Efficient community-based control strategies in adaptive networks

Hui Yang¹, Ming Tang¹,³ and Hai-Feng Zhang²,³

¹ Web Sciences Center, University of Electronic Science and Technology of China, Chengdu 610054, People’s Republic of China
² School of Mathematical Science, Anhui University, Hefei 230601, People’s Republic of China
E-mail: tangminghuang521@hotmail.com and haifeng3@mail.ustc.edu.cn

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Abstract. Most studies on adaptive networks concentrate on the properties of steady state, but neglect transient dynamics. In this study, we pay attention to the emergence of community structure in the transient process and the effects of community-based control strategies on epidemic spreading. First, by normalizing the modularity, we investigate the evolution of community structure during the transient process, and find that a strong community structure is induced by the rewiring mechanism in the early stage of epidemic dynamics, which, remarkably, delays the outbreak of disease. We then study the effects of control strategies started at different stages on the prevalence. Both immunization and quarantine strategies indicate that it is not ‘the earlier, the better’ for the implementation of control measures. And the optimal control effect is obtained if control measures can be efficiently implemented in the period of a strong community structure. For the immunization strategy, immunizing the susceptible nodes on susceptible–infected links and immunizing susceptible nodes randomly have similar control effects. However, for the quarantine strategy, quarantining the infected nodes on susceptible–infected links can yield a far better result than quarantining infected nodes randomly. More significantly, the community-based
quarantine strategy performs better than the community-based immunization strategy. This study may shed new light on the forecast and the prevention of epidemics among humans.

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1. Introduction

In various real-world systems, the structures are constantly changing with the states of the network and vice versa [1]. For instance, frequent traffic congestion on a road may lead to the construction of new roads, while the new roads will influence the traffic flow on other roads [2]. These phenomena are characterized by the existence of a feedback loop between the dynamics on networks and the dynamics of networks. Networks with such a feedback loop are called co-evolutionary or adaptive [2, 3]. Until recently, a variety of adaptive networks have been studied, such as biological networks [4–6], ecological systems [7–9], technological networks [10, 11] and social networks [12, 13]. Many interesting phenomena have been found, including the robust self-organization in biological nervous systems [14–16], the promotion of cooperation in evolutionary game [17, 18], the emergence of community structure in opinion spreading [19, 20], etc.

From the epidemiological viewpoint, when an infectious disease appears in a population, human self-protection behaviors can significantly change the predicted course of epidemic dynamics [21, 22]. In the extreme, susceptible people may break their contacts with infected partners. This will significantly alter the structure of the contact network, thus influencing the pathway of epidemic spreading. Gross et al [23] first studied the dynamics of the susceptible–infected–susceptible (SIS) model in adaptive networks and found that different transmission rates and rewiring rates can lead to fascinating phenomena, such as bistability and oscillations. Shaw and Schwartz considered a susceptible–infected–recovery–susceptible model in adaptive networks, and observed a similar phenomenon [24]. Subsequently, Marceau et al [25] developed a more precise analytical method for the adaptive networks in the framework of the model of Gross. In addition, quite a few studies showed that adaptive networks can effectively change the dynamics of epidemics [26, 27] and contact switching is an effective control strategy for epidemic outbreaks [28–30].

Previous studies of epidemic spreading in adaptive networks mainly focus on the steady state while ignoring the transient dynamics. However, an understanding of the transient dynamics can contribute to the design of efficient and timely control strategies. In this paper, we look into the adaptive SIS model of Gross et al [23] again, and find that a very strong community
structure is induced by the rewiring mechanism in the early stage of epidemic spreading. Then two community-based control strategies are proposed, and a counter-intuitive conclusion is discovered: it is not ‘the earlier, the better’ for the implementation of control measures.

The paper is organized as follows. In section 2, we formalize the problem by analyzing community structure in the transient process, and propose the normalized modularity parameter. In section 3, we investigate the effects of adaptive community structure on the prevention of epidemics by considering two types of control measures, i.e. the immunization strategy and the quarantine strategy. Finally, the conclusions and discussions are presented in section 4.

2. Problem formulation and parameter normalization

The model considers SIS dynamics in a random network with fixed \( N \) nodes and \( K \) undirected links [23], where a node may be in the susceptible (S) or the infected (I) state. For each SI link, the I node infects the S node with the fixed probability \( p \) per time step. And the I node recovers from the disease with probability \( r \), becoming susceptible again. Meanwhile, with probability \( w \) for every SI link, the S node breaks the link to the I node and forms a new link to a randomly selected S node. Multiple connections and self-connections are excluded. The results presented in the following are for \( N = 10^4 \) and \( K = 10^5 \).

For different values of \( w \) and \( p \), the system can be divided into four different phases [23]: endemic state, bistable state, oscillatory state and healthy state (see figure 1). Note that, in the
bistable state and the oscillatory state, different initial densities of infected individuals \( i(0) \) can yield completely different results [31]. In figure 1(b), for \( i(0) = 0.1 \), a large rewiring rate \( w \) can effectively isolate I nodes and prevent S nodes from infection, leading to the dying out of epidemic. For \( i(0) = 0.3 \), the rewiring behavior can lower the outbreak velocity in the early stage, but cannot prevent the outbreak of the epidemic. For \( i(0) = 0.95 \), the impact of rewiring behavior is negligible due to the huge number of I nodes. When \( w \) increases to 0.6, there is an oscillatory state for large \( i(0) = 0.95 \) in figure 1(c). Although the large rewiring rate \( w \) can prevent the prevalence to some extent, the gradually increasing S nodes will form a giant and tight cluster with the smaller threshold, which causes re-outbreak in S cluster. This process repeats itself, leading to stable oscillations.

From the above analysis, we know that the rewiring behavior can lead transiently to the formation of two separated but internally compact clusters; that is, a strong community structure emerges during the evolution of the adaptive network. Naturally, we can define the community structure of the adaptive network according to the different states of nodes [32–35]. A strong community structure implies that both S and I nodes are more likely to connect to the nodes with the same state and the number of SI links is relatively small, while a weak community structure indicates that S and I nodes are mixed more fully in the network. In view of the significant role of modularity in the dynamics on networks, it has become increasingly important to study the community structures at the mesoscale level [36–41].

Quantifying the strength of community structures in real-world networks remains a challenge [42–45]. The network modularity \( Q \), a popular indicator for measuring community structure [44, 45], is defined as

\[
Q = \sum_{s=1}^{C} \left[ \frac{l_s}{L} - \left( \frac{d_s}{2L} \right)^2 \right],
\]

where \( l_s \) and \( d_s \) represent the number of intra-links and the sum of degrees of the nodes in community \( s \), respectively, \( L \) denotes the number of links in the network, \( C \) is the number of communities and \( \sum_{s=1}^{C} d_s = 2L \). Here \( 0 \leq Q \leq 1 \), the larger the \( Q \) is, the stronger the community structure is.

Figure 2 shows the time evolution of \( Q(t) \) in different phases. One can find that, sometimes, the indicator \( Q(t) \) cannot characterize the community strength of the network well. For example, when the system is in the healthy state, most of SI links can be rapidly broken by the large rewiring rate \( w \), which may bring about the complete separation of S and I clusters. Hence the community structure in the healthy state should be more obvious than in the other states. However, from figure 2(d), one can find that \( Q(t) \) is very low, especially for large times. We note that this is due to the well-known limitations of \( Q \) in assessing the modularity of structures on different scales [40, 46–48]. Specifically, for \( C = 2 \), a network consists of two communities, which we denote as community 1 and community 2. And equation (1) is expanded as

\[
Q = \sum_{s=1}^{2} \left[ \frac{l_s}{L} - \left( \frac{d_s}{2L} \right)^2 \right] = 1 - \frac{l_{12}}{L} - \left( \frac{d_1}{2L} \right)^2 - \left( \frac{d_2}{2L} \right)^2, \tag{2}
\]

where \( l_{12} \) represents the amount of inter-links between communities 1 and 2. When the network is connected randomly, \( l_1 + l_2 \simeq L \left[ \left( \frac{d_1}{2L} \right)^2 + \left( \frac{d_2}{2L} \right)^2 \right] \) and \( l_{12} = L - l_1 - l_2 \simeq (d_1 d_2)/2L \), thus \( Q = 0 \); when \( l_{12} = 0 \) and \( d_1 = d_2 \), \( Q \) reaches the maximum, i.e. \( Q = 0.5 \). Therefore \( Q \) can only range from 0 to 0.5. It is generally known that the range of values allowed for the order parameter

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Figure 2. The time evolution of the modularity $Q(t)$ (circles) and $Q_n(t)$ (triangles) under different conditions. (a) Endemic state, $i(0) = 0.3$, $w = 0.1$; (b) bistable state, $i(0) = 0.3$, $w = 0.3$; (c) oscillatory state, $i(0) = 0.95$, $w = 0.6$; (d) healthy state, $i(0) = 0.3$, $w = 0.7$. When $i(0)$ is small, the epidemic dynamics in the oscillatory state is similar to that in the healthy state; oscillation occurs only when $i(0)$ is large. So we choose $i(0) = 0.95$ in the oscillatory state. Here $p = 0.008$, $r = 0.002$.

should be 0–1. Moreover, the large difference between the sum of degrees of the nodes in communities 1 and 2, e.g. $d_1 \gg d_2$, also yields a small $Q$ even when $l_{12}$ is small. For instance, community 1 is a complete graph with 50 nodes and community 2 is a complete graph with 10 nodes. There is only one link between the two communities. According to equation (2), the modularity of such a community network is $Q = 1 - 1/1271 - (2451/2542)^2 - (91/2542)^2 \approx 0.068$. Such a small $Q$ means a very weak community structure, which clearly does not capture the perceived modularity of such a network.

Above all, $Q$ is not an accurate index to characterize the community strength of the network with two communities. To address this limit, like in [49], the normalized $Q_n$ is defined as

$$Q_n = \frac{Q - Q_{\text{rand}}}{Q_{\text{max}} - Q_{\text{rand}}},$$

where $Q_{\text{rand}}$ corresponds to the random network with the same degree sequence, and $Q_{\text{max}}$ is the modularity of the network without inter-community links, i.e. $l_{12} = 0$. After this normalization, $Q_n$ is in the range $[0, 1]$.

The normalized $Q_n$, in comparison to $Q$, can reflect the community structure in the adaptive network more accurately. In the endemic state, as shown in figure 2(a), although the small rewiring rate $w$ cannot prevent the outbreak of epidemic, it can also induce a certain degree of community structure. In the bistable state, figure 2(b) demonstrates that $w = 0.3$ can give rise to the form of large and tight S clusters in the early stage ($t \leq 50$). And a subsequent re-outbreak in...
the $S$ cluster ($50 < t \leq 110$) results in the weakening of the community strength. When $t > 110$, the community strength of the network will keep a stable value. In the oscillatory state, $w = 0.6$ can isolate I nodes quickly; hence the community strength becomes large rapidly. But as the $S$ cluster becomes larger and tighter, the threshold of outbreak becomes smaller. Finally, the epidemic prevails in the $S$ cluster at some point, such as $t \approx 200$ in figure 2(c), resulting in the weakening of the community structure. Then this process will repeat again and again. In the healthy state, the very large $w$ will separate the $S$ and the I cluster completely and rapidly; thus the community structure is increasingly strong in the early stage and keeps $Q_n \approx 1$ after the $S$ and the I clusters are separated (see figure 2(d)).

3. Effects of the community structure on the prevention of epidemics

From the above, S and I nodes can be divided into two loosely coupled but internally tight communities due to the rewiring mechanism in the transient process. One significant question is whether this property is helpful for the control of the disease. In this viewpoint, we study the impacts of the emerging community structure on the control of disease, and search for efficient control strategies. Look back at the transient processes in the four phases of the system. In the endemic state, a disease spreads so rapidly that it is difficult to detect the community structure and control the epidemic. In the healthy state, there is no need for controlling the disease. In the oscillatory state, the oscillation occurs only when $i(0)$ is very large, such as $i(0) > 0.9$, but this is unrealistic for real-world diseases. In the bistable state, a small $i(0)$ can cause the outbreak, and there is a very strong community structure for a long time. Therefore, we only focus on the bistable state.

In the transient process, the strength of community structure is always changing. To understand the effects of the emergent community structure on the prevention of epidemics, we compare the control effects of counter measures applied at different times. As we all know, immunization and quarantine are two basic measures to control epidemic spreading. Here we consider the immunization strategy and quarantine strategy, respectively. To evaluate the effect of the control strategy, we let $T_0$ be the starting time of immunization (quarantine), $\Delta i_0(T_0)$ be the difference between the density of infected individuals $i_0(T_0)$ at time $T_0$ and the maximal density of infected individuals $i_{\text{max}}(T_0)$ that can reach after immunization (quarantine), i.e. $\Delta i_0(T_0) = i_{\text{max}}(T_0) - i_0(T_0)$. $\Delta t(T_0)$ denote the time interval of this process, and $r(T_0)$ denote the total percentage of immunized (quarantined) nodes in $\Delta t(T_0)$ (see the inset of figure 3(b)).

3.1. Immunization strategy

Immunization strategy—immunizing a fraction $f$ of S nodes at each time step. Two approaches to choose S nodes are compared: (i) randomly choose S nodes from the network (labeled IMR); (ii) randomly select S nodes from SI links (labeled IMSI).

In figure 3, the results of the IMR and the IMSI strategies are compared. Figures 3(a), (c) and (d) show that these curves follow similar trends. What is more interesting is that three parameters are minimal for $T_0 \in [20, 35]$. So the optimal starting time of immunization is $T_0 \in [20, 35]$, which is somewhat against our intuition—the earlier the immunization starts, the better the control effect is. To explicitly explain such a phenomenon, the time window of $T_0$ is divided into four regions according to the trend of $\Delta i(T_0)$ (see figure 3(a)): $\Delta i(T_0)$ decreases
Figure 3. The effects of different immunization strategies versus the starting time of immunization \( T_0 \). (a) \( \Delta i(T_0) \), (b) \( i_0, i_{\text{max}} \), (c) \( \Delta t(T_0) \) and (d) \( r(T_0) \). IMR represents the immunization strategy that randomly chooses \( S \) nodes from the network, while IMSI represents the immunization strategy that randomly selects \( S \) nodes from SI links. Here \( i(0) = 0.3, w = 0.3, r = 0.002, p = 0.008 \) and \( f = 0.008 \). ‘Stars’ in (a) represent the time evolution of \( Q_n(t) \). The inset of (b) shows the definition of \( \Delta i(T_0) \) and \( \Delta t(T_0) \). Each point represents an average over 200 realizations.

For \( T_0 \in [0, 20] \), remains stable for \( T_0 \in [20, 35] \), increases gradually for \( T_0 \in [35, 75] \) and drops again for \( T_0 \in [75, 100] \).

For \( T_0 \in [0, 20] \), the strength of community structure and the density of infected individuals \( i(t) \) increase rapidly. When the community strength is weak, immunizing a few \( S \) nodes cannot effectively break the bridges between the \( S \) cluster and the I cluster. Thus, the increase of the prevalence is large, e.g. \( \Delta i(T_0 = 0) \approx 0.2 \). With the delaying of the starting time \( T_0 \), the community structure will be enhanced, and thus the IMSI strategy can break the connections between the two communities more thoroughly, which causes the prevalence to soon be brought under control. Consequently, \( \Delta i(T_0) \) decreases with increasing \( T_0 \), which is the opposite of the traditional concept ‘the earlier, the better’. Meanwhile, the epidemic can be inhibited faster than before (i.e. the smaller \( \Delta t(T_0) \) in figure 3(c)) with immunizing fewer \( S \) nodes on SI links (i.e. \( r(T_0) \) in figure 3(d)).

For \( T_0 \in [20, 35] \), the community structure is very strong and the density of infected individuals \( i(t) \) increases slowly. Owing to the very strong community structure, immunizing \( S \) nodes on SI links can hold back the re-outbreak in the \( S \) cluster in a timely fashion, which results in the minimum of \( \Delta i(T_0) \). In figures 3(c) and (d), \( \Delta t(T_0) \) and \( r(T_0) \) also reach their minimum and remain stable.

For \( T_0 \in [35, 70] \), the adaptive network still has a very strong community structure. However, it will certainly take some time to prevent the increase of the prevalence, e.g.
Figure 4. The temporal evolution of $S_r/S$ and $I_r/I$ in the bistable state. $S_r/S$ (circles) represents the percentage of S nodes located on SI links in all S nodes and $I_r/I$ (triangles) represents the percentage of I nodes located on SI links in all I nodes. The results are derived from an average over 200 realizations.

$\Delta t(T_0) \approx 20$ for $T_0 = 20$. Suppose the immunization starts after $T_0 = 35$, there is not enough time left for control, because the tight S cluster is invaded gradually by the disease again, which leads to the weakening of community structure at $t \approx 50$ (see figure 1(b)). So a re-outbreak is inevitable due to the lack of sufficient controlling time. Therefore, the later the starting immunization time $T_0$, the worse the control effect of the IMSI strategy.

For $T_0 \in [70, 100]$, the prevalence increases rapidly, and the strength of community structure decreases dramatically. Although the trends of these curves are similar to that in the region $T_0 \in [0, 20]$, the reason is completely different. For $T_0 \in [70, 100]$ (see figure 3(b)), the control strategies no longer have any significance as $i_{\text{max}}(T_0) \to 1$. $\Delta i(T_0) \approx 1 - i(T_0)$ thus decreases with $T_0$.

Interestingly, in figure 3, the impact of the IMR strategy is almost equal to that of the IMSI strategy. To explain this, the percentage of the S nodes on SI links in all S nodes ($S_r/S$) is shown in figure 4. Although there is the strongest community structure at $t \approx 50$, the ratio $S_r/S \approx 0.5$ is still relatively large. Consequently, both the IMR strategy and the IMSI strategy have similar results.

3.2. Quarantine strategy

Quarantine strategy—quarantining a fraction $f$ of I nodes per time step. Similar to the immunization strategy, two approaches to choose I nodes are also considered: (i) randomly choose I nodes from the network (labeled ISR); (ii) randomly select I nodes from SI links (labeled ISSI).

The effects of the ISR strategy and ISSI strategy are compared in figure 5. The effect of the ISR strategy is much worse than that of the ISSI strategy. As illustrated in figure 4, the ratio $I_r/I$ is small; that is, most of infected nodes are not on the SI links. As a result, the ISR strategy can hardly pitch on the I nodes on SI links, and the advantage of the community structure could not be well developed by the ISR strategy. However, the effect of the ISSI strategy is striking.
Figure 5. The effect of different quarantine strategies versus the starting time of quarantine $T_0$. (a) $\Delta i(T_0)$, (b) $i_0$, $i_{\text{max}}$, (c) $\Delta t(T_0)$ and (d) $r(T_0)$. ISR represents the quarantine strategy that randomly chooses I nodes from the network, while ISSI represents the quarantine strategy that randomly selects I nodes from SI links. Here $i(0) = 0.3$, $w = 0.3$, $r = 0.002$, $p = 0.008$ and $f = 0.008$. Stars in (a) represent the time evolution of $Q_n(t)$. Each point represents an average over 200 realizations.

since the ISSI strategy can efficiently cut the pathways of disease to the S cluster. In particular, $\Delta i(T_0)$ in figure 5 (a), $\Delta t(T_0)$ in figure 5(c) and $r(T_0)$ in figure 5(d) reach the minimum when $T_0 \in [20, 65]$, which corresponds to the time interval with the strongest community structure. (Here, we should note that, compared with the IMSI strategy, the ISSI strategy is more efficient in controlling the epidemic and has a larger optimal region since the ISSI strategy can directly cut more spreading pathways to the S cluster than the IMSI strategy.) The reason for the trends of these curves in figure 5 is similar to the case of the immunization strategy: in the initial stage ($T_0 \in [0, 20]$), the community strength increases with $T_0$. Therefore, a larger $T_0$ brings about a better control effect. Then it reaches the optimal region, i.e. $T_0 \in [20, 65]$, in which the community structure is strongest. The ISSI strategy can contain the epidemic spreading rapidly. So, $\Delta i(T_0)$, $\Delta t(T_0)$ and $r(T_0)$ reach the minimum and remain stable. When $T_0 \in [65, 90]$, the compact S cluster is invaded by the disease again and the community structure becomes weak gradually, so the effect of the ISSI strategy becomes poor with $T_0$. At last, $i_{\text{max}}(T_0) \approx 1$ for $T_0 > 90$, thus $\Delta i(T_0)$ decreases with $T_0$ again.

4. Conclusions and discussions

To sum up, we studied the properties of community structure in the transient process of the adaptive network by normalizing the modularity. We found that different degrees of community strength emerge from distinct rewiring conditions. In particular, in the bistable state, the very
strong community structure can hold for a long period. In view of this, the community-based immunization strategy and quarantine strategy are studied thoroughly. Because most S nodes are on SI links when an epidemic prevails in an adaptive network, immunizing S nodes randomly can give rise to similar effects as immunizing the S nodes on SI links. Nevertheless, for the quarantine strategy, quarantining the I nodes on SI links is significantly better than quarantining I nodes randomly, since the former can efficiently cut the pathways of disease invading the S cluster. Both the studies of immunization and quarantine strategies reveal a counter-intuitive conclusion: it is not ‘the earlier, the better’ for the implementation of control measures. And the optimal control effect is obtained if control measures can be efficiently implemented during the time with a strong community structure. More significantly, the community-based quarantine strategy displays a more efficient performance than the community-based immunization strategy.

The prevalence of an infectious disease can trigger the behavioral responses of people attempting to minimize the risk of being infected. If so, further study on the control strategy in adaptive networks has instructive significance. We have made a forward step in this direction. This work may provide a new perspective on the forecast and prevention of epidemics among humans.

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