Uncovering the role of signs’ configuration in epidemic spreading on a signed social network

Fujuan Gao, Long Guo, Zhongjie Luo
School of mathematics and Physics, China University of Geosciences (Wuhan),
Lumo Road, 430074, Wuhan, China
E-mail: lguo314@163.com (Long Guo)

Abstract. In this paper, we introduce a reshuffling approach to analyze how signs’ configuration affects the SIR disease-spreading process on the voting network of Wikipedia (VNW) including both positive and negative edges. Inspired by the ensemble theory in statistical mechanics, many reshuffled VNWs with different signs’ configuration are built through applying our reshuffling approach with a pair of parameters \((r_s, r_+)\) on the real VNW. Through comparing SIR simulations on those reshuffled VNWs, we find that the real signs’ configuration slows down disease spreading. Interestingly, there exists the suppressed effect of the fraction of recovered nodes \(R\) in the real VNW, which is a clear fingerprint of non-random factor that prevents the spreading of disease from infectious individuals to susceptible ones in human behaviors. Our present work provides a valuable perspective and tool to understand the tightly coupling of dynamical process and topology in complex system.

1. Introduction

Recently, complex networks have undergone a remarkable development and have emerged as an invaluable tool to describe and quantify complex systems in physics, biology and sociology. Generally, complex network is always assumed to have only positive interactions (i.e., friend, collaboration or trust) when we analyze the dynamics of and on complex network. However, many real complex networks [1, 2], especially online social networks (such as the Ebay, Slashdot and Wikipedia), also have negative links as well as positive ones, such as distrust, disproval and foe such as like and dislike in Slashdot, trust and distrust in Epinions and support and oppose in the voting network of Wikipedia (VNW). Social networks with both types of interactions are called signed networks, where edges are labeled with positive and negative signs [3].

The two fundamental issues are how signs are organized in real signed social network and how signs’ configuration dose affect the dynamics of and on signed social networks. For the first issue, there are two different theories, one is the social balance theory [4, 5, 6, 7, 8], and the other is the status theory [1, 2]. The social balance theory, which follows the common principles that “the friend of my friend is also my friend”, “the enemy of my friend is also my enemy”, “the friend of my enemy is my enemy” and “the enemy of my enemy is my friend”, was formulated by Heider to understand the signs’ configuration in undirected signed social network from the aspect of social psychology. Facchetti et al. analyzed the global level of balance in online signed social networks, such as Epinions and Slashdot, and found that those undirected signed networks are indeed extremely balanced [9]. While for directed signed social networks, the status theory, developed by Leskovec and his collaborators [1, 2], explained signs’ configuration. According to
the status theory, a positive edge \((u,v)\) means that \(u\) regards \(v\) as having higher status than himself/herself while a negative edge \((u,v)\) means that \(u\) regards \(v\) as having lower status than himself/herself in a directed signed social network.

Commonly, individuals’ interactions and states are the two basic components in society. Different types of interactions show different roles in contagion processes (such as spreading of diseases, rumors or opinions), and the distribution of types of individuals’ interactions in signed social network provides a set of potential spreading channels to guide a given contagion process. While, types of individuals’ interactions will be affected by the corresponding individuals’ states. In detail, the evolution of edge signs can be affected by the evolution of nodes in signed social networks. Recently, some dynamical models have been proposed to analyzed signs’ evolution and contagion processes on signed social networks\[10, 11, 12, 13, 14, 15, 16, 17, 18\]. For example, Facchetti and his collaborators analyzed signs’ organization by exploring the landscape of near-optimal structure balance\[10\]. Fan et al. analyzed the opinion spreading based on the SIR model in homogeneous signed networks\[14\]. Altafini studied the process of opinion forming in structurally balanced signed networks\[19\]. Saeedian et al. introduced an energy function to describe the coupling problem of the sign evolution and the SI disease-spreading process based on the social balance theory and they found that balanced clusters act as obstacles to contagion processes\[18\]. Torok et al. diversified an extend opinion dynamic model to describe the rise, persistence and solution of conflicts on Wikipedia and found a transition from mainly consensus to a perpetual conflict occur when agents are replaced with new ones at a certain rate\[20\]. Y.zha et al. found a universal double-power-law distribution of time intervals between consecutive updates of an article by a set of empirical analysis of the history of millions of updates in Wikipedia and then propose a generic model to unfold collaborative human activities\[21\]. However, the study of signs’ evolution and disease spreading in realistic signed social networks, such as Epinions, Slashdot and Wikipedia, also remains a big challenge, since those online social networks provide a paramount platform to shape agents’ behavior in society.

In this paper, we focus on the issue that how signs’ configuration in the VNW affects a disease-spreading process, which is a common phenomenon of diffusion process in society\[22\]. We consider the Susceptible-Infected-Recovered (SIR) model on a given signed social network with both positive and negative edges. In the standard SIR model, one of the most investigated and classical disease-spreading models, individuals can be in one of three states depending on the stage of the disease: susceptible (S), infectious (I) and recovered (R). During the epidemic spreading, susceptible node can be infected by his/her infectious neighbors with a transmission rate \(\lambda\) in one contact with any infected neighbors, infectious node can recover from the disease and then become immune\[23, 24, 25, 26\] with rate \(\mu\). Considering difference from a simple complex network with only positive edges, signed social network has two opposite types of edges that play different roles in affecting the SIR disease-spreading process. In this context negative edges play a block role in individuals’ interactions and we assume that only positive edges have the ability to transmit disease and negative edges do not transmit disease\[18\]. Hence, signs’ configuration, i.e., a given distribution of positive and negative signs, provides a set of potential spreading channels and affects the SIR spreading process. The main question is how to rebuild different signs’ configurations as samples. Inspired by the ensemble theory in statistical mechanics, we propose a reshuffling approach that operates on edges of the VNW, then obtain different reshuffled VNWs related to parameters \(r_\text{w}\) and \(r_\text{+}\). Through comparing SIR simulations on different reshuffled VNWs, we find that the real signs’ configuration prevents the spreading of infection over the whole network and there exists an obvious suppress effect to describe the positive role of non-randomness in the real signs’ configuration.

The paper is organized as follows. In Section 2, signed social networks and our reshuffling approach are defined, and then we define a SIR disease-spreading process on different reshuffled signed networks. In Section 3, we compare SIR simulations on different VNWs and get our
interesting results. Finally, the conclusion and discussion are given in Section 4.

2. The SIR model on reshuffled signed networks
To begin with, we consider a directed signed social network \( G = (V, L, A) \), where \( V \) is the set of vertices, \( L \) is the set of directed links, \( A = \{A_{uv}\} \) describes the signed adjacency matrix with \( A_{uv} \neq 0 \) if and only if \( (u, v) \in L \) and \( A_{uv} \) quantifies the sign of edge \( (u, v) \). A positive sign \( A_{uv}(= +1) \) represents that \( u \) tags \( v \) as a friend or \( u \) supports \( v \), while a negative sign \( A_{uv}(= -1) \) represents that \( u \) tags \( v \) as enemy or \( u \) opposes \( v \). Here, we focus on analyzing the role of signs’ configuration of the voting network of Wikipedia (VNW) in disease-spreading. The VNW is a network of users that voted for and against each other in admin elections, including \( N_v = 7118 \) users and \( N_l = 103,747 \) edges where the proportion of positive edges is \( r_+ = 78\% [1, 2] \). So the VNW network can be described to signed network. We use the data of the voting network of Wikipedia (VNW) to establish signed network and simulate SIR disease-spreading process, which 7118 users on the VNW network are mapped to 7118 nodes in signed network and relationships between users are abstracted to 103,747 edges including 78% positive edges. In Fig.1, a subgraph of the real VNW is shown as an undirected signed network, where red and green edges represent the edges with negative sign and positive sign, respectively.

In order to analyze signs’ configuration in the real VNW from a viewpoint of dynamics, we choose the SIR model as a disease spreading process on the VNW. Each node, says \( i \), is labeled by \( S_i \) as its state, where \( S_i = 0 \) if the node is susceptible, \( S_i = 1 \) if it is infected and \( S_i = -1 \) if it is recovered and gets immunization. As mentioned above, the two opposite types of edges play different roles during a given scenario, and we assume that positive edges can transmit disease and negative edges cannot. Hence, a SIR disease-spreading process depends not only on nodes’ states but also on edges’ signs, since different signs arrangement provides different disease-spreading channels.

We then propose a reshuffling approach that operates on edges and rebuild the reshuffled VNWs with different signs’ configuration according to two tuning parameters \((r_s, r_+)\). Our
reshuffling approach is introduced based on the ensemble theory in statistical mechanics, since a given signed social network with constant macroscopic quantities \((N_0, N_t)\) can be regarded as an isolated system obeying the ergodic hypothesis. A signed social network can have many microscopic states according to signs’ configuration. To reset one or more signs will alter its sign’s configuration, and the system will experience another microscopic state. Different signs configuration describes different microscopic state under the same topology structure of a given system. It’s sure there exist one specific microscopic state which has the same signs configuration as the real signed social network.

In detail, our reshuffling approach is described that each edge is chosen as an activity one with the reshuffling probability \(r_s\), and then each activity edge resets its sign as positive with probability \(r_+\) and as negative with probability \((1 - r_+)\). We can obtain \(2^{N_t}\) potential reshuffled signed networks with different microscopic states according to the tuning parameters \((r_s, r_+)\). Each reshuffled signed network provides its own set of disease-spreading channels. Obviously, the fraction of positive signs of a reshuffling VNW with parameters \((r_s, r_+)\) can be written as

\[
R_+ = r_s r_+ + (1 - r_s) r_+^0
\]

Specially, a reshuffled signed network reduces to the real one when \(r_s = 0\), while all edges’ signs are reshuffled randomly when \(r_s = 1\). Note that our reshuffling approach provides the possibility and feasibility to compare roles of different signs’ configurations in contagion processes, especially the SIR disease-spreading process in this paper.

For a given reshuffled VNW with parameters \((r_s, r_+)\), we start the SIR disease-spreading process from an initial state, where a ratio of notes which were randomly infected is \(\rho_0\) and others are susceptible. At each time step, each susceptible node is infected with rate \(\lambda\) if it is connected to one or more his/her infected neighbors along the positive edges. At the same time, infected nodes are cured and acquire immunity with rate \(\mu\). Without lack of generality, we can set \(\mu = 1\). After enough time elapses, there does not exist any one infected nodes in a given system and we are interested in the fraction \(\rho_R\) of recovered nodes when the SIR disease-spreading process is finished. In order to reduce fluctuation, the SIR disease-spreading process is repeated \(N_c = 50\) times independently with the same initial condition. And we record the evolution of \(\langle \rho_R \rangle = \frac{1}{N_c} \sum \rho_R\) as a function of the infected rate \(\lambda\). Besides, we will get many reshuffled VNWs by means of our reshuffling approach with tuning parameters \((r_s, r_+)\), and repeat the SIR disease-spreading process on those reshuffled VNWs. We shed light on the evolution of \(\langle \rho_R \rangle\) as functions of \(r_s\) and \(r_+\) respectively.

3. Results
The primary interest is how signs configuration does affect the epidemic spreading on the VNW with its topology structure fixed. As show in Fig.2, the fraction of recovered nodes \(\langle \rho_R \rangle\), an order parameter, evolves as a function of infection rate \(\lambda\) for different resetting probabilities \(r_+\) and \(r_s = 0.5\). Clearly, \(\langle \rho_R \rangle\) increases as \(\lambda\) increases, which means that the larger the infection rate is, the wider the epidemic spreading is. Also, as expected, for a case of given \(\lambda\), we find that \(\rho_R\) also evolves as an increasing function of \(r_+\), which indicates that more potential spreading channel promotes the epidemic spreading. In order to quantify the difference between signs’ configurations, we assume that similar signs’ configurations, i.e, the similar potential spreading channels, have the same role in affecting the contagion process of the SIR model. Hence, a quantity \(\Delta_g(\lambda, r_s, r_+)\) (see Fig.2) is introduced to describe the difference of signs’ configuration in a reshuffled VNW and that in the real VNW under the tuning parameters \(\lambda, r_s\) and \(r_+\). By this definition, \(\Delta_g(\lambda, r_s, r_+)\) is given by

\[
\Delta_g(\lambda, r_s, r_+) = |\langle \rho_R \rangle(\lambda, r_s, r_+) - \langle \rho_R \rangle(\lambda, r_s = 0, r_+)|
\]
Figure 2. (color online) The ratio of recovered nodes $\langle p_R \rangle$ evolves as a function of the infected ratio $\lambda$ under different $r_s$ when $r_s = 0.5$ is fixed. $\Delta_g$ is a recovered gap to quantify the difference extent of the role of the two corresponding signs' configuration in the SIR spreading dynamics for a given $\lambda$.

Figure 3. (color online) The relationship between $\Delta_g$ and $r_+$ under different $r_s$ when the infected ratio $\lambda = 0.1$ (left) and $\lambda = 0.5$ (right). The inset is to zoom in the subpart in the black rectangle in the right figure.

where, $\langle p_R \rangle(\lambda, r_s = 0, r_+)$ quantifies the fraction of recovered nodes in the real VNW, and the resetting probability $r_+$ is an invalid variable since no edge is chosen as an activity one to reset its sign when $r_s = 0$. Note that the smaller the value of $\Delta_g(\lambda, r_s, r_+)$, the more similar between the signs’ configuration in the reshuffled VNW and that in the real VNW.

In Fig.3, we plot that $\Delta_g$ evolves as a function of $r_+$ under different $r_s$ in the case of low infection ratio $\lambda = 0.1$ (left) and high infection ratio $\lambda = 0.5$ (right) respectively. Obviously,
all curves have the same evolution tendency to decrease to the valley first and then increase as $r_+$ increases. The value of $\Delta_g$ at valley reaches zero, which means that signs’ configuration in a reshuffled VNW with a given pair of parameters $(r_s, r_+$) has the same role as that in the real VNW in epidemic spreading. While $\Delta_g$ increases as $r_s$ increases when $r_+$ is fixed, which indicates that the performance of our reshuffling approach provides more and more epidemic spreading channels with $r_s$ increasing. Furthermore, all curves reach the same minimal value when $r_+ = 0.8$ in the case of $\lambda = 0.1$ in the left picture of Fig.3, and $r_+ = 0.8$ is approximately equal to the real fraction of positive sign 0.78 in the VNW. Surprisingly, when we increase $\lambda$, taking $\lambda = 0.5$ in the right picture of Fig.3 for example, the positions where $\Delta_g$ reaches its minimal value are separation. $r_+|\Delta_g>0$ increases as $r_s$ decreases. For $\lambda = 0.5$, $\Delta_g$ reaches its minimal value at $r_+ \simeq 0.9$ when $r_s = 0.1$. As $r_+$ increases, the fraction of positive edges increase, that is, potential disease spreading channels increase. At this point, the rate of recovered node in reshuffled network is the same as that of the real network. In other words, there is non-randomness that prevents the spreading of disease in the real VNW network.

Next, we focus on the issue why our reshuffling approach can open additional spreading channels. Unlike atoms based on the least action principle, individual usually maximizes his/her benefits through complex psychological activity. We assume that there exist random factor and non-random factor in individual’s behavior from the macroscopic viewpoint, just like the coexistence of rational thinking and perceptual thinking in psychological activity. Then we define $W_{ran}$ and $W_{nonran}$ to quantify random factor and non-random factor and obtain

$$W_{ran} + W_{nonran} = 1 \quad (3)$$

Random factor is added in the reshuffled signs’ configuration and the weight of random factor $W_{ran}$ increases as $r_s$ increases due to the definition of our reshuffling approach. All signs are reshuffled thorough randomly and $W_{ran} = 1$ when $r_s = 1$. Specially, if there is no non-random factor in real signs’ configuration, i.e., $W_{ran} = 1$, at this time, all curves in Fig.4 should be
overlapped with each other. In order to reveal the role of non-random factor in epidemic spreading, we should suppose that the fraction of positive sign in any potential reshuffled VNW must be fixed and equal to that in the real VNW when we introduce our reshuffling approach to the real VNW. In Fig.4, we plot the evolution of $\langle \rho_R \rangle$ as a function of the infection ratio $\lambda$ under different $r_s$ when $r_p = 0.78$. It’s shown that $\langle \rho_R \rangle$ in a reshuffled VNW with $r_s \neq 0$ is larger than that in the real VNW ($r_s = 0$) when $\lambda > 0.2$, which indicates that real signs’ configuration slows down the epidemic spreading. As we already mentioned that our reshuffling process is the removing process of non-random factor, the suppressed effect of $\langle \rho_R \rangle$ is an obvious fingerprint of the non-random factor in real signs’ configuration according to the epidemic spreading. Furthermore, it is interesting that all curves are overlapping with each other when $r_s \neq 0$, which shows that role of non-random factor in real signs’ configuration in the epidemic spreading is thoroughly released when we analyze a part of the real VNW.

4. Conclusions

In summary, we have introduced a reshuffling approach to compare SIR simulations on different reshuffled VNWs including both positive and negative edges. For simplicity, we assume that only positive edges can transmit disease when considering a SIR disease-spreading process. Signs’ configuration, i.e., a distribution of positive and negative edges, provides its own set of potential disease-spreading channels to guide disease transmission. Hence, to rearrange signs’ configuration is our primary task when we analyze roles of different signs’ configuration in a disease-spreading process. Inspired by the ensemble theory in statistical mechanics, we propose a reshuffling approach to rebuild VNWs through tuning parameters $r_s$ and $r_p$. Through analyzing the order parameter $\langle \rho_R \rangle$ in different reshuffled VNWs, we find that real signs’ configuration slows down the SIR disease-spreading, since individuals have complex psychological activity, which is labeled as a non-random factor in society. Our present work provides an interesting perspective and tools to understand the coupling interaction between contagion processes and topological structure in signed social networks.

References

[1] J. Leskovec, D. Huttenlocher and J. Kleinberg 2010 Sigchi Conference on Human Factors in Computing Systems ACM. 1361-1370
[2] J. Leskovec, D. Huttenlocher and J. Kleinberg 2010 International Conference on World Wide Web ACM 641-650
[3] Y. Chen, X. L. Wang and B. Yuan and B. Z. Tang, 2014 J. Stat. Mech.: Theo. and Exp 03 03021.
[4] F. Heider 1946 The Journal of Psychology. 21 107-112
[5] T. Antal, P. L. Krapivsky and S. Redner 2005 Phys. Rev. E 72 036121
[6] S. A. Marvel, S. H. Strogatz and J. M. Kleinberg 2009 Phys. Rev. L 103 198701
[7] S. A. Marvel, J. Kleinberg, R. D. Kleinberg and S. H. Strogatz 2011 Proc. Natl. Acad. Sci. USA 108 1771
[8] A. V. Rijt 2011 Journal of Mathematical Sociology 35 94-113
[9] G. Facchetti, I. Iacono and C. Altafini 2011 Proc. Natl. Acad. Sci. USA 108 20953-20958.
[10] G. Facchetti, I. Iacono and C. Altafini 2012 Phys. Rev. E 86 036116
[11] T. H. Summers and I. Shames 2013 Europhys. Lett. 103 18001
[12] R. Nishi and N. Masuda, 2014 Europhys. Lett. 107 48003.
[13] S. Tan and J. Liu, 2016 Sci. Rep. 6, 22022.
[14] P. Fan, W. Wang, P. Li and Z. Jiang, 2012 J. Stat. Mech. Theory Exp. 8,08003.
[15] S. Righi and K. Takács, 2014 Adv. Complex Syst. 17,1450011.
[16] M. Ehsani and M. M. Sepehri 2014 Journal of Industrial and Systems Engineering 7 104-117
[17] J. Q. Jiang 2015 Phys. Rev. E 91 062805
[18] M. Saeedian, N. Azimi-Tafreshi, G. R. Jafari and J. Kertesz 2017 Phys. Rev. E 95 022314
[19] Altafini Claudio 2012 PLoS ONE 7 38135
[20] Tórók J, Iniguez G, Yasseri T, San, M. M., Kaski, K., and Kertész, J 2012 Phys. Rev. L 110 088701
[21] Zha, Y., Zhou, T., and Zhou, C. 2016 Proceedings of the National Academy of Sciences of the United States of America 113 14627.
[22] R. Pastor-Satorras, C. Castellano , P. V. Mieghem and A. Vespignani 2015 Rev. Mod. Phys. 87 925-979
[23] W. O. Kermack and A. G. McKendrick 1927 *Proceeding of the Royal Society of London A* **115** 700-721
[24] J. Zhou and Z. H. Liu 2008 *Front. Phys. China* **3** 331-348
[25] A. L Lloyd and R. M. May 2001 *Science* **292** 1316-7
[26] T. Hasegawa and K. Nemoto 2016 *Phys. Rev. E* **93** 032324