and algorithms, because it is easy to understand in this way.

2.2. Three indicators of multilayer network diversity

Based on the top-down view of a multilayer network, we can quantify the relationship between all network layers by using diversity theory, which originates in the study of ecology, evolution, and genetics. Diversity theory quantifies the differences and relationships among multiple species co-existing in an ecosystem [26]. In diversity theory, a series of statistical indicators (including richness, likeness, and evenness) is developed to greatly improving the understanding of ecological and biological systems [26].

Here, we introduce three indicators to describe the diversity of multilayer networks.

The first indicator is the richness, which is the number of network layers, r. Although the richness is the most primordial feature of multilayer networks, the effect of the richness is still a lack of clarification.

The second indicator is the likeness, which is the average number of layers involved in each multilayer edge, $l = S'(1)/r$, and describes how network layers share the same multilayer edges. $l \to 0$ indicates that few layers co-appear in a multilayer edge, whereas $l \to 1$ indicates that many layers have the same multilayer edges.

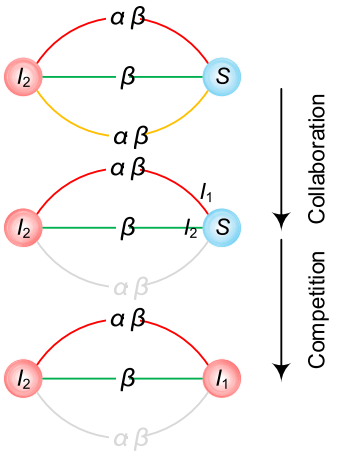

Fig. 2. Process of collaboration–competition coexisting epidemic model. Two nodes connected with each other in three network layers. One node is infected with state I_2 (i.e. infected from layer 2), and the other is susceptible. The actual infection probability is at the corresponding edges in different layers, is collaborating rate and is transmission rate. The process has two steps. In the collaboration step, the infected node transfers the viral agent to the susceptible node through edges in three layers and leads to different infection of viral agents (I_1 and I_2 in the figure). In the competition step, viral agents compete, and a uniformly randomly choosing survivor (i.e. I_1) become infection state of the susceptible node.

The third indicator is the evenness, which describes the distinction among network layers and is based on the format of Pielou's evenness index:

$$e = \frac{-\sum_{\tau=1}^{r} w_\tau \ln w_\tau}{\ln r}, \tag{5}$$

where w_τ is the proportion of edges belonging to the τth layer ($=P'_\tau(1)$) in edges of all layers ($=r \ln P'(1)/2$).

Taking the three diversity indicators (r, l and e) together, we can comprehensively describe the diversity of multilayer networks. The relationship between the level of network diversity and each of the three indicators is different. For richness and evenness, an increase in the indicators represents a rise in network diversity, but an increase in the likeness represents a fall in network diversity.

2.3. Collaboration–competition coexisting model

The diffusion process is one of the most important issues in theoretical and practical studies [3,27,28]. Here, we study epidemic process in a simple manner. In the susceptible–infected (SI) model, each node has two possible states: susceptible (S) or infected (I) [11]. For multiple layers, we extend the infected state into r states, $I_1, I_2, \ldots, I_r$, where an infected state I_τ is viral agent diffused from layer τ. Within a single layer τ, we use the common approach in which nodes represent agents, and the intra-layer edges in the layer between the nodes represent the contacts along which a viral agent can spread. Initially, all nodes are susceptible state except the one infected source with infection I_τ. Then, in discrete time steps, the viral agent can spread in the layer. At each time step t, node v_i infected at the last step $t-1$ with state I_τ could lead an susceptible neighbor node v_j to I_τ with a transmission rate β through the edge of the layer τ. The process halts once there is no further cascading transmission. In this way, the agents can only spread in a single layer; in other words, the layers are independent of each other in spreading the viral agents.

In addition to spreading processes in a single layer, we design a random collaborating mechanism to reflect interactions among multiple layers [29,13]. With collaboration rate α, an infected node v_i can spread the viral agent through edges in all layers to their neighbor v_j regardless of its infected state (Fig. 2). The resulting infected state of v_j corresponds to the layer of the actual transmitting edge. If $\alpha = 0$, network layers are independent of each other in spreading viral agents, whereas if $\alpha = 1$, the division of layers has no effect on the transmission. If a node infects one neighbor through multiple layers simultaneously, we consider a competitive mechanism among viral agents transmitted from different network layers (Fig. 2). We assume that each viral agent will kill others, and uniformly choose at random a potential network layer as the survivor, i.e. infected source. This design of collaborating mechanism adopts a minimalist approach: a node can infect its neighbors with either the edge of a single layer (without collaboration) or the edges of all layers (with collaboration). The design thus considers two extreme situations in the interaction among layers and is a reasonable approach to illustrate the effect of random collaboration. Moreover, we also randomly select the infected state of a susceptible node in the case of spreading through multiple layers from an infected node. It is a reasonable competing interaction to assume; for example, multiple strains of the same disease may compete for hosts according to some cross-immunity profile [30].

According to the integration of collaborating and competitive mechanism, we can compare the effects of the transmission rate β within network layers and the collaboration rate α among network layers with the simple competition.

3. Results and discussion

3.1. Mean-field solution of diffusion model on random multilayer networks

For a single complex network and epidemic models, the degree distribution provides a good foundation for mathematical analysis of giant component size and threshold [2,28]. Here, we will demonstrate how to use the overlaid degree distribution and the edge-type distribution to deduce threshold of collaborating epidemics in multilayer networks through the study of multiple-type branching processes [31,32]. The connection of random multilayer networks is generated by a configuration model according to $P(x)$, and the edge type is randomly chosen according to $Q(X)$. We assume that the size of the network is effectively infinite, and hence the spreading processes form a tree-like infectious component. If a node is infected to state I_τ from one of its neighbors, without collaboration, then the number of neighbors that can be infected to state I_τ from the node are represented by the distribution

$$F_\tau(x_\tau) = R_\tau(\beta x_\tau + 1 - \beta), \tag{6}$$

where $R_\tau(x) = P'_\tau(x)/P'_\tau(1)$ is the distribution of external edges of an infected node in a layer, excluding the edge along with the viral agent arrived.

With collaboration, an infected node can infect its neighbors through all layers. A collaborating node can infect a neighbor to type τ with probability

$$h_\tau = \beta \int N_\tau(\beta x + 1 - \beta)\mathrm{d}x\Big|_{x=1}, \tag{7}$$

where $N_\tau = U(\frac{\partial Q}{\partial x_\tau})$ is the average number of edges other than layer τ in a multilayer edge. Therefore, the number of neighbors that can be infected by a collaborating node to different infected states can be represented by:

$$G(X) = R\left(\sum_{\tau=1}^{r} h_\tau x_\tau + 1 - \sum_{\tau=1}^{r} h_\tau\right). \tag{8}$$

In sum, for a node with state I_τ, the process in which it infects its neighbors is described by:

$$Z_\tau(X) = \alpha G(X) + (1-\alpha)F_\tau(x_\tau). \tag{9}$$

In the language of multi-type branching processes [32], Z_τ determines the distribution of the number of offspring of various infected states produced by an I_τ node. Therefore, the mean matrix of the branching processes defined by Z_τ is

$$M = \begin{bmatrix} \frac{\partial Z_1}{\partial x_1}(1) & \frac{\partial Z_1}{\partial x_2}(1) & \cdots & \frac{\partial Z_1}{\partial x_r}(1) \\ \frac{\partial Z_2}{\partial x_1}(1) & \frac{\partial Z_2}{\partial x_2}(1) & \cdots & \frac{\partial Z_r}{\partial x_r}(1) \\ \cdots & \cdots & \cdots & \cdots \\ \frac{\partial Z_r}{\partial x_1}(1) & \frac{\partial Z_r}{\partial x_2}(1) & \cdots & \frac{\partial Z_r}{\partial x_r}(1) \end{bmatrix}. \tag{10}$$

This mean matrix has the maximal eigenvalue, which is positive and simple. According to the extinction probability in branching processes, if the maximal eigenvalue of M $eig^{\max}(M) > 1$, then the extinction probability of the branching process is less than 1. In this situation, the viral agent can occupy the network to form a giant component, whose size has the same infinite order as the size of the network. Therefore, the condition of spreading threshold of collaborating epidemics on a random multilayer networks is

$$eig^{\max}(M) = 1. \tag{11}$$

Additionally, the survival probability (i.e. the probability of occupying the entire network) when $eig^{\max}(M) > 1$ can be deduced from the following system of equations [32]:

$$\rho = \begin{bmatrix} \rho_1 \\ \rho_2 \\ \vdots \\ \rho_r \end{bmatrix} = Z(X) = \begin{bmatrix} Z_1(\rho) \\ Z_2(\rho) \\ \vdots \\ Z_r(\rho) \end{bmatrix}, \tag{12}$$

where ρ_τ is the extinction probability from a node with I_τ excluding the edge along which the viral agent arrived. By setting $R_\tau(x) = P_\tau(x)$ and $R(x) = P(x)$, we can obtain $Z'(X)$ by repeating the calculation from Eqs. (6), (8), and (9). Therefore, by randomly choosing a node and infected state as index case, the average survival probability is

$$\langle s \rangle = 1 - \frac{\sum_{\tau=1}^{r} Z'_\tau(\rho)}{r}. \tag{13}$$

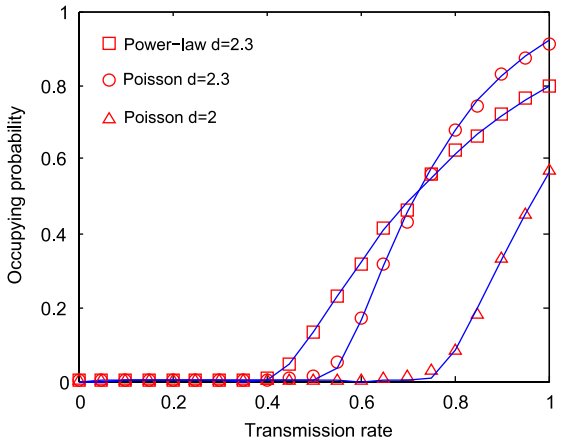

Fig. 3. Validation of the mean-field solution. Simulation results and mean-field solutions are compared for the three networks. The first network is a power-law distribution with mean degree $d = 2.3$, and the other two networks are Poisson distributions with mean degree $d = 2.0$. The points with different shapes represent the results from a Monte Carlo simulation, and the solid lines represent the results from a mean-field solution. The collaboration rate is kept at $\alpha = 1$, while the transmission rate β is varied for calculating occupying probability.

The theoretical threshold and occupying probability are identified from the simulation result (Fig. 3). In summary, adopting the top-down view and the edge-type distribution can provide a mathematical solution of epidemic processes in multilayer networks.

3.2. Diversity effects on minimal collaboration probability

One of the core problems in studying multilayer networks is the role of the structural correlation among layers in shaping dynamic processes. In previous work, Funk and Jansen [6] studied the degree correlation between two network layers. Furthermore, Lee et al. [33] considered the influence of degree correlation on network robustness. However, a general description of correlation among multiple network layers is still absent. In this section, we focus on the effect of diversity on the threshold of both collaboration probability α between layers and the transmission probability β within single layers.

We consider the role of diversity on the minimal collaboration rate (α_m) and transmission rate (β_m) to maintain $eig^{\max}(M) > 1$ with the constant number of total edges in all layers. From the previous section, the survival probability increases monotonically with the increase of either α or β. Therefore we can find α_m (or β_m) by setting $\beta = 1$ (or $\alpha = 1$) and solving the formula $eig^{\max}(M) = 1$ numerically. We evaluate the response of the α_m and β_m to changes in three diversity indicators in random multilayer networks.

First, Fig. 4 shows that the richness and evenness of multilayer networks do not affect the value of β_m. It is a rational result because the calculation of β_m depends on taking $\alpha_m = 1$ (i.e. the viral agent can spread through all layers with probability 1 within all nodes). In this situation, the number of edges in each layer does not affect β_m and only the number of total edges in all layers has the influence on the value. In contrast, the richness and evenness impact the value of α_m with a phase-transition-like character (Fig. 4). When the two indicators are below a "threshold", α_m remains about 0, but when they are larger than the threshold, α_m increases rapidly with the indicators. These results suggest that epidemic dynamics on low-diversity multilayer networks tend to rely on a single layer ($\alpha_m = 0$ indicates that a global infection does not require the collaboration mechanism). However, epidemic dynamics on high-diversity multilayer networks are more dependent on collaboration among layers. Therefore, for low-diversity networks, we should focus on determining the critical layer and controlling the transmission rate within the layer. However, for high-diversity networks, we should pay more attention to controlling the interaction between layers. The measure of network diversity could offer useful guidance to develop effective control strategies.

Second, we find that the effect of likeness on α_m and β_m is distinct to richness or evenness. For transmission rate, larger likeness lead to more edges in different layers linking the same pair of nodes (Fig. 4), and thus raises the value of β_m. For collaboration rate, the effect of likeness on α_m does not have a phase-transition-like character (Fig. 4). If $\alpha_m > 0$ (or $\alpha_m = 0$) when the likeness is equal to 1 (its minimum value), then $\alpha_m > 0$ (or $\alpha_m = 0$) also when the likeness is larger than 1. The result may seem surprising, but it does make sense. $\alpha_m = 0$ when $l = 1$ indicates that one layer can lead to a global epidemics (i.e. $eig^{\max}(M) > 1$), and the change of the likeness does not alter the features. Therefore the value of α_m will remain 0 for $l > 1$. By considering the relationship between three indicators and diversity, we find that the likeness has opposite effects on diffusion dynamics to richness and evenness. In a multilayer network, diseases prefer to high diversity at the aspect of richness or evenness but low diversity at the aspect of likeness. The result suggests that the effect of multilayer diversity is complex, and different aspects of network diversity will lead to opposite influences on diffusion dynamics. Therefore, not only the degree of diversity but also its type is critical to find out effective control strategies for multilayer networks.

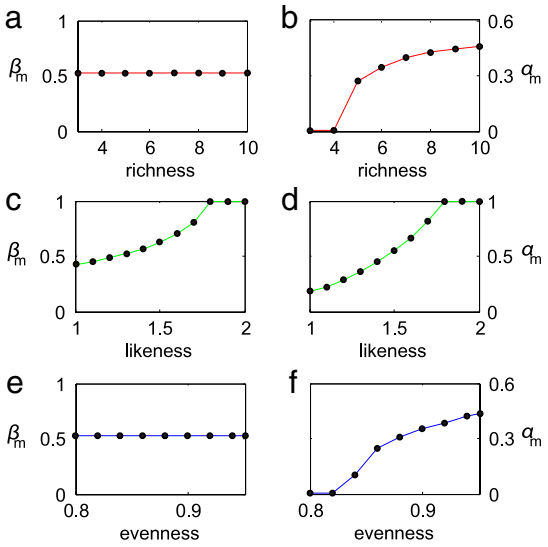

Fig. 4. Effect of diversity on minimum transmission rate (β_m) and minimum collaboration rate (α_m). The curves were calculated for a network with a Poisson degree distribution ($d = 2.3$). When evaluating the effect of one diversity indicator on the minimum transmission and collaboration rates, the other two indicators were kept constant. (a) and (b) Effect of richness: the likeness is 1.3 and the evenness is 0.9; (c) and (d) Effect of likeness: the richness is 6 and the evenness is 0.9; (e) and (f) Effect of evenness: the richness is 6 and the likeness is 1.3. The results are based on the mean-field solution.

3.3. Effect of diversity on trade-off between transmission and collaboration

In addition to the minimum values of the transmission and collaboration rates, we also consider the trade-off between the two rates. We find that, although the richness and evenness do not affect the value of β_m, these two diversity indicators influence the trade-off between the values of α and β (Fig. 5). For example, for a high-richness multilayer networks, the collaboration rate immediately increases with the decrease in the transmission rate. Contrarily, for a low-richness multilayer network, there is a critical value of transmission rate (Fig. 5a). Above the critical value, the collaboration rate does not change with a decrease in the transmission rate, but the collaboration rate begins to increase when the transmission rate is below the critical value. The effect of evenness on the trade-off between the two rates shows the same tendency. Similarly, the likeness affects the trade-off between two rates, but the effect does not have such critical points (Fig. 5b and c). The result suggests that the relationship between the rates in two-scale epidemics dynamics is controlled by network diversity.

4. Conclusions

The top-down framework used in this paper provides an efficient method for analyzing interacting epidemics in multilayer networks. Based on this framework, we have proved that both the level and type of network diversity can affect the threshold and trade-off of transmission and collaboration processes in multilayer networks. This result reverifies the power of the top-down framework and illustrates the role of richness in shaping epidemic processes. Compared with the previous bottom-up framework, our framework provides a more compact definition of multilayer networks, and the compact definition can be a concrete foundation for seeking a mathematical solution of interacting epidemics on multilayer networks [19]. Moreover, for a single complex network, a series of measurements have been developed to describe the different features of a complex network [34], although the global measurements for multilayer networks are still absent. From our proof of the relationship between three indicators and epidemic dynamics on multilayer networks, it appears that these indicators are potentially useful measurements for providing a description of a number of aspects of these networks.

Even so, the current framework also has some limitations. One of these is that one pair of overlaid degree and edge-type distributions cannot describe the variance in the coupling of multiple network layers. The definition of edge-type distribution leads to a description of degree correlation in an average situation. However, it may be possible to solve this problem by adopting a group of pairs of overlaid degree and edge-type distributions. Each pair of distributions describes the set of nodes with the similar type links of the two distributions. Another limitation is about the relationship between nodes degree and the type distribution of their edges. The problem can also be solved by introducing more edge-type distributions. As a result, the middle ground of the "top-down" and "bottom-up" approach might be more powerful to deal with multilayer networks.

In summary, the top-down framework provides a potential way to analyze multilayer networks, and investigation of the effect of diversity provides some global measurements for indicating the properties of multilayer networks.

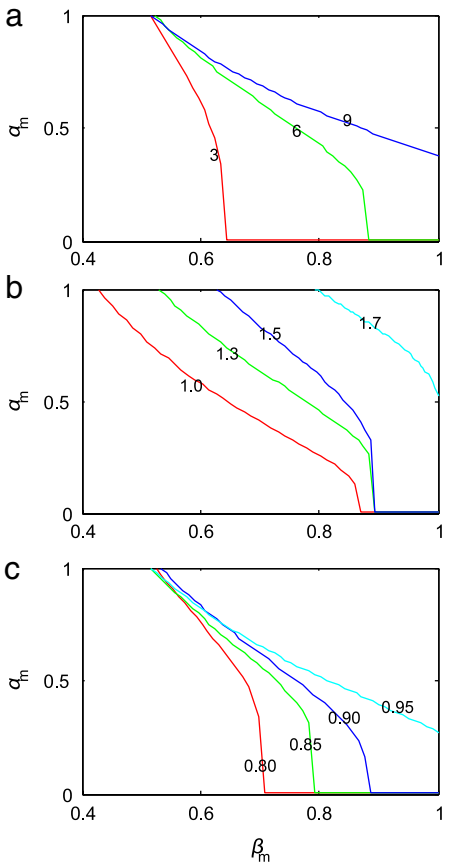

Fig. 5. Effect of diversity on the trade-off between the minimum transmission rate (β_m) and the proliferation rate (α_m). The curves were calculated for a network with a Poisson degree distribution ($d = 2.3$). When evaluating the effect of one diversity indicator on the minimum transmission and proliferation rates, the other two indicators were kept constant. (a) Effect of richness: the likeness is 1.3, and the evenness is 0.9. (b) Effect of likeness: the richness is 6, and the evenness is 0.9; (c) Effect of evenness: the richness is 6, and the likeness is 1.3. The numbers marked on the curves represent the value of the corresponding diversity indicator.

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