EPIDEMIC DYNAMICS OF A FRACTIONAL MULTISTAGE SIR NETWORK MODEL

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In this paper, the epidemic dynamics of a fractional multistage SIR epidemic model on heterogeneous networks is analyzed. Specifically, the expression of the basic production number is provided. By constructing suitable Lyapunov functions, the global stability of the disease-free equilibrium of the model is investigated. Besides, the stability of the endemic equilibrium is also analyzed using the next generation matrix method. Finally, an example is presented to demonstrate the effectiveness of the theoretical results. Such results, as we believe, shall help adopt proper pragmatic treatments for prevention and control of disease.

Keywords: Epidemic dynamics; Fractional-order; SIR network model; Multistage

1. Introduction

Infectious disease spreading in social networks has been studied intensely in the last decades. Such studies have been helping public health authorities to assess situations more quickly, taking and enforcing informed decisions, as well as improving vaccination and drug delivery policies. Among such studies include those on analyzing infectious disease dynamics with a solid mathematical basis. With mathematical analyses and computer simulations, dynamic mathematical modeling of infectious disease links up epidemiology with the latest knowledge of social systems to reflect disease propagation more accurately through social interactions. A most notable development along this direction is that, with the development of network theory, the classical infectious disease models, such as Susceptible-Infected (SI), Susceptible-Infected-Susceptible (SIS), Susceptible-Infected-Recovered (SIR), Susceptible-Infected-Recovered-Susceptible (SIRS), and Susceptible-Exposed-Infected- Recovered (SEIR) models, have been extended to be based on various complex networks for investigating the transmission of epidemic diseases, computer viruses, innovations and rumors in various systems [1-10].

Some significant results have been achieved in network-based transmission dynamics analysis. The most influential result is probably the SIS and SIR model established in [3] through mean field theory. It is found that

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network heterogeneity has a key impact on disease transmission in degree-independent networks, especially in large scale-free networks, where diseases will persist no matter how small the transmission intensity is. Pastor-Satorras et al. [2] found that the transmission behaviors in small-world networks are similar to those in regular networks and random networks, and they all exist nonzero critical values of infection rate. Theoretical studies have also been carried out on disease transmission in multi-layer coupled networks. Kan et al. [8] introduced the self-consciousness variable and found that both the infection threshold and the infected density can be affected by the existence of the consciousness network. Gao et al. [9] proposed a nonlinearly coupled information epidemic model (I-E model) and conducted a comprehensive analysis on a rather general scenario where the upload rate and deletion rate may be different for different classes of nodes (e.g., nodes in unaware and aware states).

Since fractional calculus, especially the Caputo fractional derivative [27], has memory function, which ensures revealing the influences of historical information on the present and future, combining fractional calculus and the classical disease models is very effective in analyzing the dynamics of infectious diseases [11-18], helping to describe many real-life problems in a more accurate way than classic partial/ordinary differential equation approaches. It is no doubt that Lyapunov direct method provides a very effective approach to analyze stability of nonlinear systems. Since Matignon [19] established a well-known stability theorem of linear fractional systems, various kinds of stability of fractional linear and nonlinear systems including finite-time stability, Mittag-Leffler stability, asymptotic stability have been widely studied [20-21]. Motivated by such an observation, the stability of fractional disease systems has also been a hot topic in the last decades, for which many results have been achieved. Based on the basic regeneration number and the stability theory of Lyapunov, Zafar et al. [11] analyzed the stability of the equilibrium point of a fractional order HIV/AIDS model and its spread control. Rostamy et al. [13] discussed the existence of multiple equilibrium points in the SIR model and showed that selecting appropriate fractional order parameters can expand the stable region of equilibrium points. In addition, for the fractional-order infectious disease models, some scholars have proposed vaccination control strategies to prevent the spread of the disease. Sen et al. [22] proposed a simple linear vaccination control strategy for a class of SEIR infectious disease models and tracked immunized individuals in the entire population. Applying Lyapunov function and Kirchhoff matrix tree theorem, Huo et al. [23] analyzed the stability of a fractional order SIR model with birth and death rates on a heterogeneous network and compared the effects of various immunization programs.

However, some infectious disease outbreaks have highlighted the urgent need for a more accurate description of the disease dynamics to be used for

developing measures for disease control and prevention. For infectious diseases that progress through a long and complex infectious period, infectivity or infectiousness can vary significantly in time. In different stages of the disease, infected people show different degrees of disease symptoms, with different infectivity, which has an important impact on the spread of infectious diseases. In [24], behavior of multistage infections with particular emphasis on contact networks was discussed. The number of stages modified the temporary profile of an outbreak but did not affect the final epidemic size or the condition for disease outbreak, which reflected that the number of stages plays a much more prominent role. In [25], a general class of multistage models was provided to incorporate disease progression and amelioration of individual hosts to account for more realistic situations, where the dynamics of multistage models were completely determined by the basic reproduction number. A key new feature was to allow individual hosts to move with certain probability from any stage of the disease to any other stage either forward or backward. Furthermore, a multistage SEIR model was formulated for infectious diseases with continuous age structure for each successive infectious stage during a long infective period in [26]. The proposed model could describe disease progression through multiple infectious stages as that in the cases of HIV, hepatitis B, and hepatitis C. Mathematical analysis showed that the global dynamics were completely determined by the basic reproductive number. The basic reproduction number R_0 is a threshold parameter for a disease transmission model. Definitions and stability conditions about the equilibria for compartmental models of disease transmission are presented in [28]. It is shown that, if $R_0 < 1$, then the disease free equilibrium is locally asymptotically stable; whereas if $R_0 > 1$, then it is unstable. In this report, stability about the equilibrium points will be analyzed based on the basic reproduction number.

Although there have been many fractional order models for describing the dynamics of infectious diseases, few existing studies have investigated the multistage fractional infectious models on complex networks. For the normal infectious diseases, according to their realistic transmission characteristic, there are four infectious stages, namely, the incubation, early, middle, and late stages, respectively. To more accurately describe the transmission dynamics of infectious diseases, in this report, a multistage fractional SIR network model is generated for the epidemic spread on the network. This model effectively illustrates the transmission rules between different states. The stability of disease-free equilibrium and endemic equilibrium are also analyzed in detail via Lyapunov stability theorem and the next generation matrix method, respectively.

The organization of this manuscript is as follows. In Section 2, the multistage SIR fractional disease model is described. The basis reproduction

number is provided in Section 3. The stability of the disease-free equilibrium point and the endemic equilibrium point are separately analyzed in detail in Section 4. In Section 5, an example is shown to illustrate the effectiveness of the theoretical results. Finally, a conclusion is given in Section 6.

2. Model description

Suppose a node with degree k in a network has three different states S_k, $I_{ik}(i=1,2,3,4)$, R_k ($k=1,2,\cdots,n$), which represent the relative densities of the susceptible, infected and recovered nodes. I_{1k} denotes the nodes in the incubation stage which are infective but show no disease symptoms, I_{2k} denotes the nodes in the early stage which show mild symptoms of illness, I_{3k} denote the nodes in the middle stage which show severe symptoms of illness, I_{4k} denote the nodes in the late stage which show mild symptoms of illness and will recover, respectively.

Consider the following multistage fractional-order SIR network model:

$$\begin{aligned}
&D^{\alpha}S_k = A-\beta_1 kS_k\theta(t)+\delta R_k-d_0S_k,\\
&D^{\alpha}I_{1k} = \beta_1 kS_k\theta(t)-\beta_2I_{1k}-\gamma_1I_{1k}-d_0I_{1k}-d_1I_{1k},\\
&D^{\alpha}I_{2k} = \beta_2I_{1k}-\beta_3I_{2k}-\gamma_2I_{2k}-d_0I_{2k}-d_2I_{2k},\\
&D^{\alpha}I_{3k} = \beta_3I_{2k}-\beta_4I_{3k}-\gamma_3I_{3k}-d_0I_{3k}-d_3I_{3k},\\
&D^{\alpha}I_{4k} = \beta_4I_{3k}-\gamma_4I_{4k}-d_0I_{4k}-d_4I_{4k},\\
&D^{\alpha}R_k = \gamma_1I_{1k}+\gamma_2I_{2k}+\gamma_3I_{3k}+\gamma_4I_{4k}-\delta R_k-d_0R_k,
\end{aligned} \tag{1}$$

where D^{α} denotes the Caputo fractional differential ($0<\alpha\le 1$), $\theta(t)=\dfrac{\sum_{k=1}^{n}kP(k)I_k(t)}{\langle k\rangle}$ gives the probability that a randomly chosen link emanating from a node leads to an infected node in a network with $I_k=I_{1k}+I_{2k}+I_{3k}+I_{4k}$. $\langle k\rangle=\sum_{k=1}^{n}kP(k)$ denotes the average degree of the network, $P(k)$ is the degree distribution, which means the occurrence probability of nodes of degree k. A is a constant recruitment rate, d_0 is the natural death rate. d_1, d_2, d_3, d_4 are the death rates on account of disease at the four infectious stages, respectively. β_1, β_2, β_3,

β_4 are the corresponding the infectious rates and γ_1, γ_2, γ_3, γ_4 the recovery rates, which are depicted in Fig.1.

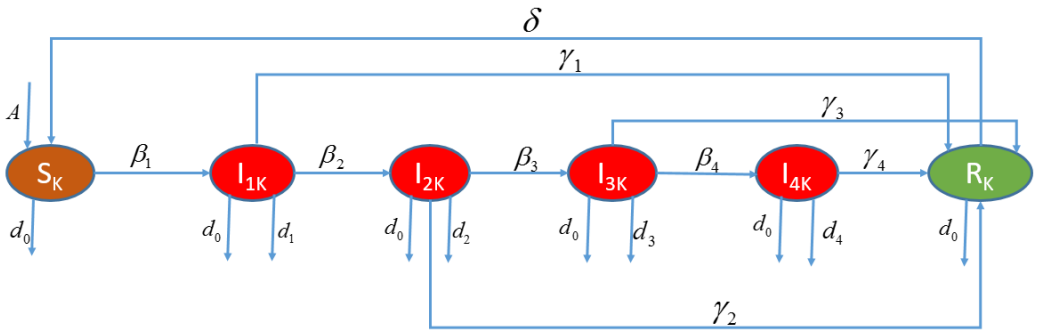

Fig.1 Flows diagram of disease transmission

3. Basic reproduction number

The disease-free equilibrium of model (1) is

$$E^0 = (S_0, I_{10}, I_{20}, I_{30}, I_{40}, R_0) = (\frac{A}{d_0}, 0, 0, 0, 0, 0).$$

Letting the right hand of model (1) be zero, we see that its endemic equilibrium $E^* = (S_k^*, I_{1k}^*, I_{2k}^*, I_{3k}^*, I_{4k}^*, R_k^*)$ is determined by the following algebraic equations:

$$S_k^* = \frac{A + \delta R_k^*}{\beta_1 k\theta^* + d_0},$$

$$I_{1k}^* = \frac{\beta_1 k\theta^*}{\beta_2 + \gamma_1 + d_1 + d_0} S_k^* = \frac{\beta_1 k\theta^*}{B_1} S_k^*,$$

$$I_{2k}^* = \frac{\beta_2}{\beta_3 + \gamma_2 + d_2 + d_0} I_{1k}^* = \frac{\beta_2}{B_2} I_{1k}^*,$$

$$I_{3k}^* = \frac{\beta_3}{\beta_4 + \gamma_3 + d_3 + d_0} I_{2k}^* = \frac{\beta_3}{B_3} I_{2k}^*,$$

$$I_{4k}^* = \frac{\beta_4}{\gamma_4 + d_4 + d_0} I_{3k}^* = \frac{\beta_4}{B_4} I_{3k}^*,$$

$$(\delta + d_0) R_k^* = \gamma_1 I_{1k}^* + \gamma_2 I_{2k}^* + \gamma_3 I_{3k}^* + \gamma_4 I_{4k}^*.$$

$$A - d_0(S_k^* + R_k^*) = (d_0 + d_1) I_{1k}^* + (d_0 + d_2) I_{2k}^* + (d_0 + d_3) I_{3k}^* + (d_0 + d_4) I_{4k}^*,$$

then

$$S_k^* = \frac{\dfrac{A}{\beta_1 k\theta^*(t)+d_0}}{1-\dfrac{\delta}{\beta_1 k\theta^*(t)+d_0}\cdot\dfrac{\gamma_1+\gamma_2\dfrac{\beta_2}{B_2}+\gamma_3\dfrac{\beta_3}{B_3}\dfrac{\beta_2}{B_2}+\gamma_4\dfrac{\beta_4}{B_4}\dfrac{\beta_3}{B_3}\dfrac{\beta_2}{B_2}}{d_0+\delta}\cdot\dfrac{\beta_1 k\theta^*(t)}{B_1}}, \tag{2}$$

$$I_{1k}^* = \frac{A\beta_1 k\theta^*(t)}{B_1(\beta_1 k\theta^*(t)+d_0)(1-\dfrac{\delta}{\beta_1 k\theta^*(t)+d_0}\cdot\dfrac{\Omega}{d_0+\delta}\cdot\dfrac{\beta_1 k\theta^*(t)}{B_1})} \tag{3}$$

with $\Omega = \gamma_1+\gamma_2\dfrac{\beta_2}{B_2}+\gamma_3\dfrac{\beta_3}{B_3}\dfrac{\beta_2}{B_2}+\gamma_4\dfrac{\beta_4}{B_4}\dfrac{\beta_3}{B_3}\dfrac{\beta_2}{B_2}$.

Therefore, substituting equations (2) and (3) into $\theta(t)$, we have

$$\theta^*(t) = \frac{\sum_{k=1}^{n} kP(k)I_k^*(t)}{\langle k\rangle}$$

$$= \frac{\sum_{k=1}^{n} kP(k)\dfrac{A\beta_1 k\theta^*(t)}{B_1(\beta_1 k\theta^*(t)+d_0)(1-\dfrac{\delta}{\beta_1 k\theta^*(t)+d_0}\cdot\dfrac{\Omega}{d_0+\delta}\cdot\dfrac{\beta_1 k\theta^*(t)}{B_1})}}{\langle k\rangle}$$

Let $\theta^*(t) = f(\theta^*)$, where $f(\bullet)$ is a continuous and differentiable function, it is easy to see that $f(0)=0$, $f(1)<1$. Thus, nonzero $\theta^*\in(0,1)$ exists if $f'(0)>1$, which leads to

$$f'(\theta)\big|_{\theta=0} = \frac{(1+\dfrac{\beta_2}{B_2}+\dfrac{\beta_3}{B_3}\dfrac{\beta_2}{B_2}+\dfrac{\beta_4}{B_4}\dfrac{\beta_3}{B_3}\dfrac{\beta_2}{B_2})A\beta_1\langle k^2\rangle}{B_1 d_0\langle k\rangle}.$$

Define the basic reproduction number as follows

$$R_0 = \frac{(1+\dfrac{\beta_2}{B_2}+\dfrac{\beta_3}{B_3}\dfrac{\beta_2}{B_2}+\dfrac{\beta_4}{B_4}\dfrac{\beta_3}{B_3}\dfrac{\beta_2}{B_2})A\beta_1\langle k^2\rangle}{B_1 d_0\langle k\rangle}.$$

It is clear that if $R_0<1$, model (1) only has a disease-free equilibrium E^0, while if $R_0>1$ model (1) has a unique positive endemic equilibrium E^*.

4. Stability analysis

Theorem 1 For system (1), if $R_0 < 1$ and $\frac{\beta_1 \langle k^2 \rangle}{B_1 \langle k \rangle}(S_k^0 + \varepsilon) - 1 \le 0$, for any $\varepsilon > 0$, then the disease free equilibrium is asymptotically stable, i.e., the infection will gradually die out.

Proof:

Let

$$D^\alpha S_k = g(t) = A - \beta_1 k S_k \theta(t) + \delta R_k - d_0 S_k, \quad S_k^0 = g(0),$$
$$D^\alpha S_k^{'} = G(t) = A + \delta - d_0 S_k^{'}, \quad S_k^{'}(0) = S_k^0 + \varepsilon = G(0),$$

since $g(t) \le G(t)$, according to the comparative theorem of fractional order systems [29], then $S_k(t) \le S_k^{'}(t)$.

Additionally, $D^\alpha S_k^{'} = A + \delta R_k - d_0 S_k^{'}$ is a asymptotically stable for $S_k^{'}$, then $S_k^{'}(t) < S_k^{'}(0)$, thus $S_k(t) \le S_k^{'}(t) < S_k^{'}(0) = S_k(t) + \varepsilon$, and for any $\varepsilon > 0$, $D^\alpha I_{1k} \le \beta_1 k (S_k^0 + \varepsilon)\theta(t) - B_1 I_{1k}$.

We consider the following auxiliary system

$$D^\alpha I_{1k} = \beta_1 k (S_k^0 + \varepsilon)\theta(t) - B_1 I_{1k},$$

and choose a Lyapunov function

$$V(t) = \sum_{k=1}^{n} (\tilde{b}_1 I_{1k} + \tilde{b}_2 I_{2k} + \tilde{b}_3 I_{3k} + \tilde{b}_4 I_{4k}),$$

where $\tilde{b}_1 = b_1 b_0$, $\tilde{b}_2 = b_2 b_0$, $\tilde{b}_3 = b_3 b_0$, $\tilde{b}_4 = b_4 b_0$, $b_0 = \frac{kP(k)}{\langle k \rangle}$, then

$$\begin{aligned} D^\alpha V(t) = \sum_{k=1}^{n} \Big(& b_1 \frac{kP(k)}{\langle k \rangle} \beta_1 k (S_k^0 + \varepsilon)\theta(t) - b_1 \frac{kP(k)}{\langle k \rangle} B_1 I_{1k} + b_2 \frac{kP(k)}{\langle k \rangle} \beta_2 I_{2k} - b_2 \frac{kP(k)}{\langle k \rangle} B_2 I_{2k} \\ & + b_3 \frac{kP(k)}{\langle k \rangle} \beta_3 I_{3k} - b_3 \frac{kP(k)}{\langle k \rangle} B_3 I_{3k} + b_4 \frac{kP(k)}{\langle k \rangle} \beta_4 I_{4k} - b_4 \frac{kP(k)}{\langle k \rangle} B_4 I_{4k} \Big) \\ = \frac{b_1 \beta_1 \langle k^2 \rangle}{\langle k \rangle} & (S_k^0 + \varepsilon)\theta - \sum_{k=1}^{n} \frac{kP(k)}{\langle k \rangle} \left[b_1 B_1 I_{1k} + b_2 (B_2 - \beta_2) I_{2k} + b_3 (B_3 - \beta_3) I_{2k} + b_4 (B_4 - \beta_4) I_{2k} \right]. \end{aligned}$$

Let $b_1 = \frac{1}{B_1}, b_2 = \frac{1}{B_2 - \beta_2}, b_3 = \frac{1}{B_3 - \beta_3}, b_4 = \frac{1}{B_4 - \beta_4}$, then we can obtain that

$$D^{\alpha}V(t)=\frac{\beta_1\langle k^2\rangle}{B_1\langle k\rangle}(S_k^0+\varepsilon)\theta-\sum_{k=1}^{n}\frac{kP(k)}{\langle k\rangle}(I_{1k}+I_{2k}+I_{2k}+I_{2k})$$
$$=\theta\left[\frac{\beta_1\langle k^2\rangle}{B_1\langle k\rangle}(S_k^0+\varepsilon)-1\right].$$

Fixing $\varepsilon>0$ to be small enough such that if $\frac{\beta_1\langle k^2\rangle}{B_1\langle k\rangle}(S_k^0+\varepsilon)-1\le 0$, then $D^{\alpha}V(t)<0$ and the disease-free equilibrium is asymptotically stable.◆

Subsequently, we will analyze the stability of the endemic equilibrium point. Following the theoretical result in [28], let

$$F=\begin{pmatrix}\beta_1 kS_k\theta\\ \beta_2 I_{1k}\\ \beta_3 I_{2k}\\ \beta_4 I_{3k}\\ 0\\ 0\end{pmatrix}\le\begin{pmatrix}\beta_1 S_k\dfrac{\sum_{k=1}^{n}k^2P(k)I_k}{\langle k\rangle}\\ \beta_2 I_{1k}\\ \beta_3 I_{2k}\\ \beta_4 I_{3k}\\ 0\\ 0\end{pmatrix},\quad V=\begin{pmatrix}\beta_2 I_{1k}+B_1 I_{1k}\\ B_2 I_{2k}\\ B_3 I_{3k}\\ B_4 I_{4k}\\ -A+\beta_1 kS_k\theta(t)-\delta R_k+d_0 S_k\\ \delta R_k+d_0 R_k-\gamma_1 I_{1k}-\gamma_2 I_{2k}-\gamma_3 I_{3k}-\gamma_4 I_{4k}\end{pmatrix},$$

then

$$f=\frac{\partial F}{\partial I_{ik}}\le\begin{pmatrix}\frac{\langle k^2\rangle S_k}{\langle k\rangle}\beta_1 & \frac{\langle k^2\rangle S_k}{\langle k\rangle}\beta_1 & \frac{\langle k^2\rangle S_k}{\langle k\rangle}\beta_1 & \frac{\langle k^2\rangle S_k}{\langle k\rangle}\beta_1\\ \beta_2 & 0 & 0 & 0\\ 0 & \beta_3 & 0 & 0\\ 0 & 0 & \beta_4 & 0\end{pmatrix}$$
$$\le\begin{pmatrix}\frac{\beta_1\langle k^2\rangle}{\langle k\rangle} & \frac{\beta_1\langle k^2\rangle}{\langle k\rangle} & \frac{\beta_1\langle k^2\rangle}{\langle k\rangle} & \frac{\beta_1\langle k^2\rangle}{\langle k\rangle}\\ \beta_2 & 0 & 0 & 0\\ 0 & \beta_3 & 0 & 0\\ 0 & 0 & \beta_4 & 0\end{pmatrix},$$

$$v = \frac{\partial V}{\partial I_{ik}} = \begin{pmatrix} \beta_2 + B_1 & & & \\ & B_2 & & \\ & & B_3 & \\ & & & B_4 \end{pmatrix}.$$

The basic reproduction number R_0 is defined as the spectral radius of the matrix fv^{-1}. Let

$$fv^{-1} = \begin{pmatrix} \frac{\beta_1\langle k^2\rangle}{\langle k\rangle} & \frac{\beta_1\langle k^2\rangle}{\langle k\rangle} & \frac{\beta_1\langle k^2\rangle}{\langle k\rangle} & \frac{\beta_1\langle k^2\rangle}{\langle k\rangle} \\ \beta_2 & 0 & 0 & 0 \\ 0 & \beta_3 & 0 & 0 \\ 0 & 0 & \beta_4 & 0 \end{pmatrix} \begin{pmatrix} \beta_2 + B_1 & & & \\ & B_2 & & \\ & & B_3 & \\ & & & B_4 \end{pmatrix}^{-1}$$

$$= \begin{pmatrix} \frac{\beta_1\langle k^2\rangle}{\langle k\rangle(\beta_2 + B_1)} & \frac{\beta_1\langle k^2\rangle}{\langle k\rangle B_2} & \frac{\beta_1\langle k^2\rangle}{\langle k\rangle B_3}\frac{1}{B_3} & \frac{\beta_1\langle k^2\rangle}{\langle k\rangle B_4} \\ \frac{\beta_2}{\beta_2 + B_1} & 0 & 0 & 0 \\ 0 & \frac{\beta_3}{B_2} & 0 & 0 \\ 0 & 0 & \frac{\beta_4}{B_3} & 0 \end{pmatrix}.$$

Then the characteristic equation of matrix fv^{-1} is

$$q^4 - \frac{\beta_1\langle k^2\rangle}{\langle k\rangle}\frac{q^3}{\beta_2 + B_1} - \frac{\beta_1\langle k^2\rangle}{\langle k\rangle}\frac{\beta_2 q^2}{(\beta_2 + B_1)B_2} + \frac{\beta_1\langle k^2\rangle}{\langle k\rangle}\frac{\beta_2\beta_3\beta_4}{(\beta_2 + B_1)B_2B_3B_4} = 0. \quad (4)$$

where q_i is the eigenvalues of equation (4).

Thus, for system (1), if $\max\{|q_i|, i = 1,2,3,4\} < 1$,then the endemic equilibrium point is locally asymptotically stable. Conversely, if $\max\{|q_i|, i = 1,2,3,4\} > 1$, the endemic equilibrium point is unstable.

5. Numerical simulations

In this section, numerical simulations are present to illustrate the theoretical results above mentioned, and the finite difference method is adopted to solve the fractional equations.

Without loss of generality, for model (1), choose a BA random scale-free network size $n = 400, k = 15, P(j) = 2j^{-3}$, and the parameters are set $A = 0.002$, $d_0 = 0.001$, $d_1 = 0.003$, $d_2 = 0.002$, $d_3 = 0.001$, $d_4 = 0.001$, $\beta_1 = 0.001$, $\beta_2 = 0.002$, $\beta_3 = 0.003$, $\beta_4 = 0.004$, $\gamma_1 = 0.25$, $\gamma_2 = 0.27$, $\gamma_3 = 0.21$, $\gamma_4 = 0.20$, $\delta = 0.02$. The initial condition is [0.52, 0.038, 0.032, 0.035, 0.031, 0.34].

We can calculate that the basic reproduction number is 0.0315<1, thus there exists only a disease-free equilibrium. From Fig. 2, we can find that the disease-free equilibrium point is global asymptotic stable. Even more, Fig. 3 shows that the states at every infective stage are converged to zero, which means that the disease will disappear ultimately. From the above, we can conclude that the theoretical results are correct and the simulation results are effective .

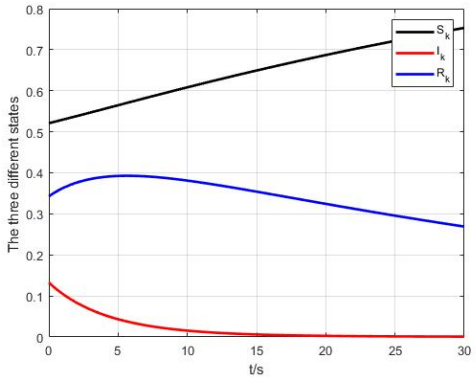

Fig.2. The graphical results for model (1) when $\alpha = 0.98$

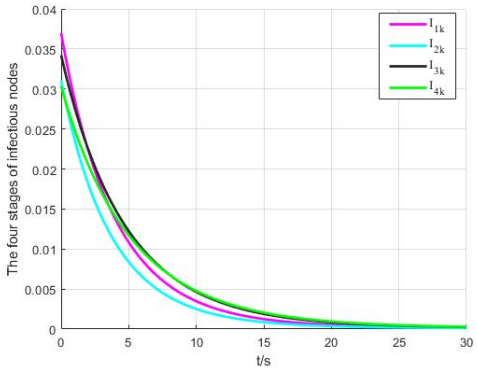

Fig.3. The graphical results for the disease at every stage $\alpha = 0.98$

6. Conclusions

In this article, a multistage fractional SIR complex network model has been provided. Based on the basic reproduction number, the stability of the disease-free equilibrium point has been analyzed. When $R_0 < 1$,there only exists

an asymptotically stable disease free equilibrium, which means that the disease will become extinct ultimately regardless of the initial density of the infected individuals. Furthermore, the stability of the endemic equilibrium point has also been studied via the next generation matrix method, and some results have been derived. Finally, numerical simulations were presented to illustrate the correctness of the theoretical results. Note that since the multistage characteristic has great influence on the propagation dynamics, there may be a need to further construct specific models for certain specific infectious diseases, e.g., cholera, flu, HIV, etc., which shall be of our future research interest.

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