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Research article

Global stability for a respiratory disease model with distributed or discrete delay on complex network

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Abstract: Distributed delay is a core concept in time-delay systems and it has been incorporated into the networked respiratory disease model that elucidates the occurrence of respiratory diseases induced by air pollution. Next we studied a respiratory disease model with distributed delay and discrete delay. By analyzing the linearized system, we showed that if the disease-free equilibrium E_0 exists, exhibits global asymptotic stability without any constraint on the variable space. In addition, we proved the global stability of the endemic equilibrium E_* by constructing the Liapunov functional. Our findings contributed networked reaction-diffusion models with distributed delay and discrete delay to the existing body of knowledge. Our research found that distributed delay altered the transmission rhythm of respiratory diseases, which weakened local stability and disrupted global stability, which leads to disease recurrence. Discrete delay could disrupt the ”synchrony” of respiratory disease transmission, thereby inducing Hopf branches that lead to periodic disease outbreaks and undermined global stability, making it impossible to completely eradicate the disease.

Keywords: network; respiratory diseases; distributed delay; discrete delay; Lyapunov functional

1. Introduction

Currently, people face a serious air pollution problem, mainly characterized by elevated concentrations of suspended particles in the atmosphere [1]. The adverse effects of air pollution on human health are multifaceted, with most pollutants directly impacting the respiratory and cardiovascular systems [2]. The investigation of $PM_{2.5}$'s influence on respiratory diseases through mathematical modeling has received significant attention in contemporary research [3, 4]. The infectious disease dynamics model is the key to analyzing the spread and control of respiratory diseases. Assuming that the number of infected individuals reaches a relatively high level, the infection saturation effect will play a dominant role. This makes the spread of the disease no longer exhibit linear characteristics, ultimately leading to a non-linear

change in the incidence rate. Therefore, mathematical modeling of the nonlinear incidence of infectious diseases has become a key issue in exploring the spread and prevention of respiratory diseases [5, 6].

To our knowledge, in previous models of respiratory diseases, the important factor of uneven spatial distribution of species was ignored [7–9]. Turing bifurcation is one of the core problems of the reaction-diffusion system, which has been widely used in biology and neurophysiology. In recent years, reaction-diffusion epidemic models have focused on spatial heterogeneous diffusion and pattern dynamics, combining complex/multi-layer networks to optimize prevention and control strategies, and have been extended to spatio-temporal multi-chamber models. The network delay system focuses on the coupled analysis of mixed time delays (discrete + distributed), explores stability and bifurcation through methods such as Lyapunov functionals, and incorporates node heterogeneity and time-varying network characteristics [10–15]. The latest research strengthens empirical calibration and dynamic mechanisms, but still needs to improve the estimation of time-delay parameters and the analysis of coupled network propagation. In recent years, the application of big data has introduced a networked structure to reaction-diffusion systems, helping to improve the accuracy of accurate models. Specifically, the Gaussian diffusion direction of classical reaction-diffusion systems is isotropic, whereas the diffusion direction of data-driven diffusion systems is anisotropic. Recently, in the study of anisotropic diffusion, many scholars have introduced weighted network structures in classical reaction-diffusion systems [16]. Inspired by this, in order to characterize population movements between different regions, Shi et al. introduced the Laplacian graph equation to improve the classical respiratory disease model into a networked respiratory disease model with discrete delay [17].

According to our research observations, when the number of infected individuals reaches a certain scale, the speed of disease transmission may no longer follow the linear response pattern. This is due to the manifestation of the infection saturation effect, which makes the incidence rate show nonlinear characteristics. Therefore, establishing a mathematical model of infectious diseases that includes nonlinear incidence rates has become a key issue in systematically exploring the transmission mechanism of respiratory diseases and optimizing prevention and control plans [18, 19].

Most models only consider a single type of delay (such as only discrete delay or distributed delay only), without taking into account the "multipath delay characteristics" of network propagation (such as the scenario where fixed contact delay and random exposure delay coexist in social networks) [20–22]. In order to improve this situation, we tried a new modeling idea. Suppose that the time between the susceptible population inhaling $PM_{2.5}$ and becoming a patient is constant τ (lag days of onset). The susceptible population at time $t-\tau$ will become a patient at time t, and the nonnegative constant τ is the lag days. However, it may be more realistic to assume that τ is a distributed parameter and the force of infection must be substituted by $\beta_2 S(x,t)\int_{\tau=0}^{h} f(\tau) I(x,t-\tau)\,d\tau$, where $f(\tau)$ represents the fraction of the vector population in which the time required to become infectious is τ. In addition, $f(\tau)$ is assumed to be non-negative, square integrable in $R_{+0} = [0,\infty)$ and satisfies $\int_0^{+\infty} f(\tau)\,d\tau = 1$, $\int_0^{+\infty} \tau f(\tau)\,d\tau < +\infty$.

In the infectious disease model established in this paper, the human population is divided into several different regions, and each region is further subdivided into two subgroups: susceptible individuals and infected individuals. Specifically, there are two ways for the respiratory diseases we are considering to spread. One way is that susceptible people inhale $PM_{2.5}$ carrying pathogens of respiratory diseases, which directly leads to infection and illness. Assuming that the onset of the disease rules out human-to-human transmission, that is, the patient's illness was not caused by the infection of other cases. Another

approach is that patients with respiratory diseases act as sources of infection, spreading the virus in their bodies to susceptible individuals, causing them to become infected and exhibit clinical symptoms. Of course, in both cases, respiratory diseases can be treated and patients can return to a susceptible state. $S(x,t)$ and $I(x,t)$ denote the densities of susceptible and infected populations, respectively.

To visually present the grid respiratory disease model architecture with distributed time delay, the fractional-order infectious disease transmission framework with a network scale of N = 5 is now visualized. For details, please refer to Figure 1.

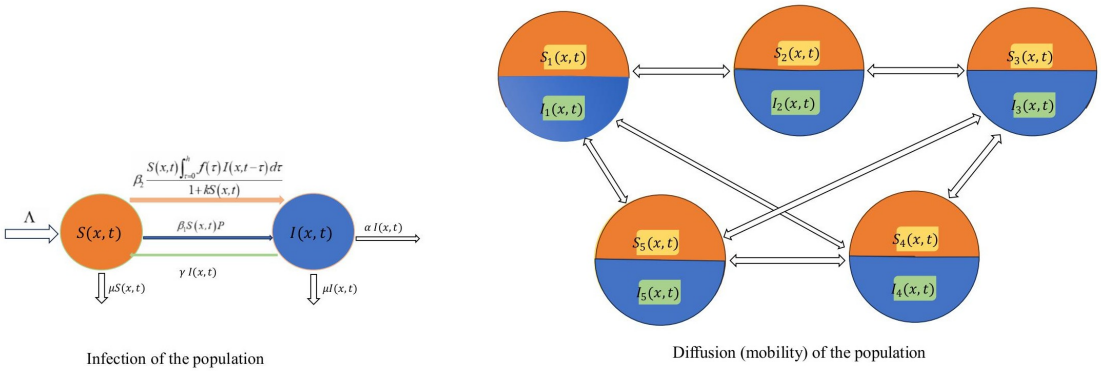

Figure 1. The left figure shows the disease transmission dynamics within node i, $i = 1, 2, 3, 4, 5$; in the right figure, each node contains two types of individuals - susceptible individuals ($S(x,t)$) and infected individuals ($I(x,t)$). The white arrows between the nodes represent the connection relationship of population diffusion (mobility).

By simultaneously considering the impact of distributed delays [5] and heterogeneous spatial distribution on the respiratory disease model, we formulate the following weighted networked differential equation model:

$$\begin{cases} \frac{\partial S}{\partial t} - d_1\Delta_\omega S(x,t) = \Lambda + \gamma I(x,t) - \beta_1 S(x,t) P - \beta_2 \frac{S(x,t)\int_{\tau=0}^{h} f(\tau) I(x,t-\tau)d\tau}{1+kS(x,t)} \\ -\mu S(x,t), \quad (x,t) \in V \times (0,\infty), \\ \frac{\partial I}{\partial t} - d_2\Delta_\omega I(x,t) = \beta_1 S(x,t) P + \beta_2 \frac{S(x,t)\int_{\tau=0}^{h} f(\tau) I(x,t-\tau)d\tau}{1+kS(x,t)} \\ -(\mu+\alpha+\gamma) I(x,t), \quad (x,t) \in V \times (0,\infty), \\ S(x,t) = S_0(x,t), I(x,t) = I_0(x,t), \quad (x,t) \in V \times [-\tau, 0], \end{cases} \tag{1.1}$$

where Λ is the recruitment rate, P denotes the air pollution index, β_1 is the conversion rate of susceptible individuals who directly got sick by inhaling air pollutants per unit of time, β_2 is the infection rate of susceptible individuals who are indirectly sick from infection by patients with respiratory diseases per unit of time, μ is the natural death rate, α is the disease-induced death rate, γ is the cure rate of infected. For a sufficiently large group of susceptible individuals, the nonlinear incidence rate $\frac{S(x,t)I(x,t-\tau)}{1+kS(x,t)}$ tends to a saturation level, where k is the saturation factor. Furthermore, d_1 and d_2 are the diffusion rates. Δ_ω is the Laplacian diffusion graph, which describes networked mobility [18, 19].

In addition, all model parameters are positive throughout the paper. Our main aim is to investigate the impact of delay on the dynamic process of transmission of respiratory diseases.

2. Previous results

Remark 1. *The region* $\Omega = \left\{(S, I) \in R^2_{\geq 0} : S + I \leq \frac{\Lambda}{\mu}\right\}$ *is positively invariant and attracts.*

Based on the traditional method for solving the positive invariant set of a system as stipulated in reference [3,4], Remark 1 above can be obtained. The specific proof process will not be elaborated here.

In order to study the spatial process of the spread of respiratory diseases, the asymptotic stability of the endemic equilibrium should be considered.

Remark 2. *Using the classic next-generation matrix method [23, 24], the basic reproduction number of system* (1.1) *is derived as* $R_0 = \frac{\mu\beta_2}{[\beta_1 P(\mu+\alpha)+\mu(\mu+\alpha+\gamma)]k}$.

Theorem 1. *1) It is easy to see that the model* (1.1) *has the disease-free equilibrium given by* $E_0 = \left(\frac{\Lambda}{\beta_1 P+\mu}, 0\right)$ *for all parameter values.*

2) If $R_0 > 1$*, then there is an unique endemic equilibrium given by* $E_* = (S_*, I_*)$*, where* $S_* = \frac{\Lambda-(\mu+\alpha)I_*}{\mu}, I_* = \frac{-B+\sqrt{B^2-4AC}}{2A}$*, and*

$$
\begin{aligned}
A &= (\mu+\alpha)\left[\mu\beta_2 - (\beta_1 P(\mu+\alpha) + \mu(\mu+\alpha+\gamma))k\right];\\
B &= -\Lambda\left[\mu\beta_2 - (\beta_1 P(\mu+\alpha) + \mu(\mu+\alpha+\gamma))k\right] + \mu^2(\mu+\alpha+\gamma) + \beta_1 P(\mu+\alpha)(\mu+k\Lambda);\\
C &= -\beta_1 P\Lambda(\mu+k\Lambda) < 0.
\end{aligned}
$$

The basic reproduction number $R_0 > 1$ indicates that "the average number of new infections caused by each infected person during the infectious period exceeds 1", which is the biological basis for the continuous spread of the disease. In reality, when respiratory diseases (such as influenza variants and the original strain of COVID-19) are highly contagious, the population's immunity is low (such as unvaccinated), and social contact is frequent, R_0 is very likely to exceed 1. At this point, the disease cannot be quickly contained, and there is a prerequisite for the formation of a sustained epidemic, which is completely consistent with the epidemiological definition of "endemic".

Then, the local asymptotic stability of the equilibria appeared.

3. Distributed delay

3.1. Local stability

Theorem 2. *The disease-free equilibrium* E_0 *is always locally asymptotically stable for all parameter values.*

Proof. First of all, the Jacobian matrix of system (1.1) at disease-free equilibrium E_0 can be derived as

$$
J_{E_0} = \begin{bmatrix} -\beta_1 P - \mu & \gamma - \frac{\beta_2\Lambda}{\beta_1 P+\mu+k\Lambda} \\ \beta_1 P & -(\mu+\alpha+\gamma) \end{bmatrix}.
$$

Then, the characteristic equation of system (1.1) without reaction diffusion terms at disease-free equilibrium E_0 is

$$
\left|\lambda E - J_{E_0}\right| = 0.
$$

Assuming λ_1 and λ_2 are the characteristic roots, then we have $\lambda_1\lambda_2 = det(J)$ and $\lambda_1 + \lambda_2 = tr(J)$, where

$$\begin{aligned} det(J) &= \beta_1 P(\mu+\alpha) + \mu(\mu+\alpha+\gamma) > 0, \\ tr(J) &= -[(\beta_1 P+\mu) + (\mu+\alpha+\gamma)] < 0. \end{aligned}$$

Then, the local asymptotic stability of the equilibria E_0 can be easily proved by using the eigenvalue method in reference [23, 24]. □

Theorem 3. *Whenever the endemic equilibrium E_* of the model* (1.1) *exists, it is locally asymptotically stable.*

Proof. The System (1.1) is centered on E_* by introducing $x_1 = S - S_*$, $x_2 = I - I_*$, and its linear part becomes

$$\begin{cases} \dot{x}_1(t) = -(\beta_1 P+\mu)x_1 + \gamma x_2 - \frac{\beta_2 S_*}{1+kS_*}\int_0^\tau f(\tau)x_2(t-\tau)d\tau + d_1\Delta_\omega x_1, \\ \dot{x}_2(t) = \beta_1 P x_1 - (\mu+\alpha+\gamma)x_2 + \frac{\beta_2 S_*}{1+kS_*}\int_0^\tau f(\tau)x_2(t-\tau)d\tau + d_2\Delta_\omega x_2. \end{cases} \tag{3.1}$$

Let us consider the Liapunov functional

$$V(x_t) = \sum_{x\in V}\frac{1}{2}x_2^2(t) + \sum_{x\in V}\frac{1}{2}\phi(x_1(t)+x_2(t))^2 + \sum_{x\in V}\frac{1}{2}\frac{\beta_2 S_*}{1+kS_*}\int_0^{+\infty} f(s)\int_{t-\tau}^{+\infty} x_2^2(s)\,ds\,d\tau, \tag{3.2}$$

where $\phi > 0$ is a constant. Let us observe that

$$V(x_t) \geq \sum_{x\in V}\omega_1(|x(t)|) = \sum_{x\in V}\frac{1}{2}x_2^2(t) + \sum_{x\in V}\frac{1}{2}\phi(x_1(t)+x_2(t))^2, \tag{3.3}$$

here ω_1 is a positive definite quadratic form of x_1 and x_2, since $\phi > 0$. Hence, $\omega_1 \geq 0$, $\omega_1 = 0$ if and only if $|x(t)| = 0$ and $\lim_{|x(t)|\to+\infty}\omega_1(|x(t)|) = +\infty$.

Furthermore, the time derivative of $V(x_t)$ along the solution of system (1.1) becomes

$$\begin{aligned} \dot{V}(x_t)|_{(2)} = &\sum_{x\in V}\left(-\phi\mu x_1^2 + (\gamma - \phi(\mu+\alpha))x_2^2 + (-(\beta_1 P+\mu) + \phi\mu - \phi(\mu+\alpha))x_1x_2\right) \\ &+ \phi d_1\Delta_\omega x_1^2 + \phi d_2\Delta_\omega x_2^2 + \phi d_1\Delta_\omega x_1x_2 + \phi d_2\Delta_\omega x_1x_2 \\ &+ \sum_{x\in V}\left(\frac{1}{2}\frac{\beta_2 S_*}{1+kS_*}x_2^2 + \frac{\beta_2 S_*}{1+kS_*}x_2\int_0^{+\infty} f(\tau)x_2(t-\tau)d\tau\right) \\ &- \sum_{x\in V}\left(\frac{1}{2}\frac{\beta_2 S_*}{1+kS_*}x_2\int_0^{+\infty} f(\tau)x_2^2(t-\tau)d\tau\right). \end{aligned}$$

According to Remark1 and the nature of the model, it is known

$$\begin{aligned} \sum_{x\in V}\phi d_1\Delta_\omega x_1^2 &\leq \phi d_1\frac{\Lambda}{\beta_1 P+\mu}\Delta_\omega x_1 \leq \phi d_1\frac{\Lambda}{\beta_1 P+\mu}\Delta_\omega\left(1-\frac{x_1^*}{x_1}\right), \\ \sum_{x\in V}\phi d_1\Delta_\omega x_1x_2 &\leq \phi d_1\frac{\Lambda}{\beta_1 P+\mu}\Delta_\omega x_2 \leq \phi d_1\frac{\Lambda}{\beta_1 P+\mu}\Delta_\omega\left(1-\frac{x_2^*}{x_2}\right). \end{aligned}$$

From the Lemma 2.1 in reference [6], one has

$$\begin{aligned}\sum_{x\in V}\left(1-\frac{x_1^*}{x_1}\right)\Delta_\omega x_1 &= -\sum_{x\in V}\frac{x_1^*}{x_1}\Delta_\omega x_1 = -\frac{1}{2}x_1^*\sum_{x\in V}\left(x_1(y)-x_1(z)\right)\\ &\quad\cdot\left(\frac{1}{x_1(y)}-\frac{1}{x_1(z)}\right)\omega(z,y)\\ &= -\frac{x_1^*}{2}\sum_{x\in V}\frac{\left(x_1(y)-x_1(z)\right)^2}{x_1(z)\,x_1(y)}\omega(z,y)\le 0.\end{aligned}$$

Summarizing the above process, it can be concluded

$$\begin{aligned}\dot{V}(x_t)\,|_{(2)} &\le \sum_{x\in V}\left(-\phi\mu x_1^2+(\gamma-\phi(\mu+\alpha))\,x_2^2+\frac{1}{2}\frac{\beta_2 S_*}{1+kS_*}x_2^2\right)\\ &\le \sum_{x\in V}\left(-\phi\mu\left(x_1^2+x_2^2\right)\right)\\ &= -\sum_{x\in V}\omega_2\left(|x(t)|\right).\end{aligned}$$

The above formula is obtained by choosing ϕ as $\omega_2(|x(t)|) = \phi\mu\left(x_1^2+x_2^2\right) = \phi\mu|x(t)|^2$ and $\Lambda = \beta_1 S_* P + \frac{\beta_2 S_* I_*}{1+kS_*} + \mu S_* - \gamma I_*, \beta_1 S_* P + \frac{\beta_2 S_* I_*}{1+kS_*} = (\mu+\alpha+\gamma)I_*$.

From the above theory and Theorem 1 in [25], the endemic equilibrium is locally asymptotically stable. □

The local stability of the equilibrium point in model (1.1) has been proven previously. Based on the uniform persistence of the disease-free equilibrium points in references [7–9], a bridge can be built from "local stability (convergence of small disturbances) to uniform persistence (even slightly larger disturbances can contract to a small range)", paving the way for the proof of global stability.

3.2. Global stability

Theorem 4. *The disease-free equilibrium E_0 of the model* (1.1) *is global asymptotically stable with respect to* Ω.

Proof. System (1.1) is centered on E_0 by introducing $\eta_1 = S - S_0$, $\eta_2 = I - I_0$, and its linear part becomes

$$\begin{cases}\dot{\eta}_1(t) = -(\beta_1 P+\mu)\eta_1+\gamma\eta_2-\frac{\beta_2 S_0}{1+kS_0}\int_0^\tau f(\tau)\,\eta_2(t-\tau)\,d\tau+d_1\Delta_\omega\eta_1,\\ \dot{\eta}_2(t) = \beta_1 P\eta_1-(\mu+\alpha+\gamma)\eta_2+\frac{\beta_2 S_0}{1+kS_0}\int_0^\tau f(\tau)\,\eta_2(t-\tau)\,d\tau+d_2\Delta_\omega\eta_2.\end{cases}\tag{3.4}$$

The disease-free equilibrium $E_0 = (\frac{\Lambda}{\beta_1 P+\mu}, 0)$ in $\Omega = \left\{(S,I)\in R^2_{\ge 0} : S+I\le\frac{\Lambda}{\mu}\right\}$ simply becomes $E_0 = (0,0)$ for (3.4).

Let us consider the Liapunov functional

$$V(\eta_t) = \sum_{x\in V}\eta_1(t)+\sum_{x\in V}\varphi\eta_2(t)+\sum_{x\in V}\beta_2\int_0^{+\infty}f(\tau)\int_{t-\tau}^{t}\eta_2(s)\,ds\,d\tau,\tag{3.5}$$

where $\varphi>0$ is a constant. Then $V(\eta_t)\ge\sum\limits_{x\in V} min(1,\varphi)(\eta_1(t)+\eta_2(t))$ for any $t\ge 0$.

Furthermore, the time derivative of $V(\eta_t)$ along the solution of system (1.1) becomes

$$\begin{aligned}
\dot{V}(\eta_t)|_{(5)} &= \sum_{x\in V}\left(-(\beta_1 P+\mu)\eta_1(t)+\gamma\eta_2(t)+d_1\Delta_\omega\eta_1+\varphi\beta_1 P\eta_1(t)-\varphi(\mu+\alpha+\gamma)\eta_2(t)\right)\\
&+\sum_{x\in V}\left(\varphi d_2\Delta_\omega\eta_2+\frac{\beta_2\eta_1(t)}{1+k\eta_1(t)}\int_0^{+\infty}f(\tau)\eta_2(t-\tau)d\tau-\beta_2\int_0^{+\infty}f(\tau)\eta_2(t-\tau)d\tau\right)\\
&\le \sum_{x\in V}\left(-(\beta_1 P+\mu)\eta_1(t)+\gamma\eta_2(t)+\varphi\beta_1 P\eta_1(t)-\varphi(\mu+\alpha+\gamma)\eta_2(t)\right)\\
&+\sum_{x\in V}d_1\Delta_\omega\left(1-\frac{\eta_1^*}{\eta_1}\right)\eta_1+\sum_{x\in V}\varphi d_2\Delta_\omega\left(1-\frac{\eta_2^*}{\eta_2}\right)\eta_2\\
&+\sum_{x\in V}\left(\frac{\beta_2\eta_1(t)}{1+k\eta_1(t)}\int_0^{+\infty}f(\tau)\eta_2(t-\tau)d\tau-\beta_2\int_0^{+\infty}f(\tau)\eta_2(t-\tau)d\tau\right).
\end{aligned}$$

In view of Lemma 2.1 in reference [6], we have

$$\begin{aligned}
\sum_{x\in V}\left(1-\frac{\eta_1^*}{\eta_1}\right)\Delta_\omega\eta_1 &= -\sum_{x\in V}\frac{\eta_1^*}{\eta_1}\Delta_\omega\eta_1=-\frac{1}{2}\eta_1^*\sum_{x\in V}(\eta_1(y)-\eta_1(x))\\
&\cdot\left(\frac{1}{\eta_1(y)}-\frac{1}{\eta_1(x)}\right)\omega(x,y)\\
&=-\frac{\eta_1^*}{2}\sum_{x\in V}\frac{(\eta_1(y)-\eta_1(x))^2}{\eta_1(x)\eta_1(y)}\omega(x,y)\le 0.
\end{aligned}$$

Therefore, it can be obtained

$$\begin{aligned}
\dot{V}(\eta_t)|_{(5)} &\le \sum_{x\in V}\left(-\beta_1 P\eta_1(t)+\gamma\eta_2(t)+\varphi\beta_1 P\eta_1(t)-\varphi(\mu+\alpha+\gamma)\eta_2(t)+\beta_2\eta_2(t)\right)\\
&=\sum_{x\in V}\left(-\beta_1 P(1-\varphi)\eta_1(t)-\left[\varphi(\mu+\alpha+\gamma)-\beta_2-\gamma\right]\eta_2(t)\right).
\end{aligned}$$

Here the last inequality is true because that $0\le\eta_1(t)\le\frac{\Lambda}{\mu}$. Choose $\varphi=\frac{\beta_2+\gamma}{2(\mu+\alpha+\gamma)}$ which is positive. Then, it can be obtained

$$\begin{aligned}
\dot{V}(\eta_t)|_{(5)} &\le \sum_{x\in V}\left(-\beta_1 P\eta_1(t)-\frac{1}{2}(\beta_2+\gamma)\eta_2(t)\right)\\
&\le \sum_{x\in V}\left(-\psi(\eta_1(t)+\eta_2(t))\right)\\
&=-\sum_{x\in V}\left(\psi|\eta(t)|_1\right)
\end{aligned}$$

for any $t\ge 0$, where $\psi=min\left\{\beta_1 P,\frac{1}{2}(\beta_2+\gamma)\right\}$.

From the above theory and Theorem 5 in [26], the equilibria E_0 is global asymptotically stable. □

This conclusion indicates that when $R_0<1$, even if there is a distribution delay, the disease can be completely eliminated across the entire network, and there will be no local rebound or long-term

prevalence. From the perspective of real-world scenarios: No matter how dispersed the transmission delay caused by population movement is, the disease will eventually disappear in all network nodes (such as communities, schools, and cities), achieving a "global disease-free" state. This provides theoretical support for strategies such as "dynamic zero-COVID" and "precise prevention and control".

Next, let's discuss the global asymptotic stability of the endemic equilibrium.

Theorem 5. *The endemic equilibrium* $E_* = (S_*, I_*)$ *of model* (1.1) *is globally asymptotically stable if* $R_0 > 1$, $min\,(\beta_1 P + \mu, \mu + \alpha + \gamma) > \frac{1}{2}$.

Proof. Evaluating both sides of (1.1) at E_* and recalling that $\int_{\tau=0}^{h} f(\tau)\,d\tau = 1$, gives

$$\Lambda = \beta_1 S_* P + \frac{\beta_2 S_* I_*}{1 + kS_*} + \mu S_* - \gamma I_*, \tag{3.6}$$

$$\beta_1 S_* P + \frac{\beta_2 S_* I_*}{1 + kS_*} = (\mu + \alpha + \gamma) I_*, \tag{3.7}$$

which will be used as substitutions in the following calculation. Let $g(y) = y - 1 - lny$,

$$U_S(t) = \sum_{x \in V} g\left(\frac{S(t)}{S_*}\right),$$

$$U_I(t) = \sum_{x \in V} g\left(\frac{I(t)}{I_*}\right),$$

$$U_+(t) = \sum_{x \in V} \int_{\tau=0}^{h} \alpha(\tau)\, g\left(\frac{I(t-\tau)}{I_*}\right) d\tau,$$

where $\alpha(\tau) = \int_{\sigma=\tau}^{h} f(\sigma)\,d\sigma$.

Then, the behavior of the Lyapunov functional will be studied as follow

$$U(t) = \frac{\beta_2 S_* I_*^2}{1 + kS_*} U_S + \frac{\beta_2 S_* I_*}{1 + kS_*} U_I + U_+. \tag{3.8}$$

It is found that $\alpha(\tau) > 0$ for each $\tau \in [0, h)$. In addition, $g : R_{>0} \longrightarrow R_{\geq 0}$ has the global minimum $g(1) = 0$. Thus, $U(t) \geq 0$ with equality if and only if $S(t) = I(t) = 1$ and $I(t-\tau) = 1$ for almost all $\tau \in [0, h)$.

By Remark 1 and Theorem 3.4 in [5], the solutions are bounded above and bounded away from zero for a time. Without loss of generality, it may be assumed that the solution in question satisfies these bounds for all $t \geq 0$. Thus, $U(t)$ is defined (and finite) for all $t \geq 0$.

The derivatives of U_S, U_I, and U_+ will be calculated separately and then combined to get the desired quantity $\frac{dU}{dt}$

$$\frac{dU_S}{dt} = \sum_{x \in V} \frac{1}{S_*}\left(\frac{S - S_*}{S}\right)\frac{dS}{dt} = \sum_{x \in V} \frac{1}{S_*}\left(\frac{S - S_*}{S}\right) \cdot \left(\Lambda + \gamma I - \beta_1 S P - \frac{\beta_2 S \int_{\tau=0}^{h} f(\tau)\, I(t-\tau)\, d\tau}{1 + kS} - \mu S + d_1 \Delta_\omega S\right).$$

Given Lemma 1 in [6], we have

$$\sum_{x\in V}\left(1-\frac{S_*}{S}\right)\Delta_\omega S = -\sum_{x\in V}\frac{S_*}{S}\Delta_\omega S = -\frac{1}{2}S_*\sum_{x\in V}(S(y)-S(x)) \cdot\left(\frac{1}{S(y)}-\frac{1}{S(x)}\right)\omega(x,y) = -\frac{S_*}{2}\sum_{x\in V}\frac{(S(y)-S(x))^2}{S(x)S(y)}\omega(x,y)\le 0.$$

Therefore

$$\begin{aligned}\frac{dU_S}{dt} &= \sum_{x\in V}\frac{1}{S_*}\left(\frac{S-S_*}{S}\right)\left(\beta_1 S_* P+\frac{\beta_2 S_* I_*}{1+kS_*}+\mu S_*-\gamma I_*+\gamma I-\beta_1 S P-\mu S\right.\\ &\left.-\frac{\beta_2 S\int_{\tau=0}^{h} f(\tau)I(t-\tau)d\tau}{1+kS}\right)\\ &= \sum_{x\in V}\frac{1}{S_*}\left(\frac{S-S_*}{S}\right)(-(\beta_1 P+\mu)(S-S_*)+\gamma(I-I_*)+\\ &\beta_2\int_{\tau=0}^{h} f(\tau)\left(\frac{S_* I_*}{1+kS_*}-\frac{S I(t-\tau)}{1+kS}\right)d\tau\Big)\\ &= \sum_{x\in V}\left[-\frac{(\beta_1 P+\mu)}{S_*}\frac{(S-S_*)^2}{S}+\frac{1}{SS_*}(S-S_*)(I-I_*)\right.\\ &\left.+\beta_2\int_{\tau=0}^{h} f(\tau)\frac{1}{S_*}\left(1-\frac{S_*}{S}\right)\left(\frac{S_* I_*}{1+kS_*}-\frac{S I(t-\tau)}{1+kS}\right)d\tau\right]\\ &\le \sum_{x\in V}\left[-\frac{(\beta_1 P+\mu)}{S_*}\frac{(S-S_*)^2}{S}+\frac{1}{2SS_*}(S-S_*)^2+\frac{1}{2II_*}(I-I_*)^2\right.\\ &\left.+\frac{\beta_2 S_* I_*^2}{1+kS_*}\int_{\tau=0}^{h} f(\tau)\left(1-\frac{S_*}{S}\frac{I(t-\tau)}{I_*}\right)+\left(1-\frac{I(t-\tau)}{I_*}\right)d\tau\right].\end{aligned}$$

Let $x=\frac{S(t)}{S_*}$, $y=\frac{I(t)}{I_*}$, $z=\frac{I(t-\tau)}{I_*}$, then

$$\frac{dU_S}{dt} = \sum_{x\in V}\left[-\frac{(\beta_1 P+\mu)}{S_*}\frac{(S-S_*)^2}{S}+\frac{1}{2SS_*}(S-S_*)^2+\frac{1}{2II_*}(I-I_*)^2+\right.$$
$$\left.\frac{\beta_2 S_* I_*^2}{1+kS_*}\int_{\tau=0}^{h} f(\tau)\left(1-\frac{1}{x}-xz+z+1-\frac{1}{x}-z+\frac{z}{x}\right)d\tau\right]. \tag{E.1}$$

On the other hand

$$\frac{dU_I}{dt} = \sum_{x\in V}\frac{1}{I_*}\left(1-\frac{I_*}{I}\right)\left(\beta_1 S P+\frac{\beta_2 S\int_{\tau=0}^{h} f(\tau)I(t-\tau)d\tau}{1+kS}-(\mu+\alpha+\gamma)I\right)$$
$$= \sum_{x\in V}\frac{1}{I_*}\left(1-\frac{I_*}{I}\right)\left(\beta_1 P S_*\frac{S}{S_*}+\frac{\beta_2 S_* I_*}{1+kS}\int_{\tau=0}^{h} f(\tau)\frac{S}{S_*}\frac{I(t-\tau)}{I_*}d\tau-(\mu+\alpha+\gamma)I\right).$$

Using $\beta_1 S_* P + \frac{\beta_2 S_* I_*}{1+kS_*} = (\mu+\alpha+\gamma)I_*$ to replace it gives

$$\begin{aligned}\frac{dU_I}{dt} &= \sum_{x\in V}\frac{1}{I_*}\left(1-\frac{I_*}{I}\right)\Bigg((\mu+\alpha+\gamma)I_* + \frac{\beta_2 S_* I_*^2}{(1+kS_*)I} - \\ &\quad \frac{\beta_2 S_* I_*}{1+kS}\int_{\tau=0}^{h} f(\tau)\frac{S}{S_*}\frac{I^2(t-\tau)}{I_*^2}d\tau - (\mu+\alpha+\gamma)I\Bigg) \\ &\le \sum_{x\in V}\frac{1}{I_*}\left(1-\frac{I_*}{I}\right)\Bigg(-(\mu+\alpha+\gamma)(I-I_*) + \frac{\beta_2 S_* I_*}{(1+kS_*)} \\ &\quad \int_{\tau=0}^{h} f(\tau)\left(\left(\frac{S}{S_*}\frac{I(t-\tau)}{I_*} - \frac{I}{I_*}\right)\right)d\tau\Bigg) \\ &= \sum_{x\in V} -\frac{(\mu+\alpha+\gamma)}{II_*}(I-I_*)^2 + \frac{\beta_2 S_* I_*}{(1+kS_*)}\int_{\tau=0}^{h} f(\tau)\left(xz-1-\frac{xz}{y}+\frac{1}{y}\right)d\tau.\end{aligned}$$

Hence

$$\frac{dU_I}{dt} = \sum_{x\in V} -\frac{(\mu+\alpha+\gamma)}{II_*}(I-I_*)^2 + \frac{\beta_2 S_* I_*}{(1+kS_*)}\int_{\tau=0}^{h} f(\tau)\left(xz-1-\frac{xz}{y}+\frac{1}{y}\right)d\tau. \tag{E.2}$$

Then, the derivative of $U_+(t)$ is calculated as follows:

$$\frac{dU_+}{dt} = \sum_{x\in V}\frac{d}{dt}\int_h^{\tau=0}\alpha(\tau)\,g\left(\frac{I(t-\tau)}{I_*}\right)d\tau = \sum_{x\in V}\int_h^{\tau=0} f(\tau)\left(g\left(\frac{I}{I_*}\right) - g\left(\frac{I(t-\tau)}{I_*}\right)\right)d\tau. \tag{E.3}$$

According to (E.1)–(E.3), it can be obtained

$$\begin{aligned}\frac{dU}{dt} &= \sum_{x\in V}\Bigg[-\left(\frac{2(\beta_1 P+\mu)-1}{2SS_*}\right)(S-S_*)^2 - \left(\frac{2(\mu+\alpha+\gamma)-1}{2II_*}\right)(I-I_*)^2 \\ &\quad -\int_{\tau=0}^{h} f(\tau)C(\tau)\,d\tau\Bigg].\end{aligned}$$

where

$$\begin{aligned}C(\tau) &= \left(-1+\frac{1}{x}+xz-z\right)+\left(-1+\frac{1}{x}+z-\frac{z}{x}\right)+\left(xz-1-\frac{xz}{y}+\frac{1}{y}\right)+lny+lnz \\ &= 2\left(\frac{1}{x}-1-ln\frac{1}{x}\right)+2(xz-z-lnxz)+\frac{z}{x}-1-ln\frac{z}{x}+\frac{xz}{y}-1-ln\frac{xz}{y} \\ &\quad +2ln\frac{1}{x}+2lnxz+ln\frac{z}{x}+ln\frac{xz}{y}+lny+lnz \ge 0.\end{aligned}$$

It is observed that $\frac{dU(t)}{dt} \le 0$ if $R_0 > 1$, $min(\beta_1 P+\mu, \mu+\alpha+\gamma) > \frac{1}{2}$.

Thus, $\frac{dU}{dt} \le 0$. By Theorem 5.3.1 of [5], solutions limit to the largest invariant subset of $\left\{\frac{dU}{dt}=0\right\}$. It is shown that Ω comprises only the endemic equilibrium E_*.

Note that $C(\tau) = 0$ if and only if $x=1$, $y=1$, $z=1$ or equivalently, if and only if $S(t) = S_*$, $I(t) = I(t-\tau)$. Thus, $\left\{\frac{dU}{dt}=0\right\}$ if and only if

$$S(t) = S_* \text{ and } f(\tau)I(t) = f(\tau)I(t-\tau) \tag{3.9}$$

for almost all $\tau \in [0, h]$. For each element of Ω, we have $S(t) = S_*$ and, since Ω is invariant, $\frac{dS}{dt} = 0$. Using equalities (3.6) and (3.7), we obtain

$$\begin{aligned} 0 &= \frac{dS}{dt} \\ &= \Lambda + \gamma I_* - \beta_1 S_* P - \frac{\beta_2 S_* \int_{\tau=0}^{h} f(\tau) I(t-\tau) d\tau}{1 + kS_*} - \mu S_* \\ &= \Lambda + \gamma I_* - \beta_1 S_* P - \frac{\beta_2 S_* \int_{\tau=0}^{h} f(\tau) I(t) d\tau}{1 + kS_*} - \mu S_*. \end{aligned}$$

Rearranging gives $I(t) = \frac{\Lambda - \beta_1 S_* P - \mu S_*}{\frac{\beta_2 S_*}{1+kS_*} - \gamma} = I_*$.

Because of $S(t) = S_*$, $I(t) = I_*$ for all t, we can gains $\frac{dU(t)}{dt} = 0$. It seems that $\lim_{t\to\infty} (S(t), I(t)) = (S_*, I_*)$. Further, it now follows that E_* is globally asymptotically stable if $R_0 > 1$, $min(\beta_1 P + \mu, \mu + \alpha + \gamma) > \frac{1}{2}$. □

3.3. Discussion

The construction of the entire Lyapunov functional is not merely a mathematical technique, but a "mathematical mapping" of the transmission mechanism of respiratory diseases. This paper, by characterizing the core transmission links such as the fluctuation of susceptible individuals, the scale of infection, rehabilitation protection, network spread, and time delay accumulation item by item, presents the biological logic of "reduced transmission risk → system stabilization". It is transformed into a mathematical determination of "decreasing functional value → convergence to disease-free equilibrium point", which not only ensures the rigor of the stability proof but also deeply integrates mathematical tools with biological reality.

Under normal circumstances, distributed delay will change the transmission rhythm of network respiratory diseases through the "time accumulation effect", thereby affecting the stability of the model. In the short term, it may weaken local stability and cause fluctuations. If the distribution delay is significant, even if the overall infection rate is not high, there may still be short-term small outbreaks where "one wave has not subsided and another has emerged", and the local stability of the corresponding model will be weakened.

In the long term, distributed delay may undermine global stability and lead to the recurrence of the disease. The conclusions of Theorems 4 and 5 in this paper indicate that distributed time delays may cause the "basic regeneration number $R_0 < 1$" to fluctuate over the long term. Even if the theoretical $R_0 < 1$, the cumulative delayed propagation may lead to the disease not being completely eliminated. The more difficult it is for the disease to achieve "global extinction", the more the global stability of the corresponding model is disrupted.

4. Discrete delay

The previous text has conducted a comprehensive and detailed analysis of the infectious disease model with distributed time delay (1.1), systematically exploring the influence of distributed time delay on the dynamic behavior of disease transmission and related key properties. To further improve the

applicability of the model and cover time delay types that are closer to actual propagation scenarios, the case of discrete delay is considered, then system (1.1) becomes the following model

$$\begin{cases} \frac{\partial S}{\partial t} - d_1\Delta_\omega S\,(x,t) = \Lambda + \gamma I\,(x,t) - \beta_1 S\,(x,t)\,P - \beta_2 \frac{S(x,t)I(x,t-\tau)e^{-\mu\tau}}{1+kS(x,t)} - \mu S\,(x,t), \\ \quad (x,t) \in V \times (0,\infty), \\ \frac{\partial I}{\partial t} - d_2\Delta_\omega I\,(x,t) = \beta_1 S\,(x,t)\,P + \beta_2 \frac{S(x,t)I(x,t-\tau)e^{-\mu\tau}}{1+kS(x,t)} - (\mu+\alpha+\gamma)I\,(x,t)\,, \\ \quad (x,t) \in V \times (0,\infty), \\ S\,(x,t) = S_0\,(x,t)\,, I(x,t) = I_0(x,t), \quad (x,t) \in V \times [-\tau,0]. \end{cases} \tag{4.1}$$

To clearly present the model structure, we visualized the fractional-order infectious disease transmission framework with a network scale of N = 5, as shown in the specific Figure 2.

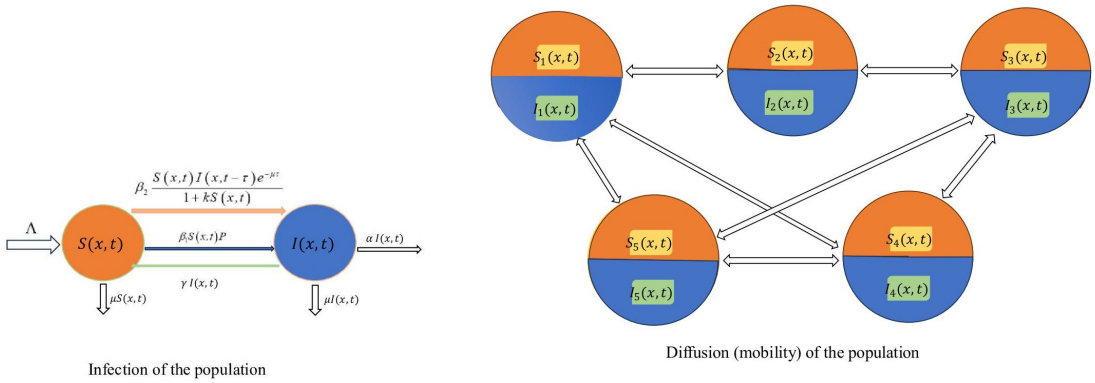

Figure 2. The left figure shows the disease transmission dynamics within node i, $i = 1,2,3,4,5$; In the right figure, each node contains two types of individuals - susceptible individuals ($S(x,t)$) and infected individuals ($I(x,t)$). The white arrows between the nodes represent the connection relationship of population diffusion (mobility).

4.1. Delay induced Hopf bifurcation

In general, the system does not lose stability at the disease-free equilibrium and produces Hopf bifurcation. Therefore, the Hopf bifurcation of system (4.1) at the endemic equilibrium is studied mainly in this section.

Lemma 1. *From the eigenvalue problem*

$$\begin{cases} -\Delta_\omega \phi\,(x) = \iota\phi\,(x)\,, x \in V, \\ \int_V \phi^2\,(x) = 1, \end{cases} \tag{4.2}$$

exists a series of eigenvalues $\{\iota_i\}_{i=1}^n : 0 = \iota_1 < \iota_2 \leq \cdots \leq \iota_n$, *whose associated eigenfunctions are* $\{\phi_i\}_{i=1}^n$. *Moreover, letting* E_i *be the space of eigenfunctions corresponding to* ι_i *such that* $E_i := \{c \cdot \phi_i : c \in \Re\}$, *one has the following space decomposition:*

$$\left[L^2\,(V)\right]^2 = \bigoplus_{i=1}^{n} E_i. \tag{4.3}$$

In order to determine the stability of $\left(\tilde{S}, \tilde{I}\right)$, we define the small perturbations around $\left(\tilde{S}, \tilde{I}\right)$ are θ_S and θ_I. The linearized system (4.1) around $\left(\tilde{S}, \tilde{I}\right)$ is

$$\begin{pmatrix} \dot{\theta_S} \\ \dot{\theta_I} \end{pmatrix} = J_1 \begin{pmatrix} \theta_S \\ \theta_I \end{pmatrix} + J_2 \begin{pmatrix} \theta_{S_\tau} \\ \theta_{I_\tau} \end{pmatrix} + D \begin{pmatrix} \Delta_\omega \theta_S \\ \Delta_\omega \theta_I \end{pmatrix}, \tag{4.4}$$

where $\theta_{I_\tau} = \theta_I (t - \tau)$, and

$$J_1 = \begin{pmatrix} a_{11} & a_{12} \\ a_{21} & a_{22} \end{pmatrix}, J_2 = \begin{pmatrix} 0 & a_{13} \\ 0 & a_{23} \end{pmatrix}, D = \begin{pmatrix} d_1 & 0 \\ 0 & d_2 \end{pmatrix}. \tag{4.5}$$

Here $a_{11} = -\beta_1 P - \mu - \frac{\beta_2 \tilde{I} e^{-\mu\tau}}{\left(1+k\tilde{S}\right)^2}, a_{12} = \gamma, a_{21} = \beta_1 P + \frac{\beta_2 \tilde{I} e^{-\mu\tau}}{\left(1+k\tilde{S}\right)^2}, a_{22} = -(\mu + \alpha + \gamma), a_{13} = -\frac{\beta_2 \tilde{I} e^{-\mu\tau}}{\left(1+k\tilde{S}\right)^2}, a_{23} = \frac{\beta_2 \tilde{I} e^{-\mu\tau}}{\left(1+k\tilde{S}\right)^2}$.

On the basis of Lemma 4.1, the space E_i is invariant under the operator $-D\Delta_\omega$, and λ_i is an eigenvalue of this operator on E_i, if and only if it is an eigenvalue of the matrix $\lambda_i D$. For any small perturbation $(\theta_S, \theta_I)^T$ from $(0, 0)$, the basis decomposition is as follows:

$$\begin{pmatrix} \theta_S \\ \theta_I \end{pmatrix} = \sum_{i=1}^{n} \begin{pmatrix} c_1^i \\ c_2^i \end{pmatrix} e^{\lambda_i t} \phi_i, \ (i = 1, 2, \cdots, n). \tag{4.6}$$

Inserting (4.6) in (4.4), noticing that $-\Delta_\omega \phi_i = \iota_i \phi_i$ and using the orthogonality of the eigenvectors, we get for each mode $\phi_i (1, 2, \cdots, n)$ that

$$\lambda_i \begin{pmatrix} c_1^i \\ c_2^i \end{pmatrix} e^{\lambda_i t} = J_1 \begin{pmatrix} c_1^i \\ c_2^i \end{pmatrix} e^{\lambda_i t} + J_2 \begin{pmatrix} c_1^i \\ c_2^i \end{pmatrix} e^{\lambda_i (t-\tau)} - \iota_i D \begin{pmatrix} c_1^i \\ c_2^i \end{pmatrix} e^{\lambda_i t}.$$

Therefore, the characteristic equation is as follows:

$$\prod_{i=1}^{n} \Delta (\lambda_i, \tau) = 0, where \ \Delta (\lambda_i, \tau) = det \left(\lambda_i I - \left(J_1 + e^{-\lambda_i \tau} J_2 - \iota_i D\right)\right). \tag{4.7}$$

Let's put (4.7) into (4.5), the characteristic equation becomes

$$\begin{aligned} \Delta (\lambda_i, \tau) = \lambda_i^2 &+ \left(-a_{11} - a_{22} + (d_1 + d_2) \iota_i - a_{23} e^{-\lambda_i \tau}\right) \lambda_i \\ &+ a_{11} a_{22} - a_{12} a_{21} - (a_{11} d_2 + a_{22} d_1) \iota_i + d_1 d_2 \iota_i^2 \\ &+ (a_{11} + d_2 \iota_i) a_{23} e^{-\lambda_i \tau}. \end{aligned} \tag{4.8}$$

It can be obtained by calculation as follow

$$\Delta (\lambda_i, \tau) = \lambda_i^2 + A_1 \lambda_i + A_2 + A_3 e^{-\lambda_i \tau}, \tag{4.9}$$

where $A_1 = -a_{11} - a_{22} + (d_1 + d_2) \iota_i - a_{23} e^{-\lambda_i \tau}, A_2 = a_{11} a_{22} - a_{12} a_{21} - (a_{11} d_2 + a_{22} d_1) \iota_i + d_1 d_2 \iota_i^2, A_3 = (a_{11} + d_2 \iota_i) a_{23}$.

The following results are found on the stability of $\left(\tilde{S}, \tilde{I}\right)$ of the model (4.1) by analyzing the characteristic Eq (4.7).

Lemma 2. *(i) For the case without the delay, all characteristic roots of the Eq (4.7) have negative real parts.*
(ii) For the other case with the delay, the characteristic equation has a pair of purely imaginary roots $\pm i\omega^*$ *at* $\tau = \tau_j$*, also*

$$(\omega^*)^2 = \frac{1}{2}\left(2A_2 - A_1^2 + \sqrt{\left(2A_2 - A_1^2\right)^2 - 4\left(A_2^2 - A_3^2\right)}\right) \tag{4.10}$$

and

$$\tau_j = \frac{1}{\omega^*}\left(2j\pi + \arccos\left(\frac{(\omega^*)^2 - A_2}{A_3}\right)\right), j = 0, 1, 2\cdots. \tag{4.11}$$

where, $A_i, i = 1, 2, 3$ *is given in the following Eq (4.13).*

Proof. (i) For the first case $\tau = 0$. Plugging $\tau = 0$ to (4.8) yields

$$\Delta(\lambda_i, \tau) = \lambda_i^2 + A_1\lambda_i + A_2 + A_3, \tag{4.12}$$

where

$$\begin{aligned}
A_1 &= (\beta_1 P + \mu) + (\mu + \alpha + \gamma) + (d_1 + d_2)\,\iota_i,\\
A_2 &= \mu(\mu + \alpha + \gamma) + (\mu + \alpha)\left(\beta_1 P + \frac{\beta_2 \tilde{I} e^{-\mu\tau}}{\left(1 + k\tilde{S}\right)^2}\right) +\\
&\quad \left[(\mu + \alpha + \gamma)\, d_1 + \left(\beta_1 P + \mu + \frac{\beta_2 \tilde{I} e^{-\mu\tau}}{\left(1 + k\tilde{S}\right)^2}\right) d_2\right]\iota_i,\\
A_3 &= \left(\beta_1 P + \mu + \frac{\beta_2 \tilde{I} e^{-\mu\tau}}{\left(1 + k\tilde{S}\right)^2} + d_2\iota_i\right)\frac{\beta_2 \tilde{I} e^{-\mu\tau}}{\left(1 + k\tilde{S}\right)^2}.
\end{aligned} \tag{4.13}$$

Because of $0 = \iota_1 < \iota_2 \le \cdots \le \iota_n$, and $A_1 > 0, A_2 + A_3 > 0$ holds, then the real parts of the roots of $\Delta(\lambda_i, \tau) = 0$ are negative. Given that the characteristic equation is $\prod_{i=1}^{n}\Delta(\lambda_i, \tau) = 0$, all the characteristic roots of (4.7) have real negative parts.

(ii) Set $\pm i\omega$ be a pair of pure imaginary roots. Putting $\pm i\omega$ into $\Delta(\lambda_i, \tau) = 0$ and separating real and imaginary parts, we can know

$$\begin{cases} -\omega^2 + A_2 + A_3\cos\omega\tau = 0,\\ A_1\omega - A_3\sin\omega\tau = 0, \end{cases} \tag{4.14}$$

which leads to

$$\omega^4 + \left(A_1^2 - 2A_2\right)\omega^2 + A_2^2 - A_3^2 = 0. \tag{4.15}$$

The Eq (4.15) has a unique positive real root $(\omega^*)^2$ if and only if $\left(A_1^2 - 2A_2\right)^2 > 4\left(A_2^2 - A_3^2\right)$. Then, the expression of ω^* is obtained in the form of (4.10) by solving the above Eq (4.15). Hence, the corresponding τ_j of (4.11) can be obtained by substituting (4.10) into (4.12). □

Theorem 6. *When $\tau = \tau_0$, the model* (4.1) *will undergo a Hopf bifurcation at the endemic equilibrium* $\left(\tilde{S}, \tilde{I}\right)$, *where* $\tau_0 = \frac{1}{\omega^*}\left(\arccos\left(\frac{(\omega^*)^2 - A_2}{A_3}\right)\right)$.

Proof. By the Lemma 2, it is only needs to show the following transversality condition

$$\frac{\mathrm{d}}{\mathrm{d}t} Re\lambda(\tau)\,|_{\tau=\tau_0} > 0, \tag{4.16}$$

with $\iota_i \equiv \iota_1$. Hence, the following analyzes the characteristic equation (4.9), where the parameter A_i has already been given in the previous equation (4.13).

It is supposed that $\lambda_i = \iota + i\omega$ is a complex eigenvalue of (4.16). It can be obtained by calculation

$$\begin{cases} \iota^2 - \omega^2 + A_1\iota + A_2 + A_3 e^{-\iota\tau}\cos\omega\tau = 0, \\ 2\iota\omega + A_1\omega - A_3 e^{-\iota\tau}\sin\omega\tau = 0. \end{cases} \tag{4.17}$$

Differentiating (4.17) to τ, one gets

$$\begin{cases} (2\iota + A_1 - A_3\tau e^{-\iota\tau}\cos\omega\tau)\frac{\mathrm{d}\iota}{\mathrm{d}\tau} - (2\omega + A_3\tau e^{-\iota\tau}\sin\omega\tau)\frac{\mathrm{d}\omega}{\mathrm{d}\tau} = \\ A_3\tau e^{-\iota\tau}(\iota\cos\omega\tau + \omega\sin\omega\tau), \\ (2\omega + A_3\tau e^{-\iota\tau}\sin\omega\tau)\frac{\mathrm{d}\iota}{\mathrm{d}\tau} + (2\iota + A_1 - A_3\tau e^{-\iota\tau}\cos\omega\tau)\frac{\mathrm{d}\omega}{\mathrm{d}\tau} = \\ A_3\tau e^{-\iota\tau}(\iota\sin\omega\tau + \omega\cos\omega\tau). \end{cases} \tag{4.18}$$

Eliminating $\frac{d\omega}{d\tau}$ of the above two equations, one has

$$\begin{aligned} &\left[(2\iota + A_1 - A_3\tau e^{-\iota\tau}\cos\omega\tau)^2 + (2\omega + A_3\tau e^{-\iota\tau}\sin\omega\tau)^2\right]\frac{d\iota}{d\tau} \\ &= A_3\tau e^{-\iota\tau}\left[(\iota\cos\omega\tau + \omega\sin\omega\tau)(2\iota + A_1 - A_3\tau e^{-\iota\tau}\cos\omega\tau)\right] \\ &+ A_3\tau e^{-\iota\tau}\left[(\iota\sin\omega\tau + \omega\cos\omega\tau)(2\omega + A_3\tau e^{-\iota\tau}\sin\omega\tau)\right]. \end{aligned} \tag{4.19}$$

In view of (4.17), to prove $\frac{d\iota}{d\tau} > 0$, it needs to verify that

$$\begin{aligned} &(\iota\cos\omega\tau + \omega\sin\omega\tau)(2\iota + A_1 - A_3\tau e^{-\iota\tau}\cos\omega\tau) \\ &+ (\iota\sin\omega\tau + \omega\cos\omega\tau)(2\omega + A_3\tau e^{-\iota\tau}\sin\omega\tau) > 0. \end{aligned} \tag{4.20}$$

In fact, when $\tau = \tau_0$ and $\omega = \omega^*$, the above inequality satisfies $\iota = 0$. In the case of $\tau = \tau_0, \iota = 0, \omega = \omega^*$, (4.17) becomes

$$\begin{cases} -(\omega^*)^2 + A_2 + A_3\cos\omega^*\tau = 0, \\ A_1\omega - A_3\sin\omega^*\tau = 0, \end{cases} \tag{4.21}$$

and (4.20) becomes

$$\omega^*\sin\omega^*\tau_0(A_1 - A_3\tau_0\cos\omega^*\tau_0) + \omega^*\cos\omega^*\tau_0(2\omega^* + A_3\tau_0\sin\omega^*\tau_0) > 0. \tag{4.22}$$

It can be simplified further from (4.22) that

$$A_1\omega^*\sin\omega^*\tau_0 + 2(\omega^*)^2\cos\omega^*\tau_0 > 0. \tag{4.23}$$

If substitute (4.21) into (4.23), you get this

$$\frac{1}{A_3}A_1^2(\omega^*)^2 - \frac{1}{A_3}\left[2(\omega^*)^2\left(A_2 - 2(\omega^*)^2\right)\right] > 0. \tag{4.24}$$

To make $A_3 > 0$, we have to verify formula

$$2(\omega^*)^4 + \left(A_1^2 - 2A_2\right)(\omega^*)^2 > 0. \tag{4.25}$$

On account of $A_1^2 > 2A_2$ always valid, the inequality (4.25) is true. □

4.2. Global stability

Theorem 7. *It is easy to know that model* (4.1) *has an unique endemic equilibrium given by* $E_* = (S_*, I_*)$, *and it is globally asymptotically stable if* $I_* < \frac{2\cdot min(\beta_1 P+\mu,\mu+\alpha+\gamma)}{\gamma+\beta_1 P S_*}$.

Proof. We decide to choose the Lyapunov function of the following form

$$\begin{aligned} E(t) &= \frac{S_* I_*}{\beta_2 e^{-\mu\tau}(1+kS_*)} E_S + \frac{S_* I_*}{\beta_2 e^{-\mu\tau}(1+kS_*)} E_I + E_- \\ &= \frac{S_* I_*}{\beta_2 e^{-\mu\tau}(1+kS_*)} \sum_{x\in V} \left(\frac{S}{S_*} - 1 - \ln\left(\frac{S}{S_*}\right)\right) \\ &+ \frac{S_* I_*}{\beta_2 e^{-\mu\tau}(1+kS_*)} \sum_{x\in V} \left(\frac{I}{I_*} - 1 - \ln\left(\frac{I}{I_*}\right)\right) \\ &+ \sum_{x\in V} \int_{\tau=0}^{h} g\left(\frac{I(t-\tau)}{I_*}\right) d\tau. \end{aligned}$$

Then $E(t) \geq 0$ for all $t \geq 0$, and $E(t) = 0$ if and only if $(S, I) = (S_*, I_*)$. It can be obtained by calculation

$$\begin{aligned} E'(t) &= \frac{S_* I_*}{\beta_2 e^{-\mu\tau}(1+kS_*)} \sum_{x\in V} \left(1 - \frac{S_*}{S}\right) \\ &\cdot \left(d_1 \Delta_\omega S + \left(\Lambda + \gamma I - \beta_1 S P - \beta_2 \frac{S I(t-\tau) e^{-\mu\tau}}{1+kS} - \mu S \right)\right) \\ &+ \frac{S_* I_*}{\beta_2 e^{-\mu\tau}(1+kS_*)} \sum_{x\in V} \left(1 - \frac{I_*}{I}\right) \\ &\cdot \left(d_2 \Delta_\omega I + \left(\beta_1 S P + \beta_2 \frac{S I(t-\tau) e^{-\mu\tau}}{1+kS} - (\mu+\alpha+\gamma) I \right)\right) \\ &+ \sum_{x\in V} \int_0^{\tau} \frac{d}{dt} g\left(\frac{I(t-\tau)}{I_*}\right) d\tau. \end{aligned}$$

First of all, we know

$$\begin{aligned} &\frac{S_* I_*}{\beta_2 e^{-\mu\tau}(1+kS_*)} \sum_{x\in V} \frac{dE_S}{dt} = \frac{S_* I_*}{\beta_2 e^{-\mu\tau}(1+kS_*)} \sum_{x\in V} \frac{1}{S_*} \left(\frac{S-S_*}{S}\right) \frac{dS}{dt} \\ &\leq -\frac{(\beta_1 P+\mu) I_*}{\beta_2 e^{-\mu\tau}(1+kS_*) S} (S-S_*)^2 + \frac{\gamma I_*}{2S\beta_2 e^{-\mu\tau}(1+kS_*)} \left[(S-S_*)^2 + (I-I_*)^2 \right] \\ &+ \left(1 - \frac{S_*}{S}\right)\left(1 - \frac{S}{S_*}\frac{I(t-\tau)}{I_*}\right). \end{aligned}$$

Let $g(a) = a - 1 - \ln(a) > 0$ [3], and have $g(1) = 0$. Set $x = \frac{S(t)}{S_*}$, $y = \frac{I(t)}{I_*}$, $z = \frac{I(t-\tau)}{I_*}$, then

$$\begin{aligned} \frac{S_* I_*}{\beta_2 e^{-\mu\tau}(1+kS_*)} \sum_{x\in V} \frac{dE_S}{dt} &\leq -\frac{(2(\beta_1 P+\mu)-\gamma) I_*}{2\beta_2 e^{-\mu\tau}(1+kS_*) S} (S-S_*)^2 + \frac{\gamma I_* (I-I_*)^2}{2S\beta_2 e^{-\mu\tau}(1+kS_*)} \\ &+ \left(1 - \frac{1}{x}\right)(1-xz). \end{aligned} \tag{E.4}$$

From the Lemma 1 in [19], one receives

$$\sum_{x\in V}\left(1-\frac{I_*}{I}\right)\Delta_\omega I=-\frac{I_*}{2}\sum_{x\in V}\left(I\left(y\right)-I\left(x\right)\right)\left(\frac{1}{I\left(y\right)}-\frac{1}{I\left(x\right)}\right)\omega\left(x,y\right)$$
$$=-\frac{I_*}{2}\sum_{x\in V}\frac{\left(I\left(y\right)-I\left(x\right)\right)^2}{I\left(x\right)I\left(y\right)}\omega\left(x,y\right)\leq 0.$$

On the other hand

$$\begin{aligned}
&\frac{S_*I_*}{\beta_2e^{-\mu\tau}\left(1+kS_*\right)}\sum_{x\in V}\frac{dE_I}{dt}\\
&=\sum_{x\in V}\frac{S_*}{\beta_2e^{-\mu\tau}\left(1+kS_*\right)}\left(1-\frac{I_*}{I}\right)\left[\beta_1SP+\beta_2\frac{SI\left(t-\tau\right)e^{-\mu\tau}}{1+kS}-\left(\mu+\alpha+\gamma\right)I\right]\\
&\leq\sum_{x\in V}\left(1-\frac{I_*}{I}\right)\left(\frac{\beta_1PS_*I_*}{\beta_2e^{-\mu\tau}\left(1+kS_*\right)}\left(S-S_*\right)-\frac{\left(\mu+\alpha+\gamma\right)S_*I_*}{\beta_2e^{-\mu\tau}\left(1+kS_*\right)}\left(I-I_*\right)\right.\\
&\left.+\left(1-\frac{S}{S_*}\frac{I\left(t-\tau\right)}{I_*}\right)\right)\\
&\leq\sum_{x\in V}\left(\frac{\beta_1PS_*I_*\left(S-S_*\right)^2}{2\beta_2e^{-\mu\tau}\left(1+kS_*\right)I}-\frac{\left(2\left(\mu+\alpha+\gamma\right)-\beta_1P\right)S_*I_*\left(I-I_*\right)^2}{2\beta_2e^{-\mu\tau}\left(1+kS_*\right)I}\right.\\
&\left.+\left(1-\frac{1}{y}\right)\left(1-xz\right)\right),
\end{aligned}$$

that is

$$\begin{aligned}
\frac{S_*I_*}{\beta_2e^{-\mu\tau}\left(1+kS_*\right)}\sum_{x\in V}\frac{dE_I}{dt}=\sum_{x\in V}\left[\right.&\frac{\beta_1PS_*I_*\left(S-S_*\right)^2}{2\beta_2e^{-\mu\tau}\left(1+kS_*\right)I}\\
&-\frac{\left(2(\mu+\alpha+\gamma)-\beta_1P\right)S_*I_*\left(I-I_*\right)^2}{2\beta_2e^{-\mu\tau}\left(1+kS_*\right)I}\\
&\left.+\left(1-\frac{1}{y}\right)\left(1-xz\right)\right],
\end{aligned}\tag{E.5}$$

Moreover

$$\sum_{x\in V}\frac{dE_-}{dt}=\sum_{x\in V}\frac{d}{dt}\int_0^\tau g\left(\frac{I\left(t-\tau\right)}{I_*}\right)d\tau=\sum_{x\in V}\int_0^\tau\frac{d}{dt}g\left(\frac{I\left(t-\tau\right)}{I_*}\right)d\tau,$$

hence

$$\sum_{x\in V}\frac{dE_-}{dt}=\sum_{x\in V}\left(g\left(\frac{I\left(t-\tau\right)}{I_*}\right)-g\left(\frac{I\left(t\right)}{I_*}\right)\right)=\sum_{x\in V}\left(g\left(z\right)-g\left(x\right)\right).\tag{E.6}$$

By the Eqs (E.4)–(E.6), it can be acquired

$$\begin{aligned}
\frac{dE\left(t\right)}{dt}&=\sum_{x\in V}\frac{S_*I_*}{\beta_2e^{-\mu\tau}\left(1+kS_*\right)}\frac{dE_S}{dt}+\sum_{x\in V}\frac{S_*I_*}{\beta_2e^{-\mu\tau}\left(1+kS_*\right)}\frac{dE_I}{dt}+\sum_{x\in V}\frac{dE_-}{dt}\\
&=-\sum_{x\in V}\frac{2\left(\beta_1P+\mu\right)-\gamma I_*-\beta_1PS_*I_*}{2\beta_2e^{-\mu\tau}\left(1+kS_*\right)S_*}\left(S-S_*\right)^2
\end{aligned}$$

$$-\frac{2(\mu+\alpha+\gamma)-\gamma I_*-\beta_1 P S_* I_*}{2\beta_2 e^{-\mu\tau}(1+kS_*)}(I-I_*)^2-\sum_{x\in V}C(\tau),$$

where

$$\begin{aligned}C(\tau)&=g(x)-g(z)-\left(1-\frac{1}{x}\right)(1-xz)-\left(1-\frac{1}{y}\right)(1-xz)\\&=x-1-\ln x+z-1-\ln z-\left(1-xz-\frac{1}{x}+z\right)-\left(1-xz-\frac{1}{y}+\frac{xz}{y}\right)\geq 0.\end{aligned}$$

It is want to make $\frac{dE(t)}{dt}\leq 0$, it has to be $I_*<\frac{2\cdot min(\beta_1 P+\mu,\mu+\alpha+\gamma)}{\gamma+\beta_1 P S_*}$.

Therefore, $C(\tau)=0$ if and only if $x=1$, $y=1$, $z=1$ or $S(t)=S_*$, $I(t)=I_*$, $I(t-\tau)=I_*$. Hence, $\frac{dE(S,I)}{dt}=0$ if and only if $S(t)=S_*, I(t)=I_*, C(\tau)I(t)=C(\tau)I(t-\tau)$. It is found that $\lim_{t\to\infty}(S(t),I(t))=(S_*,I_*)$ because of $S(t)=S_*$, $I(t)=I_*$ for all t. Then, the model (4.1) is globally asymptotically stable at unique endemic equilibrium if $I_*<\frac{2\cdot min(\beta_1 P+\mu,\mu+\alpha+\gamma)}{\gamma+\beta_1 P S_*}$. □

4.3. Discussion

According to the theoretical analysis results, in order to achieve global asymptotic stability for networked respiratory diseases with discrete time delay, the parameters β_1, P, γ in the model needs to satisfy specific relationships. It shows that the threshold condition interval of the parameters needed for global asymptotic stability of the model is smaller and the threshold condition is more stringent. This study will contribute to a better understanding of the transmission mechanism of respiratory diseases and the dynamic complexity of their corresponding ecosystems.

Discrete delay can disrupt the "synchronicity" of network respiratory disease transmission through "fixed time difference propagation delay", which may not only induce Hopf bifurcation and lead to periodic disease outbreaks, but also undermine global stability and prevent the disease from being completely eradicated. The essence of the Hopf bifurcation is the critical state where the model changes from "stable equilibrium" to "periodic oscillation", which corresponds to the "seasonal outbreak" and "periodic rebound" of diseases in reality.

In addition, from the conclusion of Theorem 7, it can be known that the discrete delay will fix the "effective transmission period" : Even if $R_0<1$, the fixed-period transmission feedback may still keep the disease at a "low-level periodic prevalence" and fail to reach the globally stable "disease-free equilibrium point". For instance, the seasonal outbreak of influenza is essentially a discrete time lag effect of "the low temperature in winter prolongs the virus's survival time (fixed time lag) + the population movement during the Spring Festival (fixed transmission delay)", which makes it impossible to be completely eradicated. It can only maintain a periodic stability of "outbreak in winter and subside in summer each year", rather than a global stability of complete elimination.

5. Conclusions

Currently, it is one of the hot spots in the prevention and control of respiratory infectious diseases to adopt epidemiological analysis technology and consider the analysis of the modeling of infectious diseases of different transmission routes. In recent years, many researchers have extensively analyzed

epidemic models with nonlinear incidence to understand the spread of disease [6–8]. Incidence plays a crucial role in the construction of epidemic models, with various incidence rates introduced by different authors. Bilinear and standard bilinear incidence are the two most widely used incidence rates in classical epidemic models [9, 17]. If the number of infected individuals becomes high, the spread of the disease may be slower than the linear response. In this case, the effect of infection saturation can lead to nonlinear morbidity [18, 19, 25].

In this paper, we take into account the respiratory disease model with nonlinear rate and distributed delay and discrete delay. For each model, the global stability of the endemic equilibrium E_* have proved by constructing the Liapunov functional. It is worth mentioning that owing to the introduction of a network and distributed delay and discrete delay, our theoretical analysis has brought out some interesting dynamic features.

This paper studies the influence mechanisms of respiratory diseases under air pollution corresponding to distributed time delay and discrete time delay, and adopts the linkage mechanism of "cross-regional population flow in polluted environment- disease transmission", enriching the understanding of the transmission laws of such diseases. Theoretical results reveal that the time lag effect in areas with high pollution incidence will amplify the transmission risk, verifying the positive correlation of "air pollution exposure - time lag - disease transmission", and providing quantitative theoretical support for analyzing the outbreak triggers of such diseases. In terms of public health impact, it is recommended to prioritize reducing the frequency of population contact in severely polluted areas to shorten the delay in disease transmission. In light of the impact of time lag on the persistence of the disease, monitoring and intervention should be initiated in advance to prevent the spread of the epidemic due to the time lag effect.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors declare there are no conflicts of interest.

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