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Bursts of Node Activation and Asynchronous Communication in Temporal Networks

Yixin Zhu*, Dongfen Li, Wenqiang Guo, Fengli Zhang
School of Computer Science and Engineering, University of Electronic Science and Technology of China, No. 2006 Xiyuan Avenue, West Hi-Tech Zone, Chengdu, Sichuan 611731, China
*Corresponding author, e-mail: xjzhuyixin@163.com

Abstract

Development of sensor technologies and the prevalence of electronic communication services provide us with a huge amount of data on human communication behavior, including face-to-face conversations, e-mail exchanges, phone calls, message exchanges and other types of interactions in various online forums. These indirect or direct interaction form potential bridges of the virus spread. For a long time, the study of virus spread is based on the aggregate static network. However, the interaction patterns containing diverse temporal properties may affect dynamic processes as much as the network topology does. Some empirical studies show, the activation time and duration of vertices and links are highly heterogeneous, which means intense activity may be followed by longer intervals inactivity. We take heterogeneous distribution of the node inter-activation time as the research background to build an asynchronous communication model. The two sides of the communication don't have to be active at the same time. One derives the threshold of virus spreading on the communication mode and analyzes the reason the heterogeneous distribution of the vertex inter-activation time suppress the spread of virus. At last, the analysis and results from the model are verified on the BA network.

***Keywords**: complex networks, epidemic threshold, inter-activation time, effective transmission rate*

1. Introduction

The network topology which is formed by the interaction between individuals plays a fundamental role in the process of determining the epidemic spread [1]. The original study of epidemiology [2] is based on homogeneous mixing hypothesis, assuming that all people have the same opportunity to contact with other individuals in the populations. The assumption and the corresponding results were challenged by the empirical study, the interactions in the populations can use a meaningful network structure to better describe [3]. A large number of empirical studies show that the node degree distribution in many of reality network obey power-law distribution with heavy-tailed, which is conducive to the spread of virus.

Communication between individuals is the basis of the human society. Nowadays technology, such as sensor devices and online communication services provide us with a large number of records of interaction between individuals, including face-to-face meetings, E-mail, and telephone communication etc.. A traditional way to describe these data is to represent them as an aggregate static network, in which an edge is established if interaction between the two ends of it taked place at least once [3].

Another richer representation of this type of data is the temporal network model [4-13], in which the connection between two nodes only exist at the time of an event. A large number of these data usually consists of a sequence of interactive events. Every event is a triplet, i.e., the IDs of two individuals involved in the event and the time of the event. Some studies of the temporal network focused on the impact of interevent time bursty on the spread of information or virus.

However, many human interactions are not always face to face or synchronous communication mode, such as E-mail exchange, short message, Twitter, WeChat etc.. Not all sent information can be accepted by the recipient, such as the recipient refuses to open a suspicious mail or refuse to click the link received etc.. This kind of asynchronous communication mode can be represented by a sequence of two-tuples, which consist of the ID of an individual and the individual activation time when the individual send or accept some

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object, such as an E-mail, short message, to or from another. When a node i sends a message to another node j at the time t1, node j in its active time t2 to decide whether to accept this message, where t1<t2 . It is worthy of attention what the heterogeneous distribution of individual inter-activation time come into being the impact on the asynchronous information transmission and virus propagation and how the heterogeneous behaviour pattern of individuals impact on asynchronous transmission.

2. Model

In some temporal network literature, any two active nodes are likely to build a temporal edge. But the reality is that the nodes contacted with a node, which are called neighbor nodes of the node, are at a certain scope. The various factors decide the range of an individul contact, such as geographical areas of individual activity, the social circle of individual life and learning, kinship and hobby and so on. Between one node and all of its possible interaction nodes are established links, which constitutes a static aggregation network descripting node activity range and is denoted by G. Email exchange system, for example, nodes is formed by email account address in system and edges are established between each email user and users of his or her email address list, which constitute a static network. So in the network, the vast majority of activities are carried out between the adjacent nodes. Does not rule out, a very small amount of interactions don't take place between the adjacent nodes, it will lead to some small changes in originally static network structure. When a node is activated, it can interact with its neighbours rather than any other node in the network. The static network topology and node activation sequence properties affect the spread behavior on networks together.

The mathematical epidemiological model that is probably the most widely used for theorizing about and emulating epidemics is the so-called the SIR (Susceptible-Infected-Recovered) model. In the SIR model, with which we are concerned in the present report, each individual belongs to either a S (susceptible), I (infected), or R (recovered) state at any given time. When a susceptible individual contact with an infected individual, the former may be infected at an infection rate.

In our model, an action of an individual, such as sending a short message or receiving a email, is called as an activation event of the node. There is a difference in meaning between the inter-activation time of a node and the inter-event time of an edge. The former is based on the behavior of an individual and the latter is based on the interaction between two individuals.

In the model of the bursts of node inter-activation time from a recent literature [11], at each time point, an activated node choose randomly another activated node to build an edge between them. If one of the two nodes is I state node and the other is S state node, the I state node will infect the S state node with some probability. Clearly, the model and the previous models have one thing in common, that is, the synchronous interaction, such as phone call, video meeting, real-time files, etc. However, many cases are closer to the asynchronous communication, such as E-mail exchange, SMS, Twitter, BBS and other network communication way, which the two sides of the communication can be active at different times. At each time t, each active node in the model can accept from neighboring nodes some information or send some information to a neighbor. In reality, user may send or receive a group of information to or from more users at the same time. For simplicity, as long as the time scale is small enough, it can be considered that information is only sent to one of its adjacent node from an activated node at a time.

In our model, all nodes are S state at initial moment except from a node i which is I state. When the initial infected node i is activated, it choose randomly one of its neighbor nodes j and send node j a message containing infection content no matter whether node j is currently activated . Then the node i becomes inactive state at next time. At each time t, every activated node i will accept one or more messages containing virus in accordance with a certain probability for each message of them and then change from S state to I state at the next moment if it has received messages containing virus sent from its neighbor nodes and the node i is S state before time t; If the activated node i is I state, it will choose a neighbor from some address book, such as E-mail address book, the telephone communication book, MSN friends list, to send a message containing virus. At each time t, an infected node recover to R state with some probability.

To facilitate the narrative of node state transition in the model, we will distinguish the S state nodes not received the messages containing virus from the S states nodes received the messages containing virus. When a S state node received messages containing viruse from other nodes, the node is at the risk of infected. Its state is denoted by D. When a D state node is activated, it has the potential to accept this suspicious message and then its state change from D into I.

At each time t, for each activated node i, it is subject to the following rule:

1) if the node i is I state, it send a message containing viruse to an its neighbor node j randomly choosed. If the node j is S state at present, it become D state at next moment t+1; If the node j is D state, I state or R state, it will maintain the current state.

2) if the node i is D state, that is it received one or more messages containing viruse from neighbors at one point t' ($t' < t$), it turn into I state if it accepte the message with probability β, which the transmission time delay is $t - t'$; it recover to S state if it refuse to accept the message with probability 1-β.

3) if the node i is in the S state or R state, it don't do any action. The sent message that does not contain virus does not affect the propogation process of virus and therefore not be considered in the model.

At each time t, no matter whether a node i is activated, it is subject to the following rule:

4) if it is I state node, it will back into R state with probability μ.

In the second point, we assume if a user first saw the suspicious messages, suspicious information or suspicious links and refused to accept them, then he or she will never accpte them. So, the corresponding node state can be changed into S state from D state at next moment.

In many types of empirical data, a wide range of patterns of human activity are known to exhibit long-tailed dynamics[14-16]. Here, we model the node inter-activation time heavy-tailed distribution with the power law distribution. Node inter-activation time τ obey power-law distribution with lower bound [17]:

$$P(\tau) = \frac{\alpha - 1}{\tau_{\min}} \left(\frac{\alpha}{\tau_{\min}} \right)^{-\alpha} \tag{1}$$

Where $\tau_{\min}$ is a lower bound of node inter-activation time τ and α is the exponent or scaling parameter of the power-law distribution.

3. Epidemic Threshold

Key quantities for epidemic dynamics are the so-called transmissibility T and the secondary reproductive number R [18]. T is the probability that an infected individual would transmit virus to a susceptible neighbor before it recovers, and R is the expected number of new nodes infected by an infected nodes.

An infected node restore into recovered state within a time step with the probability of μ, which obey the binomial distribution of the mean for 1/μ. So the average time that a infected node of network changes into a recovered node is 1/μ. When an infected node is activated, it will randomly select an its neighborhood to send an information containing virus.The neighbour accept the information at some futural time with probability of β and will be infected as a consequence if it is previously S state. The inter-activation time τ for each node of network is subject to identically independent distribution. According to the theory of update [19], the transmissibility T for the dynamics can be obtained as:

$$\begin{aligned} T &= \frac{\beta}{\mu} \int_0^{\frac{1}{\mu}} g(\Delta) d\Delta \\ &= \frac{\beta}{\mu} (1 - \int_{\frac{1}{\mu}}^{\infty} g(\Delta) d\Delta) \end{aligned} \tag{2}$$

Where $g(\Delta)=\frac{1}{<\tau>}\int_{\Delta}^{\infty}P(\tau)d\tau$, g(Δ) is to generate time distribution [20], <τ> is the mean of node inter-activation time, P(τ) is the density distribution function of node inter-activation time τ. Where the node inter-activation time τ obey power-law distribution with exponent α, given by Equation (1), transmissibility T can be written as:

$$T=\begin{cases}\frac{\beta}{\mu}\frac{(\alpha-2)}{(\alpha-1)}\frac{1}{\mu\tau_{\min}}, & \frac{1}{\mu}\le\tau_{\min}\\ \frac{\beta}{\mu}[1-\frac{(\mu\tau_{\min})^{\alpha-2}}{(\alpha-1)}], & \frac{1}{\mu}\le\tau_{\min}\end{cases} \tag{3}$$

The node that we arrive at by following a randomly chosen edge has the number of remaining outgoing edges excluding we along [21], denoted by k'. When a node i infected by its neighbor node j, node i selects randomly one of its neighbor nodes as the spread object and the probability the selected node is not node j is $k'/(k'+1)$. Thus the reproductive number R equal $T<k'>/(<k'>+1)$ in our model where $<k'>$ is the average remaining degree of network nodes. It can be expressed by node average degree < k > and the second order of node degrees $<k^2>$ [18, 21], i.e., $<k'>=(<k^2>-<k>)/<k>$. Hence the reproductive number $R=T^*(<k^2>-<k>)/<k^2>$. A basic condition that virus epidemic in network is that the reproductive number R must be greater than one, combined with Equation (3), we can obtain the epidemic threshold as:

$$\lambda_c=\begin{cases}C\mu\tau_{\min}\frac{(\alpha-1)}{(\alpha-2)}, & \frac{1}{\mu}\le\tau_{\min}\\ C[1-\frac{(\mu\tau_{\min})^{\alpha-2}}{(\alpha-1)}]^{-1}, & \frac{1}{\mu}\le\tau_{\min}\end{cases} \tag{4}$$

Where $\lambda\equiv\beta/\mu$, which is the effective transmission rate of virus, λ_c is epidemic thresthold, $C=<k^2>/(<k^2>-<k>)$. Parameter C is only related to the structure of the static network G, and has nothing to do with the dynamic activation properties of nodes.

4. Results and Analysis

Under the condition of nodes dynamic activation, the characteristics of the epidemic threshold of virus are analyzed firstly. BA network [22] is in a typical heterogeneous structure network. Each new node connects m existing nodes of the network and the final total number of the network nodes is N. For a limited scale of BA network [23], the node degree distribution $P(k)=2m^2k^{-3}/(1-N^{-1})$, the node average degree $<k>= 2m$, the node max degree $k_c=mN^{(1/2)}$. We can get the parameter C of BA network as:

$$C_{BA}=\frac{m\ln N}{m\ln N-2(1-N^{-1})} \tag{5}$$

In Figure 1, the epidemic threshold of virus is calculated by Equation (4) and Equation (5) according to the following conditions: the static network G is the BA network of node average degree for 10, the total number of nodes N = 5000, the node inter-activation time τ obey power-law distribution given by Equation (1), the minimum value of node inter-activation time τ_{min}=1, node average recovery time were shown in the illustration in Figure 1.

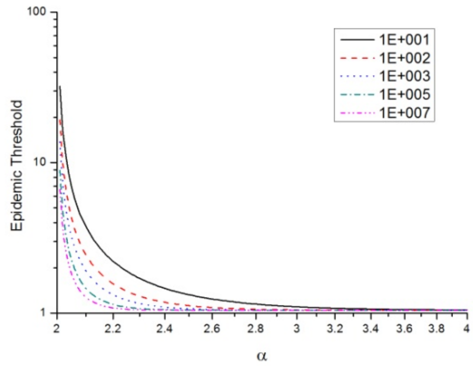

Figure 1. The Epidemic Threshold of Virus λ_c as a Function of Exponent α of Power-law Distribution which Node Inter-activation Time τ obey, for the Different Average Recovery Time

As we can see from Figure 1 three points: First, the epidemic threshold become larger as the increase of the heterogeneous of node inter-activation time distribution (i.e., α decrease) for different average recovery time of infected node. The smaller the power-law exponent of node inter-activation time distribution is, the greater the average value of node inter-activation time derived by Equation (1) is, i.e., the fewer the average times of node activation is in same time. That means an infected node has less chance to spread virus to its adjacent nodes before it recover. Thus only high effective transmission rate of virus ensure its epidemic under the circumstances. Second, the greater the average recovery time of infected node 1/μ is, which means infected nodes have more chance to be activated and transmit virus to their adjacent nodes. Hence the smaller the epidemic threshold is. Thirdly, as the power-law exponent α of node inter-activation time τ increase, propagation threshold is tending to a same value no matter what value node average recovery time 1/μ is. The increase of the power-law exponent α of node inter-activation time τ make the heterogeneity and mean of τ diminished so that there are a large number of nodes of network activated at every moment. Until most of the nodes remain active, dynamic activation network gradually close to the static network G. In this case, epidemic threshold on temporal network is only related to the topology of cumulative static network G, which can be proved from Equation (4).

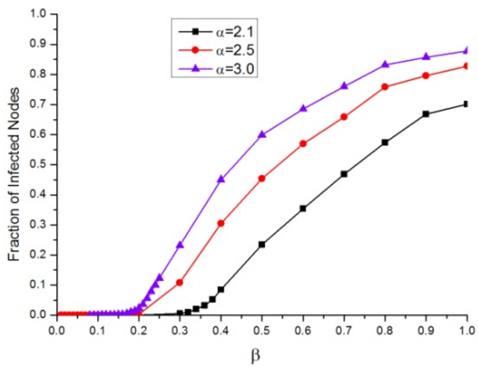

Figure 2. The node density infected by virus as a function of virus transmission rate β, for the different exponent α of power-law distribution which node inter-activation time τ obey. Network node number N = 5000, new edge number from each node m = 5, the recovery rate of the virus spread μ= 0.1, τ_{min} =1

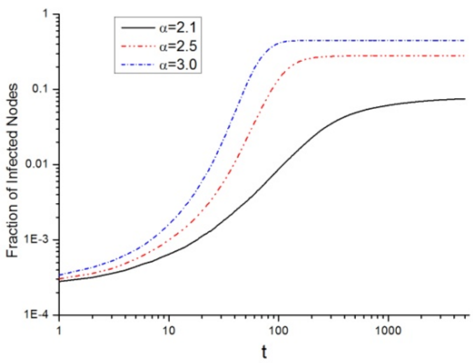

Figure 3. The node density infected by virus as a function of time t, for the different exponent α of power-law distribution which node inter-activation time τ obey. Network node number N = 5000, new edge number from each node m = 5, virus transmission rate β=0.4, the recovery rate of the virus spread μ= 0.1, τ_{min}=1

Model simulation based on static BA network, network scale is 5000 nodes. Each new node connects existing 5 nodes of network. A randomly selected node is set initially to infected state, namely seed node. The average recovery probability μ of infected nodes is 0.1. The node inter-activation time τ obey the power law distribution forms of Equation (1) and the minimum value of node inter-activation time τ_{min}=1. The exponent α is 2.1, 2.5 and 3.0, respectively. Figure 2 show the node density infected by virus change along with virus transmission rate β. It is observed that the stronger the heterogeneity of node inter-activation time τ is, the greater the epidemic threshold of virus is and the less the final spread scope of virus is. The node density infected by virus change along with time in Figure 3. As Figure 3 shown, the stronger the heterogeneity of node inter-activation time τ is, the slower the spread speed of the virus is. That the heterogeneity of node inter-activation time τ inhibits the propagation of virus is illustrated from two different aspects of the scale and the speed of virus propagation respectively in Figure 2 and Figure 3. Which demonstrate that the data simulation results accords with the theoretical analysis results of Figure 1.

5. Conclusion

Different from previous studies that the heterogeneous of inter-event time distribution affect the spread of the virus, this work is based on the heterogeneous distribution of node inter-activation time and establishes the asynchronous communication model, which is more obviously universality than the former. Asynchronous interaction style is suitable for the case that the two sides of interaction are not always active at the same time, which is prevailing in the applications from internet and mobile internet. Where node inter-activation time follows power-law distribution, epidemic threshold of the model is deduced by means of the theory of updates. Simulating in BA network, it is concluded that the stronger the heterogeneity of node inter-activation time is, the greater the epidemic threshold of virus is and the smaller the scale and speed of virus propagation is, which consistents with the results of threshold theoretical derivation.

In this work, asynchronous communication is elaborated by means of the example of sending and receiving E-mails and messages, and epidemic threshold is derived by using the power-law distribution as the heterogeneous distribution of node inter-activation time. But time statistics of human behavior is far from so simple. Different data sets, such as the data sets from mobile phone text messages, blog, BBS, online services, etc., have different heterogeneous time distribution of individual behavior [13], so the time distribution of individual behavior itself is a complicated and worth studying issue.

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References
[1] Keeling MJ, KT Eames. Networks and epidemic models. *Journal of the Royal Society Interface*. 2005; 2(4): 295-307.
[2] Anderson RM, RM May, B Anderson. Infectious diseases of humans: dynamics and control. *Wiley Online Library*. 1992; 28.
[3] Newman M. Networks: an introduction. 2009: Oxford University Press.
[4] Holme P, J Saramäki. Temporal networks. *Physics reports*. 2012; 519(3): 97-125.
[5] Masuda N, P Holme. Predicting and controlling infectious disease epidemics using temporal networks. *F1000prime reports*.2013; 5.
[6] Holme P, J Saramäki. Temporal Networks as a Modeling Framework, in Temporal Networks. Springer. 2013: 1-14.
[7] Takaguchi T, N Masuda, P Holme. Bursty Communication Patterns Facilitate Spreading in a Threshold-Based Epidemic Dynamics. *PLoS ONE*, 2013; 8(7): e68629.
[8] Masuda N, K Klemm, VM Eguíluz. *Temporal networks: slowing down diffusion by long lasting interactions.* arXiv preprint arXiv:1305.2938. 2013.
[9] Lee S, et al., Exploiting temporal network structures of human interaction to effectively immunize populations. *PloS one*. 2012; 7(5): e36439.
[10] Karimi F, P Holme. A Temporal Network Version of Watts's Cascade Model, in Temporal Networks. Springer. 2013; 315-329.
[11] Rocha LE, A Decuyper, VD Blondel. Epidemics on a stochastic model of temporal network, in Dynamics On and Of Complex Networks. Springer. 2013; 2: 301-314.
[12] Yang Z, AX Cui, T Zhou. Impact of heterogeneous human activities on epidemic spreading. *Physica A: Statistical Mechanics and its Applications*. 2011; 390(23): 4543-4548.
[13] Tao Z, et al. Statistical Mechanics on Temporal and Spatial Activities of Human. *Journal of University of Electronic Science and Technology of China*. 2013; 42(4).
[14] Eckmann JP, E Moses, D Sergi. *Entropy of dialogues creates coherent structures in e-mail traffic.* Proceedings of the National Academy of Sciences of the United States of America. 2004; 101(40): 14333-14337.
[15] Vázquez A, et al. Modeling bursts and heavy tails in human dynamics. *Physical Review E*. 2006; 73(3): 036127.
[16] Barabasi AL. The origin of bursts and heavy tails in human dynamics. *Nature*. 2005; 435(7039): 207-211.
[17] Clauset A, CR Shalizi, ME Newman. Power-law distributions in empirical data. *SIAM review*. 2009; 51(4): 661-703.
[18] Newman ME. Spread of epidemic disease on networks. *Physical review E.* 2002; 66(1): 016128.
[19] Feller W. An Introduction to Probability Theory and Its Applications. 1974; II POD.
[20] Vazquez A. Polynomial growth in branching processes with diverging reproductive number. *Physical review letters*. 2006; 96(3): 038702.
[21] Newman ME, SH Strogatz, DJ Watts. Random graphs with arbitrary degree distributions and their applications. *Physical Review E*. 2001; 64(2): 026118.
[22] Barabási AL, R Albert. Emergence of scaling in random networks. *Science*. 1999; 286(5439): 509-512.
[23] Pastor-Satorras R, A Vespignani. Epidemic dynamics in finite size scale-free networks. *Physical Review E*. 2002; 65(3): 035108.