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During the spread of an epidemic, individuals in realistic networks may exhibit collective behaviors. In order to characterize this kind of phenomenon and explore the correlation between collective behaviors and epidemic spread, in this paper, we construct several mathematical models (including without delay, with a coupling delay, and with double delays) of epidemic synchronization by applying the adaptive feedback motivated by real observations. By using Lyapunov function methods, we obtain the conditions for local and global stability of these epidemic synchronization models. Then, we illustrate that quenched mean-field theory is more accurate than heterogeneous mean-field theory in the prediction of epidemic synchronization. Finally, some numerical simulations are performed to complement our theoretical results, which also reveal some unexpected phenomena, for example, the coupling delay and epidemic delay influence the speed of epidemic synchronization. This work makes further exploration on the relationship between epidemic dynamics and synchronization dynamics, in the hope of being helpful to the study of other dynamical phenomena in the process of epidemic spread. *Published by AIP Publishing*. [http://dx.doi.org/10.1063/1.4984217]

During the spread of an infectious disease in a population, the behavioral synchronization of individuals, such as avoiding assemblage, washing hands, etc., emerges in some way to protect themselves. On the other hand, this behavioral response, usually with delay, will reduce the disease spread to some degree. In order to precisely analyze this behavioral synchronization of individuals and disease spreading dynamics and understand their interplay mathematically and numerically, the first step should be the construction of a suitable model which can display similar properties for these real dynamical networks. Taking account of the behavioral synchronization of individuals, the current manuscript investigates the coupling dynamics of the disease spread and behavioral changes during an epidemic. Three models are proposed focusing on various aspects: without delay; with one delay to incorporate the time lag in epidemic information processing and individuals' active response; with two delays where the other delay representing the incubation period of the vector-borne pathogen in the vector population. The local and global stability of these synchronization models is studied. Furthermore, the effects of time delays are evaluated through numerical simulations, which reveal some unexpected phenomena.

I. INTRODUCTION

Many natural and artificial systems, such as biological systems, electrical power systems, and social systems, can be described by complex networks, with each node bearing different meanings in various fields.[1–4] Complex networks, as a subject of interdisciplinary research, have received much attention from researchers in the fields of epidemiology, physics, and social sciences. Complex networks have been successfully used to study epidemic spread,[5] where each node represents an individual, while the link between two nodes denotes the interaction between two individuals.

In general, when an epidemic occurs, individuals adaptively change their respective behaviors to avoid being infected based on epidemic information, such as washing hands frequently, wearing masks, seeking medical care, and avoiding contact with infected individuals.[6] When the infection prevalence becomes larger, individuals send/receive safeguard information more frequently to protect themselves. Furthermore, with the evolution of epidemics, the risk-averse behaviors of individuals may tend to be consistent.[7] These consistent human behaviors, which result from the emergence and spread of epidemics, are defined as epidemic synchronization, independent of the actual epidemic pathology.[8]

Synchronization is a basic motion in nature that has been studied for a long time.[8–12] In the past decade, the synchronization of complex dynamical networks, such as small-world and scale-free dynamical networks,[13,14] has been studied extensively. Criteria for complete synchronization of dynamical networks are derived, where the interaction between network topologies and nodes' dynamics is crucial.[15,16] Unfortunately, little work is devoted to the study of synchronization during epidemic spread.[8,17] Li *et al.*[8] proposed the models of SIS (susceptible-infected-susceptible) and SIR (susceptible-infected-recovered) epidemic synchronization based on heterogeneous mean-field (HMF) theory and studied the dynamics of SIS epidemic synchronization. Li *et al.*[17] also investigated the interplay between collective

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behaviors and spreading dynamics on complex networks and analyzed the control problem of spreading behaviors.

The spread of an epidemic can trigger human behavioral changes, which in turn affect the evolution of the epidemic.[8,17–19] Particularly, some certain synchronization patterns of individual behaviors can greatly weaken the infection prevalence.[17] The previous study[8] focused on how the epidemic spread influences the synchronization behaviors of individuals, while work[17] not only investigated the effects of disease transmission on the synchronization behaviors of individuals, but also studied the effects of behavioral synchronization on the epidemic dynamics. However, these two papers[8,17] were performed based on heterogeneous mean-field (HMF) theory. Here, we adopt a more accurate theory —quenched mean-field (QMF) theory—in predicting epidemic synchronization. In order to better evaluate the influence of epidemic spread on synchronization dynamics in complex networks, we not only consider the effects of individual's protective behaviors on epidemic dynamics which change the epidemic threshold and reduce the risk of infection,[17] but also incorporate the following factors:

(i) In actual information networks, due to the finiteness of signal transmission and switching speeds, the coupling delay is non-negligible and should be explicitly incorporated into our model in order to describe more realistic scenarios.[20,21]

(ii) Meanwhile, time delay also plays an important role in the propagation process of epidemics, and temporal delay in epidemic models can be used to describe the effects of latency period or immunity period.[22,23] Classical epidemic models with time delay have been studied extensively, but very little attention has been paid to the effect of the time delay on heterogeneous networks.[24,25]

(iii) Some pathogens are transmitted through direct contact between individuals, while some others, such as malaria and avian flu, are transmitted to humans through the vectors (e.g., mosquitoes, birds, etc.). This aspect leads directly to the actions that human beings will take to reduce the contact with vectors by direct ways, such as using mosquito-repellent incense, sparge agent, mosquito-curtain, screen window, or indirect ways, such as killing mosquitos, which are very different from human responses to a directly transmitted disease. In this context, individuals' protective behaviors are induced by epidemics, however, are aiming for vectors. Moreover, the speed of behavioral synchronization of individuals with respect to vector diseases may be slower than that with respect to directly transmitted diseases. This may be because in vector-borne diseases, the newly infected vectors by infectious individuals cannot directly infect humans, while transmitting this infection to humans after a period of time.

Therefore, in this paper, in view of the above factors that cannot be ignored in the process of actual epidemic and information dissemination, we construct three different mathematical models to make a further exploration of the correlation between collective behaviors and epidemic spread. Here, we introduce the coupling delay between individuals' information communications and epidemic delay in the process of epidemic spread. Further, we investigate the effects of double delays on synchronization dynamics in epidemic networks.

The rest of this paper is organized as follows. In Sec. II, we introduce a complex dynamical network model with time-varying coupling strength and the standard SIS (susceptible-infected-susceptible) model in quenched networks. In Sec. III, we propose three different SIS epidemic synchronization models (including without delay, with a coupling delay, and with double delays). Then, we investigate the local and global stability of these epidemic synchronization models with respect to the epidemic spreading rate in Secs. IV–VI, respectively. In Sec. VII, we discuss two theoretical methods (heterogeneous mean-field theory and quenched mean-field theory) to the dynamics of the SIS model in networks, which justify the use of a quenched mean-field model to study epidemic synchronization. In Sec. VIII, some numerical simulations are carried out to complement our theoretical results obtained in Secs. IV–VII. Finally, Sec. IX concludes this paper and proposes interesting questions for further investigation.

II. MODELS AND PRELIMINARIES

A. Complex dynamical network model

Consider a complex dynamical network consisting of N linearly and diffusively coupled identical nodes, each of which is an n-dimensional dynamical system. The state equations of the network with time-varying coupling strength can be described by

$$\dot{x}_i(t) = f(x_i(t)) + c(t)\sum_{j=1}^{N} a_{ij}\Gamma\left[x_j(t) - x_i(t)\right], \quad i = 1, 2, \ldots, N, \tag{1}$$

where $x_i(t) = (x_{i1}(t), x_{i2}(t), \ldots, x_{in}(t))^T \in R^n$ is the state variable of the i-th node at time t, $t \in [0, +\infty)$. In this model, $f : R^n \to R^n$ is a continuously nonlinear function which describes the local dynamics of nodes; $c(t) > 0$ denotes the coupling strength; $\Gamma = diag(\gamma_1, \gamma_2, \ldots, \gamma_n) \in R^{n\times n}$ represents the inner-coupling matrix which is a positive definite diagonal matrix; $A = (a_{ij})_{N\times N}$ is the adjacency matrix of the network, with element a_{ij} defined as follows: if there is a connection between node i and node $j\,(i \neq j)$, then $a_{ij} = a_{ji} = 1$; otherwise, $a_{ij} = a_{ji} = 0$. Based on the adjacency matrix A, the Laplacian matrix $L = (l_{ij})_{N\times N}$ of the network can be defined as follows:

$$l_{ij} = \begin{cases} -a_{ij}, & i \neq j, \\ \sum\limits_{\substack{j=1 \\ j\neq i}}^{N} a_{ij}, & i = j. \end{cases}$$

Obviously, the diagonal elements of the Laplacian matrix L satisfy the following condition:

$$l_{ii} = -\sum_{\substack{j=1 \\ j\neq i}}^{N} l_{ij} = -\sum_{\substack{j=1 \\ j\neq i}}^{N} l_{ji} = k_i, \quad i = 1, 2, \ldots, N, \tag{2}$$

where k_i denotes the degree of node i.

Assume that L is an irreducible matrix, which means that the network is strongly connected in the sense of having no isolated clusters. It follows from Ref. 26 that zero is the smallest eigenvalue of matrix L with multiplicity 1 and all the other eigenvalues are strictly positive. Since L is an irreducible real symmetric matrix with the condition (2), there exists a unitary matrix U such that $L = U\Lambda U^T$, where $U^T U = I$ and $\Lambda = diag(\lambda_1, \lambda_2, \ldots, \lambda_N)$ with $0 = \lambda_1 < \lambda_2 \leq \cdots \leq \lambda_N$.

Then, the dynamical network model (1) can be reformulated in terms of the Laplacian matrix L as:

$$\dot{x}_i(t) = f(x_i(t)) - c(t)\sum_{j=1}^{N} l_{ij}\Gamma x_j(t), \quad i = 1, 2, \ldots, N. \tag{3}$$

Let $s(t) = \frac{1}{N}\sum_{i=1}^{N} x_i(t)$, which can be an equilibrium point, a periodic orbit, or even a chaotic attractor, satisfying $\dot{s}(t) = f(s(t))$ by Eq. (3). We define the state error variables $e_i(t) = x_i(t) - s(t)$ for $i = 1, 2, \ldots, N$, obviously, we can obtain $\sum_{i=1}^{N} e_i(t) = 0$. The dynamical network (3) is said to achieve asymptotical synchronization if

$$\lim_{t\to\infty} ||e_i(t)|| = 0, \quad i = 1, 2, \ldots, N.$$

B. Epidemic network model

The SIS model is a paradigmatic epidemic spreading model, in which each node can be in one of two states, either susceptible or infected. Infected nodes become susceptible with unit rate, and each susceptible node becomes infected by its infective neighbors with the infection rate λ. We consider the standard SIS model in a quenched network of size N. Let $\rho_i(t)$ denote the infection probability of node i at time t. Neglecting correlations between infected and susceptible nodes, the evolution equation[27–29] of node i can be described by

$$\dot{\rho}_i(t) = -\rho_i(t) + \lambda\left[1 - \rho_i(t)\right]\sum_{j=1}^{N} a_{ij}\rho_j(t), \quad i = 1, 2, \ldots, N, \tag{4}$$

where the infection rate $\lambda \in (0, 1]$ denotes the probability that each susceptible node is infected if it is connected to one infected node, a_{ij} is an element of the adjacency matrix assigned with 1 if there is an edge between nodes i and j or 0 otherwise.

III. EPIDEMIC SYNCHRONIZATION MODELS

Before formulating concrete epidemic synchronization models, we make the following basic assumptions:

(A1) There is a weakly linear coupling between individuals in the dynamical network when an epidemic begins to spread;

(A2) Individuals will transmit protective information more frequently to protect themselves when the infection prevalence (measured by the average infection density $\rho(t) = \frac{1}{N}\sum_{i=1}^{N} \rho_i(t)$) becomes larger, which implies that the rate of change of the coupling strength, $\dot{c}(t)$, is directly proportional to the infection prevalence $\rho(t)$;

(A3) When the collective protective behaviors increase significantly, the communication of safeguard information among individuals will become saturated because they have come to an agreement of protection. Thus, the proportional relationship between the rate of change of the coupling strength, $\dot{c}(t)$, and the synchronization error $\sum_{i=1}^{N} e_i^T(t)e_i(t)$ always remains valid, where $e_i(t) = x_i(t) - s(t)$ for $i = 1, 2, \ldots, N$.

Based on these assumptions, in Subsections III A–III C, we propose three models in quenched networks to investigate the coupling dynamics of the epidemic transmission and behavioral changes.

A. Epidemic synchronization model without time delay

Based on the assumptions (A1)–(A3), we use model (4) to characterize epidemic spread and model (3) to describe the evolution process of protective behaviors of individuals in the process of epidemic spread, then we can construct the SIS epidemic synchronization model as follows:

$$\begin{cases} \dot{x}_i(t) = f(x_i(t)) - c(t)\sum_{j=1}^{N} l_{ij}\Gamma x_j(t), \\ \dot{\rho}_i(t) = -\rho_i(t) + \lambda\left[1 - \rho_i(t)\right]\sum_{j=1}^{N} a_{ij}\rho_j(t), \\ \dot{c}(t) = \alpha\rho(t)\sum_{j=1}^{N} e_j^T(t)e_j(t), \end{cases} \tag{5}$$

where $i = 1, 2, \ldots, N$, parameter $\alpha > 0$ and $\rho(t) = \frac{1}{N}\sum_{i=1}^{N} \rho_i(t)$ is the infection prevalence. The initial condition of system (5) can be set as follows: the initial state $x_i(0) = \left(x_{i1}(0), x_{i2}(0), \ldots, x_{in}(0)\right)^T \in R^n$, the initial coupling strength $c(0) > 0$, and the initial infection probability $\rho_i(0) = \epsilon$ of node i, where $0 < \epsilon \ll 1$.

B. Epidemic synchronization model with a coupling delay

Sometimes, due to the limitation of the switching rate and the physical distance between subsystems in real networks, the coupling delay often emerges in the procedure of information transmission.[20,21] That is, it takes non-negligible time to receive information from its neighboring nodes. To incorporate this reality, we set the following assumption:

(A4) The behavioral information that individuals obtain from their neighbors may be a kind of delayed information during the transmission of epidemics.

Therefore, it is necessary to consider the influence of the coupling delay on epidemic synchronization. In this subsection, we consider the following linearly coupled system with a coupling delay

$$\dot{x}_i(t) = f(x_i(t)) - c(t)\sum_{j=1}^{N} l_{ij}\Gamma x_j(t-\tau_1), \quad i = 1, 2, \ldots, N, \tag{6}$$

where $\tau_1 > 0$ represents the coupling delay.

Based on the assumptions (A1)–(A4) and models (4) and (6), we can construct the SIS epidemic synchronization model with a coupling delay as follows:

$$\begin{cases} \dot{x}_i(t) = f(x_i(t)) - c(t)\sum_{j=1}^{N} l_{ij}\Gamma x_j(t-\tau_1), \\ \dot{\rho}_i(t) = -\rho_i(t) + \lambda[1-\rho_i(t)]\sum_{j=1}^{N} a_{ij}\rho_j(t), \\ \dot{c}(t) = \alpha\rho(t)\sum_{j=1}^{N} e_j^T(t)e_j(t), \end{cases} \tag{7}$$

where $i = 1, 2, \ldots, N$, parameter $\alpha > 0$ and $\rho(t) = \frac{1}{N}\sum_{i=1}^{N}\rho_i(t)$ is the infection prevalence. The initial condition of system (7) can be set as follows: the initial state $x_i(\theta) = \left(x_{i1}(\theta), x_{i2}(\theta), \ldots, x_{in}(\theta)\right)^T \in R^n, \theta \in [-\tau_1, 0]$, the initial coupling strength $c(0) > 0$, and the initial infection probability $\rho_i(0) = \epsilon$ of node i, where $0 < \epsilon \ll 1$.

C. Epidemic synchronization model with double delays

During the process of epidemic spread, the influence of the past state of individuals on the current state has been investigated by introducing a temporal delay and constructing delayed epidemic models.[22–25,30] It is interesting to perform a comparative study by observing the difference between the synchronization behaviors of epidemic models with time delay and those without time delay. In this subsection, we investigate a vector-borne disease synchronization model with a coupling delay and an epidemic delay. Before we construct a vector-borne disease model in quenched networks, we first propose the assumptions, similar to those in Ref. 30:

(A5) The pathogen can only be transmitted to host individuals by a vector, such as a mosquito. That is, susceptible hosts receive the infection from infected vectors, and susceptible vectors get infected through contact with infectious hosts.
(A6) Hosts confer negligible immunity and the pathogen does not result in death or isolation.
(A7) Births, deaths, and migration of individuals are ignored, i.e., the total number of individuals is stabilized at a constant state.
(A8) When a susceptible vector is infected by an infected host, there is an incubation period (denoted by τ_2), during which, the virus develops in the vector. At the end of this time, the vector can infect a susceptible individual.
(A9) Hosts and vectors are homogeneously mixed, and therefore, the density of infected vectors at time t is simply assumed to be proportional to the density of infected hosts at a previous instant $t - \tau_2$.
(A10) Infected individuals become susceptible (or healthy) at a rate that, without loss of generality, is set to be unity.

Based on the assumptions (A5)–(A10), we can describe the vector-borne disease transmission in quenched networks through the following delay differential equations:

$$\begin{cases} \dot{S}_i(t) = \rho_i(t) - \lambda S_i(t)\sum_{j=1}^{N} a_{ij}\rho_j(t-\tau_2), \\ \dot{\rho}_i(t) = -\rho_i(t) + \lambda S_i(t)\sum_{j=1}^{N} a_{ij}\rho_j(t-\tau_2), \end{cases} \tag{8}$$

where $i = 1, 2, \ldots, N$, $\lambda > 0$, the variable $S_i(t)$ denotes the probability of being uninfected of node i at time t and τ_2 represents the incubation period of the virus in infected vectors.

Initial functions of model (8) are set as follows:

$$\begin{cases} \dot{S}_i(t) = \rho_i(t), \\ \dot{\rho}_i(t) = -\rho_i(t), \end{cases} \quad -\tau_2 \le t \le 0, \quad i = 1, 2, \ldots, N.$$

This assumption is reasonable since no vector can infect susceptible individuals during $[-\tau_2, 0]$. Actually, there may be some susceptible vectors that receive the infection from infected individuals during $[-\tau_2, 0]$, but these vectors can infect susceptible individuals only after they undergo a time interval τ_2.

Based on the assumptions (A1)–(A10), models (6) and (8), we can construct the SIS epidemic synchronization model with double delays as follows:

$$\begin{cases} \dot{x}_i(t) = f(x_i(t)) - c(t)\sum_{j=1}^{N} l_{ij}\Gamma x_j(t-\tau_1), \\ \dot{\rho}_i(t) = -\rho_i(t) + \lambda[1-\rho_i(t)]\sum_{j=1}^{N} a_{ij}\rho_j(t-\tau_2), \\ \dot{c}(t) = \alpha\rho(t)\sum_{j=1}^{N} e_j^T(t)e_j(t), \end{cases} \tag{9}$$

where $i = 1, 2, \ldots, N$, parameter $\alpha > 0$ and the infection prevalence is $\rho(t) = \frac{1}{N}\sum_{i=1}^{N}\rho_i(t)$. The initial condition of system (9) can be set as follows: the initial state $x_i(\theta) = \left(x_{i1}(\theta), x_{i2}(\theta), \ldots, x_{in}(\theta)\right)^T \in R^n, \theta \in [-\tau_1, 0]$, the initial coupling strength $c(0) > 0$, and the initial infection probability $\rho_i(s) \simeq 0, s \in [-\tau_2, 0]$ for $i = 1, 2, \ldots, N$.

IV. STABILITY ANALYSIS OF EPIDEMIC SYNCHRONIZATION MODEL WITHOUT TIME DELAY

The steady state of the infection probability of system (4) for each node i, ρ_i, is determined by the following nonlinear equation

$$\rho_i = \frac{\lambda\sum_j a_{ij}\rho_j}{1 + \lambda\sum_j a_{ij}\rho_j}, \quad i = 1, 2, \ldots, N.$$

The existence of a positive steady state, dependent on an epidemic threshold $\lambda = \lambda_c^{qmf} = 1/\Lambda_N$ (Λ_N is the largest eigenvalue of the adjacency matrix A), has been investigated in several papers, see, e.g., Refs. 31 and 32. Here, we state the fundamental lemma for the N-intertwined SIS model in Ref. 31 for reader's convenience.

Lemma 1. *There exists a value $\lambda_c^{qmf} = 1/\Lambda_N > 0$, and, for $\lambda \le \lambda_c^{qmf}$, there is only the trivial steady state solution $\rho_i = 0$ for all i. Apart from the trivial steady state solution $\rho_i = 0$, $i = 1, 2, \ldots, N$, there is a second, nonzero steady state solution for all $\lambda > \lambda_c^{qmf}$.*

By Lemma 1, it is easy to show that the zero solution becomes unstable and for system (4) there exists a nonzero solution $\rho_i > 0$ if the effective spreading rate λ is larger than the so-called epidemic threshold λ_c^{qmf}. In this case, the final infection prevalence $\rho = \frac{1}{N}\sum_{i=1}^{N} \rho_i$ is nonzero.

In Secs. IV A–IV B, we will investigate local and global stability of the epidemic synchronization model without time delay. Before we study the dynamical properties of the model, we first present the definition of the synchronization manifold for system (5).

Definition 1. *The synchronization manifold for system (5) is defined as $S = \{(x_1^T, x_2^T, \ldots, x_N^T)^T \in R^{nN} : x_i = x_j, i,j = 1, 2, \ldots, N\}$, where $x_i = (x_{i1}, x_{i2}, \ldots, x_{in}) \in R^n$, $i = 1, 2, \ldots, N$, and x_i^T represents the transpose of x_i.*

A. Local stability of the epidemic synchronization model without time delay

Substituting the error variable $e_i(t) = x_i(t) - s(t)$ into equation (3), i.e., the first equation of system (5), we can obtain the variational equation (via first order approximation) of system (3) near the synchronization state $s(t)$ as follows:

$$\dot{e}_i(t) = D(f(s(t))e_i(t) - c(t)\sum_{j=1}^{N} l_{ij}\Gamma e_j(t), \quad i = 1, 2, \ldots, N, \tag{10}$$

where $D(f(s(t))$ is the Jacobian matrix of $f(x(t))$ with respect to the synchronization state $s(t)$.

Denote $e(t) = \left(e_1^T(t), e_2^T(t), \ldots, e_N^T(t)\right)^T \in R^{nN}$, then the variation equation (10) can be written as

$$\dot{e}(t) = \left[I_N \otimes D(f(s(t))\right]e(t) - c(t)(L \otimes \Gamma)e(t),$$

where $\otimes$ represents the Kronecker product.

Let $y(t) = \left(y_1^T(t), y_2^T(t), \ldots, y_N^T(t)\right)^T = (U^T \otimes I_n)e(t)$, the variational equation in terms of $y(t)$ can be rewritten as

$$\dot{y}(t) = \left[I_N \otimes D(f(s(t))\right]y(t) - c(t)(\Lambda \otimes \Gamma)y(t). \tag{11}$$

Note that $\lambda_1 = 0$ is an eigenvalue of the Laplacian matrix L, and its corresponding eigenvector is $u_1 = \frac{1}{\sqrt{N}}(1, 1, \ldots, 1)$. Therefore, we have

$$y_1(t) = \frac{1}{\sqrt{N}}\sum_{i=1}^{N}[x_i(t) - s(t)] = 0. \tag{12}$$

According to Eq. (12), Eq. (11) can be written as

$$\dot{y}_1(t) = D(f(s(t))y_1(t) = 0, \tag{13}$$

and

$$\dot{y}_k(t) = [D(f(s(t)) - c(t)\lambda_k\Gamma]y_k(t), \quad k = 2, 3, \ldots, N. \tag{14}$$

To proceed, we need the following result from previous studies:[33,34]

Proposition 1. *If all transverse Lyapunov exponents are negative for system (14), then the synchronization manifold S is locally exponentially stable for the coupled system (3).*

When $\Gamma = I_n$, the transverse Lyapunov exponents can be calculated in the way proposed in Ref. 35. Combing the calculation of transverse Lyapunov exponents in Ref. 35 and Proposition 1, we have the following corollary:[8]

Corollary 1. *Suppose that $h_i, i = 1, 2, \ldots, n$ are the Lyapunov exponents of variational equation (13) and λ_k, $k = 2, 3, \ldots, N$ are all non-zero eigenvalues of the Laplacian matrix L for $\Gamma = I_n$. Then, the Lyapunov exponents of the variational equation (14), denoted as $\mu_i(\lambda_k), i = 1, 2, \ldots, n$, are given by*

$$\mu_i(\lambda_k) = h_i - \limsup_{t\to\infty} c(t)\lambda_k.$$

If there is $T > 0$ such that the largest Lyapunov exponent $\mu_{\max}(\lambda_k) = h_{\max} - c(t)\lambda_2 < 0$ for $t > T$, then, the synchronization manifold S is locally exponentially stable for the coupled system (3).

Based on the previous corollary, we can establish the following synchronization results:

Theorem 1. Suppose that $\lambda_c^{qmf} = 1/\Lambda_N > 0$ is the epidemic threshold of system (4). When $\Gamma = I_n$, if the effective spreading rate satisfies $\lambda > \lambda_c^{qmf}$, then system (5) can achieve synchronization.

Proof. If the effective spreading rate $\lambda > \lambda_c^{qmf}$, by Lemma 1, we know there exists $\rho^* \in (0, 1]$, such that $\lim_{t\to+\infty} \rho(t) = \rho^*$. Then, the rate of change of the coupling strength, $\dot{c}(t)$, admits $\dot{c}(t) = \alpha\rho(t)\sum_{j=1}^{N} e_j^T(t)e_j(t) \ge 0$. Assume to the contrary that system (5) cannot achieve synchronization, then $\lim_{t\to\infty} \dot{c}(t) \ne 0$, which implies $\lim_{t\to\infty} c(t) = +\infty$. Therefore, there exists $t^* > 0$ such that $\mu_{\max}(\lambda_k) = h_{\max} - c(t)\lambda_2 < 0$ when $t > t^*$. According to Corollary 1, the synchronization manifold S is locally exponentially stable for the coupled system (5), a contradiction. Hence, the synchronization can be achieved. ■

Theorem 2. *Consider the infinite state of coupling strength $c(t)$ in system (5) with $\Gamma = I_n$, for arbitrary $\lambda \in (0, 1)$, there is always a constant $c^* > 0$ such that $\lim_{t\to\infty} c(t) = c^*$.*

Proof. Suppose that $\lambda_c^{qmf} = 1/\Lambda_N > 0$ is the epidemic threshold of system (4), obviously, we have $\lambda_c^{qmf} \in (0, 1)$. If the effective spreading rate λ satisfies $\lambda_c^{qmf} < \lambda < 1$, by Theorem 1, we know system (5) can achieve synchronization, which implies that $\lim_{t\to\infty} e_i(t) = 0$ for $i = 1, 2, \ldots, N$. In addition, when $\lambda > \lambda_c^{qmf}$, it is easy to see that $0 < \alpha\rho(t) \le \alpha$. Then, by the expression, $\dot{c}(t) = \alpha\rho(t)\sum_{j=1}^{N} e_j^T(t)e_j(t)$, we obtain $\lim_{t\to\infty} \dot{c}(t) = 0$. Since $c(0) > 0, \dot{c}(t) \ge 0$, we know that there always exists a constant $c^* > 0$, such that

$\lim_{t\to\infty} c(t) = c^*$. If the effective spreading rate λ satisfies $0 < \lambda \le \lambda_c^{qmf}$, by Lemma 1, we know $\lim_{t\to\infty} \rho(t) = 0$ for system (5). Moreover, $\alpha \sum_{j=1}^{N} e_j^T(t) e_j(t)$ is bounded. Thus, we obtain $\lim_{t\to\infty} \dot{c}(t) = 0$. Combining the condition $c(0) > 0$ and $\dot{c}(t) \ge 0$, we know that there always exists a constant $c^* > 0$ such that $\lim_{t\to\infty} c(t) = c^*$. ■

Next, we study the global stability problem of epidemic synchronization model without a time delay.

B. Global stability of epidemic synchronization model without a time delay

The system for the error variables $e_i(t) = x_i(t) - s(t)$, $i = 1, 2, \ldots, N$ with respect to equation (3), i.e., the first equation of system (5) can be written as follows:

$$\dot{e}_i(t) = f(x_i(t)) - f(s(t)) - c(t)\sum_{j=1}^{N} l_{ij}\Gamma e_j(t) + g(t), \quad i = 1, 2, \ldots, N, \tag{15}$$

where $g(t) = f(s(t)) - \frac{1}{N}\sum_{i=1}^{N} f(x_i(t))$.

Let $F(t) = \left(f^T(x_1(t)) - f^T(s(t)), f^T(x_2(t)) - f^T(s(t)), \ldots, f^T(x_N(t)) - f^T(s(t))\right)^T$ and $G(t) = \left(g^T(t), g^T(t), \ldots, g^T(t)\right)^T$, then Eq. (15) can be rewritten by using the Kronecker product into a compact form

$$\dot{e}(t) = F(t) - c(t)(L \otimes \Gamma)e(t) + (I_N \otimes I_n)G(t). \tag{16}$$

By the definition of the error variables $e_i(t) = x_i(t) - s(t)$, we have $\sum_{i=1}^{N} e_i(t) = 0$. Therefore, the following relationship always holds:

$$e^T(t)(I_N \otimes P)G(t) = \sum_{i=1}^{N} e_i^T(t) P G(t) = \left[\sum_{i=1}^{N} e_i(t)\right]^T P G(t) = 0.$$

We first propose an assumption on the nonlinear function f as follows:

Assumption 1. There exists a positive definite diagonal matrix $P = diag(p_1, p_2, \ldots, p_n)$ and a constant ξ, such that the nonlinear vector-valued continuous function $f(x(t))$ satisfies

$$(x(t) - y(t))^T P[f(x(t)) - f(y(t))] \le \xi (x(t) - y(t))^T (x(t) - y(t)),$$

for all $x(t), y(t) \in R^n$ and $t \ge 0$.

Under the above assumption, we can establish the global stability result for the synchronization manifold S of system (5).

Theorem 3. *Suppose that $\lambda_c^{qmf} = 1/\Lambda_N > 0$ is the epidemic threshold of system (4). If the effective spreading rate satisfies $\lambda > \lambda_c^{qmf}$, then the synchronization manifold S of system (5) is globally asymptotically stable.*

Proof. See Appendix A. ■

Remark 1. *In fact, in addition to the epidemic threshold condition, we only need to find the constant ξ in assumption 1 to realize the global stability of the synchronization manifold of the epidemic synchronization model without a time delay. If the local dynamic function of human behaviors is known, it is convenient for us to apply Theorem 3 to analyze practical problems.*

Remark 2. *From the results obtained in Theorems 1 and 3, we show that the synchronization condition depends mainly on the relationship between the transmission rate and the epidemic threshold, which is independent of the specific epidemic process. If we consider simultaneously the effects of synchronization dynamics on epidemics, the synchronization condition in Theorems 1 and 3 is still valid even when the epidemic threshold changes.*

V. STABILITY ANALYSIS OF THE EPIDEMIC SYNCHRONIZATION MODEL WITH A COUPLING DELAY

To discuss epidemic synchronization system (7) with a coupling delay, the synchronization space should be revised accordingly as follows:

Definition 2. *The set $\tilde{S} = \{x = \left(x_1^T(\theta), \ldots, x_N^T(\theta)\right)^T : x_i(\theta) \in C_{\tau_1}, x_i(\theta) = x_j(\theta), i, j = 1, 2, \ldots, N, \theta \in [-\tau_1, 0]\}$ is called the synchronization space for system (7) with a coupling delay, where C_{τ_1} denotes the Banach space $C([-\tau_1, 0], R^n)$. The synchronization manifold of system (7) can be defined by $U = \{\left(\phi^T, \phi^T, \ldots, \phi^T\right)^T : \phi \in C_{\tau_1}\}$.*

A. Local stability of the epidemic synchronization model with a coupling delay

Similar to the analysis of system (5) in Subsection IV A, the variational equation of $y(t)$ corresponding to equation (6), i.e., the first equation of system (7) near the synchronization state $s(t)$ can be rewritten as follows:

$$\dot{y}(t) = [I_N \otimes Df(s(t)]y(t) - c(t)(\Lambda \otimes \Gamma)y(t - \tau_1).$$

To study local stability problems of the epidemic synchronization model with a coupling delay, we need the following lemma from.[37]

Lemma 2. *Consider the delayed dynamical system (6). If the zero solutions of the following $N-1$-dimensional linear time-varying delayed differential equations are asymptotically stable:*

$$\dot{\varphi}(t) = Df(s(t))\varphi(t) - c(t)\lambda_k \Gamma \varphi(t - \tau_1), \quad k = 2, 3, \ldots, N, \tag{17}$$

then the synchronization manifold U of the coupled system (6) is locally stable.

Based on Lemma 2, we can establish the following theorem:

Theorem 4. *Denote the Jacobian matrix $D(t) =: Df(s(t))$. If there exists a diagonal matrix $P = diag(p_1, p_2, \ldots, p_n)$ with positive diagonal elements, and a constant $\delta > 0$, such that*

$$PD(t) + D^T(t)P + P\Gamma \le -\delta I_n,$$

and

$$c(t) < \frac{1}{\lambda_N}\sqrt{\frac{\delta}{\lambda_{\max}(P\Gamma)}},$$

for all $t>0$, where I_n is an $n\times n$ identity matrix, then the synchronization manifold U of system (6) is locally stable.

Proof. See Appendix B. ■

Furthermore, we have the following result:

Theorem 5. *Suppose that $\lambda_c^{qmf} = 1/\Lambda_N > 0$ is the epidemic threshold of system (4). If the effective spreading rate satisfies $\lambda > \lambda_c^{qmf}$ and there exist $\delta > 0$ and a positive definite diagonal matrix P such that $PD(t) + D^T(t)P + P\Gamma \le -\delta I_n$ and $c(t) < \frac{1}{\lambda_N}\sqrt{\frac{\delta}{\lambda_{\max}(P\Gamma)}}$ for all $t>0$, then system (7) can achieve synchronization.*

Proof. If the effective spreading rate $\lambda > \lambda_c^{qmf}$ for system (7), there exists $\rho^* \in (0,1]$, such that $\lim_{t\to+\infty}\rho(t) = \rho^*$ according to Lemma 1. Then, the rate of change of the coupling strength, $\dot{c}(t)$, satisfies $\dot{c}(t) = \alpha\rho(t)\sum_{j=1}^{N} e_j^T(t)e_j(t) \ge 0$. Assume to the contrary that system (7) cannot achieve synchronization, then $\lim_{t\to\infty}\dot{c}(t) \neq 0$, which implies $\lim_{t\to\infty} c(t) = +\infty$. Hence, there exist positive constants μ and t^* such that $c(t^*) = \mu$, where $\mu = \frac{1}{\lambda_N}\sqrt{\frac{\delta}{\lambda_{\max}(P\Gamma)}}$. Since $c(t)$ is strictly increasing, we have $c(t) > c(t^*) = \frac{1}{\lambda_N}\sqrt{\frac{\delta}{\lambda_{\max}(P\Gamma)}}$, a contradiction to the condition that $c(t) < \frac{1}{\lambda_N}\sqrt{\frac{\delta}{\lambda_{\max}(P\Gamma)}}$ for all $t>0$. Hence, the synchronization can be achieved. ■

B. Global stability of the epidemic synchronization model with a coupling delay

The error system with respect to equation (6), i.e., the first equation of system (7) can be written as

$$\dot{e}_i(t) = f(x_i(t)) - f(s(t)) - c(t)\sum_{j=1}^{N} l_{ij}\Gamma e_j(t-\tau_1) + g(t),$$
$$i = 1,2,\ldots,N, \tag{18}$$

where $g(t) = f(s(t)) - \frac{1}{N}\sum_{i=1}^{N} f(x_i(t))$.

Rewrite (18) by using the Kronecker product into a compact form

$$\dot{e}(t) = F(t) - c(t)(L\otimes\Gamma)e(t-\tau_1) + (I_N\otimes I_n)G(t), \tag{19}$$

with $F(t) = \left(f^T(x_1(t)) - f^T(s(t)), f^T(x_2(t)) - f^T(s(t)), \ldots, f^T(x_N(t)) - f^T(s(t))\right)^T$ and $G(t) = \left(g^T(t), g^T(t), \ldots, g^T(t)\right)^T$. Then we can establish the following result:

Theorem 6. *Suppose that $\lambda_c^{qmf} = 1/\Lambda_N > 0$ is the epidemic threshold of system (4). If the effective spreading rate satisfies $\lambda > \lambda_c^{qmf}$, then for a certain range of $c(t)$, the synchronization manifold U of system (7) is globally asymptotically stable.*

Proof. See Appendix C. ■

Remark 3. *Actually, in addition to the epidemic threshold condition, in Theorem 4, we need to determine the positive constant δ and positive definite diagonal matrix P in Theorem 4 to realize the epidemic synchronization. However, in Theorem 6, we have determined the range of the constant ξ. If the local dynamic function of human behaviors is known, it is easy for us to prove their existence.*

VI. STABILITY ANALYSIS OF THE EPIDEMIC SYNCHRONIZATION MODEL WITH DOUBLE DELAYS

Let C be the Banach space of continuous functions on $[-\tau_2, 0]$ with uniform norm. We consider model (8) in the phase space $X = \prod_{i=1}^{N}(R\times C)$, and nonnegative initial condition $S_i(0) \in R_+, \rho_{i0} = \phi_i \in C, \phi_i(s) \ge 0$ for $-\tau_2 \le s \le 0$, $i = 1,2,\ldots,N$. It can be easily verified that solutions with the above initial condition remain nonnegative and the following set Γ remains invariant:

$$\Gamma = \{(S_1,\rho_1(\cdot),\ldots,S_N,\rho_N(\cdot)) \in X | 0 \le S_i \le 1,$$
$$0 \le S_i + \rho_i(0) \le 1, \rho_i(s) \ge 0, s\in[-\tau_2,0],$$
$$i = 1,2,\ldots,N\}.$$

Lemma 3. *Γ is positively invariant for model (8), and if there exists some j such that $\rho_j(0) > 0$, then when $t>0$, we always have $\rho_i(t) > 0$ for all $i = 1,2,\ldots,N$.*

Proof. We can get $S_i(t) + \rho_i(t) = S_i(0) + \rho_i(0) = 1$ by model (8). According to the first equation of model (8), we have

$$S_i(t) = S_i(0)e^{-\int_0^t\left[\lambda\sum_{j=1}^{N} a_{ij}\rho_j(\eta-\tau_2)+1\right]d\eta} + \int_0^t e^{-\int_\xi^t\left[\lambda\sum_{j=1}^{N} a_{ij}\rho_j(\eta-\tau_2)+1\right]\eta} d\xi,$$

which implies that when $t>0$, $S_i(t) > 0, i = 1,2,\ldots,N$.

When $0 < t \le \tau_2$, by the second equation of model (8), we get

$$\rho_i(t) = \rho_i(0)e^{-t} + \int_0^t \lambda S_i(\eta)\sum_{j=1}^{N} a_{ij}\rho_j(\eta-\tau_2)e^{-(t-\eta)}d\eta.$$

It shows that if there exists some j, $\rho_j(0) > 0, 1 \le j \le N$, then when $0 < t \le \tau_2$, we always have $\rho_i(t) > 0$.

We can extend this procedure to the intervals $[(r-1)\tau_2, r\tau_2]$ for all $r = 1,2,\ldots$. Therefore, $\rho_i(t) > 0$ for all t and any $i \in [1,N]$ if there exists j such that $\rho_j(0) > 0$. ■

The above results show that our model is biologically meaningful. Then, we will analyze the epidemic threshold of model (8) by the next generation matrix.[39]

Let

$$\Gamma^\circ = \{(S_1,\rho_1(\cdot),\ldots,S_N,\rho_N(\cdot)) \in X | 0 < S_i < 1,$$
$$0 < S_i + \rho_i(0) < 1, \rho_i(s) > 0, s\in[-\tau_2,0],$$
$$i = 1,2,\ldots,N\}.$$

It can be shown that Γ° is the interior of Γ. In Γ, model (8) has the disease-free equilibrium $E_0 = \{1,0,\ldots,1,0\}$, and possibly an endemic equilibrium $E^* = \{S_1^*,\rho_1^*;\cdots,S_N^*,\rho_N^*\}$ satisfying $S_i^*, \rho_i^* > 0$ with

$$\rho_i^* = \lambda S_i^*\sum_{j=1}^{N} a_{ij}\rho_j^*. \tag{20}$$

Linearizing at the disease-free equilibrium E_0 and considering the infectious compartments only, then the matrix of new infection is $F = \lambda A$, and the transfer matrix is $V = E$. Clearly, F is non-negative and V is a non-singular M-matrix. Let $\psi(-\tau_2)$ be the initial number of infected individuals, and $\psi(t)$ be the number of these initially infected individuals remaining after t time units. By the second equation of system (8), we know $\psi(t)$ satisfies $\dot{\psi}(t) = -V\psi(t)$, which has the unique solution $\psi(t) = e^{-V(t+\tau_2)}\psi(-\tau_2)$.

Since F is the infection rate matrix, $Fe^{-V(t+\tau_2)}\psi(-\tau_2)$ gives the new infection rate reproduced by remaining infected members at time t. Next, we consider the number of new infections, which contains two scenarios: (1) for $-\tau_2 \le t < 0$, there are no new infections; (2) for $t \ge 0$, the new infection rate at t is $F\psi(t-\tau_2) = Fe^{-Vt}\psi(-\tau_2)$. Therefore, the total number of new infections is given by

$$\begin{aligned}\int_0^\infty F\psi(t-\tau_2)dt &= \int_0^\infty Fe^{-Vt}\psi(-\tau_2)dt,\\ &= \int_{-\tau_2}^\infty Fe^{-V(t+\tau_2)}\psi(-\tau_2)dt,\\ &= FV^{-1}\psi(-\tau_2).\end{aligned}$$

The basic reproduction number R_0 of model (8) can be defined as $R_0 = \rho(FV^{-1}) = \lambda\rho(A) = \lambda\Lambda_N$, the spectral radius of FV^{-1}.

Using the relation $R_0 = \lambda/\lambda_c$ between the epidemic threshold λ_c and the basic reproduction number R_0, we have $\lambda_c = \lambda/R_0 = 1/\Lambda_N = \lambda_c^{qmf}$. The following result is standard and its proof is omitted.

Proposition 2. *Assume that the matrix $A = (a_{ij})$ is irreducible. Then, the following results hold:*

(1) *If $R_0 \le 1$, i.e., $\lambda \le \lambda_c^{qmf}$, then E_0 is the unique equilibrium of model (8) and it is globally stable in Γ;*
(2) *If $R_0 > 1$, i.e., $\lambda > \lambda_c^{qmf}$, then E_0 is unstable and there exists a unique endemic equilibrium E^* for model (8).*

Based on Proposition 2, the main result of model (8) is given in the following:

Theorem 7. *Assume that the matrix $A = (a_{ij})$ is irreducible. If $R_0 > 1$, i.e., $\lambda > \lambda_c^{qmf}$, then there exists a unique endemic equilibrium E^* for model (8) which is globally asymptotically stable in Γ°.*

Proof. See Appendix D. ■

According to Theorem 7, the following theorems of the epidemic synchronization model with double delays hold. Since the proof is similar to that in Sec. V, we omit the proof here.

Theorem 8. *Suppose that $\lambda_c^{qmf} = 1/\Lambda_N > 0$ is the epidemic threshold of system (4). If the effective spreading rate satisfies $\lambda > \lambda_c^{qmf}$ ($R_0 > 1$) and there exist $\delta > 0$ and a positive definite diagonal matrix P such that $PD(t) + D^T(t)P + P\Gamma \le -\delta I_n$ and $c(t) < \frac{1}{\lambda_N}\sqrt{\frac{\delta}{\lambda_{\max}(P\Gamma)}}$ for all $t > 0$, then system (9) can achieve synchronization.*

We also have the global stability result for the synchronization manifold U of system (9):

Theorem 9. *Suppose that $\lambda_c^{qmf} = 1/\Lambda_N > 0$ is the epidemic threshold of system (4). If the effective spreading rate satisfies $\lambda > \lambda_c^{qmf}$ ($R_0 > 1$), then for a certain range of $c(t)$, the synchronization manifold U of system (9) is globally asymptotically stable.*

VII. DISCUSSIONS

Many real networks exhibit a heterogeneous degree distribution, scaling as a power-law (PL) $P(k) \sim k^{-\gamma}$.[2,43,44] The heterogeneous pattern of the network can have dramatic effects on the behavior of dynamical processes running on top of it.[45]

The first approach to the dynamics of the SIS model in networks[46] was based on the so-called heterogeneous mean-field (HMF) theory,[45,47] which neglects both dynamical and topological correlations. According to HMF theory, the epidemic threshold of the SIS model in uncorrelated networks takes the form $\lambda_c^{hmf} = \langle k\rangle/\langle k^2\rangle$,[46] where $\langle k^n\rangle = \sum_k k^n P(k)$ are n-th moment of the network's degree distribution $P(k)$.[2] While HMF theory represents an exact result in the case of annealed networks,[48,49] its validity for real (quenched) networks is limited. Recently, a more refined approach than HMF theory, incorporating the effects of the quenched topological structure of the network, while neglecting dynamical correlations, is given by the quenched mean-field theory (QMF).[27–29] Within this framework, the epidemic threshold for SIS is predicted to be $\lambda_c^{qmf} = 1/\Lambda_N$, where Λ_N is the largest eigenvalue of the adjacency matrix. Given the scaling of Λ_N with the maximum degree, $\Lambda_N \sim \max\{\sqrt{k_{\max}}, \langle k^2\rangle/\langle k\rangle\}$,[50] the threshold value becomes[51]

$$\lambda_c^{qmf} \simeq \begin{cases} \dfrac{1}{\sqrt{k_{\max}}}, & \gamma > 5/2,\\ \dfrac{\langle k\rangle}{\langle k^2\rangle}, & 2 < \gamma < 5/2.\end{cases}$$

This threshold value implies that QMF theory predicts the same result as HMF theory for $2 < \gamma < 5/2$, while the threshold becomes larger, that is $\lambda_c^{qmf} < \lambda_c^{hmf}$ when $\gamma > 5/2$.

Here, we consider the SIS epidemic synchronization model based on the heterogeneous mean-field (HMF) theory,[8] and make a comparison with the SIS epidemic synchronization system based on the quenched mean-field (HMF) theory in the form of the following system:

$$\begin{cases} \dot{x}_i(t) = f(x_i(t)) - c(t)\sum_{j=1}^N l_{ij}\Gamma x_j(t),\\ \dot{\rho}_k(t) = -\rho_k(t) + \lambda k\left[1-\rho_k(t)\right]\Theta(t,k),\\ \dot{c}(t) = \alpha\rho(t)\sum_{j=1}^N e_j^T(t)e_j(t),\end{cases} \tag{21}$$

where $i = 1,2,\ldots,N$, $k = 1,2,\ldots,k_{\max}$, parameter $\alpha > 0$, $\Theta(t,k) = \frac{1}{\langle k\rangle}\sum_{k'} k'P(k')\rho_{k'}(t)$ and the infection prevalence is $\rho(t) = \sum_{k=1}^{k_{\max}}\rho_k(t)P(k)$. The initial condition of system (21) can be set as follows: the initial state $x_i(0) = (x_{i1}(0), x_{i2}(0),\ldots,x_{in}(0))^T \in R^n$, the initial coupling strength $c(0) > 0$, and the initial density $\rho_k(0) = \epsilon$ of infected nodes with degree k, where $0 < \epsilon \ll 1$.

Through a similar discussion to Sec. IV, we can obtain the condition $\lambda > \lambda_c^{hmf}$ such that system (21) can achieve

synchronization, which has been predicted through QMF theory for $2 < \gamma < 5/2$. However, system (21) cannot achieve synchronization if $\lambda_c^{qmf} < \lambda < \lambda_c^{hmf}$ when $\gamma > 5/2$, which is totally different from the result through QMF theory.

VIII. NUMERICAL SIMULATIONS

In order to illustrate the main results of the above theoretical analysis, in this section, we consider the scale-free networks with size $N = 100$ which are generated by the Barabási-Albert (BA) preferential attachment algorithm.[52] Starting with $m_0 = 4$ fully connected nodes, at each time step a new node is added and connected to $m = 3$ existing nodes in the network with the probability $\prod_i = k_i / \sum_j k_j$, which is a linear preferential attachment strategy.

From the point of view of the physical propagation process, so far, it is very difficult for us to identify the behavior dynamics of individuals within a community. Without loss of generality, we assume that the local dynamics of the dynamical behavior network are identical and take the node's dynamics to be the three-dimensional classical Lorenz systems in the following simulations. Here, a single Lorenz oscillator, as the desired orbit, can be described from the system

$$\begin{cases} \dot{s}_1 = \sigma(s_2 - s_1), \\ \dot{s}_2 = r s_1 - s_2 - s_1 s_3, \\ \dot{s}_3 = s_1 s_2 - b s_3, \end{cases}$$

where $\sigma = 10$, $r = 28$, and $b = 8/3$.

In the following simulations, we choose the inner-coupling matrix $\Gamma = I_n$, the initial coupling strength $c(0) = 0.001$, $\alpha = 0.001$, and define the synchronization error as $E(t) = 1/(N-1)\sum_{i=2}^{N}[x_1(t) - x_i(t)]^2$.

From Fig. 1, we can see that the infection prevalence $\rho(t)$ reaches a peak, then rapidly drops and finally converges to zero for different effective spreading rates λ whenever $\lambda < \lambda_c^{qmf}$. This shows that the disease will not spread out if the effective spreading rate $\lambda < \lambda_c^{qmf}$. However, a larger effective spreading rate implies slower convergence speed of $\rho(t)$ towards zero. When $\lambda < \lambda_c^{qmf}$, the synchronization error $E(t)$ does not converge to zero, which means that the epidemic dynamics cannot successfully induce behavioral synchronization of individuals under small spreading rates. In addition, when $\lambda < \lambda_c^{qmf}$, the larger effective spreading rate λ, the greater steady state value that the coupling strength $c(t)$ reaches.

From Fig. 2, we can see that $\rho(t)$ converges to different positive numbers and $E(t)$ converges to zero for different effective spreading rates λ when $\lambda > \lambda_c^{qmf}$, which implies that the epidemic dynamics can successfully induce behavioral synchronization of individuals with large spreading rates. The larger spreading rate λ implies the faster convergence speed of $\rho(t)$ towards the positive prevalence value and the larger steady state value, ρ^*. Increasing the spreading rate λ from 0.12 to 0.20, we find that the epidemic dynamics not only induce behavioral synchronization of individuals, but also accelerate the speed of synchronization.

We notice from Fig. 3 that under the same generating network structure, parameters, and initial condition, QMF and HMF theoretical approaches have different epidemic thresholds λ_c^{qmf} and λ_c^{hmf}. When the effective spreading rate is set as $\lambda = 0.1105$ ($\lambda_c^{qmf} < \lambda < \lambda_c^{hmf}$), we observe that $\rho_q(t)$ experiences a valley and then gradually increases, but $\rho_h(t)$ converges to zero. The synchronization error $E_q(t)$ with respect to quenched mean-field theory converges to zero, while the synchronization error $E_h(t)$ with respect to heterogeneous mean-field theory converges to a positive number, which indicates that quenched mean-field theory is more accurate compared with the heterogeneous mean-field theory in the prediction of epidemic synchronization.

We can see from Fig. 4 that under the same generating network structure, parameters, and initial condition, the coupled delayed system (7) can achieve synchronization eventually for different coupling delays τ_1 if the effective spreading rate $\lambda > \lambda_c^{qmf}$. Furthermore, the synchronization speed of the coupled delayed system (7) is slower than system (5) without delay, and the coupling delay τ_1 can enhance the speed of synchronization for delayed system (7). We also find that

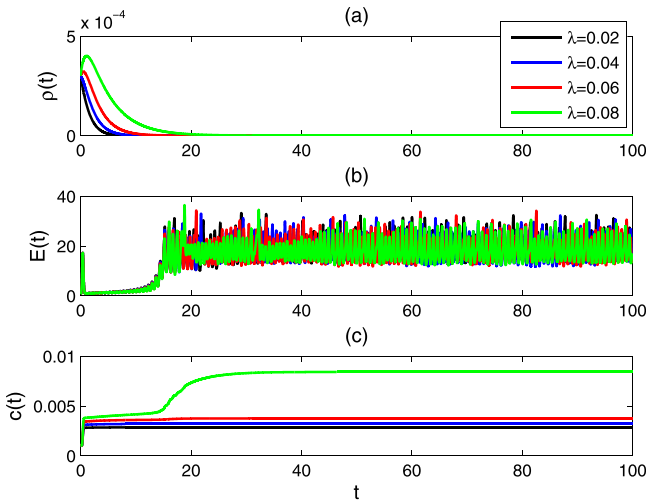

FIG. 1. When the effective spreading rate $\lambda < \lambda_c^{qmf}$, the influence of the effective spreading rate λ on the epidemic synchronization for model (5) with $\lambda_c^{qmf} = 0.1040$.

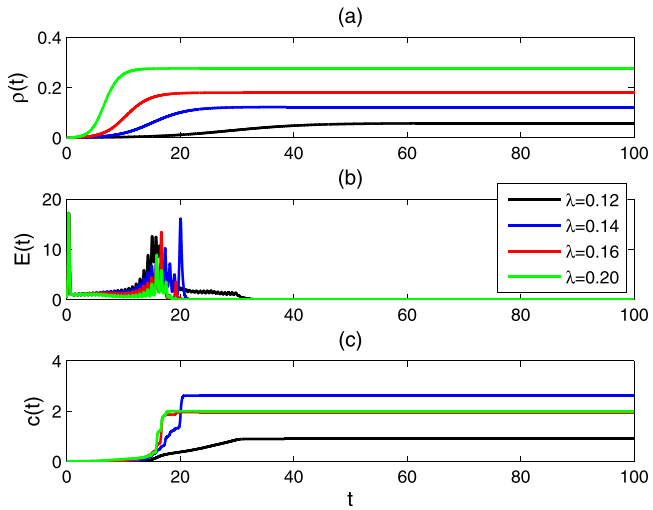

FIG. 2. When the effective spreading rate $\lambda > \lambda_c^{qmf}$, the influence of the effective spreading rate λ on the epidemic synchronization for model (5) with $\lambda_c^{qmf} = 0.1040$.

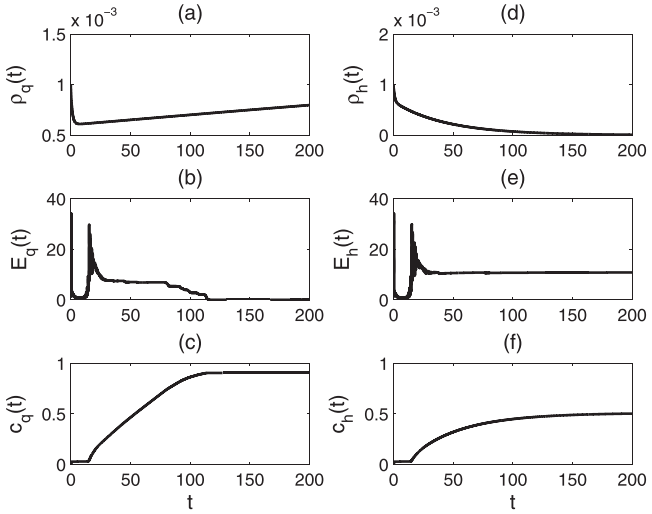

FIG. 3. The comparison of QMF and HMF theoretical approaches in the prediction of epidemic synchronization for models (5) and (21) under the same generating network structure, parameters, and initial condition. Here, $\lambda_c^{qmf} = 0.1102$, $\lambda_c^{hmf} = 0.1130$ and the effective spreading rate $\lambda = 0.1105$.

with the increase of the coupling delay τ_1 ($\tau_1 \neq 0$), the steady state value of the coupling strength $c(t)$ becomes smaller.

From Fig. 5, we can see that $\rho(t)$ converges to the same positive number for different epidemic delays τ_2 if the effective spreading rate $\lambda > \lambda_c^{qmf}$, and the convergence speed of the infection prevalence $\rho(t)$ becomes slower with a larger delay τ_2. The synchronization error $E(t)$ converges to zero for different epidemic delays τ_2 whenever $\lambda > \lambda_c^{qmf}$, which means that when $\lambda > \lambda_c^{qmf}$, the epidemic delay size does not affect synchronization. However, we can observe that the time taken to synchronization is different, with the increase of the epidemic delay τ_2, the time that system (9) ($\tau_1 = 0$, $\tau_2 \neq 0$) takes to arrive synchronization becomes longer, while the steady state value for the coupling strength $c(t)$ becomes smaller. It is obvious that the synchronization speed of system (9) ($\tau_1 = 0, \tau_2 \neq 0$) is slower than that of system (5) without a delay.

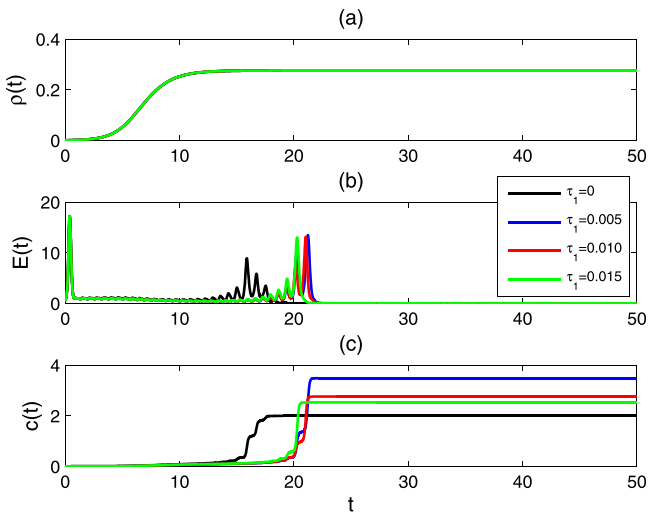

FIG. 4. The influence of the coupling delay on the epidemic synchronization for model (7). Here, $\lambda_c^{qmf} = 0.1040$ and the effective spreading rate $\lambda = 0.2$.

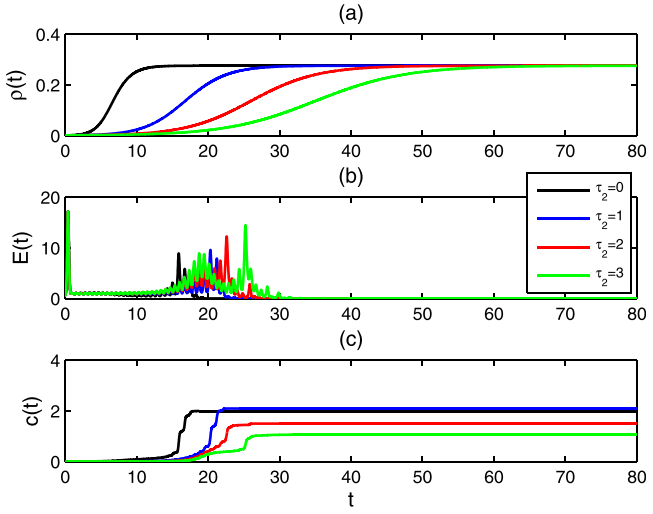

FIG. 5. The influence of the epidemic delay on the epidemic synchronization for model (9) with $\tau_1 = 0$. Here, $\lambda_c^{qmf} = 0.1040$ and the effective spreading rate $\lambda = 0.2$.

From Fig. 6, we can observe the apparent phenomenon that the synchronization speed of system (9) with double delays is slower than that of system (5) without delay if the effective spreading rate $\lambda > \lambda_c^{qmf}$. When $\lambda > \lambda_c^{qmf}$, if we fix the epidemic delay $\tau_2 = 1$, with the increase of the coupling delay τ_1, the infection prevalence $\rho(t)$ converges to the same positive number with the same convergence speed, and the synchronization error $E(t)$ converges to zero. Hence, system (9) can achieve synchronization. However, the coupling strength $c(t)$ reaches a higher steady state with larger τ_1, which is different from system (7) without epidemic delay. If we fix the coupling delay $\tau_1 = 0.010$, with different values of epidemic delay τ_2, $\rho(t)$ converges to the same positive value and the synchronization error $E(t)$ converges to zero. The steady state value that the coupling strength $c(t)$ reaches becomes smaller, and the convergence speed of $\rho(t)$ and the speed of synchronization become slower with larger τ_2, which is the same as system (9) with epidemic delay τ_2 ($\tau_1 = 0$, $\tau_2 \neq 0$).

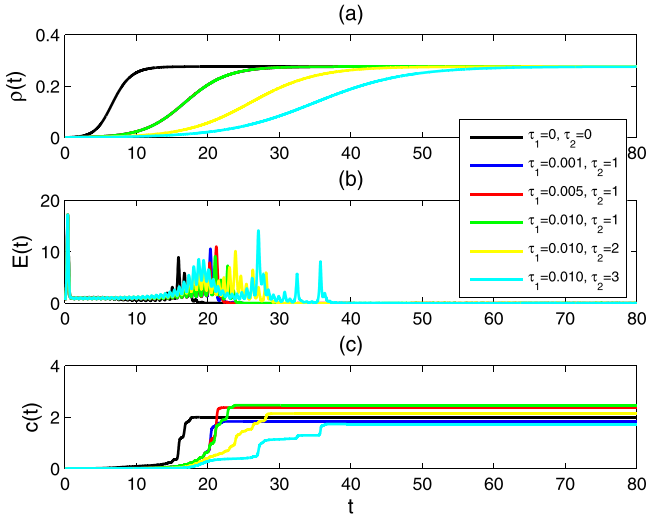

FIG. 6. The influence of double delays on the epidemic synchronization. Here, $\lambda_c^{qmf} = 0.1040$ and the effective spreading rate $\lambda = 0.2$.

IX. CONCLUSIONS

With special focus on behavioral synchronization of individuals induced by epidemic dynamics in real epidemic networks, this paper constructs several epidemic synchronization models for different types of infections. We investigate the relationship between the epidemic spreading rate and synchronization stability for model (5) without delay, and obtain an explicit condition $\lambda > \lambda_c^{qmf}$ for local and global synchronization with respect to the epidemic spreading rate, that is, if the epidemic breaks out more easily, then the epidemic dynamics can induce behavioral synchronization of individuals more effectively, which accords very well with the characteristics of real epidemic networks. For model (7) with a coupling delay and model (9) with double delays, we identify the conditions to ensure local and global synchronization, which are independent of time delays. These theoretical analyses can be applicable to networks with an arbitrary topological structure and size. The results illustrate the relationship between behavioral synchronization and model parameters, such as the effective spreading rate λ, the coupling strength $c(t)$, the coupling delay τ_1, and the epidemic delay τ_2. Then, we employ heterogeneous mean-field theory and quenched mean-field theory to the dynamics of the SIS model in networks, and illustrate that quenched mean-field (QMF) theory is much more accurate than heterogeneous mean-field (HMF) theory in predicting epidemic synchronization. Finally, some numerical simulations are performed to complement the above theoretical analysis. The simulations also reveal some complicated inherent characteristics for the speed of epidemic synchronization. When an epidemic starts to spread, if there is a delay in individual's communication, in order to facilitate individual's behaviors to achieve synchronization, individuals need to strengthen the frequency of communication about protective behaviors.

There are still some deficiencies in our models, and we will extend our future work in the following aspects: (i) Seeking a statistic to describe the speed of synchronization; (ii) Network modeling with different delays of nodes, and with the time-varying coupling delays, as well as their dynamics and control; (iii) Network modelling with both community structure and distinct individual behaviors, and the dynamics, and control of such models.

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APPENDIX A: PROOF OF THEOREM 3

We consider the following Lyapunov functional candidate:

$$V(t) = \frac{1}{2}e^T(t)(I_N \otimes P)e(t) + \frac{\beta}{2\alpha}[c^* - c(t)]^2,$$

where $\beta = \lambda_2\lambda_{\min}(P\Gamma) > 0$. Then, the derivative of $V(t)$ along the trajectory of system (16) admits

$$\begin{aligned}
\frac{dV(t)}{dt} &= e^T(t)(I_N \otimes P)F(t) - c(t)e^T(t)(L \otimes P\Gamma)e(t) \\
&\quad + e^T(t)(I_N \otimes P)G(t) - \frac{\beta}{\alpha}\dot{c}(t)[c^* - c(t)], \\
&= \sum_{i=1}^{N} e_i^T(t)P[f(x_i(t)) - f(s(t))] \\
&\quad - c(t)e^T(t)(L \otimes P\Gamma)e(t) + \beta c(t)\rho(t) \\
&\quad \times \sum_{i=1}^{N} e_i^T(t)e_i(t) - \beta c^*\rho(t)\sum_{i=1}^{N} e_i^T(t)e_i(t) \\
&\le \xi\sum_{i=1}^{N} e_i^T(t)e_i(t) - c(t)e^T(t)(L \otimes P\Gamma)e(t) \\
&\quad + \beta c(t)\sum_{i=1}^{N} e_i^T(t)e_i(t) - \beta c^*\rho(t)\sum_{i=1}^{N} e_i^T(t)e_i(t).
\end{aligned}$$

Introducing a transformation $y(t) = \left(y_1^T(t), y_2^T(t), \ldots, y_N^T(t)\right)^T = (U^T \otimes I_n)e(t)$ in Subsection IV A, then we have

$$\sum_{i=1}^{N} e_i^T(t)e_i(t) = \sum_{i=1}^{N} y_i^T(t)y_i(t).$$

Thus, we can obtain the following inequality

$$\begin{aligned}
e^T(t)(L \otimes P\Gamma)e(t) &= y^T(t)(\Lambda \otimes P\Gamma)y(t), \\
&= \sum_{i=1}^{N} \lambda_i y_i^T(t)P\Gamma y_i(t) \\
&\ge \lambda_2\lambda_{\min}(P\Gamma)\sum_{i=1}^{N} y_i^T(t)y_i(t), \\
&= \lambda_2\lambda_{\min}(P\Gamma)\sum_{i=1}^{N} e_i^T(t)e_i(t). \qquad \text{(A1)}
\end{aligned}$$

By inequality (A1), we have

$$\begin{aligned}
\frac{dV(t)}{dt} &\le \xi\sum_{i=1}^{N} e_i^T(t)e_i(t) - c(t)\lambda_2\lambda_{\min}(P\Gamma)\sum_{i=1}^{N} e_i^T(t)e_i(t) \\
&\quad + \beta c(t)\sum_{i=1}^{N} e_i^T(t)e_i(t) - \beta c^*\rho(t)\sum_{i=1}^{N} e_i^T(t)e_i(t), \\
&= [\xi - \beta c^*\rho(t)]\sum_{i=1}^{N} e_i^T(t)e_i(t) \\
&\quad + c(t)\left[\beta - \lambda_2\lambda_{\min}(P\Gamma)\right]\sum_{i=1}^{N} e_i^T(t)e_i(t), \\
&= [\xi - \beta c^*\rho(t)]\sum_{i=1}^{N} e_i^T(t)e_i(t).
\end{aligned}$$

If the effective spreading rate $\lambda > \lambda_c^{qmf}$ for system (5), then there exists $\rho^* \in (0, 1]$, such that $\lim_{t\to+\infty}\rho(t) = \rho^*$, which implies that there exist $\varepsilon \in (0, \rho^*]$ and $t_0 > 0$, such that $\rho(t) > \rho^* - \varepsilon \ge 0$ for all $t > t_0$. Therefore when $t > t_0$, we have

$$\frac{dV(t)}{dt} \le [\xi - \beta c^*(\rho^* - \varepsilon)] \sum_{i=1}^{N} e_i^T(t) e_i(t).$$

Thus, we can select a sufficiently large constant c^* such that $\frac{dV(t)}{dt} \le 0$, which implies that $V(t)$ is non-increasing. Furthermore, we have the following inequalities:

$$\frac{\lambda_{\min}(P)}{2} \sum_{i=1}^{N} e_i^T(t) e_i(t) + \frac{\beta}{2\alpha}[c^* - c(t)]^2 \le V(t) \le \frac{\lambda_{\max}(P)}{2} \sum_{i=1}^{N} e_i^T(t) e_i(t) + \frac{\beta}{2\alpha}[c^* - c(t)]^2.$$

By the LaSalle-Yoshizawa theorem,[36] we conclude that the solutions of system (16) at $e(t) = 0$ and $c^* - c(t) = 0$ are globally uniformly stable, that is, the synchronisation manifold S of system (5) is globally asymptotically stable. ■

APPENDIX B: PROOF OF THEOREM 4

We choose the following Lyapunov-Krasovskii functional candidate

$$V_k(t) = \varphi_k^T(t) P \varphi_k(t) + \int_{t-\tau_1}^{t} \varphi_k^T(\theta) P \Gamma \varphi_k(\theta) d\theta. \tag{B1}$$

The derivative of $V_k(t)$ along the solution of system (17) satisfies

$$\begin{aligned}
\frac{dV_k(t)}{dt} &= \dot{\varphi}_k^T(t) P \varphi_k(t) + \varphi_k^T(t) P \dot{\varphi}_k(t) + \varphi_k^T(t) \\
&\quad \times P\Gamma\varphi_k(t) - \varphi_k^T(t-\tau_1) P\Gamma\varphi_k(t-\tau_1), \\
&= \left[D(t)\varphi_k(t) - c(t)\lambda_k \Gamma \varphi_k(t-\tau_1)\right]^T P \varphi_k(t) \\
&\quad + \varphi_k^T(t) P \left[D(t)\varphi_k(t) - c(t)\lambda_k \Gamma \varphi_k(t-\tau_1)\right] \\
&\quad + \varphi_k^T(t) P\Gamma\varphi_k(t) - \varphi_k^T(t-\tau_1) P\Gamma\varphi_k(t-\tau_1), \\
&= \varphi_k^T(t)\left[PD(t) + D^T(t)P + P\Gamma\right]\varphi_k(t) \\
&\quad - c(t)\lambda_k \varphi_k^T(t-\tau_1)\Gamma^T P \varphi_k(t) - c(t)\lambda_k \varphi_k(t)^T \\
&\quad \times P\Gamma\varphi_k(t-\tau_1) - \varphi_k^T(t-\tau_1) P\Gamma\varphi_k(t-\tau_1) \\
&\le -\delta \varphi_k^T(t) I_n \varphi_k(t) - c(t)\lambda_k \varphi_k^T(t-\tau_1)\Gamma^T \\
&\quad \times P\varphi_k(t) - c(t)\lambda_k \varphi_k(t)^T P\Gamma\varphi_k(t-\tau_1) \\
&\quad - \varphi_k^T(t-\tau_1) P\Gamma\varphi_k(t-\tau_1), \\
&= \begin{bmatrix} \varphi^T(t) & \varphi^T(t-\tau_1) \end{bmatrix} \begin{bmatrix} B_{11} & B_{12} \\ B_{12}^T & B_{22} \end{bmatrix} \begin{bmatrix} \varphi(t) \\ \varphi(t-\tau_1) \end{bmatrix},
\end{aligned}$$

where $B_{11} = -\delta I_n, B_{12} = -\lambda_k c(t) P\Gamma$ and $B_{22} = -P\Gamma$.

From Schur complements theorem,[38] we know $B < 0$ if and only if $B_{11} - B_{12}B_{22}^{-1}B_{12}^T < 0$. Clearly, the condition can be further rewritten as

$$B_{11} < B_{12}B_{22}^{-1}B_{12}^T = -\lambda_k^2 c^2(t) P\Gamma.$$

By the specific expressions of B_{11}, B_{12}, and B_{22}, we have

$$-\lambda_N^2 \lambda_{\max}(P\Gamma) c^2(t) I_n \le B_{12}B_{22}^{-1}B_{12}^T \le -\lambda_2^2 \lambda_{\min}(P\Gamma) c^2(t) I_n.$$

Since $c(t) < \frac{1}{\lambda_N}\sqrt{\frac{\delta}{\lambda_{\max}(P\Gamma)}}$ for all $t > 0$, then we have $B < 0$, namely, $\frac{dV_k(t)}{dt} \le 0$. Therefore, the synchronization manifold U is locally asymptotically stable. ■

APPENDIX C: PROOF OF THEOREM 6

We consider the following Lyapunov functional candidate

$$V(t) = e^T(t)(I_N \otimes P)e(t) + \int_{t-\tau_1}^{t} e^T(s)(I_N \otimes I_n)e(s)ds + \frac{1}{2\alpha}[c_0 - c(t)]^2.$$

The derivative of $V(t)$ along the trajectory of system (19) satisfies

$$\begin{aligned}
\frac{dV(t)}{dt} &= 2e^T(t)(I_N \otimes P)\left[F(t) - c(t)(L \otimes P\Gamma)e(t-\tau_1) \right. \\
&\quad \left. + (I_N \otimes I_n)G(t)\right] + e^T(t)(I_N \otimes I_n)e(t) \\
&\quad - e^T(t-\tau_1)(I_N \otimes I_n)e(t-\tau_1) - \frac{1}{\alpha}\dot{c}(t)[c_0 - c(t)] \\
&\le 2\xi \sum_{i=1}^{N} e_i^T(t) e_i(t) - 2c(t)e^T(t)(L \otimes P\Gamma)e(t-\tau_1) \\
&\quad + e^T(t)(I_N \otimes I_n)e(t) + c(t)e^T(t)(I_N \otimes I_n)e(t) \\
&\quad - c_0\rho(t)e^T(t)(I_N \otimes I_n)e(t) \\
&\quad - e^T(t-\tau_1)(I_N \otimes I_n)e(t-\tau_1).
\end{aligned}$$

If the effective spreading rate $\lambda > \lambda_c^{qmf}$ for system (7), then there exists $\rho^* \in (0, 1]$, such that $\lim_{t\to+\infty} \rho(t) = \rho^*$, which implies that there exist $\varepsilon \in (0, \rho^*]$ and $t_0 > 0$, such that $\rho(t) > \rho^* - \varepsilon \triangleq \tilde{\rho}^* \ge 0$ for all $t > t_0$. Hence when $t > t_0$, we have

$$\begin{aligned}
\frac{dV(t)}{dt} &\le 2\xi \sum_{i=1}^{N} e_i^T(t) e_i(t) - 2c(t)e^T(t)(L \otimes P\Gamma)e(t-\tau_1) \\
&\quad + e^T(t)(I_N \otimes I_n)e(t) + c(t)e^T(t)(I_N \otimes I_n)e(t) \\
&\quad - c_0\tilde{\rho}^* e^T(t)(I_N \otimes I_n)e(t) - e^T(t-\tau_1)(I_N \otimes I_n)e(t-\tau_1) \\
&= \begin{bmatrix} e^T(t) & e^T(t-\tau_1) \end{bmatrix} \begin{bmatrix} H_{11} & H_{12} \\ H_{12}^T & H_{22} \end{bmatrix} \begin{bmatrix} e(t) \\ e(t-\tau_1) \end{bmatrix},
\end{aligned}$$

where $H_{11} = \left[2\xi + 1 - c_0\tilde{\rho}^* + c(t)\right] I_{Nn}, H_{12} = -c(t)(L \otimes P\Gamma)$ and $H_{22} = -I_{Nn}$.

From Schur complements theorem,[38] we know $H < 0$ if and only if $H_{11} - H_{12}H_{22}^{-1}H_{12}^T < 0$. Clearly, this condition can be further written as

$$H_{11} < -H_{12}H_{12}^T = -c^2(t)(LL^T \otimes P\Gamma^2 P). \tag{C1}$$

By the specific expressions of H_{12}, the right-hand side of inequality (C1) satisfies

$$-\lambda_N^2 \max_i\{(p_i\gamma_i)^2\} c^2(t) I_{Nn} \le -H_{12}H_{12}^T \le 0.$$

In order to ensure $H < 0$, we have

$$2\xi + 1 - c_0\tilde{\rho}^* + c(t) < -\lambda_N^2 \max_i\{(p_i\gamma_i)^2\} c^2(t). \tag{C2}$$

By solving inequality (C2), we obtain the range of $c(t)$ as follows:

$$c(t) < \frac{-1+\sqrt{1-4\lambda_N^2 \max_i\{(p_i\gamma_i)^2\}\left[2\xi+1-c_0\tilde{\rho^*}\right]}}{2\lambda_N^2 \max_i\{(p_i\gamma_i)^2\}},$$

where $\xi < -\frac{1}{2}$ and positive constant c_0 satisfies

$$0 < c_0 \le \frac{\widehat{\rho^*}+\sqrt{\widehat{\rho^*}^2-4\lambda_N^2 \max_i\{(p_i\gamma_i)^2\}(2\xi+1)}}{2\lambda_N^2 \max_i\{(p_i\gamma_i)^2\}},$$

where $\widehat{\rho^*} = \rho^* - \varepsilon - 1$.

Thus, we have $\frac{dV(t)}{dt} \le 0$. By the LaSalle-Yoshizawa theorem,[36] we conclude that the solutions of system (19) at $e(t)=0$ and $c(t)=c_0$ are globally uniformly stable, that is, the synchronization manifold U of system (7) is globally asymptotically stable. ■

APPENDIX D: PROOF OF THEOREM 7

Let $E^* = \{S_1^*, \rho_1^*; \cdots, S_N^*, \rho_N^*\}$ with $S_i^*, \rho_i^* > 0$ for $1 \le i \le N$, and denote the unique endemic equilibrium of model (8). We construct the following Lyapunov functional:

$$\begin{aligned} V_i(t) &= S_i(t) - S_i^* - S_i^* \ln\frac{S_i(t)}{S_i^*} \\ &\quad + \rho_i(t) - \rho_i^* - \rho_i^* \ln\frac{\rho_i(t)}{\rho_i^*} \\ &\quad + \lambda\sum_{j=1}^{N} a_{ij} S_i^* \rho_j^* \int_{t-\tau_2}^{t} \left[\frac{\rho_j(s)}{\rho_j^*} - 1 - \ln\frac{\rho_j(s)}{\rho_j^*}\right] ds. \end{aligned}$$

Next, we show that $V_i(t)$ satisfies the assumptions of Theorem 3.1 in Ref. 40.

$$\begin{aligned} \frac{dV_i(t)}{dt} &= \left[1-\frac{S_i^*}{S_i(t)}\right]\frac{dS_i(t)}{dt} + \left[1-\frac{\rho_i^*}{\rho_i(t)}\right]\frac{d\rho_i(t)}{dt} \\ &\quad + \lambda\sum_{j=1}^{N} a_{ij} S_i^* \rho_j^* \left[\frac{\rho_j(t)}{\rho_j^*} - \frac{\rho_j(t-\tau_2)}{\rho_j^*} + \ln\frac{\rho_j(t-\tau_2)}{\rho_j(t)}\right], \\ &= \left[1-\frac{S_i^*}{S_i(t)}\right]\left[1 - S_i(t) - \lambda S_i(t)\sum_{j=1}^{N} a_{ij}\rho_j(t-\tau_2)\right] \\ &\quad + \left[1-\frac{\rho_i^*}{\rho_i(t)}\right]\left[-\rho_i(t) + \lambda S_i(t)\sum_{j=1}^{N} a_{ij}\rho_j(t-\tau_2)\right] \\ &\quad + \lambda\sum_{j=1}^{N} a_{ij} S_i^* \rho_j^* \left[\frac{\rho_j(t)}{\rho_j^*} - \frac{\rho_j(t-\tau_2)}{\rho_j^*} + \ln\frac{\rho_j(t-\tau_2)}{\rho_j(t)}\right]. \end{aligned}$$

Using the equilibrium equations (20) and

$$1 = S_i^* + \lambda S_i^* \sum_{j=1}^{N} a_{ij}\rho_j^*,$$

we can obtain

$$\begin{aligned} \frac{dV_i(t)}{dt} &= \left[1-\frac{S_i^*}{S_i(t)}\right]\left[S_i^* + \lambda S_i^*\sum_{j=1}^{N} a_{ij}\rho_j^* - S_i(t)\right. \\ &\quad \left. - \lambda S_i(t)\sum_{j=1}^{N} a_{ij}\rho_j(t-\tau_2)\right] + \left[1-\frac{\rho_i^*}{\rho_i(t)}\right] \\ &\quad \times \left[-\rho_i^* + \lambda S_i^*\sum_{j=1}^{N} a_{ij}\rho_j^* - \rho_i(t) + \lambda S_i(t)\sum_{j=1}^{N} a_{ij}\rho_j(t-\tau_2)\right] \\ &\quad + \lambda\sum_{j=1}^{N} a_{ij} S_i^* \rho_j^* \left[\frac{\rho_j(t)}{\rho_j^*} - \frac{\rho_j(t-\tau_2)}{\rho_j^*} + \ln\frac{\rho_j(t-\tau_2)}{\rho_j(t)}\right] \\ &= -\frac{1}{S_i(t)}\left[S_i(t)-S_i^*\right]^2 + \lambda\sum_{j=1}^{N} a_{ij} S_i^* \rho_j^* \left[2 - \frac{S_i^*}{S_i(t)} - \frac{\rho_i(t)}{\rho_i^*}\right. \\ &\quad \left. - \frac{S_i(t)\rho_j(t-\tau_2)\rho_i^*}{S_i^*\rho_j^*\rho_i(t)} + \frac{\rho_j(t)}{\rho_j^*} + \ln\frac{\rho_j(t-\tau_2)}{\rho_j(t)}\right]. \end{aligned}$$

Let $\tilde{a}_{ij} = \lambda a_{ij} S_i^* \rho_j^*, G_i(\rho_i) = -\frac{\rho_i}{\rho_i^*} + \ln\frac{\rho_i}{\rho_i^*}, \Phi(a) = 1 - a + \ln a$ and

$$\begin{aligned} F_{ij}\left(S_i, I_i, I_j(\cdot)\right) &= 2 - \frac{S_i^*}{S_i(t)} - \frac{\rho_i(t)}{\rho_i^*} - \frac{S_i(t)\rho_j(t-\tau_2)\rho_i^*}{S_i^*\rho_j^*\rho_i(t)} \\ &\quad + \frac{\rho_j(t)}{\rho_j^*} + \ln\frac{\rho_j(t-\tau_2)}{\rho_j(t)}. \end{aligned}$$

Then $\frac{dV_i(t)}{dt} \le \sum_{i,j=1}^{N} \tilde{a}_{ij} F_{ij}\left(S_i, I_i, I_j(\cdot)\right)$ with

$$\begin{aligned} F_{ij}\left(S_i, I_i, I_j(\cdot)\right) &= G_i(\rho_i) - G_j(\rho_j) + \Phi\left(\frac{S_i^*}{S_i}\right) \\ &\quad + \Phi\left(\frac{S_i\rho_j(t-\tau_2)\rho_i^*}{S_i^*\rho_j^*\rho_i}\right) \\ &\le G_i(\rho_i) - G_j(\rho_j). \end{aligned}$$

Therefore, $V_i, F_{ij}, G_i, \tilde{a}_{ij}$ satisfy the assumptions of Theorem 3.1 and Corollary 3.3 in Ref. 40, and the functional $V = \sum_{i=1}^{N} c_i V_i$ as defined in Theorem 3.1 in Ref. 40 is a Lyapunov functional for system (8), namely, $\frac{dV}{dt} \le 0$ for $\left(S_1, \rho_1(\cdot), \ldots, S_N, \rho_N(\cdot)\right) \in \Gamma^\circ$. It can be verified by a similar argument as in Sec. IV of Ref. 40 that the only compact invariant set where $\frac{dV}{dt} = 0$ is the singleton $\{E^*\}$. By the LaSalle's Invariance Principle for delayed systems (see Theorem 5.3.1 of Ref. 41 or Theorem 3.4.7 of Ref. 42) we can conclude that E^* is globally asymptotically stable in Γ° if $R_0 > 1$ ($\lambda > \lambda_c^{qmf}$). ■

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