Study on the Dynamic Behavior of a Class of Heterogeneous Network Systems With Time Delay

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ABSTRACT Successful treatment of COVID-19 that outbroke worldwide since the beginning of 2020 has demonstrated the importance of effective isolation, which is aimed at asymptomatic and symptomatic infected persons in the incubation period. In this paper, to further analyze the transmission dynamics behavior of epidemics with the latent state, we construct a class of health state - latent state - infection - recovery state (SEIR) infectious disease model with heterogeneity and time delay characteristic based on considering the nonlinear incidence rate formed by psychological inhibition factors. Also, the dynamics of the epidemic, the threshold condition, and stability are studied by creating Lyapunov functions reasonably, applying LaSalle’s Invariance Principle and mean-field equation theory. The research shows that, the basic reproduction number \( R_0 \) of the system depends on birth rate, death rate, recovery rate, disease transmission rate, and network topology. If \( R_0 < 1 \), the system is stable at the disease-free equilibrium point \( E_0 \), and if \( R_0 > 1 \), the system is sound at the endemic equilibrium point \( E^* \). Moreover, it is also proved that latent delay and psychological inhibitory factors can influence the peak and rate of the infected nodes in the system before their convergence to the equilibrium point, but not the system’s global stability. Meanwhile, the theoretical results are verified by numerical simulation finally.

INDEX TERMS Asymptotic stability, heterogeneity, Lyapunov functions, psychological inhibition, time delay.

I. INTRODUCTION

It is well known that infectious diseases caused by pathogens or viruses will pose a significant threat to human health and social security once they develop into epidemics. Therefore, it is of great theoretical value and practical significance to use mathematical models and numerical simulation techniques to study infectious disease’s dynamic process and stability characteristics.

Common transmission dynamics models are widely used in the study of Ebola, SARS, dengue fever, COVID-19, and other infectious diseases, and often introduced into other fields. In the area of computer [1]–[3], Mishra et al. use the SEIR model to study the propagation behavior of the worm virus on computer networks and observe that when the basic reproduction number is less than or equal to unity, the infected nodes tend to zero, and simultaneously the worm virus on the networks disappear [1]. To investigate how to limit the spread of network virus, Ren et al. introduce the installation of anti-virus software as an inhibition parameter into the SAIR model, and find that according to the different anti-virus capability of anti-virus software, the model will appear backward fork or Hopf bifurcation [2]. In the social field, Jia et al. use big data such as Twitter and Weibo to explore the public opinion communication discussing the stability and divarication of equilibrium point [4]–[6]. Zhu et al. further analyze the parameter sensitivity of public opinion communication and propose that the government effectively control public opinion communication by popularizing security information [6]. Some scholars focus on common problems in life, such as alcoholism, to analyze the causes of these social phenomena through the transmission dynamics model and find solutions [7]. Such above transmission dynamics models have also been introduced into the field of economy and finance; meanwhile, the risk contagion mechanism of banks [8]–[10] and the rule of capital flow...
in the stock market are discussed [11], [12]. Cao et al. use the SIR model to simulate the risk contagion process of banks by combining knowledge of risk contagion process, bank risk characteristics, and complex network structure. They find that network structure and effective infection rate would affect the number of immunized banks in the end. The immunization strategy of preferentially rescuing banks with a high degree could effectively prevent bank crisis contagion [9]. Zhou et al. use the propagation dynamics model to simulate the capital flow mechanism in the stock market and verify the existence of apparent herd behavior in the stock market through the classification modeling of all kinds of people with capital intentions. Further quantitative analysis is carried out [12]. It can be seen that the practicability and application range of the propagation dynamics models increase gradually, and at the same time, how to establish a more accurate transmission dynamics model and improve universality of the model need to be further considered.

Early studies on infectious disease dynamics are mainly based on the hypothesis of uniform individual mixing. All individuals in the system are uniformly mixed and have the same contact rate, which is inconsistent with the heterogeneous characteristics of disease transmission in real life. Especially after Barabási and ALbert proposing that the nodes distribution of many real networks (such as transportation network, cooperative paper network, and computer integrated network) conforms to power-law characteristics [13], many scholars have begun to study the propagation dynamics in such heterogeneous networks (scale-free networks). Xu et al. research the influence of community structure on disease transmission in scale-free networks and apply the comparison principle and Gregorin theorem to solve the threshold of the transmission dynamics model [14]. Because of the characteristics of drug resistance in some parts of the system, Wan further draws the inoculation population V into the SIR model and explores the system dynamics characteristics of the SVIRS model by using the theory of mean-field equation, it is revealed that the existence of the disease-free equilibrium point in the system depends on the basic reproduction number. The relationship between the basic reproduction number and the scale-free network topology is analyzed in detail [15]. Yang et al. [16] pull the power-law structure characteristics of scale-free networks into the average field equation for studying the effects of its topology structure on the propagation of network malware, and propose that adjusting the network structure to make the propagation threshold less than unity could effectively control the propagation of network malware. Wu et al. explore dynamic urban traffic flow behavior on scale-free networks and claim that the capacity of traffic flow would increase under scale-free network structure, which provides a new idea for solving traffic congestion problems [17]. Up to now, a large number of research results have confirmed that the heterogeneous characteristics of the basic network topology structure play a key role in the study of epidemic transmission and stability. Therefore, it is necessary to draw the characteristics of scale-free networks into the transmission dynamics models and quantify the impact of their structural factors on the transmission dynamics process.

With the gradual deepening of scholars’ knowledge about the transmission path of infectious disease model, it can be saw that the propagation dynamics models with single or multi-stage delay factors, such as the incubation period of infectious diseases [18]–[22], the infection period [23], [24], the recovery period [20], [25], are more realistic than conventional ordinary differential model of propagation. Xia adds infectious time delay and propagation vector into the basic SIR model to explore its influence on the dynamic process [23]. Jiang et al. introduce time delay into the infectious disease model and further discuss the global stability of the SIS model with time delay and the disease control strategy under the assumption of limited government resources [20]. Zhang et al. focus on the mutual influence of disease recovery time delay and short-term immunity on transmission dynamics, by applying Lyapunov functions and LaSalle’s Invariance Principle, the global stability of the epidemic model with recovery delay is proved. The Nyquist criterion is used to estimate the range of recovery time delay to maintain the global stability at the equilibrium point, which offers scholars a new way to further study the propagation dynamics model with time delay [25]. Based on previous work, Du pulls the latent delay and immune recovery period of diseases in the SIR model, and comprehensively considers the influence of mixed delay on the spread of computer virus. The result shows that, when considering the time delay factors, the peak value of the infected nodes and the asymptotic convergence rate will be affected before reaching the equilibrium [24].

It should be clear that in the process of epidemic infection, it first develops from susceptible nodes to exposed nodes. Then according to the individual’s autoimmune ability and physical fitness, after a certain incubation period, exposed nodes enter the infection group with a certain proportion [26]. And in medicine, this incubation delay is usually defined as the period from the pathogen invading the human body to showing obvious clinical symptoms. The longer it is, the more challenging to prevent and control the epidemic. There are many scholars concern about the incubation period of different diseases. Liu et al. are committed to the dynamic process of mumps, and introduce the Et which means the nodes in the incubation period into the model. It is concluded that the latent period of the general mumps was 18 days [27]. Bonyah et al. simulate the transmission mechanism of the Ebola virus and put forward that the latent period of the Ebola virus is 2 to 21 days; therefore, it is more realistic to draw Et into the model [28]. More general studies pay attention to diseases such as AIDS and dengue fever that have aroused broad concern worldwide. Zhang et al. carry out the transmission dynamics modeling of AIDS, which shows that the average incubation period of AIDS was 9-10 years, and further leads the age structure into the SEIR model to discuss the effects of age structure and threshold [29]. Similarly,
Enduri et al. elaborate that the latent period of dengue fever is 5-7 days. The effective transmission of dengue fever could be minimized through early detection and design of vector control strategy [30]. All above confirms that there is a big gap between the latent period of different infectious diseases; and a lack of effective targeted vaccine and treatment methods for dengue fever and AIDS. In a word, the earlier you find it, the better you control it, especially for AIDS, which is in an exposed state for a long time, the curative effect of different periods is very different. Besides, there are many cases that one asymptomatic infected person infecting more than one person in COVID-19 2020, and the National Health Commission has repeatedly mentioned that the focus of current prevention is to strictly control the infection of asymptomatic people, to avoid a new peak of the epidemic [31]. From these studies, it can be sure that paying attention to the changes of exposed-state population in the incubation period can significantly improve the model’s reality. By the way, it is helpful to control infectious diseases better.

This paper also focuses on the nonlinear incidence rate with psychological factors in infectious diseases, besides considering the heterogeneity, delay factors, and exposed nodes to establish a more accurate propagation dynamics model. It plays a vital role in ensuring that the model can reasonably and approximately describe the disease transmission dynamics. In the existing communication dynamics models, the incidence rate is usually set as bilinear function [32], [33] or exponential function [34], [35]. However, in practice, the direct isolation [36]–[38] or other indirect measures (for instance network education which can directly enhance the alertness of the public and protect themselves from disease) can usually reduce the number of effective contacts between infected and suspected people, even at a high infection level. For simulating the effects of such inhibition, basing the SIS infected and suspected people, even at a high infection level. can usually reduce the number of effective contacts between

epidemic model studied in this paper can be represented in FIGURE 1 below. The dynamic process can be described by formula (1).

Given the model (1), the parameters $b$, $\delta$, $\epsilon$, $\gamma$ are all positive. Where $k$ is the degree value of scale-free networks, the description of the epidemic transmission is considered with average number (density) of susceptible, exposed, infected and recovered individuals in nodes of connectivity $k(k = 1, 2, \cdots, n)$ at time $t$ in the scale-free network, here denoted by $S_k(t)$, $E_k(t)$, $I_k(t)$ and $R_k(t)$. $\lambda$ is the transmission rate, $\delta$ is the vaccination rate and $\alpha$ is the psychological inhibition factor. $\epsilon$ means the probability of changing from risk exposed nodes to infected nodes, $\gamma$ is the ratio of infected nodes to recovered nodes. The delay term $\tau(t - \tau)$ representing the average incubation period is a time quantity, during which the infectious agents develop in the vector. And at the end of the incubation period, the vector enters the contagious state and is transferable. Besides, the model (1) also assumes that all newborns are susceptible, and the crude birth rate is equal to the natural mortality rate, that is, both the birth rate and mortality rate are $b$. $\Theta(t - \tau)$ represents the average number (density) of a link from one infected node to another infected node, and the expression is

$$\Theta(t - \tau) = \sum_k p(j | k) I_j(t - \tau) \tag{2}$$

In (2), $p(j | k)$ is the conditional probability that a node with degree $k$ connected to another node with degree $j$, and its expansion formula is $p(j | k) = \frac{\rho(k)}{\sum_k k \rho(k)}$, where $(k) = \sum_k k \rho(k)$ is the average network degree.

### B. BOUNDNESS OF SOLUTIONS AND EPIDEMIC THRESHOLD

The initial condition of the system (1) has the following form

$$S_k(\theta) = \phi_k(\theta), \quad E_k(\theta) = \phi_{k2}(\theta), \quad I_k(\theta) = \phi_{k3}(\theta), \quad R_k(\theta) = \phi_{k4}(\theta)$$

$$\phi_{ki}(\theta) \geq 0, \quad \theta \in [-\tau, 0], \quad \phi_{ki}(0) > 0 \quad (i = 1, 2, 3, 4), \quad k = 1, \cdots, n \tag{3}$$

**Lemma 1:** Let $(S_1, E_1, I_1, R_1, \cdots, S_n, E_n, I_n, R_n)$ be the solution of system (1) with the initial condition (3), then for all $t > 0$, there are $0 < S_k(t), E_k(t), I_k(t), R_k(t) < 1$ and $0 < \Theta(t) < 1$.

**Proof:** For suspected nodes $S_k(t)$, there is $\lim_{t \to \infty} \sup S_k(t) = \frac{b}{\tau + \delta} = S_0^k$. So according to the first equation

$$\frac{dS_k(t)}{dt} = b - \frac{\lambda k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_k(t) - b S_k(t) - \delta S_k(t)$$

$$\frac{dE_k(t)}{dt} = \frac{\lambda k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_k(t) - b E_k(t) - \delta E_k(t)$$

$$\frac{dI_k(t)}{dt} = \epsilon E_k(t) - b I_k(t) - \gamma I_k(t)$$

$$\frac{dR_k(t)}{dt} = \gamma I_k(t) - b R_k(t) + \delta S_k(t)$$
of system (1), we have
\[
\frac{dS_k(t)}{dt} \leq b - (b + \delta)S_k(t)
\]  
(4)

There needs a classification. If \(b - (b + \delta)S_k(t) \neq 0\), then the original function of \(S_k(t)\) can be calculated as follows
\[
\int \frac{dS_k(t)}{b - (b + \delta)S_k(t)} \leq \int 1\,dt
\]
\[
\ln[b - (b + \delta)S_k(t)] \leq -(b + \delta) (t + c)
\]
\[
S_k(t) = \frac{b}{b + \delta} + \frac{b}{b + \delta}, \quad c > 0
\]  
(5)

Obviously, the first part of the above equation (5) is negative, and then the following inequality can be obtained
\[
\lim_{t \to \infty} S_k(t) \leq \frac{b}{b + \delta} + \psi, \quad \psi > 0,
\]  
(6)

If \(b - (b + \delta)S_k(t) = 0\), then we obtain \(S_k(t) = \frac{b}{b + \delta}\).

Taken together, \(S_k(t) \leq \frac{b}{b + \delta}\). So the suspected nodes \(S_k(t)\) are bounded. Adding to the first two equation of the system (1) to get the following expression
\[
\frac{d[S_k(t) + E_k(t)]}{dt} = b - b[S_k(t) + E_k(t)] - \delta S_k(t) - \varepsilon E_k(t)
\]
\[
= b - (b + \delta)[S_k(t) + E_k(t)] + (\delta - \varepsilon)E_k(t)
\]  
(7)

which is equivalent to equation (8)
\[
\frac{d[S_k(t) + E_k(t)]}{dt} = b - (b + \varepsilon)[S_k(t) + E_k(t)] + (\varepsilon - \delta)S_k(t)
\]  
(8)

There needs a classification. If \(\delta \leq \varepsilon\), the first formula can be transformed into the following inequality
\[
\frac{d[S_k(t) + E_k(t)]}{dt} \leq b - (b + \delta)[S_k(t) + E_k(t)] < (b + \delta)[1 - S_k(t) + E_k(t)]
\]  
(9)

According to the inequality (9) to find the original function of \(S_k(t) + E_k(t), [S_k(t) + E_k(t)] \leq 1\) can be proved. The specific process is as follows
\[
\int \frac{d[S_k(t) + E_k(t)]}{(b + \delta)[1 - S_k(t) + E_k(t)]} < (b + \delta) \, dt
\]
\[
[1 - S_k(t) + E_k(t)] < -ce^{-(b+\delta)t} + 1, \quad c > 0
\]  
(10)

It can be clear that \([S_k(t) + E_k(t)] \leq 1\), and suspected nodes \(S_k(t)\) are bounded, so we can get the conclusion that \(E_k(t)\) are limited too.

If \(\delta > \varepsilon\), equation (8) can be reduced to \((b + \varepsilon)[S_k(t) + E_k(t)] \leq b\), then
\[
[S_k(t) + E_k(t)] \leq \frac{b + \varepsilon}{b + \varepsilon}E_k(t) \leq \frac{b}{b + \varepsilon}
\]  
(11)

To sum up, \(E_k(t)\) are vividly bounded. Similarly, totaling the first third equation of system (1)
\[
\frac{d[S_k(t) + E_k(t) + I_k(t)]}{dt} = b - b[S_k(t) + E_k(t) + I_k(t)] - \delta S_k(t) - \gamma I_k(t)
\]  
\[
< b - b[S_k(t) + E_k(t) + I_k(t)]
\]  
(12)

Then, we have
\[
[S_k(t) + E_k(t) + I_k(t)] < -ce^{-bt} + 1, \quad c > 0
\]  
(13)

Evidently, \(S_k(t) + E_k(t) + I_k(t) < 1\). It has been proved that \(S_k(t)\) and \(E_k(t)\) are bounded, so \(I_k(t)\) are framed too. Finally, based on \(0 \leq \lim inf N_k(t) \leq \lim sup N_k(t) \leq 1\) and three bounded state variables above proved, we can conclude that \(R_k(t)\) are bounded.

In summary, the four state variables \(S_k(t), E_k(t), I_k(t), R_k(t)\) are considered to be specified, and the proof is complete.

Next, the paper will find all feasible solutions to the system (1).

First of all, it is easy to know that the system (1) has a disease-free equilibrium point \(E^0 = \left(\frac{b}{b+\delta}, 0, 0, \frac{\delta}{b+\delta}, \ldots, \frac{b}{b+\delta}, 0, 0, \frac{\delta}{b+\delta}\right)\), for further solving the endemic equilibrium point \(E^* \left(S_1^*, E_1^*, I_1^*, R_1^*, \ldots, S_n^*, E_n^*, I_n^*, R_n^*\right)\), let the
derivative value at the equilibrium point be zero, namely

\[
\begin{align*}
\frac{dS_k(t)}{dt} &= b - \frac{\lambda k \Theta (t - \tau)}{1 + \alpha \Theta (t - \tau)} S_k(t) - b S_k(t) \\
- \delta S_k(t) &= 0 \\
\frac{dE_k(t)}{dt} &= \frac{\lambda k \Theta (t - \tau)}{1 + \alpha \Theta (t - \tau)} S_k(t) - b E_k(t) \\
- \epsilon E_k(t) &= 0 \\
\frac{dI_k(t)}{dt} &= \epsilon E_k(t) - b I_k(t) - \gamma I_k(t) = 0 \\
\frac{dR_k(t)}{dt} &= \gamma I_k(t) - b R_k(t) + \delta S_k(t) = 0
\end{align*}
\]

(14)

It is easy to know that $S_k(t)$, $E_k(t)$, $R_k(t)$ are linear function concerning $I_k(t)$, that is

\[
S_k(t) = \frac{(b + \epsilon)(b + \gamma)(1 + \alpha \Theta)}{\epsilon \lambda k \Theta (t - \tau)} I_k(t)
\]

\[
E_k(t) = \frac{(b + \gamma)}{\epsilon} I_k(t)
\]

\[
R_k(t) = 1 - S_k(t) - E_k(t) - I_k(t)
\]

(15)

Then, the expression of the endemic equilibrium point $E^*(S^*, E^*, I^*, R^*)$, can be obtained

\[
S_k^* = \frac{b(1 + \alpha \Theta^*)}{(b + \delta)(1 + \alpha \Theta^*) + \lambda k \Theta^*}
\]

\[
E_k^* = \frac{\lambda k \Theta^* + (b + \delta)(1 + \alpha \Theta^*)}{b \lambda k \Theta^*}
\]

\[
I_k^* = \frac{(\lambda k \Theta^* + (b + \delta)(1 + \alpha \Theta^*))}{b \lambda k \Theta^*}
\]

\[
R_k^* = 1 - S_k^* - E_k^* - I_k^*
\]

(16)

Drawing the $I_k^*(t)$ in equation (16) into the time function $\Theta(t)$ and expand it, we define such an equation as self-consistency equality

\[
\Theta = \frac{b e \lambda}{(b + \gamma)(b + \epsilon)} \sum_{j} \frac{j p(j)}{k \lambda k \Theta + (b + \delta)(1 + \alpha \Theta)} = f(\Theta)
\]

(17)

It is clear that $\Theta = 0$ is a solution of (17), and when $\Theta = 1$

\[
f(1) = \frac{b e \lambda}{(b + \gamma)(b + \epsilon)} + \lambda k \Theta + (b + \delta)(1 + \alpha \Theta) < 1
\]

(18)

Therefore, to ensure that self-consistency equality (17) on the time function $\Theta(t)$ has a unique non-zero solution in the interval from zero to unity, the relationship that needs to be satisfied is as follows

\[
\left. \frac{df(\Theta)}{d\Theta} \right|_{\Theta = 0} = \frac{b e \lambda}{(b + \gamma)(b + \epsilon)(b + \delta)} \frac{\{k^2\}}{k} > 1
\]

(19)

Hence, the basic reproduction number $R_0$ of system (1) is given by

\[
R_0 = \frac{b e \lambda}{(b + \gamma)(b + \epsilon)(b + \delta)} \frac{\{k^2\}}{k}
\]

where, $\{k^2\} = \sum_k k^2 p(k)$. From the formula (20), we can conclude that the basic reproduction number $R_0$ is not dependent on delay $\tau$, and if $R_0 < 1$, the disease will gradually die out, otherwise, the disease will spread on the scale-free networks.

III. STABILITY ANALYSIS

In this section, we will analyze the global stability of disease-free equilibrium point $E^0$ and endemic diseases equilibrium point $E^*$.

Lemma 2: All feasible solutions of the plan (1) belong to the following feasible region

\[
\Omega = \left\{ S_k(t), E_k(t), I_k(t), R_k(t) \in R_{1n}^k, k = 1, \cdot \cdot \cdot , n \right\}
\]

(21)

Proof: According to the four equations of the system (1), we can get

\[
\frac{dN_k(t)}{dt} = b - b [S_k(t) + E_k(t) + I_k(t) + R_k(t)]
\]

(22)

Due to $N_k(\theta) > 0, \theta \in (-\tau, 0)$, when time tends to infinity, $N_k(t)$ cannot be infinite. It follows that

\[
0 \leq \lim \inf N_k(t) \leq \lim \sup N_k(t) \leq 1
\]

(23)

All feasible solutions fall in the feasible region $\Omega$, and if $R_0 < 1$, system (1) exists a disease-free equilibrium point $E^0 = (S_1^0, 0, 0, R_1^0, \cdot \cdot \cdot, S_n^0, 0, 0, R_n^0)$, where $S_1^0 = S_2^0 = \cdot \cdot \cdot = S_n^0 = b$ and $R_1^0 = R_2^0 = \cdot \cdot \cdot = R_n^0 = \frac{\delta}{b + \epsilon}$, and the point is on the edge of the feasible area. Otherwise, system has a unique endemic disease equilibrium point $E^*(S_1^*, E_1^*, I_1^*, \cdot \cdot \cdot, S_n^*, E_n^*, I_n^*, R_n^*)$.

The stability analysis of the equilibrium points of the system (1) is completed on the basis of that all kinds of nodes are positive definite and bounded. Lemma 1 above has proved the boundedness of various nodes, and the next turn is positivity.

According to (3), the initial condition of the model is $S_k(0) > 0, E_k(0) > 0, I_k(0) > 0, R_k(0) > 0$, and in the following, proof by contradiction is used to certify that $S_k(t), E_k(t), I_k(t), R_k(t) > 0$ is constant for all $t > 0$. That is, suppose there is $t \in (0, \tau)$, so that $S_k(t) < 0$.

Combining with the first formula of system (1), we reach

\[
\frac{\dot{S}_j(t)}{1 + \alpha \Theta (t - \tau) - \lambda k \Theta (t - \tau)} + b S_j(t) + \delta S_j(t) - b = 0
\]

(24)

Obviously, $\dot{S}_j(t) + \lambda k S_j(t) + b S_j(t) + \delta S_j(t) > 0$. Proceed to the next step

\[
S_j(t) > S_j(0) e^{-(b + \delta + \lambda k) t} > 0, t \in (0, t_1), t \in kN
\]

(25)

It can be seen from (25) that the hypothesis is not true, and the susceptible nodes $S_k(t)$ are positive in the time domain. Similarly, for exposed nodes $E_k(t)$, the hypothesis exists that $E_k(t) < 0$ is satisfied for $t \in (0, \tau^*)$. 

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Be clear that $E_k(0) > 0$ and $E_k(t)$ is continuous, so there is a sufficiently small $\eta$, such that $E_k(t) > 0$ for $t \in (0, \eta)$. Moreover, there exists $j \in N_n$ and $t_2 \geq \eta > 0$ to make $E_j(t) = 0$ satisfied, so $E_j(t) > 0$ for all $t \in (0, t_2)$.

For the second equation of the system (1), we have

$$\dot{E}_j(t) - \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_j(t) + bE_j(t) + \varepsilon E_j(t) = 0 \quad (26)$$

Obviously, $\dot{E}_j(t) + bE_j(t) + \varepsilon E_j(t) > 0$, and proceed to the next step to calculate the original of $E_j(t)$

$$\dot{E}_j(t) - \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_j(t) + bE_j(t) + \varepsilon E_j(t) = 0 \quad (27)$$

Precisely, because of the equation (27), hypothesis is not correct and the exposed nodes $E_k(t)$ are positive in the time domain. And then, it is proved that the infected nodes $I_k(t)$ meet the positive qualitative requirement. Suppose there is $I_k(t) < 0$ for $t \in (0, t^*)$.

Note that $I_k(0) > 0$ and $I_k(t)$ is continuous, so there is a sufficiently small $\eta$, such that $I_k(t) > 0$ for $t \in (0, \eta)$. Meanwhile, there exists $j \in N_n$ and $t_3 \geq \eta > 0$ to make $I_j(t) = 0$ satisfied, so $I_j(t) > 0$ for all $t \in (0, t_3)$. Uniting with the third formula of the system (1)

$$\dot{I}_j(t) - \varepsilon E_k + (b + \gamma) I_j(t) = 0 \quad (28)$$

Explicitly, $\dot{I}_j(t) + (b + \gamma) I_j(t) > 0$, and then

$$I_j(t) \geq I_j(0) e^{-(b + \gamma) t} > 0, \quad t \in (0, t_3), j \in kN \quad (29)$$

\[ \frac{dV_{k1}(t)}{dt} = \frac{\varepsilon}{b + \varepsilon} \sum_{k} g(k) \left(1 - \frac{S_0^k}{S_k(t)}\right) \left(b - \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_k(t) - bS_k(t) - \delta S_k(t) - b + \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_0^k + bS_0^k + \delta S_0^k\right) + \frac{\varepsilon}{b + \varepsilon} \sum_{k} g(k) \left[\frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_k(t) - bE_k(t) - \varepsilon E_k(t)\right] + \sum_{k} g(k) \left[\varepsilon E_k(t) - bI_k(t) - \varepsilon I_k(t)\right] \]

\[ = \frac{\varepsilon}{b + \varepsilon} \sum_{k} g(k) \left(1 - \frac{S_0^k}{S_k(t)}\right) \left(- \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_k(t) + \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_0^k + \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_0^k - \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_k(t) + \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_0^k\right) + \sum_{k} g(k) \left[\varepsilon E_k(t) - bI_k(t) - \varepsilon I_k(t)\right] \]

\[ = \frac{\varepsilon}{b + \varepsilon} \sum_{k} g(k) \left(1 - \frac{S_0^k}{S_k(t)}\right) \left(- \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_k(t) + \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_0^k + \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_0^k - \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_k(t) + \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_0^k\right) + \sum_{k} g(k) \left[\varepsilon E_k(t) - bI_k(t) - \varepsilon I_k(t)\right] \]

\[ = \frac{\varepsilon}{b + \varepsilon} \sum_{k} g(k) \left(1 - \frac{S_0^k}{S_k(t)}\right) \left(- \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_k(t) + \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_0^k + \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_0^k - \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_k(t) + \frac{\lambda_k \Theta(t - \tau)}{1 + \alpha \Theta(t - \tau)} S_0^k\right) + \sum_{k} g(k) \left[\varepsilon E_k(t) - bI_k(t) - \varepsilon I_k(t)\right] \]
The assumption fails on the basis of (29), the infected nodes $I_k(t)$ are positive in the time domain. In the same way, for recovery nodes $R_k(t)$, we suppose that $R_k(t) < 0$ for $t \in (0, t^*)$.

Notice that $R_k(0) > 0$ and $R_k(t)$ is continuous, so there is a sufficiently small $\varepsilon$ such that $R_k(t) > 0$ for $t \in (0, t_4)$. At the same time, there exists $j \in N_\varepsilon$ and $t_4 \geq \eta > 0$ to hold $R_j(t) = 0$ satisfied, so $R_j(t) > 0$ for all $t \in (0, t_4)$. Integrating with the fourth formula of system (1)

$$\dot{R}_j(t) - \gamma_j I_j(t) - \delta S_j(t) + bR_j(t) = 0$$

Obviously, $\dot{R}_j(t) + bR_j(t) > 0$. Go a step further

$$R_j(t) \geq R_j(0) e^{-bt} > 0, t \in (0, t_4), j \in kN$$

Owing to Eq. (31), the positivity of recovery nodes $R_k(t)$ is proved. Let $t^* = \min(t_1, t_2, t_3, t_4)$, according to the continuity of the function, equation (32) is always true when $t \in (0, t^*)$. Namely

$$S_k(t) > 0, E_k(t) > 0, I_k(t) > 0, R_k(t) > 0; k \in kN$$

Above, the positivity analysis is accomplished, and then proves the stability of disease-free equilibrium point $E^0$ and the endemic equilibrium point $E^*$.

**Theorem 3:** Disease-free equilibrium point $E^0$ is global asymptotic stable if $R_0 = \frac{b \gamma I_0}{(b+\gamma)(b+\gamma+\beta)} < 1$, otherwise, it is unstable.

**Proof:** First of all, it is clear that $\lim_{t \to \infty} S_k(t) = S^0_k$ according to the analysis of boundedness in the previous section, and the Lyapunov function is constructed as follows, where $g(k) = \frac{k \rho(k)}{(b+\gamma)(b+\beta)}$, $k = 1, \ldots, n$

$$V_{k1}(t) = \frac{e}{b+\varepsilon} \sum_k g(k) \left(S_k(t) - S^0_k - S^0_k \ln \frac{S_k(t)}{S^0_k} \right) + \frac{e}{b+\varepsilon} \sum_k g(k)E_k(t) + \sum_k g(k)I_k(t)$$

$$V_{k2}(t) = \frac{e}{b+\varepsilon} \sum_k g(k) \int_{t-1}^t \Theta(x) dx$$

$$V_k(t) = V_{k1}(t) + V_{k2}(t)$$

Expanding out the derivative of the function $V_{k1}(t)$

$$\frac{dV_{k1}(t)}{dt} = \frac{dV_{k1}(t)}{dt} + \frac{dV_{k2}(t)}{dt}$$

$$\leq \frac{e}{b+\varepsilon} \sum_k g(k) \left(1 - \frac{S^0_k}{S_k(t)} \right) \left(b + \delta \right) \left(S^0_k - S_k(t) \right) + \frac{e}{b+\varepsilon} \sum_k g(k) \frac{\lambda S_k(t)}{1 + \alpha \Theta(t-\tau)} - \sum_k g(k) \left(b + \gamma \right) I_k(t)$$

The derivative of this function is divided into three parts. Since the value at the disease-free equilibrium point satisfies equation (14), substituting the first equation of (14) into the first part of the derivative, and the following equation can be obtained.

$$\frac{e}{b+\varepsilon} \sum_k g(k) \left(1 - \frac{S^0_k}{S_k(t)} \right) \left(b + \delta \right) \left(S^0_k - S_k(t) \right) + \frac{e}{b+\varepsilon} \sum_k g(k) \frac{\lambda S_k(t)}{1 + \alpha \Theta(t-\tau)} - \sum_k g(k) \left(b + \gamma \right) I_k(t)$$

Then, differentiating $V_{k2}(t)$, we have

$$\frac{dV_{k2}(t)}{dt} = \sum_k g(k) \frac{e}{b+\varepsilon} \lambda S^0_k \Theta(t-\tau)$$

A further relation is given as (40), as shown at the bottom of the page.

According to the comparison principle, there is

$$\frac{\lambda \Theta(t-\tau)}{1+\alpha \Theta(t-\tau)} S_k^0 \leq \lambda \Theta(t-\tau) S_k^0$$

Then the value of disease-free equilibrium point is put into Equation (42), as shown at the bottom of the next page. By calculating, we have

Evidently, $\frac{e}{b+\varepsilon} \sum_k g(k) \left(1 - \frac{S^0_k}{S_k(t)} \right) \left(b + \delta \right) \left(S^0_k - S_k(t) \right)$ is negative, and the second part of Eq. (42) is negative too when $R_0 < 1$. In conclusion, by LaSalle’s Invariance Principle[40]. The disease-free equilibrium point of the system (1) is globally asymptotically stable if $R_0 < 1$, correspondingly, this point is unstable. Theorem 3 is verified.

**Theorem 4:** According to Lemma 2, for any non-negative time delay $\tau$, there exists a unique endemic equilibrium point $E^*(S^*_1, E^*_1, I^*_1, R^*_1, \ldots, S^*_n, E^*_n, I^*_n, R^*_n)$ with global asymptotic stability when $R_0 = \frac{b \gamma I_0}{(b+\gamma)(b+\beta)} > 1$.

**Proof:** Considering the endemic disease equilibrium point of SEIR system (1) with initial value condition (3), the Lyapunov function is constructed as follows

$$V_{k1}(t) = S_k(t) - \int_{S_k^0}^{S_k(t)} \frac{E_k^*(s)}{E_k^*(s)} + E_k(t)$$

$$\int_{I_k^*(s)}^{I_k(t)} \frac{E_k^*(s)}{I_k^*(s)} + I_k(t) - \int_{I_k^*(s)}^{I_k(t)} \frac{E_k^*(s)}{I_k^*(s)}$$

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For this function, Wang J [41] mentioned that the endemic disease equilibrium point is the only internal equilibrium point in the feasible region, and which is simultaneously corresponding to the minimum value point of the function, which means the function has a lower bound.
Denote $V_{k2}(t) = \int_0^t \left( \frac{I_j(t-\theta)}{I_j^*} - 1 - \ln \frac{I_j(t-\theta)}{I_j^*} \right) d\theta$, $\theta \in [0, \tau], j, k \in N_n$, it is easy to see that the differential of $V_{k2}(t)$ is not negative, and the value of this function equals zero if and only if $I_j(t-\theta) = I_j^*$, where $\theta \in (0, \tau)$.

Consequently, the Lyapunov function below is defined

$$V_k(t) = V_{k1}(t) + (b + \varepsilon)E_k^*V_{k2}(t) \quad (44)$$

Then, differentiating $V_k(t)$ and every formula of the system (1) is substituted into

$$\frac{dV_k(t)}{dt} = \left(1 - \frac{S_k^*}{S_k(t)} \right) \frac{dS_k(t)}{dt} + \left(1 - \frac{E_k^*}{E_k(t)} \right) \frac{dE_k(t)}{dt} + \left(1 - \frac{I_k^*}{I_k(t)} \right) \frac{dI_k(t)}{dt} b + \varepsilon \frac{dE_k(t)}{dt} - b + \varepsilon$$

$$= \left(1 - \frac{S_k^*}{S_k(t)} \right) \left[ b - \frac{\lambda^* \Theta(t-\tau)}{1 + \alpha \Theta(t-\tau)} S_k(t) \right] - bE_k(t) - \varepsilon E_k(t)$$

$$+ \left(1 - \frac{E_k^*}{E_k(t)} \right) \left[ \frac{\lambda^* \Theta(t-\tau)}{1 + \alpha \Theta(t-\tau)} - \delta S_k(t) \right] - bE_k(t) - \varepsilon E_k(t)$$

$$+ \left(1 - \frac{I_k^*}{I_k(t)} \right) \left[ \gamma E_k(t) - bI_k(t) - \gamma I_k(t) \right] b + \varepsilon$$

$$= \left(1 - \frac{S_k^*}{S_k(t)} \right) \left[ b - \frac{\lambda^* \Theta(t-\tau)}{1 + \alpha \Theta(t-\tau)} - bS_k^* - \delta S_k^* \right]$$

$$- \varepsilon E_k(t)$$

Since the endemic disease equilibrium point $E^*$ satisfies equation (14), that is $b = \frac{\lambda^* \Theta(t-\tau)}{1 + \alpha \Theta(t-\tau)} S_k(t) - \delta S_k^*$, plugging the first formula into and further simplifying in (46), as shown at the bottom of the previous page.

Expanding and sorting out the derivative of $V_{k1}(t)$ about $E_k(t)$

$$\left(1 - \frac{E_k^*}{E_k(t)} \right) \left( \frac{\lambda^* \Theta(t-\tau)}{1 + \alpha \Theta(t-\tau)} S_k(t) - bE_k(t) - \varepsilon E_k(t) \right)$$

$$= \left(1 - \frac{E_k^*}{E_k(t)} \right) \left[ \frac{\lambda^* \Theta(t-\tau)}{1 + \alpha \Theta(t-\tau)} S_k(t) \right] - bE_k(t) - \varepsilon E_k(t)$$

$$+ \left(1 - \frac{E_k^*}{E_k(t)} \right) \left[ -bE_k(t) - \varepsilon E_k(t) \right]$$

Then, (48), as shown at the bottom of the previous page.

As shown above, the derivative of $V_{k1}(t)$ is divided into six parts, obviously, \[ \left(1 - \frac{S_k^*}{S_k(t)} \right) (b + \delta) \left[S_k^* - S_k(t) \right] \leq 0 \]

and then just focus on the rest.

From the foregoing, it can be known that the endemic disease equilibrium point $E^*$ satisfies the following relation

$$\left(1 - \frac{E_k^*}{E_k(t)} \right) \left[ \frac{\lambda^* \Theta(t-\tau)}{1 + \alpha \Theta(t-\tau)} S_k(t) \right] = bE_k(t) + \varepsilon E_k^*$$

Then convert the five small parts (2) to (6) in Eq.(48) on the basis of (49), we have (50), as shown at the bottom of the previous page.

Ulteriorly, the derivative of $V_{k1}(t)$ is rearranged (51), as shown at the bottom of the next page.

Go a step further, differentiating $(b + \varepsilon)E_k^* \frac{dV_{k2}(t)}{dt}$, the second part of $V_k(t)$

$$= (b + \varepsilon)E_k^* \frac{d}{d\theta} \int_0^\tau \left( \frac{I_j(t-\theta)}{I_j^*} - 1 - \ln \frac{I_j(t-\theta)}{I_j^*} \right) d\theta$$

$$= (b + \varepsilon)E_k^* \left[ \frac{I_j(t-\theta)}{I_j^*} + \frac{I_j(t-\theta)}{I_j^*} + \ln \frac{I_j(t-\theta)}{I_j^*} \right]$$

$$= \left(\frac{I_j(t-\theta)}{I_j^*} + \frac{I_j(t-\theta)}{I_j^*} + \ln \frac{I_j(t-\theta)}{I_j^*} \right)$$

Owing to the common factor $(b + \varepsilon)E_k^*$ between $(b + \varepsilon)E_k^* \left[ \frac{I_j(t-\theta)}{I_j^*} - \frac{I_j(t-\theta)}{I_j^*} - \ln \frac{I_j(t-\theta)}{I_j^*} \right]$ and $(b + \varepsilon)E_k^* \left[ \frac{I_j(t-\theta)}{I_j^*} + \frac{I_j(t-\theta)}{I_j^*} + \ln \frac{I_j(t-\theta)}{I_j^*} \right]$, which is always positive, the derivative of $V_k(t)$ is written as (53), as shown at the bottom of the next page,

$$- \left(\frac{S_k^*}{S_k(t)} \right) - 1 - \ln \frac{S_k^*}{S_k(t)} \right) - \left(\frac{I_j(t-\theta)}{I_j^*} - 1 - \ln \frac{I_j(t-\theta)}{I_j^*} \right)$$

all satisfy the Volterra-type function $f(x) = -(x - 1 - \ln x) \leq 0$, which means the four terms are all non-positive. Let $a_{jk} = \beta k S_k^* \sum_j \frac{R_0}{I_j^*} I_j^* G_k(I_k(t)) = \frac{b}{I_k^*} + \ln \frac{I_k(t)}{I_k^*}$, $j, k \in N_n$, readily to get that

$$V_k(S_k(t), E_k(t), I_k(t), R_k(t)) = F_k$$

$$= G_k(I_k(t)) - G_j(I_j(t)) - f \left(\frac{S_k^*}{S_k(t)} \right) - f \left(\frac{E_k^*}{E_k(t)} \right)$$

$$- f \left(\frac{E_k^*}{E_k(t)} \right) - f \left(\frac{I_j(t)}{I_j^*} \right) - \left(\ln \frac{I_j(t)}{I_j^*} \right)$$

$$\leq G_k(I_k(t)) - G_j(I_j(t)) - \left(\ln \frac{I_j(t)}{I_j^*} \right)$$

Combining with $\left(\ln \frac{I_k(t)}{I_k^*} \right) = 0$, and $V_k(S_k, E_k, I_k, R_k)$ satisfy the assumptions of Theorem 3.1 and Corollary 3.3 in reference [42], namely

$$V_k(S_k(t), E_k(t), I_k(t), R_k(t)) \leq 0 \quad (55)$$

So it can be certified that for all non-negative time delay $\tau$, the endemic equilibrium point $E^*$ of system (1) tends to global asymptotic stable if $R_0 = \frac{bc\lambda}{(b+\gamma)(b+\tau)(b+\tau)} [\frac{\kappa}{\kappa}] > 1$.

IV. NUMERICAL ANALYSIS

For SEIR system (1), the previous paper theoretically studied the system threshold, the stability of equilibrium, and the influence of time delay and psychological inhibitory factor on
and psychological inhibition factor. In this section, to further verify the correctness of the previous theoretical research, several numerical results will be carried out on the system (1), mainly focusing on presenting the variation trend of various nodes in the system dynamic process. In this section, the numerical analysis of the system (1) is based on the heterogeneous network system (i.e., the time domain, and analyzing the influence of time delay $\tau$ and psychological inhibition factor $\alpha$.

In this section, the numerical analysis of the system (1) is based on the heterogeneous network system (i.e., the

\[
\frac{dV_{k1}}{dt} (t) = \left(1 - \frac{S_k^*(t)}{S_k(t)}\right)(b + \delta) \left(S_k^*(t) - S_k(t)\right),
\]

\[
+ (b + \varepsilon) E_k^* \left[ \left(2 - \frac{S_k^*(t)}{E_k^*(t)}\right) - \frac{E_k^*(t)}{S_k^*(t)} \right] - \frac{E_k(t)}{E_k^*} + 1 + \left(1 - \frac{I_k^*(t)}{I_k(t)}\right) \left(\frac{E_k(t)}{E_k^*} - \frac{1}{I_k(t)}\right)
\]

\[
= \left(1 - \frac{S_k^*(t)}{S_k(t)}\right)(b + \delta) \left(S_k^*(t) - S_k(t)\right) + (b + \varepsilon) E_k^* \left[ 4 - \frac{E_k(t)}{S_k^*(t)} - \frac{I_k^*(t)}{E_k^*} - I_k(t) \right]
\]

\[
\frac{d}{dt} \left(\frac{V_k}{I_j}\right) \leq G_k(I_k(t)) - G_j(I_j(t)) - f \left(\frac{S_k^*(t)}{E_k^*(t)}\right) - f \left(\frac{E_k^*(t)}{S_k(t)}\right) - f \left(\frac{I_k(t - \theta)}{I_j^*}\right) - \left(\ln \frac{I_j(t)}{I_j^*}\right)
\]
FIGURE 3 compares the system’s equilibrium conditions under two limit thresholds (i.e., approaching unity or less than one). The variation trend of susceptible nodes $S_t$ and infected nodes $I_t$ over time is shown in FIGURE 3 (a) (b) respectively.

A. STABILITY OF EQUILIBRIUM POINT

In FIGURE 2, the basic reproduction number $R_0 = 0.6324 < 1$ is given with the parameters $b = 0.4, \delta = 0.02, \alpha = 0.1, \gamma = 0.3, \varepsilon = 0.2, \lambda = 0.1, \tau = 3$, and the disease-equilibrium point is global asymptotically stable in FIGURE 2(a). Correspondingly, the basic reproduction number $R_0 = 13.9280 > 1$ is given with the parameters $b = 0.04, \delta = 0.02, \alpha = 0.1, \gamma = 0.01, \varepsilon = 0.13, \lambda = 0.3, \alpha = 0.1, \tau = 3$, and the endemic disease equilibrium point tends to global asymptotically stable.

To further verify the rigor of the theoretical results, FIGURE 3 compares the system’s equilibrium conditions under two limit thresholds (i.e., approaching unity or approaching zero). The variation trend of susceptible nodes $S_t$ and infected nodes $I_t$ over time is shown in FIGURE 3 (a) (b) respectively.

In FIGURE 3, the basic reproduction number $R_0 = 0.0529 \ll 1$ is given with the parameters $b = 0.4, \delta = 0.02, \alpha = 0.1, \gamma = 0.01, \varepsilon = 0.5, \lambda = 0.009, \tau = 0.5$ and $R_0 = 0.9920 < 1$ when $b = 0.4, \delta = 0.02, \alpha = 0.1, \gamma = 0.11, \varepsilon = 0.5, \lambda = 0.21, \tau = 0.5$, and the disease equilibrium point $E^0$ of global asymptotic stability. At the same time, FIGURE 3 reveals that the smaller $R_0$ is, the faster the system (1) approaches disease-free equilibrium point; especially, infection disease cannot spread rapidly on scale-free networks when $R_0 \ll 1$.

Considering the evolution of the system (1) with fixed parameters $b = 0.04, \delta = 0.02, \alpha = 0.1, \gamma = 0.01, \varepsilon = 0.13, \tau = 0.5$, the basic reproduction number $R_0 = 4.1784 > 1$ and $R_0 = 13.9280 > 1$ can be obtained when $\lambda = 0.09$ and $\lambda = 0.3$ is solely given. Further evolution mechanism is shown in FIGURE 4.

Observing FIGURE 4, if $R_0 > 1$, with the gradual increase of $R_0$, the number of suspected nodes $S_t$ in the system will decrease, on the contrary, the number of infected nodes will increase and the level of disease infection will rise accordingly, which shows that controlling the size of the basic reproduction number $R_0$ is the most effective method to prevent the spread of the disease when the disease occurs.
B. PSYCHOLOGICAL INHIBITOR

The psychological inhibitory factor $\alpha$ is a key parameter that constitutes the non-linear incidence function in the system (1). It represents the intention of people in the scale-free network to actively take measures to isolate the source of infection due to some direct or indirect effects. In the same way, this paper will study the influence of psychological inhibitory factors, and pay attention to the trend of suspected nodes $S_t$ and infected nodes $I_t$ over time.

Now set the parameters $b = 0.1, \delta = 0.02, \gamma = 0.3, \epsilon = 0.2, \lambda = 0.1, \tau = 3$ and $\alpha = 1, 10, 20, 50, 100$ to discuss the evolution of susceptible nodes $S_t$ and infected nodes $I_t$ over time.

Through comparison, it can be found that when $R_0 < 1$, with the gradual increase of psychological inhibitory factors $\alpha$, the rate at which the system tends to the disease-free equilibrium point increases, and the peak value that the number of infected nodes $I_t$ can reach before it tends to disappear gradually decreases.

In FIGURE 6, the basic reproduction number $R_0 = 13.9280 > 1$ is given the parameters $b = 0.04, \delta = 0.02, \gamma = 0.01, \epsilon = 0.13, \lambda = 0.3, \tau = 0.5$, comparing the changes of suspected nodes $S_t$ and infected nodes $I_t$ in the system (1) when $\alpha = 1, 10, 20, 50, 100$.

Because of $R_0 = 13.9280 > 1$, there will be both suspected nodes $S_t$ and infected nodes $I_t$ in the system. It is worth noting that the number of susceptible nodes increased while the number of infected nodes decreased with the gradual increase of psychological inhibitory factors $\alpha$. This altogether indicates that psychological inhibitors can reduce the number of infected nodes in the system and inhibit the spread of infectious diseases.

C. DELAY TERM

The effect of delay term on the system (1) is analyzed in two cases, $R_0 < 1$ and $R_0 > 1$. The basic reproduction number is given with the parameters $b = 0.1, \delta = 0.02, \alpha = 0.1, \gamma = 0.3, \epsilon = 0.2, \lambda = 0.1$, go a step further to simulate the evolution of the suspected nodes $S_t$ and the infected nodes $I_t$ when $\tau = 0.5, 2.5, 5, 9, 11$ independently, such results are presented in FIGURE 7. And the figure reveals that with the progress of delay term $\tau$, the system's period to reach the disease-free equilibrium point is prolonged and the rate is
slowed down when \( R_0 < 1 \). Meanwhile, the value of \( \tau \) does not affect the final equilibrium point.

In the same way, the basic reproduction number \( R_0 = 13.9280 > 1 \) can be obtained with the fixed parameters \( b = 0.04, \delta = 0.02, \alpha = 0.1, \gamma = 0.01, \varepsilon = 0.13, \lambda = 0.3 \), take a set of variable delay terms \( \tau = 0.5, 2.5, 5, 9, 11 \) to contrast the track of suspected nodes \( S_t \) and infected nodes \( I_t \), shown in FIGURE 8.

FIGURE 8 illustrates that the asymptotic convergence rate of the system is affected by the time delay to a certain extent, and when it increases to 11, the endemic disease equilibrium point \( E^* \) changes, indicating that there is obviously a critical value of the time delay term \( \tau \), beyond which the value of the time delay will affect the endemic equilibrium \( E^* \).

V. CONCLUSION

In this paper, a nonlinear incidence SEIR propagation dynamic model with time delay based on a scale-free network is presented. Among them, psychological inhibitory factor reflects the strength of people’s intention to take active measures to isolate the source of infection due to some direct or indirect influences in scale-free networks. In addition, there is really a time delay \( \tau \) when the suspected nodes \( S_t \) are converted to infected nodes \( I_t \). Furthermore, the existence of time delay \( \tau \) makes the system’s stability more difficult. For example, Lyapunov functions with time delay must be constructed in order to study the stability of system equilibrium points. And in this paper, the SEIR model is taken as an example to provide a reference for analyzing the dynamic problems of a class of delay systems by using delay differential equations.

In the work of this paper, the influence of time delay and psychological inhibition factor on the propagation dynamics is emphasized, and on this basis, how the topology of the propagation network affects the propagation of the system (1) is further investigated. Firstly, the propagation threshold \( R_0 \) of SEIR model is explicitly calculated, where \( \langle k^2 \rangle / \langle k \rangle \) means the influence of the degree distribution of scale-free network nodes on the propagation process of the system (1). Then, the dynamic propagation characteristics of the system (1) are analyzed in theory, the global asymptotic stability of the disease-free equilibrium point and the endemic equilibrium point is proved by using Lyapunov function and mean-field theory. The results illustrate that: if \( R_0 < 1 \), the disease-free equilibrium point is global asymptotically stable, otherwise, the endemic equilibrium is globally asymptotically stable. A large number of numerical simulations provide supporting evidence for these conclusions. Finally, it is proposed that both psychological inhibition factor and time delay can influence the gradual convergence rate of the system. In particular, psychological inhibitory factor can affect the peak value of various nodes in the system before reaching the equilibrium point; and when \( R_0 > 1 \), there is a critical value of time delay, beyond which the time delay will exert influence on the endemic disease equilibrium point. In fact, systems with time delay still have an extensive range of research value. For instance, cyberneticians use Pontryagin’s maximum principle and Hamiltonian function to study the optimal control of systems with time delay, also give some important arguments. Therefore, we will continue to work on a class of propagation dynamic models with time delay and nonlinear incidence in the long run.

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