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DYNAMICAL ANALYSIS OF A COVID-19 MODEL WITH HUMAN-TO-HUMAN AND ENVIRONMENT-TO-HUMAN TRANSMISSIONS AND DISTRIBUTED DELAYS

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Abstract. SARS-CoV-2 can survive in different environments and remain infectious for several days, which presents challenges to eliminating infectious diseases. It encourages researchers to study the effects of SARS-CoV-2 on the environment. In this paper, we formulate an epidemic model for SARS-CoV-2, which focuses on the transmission of the virus under environmental conditions. Two distributed delays are introduced to describe the probability of the exposed and infected individuals in different infection periods based on the transmission of the virus in the environment. The positivity and boundness of solutions of model are derived. The basic reproduction number threshold theory is established and the results demonstrate that the persistence of COVID-19 depends on the basic reproduction number. Numerical simulations are presented to verify the theoretical results. Some measures are proposed to control and eliminate COVID-19 infectious diseases.

1. Introduction

Infectious diseases, such as acquired immune deficiency syndrome (AIDS), tuberculosis, cholera, and influenza, are caused by viruses, parasites, or pathogenic microorganisms that can be transmitted from human to human, animal to animal, or human to animal [15]. Infectious diseases not only pose public health burdens for society but can bring great disasters to the national economy.

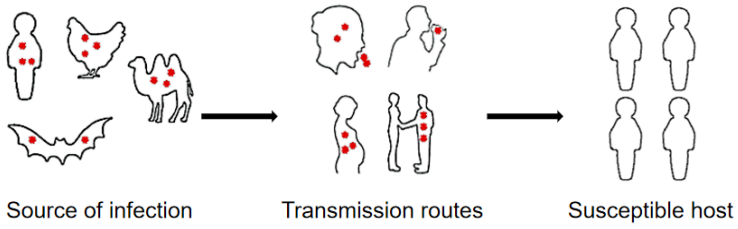

Figure 1. Transmission of infectious disease.

As Figure 1 shows, infectious diseases can spread or prevail according to the three compartments, the source of infection (people or animals that can excrete pathogens), the transmission routes (pathogen transmission routes to others), and the susceptible host (healthy people who can be infected). Cutting off one of the links completely is considered a useful way to prevent the occurrence and prevalence of this infectious disease. Therefore, it is very important to investigate the pathogenesis and transmission mechanisms of infectious diseases.

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Mathematical modeling is one of the most efficient tools for investigating the transmission of infectious diseases in the population. By analyzing the dynamic properties of the model, it is not only possible to understand the transmission mechanisms of infectious diseases but also to study the transmission patterns of infectious diseases qualitatively and quantitatively, which is important for the formulation of prevention and control measures.

In recent years, coronavirus disease 2019 (COVID-19) has been troubling the world which is caused by a novel coronavirus named SARS-CoV-2. Coronaviruses are enveloped RNA viruses that are distributed broadly among humans, other mammals, and birds and that cause respiratory, enteric, hepatic, and neurologic diseases [14, 30]. Severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS) are also caused by coronaviruses. However, different from SARS, asymptomatic infection plays a major role in the spread of SARS-CoV-2 [27]. Asymptomatic infections refer to individuals who are infected and carry viruses, but lack noticeable symptoms throughout the infection course. Johansson et al. [12] introduced an analysis study to estimate the transmission of SARS-CoV-2, which concluded that at least 50% of new SARS-CoV-2 infections are caused by asymptomatic infected individuals. Almadhi et al. [1] analyzed the data from the National COVID-19 Database in Bahrain, which supported these predictions. The presence of individuals with infection but without symptoms reduces the efficiency of identifying the asymptomatic infected individuals through symptom screening, creates difficulties in controlling the source of transmission, and leads to pandemics of infectious diseases. It is important to use mathematical modeling to study the dynamic properties of infectious diseases and propose appropriate control strategies to eliminate epidemics.

The impact of individuals who are infected and infectious but not yet symptomatic on the transmission of COVID-19 cannot be ignored, and many scholars have introduced exposed infectious into their models. Annas et al. [2] constructed an SEIR model for COVID-19 and provided stability analysis and numerical simulation of the SEIR model. They found vaccines can accelerate COVID-19 healing, and the isolation period can slow the spread of COVID-19 in Indonesia. To consider whether and for how long the dynamic zero-crown strategy adopted by China since August 2021 can be maintained, Cai et al. [7] developed an age-structured stochastic SLIRS model and suggested some factors should be the focus of future mitigation policies. Berger et al. [5] presented a standard SEIR model with testing and several other pertinent features and pointed out that testing asymptomatic individuals can stand in for economically costly quarantine measures.

By investigating a cluster of COVID-19 cases in Wenzhou, China, Cai et al. [8] realized that indirect transmission is a route of SARS-CoV-2 transmission, and they pointed out that indirect transmission perhaps results from objects contaminated by virus or aerosols containing virus. Xiao et al. [28] provided evidence for gastrointestinal infection with SARS-CoV-2, and it suggested infection may spread via faeces. Ong et al. [17] detected the presence of SARS-CoV-2 on the surfaces of objects and suggested the contaminated environment as a potential medium of transmission. Kampf et al. [13] summarized 22 studies that revealed that human coronaviruses persist on inanimate surfaces like metal, glass, or plastic for up to 9 days. It is necessary to consider the impact of SARS-CoV-2 in the environment.

Yang and Wang [29] proposed a mathematical model with non-constant transmission rates, emphasized the role of the environmental contamination in the transmission of COVID-19, and reflected the impact of the on-going disease control measures. To investigate the effect of viruses in the environment and individual mobility on infectious diseases, Rao et al. [19] established an SEIVR (Susceptible-Exposed-Infected-Environment-Remove) epidemic model with individual mobility, and suggested that customs inspection is very effective in preventing high-risk group movement. Naik et al. [16] proposed a novel nonlinear mathematical model of the COVID-19 epidemic, analyzed the effects of the

environmental virus on the transmission patterns, and determined the relative importance of the disease transmission parameters. To enhance the surveillance of wastewater for early epidemic prediction, Proverbio et al. [18] presented a novel mechanistic model-based approach to reconstruct the complete epidemic dynamics from the SARS-CoV-2 viral load in wastewater. Zou et al. [31] indicated that viral loads in the nasal and throat are related to days of infection, and higher viral loads were detected soon after symptom onset. The number of viruses in the environment is not only related to the number of individuals infected but also to the viral load in their nasal and throat. Thus, we propose a model with two distributed delays to describe the difference of viral load in nasal and throat at different stages of infection.

The main objective of this paper is to construct the DDE model for COVID-19 and analyze the stability of the model. The rest of the paper is organized as follows. In Section 2, we construct the DDE model according to the characteristics of the spread of COVID-19. The positivity, boundness, and analysis of the reproduction number of the model will be investigated in Section 3. In Section 4, we analyze the stability of two equilibria. Numerical simulations are presented in Section 5 to confirm the theoretical results. In Section 6, we conclude the paper with some discussion.

2. Mathematical modeling

To study the spread of COVID-19, Tilahun and Alemneh [24] proposed the following model

$$\begin{cases} \frac{\mathrm{d}S}{\mathrm{d}t} = \pi + \eta R - \beta(\sigma_1 I + \sigma_2 E)S - \mu S, \\ \frac{\mathrm{d}E}{\mathrm{d}t} = \beta(\sigma_1 I + \sigma_2 E)S - (\delta + \mu)E, \\ \frac{\mathrm{d}I}{\mathrm{d}t} = \tau\delta E - (\varepsilon + \rho + \mu)I, \\ \frac{\mathrm{d}R}{\mathrm{d}t} = (1-\tau)\delta E + \varepsilon I - (\mu + \eta)R. \end{cases} \tag{2.1}$$

In this model, the population is divided into four compartments: susceptible(S), exposed(E), infected (I) and recovery(R) individuals. The exposed individuals indicate those people who are infected and infectious but not yet symptomatic.

This model examined the impact of exposed infections on COVID-19 without considering the impact of viruses in the environment. Thus, in our model, W is added to characterize the density of the viruses in the environment since susceptible individuals can be infected by viruses that are transmitted to the environment by infected and exposed individuals. As the viral load in the nasal and throat is related to days of infection, two distributed delays are introduced in our model with kernel functions given by $f(\tau_1) = f_1(\tau_1)e^{-\delta_1\tau_1}$ and $g(\tau_2) = g_1(\tau_2)e^{-\delta_2\tau_2}$, where δ_1 and δ_2 are the average removal rates of exposed and infected individuals. $f_1(\tau_1)$ denotes the probability that the virus is transmitted from individuals who turn into exposed individuals at $t-\tau_1$ to the environment at moment t, and $g_1(\tau_2)$ denotes the probability that the virus is transmitted from individuals who turn into infected individuals at $t-\tau_2$ to the environment at moment t. We assume that viruses in the environment are removed at rate d. Thus, the dynamic of viruses in the environment is governed by

$$\frac{\mathrm{d}W(t)}{\mathrm{d}t} = \int_0^{+\infty} \sigma_1 E(t-\tau_1)f(\tau_1)\mathrm{d}\tau_1 + \int_0^{+\infty} \sigma_2 I(t-\tau_2)g(\tau_2)\mathrm{d}\tau_2 - dW, \tag{2.2}$$

where σ_1 and σ_2 are the respective rates of exposed and infected individuals spreading the virus to the environment.

We assume that recovery individuals would not lose their immunity, then the transfer diagram for the COVID-19 model with human-to-human and environment-to-human transmissions and distributed delays is depicted in Figure 2.

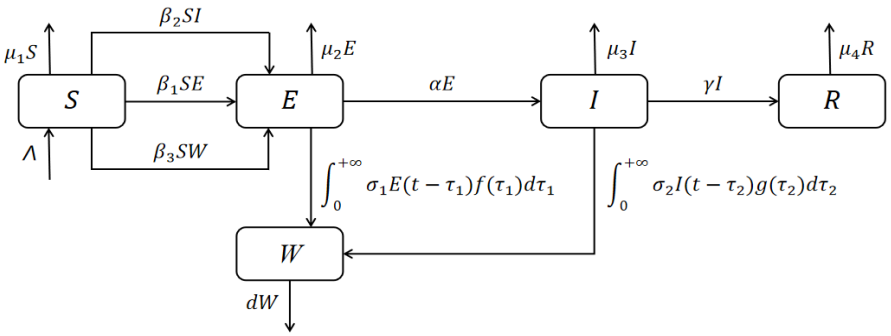


FIGURE 2. Transfer diagram for the COVID-19 model with human-to-human and environment-to-human transmissions and distributed delays.

Thus, we consider the following model

$$\begin{cases} \dfrac{\mathrm{d}S(t)}{\mathrm{d}t} = \Lambda - \beta_1 SE - \beta_2 SI - \beta_3 SW - \mu_1 S, \\ \dfrac{\mathrm{d}E(t)}{\mathrm{d}t} = \beta_1 SE + \beta_2 SI + \beta_3 SW - (\alpha + \mu_2)E, \\ \dfrac{\mathrm{d}I(t)}{\mathrm{d}t} = \alpha E - (\gamma + \mu_3)I, \\ \dfrac{\mathrm{d}R(t)}{\mathrm{d}t} = \gamma I - \mu_4 R, \\ \dfrac{\mathrm{d}W(t)}{\mathrm{d}t} = \displaystyle\int_0^{+\infty} \sigma_1 E(t-\tau_1) f(\tau_1) \mathrm{d}\tau_1 + \int_0^{+\infty} \sigma_2 I(t-\tau_2) g(\tau_2) \mathrm{d}\tau_2 - dW, \end{cases} \tag{2.3}$$

where the parameter Λ is the population influx. β_1, β_2, and β_3 represent the transmission rates between susceptible individuals and the exposed, infected, and the viruses in the environment, respectively. μ_1, μ_2, μ_3, and μ_4 are the death rates of the susceptible, exposed, infected, and recovered individuals, respectively. Due to the fact that COVID-19 increases the mortality rate of individuals, we asumme that $\min\{\mu_2, \mu_3, \mu_4\} > \mu_1$. The parameter α^{-1} indicates the incubation period between the infection, and the onset of symptoms and γ represents the rate of recovery from infection. For the kernel functions $f(\tau_1)$ and $g(\tau_2)$, we assume $f_1(\tau_1) \geq 0, g_1(\tau_2) \geq 0$ and

$$\int_0^{+\infty} f(\tau_1)\mathrm{d}\tau_1 = 1, \int_0^{+\infty} g(\tau_2)\mathrm{d}\tau_2 = 1. \tag{2.4}$$

Due to the infinite delay, we need to determine an appropriate phase space. For any $\triangle \in (0, \min\{\delta_1, \delta_2\})$, let

$$C_\triangle = \left\{\varphi : (-\infty, 0] \to R, \varphi(\theta)e^{\triangle\theta} \text{ is bounded on } (-\infty, 0]\right\},$$

and

$$Y_\triangle = \{\varphi \in C_\triangle : \varphi(\theta) \geq 0 \text{ for all } \theta \leq 0\}.$$

Define the norm on $C_\triangle$ and $Y_\triangle$ by

$$\| \varphi \| = \sup_{\theta \leq 0} \left|\varphi(\theta)e^{\triangle\theta}\right|.$$

Let $\mathbb{R}_0^+ = [0, +\infty)$, the initial conditions of (2.3) are given as

$$S(0) = s_0, E(\theta) = \varphi_1(\theta), I(\theta) = \varphi_2(\theta), R(0) = r_0, W(0) = w_0, \ \theta \in (-\infty, 0], \tag{2.5}$$

where $s_0, r_0, w_0 \in \mathbb{R}_0^+$ and $\varphi_i \in Y_\triangle$ for $i = 1, 2$.

3. Well-posedness

Theorem 3.1. *Solutions of system (2.3) satisfying the initial conditions (2.5) are non-negative.*

Proof. Asumme that there is a $t_1 > 0$ such that $S(t_1) < 0$, then there must be a $t_2 \in [0, t_1)$ such that $S(t_2) = 0$ and $S'(t_2) < 0$. By the first equation of system (2.3) it has $S'(t_2) = \Lambda > 0$ for $S(t_2) = 0$, which is a contradiction to $S'(t_2) < 0$. It follows that $S(t) \geq 0$ for all $t \geq 0$.

Suppose that there is a $t_3 > 0$ such that $E(t_3) < 0$, then there must be a $0 \leq \tilde{t} < t_3$ such that $E(t) \geq 0$ for all $t \in [0, \tilde{t}]$, $E(\tilde{t}) = 0$, and $E'(\tilde{t}) < 0$. From the second equation of system (2.3), we have $I(\tilde{t}) < 0$ or $W(\tilde{t}) < 0$. For the case of $I(\tilde{t}) < 0$, there must be a $0 \leq \hat{t} < \tilde{t}$ such that $I(t) \geq 0$ for all $t \in [0, \hat{t}]$, $I(\hat{t}) = 0$, and $I'(\hat{t}) < 0$. From the third equation of system (2.3), we have $E(\hat{t}) < 0$, which is a contradiction to $E(t) \geq 0$ for all $t \in [0, \tilde{t}]$. We can show that $W(\tilde{t}) < 0$ is a contradiction to $E(t) \geq 0$ for all $t \in [0, \tilde{t}]$ in the same way. Thus, there does not exist $t_3 > 0$ such that $E(t_3) < 0$, which implies $E(t) \geq 0$ for all $t \in [0, +\infty)$.

From the fourth equation of system (2.3), it follows that

$$I(t) = I(0)e^{-(\gamma+\mu_3)t} + \int_0^t \alpha E(\eta) e^{-(\gamma+\mu_3)(t-\eta)} \mathrm{d}\eta.$$

This shows that $I(t) \geq 0$ for all $t \in [0, +\infty)$ from $E(t) \geq 0$ and the initial conditions.

By the last two equations of system (2.3), we obtain

$$R(t) = R(0)e^{-\mu_4 t} + \int_0^t \gamma I(\eta) e^{-\mu_4(t-\eta)} \mathrm{d}\eta,$$

and

$$W(t) = W(0)e^{-dt} + \int_0^t \left[\int_0^{+\infty} \sigma_1 E(\eta - \tau_1) f(\tau_1) \mathrm{d}\tau_1 + \int_0^{+\infty} \sigma_2 I(\eta - \tau_2) g(\tau_2) \mathrm{d}\tau_2 \right] e^{-d(t-\eta)} \mathrm{d}\eta.$$

It is easy to show that $R(t) \geq 0$ and $W(t) \geq 0$ for all $t \in [0, +\infty)$ from $E(t) \geq 0$, $I(t) \geq 0$, and the initial conditions. The proof is completed. □

Theorem 3.2. *Solutions of system (2.3) is ultimately bounded, that is there exists $M > 0$ and $t_0 > 0$ such that for solutions of system (2.3) satisfy $\max\{S(t), E(t), I(t), R(t), W(t)\} \leq M$ for any $t \in [t_0, \infty)$.*

Proof. From $N(t) = S(t) + E(t) + I(t) + R(t)$, one gets

$$\frac{\mathrm{d}N(t)}{\mathrm{d}t} = \Lambda - \mu_1 S(t) - \mu_2 E(t) - \mu_3 I(t) - \mu_4 R(t) \leq \Lambda - \mu_1 N(t),$$

then we obtain

$$\limsup_{t \to \infty} N(t) \leq \frac{\Lambda}{\mu_1}.$$

This implies that there exists $N_1 > 0$ such that solutions on $[N_1, \infty)$ satisfy

$$S(t) \leq \frac{\Lambda}{\mu_1} + 1, E(t) \leq \frac{\Lambda}{\mu_1} + 1, I(t) \leq \frac{\Lambda}{\mu_1} + 1, R(t) \leq \frac{\Lambda}{\mu_1} + 1.$$

Next, we define $S(\theta) = s_0$ for all $\theta \in (-\infty, 0]$ and

$$F(t) = \frac{1}{\alpha} \int_0^{+\infty} \sigma_1 [S(t-\tau_1) + E(t-\tau_1)] f(\tau_1) d\tau_1 + \frac{1}{\gamma} \int_0^{+\infty} \sigma_2 [S(t-\tau_2) + E(t-\tau_2) + I(t-\tau_2)] g(\tau_2) d\tau_2 + W(t).$$

Combining $F(t)$ and equations of (2.3), it has

$$\begin{aligned}F'(t) =&\frac{\Lambda\sigma_1 p}{\alpha}+\frac{\Lambda\sigma_2 q}{\gamma}-\frac{1}{\alpha}\int_0^{+\infty}\sigma_1[\mu_1 S(t-\tau_1)+\mu_2 E(t-\tau_1)]f(\tau_1)d\tau_1\\&-\frac{1}{\gamma}\int_0^{+\infty}\sigma_2[\mu_1 S(t-\tau_2)+\mu_2 E(t-\tau_2)+\mu_3 I(t-\tau_2)]g(\tau_2)d\tau_2-dW(t)\\\leq&\frac{\Lambda\sigma_1 p}{\alpha}+\frac{\Lambda\sigma_2 q}{\gamma}-hF(t),\end{aligned}$$

where $h=\min\{\mu_1,d\}$ and

$$p=\int_0^{+\infty}f(\tau_1)d\tau_1,\quad q=\int_0^{+\infty}g(\tau_2)d\tau_2.$$

Then we have

$$\limsup_{t\to\infty}F(t)\leq\frac{\Lambda\sigma_1 p}{\alpha h}+\frac{\Lambda\sigma_2 q}{\gamma h},$$

which means that there exists $N_2>0$ such that

$$W(t)\leq\frac{\Lambda\sigma_1 p}{\alpha h}+\frac{\Lambda\sigma_2 q}{\gamma h}+1\ \text{ for }\ t\in[N_2,+\infty).$$

Hence,

$$M>\max\left\{\frac{\Lambda}{\mu_1}+1,\frac{\Lambda\sigma_1 p}{\alpha h}+\frac{\Lambda\sigma_2 q}{\gamma h}+1\right\}$$

and $t_0>\max\{N_1,N_2\}$ are obtained such that solutions of system (2.3) satisfy

$$\max\{S(t),E(t),I(t),R(t),W(t)\}\leq M\quad\text{for any }\ t\in[t_0,\infty).$$

□

Since the equations of S,E,I and W are decoupled in (2.3), it suffices to study the following system

$$\begin{cases}\dfrac{\mathrm{d}S(t)}{\mathrm{d}t}=\Lambda-\beta_1 SE-\beta_2 SI-\beta_3 SW-\mu_1 S,\\[2mm]\dfrac{\mathrm{d}E(t)}{\mathrm{d}t}=\beta_1 SE+\beta_2 SI+\beta_3 SW-(\alpha+\mu_2)E,\\[2mm]\dfrac{\mathrm{d}I(t)}{\mathrm{d}t}=\alpha E-(\gamma+\mu_3)I,\\[2mm]\dfrac{\mathrm{d}W(t)}{\mathrm{d}t}=\displaystyle\int_0^{+\infty}\sigma_1 E(t-\tau_1)f(\tau_1)\mathrm{d}\tau_1+\int_0^{+\infty}\sigma_2 I(t-\tau_2)g(\tau_2)\mathrm{d}\tau_2-dW.\end{cases}\tag{3.1}$$

It is obvious that system (3.1) has a disease-free equilibrium $E_0=(S_0,0,0,0)$, where $S_0=\Lambda/\mu_1$.

The infection components in this model are E,I, and W. The new infection matrix F and the transition matrix V are given by

$$F=\begin{bmatrix}\beta_1 S_0&\beta_2 S_0&\beta_3 S_0\\0&0&0\\0&0&0\end{bmatrix},$$

and

$$V=\begin{bmatrix}\alpha+\mu_2&0&0\\-\alpha&\gamma+\mu_3&0\\-\int_0^{\infty}\sigma_1 f(\tau_1)d\tau_1&-\int_0^{\infty}\sigma_2 g(\tau_2)d\tau_2&d\end{bmatrix}.$$

By $\Re_0=\rho(FV^{-1})$, we get the basic reproduction number of system (3.1) as followed

$$\Re_0=\frac{\Lambda}{\mu_1}\left(\frac{\beta_1}{(\alpha+\mu_2)}+\frac{\alpha\beta_2}{(\alpha+\mu_2)(\gamma+\mu_3)}+\frac{[(\gamma+\mu_3)\sigma_1 p+\alpha\sigma_2 q]\beta_3}{(\alpha+\mu_2)(\gamma+\mu_3)d}\right).\tag{3.2}$$

If system (3.1) has an endemic equilibrium $E_1(S^*, E^*, I^*, W^*)$ which should satisfy the following equations

$$\begin{cases} \Lambda - \beta_1 S^* E^* - \beta_2 S^* I^* - \beta_3 S^* W^* - \mu_1 S^* = 0, \\ \beta_1 S^* E^* + \beta_2 S^* I^* + \beta_3 S^* W^* - (\alpha + \mu_2) E^* = 0, \\ \alpha E^* - (\gamma + \mu_3) I^* = 0, \\ \int_0^{+\infty} \sigma_1 E^* f(\tau_1) \mathrm{d}\tau_1 + \int_0^{+\infty} \sigma_2 I^* g(\tau_2) \mathrm{d}\tau_2 - dW^* = 0. \end{cases} \tag{3.3}$$

Then we obtain the endemic equilibrium $E_1(S^*, E^*, I^*, W^*)$ exists if $\Re_0 > 1$, and

$$\begin{aligned} S^* &= \frac{\Lambda}{\Re_0 \mu_1}, \\ E^* &= \frac{\Lambda}{\alpha + \mu_2} \left(1 - \frac{1}{\Re_0}\right), \\ I^* &= \frac{\Lambda \alpha}{(\alpha + \mu_2)(\gamma + \mu_3)} \left(1 - \frac{1}{\Re_0}\right), \\ W^* &= \frac{\Lambda[(\gamma + \mu_3)\sigma_1 p + \alpha \sigma_2 q]}{(\alpha + \mu_2)(\gamma + \mu_3) d} \left(1 - \frac{1}{\Re_0}\right). \end{aligned}$$

4. Stability analysis

4.1. **Local stability.**

Theorem 4.1.

(i) *The disease-free equilibrium E_0 is locally asymptotically stable if $\Re_0 < 1$;*

(ii) *The endemic equilibrium E_1 is locally asymptotically stable if $\Re_0 > 1$.*

Proof. (i) The characteristic equation of system (3.1) at the disease-free equilibrium $E_0 = (S_0, 0, 0, 0)$ is

$$\begin{aligned} &(\lambda + \mu_1)[(\lambda - \beta_1 S_0 + \alpha + \mu_2)(\lambda + \gamma + \mu_3)(\lambda + d) - \alpha \beta_2 S_0 (\lambda + d) \\ &- (\lambda + \gamma + \mu_3)\sigma_1 \beta_3 S_0 \int_0^{+\infty} e^{-\lambda \tau_1} f(\tau_1) d\tau_1 - \alpha \sigma_2 \beta_3 S_0 \int_0^{+\infty} e^{-\lambda \tau_2} g(\tau_2) d\tau_2] = 0. \end{aligned} \tag{4.1}$$

Due to $\lambda = -\mu_1 < 0$, so the stability of E_0 depends on

$$\begin{aligned} &(\lambda - \beta_1 S_0 + \alpha + \mu_2)(\lambda + \gamma + \mu_3)(\lambda + d) = \alpha \beta_2 S_0 (\lambda + d) \\ &+ (\lambda + \gamma + \mu_3)\sigma_1 \beta_3 S_0 \int_0^{+\infty} e^{-\lambda \tau_1} f(\tau_1) d\tau_1 + \alpha \sigma_2 \beta_3 S_0 \int_0^{+\infty} e^{-\lambda \tau_2} g(\tau_2) d\tau_2. \end{aligned} \tag{4.2}$$

It follows that

$$\begin{aligned} \frac{\lambda}{S_0} - \beta_1 + \frac{\alpha + \mu_2}{S_0} =& \frac{\alpha \beta_2}{\lambda + \gamma + \mu_3} + \frac{\sigma_1 \beta_3 \int_0^{+\infty} e^{-\lambda \tau_1} f(\tau_1) d\tau_1}{\lambda + d} \\ &+ \frac{\alpha \sigma_2 \beta_3 \int_0^{+\infty} e^{-\lambda \tau_2} g(\tau_2) d\tau_2}{(\lambda + \gamma + \mu_3)(\lambda + d)}. \end{aligned} \tag{4.3}$$

Let $\lambda = a + ib$, if $a \geq 0$, we have

$$\begin{aligned}
&\left| \frac{\alpha\beta_2}{\lambda+\gamma+\mu_3} + \frac{\sigma_1\beta_3 \int_0^{+\infty} e^{-\lambda\tau_1} f(\tau_1)d\tau_1}{\lambda+d} + \frac{\alpha\sigma_2\beta_3 \int_0^{+\infty} e^{-\lambda\tau_2} g(\tau_2)d\tau_2}{(\lambda+\gamma+\mu_3)(\lambda+d)} \right| \\
&\leq \left| \frac{\alpha\beta_2}{\lambda+\gamma+\mu_3} \right| + \left| \frac{\sigma_1\beta_3 \int_0^{+\infty} e^{-\lambda\tau_1} f(\tau_1)d\tau_1}{\lambda+d} \right| + \left| \frac{\alpha\sigma_2\beta_3 \int_0^{+\infty} e^{-\lambda\tau_2} g(\tau_2)d\tau_2}{(\lambda+\gamma+\mu_3)(\lambda+d)} \right| \\
&\leq \frac{\alpha\beta_2}{|\lambda+\gamma+\mu_3|} + \frac{\sigma_1 p\beta_3}{|\lambda+d|} + \frac{\alpha\sigma_2 q\beta_3}{|(\lambda+\gamma+\mu_3)|\,|(\lambda+d)|} \\
&\leq \frac{\alpha\beta_2}{\gamma+\mu_3} + \frac{\sigma_1 p\beta_3}{d} + \frac{\alpha\sigma_2 q\beta_3}{(\gamma+\mu_3)d},
\end{aligned}$$

and

$$\left| \frac{\lambda}{S_0} - \beta_1 + \frac{\alpha+\mu_2}{S_0} \right| \geq -\beta_1 + \frac{\alpha+\mu_2}{S_0}.$$

From $\Re_0 < 1$, it has

$$\frac{\alpha\beta_2}{\gamma+\mu_3} + \frac{\sigma_1 p\beta_3}{d} + \frac{\alpha\sigma_2 q\beta_3}{(\gamma+\mu_3)d} < -\beta_1 + \frac{\alpha+\mu_2}{S_0},$$

which leads to a contradiction. Thus all characteristic roots of equation (4.1) have negative real parts. The disease-free equilibrium is locally asymptotically stable if $\Re_0 < 1$.

(ii) The characteristic equation of system (3.1) at the endemic equilibrium $E_1 = (S^*, E^*, I^*, W^*)$ is

$$\begin{aligned}
&\frac{\Lambda}{\mu_1\Re_0}(\lambda+\mu_1)\left[\beta_1(\lambda+\gamma+\mu_3)(\lambda+d) + \beta_2\alpha(\lambda+d) + \beta_3\sigma_1(\lambda+\gamma+\mu_3)\int_0^{\infty} e^{-\tau_1\lambda} f(\tau_1)d\tau_1\right. \\
&\left.+\beta_3\alpha\sigma_2\int_0^{\infty} e^{-\tau_2\lambda} g(\tau_2)d\tau_2\right] = (\lambda+\mu_1\Re_0)(\lambda+\alpha+\mu_2)(\lambda+\gamma+\mu_3)(\lambda+d).
\end{aligned} \tag{4.4}$$

It follows that

$$\begin{aligned}
&\frac{(\lambda+\mu_1)}{(\lambda+\mu_1\Re_0)}\frac{\Lambda}{\mu_1\Re_0}\left[\frac{\beta_1}{(\lambda+\alpha+\mu_2)} + \frac{\beta_2\alpha}{(\lambda+\alpha+\mu_2)(\lambda+\gamma+\mu_3)} + \frac{\beta_3\sigma_1\int_0^{\infty} e^{-\tau_1\lambda} f(\tau_1)d\tau_1}{(\lambda+\alpha+\mu_2)(\lambda+d)}\right. \\
&\left.+\frac{\beta_3\alpha\sigma_2\int_0^{\infty} e^{-\tau_2\lambda} g(\tau_2)d\tau_2}{(\lambda+\alpha+\mu_2)(\lambda+\gamma+\mu_3)(\lambda+d)}\right] = 1.
\end{aligned} \tag{4.5}$$

Let

$$\begin{aligned}
m =& \frac{\beta_1}{(\lambda+\alpha+\mu_2)} + \frac{\beta_2\alpha}{(\lambda+\alpha+\mu_2)(\lambda+\gamma+\mu_3)} + \frac{\beta_3\sigma_1\int_0^{\infty} e^{-\tau_1\lambda} f(\tau_1)d\tau_1}{(\lambda+\alpha+\mu_2)(\lambda+d)} \\
&+ \frac{\beta_3\alpha\sigma_2\int_0^{\infty} e^{-\tau_2\lambda} g(\tau_2)d\tau_2}{(\lambda+\alpha+\mu_2)(\lambda+\gamma+\mu_3)(\lambda+d)}.
\end{aligned}$$

If $\lambda = a + ib$, $a \geq 0$ is a root of (4.5), it follows

$$\begin{aligned}
|m| =& \left| \frac{\beta_1}{(\lambda+\alpha+\mu_2)} + \frac{\beta_2\alpha}{(\lambda+\alpha+\mu_2)(\lambda+\gamma+\mu_3)} + \frac{\beta_3\sigma_1 \int_0^\infty e^{-\tau_1\lambda} f(\tau_1)d\tau_1}{(\lambda+\alpha+\mu_2)(\lambda+d)} \right. \\
& \left. + \frac{\beta_3\alpha\sigma_2 \int_0^\infty e^{-\tau_2\lambda} g(\tau_2)d\tau_2}{(\lambda+\alpha+\mu_2)(\lambda+\gamma+\mu_3)(\lambda+d)} \right| \\
\leq & \left| \frac{\beta_1}{(\lambda+\alpha+\mu_2)} \right| + \left| \frac{\beta_2\alpha}{(\lambda+\alpha+\mu_2)(\lambda+\gamma+\mu_3)} \right| + \left| \frac{\beta_3\sigma_1 \int_0^\infty e^{-\tau_1\lambda} f(\tau_1)d\tau_1}{(\lambda+\alpha+\mu_2)(\lambda+d)} \right| \\
& + \left| \frac{\beta_3\alpha\sigma_2 \int_0^\infty e^{-\tau_2\lambda} g(\tau_2)d\tau_2}{(\lambda+\alpha+\mu_2)(\lambda+\gamma+\mu_3)(\lambda+d)} \right| \\
=& \frac{\beta_1}{|(\lambda+\alpha+\mu_2)|} + \frac{\beta_2\alpha}{|(\lambda+\alpha+\mu_2)|\,|(\lambda+\gamma+\mu_3)|} + \frac{\left|\beta_3\sigma_1 \int_0^\infty e^{-\tau_1\lambda} f(\tau_1)d\tau_1\right|}{|(\lambda+\alpha+\mu_2)|\,|(\lambda+d)|} \\
& + \frac{\left|\beta_3\alpha\sigma_2 \int_0^\infty e^{-\tau_2\lambda} g(\tau_2)d\tau_2\right|}{|(\lambda+\alpha+\mu_2)|\,|(\gamma+\mu_3)|\,|(\lambda+d)|} \\
\leq & \frac{\beta_1}{(\alpha+\mu_2)} + \frac{\beta_2\alpha}{(\alpha+\mu_2)(\gamma+\mu_3)} + \frac{\beta_3\sigma_1 p}{(\alpha+\mu_2)d} + \frac{\beta_3\alpha\sigma_2 q}{(\alpha+\mu_2)(\gamma+\mu_3)d} \\
=& \frac{\mu_1}{\Lambda}\Re_0.
\end{aligned}$$

If $\Re_0 > 1$, we have

$$\left| \frac{\lambda+\mu_1}{\lambda+\mu_1\Re_0} \right| = \frac{|\lambda+\mu_1|}{|\lambda+\mu_1\Re_0|} < 1.$$

Thus, one obtains

$$\left| \frac{(\lambda+\mu_1)}{(\lambda+\mu_1\Re_0)} \frac{\Lambda}{\mu_1\Re_0} m \right| < 1.$$

This leads to a contradiction. Thus all characteristic roots of equation (4.4) have negative real parts. The endemic equilibrium E_1 is locally asymptotically stable if $\Re_0 > 1$. □

4.2. **Persistence.** In this subsection, we will employ the permanence theorem of Hale and Waltman [11] which has been applied in Theorem 6.1 of Röst and Wu [20] and Theorem 2.3 of Wang et al. [25] to get the uniform persistence of system (3.1). Let $X = \mathbb{R}_0^+ \times Y_\triangle \times Y_\triangle \times \mathbb{R}_0^+$, and $T(t), t \geq 0$ denotes the family of solution operators corresponding to system (3.1). We follow the notion in [25].

Theorem 4.2. [20]. *Suppose that we have the following*

(i) X^0 *is open and dense in* X *with* $X^0 \cup X_0 = X$ *and* $X^0 \cap X_0 = \emptyset$;

(ii) *the solution operators* $T(t)$ *satisfy* $T(t) : X^0 \to X^0$, $T(t) : X_0 \to X_0$;

(iii) $T(t)$ *is point dissipative in* X;

(iv) $\gamma^+(U)$ *is bounded in* X *if* U *is bounded in* X;

(v) $T(t)$ *is asymptotically smooth;*

(vi) $\mathcal{A} = \cup_{x\in A_b}\omega(x)$ *is isolated and has an acyclic covering* N, *where* A_b *is the global attractor of* $T(t)$ *restricted to* X_0 *and* $N = \cup_{i=1}^k N_i$;

(vii) *for each* $N_i \in N$, $W^s(N_i) \cap X^0 = \emptyset$, *where* W^s *refers to the stable set.*

Then $T(t)$ *is a uniform repeller with respect to* X^0, *i.e. there is an* $\eta > 0$ *such that for any* $x \in X^0$, $\liminf_{t\to\infty} d(T(t)x, X_0) \geq \eta$.

Theorem 4.3. *System (3.1) is uniformly persistence if $\Re_0 > 1$ and the disease is initially present, that is, there exists $\eta > 0$ such that*

$$\liminf_{t\to\infty} S(t) \geq \eta, \ \liminf_{t\to\infty} E(t) \geq \eta, \ \liminf_{t\to\infty} I(t) \geq \eta \text{ and } \liminf_{t\to\infty} W(t) \geq \eta.$$

Proof. Let

$$X^0 = \{(C_1, \phi_1(\theta), \phi_2(\theta), C_2) \in X : \phi_1(\theta), \phi_2(\theta) > 0, \text{ for some } \theta < 0\},$$

$$X_0 = \{(C_1, \phi_1(\theta), \phi_2(\theta), C_2) \in X : \phi_1(\theta), \phi_2(\theta) = 0, \text{ for any } \theta \leq 0\}.$$

It is trivial to see that (i) and (ii) are satisfied, (iii) has been proved in Theorem 3.2, and (iv) can be easily proved by Theorem 3.2. Therefore, we only need to prove the other three conditions in Theorem 4.2 hold.

(v) Let

$$M := \max\left\{\frac{\Lambda}{\mu_1} + 1, \frac{\Lambda\sigma_1 p}{\alpha h} + \frac{\Lambda\sigma_2 q}{\gamma h} + 1\right\},$$

and

$$\Upsilon := \left\{\varphi \in C_\Delta : \sup_{s\leq 0} \varphi(s) e^{\frac{\Delta}{2}s} \leq M\right\}.$$

We can obtain that Υ is compact in C_Δ from Lemma 3.2 of [6]. For any bounded set $U \subset X$, let E_t and I_t be the segment of a solution with initial solution from U. Next we will demonstrate $\lim_{t\to\infty} d(E_t, \Upsilon) = 0$, and I_t can be treated similarly. By theorem 3.2, there exist $T > 0$ such that $E(t) \leq M$ for $t \geq T$ and $E(T) = M$ or $E(t) < M$ for all $t > 0$. For the first case, let K be the maximum of $E(t)$ on $[0, T]$. We define

$$\psi^t(s) := \begin{cases} E(t+s) & \text{if } T - s \leq s \leq 0, \\ M & \text{if } s \leq T - t. \end{cases}$$

It is obvious that $\psi^t \in \Upsilon$ and

$$d(E_t, \Upsilon) \leq d(E_t, \psi^t) = \sup_{s\leq 0} \left|E_t(s) - \psi^t(s)\right| e^{\Delta s}.$$

Then we have

$$\sup_{T-t\leq s\leq 0} \left|E_t(s) - \psi^t(s)\right| e^{\Delta s} = 0,$$

$$\sup_{-t\leq s\leq T-t} \left|E_t(s) - \psi^t(s)\right| e^{\Delta s} \leq (K + M) e^{\Delta(T-t)},$$

$$\sup_{s\leq -t} \left|E_t(s) - \psi^t(s)\right| e^{\Delta s} \leq (\| E_0 \| + M) e^{-\Delta t}.$$

Thus $\lim_{t\to\infty} d(E_t, \Upsilon) = 0$ is obtained. By following the same technique to the second case, we also get $\lim_{t\to\infty} d(E_t, \Upsilon) = 0$. Hence, we obtain (v).

(vi) It is obvious that $\mathcal{A} = \{E_0\}$, and it is isolated. Thus, the covering is simply $N = E_0$, which is acyclic (there is no orbit that connects E_0 to itself in X_0).

(vii) From (vi), we need to show that $W^s(E_0) \cap X^0 = \emptyset$. Suppose the contrary, that is there exists a solution $u_t \in X^0$ such that

$$\lim_{t\to\infty} S(t) = S_0, \lim_{t\to\infty} E_t = 0, \lim_{t\to\infty} I_t = 0, \lim_{t\to\infty} W(t) = 0.$$

There exists T_0 such that for any sufficiently small $\varepsilon, S(t) > S_0 - \varepsilon$ for all $t > T_0$. When $\Re_0 > 1$, we have

$$(S_0 - \varepsilon)\left[\beta_1 + \frac{\alpha\beta_2}{\gamma + \mu} + \frac{((\gamma + \mu)\sigma_1 p + \alpha\sigma_2 q)\beta_3}{(\gamma + \mu)d}\right] > \alpha + \mu.$$

There exists T_1 such that

$$(S_0-\varepsilon)\left[\beta_1+\frac{\alpha\beta_2}{\gamma+\mu}+\frac{((\gamma+\mu)\sigma_1\int_0^{T_1}f(\tau_1)d\tau_1+\alpha\sigma_2\int_0^{T_1}g(\tau_2)d\tau_2)\beta_3}{(\gamma+\mu)d}\right]>\alpha+\mu.$$

Due to $\lim_{t\to\infty}E_t=0$, $\lim_{t\to\infty}I_t=0$, there exists T_2 such that for all $t>T_2$, we have

$$\begin{aligned}&I(0)-\frac{\alpha E(t-\tau_1)}{\gamma+\mu_3}>0,\\&W(0)-\int_0^\infty\sigma_1E(t-\tau_1)e^{-\delta_1\tau_1}f_1(\tau_1)d\tau_1-\int_0^\infty\sigma_2I(t-\tau_2)e^{-\delta_2\tau_2}g_1(\tau_2)d\tau_2>0,\end{aligned}$$

and then, we can obtain

$$\begin{aligned}I(t)&\geq\frac{\alpha E(t)}{\gamma+\mu_3},\\W(t)&\geq\frac{\int_0^\infty\sigma_1E(t-\tau_1)f(\tau_1)d\tau_1+\int_0^\infty\sigma_2\frac{\alpha E(t-\tau_2)}{\gamma+\mu_3}g(\tau_2)d\tau_2}{d}\\&=\frac{\int_0^\infty[(\gamma+\mu)\sigma_1f(\tau)+\alpha\sigma_2g(\tau)]E(t-\tau)d\tau}{(\gamma+\mu_3)d}.\end{aligned}\tag{4.6}$$

Hence, for $t>T^*:=\max\{T_0,T_1,T_2\}$,

$$\begin{aligned}E'(t)\geq&(S_0-\varepsilon)\left[\beta_1E+\frac{\beta_2\alpha E}{\gamma+\mu_3}+\frac{\beta_3\int_0^\infty[(\gamma+\mu)\sigma_1f(\tau)+\alpha\sigma_2g(\tau)]E(t-\tau)d\tau}{(\gamma+\mu_3)d}\right]\\&-(\alpha+\mu_2)E\\\geq&(S_0-\varepsilon)\left[\beta_1E+\frac{\beta_2\alpha E}{\gamma+\mu_3}+\frac{\beta_3\int_0^{T^*}[(\gamma+\mu)\sigma_1f(\tau)+\alpha\sigma_2g(\tau)]E(t-\tau)d\tau}{(\gamma+\mu_3)d}\right]\\&-(\alpha+\mu_2)E.\end{aligned}$$

By the mean value theorem for integrals, we know that for any $t>T^*$ there is a $\xi_t\in[t-T^*,t]$ such that

$$\begin{aligned}E'(t)\geq&(S_0-\varepsilon)\left[\beta_1E+\frac{\beta_2\alpha E}{\gamma+\mu_3}+\frac{\beta_3E(\xi_t)\int_0^{T^*}[(\gamma+\mu)\sigma_1f(\tau)+\alpha\sigma_2g(\tau)]d\tau}{(\gamma+\mu_3)d}\right]\\&-(\alpha+\mu_2)E.\end{aligned}$$

If $E(t)\to0$, as $t\to\infty$, then by a standard comparison argument and the non-negativity, the solution $n(t)$ of

$$\begin{aligned}n'(t)=&(S_0-\varepsilon)\left[\beta_1n(t)+\frac{\beta_2\alpha n(t)}{\gamma+\mu_3}+\frac{\beta_3n(\xi_t)\int_0^{T^*}[(\gamma+\mu)\sigma_1f(\tau)+\alpha\sigma_2g(\tau)]d\tau}{(\gamma+\mu_3)d}\right]\\&-(\alpha+\mu_2)n(t),\end{aligned}$$

with initial data $n_0=E_0$ converges to 0 as well.

Define

$$G(t):=n(t)+\left[(\alpha+\mu_2)-(S_0-\varepsilon)(\beta_1+\frac{\beta_2\alpha}{\gamma+\mu_3})\right]\int_{\xi_t}^t n(s)ds.$$

Differentiating $G(t)$ with respect to t gives

$$\frac{dG}{dt}=\left\{(S_0-\varepsilon)\left[\beta_1+\frac{\beta_2\alpha}{\gamma+\mu}+\frac{\beta_3\int_0^{T^*}[(\gamma+\mu)\sigma_1f(\tau)+\alpha\sigma_2g(\tau)]d\tau}{(\gamma+\mu_3)d}\right]-\alpha+\mu\right\}n(\xi_t)\geq0.$$

Therefore, $G(t)$ goes to infinity or approaches a positive limit as $t \to \infty$. This is contrary to $\lim_{t\to\infty} G = 0$. Hence we have $W^s(E_0) \cap X^0 = \emptyset$. By Theorem 4.2 we can obtain

$$\liminf_{t\to\infty} \parallel E_t \parallel \geq \eta_0.$$

By similar estimates as in the proof of Theorem 3.2, we have

$$\liminf_{t\to\infty} E(t) \geq \eta_0.$$

Then by equations (4.6), we get

$$\begin{aligned}
&\liminf_{t\to\infty} I(t) \geq \frac{\alpha}{\gamma+\mu_3}\eta_0,\\
&\liminf_{t\to\infty} W(t) \geq \frac{(\gamma+\mu)\sigma_1+\alpha\sigma_2}{(\gamma+\mu_3)d}\eta_0.
\end{aligned}$$

It means that there exists

$$\eta := \min\left\{\eta_0, \frac{\alpha}{\gamma+\mu_3}\eta_0, \frac{(\gamma+\mu)\sigma_1+\alpha\sigma_2}{(\gamma+\mu_3)d}\eta_0\right\} > 0$$

such that

$$\liminf_{t\to\infty} S(t) \geq \eta,\ \liminf_{t\to\infty} E(t) \geq \eta,\ \liminf_{t\to\infty} I(t) \geq \eta \ \text{ and } \ \liminf_{t\to\infty} W(t) \geq \eta.$$

The proof is completed. □

4.3. **Global dynamical properties.**

Theorem 4.4.

(i) *The disease-free equilibrium E_0 is global asymptotically stable if $\Re_0 < 1$;*

(ii) *The endemic equilibrium E_1 is global asymptotically stable if $\Re_0 > 1$.*

Proof. (i) Define a Lyapunov functional V_1 as follows

$$\begin{aligned}
V_1 =&(S - S_0 - S_0 \ln\frac{S}{S_0}) + \Re_0 E + \frac{(\beta_2 d + \beta_3\sigma_2 q)S_0}{(\gamma+\mu_3)d} I + \frac{\beta_3 S_0}{d} W\\
&+ \frac{\beta_3\sigma_1 S_0}{d}\int_0^\infty\int_0^{\tau_1} E(t-s)f(\tau_1)dsd\tau_1 + \frac{\beta_3\sigma_2 S_0}{d}\int_0^\infty\int_0^{\tau_2} I(t-s)g(\tau_2)dsd\tau_2.
\end{aligned}$$

Then combining V_1 and the identity $\Lambda = \mu_1 S_0$, we obtain

$$\begin{aligned}
\frac{\mathrm{d}V_1}{\mathrm{d}t} =&\left(1-\frac{S_0}{S}\right)\frac{\mathrm{d}S}{\mathrm{d}t} + \Re_0\frac{\mathrm{d}E}{\mathrm{d}t} + \frac{(\beta_2 d+\beta_3\sigma_2 q)S_0}{(\gamma+\mu_3)d}\frac{\mathrm{d}I}{\mathrm{d}t} + \frac{\beta_3 S_0}{d}\frac{\mathrm{d}W}{\mathrm{d}t}\\
&+ \frac{\mathrm{d}}{\mathrm{d}t}\left(\frac{\beta_3\sigma_1 S_0}{d}\int_0^\infty\int_0^{\tau_1} E(t-s)f(\tau_1)dsd\tau_1\right) + \frac{\mathrm{d}}{\mathrm{d}t}\left(\frac{\beta_3\sigma_2 S_0}{d}\int_0^\infty\int_0^{\tau_2} I(t-s)g(\tau_2)dsd\tau_2\right)\\
=&\left(1-\frac{S_0}{S}\right)(\Lambda - \beta_1 SE - \beta_2 SI - \beta_3 SW - \mu_1 S) + \Re_0[\beta_1 SE + \beta_2 SI + \beta_3 SW - (\alpha+\mu_2)E]\\
&+ \frac{(\beta_2 d+\beta_3\sigma_2 q)S_0}{(\gamma+\mu_3)d}[\alpha E - (\gamma+\mu_3)I]\\
&+ \frac{\beta_3 S_0}{d}\left[\int_0^{+\infty}\sigma_1 E(t-\tau_1)f(\tau_1)\mathrm{d}\tau_1 + \int_0^{+\infty}\sigma_2 I(t-\tau_2)g(\tau_2)\mathrm{d}\tau_2 - dW\right]\\
&+ \frac{\beta_3\sigma_1 S_0}{d}\int_0^\infty [E(t)-E(t-\tau_1)]f(\tau_1)d\tau_1 + \frac{\beta_3\sigma_2}{d}\int_0^\infty [I(t)-I(t-\tau_2)]g(\tau_2)d\tau_2\\
=&\mu_1 S_0\left(2 - \frac{S_0}{S} - \frac{S}{S_0}\right) + (\Re_0 - 1)(\beta_1 SE + \beta_2 SI + \beta_3 SW).
\end{aligned}$$

Obviously, by the arithmetic-geometric mean, we have $\frac{dV_1}{dt} \leq 0$ when $\Re_0 < 1$. And $\frac{dV_1}{dt} = 0$ if and only if $E = 0, I = 0$ and $W = 0$. Let M_1 be the largest invariant set in $\left\{S(t), E(t), I(t), W(t) | \frac{dV_1}{dt} = 0\right\}$. It is easy to show that $M_1 = \{E_0\}$. It follows from LaSalle invariance principle that E_0 is global asymptotically stable if $\Re_0 < 1$.

(ii) Define a Lyapunov functional V_2 as follows

$$V_2 = V_S + V_E + V_I + V_W + V_{E^+} + V_{I^+},$$

where

$$\begin{aligned}
V_S &= S - S^* - S^* \ln \frac{S}{S^*},\\
V_E &= E - E^* - S^* \ln \frac{E}{E^*},\\
V_I &= \frac{1}{\alpha E^*}\left(\beta_2 S^* I^* + \frac{\sigma_2 I^* q \beta_3 S^* W^*}{\sigma_1 E^* p + \sigma_2 I^* q}\right)\left(I - I^* - I^* \ln \frac{I}{I^*}\right),\\
V_W &= \frac{\beta_3 S^* W^*}{\sigma_1 E^* p + \sigma_2 I^* q}\left(W - W^* - W^* \ln \frac{W}{W^*}\right),\\
V_{E^+} &= \frac{\sigma_1 E^* \beta_3 S^* w^*}{\sigma_1 E^* p + \sigma_2 I^* q}\int_0^\infty f(\tau_1)\int_0^{\tau_1}\left[\frac{E(t-s)}{E^*} - 1 - \ln \frac{E(t-s)}{E^*}\right] ds d\tau_1,\\
V_{I^+} &= \frac{\sigma_2 I^* \beta_3 S^* w^*}{\sigma_1 E^* p + \sigma_2 I^* q}\int_0^\infty g(\tau_2)\int_0^{\tau_2}\left[\frac{I(t-s)}{I^*} - 1 - \ln \frac{I(t-s)}{I^*}\right] ds d\tau_2.
\end{aligned}$$

Obviously, V_2 is non-negative at any time t. Differentiating V_S and employing the first equation of (3.3), we obtain

$$\begin{aligned}
\frac{\mathrm{d}V_S}{\mathrm{d}t} =& \left(1 - \frac{S^*}{S}\right)(\Lambda - \beta_1 SE - \beta_2 SI - \beta_3 SW - \mu_1 S)\\
=& \mu_1 S^*\left(2 - \frac{S^*}{S} - \frac{S}{S^*}\right) + \beta_1 S^* E^*\left(1 - \frac{S^*}{S} + \frac{E}{E^*} - \frac{SE}{S^* E^*}\right) + \beta_2 S^* I^*\left(1 - \frac{S^*}{S} + \frac{I}{I^*} - \frac{SI}{S^* I^*}\right)\\
&+ \beta_3 S^* W^*\left(1 - \frac{S^*}{S} + \frac{W}{W^*} - \frac{SW}{S^* W^*}\right).
\end{aligned}$$

Analogously, using V_E and the second equation of (3.3), we have

$$\begin{aligned}
\frac{\mathrm{d}V_E}{\mathrm{d}t} =& \left(1 - \frac{E^*}{E}\right)(\beta_1 SE + \beta_2 SI + \beta_3 SW - (\alpha + \mu_2)E)\\
=& \beta_1 S^* E^*\left(1 - \frac{S}{S^*} - \frac{E}{E^*} + \frac{SE}{S^* E^*}\right) + \beta_2 S^* I^*\left(1 - \frac{E}{E^*} + \frac{SI}{S^* I^*} - \frac{SIE^*}{S^* I^* E}\right)\\
&+ \beta_3 S^* W^*\left(1 - \frac{E}{E^*} + \frac{SW}{S^* W^*} - \frac{SWE^*}{S^* W^* E}\right).
\end{aligned}$$

Combining V_I and the thrid equation of (3.3), we get

$$\begin{aligned}
\frac{\mathrm{d}V_I}{\mathrm{d}t} =& \frac{1}{\alpha E^*}\left(\beta_2 S^* I^* + \frac{\sigma_2 I^* q \beta_3 S^* W^*}{\sigma_1 E^* p + \sigma_2 I^* q}\right)\left(1 - \frac{I^*}{I}\right)(\alpha E - (\gamma + \mu_3)I)\\
=& \left(\beta_2 S^* I^* + \frac{\sigma_2 I^* q \beta_3 S^* W^*}{\sigma_1 E^* p + \sigma_2 I^* q}\right)\left(1 - \frac{I}{I^*} + \frac{E}{E^*} - \frac{I^* E}{I E^*}\right).
\end{aligned}$$

Substituting the last equation of (3.3) into the differential process of V_W, we derive $\frac{\mathrm{d}V_W}{\mathrm{d}t}$ as

$$\begin{aligned}\frac{\mathrm{d}V_W}{\mathrm{d}t} =&\frac{\beta_3 S^* W^*}{\sigma_1 E^* p+\sigma_2 I^* q}\left(1-\frac{W^*}{W}\right)\left(\int_0^{+\infty}\sigma_1 E(t-\tau_1)f(\tau_1)\mathrm{d}\tau_1+\int_0^{+\infty}\sigma_2 I(t-\tau_2)g(\tau_2)\mathrm{d}\tau_2-dW\right)\\ =&\beta_3 S^* W^*\left(1-\frac{W}{W^*}\right)+\frac{\sigma_1 E^*\beta_3 S^* W^*}{\sigma_1 E^* p+\sigma_2 I^* q}\int_0^{\infty} f(\tau_1)\left[\frac{E(t-\tau_1)}{E^*}-\frac{W^* E(t-\tau_1)}{W E^*}\right]d\tau_1\\ &+\frac{\sigma_2 I^*\beta_3 S^* W^*}{\sigma_1 E^* p+\sigma_2 I^* q}\int_0^{\infty} g(\tau_2)\left[\frac{I(t-\tau_2)}{I^*}-\frac{W^* I(t-\tau_2)}{W I^*}\right]d\tau_2.\end{aligned}$$

Next, calculating the time derivative of $\frac{\mathrm{d}V_{E^+}}{\mathrm{d}t}$ and $\frac{\mathrm{d}V_{I^+}}{\mathrm{d}t}$ along (3.1), we have

$$\begin{aligned}\frac{\mathrm{d}V_{E^+}}{\mathrm{d}t} =&\frac{\sigma_1 E^*\beta_3 S^* w^*}{\sigma_1 E^* p+\sigma_2 I^* q}\int_0^{\infty} f(\tau_1)\frac{\mathrm{d}}{\mathrm{d}t}\int_0^{\tau_1}\left[\frac{E(t-s)}{E^*}-1-\ln\frac{E(t-s)}{E^*}\right]dsd\tau_1\\ =&\frac{\sigma_1 E^*\beta_3 S^* w^*}{\sigma_1 E^* p+\sigma_2 I^* q}\int_0^{\infty} f(\tau_1)\left[\frac{E(t)}{E^*}-\ln\frac{E(t)}{I^*}-\frac{E(t-\tau_1)}{E^*}+\ln\frac{E(t-\tau_1)}{E^*}\right]d\tau_1,\end{aligned}$$

and

$$\begin{aligned}\frac{\mathrm{d}V_{I^+}}{\mathrm{d}t} =&\frac{\sigma_2 I^*\beta_3 S^* w^*}{\sigma_1 E^* p+\sigma_2 I^* q}\int_0^{\infty} g(\tau_2)\frac{\mathrm{d}}{\mathrm{d}t}\int_0^{\tau_2}\left[\frac{I(t-s)}{I^*}-1-\ln\frac{I(t-s)}{I^*}\right]dsd\tau_2\\ =&\frac{\sigma_2 I^*\beta_3 S^* w^*}{\sigma_1 E^* p+\sigma_2 I^* q}\int_0^{\infty} g(\tau_2)\left[\frac{I(t)}{I^*}-\ln\frac{I(t)}{I^*}-\frac{I(t-\tau_2)}{I^*}+\ln\frac{I(t-\tau_2)}{I^*}\right]d\tau_2.\end{aligned}$$

Hence, we derive $\frac{dV_2}{dt}$ as follows

$$\begin{aligned}\frac{dV_2}{dt} =&(\mu_1 S^*+\beta_1 S^* E^*)\left(2-\frac{S^*}{S}-\frac{S}{S^*}\right)+\beta_2 S^* I^*\left(3-\frac{S^*}{S}-\frac{I^* E}{I E^*}-\frac{S I E^*}{S^* I^* E}\right)\\ &+\beta_3 S^* W^*\left(1-\frac{S^*}{S}+\ln\frac{S^*}{S}\right)+\beta_3 S^* W^*\left(1-\frac{S W E^*}{S^* W^* E}+\ln\frac{S W E^*}{S^* W^* E}\right)\\ &+\frac{\sigma_2 I^* q\beta_3 S^* W^*}{\sigma_1 E^* p+\sigma_2 I^* q}\left(1-\frac{E I^*}{E^* I}+\ln\frac{E I^*}{E^* I}\right)\\ &+\frac{\beta_3 S^* W^*\sigma_1 E^*}{\sigma_1 E^* p+\sigma_2 I^* q}\int_0^{\infty} f(\tau_1)\left[1-\frac{W^* E(t-\tau_1)}{W E^*}+\ln\frac{W^* E(t-\tau_1)}{W E^*}\right]d\tau_1\\ &+\frac{\beta_3 S^* W^*\sigma_2 I^*}{\sigma_1 E^* p+\sigma_2 I^* q}\int_0^{\infty} g(\tau_2)\left[1-\frac{W^* I(t-\tau_2)}{W I^*}+\ln\frac{W^* I(t-\tau_2)}{W I^*}\right]d\tau_2.\end{aligned}$$

Therefore, by the arithmetic-geometric mean, we have $\frac{dV_2}{dt}\leq 0$. And $\frac{dV_2}{dt}=0$ if and only if $S=S^*, EI^*=E^*I, W^*E(t-\tau_1)=WE^*, W^*I(t-\tau_2)=WI^*$ and $WE^*=W^*E$. Let M_2 be the largest invariant set in $\{S(t),E(t),I(t),W(t)\ \ |\frac{dV_2}{dt}=0\}$. It is easy to show that $M_2=\{E^*\}$. It follows from LaSalle invariance principle that E_1 is global asymptotically stable if $\Re_0>1$. □

5. Numerical simulations

In this section, some numerical simulations of system(2.3) are given to verify the above theoretical results. We employ parameters in [19], showing in Table 1. We take three kinds of kernel functions which are Dirac Delta function, weak delay kernel and strong delay kernel.

TABLE 1. Definitions and values of model parameters.

Parameters	Definition	Values	Reference
Λ	The birth rate	75 per day	Estimated
β_1	Transmission constant between S and E	3.11×10^{-8} /person/day	[23]
β_2	Transmission constant between S and I	1.62×10^{-8} /person/day	[23]
β_3	Transmission constant between S and V	1.03×10^{-8}/person/day	[29]
μ_1	Nature death rate	3.01×10^{-5}/person/day	[29]
μ_2	Death rate of E	0.0317/person/day	[4]
μ_3	Death rate of I	0.0357/person/day	[4]
$1/\alpha$	Incubation period	7 days	[22]
γ	Recovery rate	1/13 per day	[22]
σ_1	Virus shedding rate by exposed people	2.3 per person per day per ml	[29]
σ_2	Virus shedding rate by infected people	1.6 per person per day per ml	[29]
d	Removal rate of virus	1 per day	Estimated

Case 1: If $f_1(\tau_1)$ and $g_1(\tau_2)$ are determined by Dirac Delta function, let $f_1(\tau_1) = \delta(\tau_1 - \tau_3)$, $g_1(\tau_2) = \delta(\tau_2 - \tau_4)$, system(2.3) reduces to a system with discrete delay

$$\begin{cases} \dfrac{\mathrm{d}S(t)}{\mathrm{d}t} = \Lambda - \beta_1 SE - \beta_2 SI - \beta_3 SW - \mu_1 S, \\ \dfrac{\mathrm{d}E(t)}{\mathrm{d}t} = \beta_1 SE + \beta_2 SI + \beta_3 SW - (\alpha + \mu_2)E, \\ \dfrac{\mathrm{d}I(t)}{\mathrm{d}t} = \alpha E - (\gamma + \mu_3)I, \\ \dfrac{\mathrm{d}W(t)}{\mathrm{d}t} = \sigma_1 E(t-\tau_3)e^{-\delta_1\tau_3} + \sigma_2 I(t-\tau_4)e^{-\delta_2\tau_4} - dW. \end{cases} \tag{5.1}$$

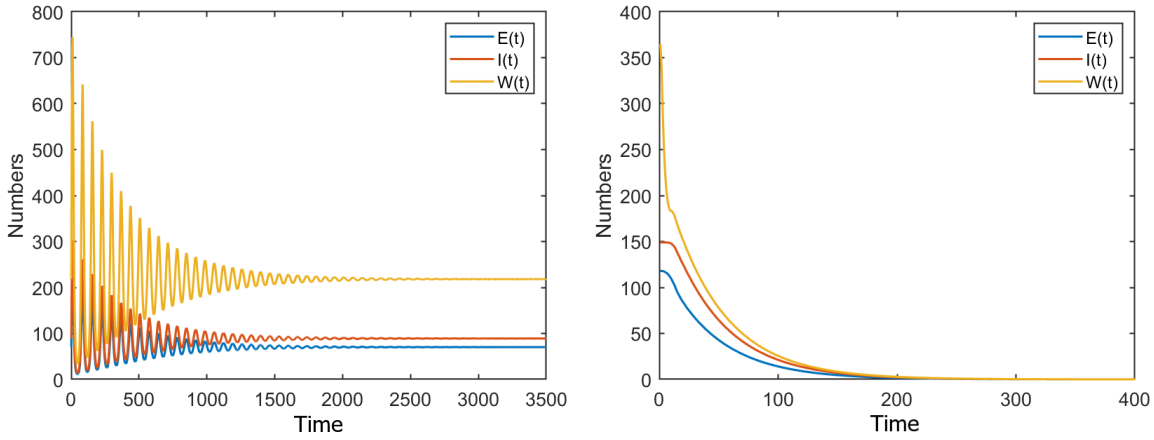
We choose initial values as $(S_0, E, I, W_0) = (2109695, 118, 149, 365)$. Let $\tau_3 = 3$, $\tau_4 = 3$, $\delta_1 = \alpha$, and $\delta_2 = \gamma$. From (3.2), we have $\Re_0 = 1.195$. As Figure 3(a) shows, the solution of (5.1) converges to the endemic equilibrium $E_1 = (2084961.584, 70.135, 88.964, 218.093)$. As SARS-CoV-2 can be efficiently inactivated by 75% ethanol, environmental disinfection is used to control the epidemic. Thus, assume that the environment is disinfected twice a day and $d = 2$. From (3.2), we have $\Re_0 = 0.966$. As Figure 3(a) shows, the solution of (5.1) is attracted by the disease-free equilibrium $E_0 = (2491694.352, 0, 0, 0)$, which means that the disease will eventually become extinct.

Case 2: Next, we consider the Gamma distribution delay kernel

$$G(u) = \frac{a^n u^{n-1} e^{-au}}{(n-1)!},\ n = 1, 2, ...,$$

where $a > 0$ is a constant and n is an integer, with the average delay $T = n/a$, and two special cases,

$$G(u) = ae^{-au}\ (n = 1),\ G(u) = a^2 ue^{-au}\ (n = 2),$$



(a) $\Re_0 = 1.195$, the time evolution of solution of (5.1). (b) $\Re_0 = 0.966$, the time evolution of solution of (5.1).

FIGURE 3. The kernel functions are determined by Dirac Delta function.

are called weak delay kernel and strong delay kernel, respectively [3]. If $f_1(\tau_1)$ and $g_1(\tau_2)$ are determined by weak delay kernel, we have

$$\begin{cases} \dfrac{\mathrm{d}S(t)}{\mathrm{d}t} = \Lambda - \beta_1 SE - \beta_2 SI - \beta_3 SW - \mu_1 S, \\ \dfrac{\mathrm{d}E(t)}{\mathrm{d}t} = \beta_1 SE + \beta_2 SI + \beta_3 SW - (\alpha + \mu_2)E, \\ \dfrac{\mathrm{d}I(t)}{\mathrm{d}t} = \alpha E - (\gamma + \mu_3)I, \\ \dfrac{\mathrm{d}W(t)}{\mathrm{d}t} = \displaystyle\int_0^{+\infty} \sigma_1 E(t-\tau_1)\frac{1}{\tau_3} e^{-(\frac{1}{\tau_3}+\delta_1)\tau_1}\mathrm{d}\tau_1 + \int_0^{+\infty} \sigma_2 I(t-\tau_2)\frac{1}{\tau_4} e^{-(\frac{1}{\tau_4}+\delta_2)\tau_2}\mathrm{d}\tau_2 - dW. \end{cases} \tag{5.2}$$

Define

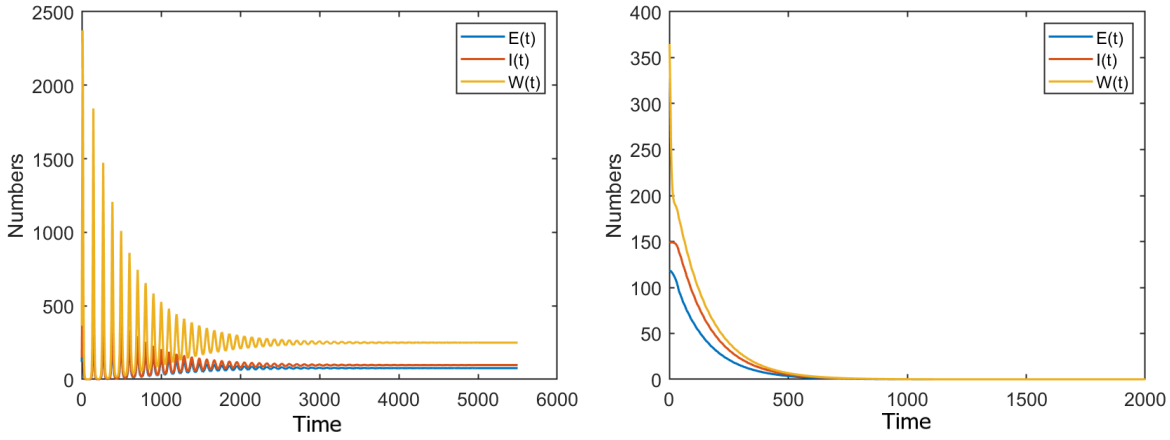
$$E_w = \int_0^{+\infty} E(t-\tau_1)\frac{1}{\tau_3} e^{-(\frac{1}{\tau_3}+\delta_1)\tau_1}\mathrm{d}\tau_1, I_w = \int_0^{+\infty} I(t-\tau_2)\frac{1}{\tau_4} e^{-(\frac{1}{\tau_4}+\delta_2)\tau_2}\mathrm{d}\tau_2,$$

then one obtains

$$\begin{cases} \dfrac{\mathrm{d}S(t)}{\mathrm{d}t} = \Lambda - \beta_1 SE - \beta_2 SI - \beta_3 SW - \mu_1 S, \\ \dfrac{\mathrm{d}E(t)}{\mathrm{d}t} = \beta_1 SE + \beta_2 SI + \beta_3 SW - (\alpha + \mu_2)E, \\ \dfrac{\mathrm{d}I(t)}{\mathrm{d}t} = \alpha E - (\gamma + \mu_3)I, \\ \dfrac{\mathrm{d}W(t)}{\mathrm{d}t} = \sigma_1 E_w + \sigma_2 I_w - dW, \\ \dfrac{\mathrm{d}E_w(t)}{\mathrm{d}t} = \dfrac{1}{\tau_3}E - (\dfrac{1}{\tau_3} + \delta_1)E_w, \\ \dfrac{\mathrm{d}I_w(t)}{\mathrm{d}t} = \dfrac{1}{\tau_4}I - (\dfrac{1}{\tau_4} + \delta_2)I_w. \end{cases} \tag{5.3}$$

We choose initial values as $(S_0, E, I, W_0) = (2109695, 118, 149, 365)$. Assuming $\tau_3 = 3, \tau_4 = 3$, and $\delta_1 = \alpha$, and $\delta_2 = \gamma$, then we have $\Re_0 = 1.217$. The time evolution of the solution of (5.2) is shown in Figure 4(a), which implies that system (5.2) transitions to a stable interior equilibrium state with an

oscillatory approach towards it. Then let $1/\mu_1 = 1$ and $1/\mu_2 = 1$, we have $\Re_0 = 0.972$. The solution of (5.2) converges to the disease-free equilibrium, and the result is shown in Figure 4(b).



(a) $\Re_0 = 1.217$, the time evolution of solution of (5.2). (b) $\Re_0 = 0.972$, the time evolution of solution of (5.2).

FIGURE 4. The kernel functions are determined by weak delay kernel.

Case 3: If $f_1(\tau_1)$ and $g_1(\tau_2)$ are determined by strong delay kernel, we can obtain

$$\begin{cases} \dfrac{\mathrm{d}S(t)}{\mathrm{d}t} = \Lambda - \beta_1 SE - \beta_2 SI - \beta_3 SW - \mu_1 S, \\ \dfrac{\mathrm{d}E(t)}{\mathrm{d}t} = \beta_1 SE + \beta_2 SI + \beta_3 SW - (\alpha + \mu_2)E, \\ \dfrac{\mathrm{d}I(t)}{\mathrm{d}t} = \alpha E - (\gamma + \mu_3)I, \\ \dfrac{\mathrm{d}W(t)}{\mathrm{d}t} = \displaystyle\int_0^{+\infty} \sigma_1 E(t-\tau_1)\frac{4}{\tau_3^2}\tau_1 e^{-(\frac{2}{\tau_3}+\delta_1)\tau_1}\mathrm{d}\tau_1 + \int_0^{+\infty} \sigma_2 I(t-\tau_2)\frac{4}{\tau_4^2}\tau_2 e^{-(\frac{2}{\tau_4}+\delta_2)\tau_2}\mathrm{d}\tau_2 - dW. \end{cases} \tag{5.4}$$

Define

$$E_{s1} = \int_0^{+\infty} E(t-\tau_1)\frac{2}{\tau_3}e^{-(\frac{2}{\tau_3}+\delta_1)\tau_1}\mathrm{d}\tau_1, E_{s2} = \int_0^{+\infty} E(t-\tau_1)\frac{4}{\tau_3^2}\tau_1 e^{-(\frac{2}{\tau_3}+\delta_1)\tau_1}\mathrm{d}\tau_1,$$

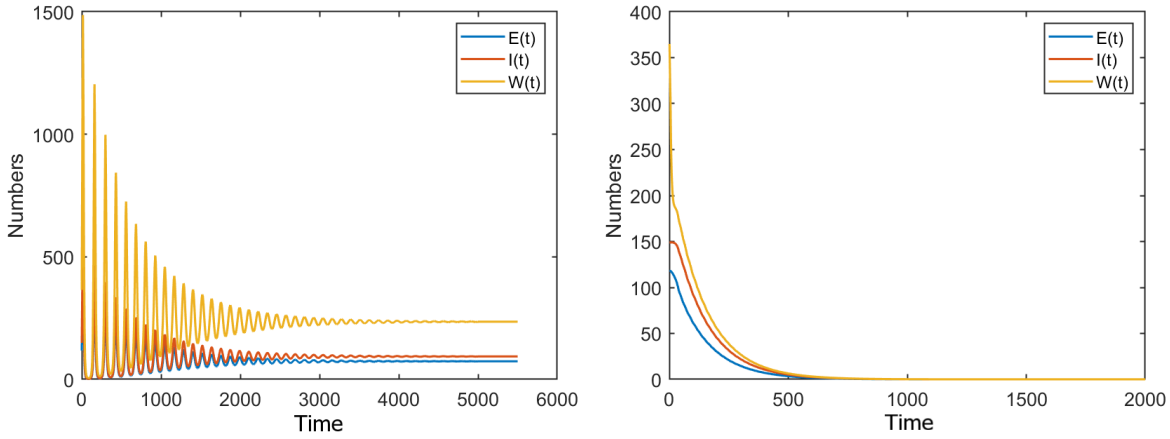
and

$$I_{s1} = \int_0^{+\infty} I(t-\tau_2)\frac{2}{\tau_4}e^{-(\frac{2}{\tau_4}+\delta_2)\tau_2}\mathrm{d}\tau_2, I_{s2} = \int_0^{+\infty} I(t-\tau_2)\frac{4}{\tau_4^2}\tau_2 e^{-(\frac{2}{\tau_4}+\delta_2)\tau_2}\mathrm{d}\tau_2,$$

then one gets

$$\begin{cases} \dfrac{\mathrm{d}S(t)}{\mathrm{d}t} = \Lambda - \beta_1 SE - \beta_2 SI - \beta_3 SW - \mu_1 S, \\ \dfrac{\mathrm{d}E(t)}{\mathrm{d}t} = \beta_1 SE + \beta_2 SI + \beta_3 SW - (\alpha + \mu_2)E, \\ \dfrac{\mathrm{d}I(t)}{\mathrm{d}t} = \alpha E - (\gamma + \mu_3)I, \\ \dfrac{\mathrm{d}W(t)}{\mathrm{d}t} = \sigma_1 E_{s2} + \sigma_2 I_{s2} - dW, \\ \dfrac{\mathrm{d}E_{s1}(t)}{\mathrm{d}t} = \dfrac{2}{\tau_3}E - (\dfrac{2}{\tau_3} + \delta_1)E_{s1}, \\ \dfrac{\mathrm{d}E_{s2}(t)}{\mathrm{d}t} = \dfrac{2}{\tau_3}E_{s1} - (\dfrac{2}{\tau_3} + \delta_1)E_{s2}, \\ \dfrac{\mathrm{d}I_{s1}(t)}{\mathrm{d}t} = \dfrac{2}{\tau_4}I - (\dfrac{2}{\tau_4} + \delta_2)I_{s1}, \\ \dfrac{\mathrm{d}I_{s2}(t)}{\mathrm{d}t} = \dfrac{2}{\tau_4}I_{s1} - (\dfrac{2}{\tau_4} + \delta_2)I_{s2}. \end{cases} \tag{5.5}$$

We choose initial values as $(S_0, E, I, W_0) = (2109695, 118, 149, 365)$. Assume that $\tau_3 = 3, \tau_4 = 3$, and $\delta_1 = \alpha$ and, $\delta_2 = \gamma$, then we have $\Re_0 = 1.207$. The evolution of the solution with time is shown in Figure 5(a), which means the solution of (5.4) eventually reaches a stable interior equilibrium state. Then let $d = 2$, we have $\Re_0 = 0.972$. The time evolution of the solution is shown in Figure 5(b), and we can find the disease will eventually die out.



(a) $\Re_0 = 1.207$, the time evolution of solution of (5.4).

(b) $\Re_0 = 0.972$, the time evolution of solution of (5.4).

FIGURE 5. The kernel functions are determined by strong delay kernel.

As shown in the above images, the stability of system (3.1) depends on the basic reproduction number $\Re_0$.

Assuming $\tau_3 = \tau_4$, Figure 6 depicts the effect of time delays on the basic reproduction number and illustrates that the basic reproduction number decreases as time delays increase. We can find that the variation of time delays have the greatest impact on the model where the kernel function is determined by the Dirac Delta function and the smallest effect on the model in which the kernel function is derived from the weak delay kernel. In a biological sense, models in which the kernel function is determined by

the Dirac Delta function indicate that exposed and infected individuals will only transmit the viruses to the environment on a given day after being infected. For this kind of model, increases in time delays mean that more exposed and infected individuals will enter other compartments before they have transmitted the viruses to the environment, and thus this type of model is more sensitive to change in time delays. Moreover, models whose kernel function is governed by a weak kernel function indicate that the largest proportion of viruses is transmitted to the environment when the exposed and infected individuals are newly infected, thus, time delays have the least effect on it.

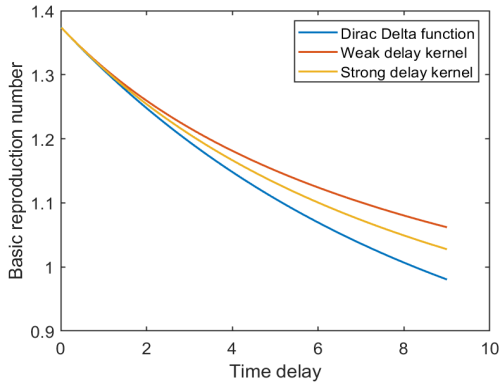
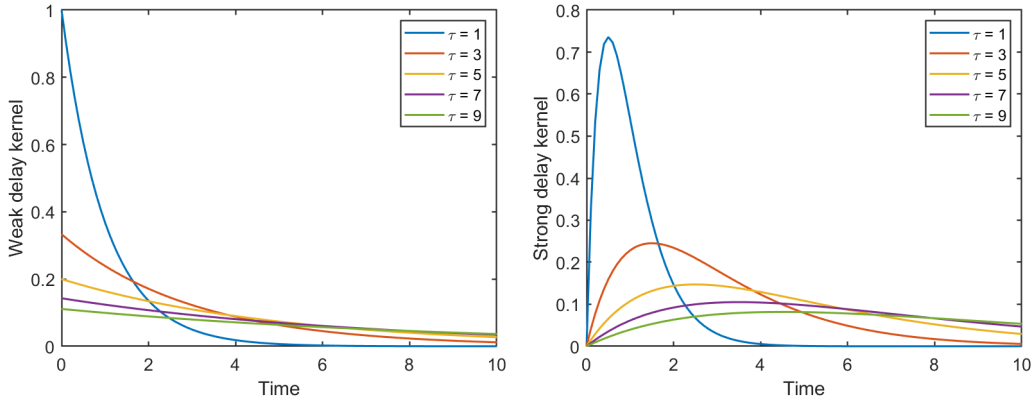


FIGURE 6. The evolution of the basic reproduction number with time delay.

Figure 7 demonstrates that the time delay affects the probability of transmitting the virus from an individual to the environment. For both weak and strong kernel functions, an increase in time delay leads to a decrease in the probability of transmitting the virus to the environment for those who are infected for a short period of time, and an increase in the probability of transmitting the virus to the environment for those who are infected for a long period of time. However, as time goes on, more individuals are removed from exposed or infected compartments, resulting in less virus transmission from individuals to the environment overall, which explains the results in Figure 6.



(a) The time evolution of week kernel functions. (b) The time evolution of strong kernel functions.

FIGURE 7. The time evolution of kernel functions.

6. Conclusions

In this paper, we have proposed an epidemic model with environmental infections by viruses, and viruses in the environment can infect susceptible individuals. The model contains two distributed delays, which describe the difference in viral load in the nasal and throat at different stages of exposed and infected individuals. By the dynamic analysis of this model, we have pointed out that the disease eventually disappears when $\Re_0 < 1$, and the disease persists when $\Re_0 > 1$.

The theoretical results indicate that environment-to-human transmission leads to the spread of infectious disease and causes difficulties in infectious disease eradication. Numerical simulations demonstrate that disinfection is considered an effective measure to control the spread of COVID-19. Moreover, the expression for the basic reproduction number indicates that reducing the potential for virus transmission from the host to the environment can decrease the level of endemic disease prevalence. Numerical simulations confirm the theoretical results.

From the perspective of the source of infection, developing specific medicine to improve the recovery rate of infected people will not only shorten the infection cycle but also effectively reduce the number of viruses in the environment. To cut off the transmission route of the virus, the government could organize environmental disinfection periodically, and enterprises should appropriately expand production to ensure the supply of protective items. Moreover, individuals could wear a mask, wash their hands frequently, and disinfect their surroundings after contacting others. For susceptible individuals, vaccination is an effective way to reduce the proportion of susceptible individuals and prevent the spread of infectious diseases.

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