Feedback pinning control of collective behaviors aroused by epidemic spread on complex networks

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Abstract

This paper investigates epidemic control behavioral synchronization for a class of complex networks resulting from spread of epidemic diseases via pinning feedback control strategy. Based on the quenched mean field theory, epidemic control synchronization models with inhibition of contact behavior is constructed, combining with the epidemic transmission system and the complex dynamical network carrying extra controllers. By the properties of convex functions and Gerschgorin theorem, the epidemic threshold of the model is obtained, and the global stability of disease-free equilibrium is analyzed. For individual’s infected situation, when epidemic spreads, two types of feedback control strategies depended on the diseases’ information are designed: the one only adds controllers to infected individuals, the other adds controllers both to infected and susceptible ones. And by using Lyapunov stability theory, under designed controllers, some criteria that guarantee epidemic control synchronization system achieving behavior synchronization are also derived. Several numerical simulations are performed to show the effectiveness of our theoretical results. As far as we know, this is the first work to address the controlling behavioral synchronization induced by epidemic spreading under the pinning feedback mechanism. It is hopeful that we may have more deeper insight into the essence between disease’s spreading and collective behavior controlling in complex dynamical networks.

Keywords: Epidemic model; Control behavioral synchronization; Pinning feedback control; Complex network

1. Introduction

Complex networks are ubiquitous around the world, such as the World Wide Web, biological networks, food chain networks, power grids and transportation networks, economic and social networks, see [1]-[10] and references therein. Since complex networks can be used as models to reflect certain intrinsic natures in the real world, a lot of achievements have been made on complex networks [11, 12]. Synchronization and propagation, two typical interesting and important dynamical behaviors in networks, have attracted much attention from various fields in recent years, including communication, engineering, physical science, mathematics, sociology and biology. On the one hand, many kinds of synchronization mechanisms have been discovered over the last decades, such as the complete synchronization [13], the cluster synchronization [13, 14], the lag synchronization [17, 18], and so on, see [19]-[21] and references therein. On the other hand, the spreading of epidemic disease in complex networks have also been received more and more attentions due to the development of network science research since 1998. When an epidemic disease spreads in a realistic network, the information about the disease transfer only along the edges, which means that there will no direct transmission in any pairs of individuals without relationship between them. Taking into consideration of the significance about spreading of epidemic diseases on complex networks, many successful works have been devoted by a lot of researchers from mathematical biology field [22]-[24]. The problem about global stability and attractivity of a network-based SIS epidemic model with nonmonotone incidence rate was investigated in [23], and the conditions guaranteeing global asymptotical stability or global attractivity of an endemic equilibrium are also obtained. And the issue of network-level reproduction number and extinction threshold for vector-borne diseases was...
studied in [26], meanwhile relationships between the basic reproduction numbers and the extinction thresholds of corresponding models are obtained by using some assumptions.

One may think that collective behaviors and spreading behaviors are two independent patterns of behavior in complex networks, however, the fact seem to be not the case. In fact, when an epidemic disease spreads in a network, individuals always change their behaviors to protect themselves from being infected by infected individuals around them. They may take steps consistently, such as washing their hands with soaps frequently, having the cure in hospital, avoiding going to the crowded and doing exercise to strengthen their resistance to infection, and so on, to combat the disease. These consistent behaviors against the epidemic disease are also called as behavior synchronization induced by contagious diseases. There are some successful works in recent years [27]-[30] taking the synchronization and epidemic transmission in complex networks into consideration simultaneously. Motivated by these interesting works, in this paper, we will further study the problem about the behavior synchronization induced by epidemic spread.

In [31], authors considered adaptive mechanism between dynamical synchronization and epidemic spreading behavior on complex networks, in which two models of epidemic synchronization were introduced for the first time to analyze the stability of epidemic synchronization and obtained local and global conditions. Following that, the interplay between collective behavior and spreading dynamics on complex networks was further investigated in [32], where several bidirectional network models of spreading phenomena were constructed, and found that the collective behavior and spreading behavior are influenced each other. But the models in the above two papers are all based on the heterogeneous mean-field theory. In order to be more accurate in predicting epidemic synchronization, authors in [33] introduced the quenched mean-field theory to construct three epidemic synchronization models without delays or with distinct delays, and got some local and global conditions to guarantee the results. Therefore, the quenched mean-field theory will be also considered in our work for more precise prediction of epidemic collective behaviors. However, models in these works are all about adaptive synchronization with adaptive exterior coupling strength. As is known to all, it is relatively difficult to synchronize a network only depending on itself evolution. Hence, we will add external controllers to achieve behavioral synchronization aroused by the epidemic disease transmission in this work.

It is acknowledged that adding external controllers to reach synchronization is an important and effective method in a scale-large complex network [34]-[36]. When an epidemic disease breaks out, it is also important to actively control collective behaviors, as the individuals may not get full information about the diseases or even do not know the event of contagion in the early stage. Therefore, in order to make individuals in a network response quickly in the early stage of an epidemic disease spreading, some external controlling methods are carried out to reach behavioral consistency for the individuals, named as controlling behavioral synchronization induced by epidemic disease transmission. Such control can be performed via broadcasting and other media of information dissemination or some certain well-directed means, by which individuals may consistently behavior, such as going to the hospital, washing their hands with soaps, etc., to protect themselves from being infected, and resist the diffusion of contagions. As far as we know, there are no works concerning epidemic behavior synchronization via a control strategy. Therefore, we will discuss the collective behaviors induced by epidemic disease transmission in the view of external pinning feedback controller introduced in [34] with the normalized outer-coupling strength in the synchronous models. The difference in this case is that the pinning feedback controllers depend upon the information about epidemic spreading, where controllers work only when contagious disease spreads. In addition, noticing a realistic fact that there are always the inhibition of contact behaviors among individuals rooted in the subconscious, especially when an contagion spreads. In other words, individuals may change their willing to contact with others relying on the risk of being infected, and the consistency with the behavioral target resulting from protecting themselves, which can be reflected as the higher of the risk of being infected, the less the individuals are willing to contact with each other. And the less distinct between behavioral targets, persons are desired to touch with others. Thus it is natural to consider the inhibition of contact behavior into our models.

Inspired by the above discussions, this paper further studies the the pinning feedback control of behavior synchronization induced by epidemic diseases spreading. The outer-coupling strength is normalization and the external pinning feedback controllers are equipped in the synchronous models. And in the epidemic spreading models, the quenched mean-field theory is introduced for more accurate reflection of the realistic situations, and more precise evaluation of the control behavior synchronization aroused from transmission of contagious. What's more, in order to simulate more real human reaction, the inhibition of contact behaviors are also considered in our epidemic spreading models. That is to say, on the one hand, the higher the risk of being infected, the less the willing to contact with each other. On the other hand, the more consistent the individuals’ behaviors are, the stronger the desires to keep in touch.
We will use distinct pinning feedback control strategies to achieve corresponding control of behavior synchronization, which means the corresponding controllers will be added to only a portion of nodes or individuals for each strategy in the network.

The main contributions of this work are listed as follows. Firstly, combined with the synchronous systems and Susceptible-Infected-Susceptible (SIS) epidemic systems, based on the quenched mean-field theory, we construct a class of models of epidemic controlled behavioral synchronization with the external controllers, in which the outer-coupling strength is normalized in the synchronous system. Secondly, in order to perform more real simulation, the inhibition of contact behavior is concerned in the epidemic system, which means that the desires to contact with others not only rely on the risk of being infected, but also depend on the difference between behavioral target states. Finally, in the epidemic extinction. The main contributions of this work are listed as follows. Firstly, combined with the synchronous systems and Susceptible-Infected-Susceptible (SIS) epidemic systems, based on the quenched mean-field theory, we construct a class of models of epidemic controlled behavioral synchronization with the external controllers, in which the outer-coupling strength is normalized in the synchronous system. Secondly, in order to perform more real simulation, the inhibition of contact behavior is concerned in the epidemic system, which means that the desires to contact with others not only rely on the risk of being infected, but also depend on the difference between behavioral target states. Finally, pinning feedback control are equipped into the epidemic synchronization for the first time. In other words, only a part of nodes will be controlled by corresponding feedback strategies, and the controllers are related to the information of the epidemic spread, which means the controllers will work only when the epidemic spreads, or failures with the epidemic extinction.

The rest of the paper is organized as follows. In Section 2 the problem is formulated and some helpful definitions, lemmas, assumptions and notations are given. In Section 3 the existence of endemic equilibrium and the global stability of disease-free equilibrium are analyzed at first in the epidemic controlling synchronization models, and then the theoretical results for distinct controlled behaviors synchronization induced by epidemic spreading under different control strategies are derived. Some numerical examples are shown to illustrate the theoretical results in Section 4. Finally, the conclusions of this paper are presented in Section 5.

Notations. Throughout the paper, \( \mathbb{R} \) is the set of real numbers, \( \mathbb{R}^n \) represents the \( n \) dimensional Euclidean space, \( \mathbb{R}_+^n \) is the \( n \) dimensional vector space with positive elements, and \( \mathbb{R}^{n \times m} \) denotes the set of real matrices with order \( n \times m \). The transposition for a vector or a matrix is represented by superscript \( T \). \( \lambda_{\min}(\cdot) \) and \( \lambda_{\max}(\cdot) \) represent the minimum and maximum eigenvalues of a square matrix, respectively. And \( I_N \) is the identity matrix of order \( N \). \( \| \cdot \| \) represents the Euclidean norm for vectors and the induced 2-norm for matrices. And \( \otimes \) is the Kronecker product. For a matrix \( P, P > 0 (< 0) \) means that \( P \) is positive (negative) definite.

2. Models and Preliminaries

The mathematical models about infectious disease dynamics and complex dynamical network systems are given in this part. And some fundamental conceptions and necessary preliminaries are introduced for later use.

2.1. Mathematical models

A. Complex dynamical network model

We now introduce a coupled complex network with \( N \) nodes or individuals under controlling

\[
\dot{x}_i(t) = f(x_i(t)) + \sum_{j=1}^{N} a_{ij} x_j(t) + u_i(t), \quad i = 1, \cdots, N, \tag{1}
\]

where \( x_i(t) = (x_{i1}(t), \cdots, x_{in}(t))^T \in \mathbb{R}^n \) represents the \( i \)-th node's state, \( f(\cdot) : \mathbb{R}^n \to \mathbb{R}^n \) is a differentiable function that represents the behavior of individual system, and \( f(x_i(t)) = (f_1(x_i(t)), f_2(x_i(t)), \cdots, f_n(x_i(t)))^T \in \mathbb{R}^n \). Matrix \( A = (a_{ij}) \in \mathbb{R}^{N \times N} \) represents the out-coupled structure, in which \( a_{ij} = a_{ji} = 1 \) if there is a connection between nodes (or individuals) \( i \) and \( j \), and \( a_{ij} = a_{ji} = 0 \) otherwise. \( u_i(t) \) is the external controller for each node \( i \), which will be defined later.

Define the Laplacian matrix as \( L = (l_{ij}) \in \mathbb{R}^{N \times N} \):

\[
l_{ij} = -a_{ij},
\]

\[
l_{ii} = -\sum_{j=1, j \neq i}^{N} l_{ij}, \quad i = 1, 2, \cdots, N
\]
Assume the Laplacian matrix $L$ is irreducible, which indicates that network with $N$ nodes is strongly connected, e.g., there exists one path from any node $i$ to $j$. From the definition of Laplacian $L$, it is obvious that $L$ has one zero eigenvalue and $N - 1$ positive eigenvalue, which means that there exists a unitary matrix $U$ such that

$$U^T L U = \text{diag}\{\lambda_1, \lambda_2, \cdots, \lambda_N\},$$

we sort these eigenvalues as $0 = \lambda_1 \leq \lambda_2 \leq \cdots \leq \lambda_N$.

**B. SIS epidemic dynamics model**

Taking into account the network’s topological structure, this paper introduces the following quenched networks for infectious disease systems (or quenched mean field model):

$$\begin{aligned}
\dot{S}_i(t) &= \rho_i(t) - \lambda \varphi_i(t) [1 - \rho_i(t)] \sum_{j=1}^{N} a_{ij} \rho_j(t) \\
\dot{I}_i(t) &= -\rho_i(t) + \lambda \varphi_i(t) [1 - \rho_i(t)] \sum_{j=1}^{N} a_{ij} \rho_j(t)
\end{aligned}$$

where $\rho_i(t)$ and $I_i(t)$ represent the infected probability and the uninfected probability of node $i$ at time $t$, respectively. $\lambda$ is the infection rate. $\varphi_i(t)$ is the probability of inhibition to contact behavior for each individual $i$, which has the representation as $\varphi_i(t) = 1 - \gamma_i(t)$, where $\gamma_i(t)$ is defined as follows,

$$\gamma_i(t) = \alpha \rho_i(t) \frac{\sum_{i=1}^{N} e_i^T(t) e_i(t)}{\sum_{i=1}^{N} e_i^T(t) e_i(t)},$$

in which $\alpha$ denotes the gain of desire for contact, and $\alpha \in (0, 1)$. And the $e_i(t)$ is the synchronization errors for the $i$-th node defined later.

**Remark 1.** Note that the inhibition to contact behavior is a natural reaction for all the population, especially in the case of breaking out of an epidemic disease. This behavior not only depends on the information about the risk of being infected by other individuals, but also on the target to reach. On the one hand, the greater the risk of being infected is, the weaker the desire to contact with others is. On the other hand, the smaller the behavior error between synchronous target is, the stronger the willing to contact with others is.

**C. SIS epidemic controlling synchronization model**

Then by combining the SIS epidemic dynamics model and complex dynamical network model, we get the epidemic controlling synchronization model with external controllers:

$$\begin{aligned}
\dot{x}_i(t) &= f(x_i(t)) - \sum_{j=1}^{N} l_{ij} x_j(t) + u_i(t) \\
\dot{\rho}_i(t) &= -\rho_i(t) + \lambda \varphi_i(t) [1 - \rho_i(t)] \sum_{j=1}^{N} a_{ij} \rho_j(t)
\end{aligned}$$

This kind of compound model mixes dynamical equation of individual’s state and equation for individual’s spreading of disease, and in the case of the controlling behavioral synchronization induced by the transmission of epidemic disease, $x_i(t)$ denotes the state variable of the individual (or node) $i$ at time $t$ in the epidemic disease network, which reflects certain daily behavioral patterns (washing the hands, traveling and having a rest, etc.). $\rho_i(t)$ represents the probability of being infected for individual $i$ at time $t$. Assume that individuals may be infected only when there exists connections among them. At this moment, model (1) and model (2) share the same topology structure. In other words, the dynamical behaviors of individuals and the epidemic spreading behaviors appear simultaneously in the same network, which indicates that the process of individuals’ dynamical behaviors will accompany the spreading behavior.
of epidemic disease in the disease network. And the $f$ defines the local dynamics that represents the summation of distinct behaviors for epidemic individuals, and the $f$ is active, dynamic as well as complex. In this paper, we realize the coupling between the model (1) and model (2) by external controllers depending on the epidemic information of individuals’ disease and the synchronous errors, then reaching the point of controlling behavioral synchronization under the premise of epidemic diseases transmission.

In the first equation of model (3), $u_i(t)$ represents the external controller for each node $i$ consisting of two parts, $\rho_i(t)$ and $c_i(t)$, in which $\rho_i(t)$ can be regarded as the information about the epidemic disease for each individual, while the other term is the essential part in the pinning control strategy that measures the synchronous state of each individual (or node). Then the controllers will be activated and play a role according to the situation about the breaking-out of the epidemic disease under this framework for controllers. Which means that the controllers will work to lead the behavioral synchronization when the epidemic disease spreads, e.g., $\rho_i(t)$ will not be zero as $t$ tends to positive infinity. When the epidemic disease dose not spread, the controllers will be invalid due to the disease’s die out, i.e., $\rho_i(t)$ goes to zero. Hence it is no needed to control individuals in the network to get the collective behavior. Thus it is meaningful to design the pinning controllers to relate to $\rho_i(t)$.

2.2. Preliminaries

For analytical requirement, some useful preliminaries are introduced in this part.

**Definition 1.** A complex dynamical network system containing $N$ individuals (or nodes) is said to reach asymptotical synchronization, if for each pair nodes $i,j$ ($i \neq j$) we have

$$\lim_{t \to \infty} \left\| x_i(t) - x_j(t) \right\| = 0.$$ 

Assume that there exists a synchronous orbit $s(t)$, then it is just needed to check $\left\| x_i(t) - s(t) \right\| \to 0$ as $t \to \infty$ to achieve asymptotical synchronization. As for the case of distinct synchronous orbits $s_k(t)$, if $\left\| x_i(t) - s_k(t) \right\| \to 0$ as $t \to \infty$, $i$ belongs to distinct groups $k$, then the dynamical network achieves cluster synchronization.

**Definition 2.** A continuous function $f(t,x) : [0, +\infty) \times \mathbb{R}^n \to \mathbb{R}^n$ is said to be in a QUAD function class, denoted as $f \in \text{QUAD}(\mathcal{F}, \Delta, \eta)$, if there exists a positive definite diagonal matrix $\mathcal{F} = \text{diag} \{ p_1, p_2, \cdots, p_n \}$, a diagonal matrix $\Delta = \text{diag} \{ \delta_1, \delta_2, \cdots, \delta_n \}$ and a constant $\eta > 0$, such that $f$ satisfies following condition:

$$(x - y)^T \mathcal{F} (f(t,x) - f(t,y) - \Delta(x - y)) \leq -\eta(x - y)^T (x - y)$$

for all $x, y \in \mathbb{R}^n$.

**Remark 2.** Notice a fact that there are many chaotic systems having the properties of the QUAD function class, for example, the Lorenz system, the Rössler system, the Chen system, the Chua’s circuit, and the logistic differential system.

**Definition 3.** Matrix $L$ is said to be class $A_1$, denoted as $L \in A_1$, if the following conditions hold:

1. $l_{ij} \leq 0, i \neq j, l_{ii} = -\sum_{j=1, j \neq i}^{N} l_{ij},$

2. $L$ is irreducible.

If $L$ is symmetric, we say $L$ belongs to class $A_2$, denoted as $L \in A_2$. We can easily obtain that if $L$ is class $A_1$, then $L$ has the property of zero row-sum, e.g., $\sum_{j=1}^{N} l_{ij} = 0$.

As for a rectangular matrix $L \in \mathbb{R}^{N_1 \times N_2}$, if its each row-sum is zero, e.g., $\sum_{j=1}^{N_2} l_{ij} = 0, i = 1, 2, \cdots, N_1$, then we call $L$ is in class $A_3$, denoted as $L \in A_3$.

**Lemma 1.** For any vectors $x, y \in \mathbb{R}^n$, there exists positive $\theta$, such that

$$2x^Ty \leq \frac{1}{\theta} x^Tx + \theta y^Ty.$$

**Lemma 2.** (Gerschgorin Theorem [48]). Let $A = (a_{ij}) \in \mathbb{C}^{n \times n}$ and let $r_i = \sum_{j=1, j \neq i}^{n} |a_{ij}|, i = 1, 2, \cdots, n$. Then all the eigenvalues of $A$ lie in the union of $n$ closed discs $\bigcup_{i=1}^{n} \{ z \in \mathbb{C} : |z - a_{ii}| \leq r_i \}$, where $\mathbb{C}$ is the set of complex numbers and $\mathbb{C}^{n \times n}$ represents the complex matrices set of order $n \times n$. 

3. Main Results

The main concern of our study is controlling the behavioral dynamics system (3) to achieve desired behavior synchronization via designed controller \( u_i(t) \) during the spread of an infectious disease.

The reason to add controllers can be explained as follows. Because of unresponsive information transmission or unawareness about the disease type, when the infectious disease outbreaks, e.g., infection rate is larger than the epidemic threshold, people may not make corresponding response to this case in a short time, or the information about disease is transmitted by medical institutions to the public after some research. At this moment, some guidance or measures should be carried out on their behaviors to reach an identical action, including getting cure in hospital and isolating, or washing hands frequently and having rest and so on, which prevent a disease from next outbreak. Obviously, this kind of behavioral consistency is resulted from the disease transmission, and is achieved under external feedback controllers.

Before analyzing behavior synchronization, the model (2)’s equilibrium will be studied first.

3.1. Analysis of the infectious disease system

Let

\[
\Gamma = \{(\rho_1, \ldots, \rho_N) \in \mathbb{R}_+^N \mid 0 \leq \rho_i \leq 1, \ i = 1, \ldots, N\}.
\]

Obviously, it is easy to know that \( \Gamma \) is the positive invariant set for system (2).

**Theorem 1.** If the infection rate \( \lambda > \frac{1 - \alpha}{\rho(A)} \), then the epidemic dynamics system (2) has a positive equilibrium \( E^* = (S_1^*, \rho_1^*, \ldots, S_N^*, \rho_N^*) \), i.e., the endemic equilibrium.

**Proof.** Denote by \( \rho_i \) the steady state of \( \rho_i(t) \), and let the right-hand side equal to zero in the first one equation (2a) of the system (2), then define

\[
\Theta = \sum_{j=1}^{N} a_{ij} \rho_j.
\]

From the system (2), we have

\[
\Theta = \sum_{j=1}^{N} a_{ij} \lambda \phi_i \frac{\Theta}{1 + \lambda \phi_i \Theta}.
\]

In order to analyze the \( \Theta \)'s property, construct the auxiliary function

\[
F(\Theta) = \Theta - \sum_{j=1}^{N} a_{ij} \frac{\lambda \phi_i \Theta}{1 + \lambda \phi_i \Theta}.
\]

Then, we can get the first-order derivative at \( \Theta \)

\[
\frac{dF(\Theta)}{d\Theta} = 1 - \sum_{j=1}^{N} a_{ij} \frac{\lambda \phi_i}{(1 + \lambda \phi_i \Theta)^2},
\]

and the second-order derivative at \( \Theta \)

\[
\frac{d^2F(\Theta)}{d\Theta^2} = \sum_{j=1}^{N} a_{ij} \frac{2 \lambda^2 \phi_i^2}{(1 + \lambda \phi_i \Theta)^3} > 0,
\]

As for the first-order derivative at \( \Theta = 0 \), we have

\[
\frac{dF(\Theta)}{d\Theta} \bigg|_{\Theta=0} = 1 - \sum_{j=1}^{N} a_{ij} \lambda \phi_i < 1 - \sum_{j=1}^{N} a_{ij} \lambda (1 - \alpha).
\]
Then, it is necessary for the equation $F(\Theta) = 0$ to have a unique positive solution within the internal $(0, 1)$ that the following relation holds

$$\lambda > \frac{1}{(1 - \alpha) \sum_{j=1}^{N} a_{ij}},$$

and inequality (9) means

$$\lambda > \frac{1}{(1 - \alpha) \max_{i} \sum_{j=1}^{N} a_{ij}}.$$ 

On the other hand, according to the Gersgorin disc theorem,

$$|\lambda_i(A)| \leq \sum_{j=1}^{N} a_{ij},$$

so we further know that

$$\max_{i} |\lambda_i(A)| \leq \max_{i} \sum_{j=1}^{N} a_{ij}.$$ 

From the definition of spectral radius for the matrix $A$, we have

$$\rho(A) = \max_{i} |\lambda_i(A)| \leq \max_{i} \sum_{j=1}^{N} a_{ij}. $$

Therefore, by the condition

$$\lambda > \frac{1}{(1 - \alpha) \rho(A)},$$

we obtain the first-order derivative of $F(\Theta)$

$$\frac{dF(\Theta)}{d\Theta} \bigg|_{\Theta=0} < 0.$$

Then the proof is completed.

It is worthy to notice that Theorem 1 discovers when the infection rate $\lambda > \frac{1}{(1 - \alpha) \rho(A)}$, there exists $\rho_i^* \in (0, 1)$, $i = 1, 2, \cdots, N$, such that

$$\lim_{t \to \infty} \rho_i(t) = \rho_i^*, \quad i = 1, 2, \cdots, N.$$

Next, the global stability of disease-free equilibrium will be considered. Some mathematical expressions are given for analytical requirement. Setting the positive left eigenvector corresponding to the maximum eigenvalue of $A$ as $(\omega_1, \cdots, \omega_N)^T \in \mathbb{R}^N$, which indicates that

$$(\omega_1, \cdots, \omega_N)A = (\omega_1, \cdots, \omega_N)\rho(A).$$

**Theorem 2.** The disease-free equilibrium in the epidemic dynamics system (2) is globally asymptotically stable if the infection rate $\lambda \leq \frac{1}{(1 + \alpha) \rho(A)}$. While it is unstable when $\lambda > \frac{1}{(1 - \alpha) \rho(A)}$. 

\[\square\]
Proof. Choose the following Lyapunov functional

\[ V(t) = \sum_{i=1}^{N} \omega_i \rho_i(t). \]  

Differentiating the function \( V(t) \) along the trajectories of Eq. (14), we obtain

\[ \dot{V}(t) = \sum_{i=1}^{N} \omega_i \dot{\rho}_i(t) \]

\[ = \sum_{i=1}^{N} \omega_i \left[ -\rho_i(t) + \lambda \varphi_i(t)(1 - \rho_i(t)) \sum_{j=1}^{N} a_{ij} \rho_j(t) \right] \]

\[ = -\sum_{i=1}^{N} \omega_i \rho_i(t) + \lambda \sum_{i=1}^{N} \sum_{j=1}^{N} \omega_i \varphi_i(t) a_{ij} \rho_j(t) - \lambda \sum_{i=1}^{N} \sum_{j=1}^{N} \omega_i \rho_i(t) \left[ 1 - \alpha \rho_i(t) \frac{e_i^T(t)e_i(t)}{\sum_{i=1}^{N} e_i^T(t)e_i(t)} \right] a_{ij} \rho_j(t) \]

\[ \leq \left[ \lambda(1 + \alpha) \rho(A) - 1 \right] \omega \rho, \]

where \( \omega = (\omega_1, \cdots, \omega_N)^T \in \mathbb{R}^N, \rho = (\rho_1(t), \cdots, \rho_N(t))^T \in \mathbb{R}^N. \)

Therefore, if \( \lambda \leq \frac{1}{(1+\alpha)\rho(A)} \), we have \( \dot{V}(t) \leq 0 \), and \( \dot{V}(t) = 0 \) if and only if \( \rho_i(t) = 0, i = 1, 2, \cdots, N. \) By LaSalle’s Invariance Principle, disease-free equilibrium is globally asymptotically stable.

On the other hand, inequality relation \( (1 - \alpha)\rho(A) < (1 + \alpha)\rho(A) \) indicates that \( \frac{1}{(1-\alpha)\rho(A)} > \frac{1}{(1+\alpha)\rho(A)} \). And from the Theorem 1 which means that the \( \dot{V}(t) > 0 \) while \( \lambda > \frac{1}{(1-\alpha)\rho(A)} \). That is to say, the disease-free equilibrium is unstable, and there is one positive equilibrium in the interior \( \Gamma \) of \( \bar{\Gamma} \).

3.2. Synchronization on infectious disease complex network system with controllers

To control the transmission of an infectious disease, we want to make the behavioral dynamics system (3) reach a suitable behavior synchronization under the designed external controllers. We will achieve distinct synchronous types according to selecting different control strategies on the epidemic controlling synchronization system. Assuming that there exist synchronous orbits \( s(t) \) and \( s_k(t) \) for two distinct synchronous types as the complete synchronization machine and the cluster synchronization for two groups based on the information of individuals’ disease, respectively.

A. Control the infected individuals or nodes

A well known fact indicates that control all nodes on complex network system is difficult to implement and unrealistic, while it is effective and enforceable to control a portion of nodes. Therefore, we design the following external controllers

\[ u_i(t) = -d_i \rho_i(t)(x_i(t) - s(t)) \]  

in which \( d_i \) is the control strength, and \( d_i > 0 \) when \( i = 1, 2, \cdots, r_1 \), and \( d_i = 0 \) otherwise.

Remark 3. Note that the designed controllers are only added to infectious individuals, under pinning feedback controllers, the whole network containing infectious and susceptible individuals achieves desired behavior synchronization.

Let \( s(t) \) be the synchronous target that satisfies the uncoupled system as follows

\[ \dot{s}(t) = f(s(t)). \]  

Define the errors between node and target as \( e_i(t) = x_i(t) - s(t), i = 1, 2, \cdots, N. \) Then we can get the error system with controllers

\[ \dot{e}_i(t) = f(x_i(t)) - f(s(t)) - \sum_{j=1}^{N} l_{ij} e_j(t) - d_i \rho_i(t) e_i(t). \]
Theorem 3. Assume the local dynamics $f$ belongs to the QUAD function class. When an infectious disease spreads, which means $\lambda > \frac{1}{(1-\eta)\rho_{\min}}$, then the endemic equilibrium is globally asymptotically stable. Under this condition, the behavioral dynamics system \(3\) can achieve behavior synchronization under the extra controllers \(16\) if there exists a positive $\eta > 0$ and the following condition holds:

$$(-\eta - \lambda_2\lambda_{\min}(P))I_{Nn} + I_N \otimes (P\Delta) - D \otimes P < 0,$$

where

$$D = \text{diag} \{ (\rho_1^* - \varepsilon), \cdots, \rho_l^* - \varepsilon, 0, \cdots, 0 \}.$$  

Proof. Choose the functional

$$V(t) = V_1(t) + V_2(t),$$

in which

$$V_1(t) = \frac{1}{2} \sum_{i=1}^{N} e_i^T(t) Pe_i(t),$$

$$V_2(t) = \sum_{i=1}^{N} c_i V_{2i}, c_i \geq 0,$$

where

$$V_{2i} = S_i(t) - S_i^* - \sum_{j=1}^{N} \rho_i(t) \ln \frac{S_i(t)}{S_i^*} + \rho_i(t) - \rho_i^* \ln \frac{\rho_i(t)}{\rho_i^*}.$$  

Obviously, $V_1(t)$ is a Lyapunov function, while $V_2(t)$ needs to be clarified further. Next, differentiating the function $V(t)$ along the trajectories of Eq. \(3\).

Firstly, according to the error systems \(18\), we can get the differential form

$$\dot{V}_1(t) = \sum_{i=1}^{N} e_i^T(t) Pe_i(t)$$

$$\leq -\eta \sum_{i=1}^{N} e_i^T(t) Pe_i(t) + \sum_{i=1}^{N} e_i^T(t) P\Delta e_i(t) - \sum_{i=1}^{N} \sum_{j=1}^{N} e_i^T(t) P l_{ij} e_j(t) - \sum_{i=1}^{N} e_i^T(t) P d_i \rho_i(t) e_i(t).$$

Let $y(t) = (y_1^T(t), y_2^T(t), \cdots, y_N^T(t))^T = (U^T \otimes I_N) e(t)$, where $U$ is the unitary matrix that satisfies $U^TU = UU^T = I$. Then, we have

$$\sum_{i=1}^{T} \sum_{j=1}^{N} e_i^T(t) P l_{ij} e_j(t) = e^T(t)(L \otimes P)e(t)$$

$$= y^T(t)(\Lambda \otimes P)y(t)$$

$$= \sum_{i=1}^{N} y_i^T(t) \lambda_i P y_i(t)$$

$$\geq \lambda_2\lambda_{\min}(P) \sum_{i=1}^{N} y_i^T(t) y_i(t).$$
\[ V_1(t) = \lambda_2 \lambda_{\text{min}}(P) \sum_{i=1}^{N} e_i^T(t)e_i(t) \]

Additionally, by the condition \( \lambda > \frac{1}{(1-\alpha_p)(\beta)} \), we have

\[ \sum_{i=1}^{N} e_i^T(t)Pd_i(\rho_i(t)e_i(t)) > \sum_{i=1}^{N} e_i^T(t)Pd_i(\rho_i^* - \varepsilon)e_i(t). \]

Therefore, one can obtain

\[ \dot{V}_1(t) \leq e^T(t) \left[ (-\eta - \lambda_2 \lambda_{\text{min}}(P))I_{1\times N} + I_N \otimes (P\Delta) - D \otimes P \right] e(t), \]

where

\[ D = \text{diag}\{ (d_1(\rho_1^* - \varepsilon), \ldots, d_{r_1}(\rho_{r_1}^* - \varepsilon), 0, \ldots, 0) \} \]

Now, as for \( V_2(t) \), we obtain the \( \dot{V}_2(t) \)'s differential along the trajectories of Eq. 3.

\[ \dot{V}_2(t) = \dot{S}_i(t) - \frac{S_i^*}{S_i(t)} \dot{S}_i(t) + \dot{\rho}_i(t) - \frac{\rho_i^*}{\rho_i(t)} \dot{\rho}_i(t) \]

\[ = \left( 1 - \frac{S_i^*}{S_i(t)} \right) \left[ \dot{\rho}_i(t) - \lambda \varphi_i(t)(1 - \rho_i(t)) \sum_{j=1}^{N} a_{ij} \rho_j(t) \right] \]

\[ + \left( 1 - \frac{\rho_i^*}{\rho_i(t)} \right) \left[ - \rho_i(t) + \lambda \varphi_i(t)(1 - \rho_i(t)) \sum_{j=1}^{N} a_{ij} \rho_j(t) \right]. \]

Since \( S_i^*, \rho_i^*, i = 1, 2, \ldots, N \), are the positive equilibrium of the epidemic dynamics system 3, e.g., there are following relations:

\[ -\rho_i^* + \lambda \varphi_i(t) S_i^* \sum_{j=1}^{N} a_{ij} \rho_j^* = 0 \]

and

\[ S_i^* + \lambda \varphi_i(t) \sum_{j=1}^{N} a_{ij} \rho_j^* = 1. \]

Then, we have

\[ \dot{V}_2(t) = \left( 1 - \frac{S_i^*}{S_i(t)} \right) \left[ S_i^* + \lambda \varphi_i(t) S_i^* \sum_{j=1}^{N} a_{ij} \rho_j^* - S_i(t) - \lambda \varphi_i(t) S_i(t) \sum_{j=1}^{N} a_{ij} \rho_j(t) \right] \]

\[ + \left( 1 - \frac{\rho_i^*}{\rho_i(t)} \right) \left[ - \rho_i^* + \lambda \varphi_i(t) S_i^* \sum_{j=1}^{N} a_{ij} \rho_j^* - \rho_i(t) + \lambda \varphi_i(t) S_i(t) \sum_{j=1}^{N} a_{ij} \rho_j(t) \right] \]

\[ = \frac{1}{S_i(t)} (S_i(t) - S_i^*) \left[ -(S_i(t) - S_i^*) + \lambda \varphi_i(t) S_i^* \sum_{j=1}^{N} a_{ij} \rho_j^* - \lambda \varphi_i(t) S_i(t) \sum_{j=1}^{N} a_{ij} \rho_j(t) \right] - \rho_i^* \]

\[ + \lambda \varphi_i(t) S_i^* \sum_{j=1}^{N} a_{ij} \rho_j^* - \rho_i(t) + \lambda \varphi_i(t) S_i(t) \sum_{j=1}^{N} a_{ij} \rho_j(t) + \frac{\rho_i^2}{\rho_i(t)} - \lambda \varphi_i(t) \frac{\rho_i^* S_i^*}{\rho_i(t)} \sum_{j=1}^{N} a_{ij} \rho_j^* \]
Let \( \rho_i(t) \) be the density of infected individuals, and \( \phi_i(t) \) be the density of uninfected individuals. Under the controllers (16), the behavioral dynamics system (3) achieves behavior synchronization.

Dividing all individuals into two groups, infected and uninfected, as shown in the following:

\[
\{1, 2, \ldots, r_1, r_1 + 1, r_1 + 2, \ldots, r_2\},
\]

infected individuals

\[
\{r_{k-1} + 1, r_{k-1} + 2, \ldots, r_k\},
\]

Uninfected individuals

where \( k = 1, 2 \), and \( r_0 = 0, r_2 = N \).
Blocking topology structure of the network system, we obtain

\[ L = \begin{bmatrix}
  l_{11} & l_{12} & \cdots & l_{1N} \\
  l_{21} & l_{22} & \cdots & l_{1N} \\
  \vdots & \vdots & \ddots & \vdots \\
  l_{N1} & l_{N2} & \cdots & l_{NN}
\end{bmatrix} \]

where \( L_{kk} \in \mathbb{R}^{(r_k-r_k-1) \times (r_k-r_k-1)} \) belongs to \( A_2 \), \( L_{kg} \in \mathbb{R}^{(r_k-r_k-1) \times q_k} \), \( q_k = N - (r_k - r_k - 1) \), belongs to \( A_3 \).

Then, we design the controllers for infected individuals and uninfected individuals, respectively, as follows:

\[
 u_i(t) = \begin{cases} 
 -d_i \rho_i(t)(x_i(t) - s_1(t)), & i \in \{1, 2, \cdots, r_1\}, \\
 -\beta \xi_i S_i(t)(x_i(t) - s_2(t)), & i \in \{r_1 + 1, r_1 + 2, \cdots, r_2\}.
\end{cases}
\]

(24)

where \( d_i \) is the infected controlling strength added to \( i \)-th infected individual, and \( d_i > 0 \) when \( i = 1, 2, \cdots, r_1 \), otherwise \( d_i = 0 \). \( \xi_i \) is the uninfected controlling strength to susceptible individuals, and \( \xi_i > 0 \) when \( i = r_1 + 1, r_1 + 2, \cdots, r_1 + s \), otherwise \( \xi_i = 0 \). \( \beta \) is the global coefficient of elasticity for susceptible individuals.

**Remark 4.** In this control strategy, individuals in the complex network system will reach two desired consistent behaviors. On the one hand, individuals in the infected group will go to hospital to receive treatment or to be isolated from getting in touch with healthy individuals. On the other hand, the susceptible individuals in the uninfected group are going to wash their hands frequently or have a rest to keep nice health state. What’s more, they are informed to avoid staying at crowded place for long time and to open the window maintaining good indoor ventilation. Namely, the epidemic behavioral cluster synchronization is reached for two groups based on the information about individuals’ infection.

Therefore, we consider the synchronization state as follows:

\[
 \dot{s}_k(t) = f(s_k(t)).
\]

(25)

Then the corresponding errors can be designed as

\[
 e_i(t) = x_i(t) - s_k(t), \quad i \in \mathbb{R}, \quad k = 1, 2.
\]

Before theoretical analysis, we give some notations that will be used later. Let \( Q = \{1, 2, \cdots, r_k - 1, r_k + 1, \cdots, N\} \), \( e_i^{(k)}(t) = (e_{r_k-1+1,j}(t), e_{r_k-1+2,j}(t), \cdots, e_{r_k,j}(t))^T \in \mathbb{R}^{r_k-r_k-1} \), and \( e_i^{(q)}(t) = (e_{1,j}(t), \cdots, e_{r_k-1,j}(t), e_{r_k+1,j}(t), \cdots, e_{N,j}(t))^T \in \mathbb{R}^{N-(r_k-r_k-1)} \).

**Theorem 4.** Assume that the local dynamics \( f \) belongs to the \textit{QUAD} function class. When the infection rate \( \lambda > \frac{1}{(1-\rho_1)\rho(A)} \), which indicates that the disease may spread, then the endemic equilibrium \( E^* \) is globally asymptotically stable, as well as the behavioral dynamics system (3) will achieve behavioral cluster synchronization for two groups based on the information about individuals’ infection under the grouped control strategies (24) if there exists a positive \( \theta > 0 \) such that:

\[
 \left( \frac{\Lambda}{\lambda_{\min}(P)} + \frac{\theta}{2} - d_k^* \right) I_{r_k-r_k-1} - L_{kk} + \frac{1}{2\theta} L_{kk} L_{kk}^T < 0, \quad k = 1, 2,
\]

(26)

where \( d_1^* = \min_{i \in \{1, 2, \cdots, r_1\}} \{d_i(\rho_i^* - \varepsilon)\} \), and \( d_2^* = \min_{i \in \{r_1+1, \cdots, r_1+s\}} \{\beta \xi_i (1 - \rho_i^* - \varepsilon)\} \).

**Proof.** Define the Lyapunov function as follows,

\[
 V(t) = V_1(t) + V_3(t),
\]

(27)
where \( V_2(t) \) has the form shown as

\[
V_2(t) = \sum_{k=1}^{2} V_2^{(k)}(t),
\]

in which

\[
V_2^{(k)}(t) = \sum_{i \in K} c_i^{(k)} V_{2i}^{(k)},
\]

and consisting with the case of different groups, \( V_{2i}^{(k)} \) is defined as

\[
V_{2i}^{(k)} = S_{k,i}(t) - S_{k,i}^* - S_{k,i}^* \ln \frac{S_{k,i}(t)}{S_{k,i}^*} + \rho_{k,i}(t) - \rho_{k,i}^* \ln \frac{\rho_{k,i}(t)}{\rho_{k,i}^*}.
\]

And also \( S_{k,i}(t), \rho_{k,i}(t) \) are supposed to satisfy the corresponding epidemic models as

\[
\begin{align*}
\dot{\rho}_{k,i}(t) &= -\rho_{k,i}(t) + \lambda \varphi_{k,i}(t)(1 - \rho_{k,i}(t)) \sum_{j=1}^{N} a_{ij} \rho_j(t) \\
\dot{S}_{k,i}(t) &= \rho_{k,i}(t) - \lambda \varphi_{k,i}(t) S_{k,i}(t) \sum_{j=1}^{N} a_{ij} \rho_j(t).
\end{align*}
\]

And the analysis for \( V_{2i}^{(k)}(t) \) is similar as in Theorem 3 for \( V_{2i}(t) \), and have the same results that \( V_{2i}^{(k)}(t) \) is a Lyapunov function, for which \( \dot{V}_{2i}^{(k)}(t) \leq 0 \) and \( \dot{V}_{2i}^{(k)}(t) = 0 \) if and only if \( S_{k,i}(t) = S_{k,i}^*, \rho_{k,i}(t) = \rho_{k,i}^*, i \in K, k = 1, 2 \). Then, \( V_2(t) \) can be claimed to be a Lyapunov function, and the invariant set of \( \dot{V}_2(t) = 0 \) is the single point set written as \( \{(S_{1,i}^+, \rho_{1,i}^+, \ldots, S_{1,r_1}^+, \rho_{1,r_1}^+, \ldots, S_{2,r_2}^+, \rho_{2,r_2}^+)\} \).

Then, \( V_3(t) \) is also selected as (21), but has another equivalence form for cluster analysis,

\[
V_3(t) = \frac{1}{2} \sum_{k=1}^{2} \sum_{i \in K} c_i^T(t) P e_i(t).
\]

At first, differentiate the \( V_3(t) \)

\[
\dot{V}_3(t) = \sum_{k=1}^{2} \sum_{i \in K} c_i^T(t) P \dot{e}_i(t)
\]

\[
= \sum_{k=1}^{2} \sum_{i \in K} c_i^T(t) P [f(x_i(t)) - f(s_k(t))] - \sum_{j=1}^{N} l_{ij} e_j(t) + u_i(t)]
\]

\[
= \sum_{k=1}^{2} \sum_{i \in K} c_i^T(t) P [f(x_i(t)) - f(s_k(t))] - \sum_{i=1}^{N} \sum_{j=1}^{N} c_i^T(t) P l_{ij} e_j(t) + \sum_{i=1}^{T} c_i^T(t) P u_i(t)
\]

Next, we analyze each term in the aspect of clustering shown above.

For the first term, from the property of QUAD function class, we have

\[
\sum_{k=1}^{2} \sum_{i \in K} c_i^T(t) P [f(x_i(t)) - f(s_k(t))] \leq \zeta \sum_{k=1}^{2} \sum_{i \in K} c_i^T(t) e_i(t)
\]

(29)
\[ \leq \frac{\zeta}{\lambda_{\min}(P)} \sum_{k=1}^{2} \sum_{i \in K} e_i^T(t) P e_i(t) \]
\[ = \frac{\zeta}{\lambda_{\min}(P)} \sum_{k=1}^{2} \sum_{i \in K} e_i^T(t) p_j e_i(t) \]
\[ = \frac{\zeta}{\lambda_{\min}(P)} \sum_{k=1}^{2} \sum_{i \in K} \sum_{j=1}^{n} (e_i^{(k)}(t))^T e_j^{(k)}(t). \] (30)

As for the second term, it may be obtained that

\[ - \sum_{i=1}^{N} \sum_{j=1}^{N} e_i^T(t) P l_{ij} e_j(t) = - \sum_{k=1}^{2} \left[ \sum_{i,j \in K} e_i^T(t) P l_{ij} e_j(t) + \sum_{i \in K, j \in Q} e_i^T(t) P l_{ij} e_j(t) \right] \]
\[ = - \sum_{k=1}^{2} \left[ \sum_{i,j \in K} l_{ij} \sum_{l=1}^{n} p_l e_i(t) e_j(t) + \sum_{i \in K, j \in Q} l_{ij} \sum_{l=1}^{n} e_i(t) p_j e_j(t) \right] \]
\[ = - \sum_{k=1}^{2} \left[ \sum_{l=1}^{n} \sum_{i,j \in K} p_l e_i(t) l_{ij} e_j(t) + \sum_{l=1}^{n} \sum_{i \in K, j \in Q} e_i(t) l_{ij} e_j(t) \right] \]
\[ = - \sum_{k=1}^{2} \left[ \sum_{l=1}^{n} \sum_{i=1}^{n} p_l (c_i^{(k)}(t))^T L_{kk} e_i^{(k)}(t) + \sum_{l=1}^{n} \sum_{i=1}^{n} p_l (c_i^{(k)}(t))^T L_{kk} e_i^{(k)}(t) \right]. \]

According to the Lemma \[ x^T y \leq \frac{1}{2 \theta} x^T x + \frac{\theta}{2} y^T y, \text{ existing } \theta > 0, \]\ we obtain the following inequality

\[ - \sum_{i=1}^{N} \sum_{j=1}^{N} e_i^T(t) P l_{ij} e_j(t) \leq - \sum_{k=1}^{2} \sum_{j=1}^{n} p_j (\tilde{e}_j^{(k)}(t))^T L_{kk} \tilde{e}_j^{(k)}(t) \]
\[ + \sum_{k=1}^{2} \sum_{j=1}^{n} p_j \left[ \frac{1}{20} (c_i^{(k)}(t))^T L_{kk} \tilde{e}_j^{(k)}(t) + \frac{\theta}{2} \tilde{e}_j^{(q)}(t))^T \tilde{e}_j^{(q)}(t) \right]. \]

Note that \( (c_i^{(q)}(t))^T \tilde{e}_j^{(q)}(t) = \sum_{k=1}^{2} \sum_{j=1}^{n} (c_i^{(k)}(t))^T \tilde{e}_j^{(k)}(t) - (c_i^{(k)}(t))^T \tilde{e}_j^{(k)}(t), \) then we get that

\[ - \sum_{i=1}^{N} \sum_{j=1}^{N} e_i^T(t) P l_{ij} e_j(t) \leq - \sum_{k=1}^{2} \sum_{j=1}^{n} p_j (\tilde{e}_j^{(k)}(t))^T L_{kk} \tilde{e}_j^{(k)}(t) \]
\[ + \frac{1}{2 \theta} \sum_{k=1}^{2} \sum_{j=1}^{n} p_j (\tilde{e}_j^{(k)}(t))^T L_{kk} \tilde{e}_j^{(k)}(t) + \sum_{k=1}^{2} \sum_{j=1}^{n} p_j (\tilde{e}_j^{(k)}(t))^T \tilde{e}_j^{(k)}(t). \] (31)

Considering the third term, we can get

\[ \sum_{i=1}^{N} e_i^T(t) P u_i(t) = - \sum_{i=1}^{r_1} e_i^T(t) P d_i \rho_i(t) (x_i(t) - s_1(t)) - \sum_{i=r_1+1}^{N} e_i^T(t) P \beta \xi_i (1 - \rho_i(t))(x_i(t) - s_2(t)) \]

From the fact that when \( \lambda > \frac{1}{1 - \beta \xi_i (1 - \rho_i(t))(x_i(t) - s_2(t))} \) there is a unique endemic equilibrium \( E^* \) for the epidemic dynamics system \[ \text{which means that there exists an } \varepsilon > 0, \text{ and } t_0, \text{ when } t \geq t_0, \text{ then } |\rho_i(t) - \rho_i^*| < \varepsilon. \] Therefore,

\[ \sum_{i=1}^{N} e_i^T(t) P u_i(t) = - \sum_{i=1}^{r_1} \sum_{j=1}^{n} d_i \rho_i(t) e_i(t) p_j e_j(t) - \sum_{i=r_1+1}^{N} \sum_{j=1}^{n} \beta \xi_i (1 - \rho_i(t)) e_i(t) p_j e_j(t) \]
\[ \sum_{i=1}^{N} e_i^T(t) P u_i(t) = - \sum_{i=1}^{r_1} \sum_{j=1}^{n} d_i \rho_i(t) e_i(t) p_j e_j(t) - \sum_{i=r_1+1}^{N} \sum_{j=1}^{n} \beta \xi_i (1 - \rho_i(t)) e_i(t) p_j e_j(t) \]
where 
\[ d_1^* = \min_{i \in \{1, 2, \ldots, r_1\}} \{d_i(\rho_i^* - \varepsilon)\}, \quad d_2^* = \min_{j \in \{r_1+1, \ldots, r_1+s\}} \{\beta \xi_i(1 - \rho_i^* - \varepsilon)\}. \]

Hereto, combining with (29), (31) and (32), we obtain

\[ \dot{V}_3(t) \leq 2 \sum_{k=1}^{r_2} \sum_{j=1}^{n} p_j (\tilde{e}_j^{(k)}(t))^T \left[ \left( \frac{\zeta}{\lambda_{\min}(P)} + \frac{\theta}{2} - d_1^* \right) I_{r_k} - r_{k-1} - L_{kk} + \frac{1}{2q} L_{kk} \right] \tilde{e}_j^{(k)}(t) \]

(33)

In conclusion, we get

\[ \dot{V}(t) = \dot{V}_2(t) + \dot{V}_3(t) < 0 \]

if conditions (26) are satisfied.

Therefore, we can claim that when \( \lambda > \frac{1}{(1 - \rho_1^*) \mu C} \), resulting in the infectious disease transmission, then the endemic equilibrium is globally asymptotically stable. And under the control strategy (24), only when conditions (26) are satisfied, the behavioral dynamics system (3) reaches behavioral cluster synchronization for two groups based on the disease information. Then the proof is completed.

\[ \square \]

4. Numerical Simulation

In this section, we shall give two numerical simulation examples to demonstrate the obtained main results in this paper. At first, using the NW small-world principle with the average degree as 4 and the rewiring probability as 0.1, and the average degree as 6 and the rewiring probability also as 0.1, to generate two parts with size of 20 nodes and 30 nodes, respectively. And those two parts are combined by the rules that existing 2 edges from the first part to the second part and 3 edges from the second to the first one. Which generates the considered network topology with the node’s dimension as 2.

Example 1. Then, as for the first control strategy, we select the local dynamics as

\[ f(x) = C \cdot x + A \cdot \tanh x, \]

where each node has dimension 2 and

\[ C = \begin{pmatrix} -1 & 0 \\ 0 & -1 \end{pmatrix}, \quad A_1 = \begin{pmatrix} -1 & -0.5 \\ -5 & 4.5 \end{pmatrix}. \]

And we select the matrices \( P \) and \( \Delta \) as

\[ P = \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix}, \quad \Delta = 0.5 \times \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix}. \]

Then we can select \( \eta = 1.4394 \) such that

\[ (x - y)^T P[f(x) - f(y) - \Delta(x - y)] \leq -\eta(x - y)^T (x - y). \]
According to the considered network, we can get the $\lambda_2 = 0.235$. Therefore, under the synchronous condition in Theorem 3 by simple computing, we can get that
\[-\eta - \lambda_2 \lambda \min(P) + 0.5 < 0\]

So, it is only needed to select the appropriate control strength and the synchronous condition is also satisfied naturally.

From the network’s topology, we can compute the $\rho(A) = 8.2574$. Let $\alpha = 0.5$, then the epidemic threshold can be obtained as $\lambda_c = \frac{1}{(1-\alpha)\rho(A)} = 0.2422$, while $\frac{1}{(1+\alpha)\rho(A)} = 0.0807$.

Next, let the initial epidemic probability $\rho_0 = 0.01$, and select the appropriate initial values for nodes. And adding the controller to the first 10 nodes with the strength $d_i = 20$, $i = 1, \ldots, 10$. We set the infection rate $\lambda = 0.3 > \lambda_c$. Then, we can obtain the Fig. 1. From which one can see that when the infection rate is larger than the epidemic threshold, there exists a positive stable value in the end referred by the average infected density $\rho(t)$’s ($\rho(t) = \sum_{i=1}^{50} \rho_i(t)$) curve, which means the disease will spread and will continuously exist to be the endemic disease. In this case, under the feedback control strategy, the error picture tends to zero which reflects that the network achieve behavior synchronization arouse by epidemic transmission. If we select $\lambda = 0.7$, then there will be a larger stable value for $\rho(t)$ as shown in Fig. 2 and the behavior synchronization resulting from disease transmission also emerge under the pinning feedback controllers.

When select the infection rate less than the value $\frac{1}{(1+\alpha)\rho(A)}$, such as $\lambda = 0.06 < 0.0807$. From the Fig. 3 one can see that the $\rho(t)$ reach to zero in the end, which means the disease shall be extinct eventually. And the network will not achieve behavior synchronization with the error going to none zero result from the invalid controllers, as shown in the error curve.

**Example 2.** Now, we assume that the first 20 nodes constitute the infectious individual group in the above concerned network, and the latter 30 ones represent the susceptible group, we shall show that when the epidemic spreads, the network can reach two different behavior synchronization states for those two groups individuals via the second control strategy in this paper. In order to demonstrate this case, we introduce two types of local dynamical behaviors with the same form as Example 1 but distinct coefficient matrices as

$$C_2 = \begin{pmatrix} -4.5 & 0 \\ 0 & -4.5 \end{pmatrix}, A_2 = \begin{pmatrix} -2 & -0.1 \\ -5 & 4.5 \end{pmatrix} \quad \text{and} \quad C_3 = \begin{pmatrix} -1.8 & 0 \\ 0 & -1.8 \end{pmatrix}, A_3 = \begin{pmatrix} 0.1 & -0.1 \\ -0.4 & 0.4 \end{pmatrix}.$$

Then, we can get the $\zeta = 2.4462$ such that
$$(x - y)^T[f(x) - f(y)] \leq \zeta(x - y)^T(x - y).$$
Fig. 2: The epidemic probability and the synchronization errors for all nodes under the first control strategy with the infection rate $\lambda = 0.7$.

Fig. 3: The epidemic probability and the synchronization errors for all nodes under the first control strategy with the infection rate $\lambda = 0.06$. 
Select $\theta = 1$, and we can easily get that

$$\lambda_{\text{max}} \left( -L_{11} + \frac{1}{2} L_{12} L_{21}^T \right) < -\left( \zeta + \frac{1}{2} \right),$$

$$\lambda_{\text{max}} \left( -L_{22} + \frac{1}{2} L_{21} L_{12}^T \right) < -\left( \zeta + \frac{1}{2} \right),$$

so the synchronization conditions in Theorem 4 are satisfied under the appropriate control strength. In this case, we select the initial infectious probabilities for two groups as $\rho_1(0) = 0.01$ and $\rho_2(0) = 0.04$, respectively.

Then let $\lambda = 0.4 > 0.2422$, by using the second control strategy, adding the controllers to a part of nodes in each group with the appropriate control strength, the epidemic probability reach a stable state, and the network achieve two types of behavior synchronization. In Fig. 4 one can see that the $\rho_1(t)$ and $\rho_2(t)$ get the positive stable values in the end. And the errors $E_1(t)$ and $E_2(t)$ in each groups and the whole errors $E(t)$ of all nodes’ states are also tend to the zeros, while the errors $E_{12}(t)$ between two groups do not tend to zero, which indicates the main results in our paper. If we select $\lambda = 0.7$, then the infectious probability for each groups will have a higher stable values, as shown in Fig. 5.

When the infection rate is less than the value $\frac{1}{1-\alpha \rho(A)}$, e.g., $\lambda = 0.07 < 0.0807$, we can obtain Fig. 6. In which, the infectious probabilities for two groups $\rho_1(t)$ and $\rho_2(t)$ tend to zero eventually. And the errors in two groups $E_1(t)$ and $E_2(t)$, as well as the whole errors for the network do not tend to zero. Which indicates that once the infection rate is less than a certain value $\frac{1}{1-\alpha \rho(A)}$, the disease will be extinct in the end, and meanwhile the controllers are invalid so that none behavior synchronization.

**Remark 5.** As for the case that the infection rate is located between the value $\frac{1}{1-\alpha \rho(A)}$ and the epidemic threshold $\frac{1}{1-\alpha \rho(A)}$, we select two values of $\lambda$ to see what changes there will be under the two control strategies. On the one hand, let $\lambda = 0.15 \in [0.0807, 0.2422]$. Under the first control strategy, the $\rho(t)$ curve and errors are shown in Fig. 7. We can see that the infectious probability tends to a stable positive value, while the network dose not get the behavior synchronization reflected by the error curve. On the other hand, let $\lambda = 0.1$, and the infectious possibilities and the errors under the second control strategy are shown in Fig. 8. In that case, the infection rate tend to zero, and the errors so not. Hence, it may be more complex and varied in the case that the infection rate belongs to the interval $[0.0807, 0.2422]$, and some corresponding measures should be discussed according to the concrete conditions.
Fig. 5: The $\rho_1(t)$, $\rho_2(t)$, the errors in each group, all errors of the whole nodes’ states in the network and the errors between each groups with the infection rate $\lambda = 0.7$ under the second control strategy.

Fig. 6: The $\rho_1(t)$, $\rho_2(t)$, the errors in each groups, all errors of the whole nodes’ states in the network and the errors between each groups with the infection rate $\lambda = 0.07$ under the second control strategy.
Fig. 7: The epidemic probability and the synchronization errors for all nodes under the first control strategy with the infection rate $\lambda = 0.15$.

Fig. 8: The $\rho_1(t), \rho_2(t)$, the errors in each groups, all errors of the whole nodes’ states in the network and the errors between each groups with the infection rate $\lambda = 0.1$ under the second control strategy.
5. Conclusions

In this paper, we investigate the epidemic controlling behavioral synchronization problem for a class of epidemic synchronous complex dynamical networks aroused from the contagions transmission, in which the quenched mean-field theory is introduced in the epidemic system. More realistically, the case of inhibition of contact behavior is discussed. Then, based on the proven technique of pinning feedback strategy, where only a part of nodes need to be controlled, and the design of two types of pinning schemes depending on nodes’ information of infection, two desired epidemic control behavior synchronization are achieved by means of the definition of behavioral synchronization. Sufficient conditions for epidemic control behavior synchronization under the contagions spreading are derived. The numerical simulations are exhibited to demonstrate the effectiveness of the main results obtained in this paper.

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