International Journal of Modern Physics B
Vol. 34, No. 28 (2020) 2050262 (13 pages)

DOI: 10.1142/S0217979220502628

Quantitative effects of network connectivity on epidemics

Zhenzhen Liu, Xiaoke Xu* and Jianyun Zhou
School of Information and Communication Engineering,
Dalian Minzu University,
Dalian 116600, P. R. China
**xuxiaoke@foxmail.com*

Received 14 May 2020
Revised 7 July 2020
Accepted 13 July 2020
Published 20 October 2020

Epidemics are affected by the connectivity of nodes in networks in addition to the cooperation of infection transmission. We investigate quantitatively the effects of node connectivity on transmission dynamics by comparing epidemic diffusion in null models with gradual connection strength. Results show that: (1) the inhomogeneity of network connectivity accelerates the spreading of epidemics, this phenomenon is more significant in the early stage of propagation; (2) the enhancement of connectivity of homogenous nodes restrains epidemic spreading, and the spreading speed correlates negatively with connection strength; (3) the spreading speed of epidemics does not change linearly with the strength of rich-club property, which means that the connectivity among hub nodes does not appreciably affect disease diffusion.

Keywords: Epidemic spreading; null model; assortativity; rich-club.

PACS numbers: 89.75.-k, 87.23.Ge, 64.60.Ht

1. Introduction

Epidemiology is a very hot topic as various pandemics have always haunted us since time immemorial. In modern societies, epidemics are usually spread through biological contact between individuals. By abstracting biological individuals into nodes and reducing interactions between individuals to edges, disease transmission can be studied based on network science.[1,2] The influence of contact structure of networks on infection process is one of the research focuses. Barabási pointed out that the most important factor that affects epidemics is network topology rather than epidemiological parameters.[1]

*Corresponding author.

When analyzing network structure, we usually start from degree distribution, which can serve as a basis for calculating most other topology features.[3–6] Different degree distributions vary in their effects on the propagation of epidemics. In comparison with homogeneous networks and small-world networks, scale-free (SF) networks with power-law degree distribution are conducive to the spreading of epidemics, where epidemic threshold does not even exist.[7–9]

Degree distribution cannot describe uniquely networks, as other features of networks with the same degree distribution could be very different. To further describe the topology structure of networks, the high-level feature (i.e., degree correlations[10–12] need to be considered. There are three main methods to describe degree correlations: combined probability distribution,[13] excess average degree[14] and assortativity coefficient.[15] Combined probability distribution is complex, and excess average degree is not good at comparing networks with different scales. Although assortativity coefficient is coarser, it can quantitatively describe degree correlations. In this study, we employ assortativity coefficient (Pearson correlation coefficient[1]) to discuss the impact of degree correlations on epidemic spreading. If nodes with high degree tend to connect to other nodes with high degree in a network, the network is called assortative network, whose assortativity coefficient is positive. Otherwise, this network is a disassortative network with negative assortativity coefficient.[15] It can be seen that the interconnection of homogenous nodes, whose degrees are on the same scale, produces assortative networks and disassortative networks result from the connection of heterogeneous nodes. Obviously, degree correlations measure the connectivity of nodes. Assortative mixing is beneficial to the spreading of infection, and leads to a lower epidemic threshold. Conversely, disassortative correlations restrain the diffusion of epidemics, and the higher threshold makes it easier to immunize individuals.[16–19]

Usually, there always exist a small number of nodes which have high degree in a real-world network. The most basic measure of node importance is degree centrality,[20] namely, the higher degree of a node means the higher importance. In this way, the nodes with high degree are important nodes. If these nodes tend to connect to each other, they construct a rich club,[1,21] which is a core sub-network of overall network. Two nodes coming from the rich club can be regarded as homogenous nodes. If two nodes belong to rich club and the reminder of network separately, they are heterogeneous. If degree correlations measure the global connection between nodes of networks, rich-club measures the local connection. Rich-club feature plays a leading role in network properties, such as network routing efficiency, redundancy and robustness. The rich-club property contributes to the spreading of disease, and the high rich-club property leads to a low epidemic threshold.[22,23]

Although the effects of network connectivity on epidemic spreading have been studied, how the change of connectivity strength affects the epidemic spreading remains largely unexplored. In this paper, we focus on the quantitative effects of network connectivity on epidemic spreading. There are two ways to study the influence of network topology on epidemic spreading. The first method is to construct a network model, which stresses an aspect of network topology, and then analyze the impact of this feature on other network properties and network phenomena.[8,24,25] The second is to use null models, which weaken some topology measure of empirical networks, and then examine the role of this feature.[26,27] In order to make connectivity strength vary in a wide range, we employ null models whose connectivity strength can be enhanced or diminished arbitrarily. It is well-known that empirical networks always have inhomogeneous degree distribution. In order to remain this nontrivial property, we introduce null models based on rewiring algorithm, which can maintain the degree distribution sequence of networks. By changing rewiring times, null models with various connectivity strengths are obtained. Then, the spreading speed of epidemics in these null models is counted. The influence of network connectivity on epidemic spreading is analyzed quantitatively by comparing the spreading speed in these null models.

Susceptible-infected (SI) model is employed to simulate the epidemic spreading process. Each individual in networks can only be in susceptible (S) state or infected (I) state at any moment in time. A susceptible individual can be infected by its infected neighbors with some probability.[28–30] In real situations, the more infected neighbors an individual has, the higher probability it is infected. Namely, the infection of a susceptible individual results from the synergistic action of its infected neighbors. Thus, we let the probability that a susceptible individual is infected depends on the number of its infected neighbors.

Our results show that the power-law degree distribution of networks accelerates the spreading of epidemics. Enhancing assortativity restrains the epidemic spreading, and the speed changes linearly with assortativity coefficient. In this case, there is a linear relationship between spreading speed and average shortest path length, and spreading speed decreases linearly with increasing average shortest path length. However, there is also no linear relationship between spreading speed and rich-club strength, and the spreading speed does not change linearly with average shortest path length in this case.

Our research framework, which is used to analyze quantitatively the influencing factors of epidemic spreading in empirical networks, proposes a new paradigm of studying information diffusion. The results of this study lay a foundation for such problems, namely, how to systematically comprehend the impact of network topology on different dynamic processes. Moreover, these results have long-term and short-term theoretical and practical value, as most contact networks involved have a wide degree distribution.

2. Influence of Degree Distribution on Epidemic Spreading

The degree distribution, $P(k)$, which denotes the probability that a randomly chosen node have k adjacent nodes, provides a good basis for further study of network

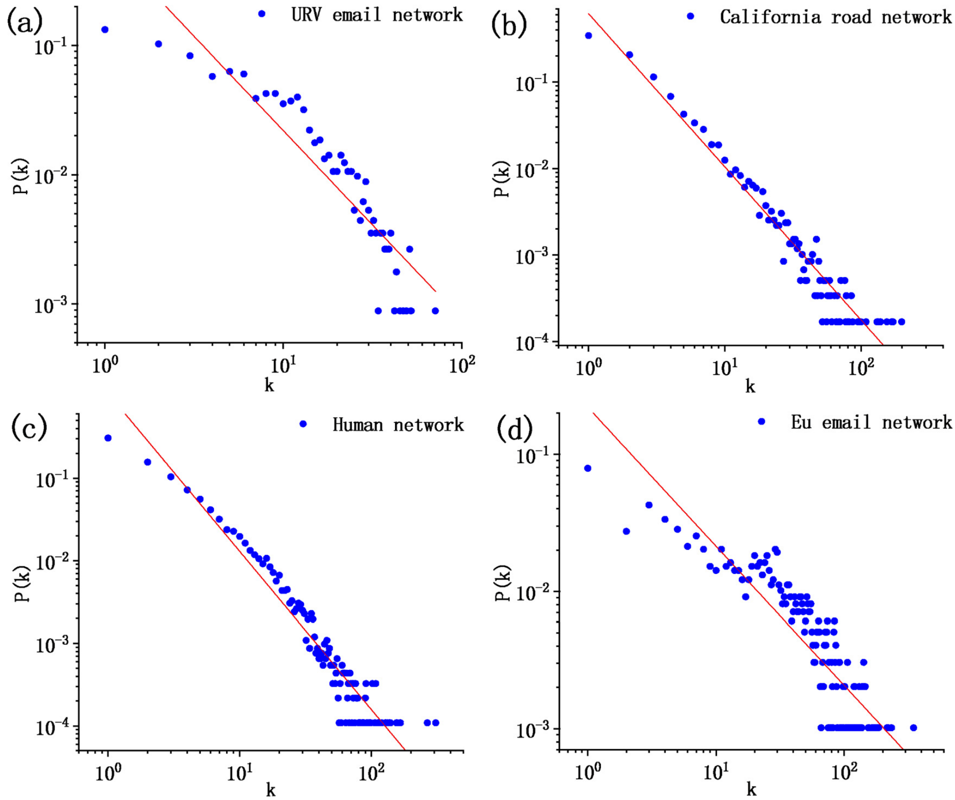

Fig. 1. (Color online) Log–log plot of degree distribution for various networks (dots) and corresponding reference lines (full lines) which indicating the power-law distribution. (a) URV email network. (b) California road network. (c) Human network. (d) EU email network.

characteristics. So, we first analyze the degree distribution of empirical networks, including the email network of University at Rovira i Virgili (hereinafter referred to as URV email network),[31] California road network,[32] BIOGRID human interactome network (hereinafter referred to as Human network)[33] and email network of a large European research institution (hereinafter referred to as EU email network).[34] In Fig. 1, we plot the degree distribution of empirical networks. As shown in Fig. 1, the degree distribution all follows a long-tail distribution. Namely, most nodes have low degree, meanwhile a small number of nodes have high degree. The degree distribution of these networks all follows the power-law distribution, which signifies the SF-property.

In order to analyze the impact of SF degree distribution on epidemic spreading, we introduce 0k null model based on rewiring algorithm. As shown in Fig. 2, the link between two randomly chosen nodes is removed, and at the same time, two disconnected nodes which are randomly chosen are connected. Repeating this process

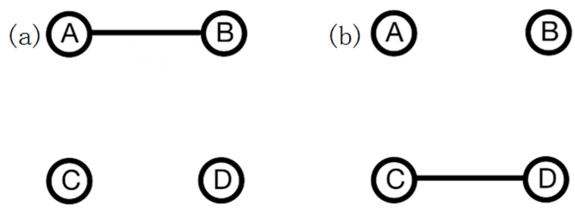

Fig. 2. Rewiring process of a link for 0k null model. (a) In original state, nodes A and B are connected. (b) A and B are disconnected, meanwhile two randomly chosen nodes C and D are connected.

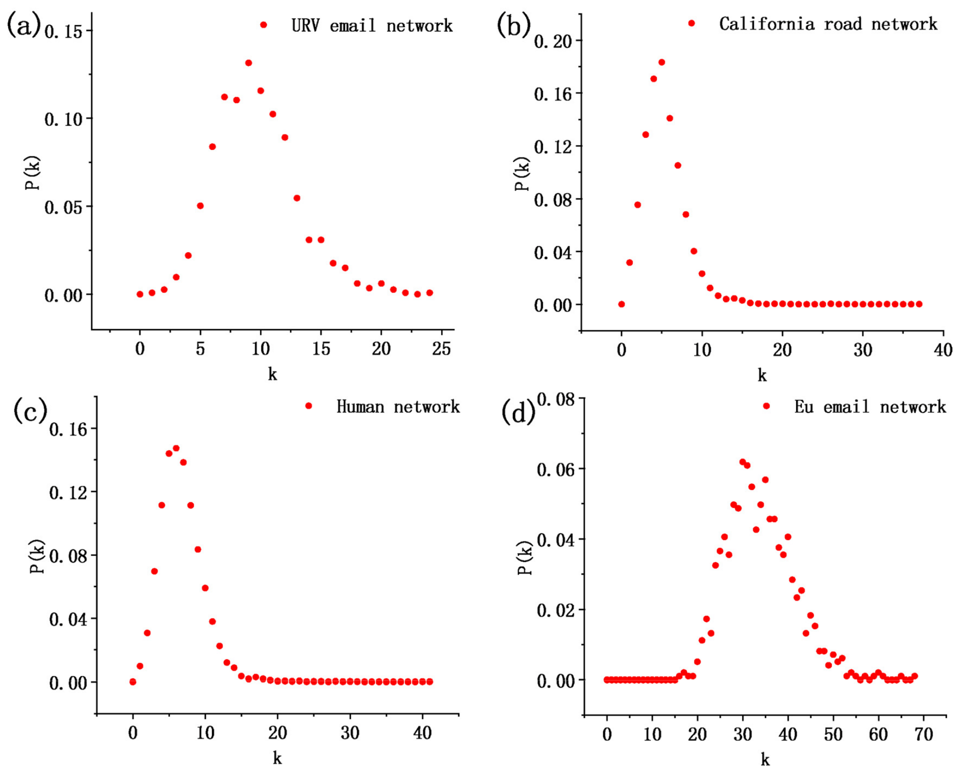

Fig. 3. (Color online) Degree distribution of 0k null models corresponding to (a) URV email network, (b) California road network, (c) Human network and (d) EU email network.

enough times results in 0k null model for an empirical network. It can be seen that 0k null model has the same numbers of nodes and links as original network, but they have different degree sequences. Figure 3 indicates that the degree of 0k null models corresponding to four empirical networks follows the normal distribution. That is, the degree of most nodes is around the peak of degree distribution. This demonstrates that 0k null model has stochastic characteristics.

In the following, we analyze the influence of power-law degree distribution on epidemic spreading by comparing propagation process in empirical networks and corresponding 0k null models. The probability of a susceptible individual is infected by one infected neighbor is denoted as α. In view of synergistic action of infected neighbors, a susceptible individual is infected by its infected neighbors with probability

$$1 - (1 - \alpha)^{k(i)}, \tag{1}$$

where $k(i)$ is the number of its infected neighbors. An infected individual becomes susceptible with probability β. Here, infection rate α is fixed to 0.09, and one individual is randomly selected as the source of infection at initial time. Without lack of generality, we set $\beta = 1$, as it only affected the time scale of the infection evolution.

In Fig. 4, we plot the evolution of fraction of infected individuals $i(t)$ in empirical networks and corresponding 0k null models, respectively. It can be seen that

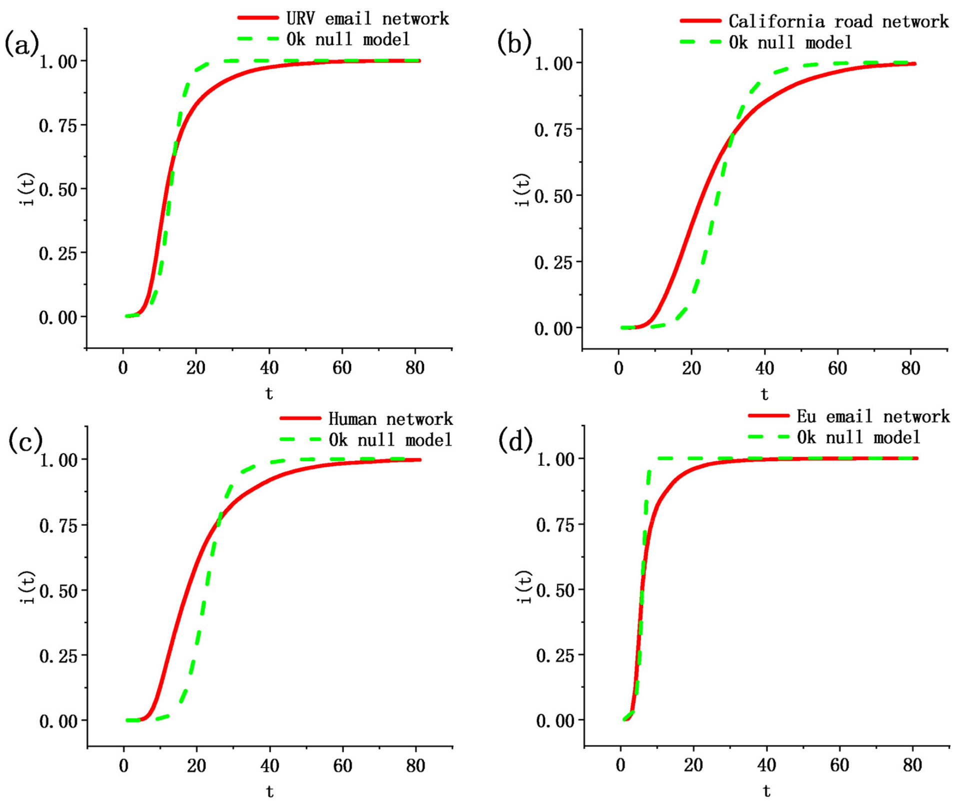

Fig. 4. (Color online) Fraction of infected individuals $i(t)$ as a function of spreading time t for various networks (full lines) and corresponding 0k null models (dotted lines). (a) URV email network. (b) California road network. (c) Human network. (d) EU email network. Results are averaged over 1000 runs.

the time of all nodes infected in 0k null models is shorter than that in empirical networks. The spreading in empirical networks is quicker than that in 0k null models in the initial transient process, and the situation is reversed in the later period. At the early stage, once a node with high degree is infected, it will infect a large number of infected nodes in empirical networks. This leads to the fast spreading of disease. As time goes by, most nodes including ones with high degree are infected, so infected nodes increase more slowly. On the other hand, the average feature of 0k null model makes spreading speed be almost uniform in the whole process.

3. Influence of Degree Correlations on Epidemic Spreading

In order to remain degree distribution sequence, we use rewiring method to build null models with different assortativity strengths. As shown in Fig. 5(a), the degree of nodes A and D is higher than that of nodes B and C in original state. After removing edges (A, C) and (B, D), edges (A, D) and (B, C) are linked [Fig. 5(b)]. In this way, assortativity is enhanced. After repeating this process enough times, we can get an assortative null model. The rewiring operation is aimed at changing the connection property of nodes. Different rewiring times lead to null models with various assortativity strengths. In other words, this rewiring algorithm can change the connection feature of original networks, and precisely adjust assortativity coefficient. Then, these null models can be used to inspect how the assortativity of networks influences epidemic spreading. Here, the assortativity of networks is quantified by Pearson correlation coefficient[15]:

$$r = \frac{M^{-1}\sum_i s_i t_i - \left[M^{-1}\sum_i \frac{1}{2}(s_{i+} t_i)\right]^2}{M^{-1}\sum_i \frac{1}{2}(s_i^2 + t_i^2) - \left[M^{-1}\sum_i \frac{1}{2}(s_{i+} t_i)\right]^2}, \tag{2}$$

where s_i, t_i are the degrees of nodes of the ith edge with $i = 1, \ldots, M$.

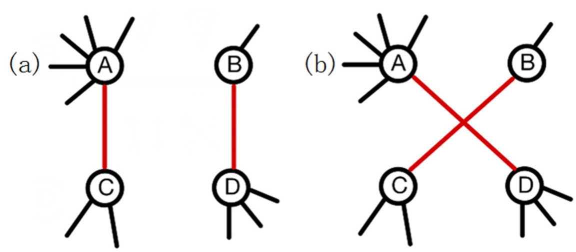

Fig. 5. (Color online) Rewiring process of links for assortativity null model. (a) In original state, nodes A and C are connected, and B and D are connected. A and D have higher degree than B and C. (b) Edges (A, C) and (B, D) are removed. Then, A and D, B and C are connected, respectively.

We set infection rate $\alpha = 0.09$. Initially, one individual is randomly selected as the source of infection. In Fig. 6, spreading speed v is plotted as a function of rewiring times of edges (hereinafter referred to as RT), where v is denoted as

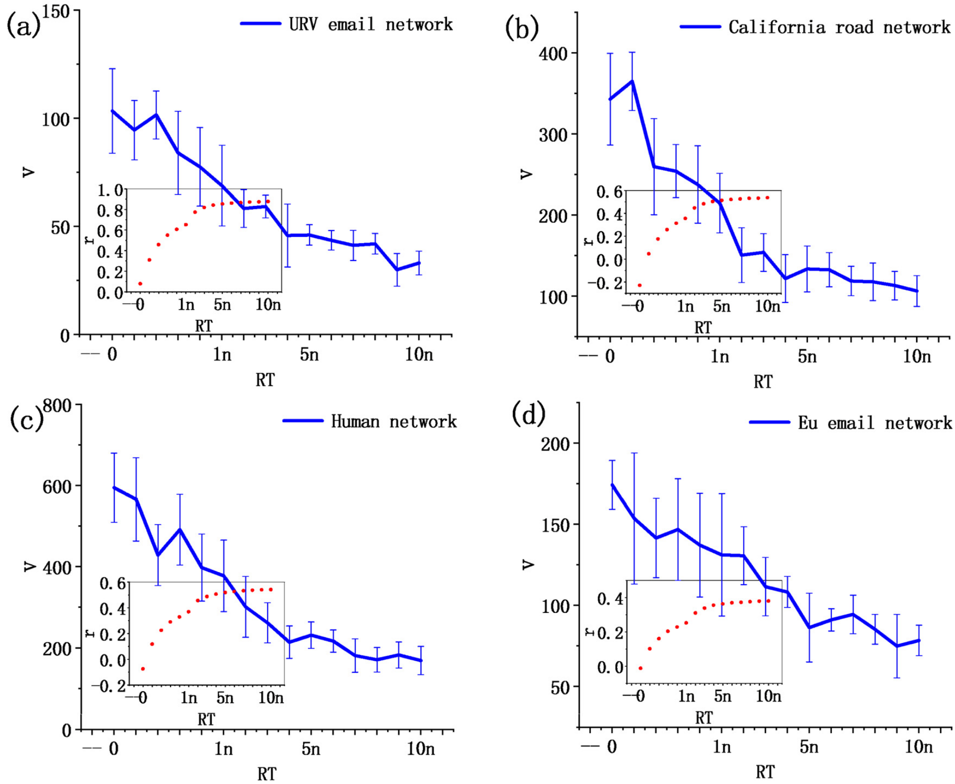

Fig. 6. (Color online) Spread speed v as a function of rewiring times RT for (a) URV email network, (b) California road network, (c) Human network and (d) EU email network. n is twice the number of edges in each network. Inset: Pearson correlation coefficient r as a function of RT. Results are averaged over 1000 runs.

the number of individuals which are infected in a unit time. Meanwhile, Pearson correlation coefficient r is plotted as a function of RT in the insets of Fig. 6. We can see that Pearson correlation coefficient r increases with rewiring times RT. This means that the rewiring operation for enhancing assortativity of networks indeed strengthens the connectivity of homogeneous nodes. At the same time, spreading speed v and RT also show a similar relationship, that is, v decreases with RT. This demonstrates that spreading speed of epidemics decreases with increasing assortativity strength. In brief, the connectivity of nodes in networks and spreading speed of epidemics are strongly correlated (i.e., network connectivity dominates the spreading of infection).

For further study of this issue, we plot the average shortest path length L of null models versus RT in Fig. 7. As shown in Fig. 7, L increases with RT, which is opposite to the change in spreading speed v along with RT (see Fig. 6). Then we plot v versus L in the insets of Fig. 7. The insets show that v and L are highly correlated,

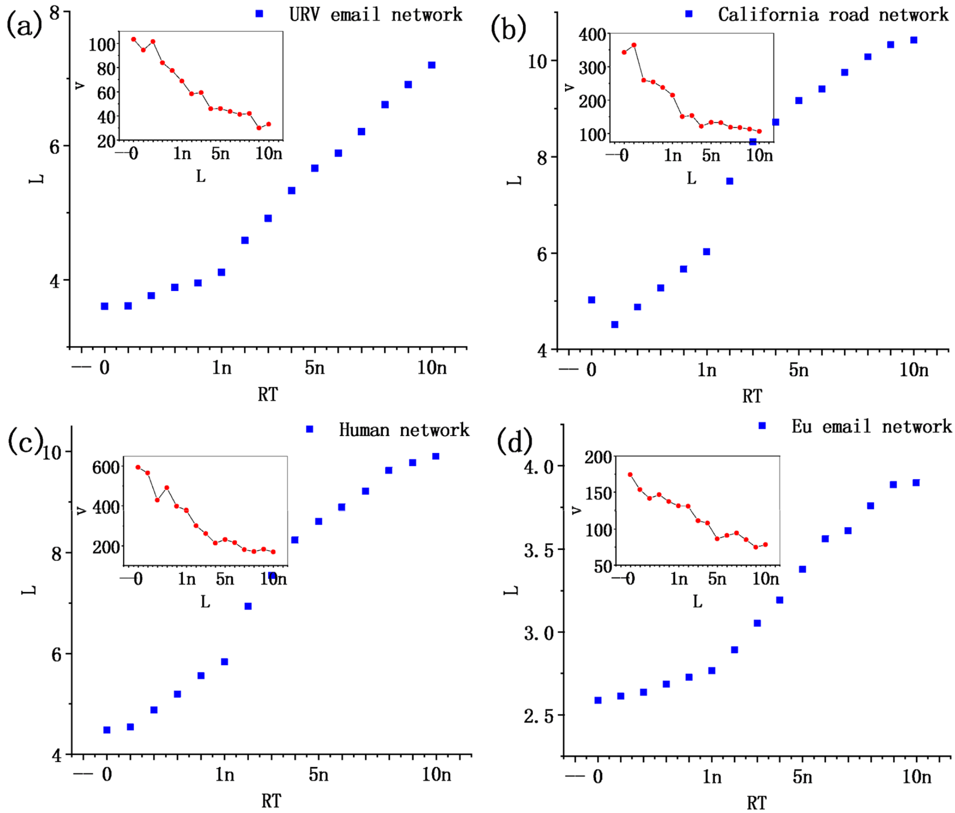

Fig. 7. (Color online) Average shortest path length L as a function of rewiring times RT for (a) URV email network, (b) California road network, (c) Human network and (d) EU email network. n is twice the number of edges in each network. Inset: Spreading speed v as a function of L. Results are averaged over 1000 runs.

and v decreases with L. This means that the enhancement of assortativity increases the average shortest path length of networks, and therefore restrains the spreading speed of epidemics.

4. Influence of Rich-Club on Epidemic Spreading

First, we introduce null models for rich-club property. In Fig. 8(a), nodes A and B have larger degree, namely, they are "rich", and nodes C and D have small degree. There exists two edges (i.e., (A, C) and (B, D) in the initial state). In order to enhance rich-club property, edges (A, C) and (B, D) are removed, and then edges (A, B) and (C, D) are added [see Fig. 8(b)]. After repeating this process enough times, a null model is obtained whose rich-club property is higher than original network. Different repetition times lead to null models with different rich-club strength. Comparing epidemic spreading in these null models, we can get the impact of rich-club property on epidemics in detail.

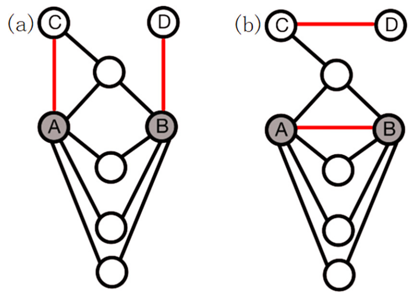

Fig. 8. (Color online) Rewiring process of links for rich-club null model. (a) In original state, nodes A and B in rich-club are connected with C and D which are not 'rich', respectively. (b) Edges (A, C) and (B, D) are removed. Then, A and B, C and D are connected.

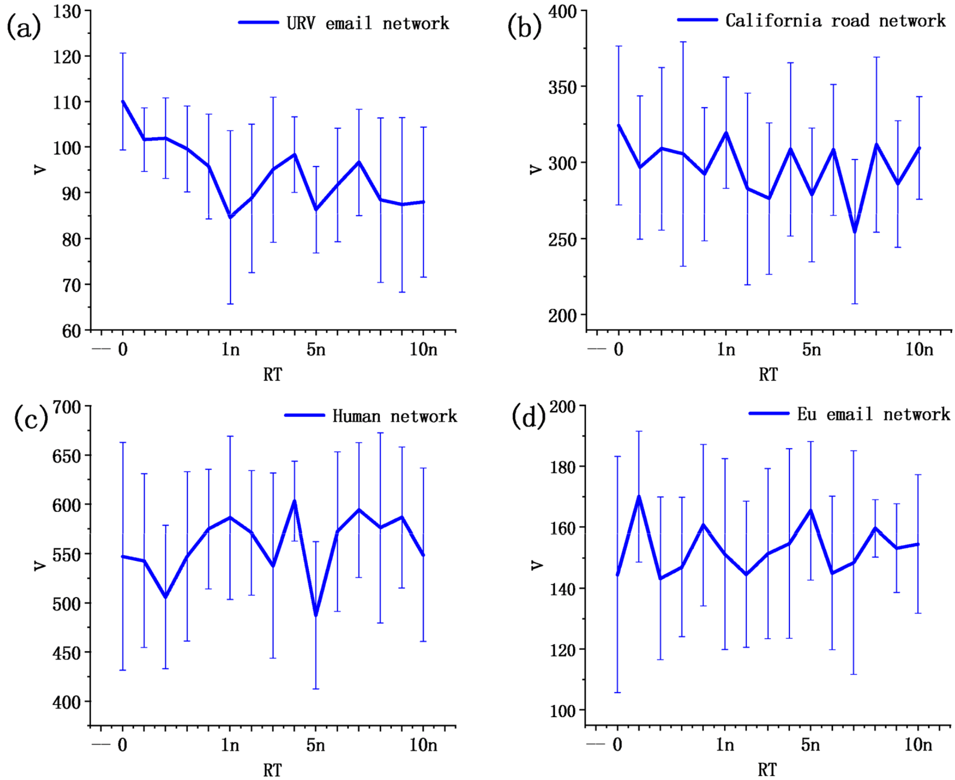

Fig. 9. (Color online) Spread speed v as a function of rewiring times RT for (a) URV email network, (b) California road network, (c) Human network and (d) EU email network. n is twice the number of edges in each network. Results are averaged over 1000 runs.

Initially, one individual is randomly selected as I state, and others are S state. α is set to 0.09. The spreading speed is plotted as a function of rewiring times of edges RT in Fig. 9. As shown in Fig. 9, the spreading speed does not linearly change

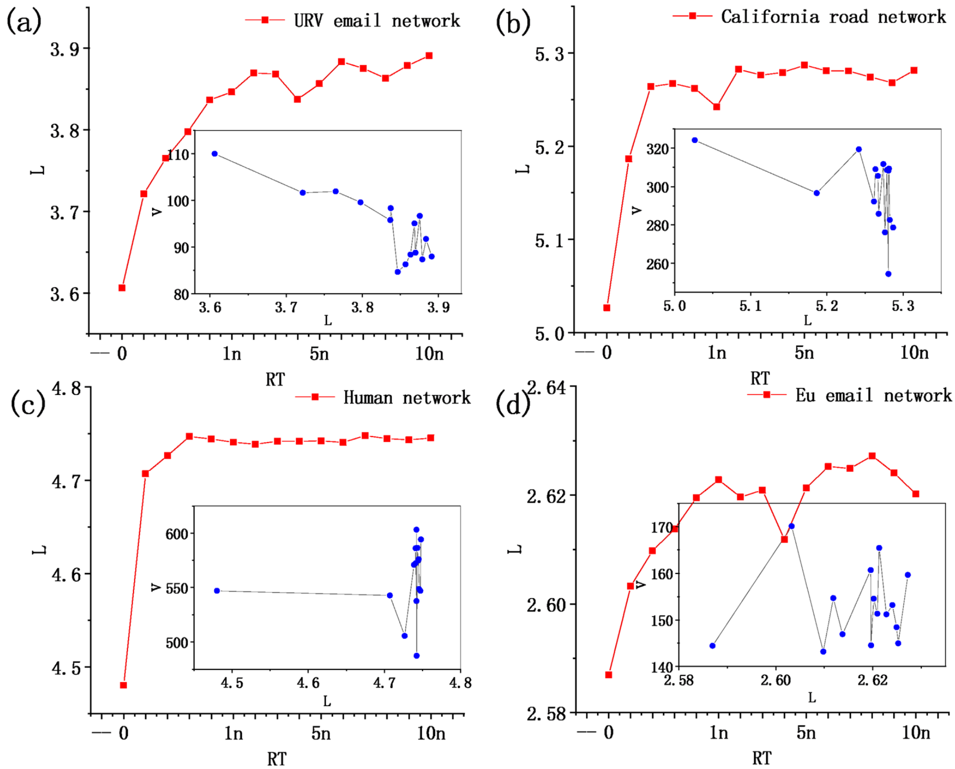

Fig. 10. (Color online) The shortest path length L as a function of rewiring times RT for (a) URV email network, (b) California road network, (c) Human network and (d) EU email network. The rewiring operation is aimed at changing the rich-club. n is twice the number of edges in each network. Inset: spreading speed v as a function of L. Results are averaged over 1000 runs.

with rewiring times. This indicates that the intensity of rich-club property does not correlate strongly with the spreading speed.

In order to discuss the relationship between average shortest path length L and spreading speed v, we plot L versus RT and v versus L in Fig. 10. As shown in Fig. 10, although L changes with increasing RT, there is no linear relationship between v and L. Combined with the above research, there is no absolute correlation between average shortest path length and spreading speed.

5. Conclusions

In summary, the role of network connectivity in transmission dynamics has been explored quantitatively. We introduced null models to measure the value of connection feature, and then explore the impact of connectivity in epidemic spreading. Null models resulting from varying rewiring times in empirical networks have different connection strength. By comparing epidemic spreading in these null models, we have analyzed the quantitative influence of connectivity on the spreading. Our results show that inhomogeneous structure of networks accelerates the diffusion of epidemics. The spreading speed and global matching coefficient are linearly related. The rewiring operation which enhances assortativity has a negative role in epidemic spreading. However, the rich-club property shows a nonlinear relationship with spreading speed of epidemics. This means that the change of global connectivity between nodes directly impacts the spreading of epidemics, but the local connectivity does not possess this influence. Further investigation shows that when the global connectivity of networks is changed, there exists a linear relationship between spreading speed and average shortest path length of networks, and the speed increases with decreasing average shortest path length. However, there is no such phenomenon in the case of changing local connectivity of networks. This means that the average shortest path length of networks is not the major factor influencing epidemic spread.

Acknowledgments

This research is partially supported by the National Natural Science Foundation of China (61773091), the LiaoNing Revitalization Talents Program (XLYC1807106), and the Program for the Outstanding Innovative Talents of Higher Learning Institutions of Liaoning (LR2016070).

References

1. A.-L. Barabási *et al.*, *Network Science* (Cambridge University Press, 2010).
2. Z. Liu, X. Wang and M. Wang, *Chaos* **20**, 023128 (2010).
3. Z. Liu, X. Wang and M. Wang, *Chin. Phys. B* **21**, 078901 (2012).
4. P. Klimek and S. Thurner, *New J. Phys.* **15**, 063008 (2013).
5. J. Tang *et al.*, *Phys. A* **450**, 635 (2016).
6. H.-X. Yang and B.-H. Wang, *Int. J. Mod. Phys. C* **27**, 1650125 (2016).
7. R. Pastor-Satorras and A. Vespignani, *Phys. Rev. Lett.* **86**, 3200 (2001).
8. J. D. J. Esquivel-Gómez and J. G. Barajas-Ramirez, *Chaos* **28**, 013119 (2018).
9. J.-X. Yang, *Int. J. Mod. Phys. B* **33**, 1950266 (2019).
10. I. Sendina-Nadal *et al.*, *Phys. Rev. E* **91**, 032811 (2015).
11. G. F. de Arruda *et al.*, *Phys. D* **323**, 5 (2016).
12. R. Burkholz and F. Schweitzer, *Phys. Rev. E* **98**, 022306 (2018).
13. R. Pastor Satorras, A. Vazquez and A. Vespignani, *Phys. Rev. Lett.* **87**, 258701 (2001).
14. S. Maslov and K. Sneppen, *Science* **296**, 910 (2002).
15. M. E. Newman, *Phys. Rev. Lett.* **89**, 208701 (2002).
16. G. D'Agostino *et al.*, *Europhys. Lett.* **97**, 68006 (2012).
17. Y. Hayashi, arXiv:cond-mat/0408264.
18. A. V. Goltsev, S. N. Dorogovtsev and J. F. Mendes, *Phys. Rev. E* **78**, 051105 (2008).
19. J. C. Miller, *Phys. Rev. E* **80**, 020901 (2009).
20. V. Colizza *et al.*, *Nat. Phys.* **2**, 110 (2006).
21. A. Alawieh *et al.*, *Sci. Rep.* **5**, 13513 (2015).
22. K. Berahmand, N. Samadi and S. M. Sheikholeslami, *Int. J. Mod. Phys. B* **32**, 1850142 (2018).
23. T. Takaguchi and R. Lambiotte, *J. Theor. Biol.* **380**, 134 (2015).

24. D. Broder-Rodgers, F. J. Perez Reche and S. N. Taraskin, *Phys. Rev. E* **92**, 062814 (2015).
25. L. G. Alvarez-Zuzek *et al.*, *PLoS one* **12** (2017).
26. C. P. Brooks and H. Zhang, *J. Theor. Biol.* **264**, 866 (2010).
27. S. Gao *et al.*, *Brain* **141**, 2711 (2018).
28. Y.-F. Chou, H.-H. Huang and R.-G. Cheng, *IEEE Commun. Lett.* **17**, 1356 (2013).
29. G. De Martino and S. Spina, *Phys. A* **438**, 634 (2015).
30. F. Ji and W. P. Tay, *IEEE Trans. Signal Process.* **65**, 2517 (2017).
31. http://konect.uni-koblenz.de/networks/arenas-email.
32. http://snap.stanford.edu/data/roadNet-CA.html.
33. https://snap.stanford.edu/data/email-Eu-core.html.
34. http://snap.stanford.edu/data/email-EuAll.html.