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Dynamics and Event-Triggered Impulsive Control of a Fractional-Order Epidemic Model with Time Delay

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Abstract: Due to the lack of timely protection measures against infectious diseases, or based on the particularity of the transmission of some infectious diseases and the time-varying connections between people, the transmission dynamics of infectious diseases in the information society are becoming more and more complex and changeable. A fractional-order epidemic mathematical model with network weighting and latency is proposed in this paper, and the stability near the disease-free equilibrium point and endemic equilibrium point are discussed separately. Subsequently, an event-triggered impulsive control strategy based on an infection rate threshold is put forward. By selecting the appropriate control gain, the Zeno phenomenon can be eliminated on the premise of ensuring the stability of the control error system. Finally, the theoretical results were validated numerically and some conclusions are presented. These findings contribute to future research on the limited-time event-triggered impulsive control of infectious diseases.

Keywords: fractional-order epidemic network model; equilibrium point; stability analysis; event-triggered impulsive control

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1. Introduction

The development of human society has been threatened by a multitude of infectious diseases. From the smallpox virus in its early stages to recent outbreaks, such as bird flu, AIDS, and COVID-19, the emergence of large-scale infectious viruses consistently inflicts significant damage upon human life and property. These successive tragedies have served as a wake-up call for researchers to engage in continuous investigations of various infectious viruses. These investigations play a crucial guiding role in formulating epidemic prevention and control policies to study the process of virus transmission, construct a mathematical model that can describe the dynamic behavior of infectious disease transmission, and even reveal its transmission mechanism.

The most typical work undertaken to study the dynamic behavior of infectious disease spread in networks is to construct mathematical differential equations for infectious diseases. The most representative result is the models established by Pastor-Satorras and Vespignani [1] based on the average field theory, namely, SIS and SIR models. Later, researchers further studied the stability and transmission threshold of SIS epidemic models with non-monotonic morbidity [2,3], multiple pathogenic strains [4], infectious agents [5], and cure functions [6].

With the in-depth study of the law of epidemic transmission, scholars found that some people do not quickly appear infected after contact with infected persons, but after some time, they become sick and infected. We often refer to this phenomenon as the incubation period. Due to the latent nature of some diseases, researchers also proposed time-delay infectious disease network models [7–10] and analyzed the influence of infection delay on transmission threshold and bifurcation dynamic behavior caused by time delay in detail. In addition, since people's awareness of epidemic information influences disease transmission

to a large extent, studies showed that the more individuals acquire disease transmission information, the fewer the number of patients will be [11–14]. In conclusion, this kind of incubation period without infectious disease (infection delay) has a great impact on the dynamic behavior of the disease transmission. It can generate more complex dynamic behavior, such as periodic oscillation, bifurcation, or chaos, which is of great significance in both mathematical theory and biology.

Moreover, with the development of research, it was found that the link strength between individuals can seriously influence epidemic transmission. The stronger the connection between two nodes, the more susceptible people become infected and the faster information about the disease becomes available to unknown people. In [15–18], some methods are provided to assess the spread of disease in weighted networks. An improved epidemiological SIS model based on adaptive weighted networks is proposed in [19], showing that as the closeness between individuals decreases, the disease will fade away. A new method based on edge weights is proposed to estimate prevalence thresholds and prevalence scales on networks with generality and weight distributions in [20]. It was found in [21] that the weights can promote epidemic transmission by expanding the basic reproduction number, and for different network structures, the impact of the internal infection rate on epidemic prevalence is greater than that of the cross-infection rate. Therefore, in order to realistically simulate the epidemic transmission process, in this study, we constructed an epidemic mathematical model on weighted networks and considered the impacts of link strength values on disease transmission.

With the rapid development of fractional-order differential equations, many researchers have tried to use fractional differential equation modeling. A fractional-order differential equation has the memory characteristic, that is, the fractional order system not only considers the current moment state but also the previous moment state. The most prominent characteristic of the immune system in the human body is memory; therefore, it is more realistic to apply fractional differential equations to model the epidemic transmission dynamics. Applying fractional-order Lyapunov stability theory, researchers studied the stability of an HIV/AIDS fractional-order infectious disease model [22], COVID-19 fractional-order infectious disease model [23], and Caputo fractional-order SIS/SIR model [24,25]. Meanwhile, a novel parameter estimation method based on improved particle swarm optimization was proposed [26], which was extended to various fractional infectious disease models. It can be found from the above research results that fractional-order models are very suitable for simulating epidemic transmission dynamics. Thus, this study intended to establish a fractional-order network epidemic model with time delay. The effects of time delay and fractional-order parameters on disease transmission dynamics were studied, especially the complex dynamic behaviors caused by time delay, which can be a good supplement to the existing research.

Impulsive control is a kind of discontinuous control method that can make the system state change suddenly. It has good application prospects in a system that cannot bear continuous control input or cannot provide continuous control input. It is well known that traditional impulsive control is triggered by time, and the information sampling and transmission of the system are frequently carried out, which may lead to the waste of system resources. Event-triggered impulsive control is a much more effective control method that can overcome this shortcoming. Compared with general impulsive control, the impulsive time of the event-triggered impulsive control is determined by the preset conditions related to the system's state, which makes the impulsive control strategy more flexible. Meanwhile, the control input of the event-triggered impulsive control is only determined by some event-triggered conditions related to the system's state. Due to many factors that affect the transmission characteristics of infectious diseases, the applications of an event-triggering mechanism in the research field of infectious diseases are relatively few [27,28]. In study [27], although the application of event-triggering control to the SIS infectious disease model achieved effective control, it only considered the event triggering based on a single parameter, namely, infection rate. This study intended to propose a

control method based on a single-factor event-triggering mechanism, taking the infection rate threshold as the trigger condition and selecting appropriate control gains to achieve effective disease control.

This paper is arranged as follows. Definitions of Caputo fractional calculus, some important lemmas, and model descriptions are given in Section 2. In Section 3, the stability of the equilibrium of the fractional-order infectious system with time delay is analyzed in detail. Section 4 proposes an event-triggered impulsive control method and analyzes the controlled system's dynamical behavior. In Section 5, a numerical example is shown to illustrate the correctness of the above theoretical results. Some conclusions are provided in Section 6, and some advice about further research is also given.

2. Preliminaries and Model Description

First, a definition of Caputo fractional-order calculus and some important lemmas are provided. Subsequently, descriptions of a fractional-order infectious model with time delay are introduced.

2.1. Preliminaries

Definition 1 [29]. *Suppose that* $k > 0; t > t_0 > 0;$ *and* $k,\ t_0,\ t \in R$. *The Caputo fractional derivative is given by*

$$ {}_{t_0}^{c}D_t^k f(t) = \frac{1}{\Gamma(n-k)} \int_{t_0}^{t} \frac{f^n(\zeta)}{(t-\zeta)^{k+1-n}} d\zeta,\ n-1 < k < n,\ n \in N^+, $$

where t_0 *denotes the initial time and* $\Gamma(\bullet)$ *is the gamma function.*

Remark 1. *For a smooth function* $f = f(t)$, *its Caputo fractional-order derivative is given by*

$$ {}_{t_0}^{c}D_t^k f(t) = \frac{1}{\Gamma(1-k)} \int_{t_0}^{t} \frac{f^n(\zeta)}{(t-\zeta)^{k}} d\zeta, $$

where $0 < k < 1$ *is the fractional-order parameter.*

For simplicity, in this paper, the Caputo fractional derivative ${}_{t_0}^{c}D_t^k$ *is always rewritten as* D^k.

Lemma 1 [30]. *If there exists a continuous* $\Phi(t)$ *that satisfies* $D^k\Phi(t) \le s\Phi(t)$ *for* $t \ge t_0$, *then*

$$ \Phi(t) \le \Phi(t_0) E_k[s(t-t_0)^k], $$

where $0 < k < 1$ *and* s *is a constant.*

Lemma 2 [31]. *For* $0 < k < 1$ *and* $t \ge t_0$, $0 \le E_k[w(t-t_0)^k] \le 1$ *holds if* $w < 0$.

Lemma 3 [32]. *If the function* $\varphi(t) \in R^n$ *is derivable, then* $D^k(\varphi^T(t)\varphi(t)) \le 2\varphi^T(t)D^k\varphi(t)$ *holds for* $\forall t \ge t_0$ *and* $0 < k < 1$.

Lemma 4 [33]. *For* $u, v \in R^n$ *and* $m > 0$, *then* $2u^Tv \le \frac{1}{m}u^Tu + kv^Tv$ *holds.*

Lemma 5 [34]. *For a fractional-order time-delay system* $D^k x(t) = \phi(x(t), x(t-\tau))$, *where* $\phi(\bullet) \in R^n$ *is a nonlinear function that satisfies the Lipschiz condition,* $0 < k < 1$, $x(t) \in R^n$, *and* $\tau \in R$ *is a constant. If a positive definite matrix* P *and a positive semi-definite matrix* Q *exist, and the following condition*

$$ x^T(t)PD^k x(t) + x^T(t)Qx(t) - x^T(t-\tau)Qx(t-\tau) \le 0 $$

is satisfied, then the system is Lyapunov stable.

2.2. Model Descriptions

In an epidemic network, nodes can be classified into three classes: susceptible nodes (S), infected nodes (I), and recovery nodes (R). Figure 1 illustrates their transmission law. a represents the constant population input rate, d represents the natural mortality rate of the population, β denotes the infection rate of susceptible individuals, α denotes the recovery rate of infected individuals, and γ represents the probability from recovery to susceptibility.

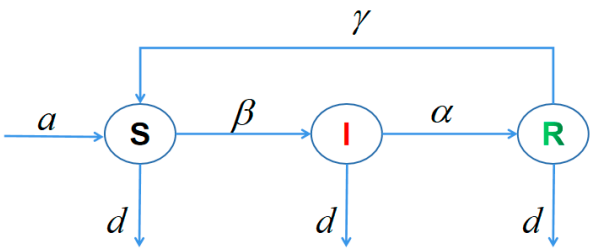

Figure 1. Propagation law between nodes between nodes.

In a social network, the connection weight between node i and another node j is denoted as ω_{ij}. If there is a connection between node i and node j, then $\omega_{ij} \neq 0$; conversely, if there is no connection, then $\omega_{ij} = 0$. In this paper, it should be noted that we only concentrate on undirected networks, which means $\omega_{ij} = \omega_{ji}$. Previous research showed that the weight of nodes plays a significant role in disease transmission.

From Figure 1, it can be observed that the susceptibility of a node i to infection by its neighboring infected nodes is $\lambda_I = 1 - (1-\beta)^{\omega_{ij}}$, as determined by the weight assigned to each connection in the social network. In cases where multiple infected nodes p are present among their neighbors, the total probability of infection is $1 - \prod_{j=1}^{p}(1-\lambda_I)$, which is calculated as a product of these individual probabilities. Based on this disease transmission relationship, a fractional-order SIR network model is proposed in the following:

$$\begin{aligned} D^k S_i(t) &= a - dS_i(t) - \left[1 - \prod_{j=1}^{p}(1-\lambda_I)\right] S_i(t) I_i(t) + \gamma R_i(t), \\ D^k I_i(t) &= -dI_i(t) + \left[1 - \prod_{j=1}^{p}(1-\lambda_I)\right] S_i(t) I_i(t) - \alpha I_i(t), \\ D^k R_i(t) &= \alpha I_i(t) - dR_i(t) - \gamma R_i(t). \end{aligned} \tag{1}$$

It is important to note that for certain diseases, there exists a latency period before an individual becomes infected. Consequently, assuming the presence of this incubation period $\tau \geq 0$, model (1) can be appropriately adjusted to incorporate a time-delayed SIR epidemiological model:

$$\begin{aligned} D^k S_i(t) &= a - dS_i(t) - \left[1 - \prod_{j=1}^{p}(1-\lambda_I)\right] S_i I_i(t-\tau) + \gamma R_i(t), \\ D^k I_i(t) &= -dI_i(t) + \left[1 - \prod_{j=1}^{p}(1-\lambda_I)\right] S_i(t) I_i(t-\tau) - \alpha I_i(t), \\ D^k R_i(t) &= \alpha I_i(t) - dR_i(t) - \gamma R_i(t). \end{aligned} \tag{2}$$

where $0 \leq \tau$ and the initial condition is $[S_i(\eta),\ I_i(\eta),\ R_i(\eta)]$, where $\eta \in [-\tau, 0]$.

3. Stability Analysis of the Equilibrium Points

3.1. The Existence and Boundedness of Solutions

For simplicity, by letting $\widetilde{\beta} = 1 - \prod_{j=1}^{p}(1-\lambda_I)$, system (2) can be transformed to

$$\begin{aligned} D^k S_i(t) &= a - dS_i(t) - \widetilde{\beta} S_i(t) I_i(t-\tau) + \gamma R_i(t), \\ D^k I_i(t) &= -dI_i(t) + \widetilde{\beta} S_i(t) I_i(t-\tau) - \alpha I_i(t), \\ D^k R_i(t) &= \alpha I_i(t) - dR_i(t) - \gamma R_i(t). \end{aligned} \tag{3}$$

For system (3), there always exists a disease-free equilibrium point $E_0 = (\frac{a}{d}, 0, 0)$. When $\tau = 0$, system (3) can be written as

$$\begin{aligned} D^k S_i(t) &= a - dS_i(t) - \widetilde{\beta} S_i(t) I_i(t) + \gamma R_i(t), \\ D^k I_i(t) &= -dI_i(t) + \widetilde{\beta} S_i(t) I_i(t) - \alpha I_i(t), \\ D^k R_i(t) &= \alpha I_i(t) - dR_i(t) - \gamma R_i(t). \end{aligned}$$

Based on the next-generation matrix method [35], we can calculate the basic reproduction number $R_0 = \frac{a\widetilde{\beta}}{d(d+\alpha)}$.

For system (3), the endemic equilibrium point $E^* = (S_i^*, I_i^*, R_i^*)$ is presented as follows:

$$S_i^* = \frac{d+\alpha}{1-\prod_{j=1}^{p}(1-\lambda_I)} = \frac{d+\alpha}{\widetilde{\beta}}, I_i^* = \frac{d+\gamma}{\widetilde{\beta}} \cdot \frac{a\widetilde{\beta} - d(d+\alpha)}{d^2 + d\alpha + d\gamma}, R_i^* = \frac{\alpha}{\widetilde{\beta}} \cdot \frac{a\widetilde{\beta} - d(d+\alpha)}{d^2 + d\alpha + d\gamma}.$$

It is obvious that $S_i^* > 0$. When $R_0 = \frac{a\widetilde{\beta}}{d(d+\alpha)} > 1$, then $I_i^* > 0$, $R_i^* > 0$, and there exists a unique endemic equilibrium point E^* for system (2). When $R_0 = \frac{a\widetilde{\beta}}{d(d+\alpha)} < 1$, then $I_i^* < 0$, $R_i^* < 0$, and system (2) has no unique endemic equilibrium point.

Let $N_i(t) = S_i(t) + I_i(t) + R_i(t)$; from Equation (3), this yields

$$D^k N_i(t) = a - dN_i(t). \tag{4}$$

Applying the Laplace transform, Equation (4) can be transformed to

$$s^k L[N_i(t)] - s^{k-1} N_i(0) = \frac{a}{s} - dL[N_i(t)]. \tag{5}$$

Thus, we can obtain that

$$N_i(s) = \frac{s^k N_i(0) + a}{s^{k+1} + sd} = \frac{s^{k-1} N_i(0)}{s^k + d} + \frac{a}{s^{k+1} + sd}. \tag{6}$$

Applying the inverse Laplace transform, Equation (6) yields

$$N_i(t) = E_{k,1}(-dt^k) N_i(0) + aE_{0,1}(-dt^0). \tag{7}$$

Since $\lim_{t\to\infty} E_{k,1}(-dt^k) = 0$ and $E_{0,1}(-dt^0) = \frac{1}{1+d}$, then from (7), we can obtain that

$$\lim_{t\to\infty} N_i(t) = \frac{a}{1+d},$$

which means that the solutions of Equation (3) are bounded.

3.2. Stability Analysis of the Equilibrium

A. Stability analysis of disease-free equilibrium point

For system (3), its linearization equation at the equilibrium point is

$$\begin{aligned} D^k S_i(t) &= (-d-\widetilde{\beta}I_i^*)S_i(t) - \widetilde{\beta}S_i^* I_i(t-\tau) + \gamma R_i(t), \\ D^k I_i(t) &= \widetilde{\beta}I_i^* S_i(t) + \widetilde{\beta}S_i^* I_i(t-\tau) - (d+\alpha)I_i(t), \\ D^k R_i(t) &= \alpha I_i(t) - dR_i(t) - \gamma R_i(t). \end{aligned} \tag{8}$$

Applying the Laplace transform yields

$$\begin{aligned} \lambda^k Y_1(\lambda) &= (-d-\widetilde{\beta}I_i^*)_1(\lambda) + \lambda^{k-1}\phi_1(0) - \widetilde{\beta}S_i^* e^{-\lambda\tau}(Y_2(\lambda) + \textstyle\int_{-\tau}^{0} e^{-\lambda\tau}\phi_1(t)dt) + Y_3(\lambda), \\ \lambda^k Y_2(\lambda) &= \widetilde{\beta}I_i^* Y_1(\lambda) + \widetilde{\beta}S_i^* e^{-\lambda\tau}(Y_2(\lambda) + \textstyle\int_{-\tau}^{0} e^{-\lambda\tau}\phi_2(t)dt) - \Big[(d+\alpha)Y_2(\lambda) + \lambda^{k-1}\phi_2(0)\Big], \\ \lambda^k Y_3(\lambda) &= \alpha Y_2(\lambda) - \Big[(d+\gamma)Y_3(\lambda) + \lambda^{k-1}\phi_3(0)\Big]. \end{aligned} \tag{9}$$

For Equation (9), let

$$B(\lambda)\begin{pmatrix} Y_1(\lambda) \\ Y_2(\lambda) \\ Y_3(\lambda) \end{pmatrix} = \begin{pmatrix} \psi_1(\lambda) \\ \psi_2(\lambda) \\ \psi_3(\lambda) \end{pmatrix},$$

Then, we can obtain that

$$B(\lambda) = \begin{pmatrix} \lambda^\alpha + d + \widetilde{\beta}I_i^* & \widetilde{\beta}S_i^* e^{-\lambda\tau} & -\gamma \\ -\widetilde{\beta}I_i^* & \lambda^\alpha - \widetilde{\beta}S_i^* e^{-\lambda\tau} + d + \alpha & 0 \\ 0 & -\alpha & \lambda^\alpha + d + \gamma \end{pmatrix},$$

and its characteristic polynomial is

$$\det(B(\lambda)) = (\lambda^k + d + \widetilde{\beta}I_i^*)(\lambda^k - \widetilde{\beta}S_i^* e^{-\lambda\tau} + d + \alpha)(\lambda^k + d + \gamma) - \alpha\gamma\widetilde{\beta}I_i^* + \widetilde{\beta}I_i^*(\lambda^k + d + \gamma)\widetilde{\beta}S_i^* e^{-\lambda\tau}. \tag{10}$$

If $\tau = 0$, Equation (9) can be transformed to

$$\det(B(\lambda)) = (\lambda^k + d + \widetilde{\beta}I_i^*)(\lambda^k - \widetilde{\beta}S_i^* + d + \alpha)(\lambda^k + d + \gamma) - \alpha\gamma\widetilde{\beta}I_i^* + \widetilde{\beta}I_i^*(\lambda^k + d + \gamma)\widetilde{\beta}S_i^*.$$

Since the disease-free equilibrium is $E_0 = (\frac{a}{d}, 0, 0)$, then

$$\det(B(\lambda)) = (\lambda^k + d)(\lambda^k - \widetilde{\beta}\frac{a}{d} + d + \alpha)(\lambda^k + d + \gamma).$$

Therefore, if $R_0 < 1$, the roots of $\det(B(\lambda))$ are all negative. Hence, when $\tau = 0$, system (3) is asymptotically stable at the disease-free equilibrium point.

If $\tau > 0$, let $\lambda = \delta i$; then, Equation (9) can be transformed to

$$\det(B(\lambda)) = ((\delta i)^k + d)((\delta i)^k - \widetilde{\beta}\frac{a}{d}e^{-\delta\tau i} + d + \alpha)((\delta i)^k + d + \gamma) = 0 \tag{11}$$

Separating the real and imaginary parts of Equation (11) produces

$$\begin{aligned} (-1)^k\delta^{2k} &= (2d+\gamma)(d+\alpha+m\cos\delta\tau) + d(d+\gamma), \\ (-\delta^2)^k &= -\tfrac{d(d+\gamma)(d+\alpha+m\cos\delta\tau)}{d+d+r+d+\alpha+m\cos\delta\tau}, \\ m\sin\delta\tau(-\delta^2)^k &+ d(d+\gamma)\sin\delta\tau = 0, \\ m\sin\delta\tau(d+\gamma)\delta^k &+ d\sin\delta\tau\delta^k = 0, \end{aligned}$$

with $m = \widetilde{\beta}\frac{a}{d}$.

Consequently, $\sin\delta\tau = 0$ and $\cos\delta\tau = \pm 1$. Then,

$$-\frac{d(d+\gamma)(d+\alpha+m\cos\delta\tau)}{d+d+r+d+\alpha+m\cos\delta\tau} = (2d+\gamma)(d+\alpha+m\cos\delta\tau) + d(d+\gamma).$$

Thus, we can conclude that $\cos \delta\tau = -1$ and

$$(-\delta^2)^k = -\frac{d(d+\gamma)(d+\alpha-\widetilde{\beta}\frac{a}{d})}{d+d+r+d+\alpha+m\cos\delta\tau}.$$

Therefore, if $R_0 = \frac{a\widetilde{\beta}}{d(\alpha+d)} < 1$, then $(-\delta^2)^k = -\frac{d(d+\gamma)(d+\alpha-\widetilde{\beta}\frac{a}{d})}{d+d+r+d+\alpha+m\cos\delta\tau} < 0$; therefore, the roots of (11) have a negative real part and the system is stable.

If $R_0 = \frac{a\widetilde{\beta}}{d(\alpha+d)} > 1$,then $(-\delta^2)^k = -\frac{d(d+\gamma)(d+\alpha-\widetilde{\beta}\frac{a}{d})}{d+d+r+d+\alpha+m\cos\delta\tau} < 0$; similarly, the roots of Equation (10) have a positive real part and the system is unstable.

From the above arguments, we have the following theorem.

Theorem 1. *For system (3), the disease-free equilibrium point* $E_0 = (\frac{a}{d}, 0, 0)$ *is locally stable when* $R_0 = \frac{a\widetilde{\beta}}{d(\alpha+d)} \leq 1$ *and unstable when* $R_0 = \frac{a\widetilde{\beta}}{d(\alpha+d)} > 1$.

B. Stability analysis of the endemic equilibrium point

When $\tau = 0$, at the endemic equilibrium $E^* = (S_i^*, I_i^*, R_i^*)$, Equation (10) can be transformed to

$$\begin{aligned}\det(B(\lambda)) &= (\lambda^k + d + \widetilde{\beta}I_i^*)(\lambda^k - \widetilde{\beta}S_i^* + d + \alpha)(\lambda^k + d + \gamma) - \alpha\gamma\widetilde{\beta}I_i^* + \widetilde{\beta}I_i^*(\lambda^k + d + \gamma)\widetilde{\beta}S_i^* \\ &= (\lambda^k)^3 + Q_1(\lambda^k)^2 + Q_2\lambda^k + Q_3,\end{aligned}$$

with $p = \widetilde{\beta}I_i^*$, $q = \widetilde{\beta}S_i^*$,

$$\begin{aligned}Q_1 &= 3d + p + \alpha + \gamma - q, \\ Q_2 &= (2d + p + \alpha - q)(d + \gamma) + (d + \alpha - q)(d + p)d + p(d + \gamma)q, \\ Q_3 &= (d + \alpha - q)(d + p)(d + \gamma) - \alpha\gamma p + p(d + \gamma)q.\end{aligned}$$

According to the Routh–Hurwitz stability criteria, if $\alpha\widetilde{\beta} - d(d + \alpha) > 0$, e.g., $R_0 = \frac{a\widetilde{\beta}}{d(\alpha+d)} > 1$, then $Q_1 > 0$, $Q_2 > 0$, $Q_3 > 0$, and $Q_1Q_2 - Q_3 > 0$. That is to say, the roots of $\det(B(\lambda)) = 0$ all have a negative real part. Therefore, there exists an endemic equilibrium for system (2) and the system is locally stable at the endemic equilibrium when $\tau = 0$.

When $\tau > 0$, Equation (10) can be transformed to

$$\begin{aligned}\det(B(\lambda)) = (\lambda^k + d + \widetilde{\beta}I_i^*)(\lambda^k - \widetilde{\beta}S_i^*e^{-\lambda\tau} + d + \alpha)(\lambda^k + d + \gamma) - \alpha\gamma\widetilde{\beta}I_i^* + \widetilde{\beta}I_i^*(\lambda^k + d + \gamma)\widetilde{\beta}S_i^*e^{-\lambda\tau} \\ = (\lambda^k)^3 + Q_1(\lambda^k)^2 + Q_2\lambda^k + Q_3,\end{aligned}$$

with $\widetilde{p} = \widetilde{\beta}I_i^*$, $\widetilde{q} = \widetilde{\beta}S_i^*e^{-\lambda\tau}$,

$$\begin{aligned}\widetilde{Q}_1 &= 3d + p + \alpha + \gamma - q, \\ \widetilde{Q}_2 &= (2d + p + \alpha - q)(d + \gamma) + (d + \alpha - q)(d + p)d + p(d + \gamma)q, \\ \widetilde{Q}_3 &= (d + \alpha - q)(d + p)(d + \gamma) - \alpha\gamma p + p(d + \gamma)q.\end{aligned}$$

For system (3), by applying Lemma 5 and letting $x_i = (S_i,\ I_i,\ R_i)^T$, $P = \begin{pmatrix} 1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{pmatrix}$, and $Q = \begin{pmatrix} 0 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 0 \end{pmatrix}$, then

$$
\begin{aligned}
& x_i^T P D^k x_i + x_i^T Q x_i - x_i^T(t-\tau) Q x_i(t-\tau) \\
&= S_i D^k S_i + I_i D^k I_i + R_i D^k R_i + I_i^2 - I_i^2(t-\tau) \\
&= S_i\left[(-d-\widetilde{\beta} I_i^*)S_i - \widetilde{\beta} S_i^* I_i(t-\tau) + \gamma R_i\right] + I_i\left[\widetilde{\beta} I_i^* S_i + \widetilde{\beta} S_i^* I_i(t-\tau) - (d+\alpha) I_i\right] \\
&\quad + R_i(\alpha I_i - d R_i - \gamma R_i) + I_i^2 - I_i^2(t-\tau) \\
&\le (-d - \tfrac{1}{2}\widetilde{\beta} I_i^* - \tfrac{1}{2}\widetilde{\beta} S_i^* + \tfrac{1}{2}\gamma) S_i^2 + (\tfrac{1}{2}\widetilde{\beta} I_i^* + \tfrac{1}{2}\widetilde{\beta} S_i^* - \tfrac{1}{2}\alpha + 1 - d) I_i^2 \\
&\quad + (\tfrac{1}{2}\alpha - d - \tfrac{1}{2}\gamma) R_i^2 - I_i^2(t-\tau) \\
&\le (-d - \tfrac{1}{2}\widetilde{\beta} I_i^* - \tfrac{1}{2}\widetilde{\beta} S_i^* + \tfrac{1}{2}\gamma) S_i^2 + (\tfrac{1}{2}\widetilde{\beta} I_i^* + \tfrac{1}{2}\widetilde{\beta} S_i^* - \tfrac{1}{2}\alpha + 1 - d) I_i^2 \\
&\quad + (\tfrac{1}{2}\alpha - d - \tfrac{1}{2}\gamma) R_i^2.
\end{aligned}
$$

Thus, if $-d - \frac{1}{2}\widetilde{\beta} I_i^* - \frac{1}{2}\widetilde{\beta} S_i^* + \frac{1}{2}\gamma < 0$, $\frac{1}{2}\widetilde{\beta} I_i^* + \frac{1}{2}\widetilde{\beta} S_i^* - \frac{1}{2}\alpha + 1 - d < 0$, and $\frac{1}{2}\alpha - d - \frac{1}{2}\gamma < 0$ are satisfied, system (7) is locally stable at the endemic equilibrium $E^* = (S_i^*,\ I_i^*,\ R_i^*)$.

C. Lyapunov stability analysis of the equilibrium point

Theorem 2. *For system (3), if* $\forall P > 0,\ Q \ge 0$, *and both* $Q + \frac{1}{2}P\beta - Pd - P\alpha \le 0$ *and* $\frac{1}{2}P\beta - Q \le 0$ *are satisfied, then the equilibrium points are Lyapunov stable; if* $Q + \frac{1}{2}P\beta - Pd - P\alpha < 0$ *and* $\frac{1}{2}P\beta - Q < 0$ *are satisfied, then the equilibrium points are globally Lyapunov asymptotic stable.*

Proof. Based on Lemma 5, choose $P > 0$ and $Q \ge 0$, then,

$$
\begin{aligned}
& I_i(t) P D^k I_i(t) + I_i(t) Q I_i(t) - I_i(t-\tau) Q I_i(t-\tau) \\
&= I_i(t) P\left[-d I_i(t) + \widetilde{\beta} S_i(t) I_i(t-\tau) - \alpha I_i(t)\right] + I_i(t) Q I_i(t) - I_i(t-\tau) Q I_i(t-\tau) \\
&= (Q - Pd - P\alpha) I_i^2(t) + P\beta S_i(t) I_i(t) I_i(t-\tau) - Q I_i^2(t-\tau) \\
&\le (Q - Pd - P\alpha) I_i^2(t) + P\beta I_i(t) I_i(t-\tau) - Q I_i^2(t-\tau) \\
&\le (Q - Pd - P\alpha) I_i^2(t) + \tfrac{1}{2}P\beta (I_i^2(t) + I_i^2(t-\tau)) - Q I_i^2(t-\tau) \\
&= (Q + \tfrac{1}{2}P\beta - Pd - P\alpha) I_i^2(t) + (\tfrac{1}{2}P\beta - Q) I_i^2(t-\tau).
\end{aligned}
$$

Thus, if $Q + \frac{1}{2}P\beta - Pd - P\alpha \le 0$ and $\frac{1}{2}P\beta - Q \le 0$ are satisfied, then for system (3), the equilibrium points are Lyapunov stable; if $Q + \frac{1}{2}P\beta - Pd - P\alpha < 0$ and $\frac{1}{2}P\beta - Q < 0$ are satisfied, then the equilibrium points are globally Lyapunov asymptotic stable. □

4. Event-Triggered Impulsive Control

Event-triggered impulsive control is only activated when a specific event occurs, such as government intervention to combat an infectious disease once the number of infected individuals surpasses a predetermined threshold. In this section, we propose a single-factor event-triggered impulsive control approach to effectively manage infectious diseases by selecting an appropriate control gain, where the threshold of infection rate serves as the trigger condition.

For system (3), the following event-triggered impulsive control is suggested:

$$
\begin{aligned}
& D^k S_i(t) = a - d S_i(t) - \widetilde{\beta} S_i(t) I_i(t-\tau) + \gamma R_i(t), \\
& D^k I_i(t) = -d I_i(t) + \widetilde{\beta} S_i(t) I_i(t-\tau) - \alpha I_i(t) + U, \\
& D^k R_i(t) = \alpha I_i(t) - d R_i(t) - \gamma R_i(t).
\end{aligned} \tag{12}
$$

where

$$U = \sum_{l=1}^{\infty} [KI_i(t_l) - LI_i(t)\delta(t - t_l)], \tag{13}$$

K and L are gain coefficients, $\delta(\cdot)$ is the Dirac function, and $l \in N$. The event-triggered sequence $\{t_l\}_{l=1}^{\infty}$ satisfies $0 < t_1 < t_2 < \cdots < t_l < \cdots$, where $\lim_{l\to\infty} t_l = \infty$. $I_i(t_l) \neq 0$ denotes the given infective rate thresholds, for example, $I_i(t_l)$ maybe indicates the number of people infected at different levels of risk in the COVID-19 outbreak.

Since the Dirac function satisfies $\delta(t - t_l) = \begin{cases} +\infty, t = t_l, \\ 0, t \neq t_l, \end{cases}$ and $\int_{-\infty}^{+\infty} h(t)\delta(t - t_l)dt = h(t_l)$, then system (12) can be transformed to

$$\begin{aligned} &D^k S_i(t) = a - dS_i(t) - \widetilde{\beta} S_i(t) I_i(t - \tau) + \gamma R_i(t), \\ &D^k I_i(t) = -dI_i(t) + \widetilde{\beta} S_i(t) I_i(t - \tau) - \alpha I_i(t) + KI_i(t_l),\ t \neq t_l, \\ &\Delta I_i(t_l) = -LI_i(t_l),\ t = t_l, \\ &D^k R_i(t) = \alpha I_i(t) - dR_i(t) - \gamma R_i(t). \end{aligned} \tag{14}$$

By letting $\varepsilon(t) = I_i(t_l) - I_i(t)$, Equation (14) can be further expressed as

$$\begin{aligned} &D^k S_i(t) = a - dS_i(t) - \widetilde{\beta} S_i(t) I_i(t - \tau) + \gamma R_i(t), \\ &D^k I_i(t) = -dI_i(t) + \widetilde{\beta} S_i(t) I_i(t - \tau) - \alpha I_i(t) + K(I_i(t) + \varepsilon(t)),\ t \neq t_l, \\ &I_i(t_l^+) = (1 - L) I_i(t_l),\ t = t_l, \\ &D^k R_i(t) = \alpha I_i(t) - dR_i(t) - \gamma R_i(t). \end{aligned} \tag{15}$$

Remark 2. *The stability of the event-triggered impulsive system discussed in this paper differs from that of conventional impulsive systems. In the context of controlling infectious diseases, our primary focus should be on treating infected individuals to restore their health. Therefore, for system (15), we only need to consider the stability of the following impulsive system:*

$$\begin{aligned} D^k I_i(t) = -dI_i(t) + \widetilde{\beta} S_i I_i(t - \tau) - \alpha I_i(t) + K(I_i(t) + \varepsilon(t)),\ t \neq t_l, \\ I_i(t_l) = (1 - L) I_i(t_{l^-}),\ t = t_l. \end{aligned} \tag{16}$$

Theorem 3. *If a constant T exists such that the following inequality $T = \inf\{t_{+1} - t_l\} > 0$ holds, then system (15) under control (13) exhibits no Zeno phenomenon.*

Proof. Let $W_i(t) = \varepsilon_i^2(t)$. Since $\varepsilon(t) = I_i(t_l) - I_i(t)$, then

$$\begin{aligned} D^\alpha W_i(t) &= 2\varepsilon_i(t) D^\alpha \varepsilon_i(t) \\ &= -2\varepsilon_i(t) D^\alpha I_i(t) \\ &= -2\varepsilon_i(t)[-dI_i + \widetilde{\beta} S_i I_i(t - \tau) - \alpha I_i + KI_i(t_l)] \\ &\leq 2\varepsilon_i(t)(d + \alpha) I_i(t) - 2\varepsilon_i(t)\widetilde{\beta} S_i I_i(t - \tau) - 2\varepsilon_i(t) KI_i(t_l) \\ &\leq 3\varepsilon_i^2(t) + (d + \alpha)^2 + \widetilde{\beta}^2 + K^2 I_i^2(t_l) \\ &\leq 3\varepsilon_i^2(t) + \eta, \end{aligned}$$

with $\eta = (d + \alpha)^2 + \widetilde{\beta}^2 + K^2 I_i^2(t_l)$.

Since $\varepsilon_i(t_l) = 0$, then for $t \in (t_l, t_{l+1}]$, $\varepsilon_i^2(t) \leq 3\eta(t - t_l)^{k-1} E_k(3(t - t_l)^k)$.

Choose the event-triggered condition $t_{l+1} = \inf\left\{ t \in (t_l, \infty) \middle| \varepsilon_i^2(t) \geq I_i^2(t) \right\}$.

By letting $\Xi(I_i(t)) = I_i^2(t)$, we obtain that

$$\Xi(I_i(t_{l+1})) \leq 3\eta(t_{l+1} - t_l)^{k-1} E_k(3(t_{l+1} - t_l)^k).$$

If $\Xi(I_i(t_{l+1})) = 0$, then $\eta = 0$, which contradicts $\eta = (d+\alpha)^2 + \widetilde{\beta}^2 + K^2 I_i^2(t_l) > 0$; therefore, $\Xi(I_i(t_{l+1})) > 0$.

Let $T_l = t_{l+1} - t_l$ and $\Xi(I_i(t_{l+1})) = 3\xi\eta(t_{l+1} - t_l)^{k-1} E_k(3(t_{l+1} - t_l)^k)$. Then, $T_l = t_{l+1} - t_l > 0$, and thus, there is no Zeno phenomenon. □

Theorem 4. *For system (16), if* $2K - d - \alpha + \widetilde{\beta} \leq 0$ *and* $0 < L < 1$ *are satisfied, then the system is stable under event-triggered impulsive control.*

Proof. Choose $V(t) = I_i^2(t)$. When $t \in (t_l, t_{l+1}]$,

$$\begin{aligned} D^k V(t) &= I_i(t) D^k I_i(t) \\ &= I_i(t)\Big[-dI_i + \widetilde{\beta} S_i I_i(t-\tau) - \alpha I_i + K(I_i(t) + \varepsilon(t))\Big] \\ &\leq (K - d - \alpha) I_i^2(t) + I_i(t)\Big[\widetilde{\beta} S_i I_i(t-\tau) + K\varepsilon(t)\Big] \\ &\leq (K - d - \alpha) I_i^2(t) + I_i(t)\widetilde{\beta} I_i(t-\tau) + \xi I_i(t) K\varepsilon(t) \\ &\leq (K - d - \alpha) I_i^2(t) + \tfrac{1}{2}\widetilde{\beta}\Big[I_i^2(t) + I_i^2(t-\tau)\Big] + \tfrac{1}{2} K\xi\Big[I_i^2(t) + \varepsilon_i^2(t)\Big]. \end{aligned} \tag{17}$$

From the event-triggered condition, we can deduce that when $t \in (t_l, t_{l+1}]$, $\varepsilon_i^2(t) \leq I_i^2(t)$ and inequality (17) can be written as

$$\begin{aligned} D^k V(t) &\leq (2K - d - \alpha) I_i^2(t) + \tfrac{1}{2}\widetilde{\beta}\Big[I_i^2(t) + I_i^2(t-\tau)\Big] \\ &\leq (2K - d - \alpha) I_i^2(t) + \widetilde{\beta} I_i^2(t) \\ &\leq (2K - d - \alpha + \widetilde{\beta}) I_i^2(t) \\ &= (2K - d - \alpha + \widetilde{\beta}) V(t). \end{aligned}$$

Let $\hat{\beta} = 2K - d - \alpha + \widetilde{\beta}$. Then, $D^k V(t) \leq \widetilde{\beta} V(t)$.

Applying Lemma 1, when $t \in (t_l, t_{l+1}]$, it is easy to obtain that

$$V(t) \leq V(t_l) E_k[\hat{\beta}(t - t_l)^k]. \tag{18}$$

When $t = t_l$,

$$V(t_l) = I_i^2(t_l) = (1 - L)^2 I_i^2(t_{l^-}). \tag{19}$$

From (18) and (19), when $t \in (t_l, t_{l+1}]$, we have that

$$\begin{aligned} V(t) &\leq V(t_l) E_k[\hat{\beta}(t - t_l)^k] \\ &= (1 - L)^2 V(t_{l^-}) E_k[\hat{\beta}(t - t_l)^k] \\ &\leq (1 - L)^2 V(t_{l-1}) E_k[\hat{\beta}(t_{l^-} - t_{l-1})^k] E_k[\hat{\beta}(t - t_l)^k]. \end{aligned}$$

By analogy, when $t \in (t_l, t_{l+1}]$,

$$\begin{aligned} V(t) &\leq (1 - L)^{2l} V(t_0) E_k[\hat{\beta}(t - t_l)^k] \prod_{i=1}^{l} E_k[\hat{\beta}(t_{i^-} - t_{i-1})^k] \\ &\leq (1 - L)^{2l} V(t_0) E_k[\hat{\beta}(t - t_l)^k] \prod_{i=1}^{l} E_k[\hat{\beta}\hat{T}^k], \end{aligned}$$

with $\hat{T} = \sup\{t_{i+1} - t_i\} > 0$, $i = 1, 2, \cdots$.

If $\hat{\beta} = 2K - d - \alpha + \widetilde{\beta} \leq 0$ and $0 < L < 1$, then we can obtain that $\lim_{t\to\infty} V(t) = 0$.

Based on the above analysis, it is clear that for system (15), $\lim_{t\to\infty} I_i(t) = 0$. □

Remark 3. *In Theorem 3, the sufficient conditions for ensuring the stability of fractional-order impulsive systems in the presence of time delays are derived. Furthermore, to the best of the author's knowledge, in the field of epidemics, related works on the stability problem of fractional-order systems under event-triggered impulsive control for finite-time cases with fractional orders of* $0 < k < 1$

have not been investigated yet. This aspect remains unexplored and requires further investigation in future studies.

5. Numerical Simulations

In this section, an example is presented to illustrate the effectiveness of the theoretical results mentioned above.

For system (2), for node i, we set $k = 0.98$, $\alpha = 0.84$, $\beta = 0.65$, $\gamma = 0.14$, a $= 0.002$, $d = 0.001$, and $p = 8$. The initial condition was [0.6411, 0.2346, 0.1243] and ω_{ij} was valued randomly between 0 and 1. When $\tau = 10$, we calculated that $R_0 \approx 2.3 > 1$, and thus, there existed an endemic equilibrium point for system (2). At this moment, the endemic equilibrium point was stable, which is presented in Figure 2.

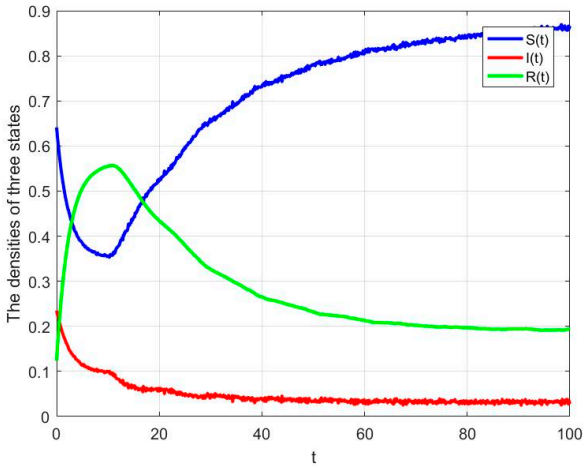

Figure 2. The time responses of all states for system (2) when $\tau = 10$ and $R_0 > 1$.

Furthermore, we also simulated the influence of time delay on the infective rate, as depicted in Figure 3. It is obvious that the disease died out much more slowly as the time delay became larger.

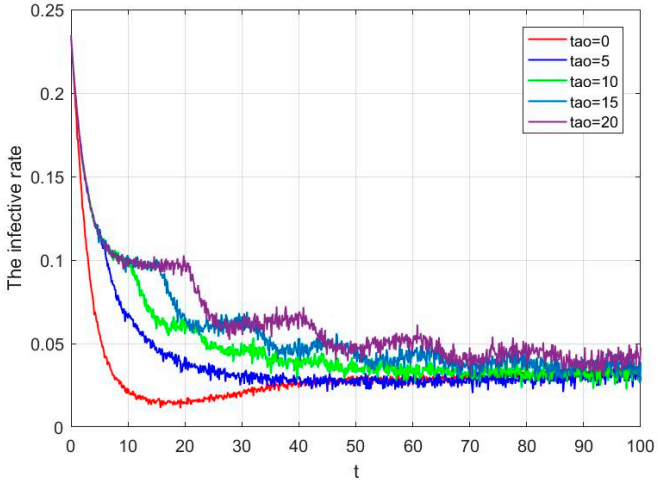

Figure 3. The time responses of infective states when τ varied and $R_0 > 1$.

For system (2), for node i, we set $\beta = 0.45$, a $= 0.001$, and $d = 0.003$, while the other parameters remained unchanged. In this case, we calculated that $R_0 \approx 0.37 < 1$, then there existed only a disease-free equilibrium point for system (2). And the disease-free equilibrium point was globally Lyapunov asymptotic stable, which is presented in Figure 4. Similarly, we also simulated the influence of the time delay on the infective rate, as depicted

in Figure 5. It is also easy to see that the smaller the time delay, the faster the infection rate declined.

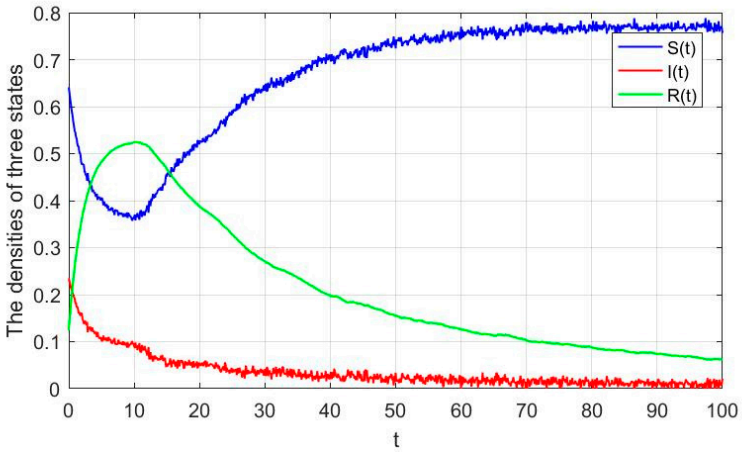

Figure 4. The time responses of all states for system (2) when $\tau = 10$ and $R_0 < 1$.

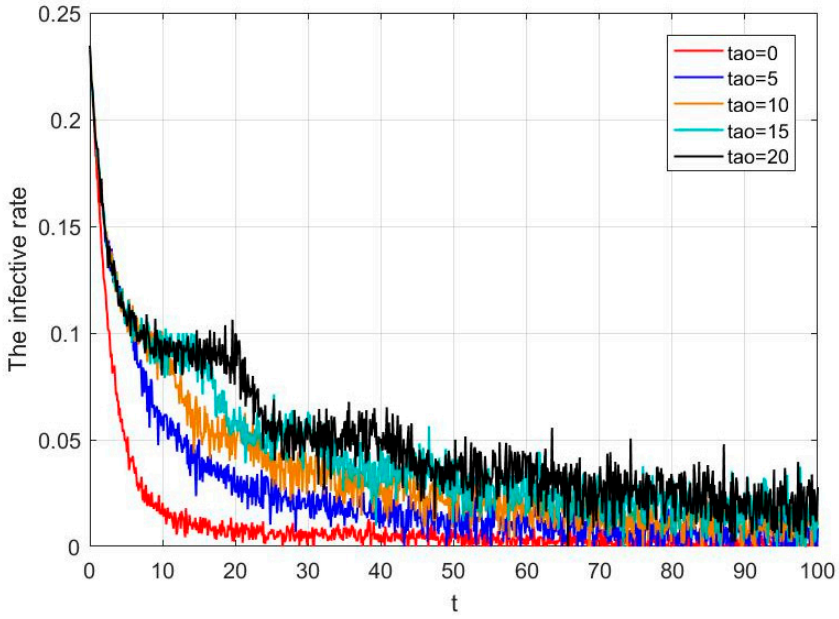

Figure 5. The time responses of the infective states when τ varied and $R_0 < 1$.

It is well known that fractional-order parameters have an important effect on the dynamic behavior of fractional-order systems. Unfortunately, we do not provide relevant theoretical analysis and proof in detail in this paper, but we could simulate and quantify the impact of fractional-order parameters on the infection rate, as shown in Figure 6. It is known that when the fractional-order parameter takes different values, the number of infected people will also be different. Clearly, the smaller the parameter values, the more people were infected.

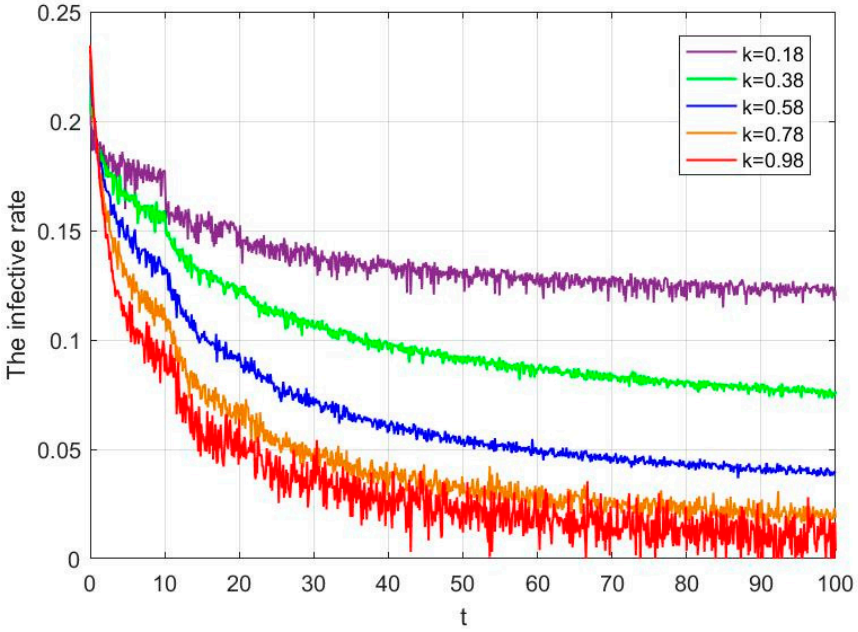

Figure 6. The time responses of the infectious states with $\tau = 10$ and $R_0 < 1$ when k varies.

Since the Chinese government's response to the COVID-19 outbreak was to divide places into different risk levels, the intensity of control for high-risk and low-risk areas varied. In this manuscript, in order to better highlight the control effect, we took the situation $R_0 > 1$ as an example to apply control. When the event-triggered control was active, the infection rates were 0.2 and 0.01, while the control gains were $L = 0.65$ and $K = -0.02$ for high-risk and low-risk areas, respectively. The other parameters were the same as in Figure 2. The simulation result is presented in Figure 7, which shows that the disease decreased faster and eventually declined to zero, and the control method was effective.

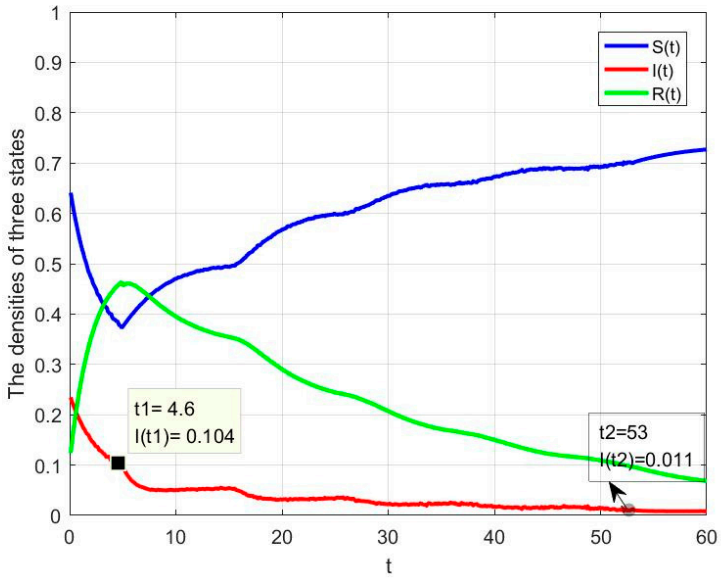

Figure 7. The time responses under event-triggered control.

Remark 4. *From Figure 7, it is obvious that for system (2) under the event-triggered impulsive control, the infectious rate decayed to zero rapidly. Compared with Figure 2, there was a stable endemic equilibrium point in the system without control, indicating that there was always a certain infected population.*

6. Conclusions

In this work, we proposed and analyzed a fractional-order epidemic model with time delay and network weights. First, the existence and stability of both a disease-free equilibrium point and an endemic equilibrium point were discussed. Then, sufficient conditions of stability for fractional-order epidemic systems with time delay are provided under event-triggered impulsive control, and the Zeno phenomenon was ruled out. Also, we found that the fractional-order parameter greatly influenced the infection rate.

For certain diseases, it is not necessary to achieve a complete eradication of the infection rate in practical scenarios. It is considered that the disease is under control when the infection rate is within a specific range. Hence, the top task we should carry out is to control the infection rate of the entire population to a certain acceptable range. Moreover, for some specific epidemics, for example, COVID-19 and cholera, people hope to suppress them quickly so that they can avoid casualties and economic losses. Therefore, our next crucial task is to propose an effective method to contain a major outbreak of an epidemic in a finite time.

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