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Research article

Dynamic analysis of a SIV Filippov system with media coverage and protective measures

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Abstract: This study aims to analyze a class of SIV systems considering the transmission rate influenced by media coverage and protective measures, in which the transmission rate is represented by a piecewise-smooth function. Firstly, for the SIV Filippov system, we take the dynamic behaviors of two subsystems into consideration, and obtain the basic reproduction number and the equilibria of the subsystems respectively. Secondly, based on the Filippov convex method, we calculate the sliding domain and the sliding mode equation, and further analyze the global dynamic behaviors of the system, through which we verify that there is no closed orbit in the system. Furthermore, we prove the global asymptotical stability of the disease-free equilibrium, two real equilibria, and the pseudo-equilibrium under certain conditions. The results demonstrate that the threshold value, the protective measures, and the media coverage could affect the number of infected individuals and the final scale of the disease. To prevent the spread of the disease, it is necessary to select an appropriate threshold and take applicable protective measures combined with media coverage. Lastly, we verify the validity of the results by numerical simulations.

Keywords: Filippov systems; global dynamics; stability; media coverage; protective measures
Mathematics Subject Classification: 34D05, 34D23, 92B05

1. Introduction

Modeling is an important method in various kinds of scientific research which has been widely used. In mathematics particularly, it abstracts the specific problem through a reasonable mathematical model, and simplifies the research object with mathematical language. A combination of quantitative and qualitative research approaches was used to reflect the characteristics of the object in biomathematical models [1–13]. For example, an infectious disease model can be analyzed based on data or transmission mechanisms to predict the scale, peak, the time duration of the spread, disease control strategy, and

the speed of the spread. In 1927, Kermack and McKendrick [14] first put forward the SIR model to study the epidemic law of infectious diseases, and took the plague in Bombay as an example to verify the feasibility of their model with data from December 1905 to July 1906. In this first proposed deterministic model, the population is split into three disjoint parts, where S denotes the number of the susceptible individuals, I denotes the infected individuals, and R denotes the number of individuals removed on account of death or recovery. Once S and I are determined, R is determined, so we can remove R from the model to get a simpler model. Based on the previously proposed SIR model, Kermack and McKendrick further proposed the SIS model in 1932 and obtained a threshold theory [15, 16]. Since then, the literature on deterministic models has proliferated, with researchers taking into account more realistic factors to make the model more realistic. For instance, considering the effect of vaccination on the spread of disease [17], another researchers further proposed the SIV model [18], in which V represents the vaccinated individuals.

Based on previous studies, we came to a conclusion that in the prevention and treatment of infectious diseases, people need to strengthen protective measures when the epidemic is getting severe and spreading. However, when the epidemic is gradually receding, if strong measures continue to be taken while the actual effect is not obvious, it will result in a great waste of social resources, which means that a parameter in the model can be considered piecewise, commonly referred to as a threshold policy (TP) [19]. In this case, consideration of a piecewise discontinuous model is the most reasonable choice, therefore the piecewise SIV model has been put forward.

Xiao et al. [20] extended the existing non-smooth model under the condition of imperfect vaccination, and proposed a general piecewise SIV model with nonlinear incidence to explore the impact of threshold policy. The stability and bifurcation of the equilibria of the system are studied by the right-hand discontinuous differential equation theory, stability theory and bifurcation theory, and the biological significance of the imperfect vaccination is revealed. The system is as follows:

$$\begin{cases} \dfrac{dS(t)}{dt} = \mu - \beta(1 - f\varepsilon(t))SI - \mu S - \phi S + \gamma I + \theta V, \\ \dfrac{dI(t)}{dt} = \beta(1 - f\varepsilon(t))SI + \sigma\beta(1 - f\varepsilon(t))VI - (\mu + \gamma)I, \\ \dfrac{dV(t)}{dt} = \phi S - \sigma\beta(1 - f\varepsilon(t))VI - \mu V - \theta V, \end{cases} \tag{1.1}$$

where

$$\varepsilon(t) = \begin{cases} 0, & I < I_c, \\ 1, & I > I_c, \end{cases}$$

here S, I, V represent the numbers of the susceptible, infected, and vaccinated individuals at the time t, with all the parameters being positive constants respectively. μ is the natural birth and death rate, β represents the coefficient of transmission rate, ϕ denotes the vaccination rate of the susceptible population, γ is the cure rate of infected individuals, θ represents the rate at which the vaccinated people lose immunity and become susceptible. f denotes the level of protective measures taken by the crowd, when the infected population reaches a certain threshold I_c, people begin to take precautionary measures, such as wearing masks in public, washing hands frequently, not gathering in public places, and self-isolation. In addition, β decreases to $\beta(1 - f\varepsilon(t))$ where ε is a piecewise function whose value depends on $I - I_c$, $\varepsilon = 0$ when $I - I_c < 0$. In practical terms, the number of infected people is relatively

small and no precautionary measures are taken by the general population, but individuals begin to get concerned and take measures to reduce their risk of infection if $I - I_c > 0$, i.e., $\varepsilon = 1$. The parameter $\sigma(0 < \sigma < 1)$ is the measurement of the effectiveness of the vaccine as a multiplier to the infection rate, where $\sigma = 0$ indicates that the vaccine is completely effective, while $\sigma = 1$ indicates the vaccine is completely ineffective. Figure 1 shows the diagram of the system (1.1).

In general, there are three links in the transmission of infectious diseases: source of infection, transmission route, and susceptible population, we can also response to infectious diseases from these three links. First, for the source of infection, we can isolate and treat the infected [21] to reduce the possibility of the spread of pathogen; second, we should reduce exposure and cut off transmission routes [22]; finally, people should take protective measures in the first place to reduce their risk of infection, for example, wearing a mask [23–25] and not gathering in public places. Society can also increase people's awareness of self-protection through media coverage [26, 27]. Besides these approaches, vaccines are also very effective [28] and people are immunized by vaccination, which provides some protection even if they are accidentally exposed to the virus.

At the early stage of an outbreak, the public and the media know little about the disease. Only when it has spread for some time and the number of infected people has reached a certain amount could the media cover it. Media coverage could raise people's awareness of the risks of infectious diseases to understand their transmission ways and means, and update essential information like the number of infections and the location of outbreaks. In this way, people could raise their awareness, avoid infected areas, and reduce their contact with infected people as well as the risk of contracting the disease. Therefore, the number of infections will decrease with the increase of media coverage, which directly affects the transmission term. But the the specific influence of media on propagation term is not the intrinsic deterministic factor, Cui et al. [29] proposed the negative exponential term function in transmission term. The coefficient of transmission rate usually expressed as β is reduced to $\beta \exp(-\alpha I)$ [30, 31]. If there is less media coverage and fewer people who know about it, infectious diseases could still spread extensively. Massive media coverage will greatly increase public awareness of infectious diseases, but it will consume large amounts of human and material resources. Therefore, we introduced the measure of media coverage combined with protective measures and the same threshold as the standard to consider the transmission rate in segments. In addition, the process during which the vaccinated individuals lose their immunity and become infected could be divided into two stages. First, the vaccinated individuals lose their immunity and become susceptible; afterward, they transform into infected individuals. Therefore, we do not consider the case that V directly transforms into I.

Based on the above reasons, this paper considers the extension model of the system (1.1) as follows:

$$\begin{cases} \dfrac{dS(t)}{dt} = \mu - \beta(1 - \varepsilon f)\exp(-\varepsilon\alpha I)SI - \mu S - \phi S + \gamma I + \theta V, \\ \dfrac{dI(t)}{dt} = \beta(1 - \varepsilon f)\exp(-\varepsilon\alpha I)SI - (\mu + \gamma)I, \\ \dfrac{dV(t)}{dt} = \phi S - \mu V - \theta V, \end{cases} \tag{1.2}$$

where

$$\varepsilon = \begin{cases} 0, & I < I_c, \\ 1, & I > I_c, \end{cases}$$

here α is the influence coefficient of media coverage. Suppose the total population is a constant, might as well we set $S + I + V = 1$, i.e., $\frac{d(S+I+V)}{dt} = 0$. Figures 2 and 3 show the diagram of the system and the threshold policy.

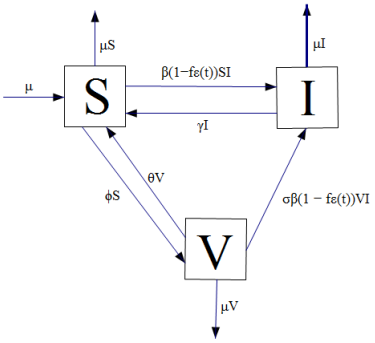

Figure 1. Diagram of infectious disease transmission system (1.1).

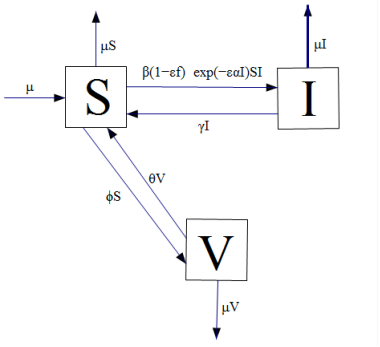

Figure 2. Diagram of infectious disease transmission system (1.2).

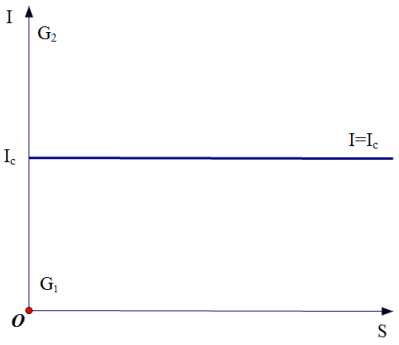

Figure 3. Schematic diagram of the threshold policy of system (1.2).

There have been extensive studies and application of discontinuous models, especially the Filippov system, which are described by differential equations with discontinuous right-hand sides. Tang et al. [30] investigated the influence of media coverage, vaccination, and treatment on disease dynamics in a non-smooth SIR Filippov system. Wang et al. [32] have studied the model of non-smooth plant diseases with economic thresholds and cultivation strategies, and a Filippov system was proposed according to a threshold of the number of infected plants. The equilibrium, heteroclitic orbit, and global dynamic behaviors of the system were analyzed. Li et al. [33] put forward the non-smooth boundary of the discontinuous plant disease model. In this system, they combined two thresholds which are the infection threshold I_T and the ratio threshold I/S and divided the plane into two regions, then the system dynamic behavior is analyzed in detail. There are many similar works and more questions for researchers to discuss. Inspired by previous studies, media coverage and protection measures are important for disease transmission dynamics, but there are few papers consider both. Then this paper aims to further investigate how the protection level f and the influence coefficient of media coverage α influence the epidemic trends. In this paper, the basic reproduction numbers of the two subsystems are different, so the global dynamic behavior analysis is more complex. In addition, the importance of the threshold I_c on the number of infected persons and the final equilibrium state of the disease

was analyzed numerically. In terms of theoretical analysis, we used the methods of discontinuous differential inclusion theory, non-smooth system analysis, Filippov convex method [34] etc.

An outline of this paper is as follows. In Section 2, we expound on the knowledge of the Filippov system and explain the positivity and boundedness of the solutions with the initial value problem. The dynamic behavior analysis of the subsystems is presented in Section 3. The local stability and global stability of the equilibria of the two subsystems are analyzed respectively by calculating the basic reproduction number. In Section 4, the sliding mode dynamics are studied. In Section 5, we first exclude the existence of the closed orbit, then the global asymptotic stability of the real equilibrium point and the pseudo-equilibrium is analyzed. Finally, there are numerical simulations and the corresponding biological implications.

2. Preliminaries

In this part, we first give some definitions about the to analyze the dynamic behaviors of the system. Then, the positivity and boundedness of the solutions with the initial value problem will be analyzed [8, 32].

For the system (1.2), suppose $V = 1 - S - I$, then the system (1.2) can be converted to

$$\begin{cases} \dfrac{dS(t)}{dt} = \mu - \beta(1-\varepsilon f)\exp(-\varepsilon\alpha I)SI - \mu S - \phi S + \gamma I + \theta(1-S-I), \\ \dfrac{dI(t)}{dt} = \beta(1-\varepsilon f)\exp(-\varepsilon\alpha I)SI - (\mu+\gamma)I. \end{cases} \tag{2.1}$$

Let $R_+^2 = \{X = (S, I)^T | S \geq 0, I \geq 0\}$, $H(X) = I - I_c$,

$$F_1 = \begin{bmatrix} \mu - \beta SI - \mu S - \phi S + \gamma I + \theta(1-S-I) \\ \beta SI - (\mu+\gamma)I \end{bmatrix},$$

and

$$F_2 = \begin{bmatrix} \mu - \beta(1-f)\exp(-\alpha I)SI - \mu S - \phi S + \gamma I + \theta(1-S-I) \\ \beta(1-f)\exp(-\alpha I)SI - (\mu+\gamma)I \end{bmatrix}.$$

So we can write the system (2.1) to the following generic planar Filippov system:

$$\frac{dX(t)}{dt} = \begin{cases} F_1(X), & X \in G_1, \\ F_2(X), & X \in G_2, \end{cases} \tag{2.2}$$

where $G_1 = \{X \in R_+^2 | H(X) < 0\}$, $G_2 = \{X \in R_+^2 | H(X) > 0\}$. Suppose $H_X(X)$ is the gradient of $H(X)$ and directs to G_2, $H_X(X) = (0, 1)^T$, $\langle \cdot, \cdot \rangle$ is the standard scalar product. Then $\Sigma = \{X \in R_+^2 | H(X) = 0\}$ can be split into three regions:

(a) $\Sigma_c \subset \Sigma$ is called the crossing region if the product $\langle H_X(X), F_1(X)\rangle\langle H_X(X), F_2(X)\rangle > 0$;

(b) $\Sigma_s \subset \Sigma$ is called the sliding region if the product $\langle H_X(X), F_1(X)\rangle > 0$, $\langle H_X(X), F_2(X)\rangle < 0$;

(c) $\Sigma_e \subset \Sigma$ is called the escaping region if the product $\langle H_X(X), F_1(X)\rangle < 0$, $\langle H_X(X), F_2(X)\rangle > 0$.

Here are some definitions about the Filippov system [31, 35–38]:

Definition 2.1. *(The classification of equilibrium) For the system* (2.2)*,*

(a) A real equilibrium X_R is described as $F_1(X_R) = 0$, $X_R \in G_1$ or $F_2(X_R) = 0$, $X_R \in G_2$.

(b) A virtual equilibrium X_V is described as $F_1(X_V) = 0$, $X_V \in G_2$ or $F_2(X_V) = 0$, $X_V \in G_1$.

(c) A pseudo-equilibrium X_p is described as $\lambda F_1(X_p) + (1-\lambda)F_2(X_p) = 0$, $0 < \lambda < 1$, where

$$\lambda = \frac{\langle H_X(X_p), F_2(X_p)\rangle}{\langle H_X(X_p), F_2(X_p) - F_1(X_p)\rangle}.$$

(d) A boundary equilibrium X_B is described as $F_1(X_B) = 0$, $H(X_B) = 0$ or $F_2(X_B) = 0$, $H(X_B) = 0$.

Definition 2.2. *(The tangent point) A tangent point X_T of the system* (2.2) *is defied as:*

(a) $F_i(X_T) \neq 0$, $i = 1, 2$,

(b) $\langle F_1(X_T), H_X(X_T)\rangle = 0$ or $\langle F_2(X_T), H_X(X_T)\rangle = 0$, i.e. the trajectory of $G_i (i = 1, 2)$ is tangent to the sliding region Σ.

In order to investigate the dynamic behaviors of the system (2.1), in the following, we give that the solutions of the system (2.1) with initial value are positive and bounded.

Proposition 2.1. *Supposing that $(S(t), I(t))$ is a solution of the system* (2.1) *with $S(0) = S_0 \geq 0$, $I(0) = I_0 \geq 0$ on $[0, T)$, where $T \in (0, +\infty]$, then $S(t) \geq 0$ and $I(t) \geq 0$ for all $t \in [0, T)$.*

Proof. According to the definition of the solution of the system (2.1) in Filippov sense [34] and the first equation of the system (2.1), we have

$$\left.\frac{dS}{dt}\right|_{S=0} = \mu + \gamma I + \theta(1 - I) > 0.$$

Consider $S(0) = S_0 \geq 0$, so that $S(t) \geq 0$, $t \in [0, T)$. Then according to the second equation of the system (2.1)

$$\frac{dI}{dt} = \beta(1 - \varepsilon f)\exp(-\varepsilon\alpha I)SI - (\mu + \gamma)I = (\beta(1 - \varepsilon f)\exp(-\varepsilon\alpha I)S - (\mu + \gamma))I. \tag{2.3}$$

Note that $I(0) = I_0 \geq 0$, then

$$I(t) = I_0 e^{\int_0^t \beta(1-\varepsilon f)\exp(-\varepsilon\alpha I(\xi))S(\xi) - (\mu+\gamma))d\xi} \geq 0.$$

Then, $I(t) \geq 0$ for $t \in [0, T)$. Thus, $S(t) \geq 0$ and $I(t) \geq 0$ for all $t \in [0, T)$. □

Suppose $(S(t), I(t))$ is a solution of the system (2.1) with $S(0) = S_0 \geq 0$ and $I(0) = I_0 \geq 0$ on $[0, T)$, where $T \in [0, +\infty)$. Since $S + I + V = 1$, it is obvious that the solution $(S(t), I(t))$ is bounded on $[0, +\infty)$. And it is easy to obtain the invariant region is defined as $\Omega = \{(S, I) \in R_+^2 | 0 < S(t) + I(t) \leq 1\}$.

3. Global dynamic analysis of subsystems

In this part, we rewrite the system (2.2) to two subsystems, then the basic reproduction numbers and their equilibria are discussed respectively, so the dynamic behaviors of the two subsystems can be analyzed through above results.

If $I < I_c$, then system (2.2) specifies to the subsystem:

$$\begin{cases} \dfrac{dS(t)}{dt} = \mu - \beta S I - \mu S - \phi S + \gamma I + \theta(1 - S - I), \\ \dfrac{dI(t)}{dt} = \beta S I - (\mu + \gamma) I. \end{cases} \tag{3.1}$$

If $I > I_c$, then system (2.2) is:

$$\begin{cases} \dfrac{dS(t)}{dt} = \mu - \beta(1 - f)\exp(-\alpha I) S I - \mu S - \phi S + \gamma I + \theta(1 - S - I), \\ \dfrac{dI(t)}{dt} = \beta(1 - f)\exp(-\alpha I) S I - (\mu + \gamma) I. \end{cases} \tag{3.2}$$

3.1. Global dynamic analysis of subsystem (3.1)

By the regeneration matrix method [39], we calculate the basic reproduction number of the system (3.1) as follows

$$R_0^1 = \frac{\beta(\mu + \theta)}{(\mu + \gamma)(\mu + \phi + \theta)}. \tag{3.3}$$

Meanwhile, the system (3.1) has the following two equilibria, a disease-free equilibrium $E_0 = (\frac{\mu+\theta}{\mu+\phi+\theta}, 0)$ and a positive endemic equilibrium $E_1 = (\frac{\mu+\gamma}{\beta}, 1 - \frac{(\mu+\gamma)(\mu+\phi+\theta)}{\beta(\mu+\theta)})$ when $R_0^1 > 1$.

Theorem 3.1. *For the subsystem* (3.1)*, the disease-free equilibrium E_0 is globally asymptotically stable (GAS) if $R_0^1 < 1$, while the endemic equilibrium E_1 is GAS if $R_0^1 > 1$.*

Proof. For the subsystem (3.1), we calculate its Jacobian matrix to be

$$J = \begin{bmatrix} -\beta I - (\mu + \phi + \theta) & -\beta S + \gamma - \theta \\ \beta I & \beta S - (\mu + \gamma) \end{bmatrix}.$$

Then on the disease-free equilibrium E_0, the Jacobian matrix can be written as

$$J_{E_0} = \begin{bmatrix} -(\mu + \phi + \theta) & -\dfrac{\beta(\mu + \theta)}{\mu + \phi + \theta} + \gamma - \theta \\ 0 & \dfrac{\beta(\mu + \theta)}{\mu + \phi + \theta} - (\mu + \gamma) \end{bmatrix}.$$

For $R_0^1 = \frac{\beta(\mu+\theta)}{(\mu+\gamma)(\mu+\phi+\theta)} < 1$, then $\frac{\beta(\mu+\theta)}{\mu+\phi+\theta} < \mu + \gamma$, i.e., $\frac{\beta(\mu+\theta)}{\mu+\phi+\theta} - (\mu + \gamma) < 0$. Additionally, $-(\mu + \phi + \theta) < 0$, thus E_0 is a locally asymptotically stable node(LAS). Furthermore, it is a saddle if $R_0^1 > 1$.

Likewise, the Jacobian matrix of the system (3.1) on the endemic equilibrium is

$$J_{E_1} = \begin{bmatrix} -\dfrac{\beta(\mu + \theta) - (\mu + \gamma)(\mu + \phi + \theta)}{(\mu + \theta)} - (\mu + \phi + \theta) & -(\mu + \theta) \\ \dfrac{\beta(\mu + \theta) - (\mu + \gamma)(\mu + \phi + \theta)}{(\mu + \theta)} & 0 \end{bmatrix}.$$

Sine $R_0^1 = \frac{\beta(\mu+\theta)}{(\mu+\gamma)(\mu+\phi+\theta)} > 1$, then $tr(J_{E_1}) = -\frac{\beta(\mu+\theta)-(\mu+\gamma)(\mu+\phi+\theta)}{(\mu+\theta)} - (\mu+\phi+\theta) < 0$, $det(J_{E_1}) = \beta(\mu+\theta) - (\mu+\gamma)(\mu+\phi+\theta) > 0$. So J_{E_1} has two negative characteristic roots or a pair of conjugate negative roots with negative real parts. So E_1 is LAS. Meanwhile, the characteristic polynomial of the system (3.1) is $\lambda^2 - tr(J_{E_1})\lambda + det(J_{E_1}) = 0$. Thus, E_1 is a stable focus if $\Delta < 0$, while it is a stable node if $\Delta > 0$, where $\Delta = tr^2(J_{E_1}) - 4det(J_{E_1})$.

Next we will prove the GAS of the two equilibria, respectively. For the system (3.1), let $F_1 = (F_1^1, F_1^2)^T$ and select the Dulac function $B(S, I) = \frac{1}{SI}$. Then

$$\begin{aligned}\frac{\partial(BF_1^1)}{\partial S} + \frac{\partial(BF_1^2)}{\partial I} &= \frac{\partial(\frac{\mu+\theta}{SI} - \beta - \frac{\mu+\phi+\theta}{I} + \frac{\gamma-\theta}{S})}{\partial S} + \frac{\partial(\beta - \frac{\mu+\gamma}{S})}{\partial I} \\ &= -\frac{\mu+\theta}{S^2 I} - \frac{\gamma-\theta}{S^2}.\end{aligned}$$

Since $I < 1$, then $-\frac{\mu+\theta}{S^2 I} < -\frac{\mu+\theta}{S^2}$, and all the parameters are positive constant, thus $\frac{\partial(BF_1^1)}{\partial S} + \frac{\partial(BF_1^2)}{\partial I} < -\frac{\mu+\theta}{S^2} - \frac{\gamma-\theta}{S^2} = -\frac{\mu+\gamma}{S^2} < 0$. According to the Bendixson-Dulac criterion [40], the subsystem (3.1) has no close orbit. So the unique endemic equilibrium E_1 of the subsystem (3.1) is GAS. Similarly, the disease-free equilibrium E_0 of the subsystem (3.1) is GAS when $R_0^1 < 1$. □

3.2. Global dynamic analysis of subsystem (3.2)

We calculate the basic reproduction number of the system (3.2) as follows

$$R_0^2 = \frac{\beta(1-f)(\mu+\theta)}{(\mu+\gamma)(\mu+\phi+\theta)}. \tag{3.4}$$

Meanwhile, it's easy to derive the disease-free equilibrium of the system (3.2) is $E_0 = (\frac{\mu+\theta}{\mu+\phi+\theta}, 0)$. Let $\frac{dS(t)}{dt} = 0$ and $\frac{dI(t)}{dt} = 0$, we obtain that

$$\begin{cases}\mu - \beta(1-f)\exp(-\alpha I_2)S_2 I_2 - \mu S_2 - \phi S_2 + \gamma I_2 + \theta(1 - S_2 - I_2) = 0, \\ S_2 = \dfrac{\mu+\gamma}{\beta(1-f)}\exp(\alpha I_2).\end{cases} \tag{3.5}$$

Then we get a equation for I_2

$$\mu + \theta - (\mu+\theta)I_2 - \frac{(\mu+\gamma)(\mu+\phi+\theta)}{\beta(1-f)}\exp(\alpha I_2) = 0.$$

Let $g(I) = \mu+\theta-(\mu+\theta)I - \frac{(\mu+\gamma)(\mu+\phi+\theta)}{\beta(1-f)}\exp(\alpha I)$. We derive that $g(0) = \mu+\theta-\frac{(\mu+\gamma)(\mu+\phi+\theta)}{\beta(1-f)} > 0$ for $R_0^2 > 1$, and $g(1) = -\frac{(\mu+\gamma)(\mu+\phi+\theta)}{\beta(1-f)}\exp(\alpha) < 0$. Moreover, we have

$$\frac{d(g(I))}{dI} = -(\mu+\theta) - \frac{\alpha(\mu+\gamma)(\mu+\phi+\theta)}{\beta(1-f)}\exp(\alpha I) < 0$$

for all $I \geq 0$. Such that there exist unique I_2 for $g(I) = 0$ in $(0, 1)$.
Since $g(I_2) = 0$, we obtain

$$\mu + \theta - (\mu+\theta)I_2 - \frac{(\mu+\gamma)(\mu+\phi+\theta)}{\beta(1-f)}\exp(\alpha I_2) = 0,$$

simply deform the above formula, we have

$$\alpha(1-I_2)\exp(\alpha(1-I_2)) = \frac{\alpha(\mu+\gamma)(\mu+\phi+\theta)}{\beta(1-f)(\mu+\theta)}\exp(\alpha).$$

According to the definition of Lambert W function [41], we let $z = \frac{\alpha(\mu+\gamma)(\mu+\phi+\theta)}{\beta(1-f)(\mu+\theta)}\exp(\alpha)$, and $W(z) = \alpha(1-I_2)$, then $W(\frac{\alpha(\mu+\gamma)(\mu+\phi+\theta)}{\beta(1-f)(\mu+\theta)}\exp(\alpha)) = \alpha(1-I_2)$, thus we derive $I_2 = 1 - \frac{1}{\alpha}W(\frac{\alpha(\mu+\gamma)(\mu+\phi+\theta)}{\beta(1-f)(\mu+\theta)}\exp(\alpha))$.

When $R_0^2 > 1$, there is a unique positive equilibrium $E_2 = (S_2, I_2) = (\frac{\mu+\gamma}{\beta(1-f)}\exp(\alpha I_2), I_2)$, where $I_2 = 1 - \frac{1}{\alpha}W(\frac{\alpha(\mu+\gamma)(\mu+\phi+\theta)}{\beta(1-f)(\mu+\theta)}\exp(\alpha))$, i.e., E_2 is the endemic equilibrium of subsystem (3.2).

Theorem 3.2. *For the subsystem (3.2), the disease-free equilibrium E_0 is GAS if $R_0^2 < 1$, whereas the endemic equilibrium E_2 is GAS if $R_0^2 > 1$.*

Proof. The proof of LAS is similar to the Theorem 3.1, so we omit it here. Now we similarly proof the GAS of the two equilibria. Select the Dulac function $B(S, I) = \frac{1}{SI}$. Then

$$\begin{aligned}\frac{\partial(BF_2^1)}{\partial S} + \frac{\partial(BF_2^2)}{\partial I} &= \frac{\partial(\frac{\mu+\theta}{SI} - \beta(1-f)\exp(-\alpha I) - \frac{\mu+\phi+\theta}{I} + \frac{\gamma-\theta}{S})}{\partial S} \\ &\quad + \frac{\partial(\beta(1-f)\exp(-\alpha I) - \frac{\mu+\gamma}{S})}{\partial I} \\ &= -\frac{\mu+\theta}{S^2 I} - \frac{\gamma-\theta}{S^2} - \alpha\beta(1-f)\exp(-\alpha I) \\ &< 0.\end{aligned}$$

According to the Bendixson-Dulac criterion , the subsystem (3.2) has no close orbit. So the disease-free equilibrium E_0 of the subsystem (3.2) is GAS if $R_0^2 < 1$, and the unique endemic equilibrium E_2 of the subsystem (3.2) is GAS if $R_0^2 > 1$. □

Remark 3.1. *The disease-free equilibrium point E_0 is always real for the subsystem (3.1), and always virtual for the subsystem (3.2). When $R_0^1 > 1$, the endemic equilibrium E_1 is real (virtual) if and only if (iff) $I_1 - I_c < 0 (> 0)$, while it is a boundary equilibrium iff $I_1 - I_c = 0$. When $R_0^2 > 1$, the endemic equilibrium E_2 is virtual (real) iff $I_2 - I_c < 0 (> 0)$, while it is a boundary equilibrium iff $I_2 - I_c = 0$. The pseudo-equilibrium will be described in Section 4.*

4. Sliding mode dynamics

In this part, we mainly solve the sliding domain and sliding mode equation of the system (2.2). According to the Section 2, the sliding domain is $\langle H_X(X), F_1(X)\rangle > 0$, and $\langle H_X(X), F_2(X)\rangle < 0$.
To obtain the sliding domain, we let

$$g_1(S) = \langle H_X(X), F_1(X)\rangle = \beta S I - (\mu+\gamma)I > 0.$$

Thus $S > \frac{\mu+\gamma}{\beta} = S_1$. Similarly, we have

$$g_2(S) = \langle H_X(X), F_2(X)\rangle = \beta(1-f)\exp(-\alpha I)S I - (\mu+\gamma)I < 0,$$

then $S < \frac{\mu+\gamma}{\beta(1-f)} \exp(\alpha I) = S_2$. It is obvious that $S_1 < S_2$. Therefore, the sliding domain is

$$\Sigma_s = \{(S, I) \in R_+^2 | \frac{\mu+\gamma}{\beta} < S < \frac{\mu+\gamma}{\beta(1-f)} \exp(\alpha I_c), I = I_c\}.$$

According to the famous Filippov convex method [34], the dynamic on the sliding domain Σ_s of the Fiippov system (2.2) is represented as follows:

$$\begin{aligned}
&\lambda F_1 + (1-\lambda)F_2 \\
&= \lambda \begin{bmatrix} \mu - \beta S I - \mu S - \phi S + \gamma I + \theta(1 - S - I) \\ \beta S I - (\mu+\gamma) I \end{bmatrix} \\
&+ (1-\lambda) \begin{bmatrix} \mu - \beta(1-f)\exp(-\alpha I) S I - \mu S - \phi S + \gamma I + \theta(1 - S - I) \\ \beta(1-f)\exp(-\alpha I) S I - (\mu+\gamma) I \end{bmatrix} \\
&= \begin{bmatrix} \mu + \theta - (\mu + \phi + \theta) S + (\gamma - \theta) I - \lambda\beta S I - (1-\lambda)\beta(1-f)\exp(-\alpha I) S I \\ -(\mu+\gamma) I + \lambda\beta S I + (1-\lambda)\beta(1-f)\exp(-\alpha I) S I \end{bmatrix},
\end{aligned}$$

with $0 \le \lambda \le 1$. Then the sliding mode equation is

$$f(S) = \mu + \theta - (\mu + \phi + \theta) S - (\mu + \theta) I_c.$$

Let $f(S) = 0$, then

$$S_p = \frac{(\mu+\theta)(1-I_c)}{\mu+\phi+\theta}.$$

At this point, $f(S)$ has only one equilibrium $E_p(S_p, I_c)$. Thus the pseudo-equilibrium exists iff $S_1 < S_p < S_2$, which is equivalent to $I_2 < I_c < I_1$.

Theorem 4.1. *E_p is a stable pseudo-equilibrium on Σ_s when it exists.*

Proof. Direct calculation yields

$$\left.\frac{d(f(S_p))}{dS}\right|_{E_p} = -(\mu + \phi + \theta) < 0.$$

Thus the solutions are attracting. □

In order to better understand the dynamic behaviors of the system (2.2), we now discuss the relationship between the sliding domain Σ_s and the invariant region Ω. Let $\frac{\mu+\gamma}{\beta} = 1 - I$, then we get $I_c^1 = \frac{\beta-(\mu+\gamma)}{\beta}$. Similarly, we let

$$\frac{\mu+\gamma}{\beta(1-f)} \exp(\alpha I) = 1 - I,$$

by the definition of Lambert W function, we obtain

$$I_c^2 = 1 - \frac{1}{\alpha} W(\frac{\alpha(\mu+\gamma)}{\beta(1-f)} \exp(\alpha)).$$

Since $S_1 < S_2$, then $I_c^1 > I_c^2$. Thus the relationship between the sliding domain Σ_s and the invariant region Ω can be divided into the following three cases:

(a) Σ_s is totally out of Ω if $I_c > I_c^1$.

(b) Σ_s is totally in Ω if $I_c < I_c^2$.

(c) Part of Σ_s is in Ω if $I_c^2 < I_c < I_c^1$.

Remark 4.1. *On the basis of the Section 4, we derive the following results:*

(a) The crossing region $\Sigma_c \subset \Sigma$ *is* $\langle H_X(X), F_1(X)\rangle > 0, \langle H_X(X), F_2(X)\rangle > 0$ *or* $\langle H_X(X), F_1(X)\rangle < 0, \langle H_X(X), F_2(X)\rangle < 0$. *By simply calculation, we derive that* $\Sigma_c = \{(S, I) \in R_+^2 \mid S < S_1, S > S_2, I = I_c\}$.

(b) The escaping region is $\Sigma_e = \emptyset$ *(the escaping region does not exist).*

(c) There are two tangent points: $T_1 = (S_1, I_c)$ *and* $T_2 = (S_2, I_c)$.

5. Analysis of global dynamics

In this part, we prove that the system (2.2) has no closed orbit, which contains a part of the closure of the sliding domain $\overline{\Sigma}_s$ or surrounds the whole sliding domain $\overline{\Sigma}_s$. And then, we analyze the global dynamics of the system using the method in [10].

5.1. Nonexistence of closed orbit

Lemma 5.1. *There is no closed orbit that contains a part of the closure of the sliding domain* $\overline{\Sigma}_s$ *for the system* (2.2).

Proof. First, there exists a pseudo-equilibrium E_p if $I_2 < I_c < I_1$, and E_p is LAS in the sliding domain Σ_s, which means the nonexistence of limit cycle that containing part of the sliding domain.

Second, we shall illustrate that there is no close orbit that contains a part of the closure of the sliding domain $\overline{\Sigma}_s$ if $I_c > I_1$ or $I_c < I_2$. Without loss of generality, we assume that $I_c > I_1$, i.e., E_1 is real (denoted by E_1^r) and E_2 is virtual (denoted by E_2^v). Then E_1^r and E_2^v are in the region $G_1 \bigcap \Omega$, and the vector field in $G_2 \bigcap \Omega$ is pointing downwards. Because $f(S) < 0$, then the trajectory moves from right to left on T_1T_2. We will illustrate that the orbit C initiating at T_1 will not hit the sliding domain Σ_s again. According to Theorem 3.1, E_1^r is a stable node or focus in region G_1, then the orbit C starting at T_1 either tends to the stable equilibrium E_1^r directly or spirally. If the latter is true, then the orbit C intersects with the horizontal isocline g_1^1 at two points O_1 and O_2, where O_2 is on the segment $T_1E_1^r$. Obviously, the two points O_1 and O_2 are below the point T_1. Hence, C starting at T_1 cannot form a cycle, as shown in Figure 4.

Consequently, there is no closed orbit that contains a part of the closure of the sliding domain $\overline{\Sigma}_s$ for the system (2.2). □

Lemma 5.2. *There is no closed orbit surrounding* $\overline{\Sigma}_s$ *for the system* (2.2).

Proof. Otherwise, there exists a closed orbit Γ that surrounding $\overline{\Sigma}_s$ (see Figure 5). Suppose that the closed orbit Γ intersects with Σ at two points N_1, N_2, where $N_1 = (P_1, I_c)$, $N_2 = (P_2, I_c)$. In addition, Γ intersects with the auxiliary line $I_c - \epsilon_1$ at two points M_1, M_2, and Γ intersects with the auxiliary line $I_c + \epsilon_1$ at two points M_3, M_4, where ϵ_1 is a sufficiently small number. Suppose D_1 denotes the lower

half region surrounded by M_1, M_2 and Γ_1, D_2 represents the upper half region surrounded by M_3, M_4 and Γ_2. Let $B(S, I) = \frac{1}{SI}$, we have

$$\begin{aligned}
&\sum_{i=1}^{2} \iint_{D_i} [\frac{\partial(BF_i^1)}{\partial S} + \frac{\partial(BF_i^2)}{\partial I}] dS\, dI \\
&= \iint_{D_1} [-\frac{\mu+\theta}{S^2 I} - \frac{\gamma-\theta}{S^2}] dS\, dI + \iint_{D_2} [-\frac{\mu+\theta}{S^2 I} - \frac{\gamma-\theta}{S^2} - \alpha\beta(1-f)\exp(-\alpha I)] dS\, dI \\
&< 0,
\end{aligned}$$

where $i = 1, 2$. Suppose that the abscissas of the points M_1, M_2, M_3 and M_4 are $P_1 + u_1(\epsilon_1), P_2 - u_2(\epsilon_1), P_2 - u_3(\epsilon_1), P_1 + u_4(\epsilon_1)$, respectively, where $u_i(\epsilon_1)$ $(i = 1, ..., 4)$ is continuous and satisfies $\lim_{\epsilon_1 \to 0} u_i(\epsilon_1) = 0$ and $u_i(0) = 0$. According to the Green's theorem, we obtain

$$\begin{aligned}
&\iint_{D_1} [\frac{\partial(BF_1^1)}{\partial S} + \frac{\partial(BF_1^2)}{\partial I}] dS\, dI \\
&= \oint_{\Gamma_1 \cup \overrightarrow{M_2 M_1}} BF_1^1 dI - \oint_{\Gamma_1 \cup \overrightarrow{M_2 M_1}} BF_1^2 dS \\
&= \int_{\Gamma_1} B(F_1^1 F_1^2 - F_1^2 F_1^1) dt + \int_{\overrightarrow{M_2 M_1}} BF_1^1 dI - \int_{\overrightarrow{M_2 M_1}} BF_1^2 dS \\
&= -\int_{\overrightarrow{M_2 M_1}} BF_1^2 dS \\
&= -\beta(P_1 - P_2 + u_1(\epsilon_1) + u_2(\epsilon_1)) + (\mu+\gamma) \ln |\frac{P_1 + u_1(\epsilon_1)}{P_2 - u_2(\epsilon_1)}|,
\end{aligned}$$

where $dS = F_1^1 dt$ and $dI = F_1^2 dt$. Similarly, for the region D_2,

$$\begin{aligned}
&\iint_{D_2} [\frac{\partial(BF_2^1)}{\partial S} + \frac{\partial(BF_2^2)}{\partial I}] dS\, dI \\
&= \oint_{\Gamma_2 \cup \overrightarrow{M_4 M_3}} BF_2^1 dI - \oint_{\Gamma_2 \cup \overrightarrow{M_4 M_3}} BF_2^2 dS \\
&= -\int_{\overrightarrow{M_4 M_3}} BF_2^2 dS \\
&= -\beta(1-f)\exp(-\alpha I_c)(P_2 - P_1 - u_3(\epsilon_1) - u_4(\epsilon_1)) + (\mu+\gamma) \ln |\frac{P_2 - u_3(\epsilon_1)}{P_1 + u_4(\epsilon_1)}|,
\end{aligned}$$

where $dS = F_2^1 dt$ and $dI = F_2^2 dt$. Since $P_1 < P_2$, then

$$\begin{aligned}
&\lim_{\epsilon_1 \to 0} \sum_{i=1}^{2} \iint_{D_i} [\frac{\partial(BF_i^1)}{\partial S} + \frac{\partial(BF_i^2)}{\partial I}] dS\, dI \\
&= \lim_{\epsilon_1 \to 0} (-\int_{\overrightarrow{M_2 M_1}} BF_1^2 dS - \int_{\overrightarrow{M_4 M_3}} BF_2^2 dS) \\
&= \beta(P_2 - P_1)(1 - (1-f)\exp(-\alpha I_c)) > 0,
\end{aligned}$$

this is a contradiction. Therefore, there is no closed orbit surrounding $\overline{\Sigma}_s$ for the system (2.2). □

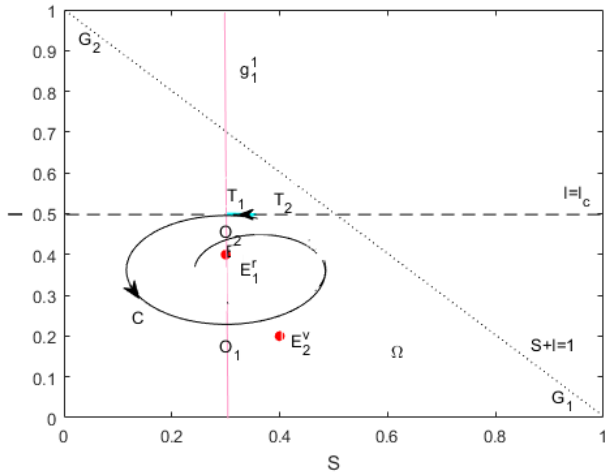

Figure 4. The possible closed orbit containing a part of $\overline{\Sigma}_s$ when E_1 is real and E_2 is virtual.

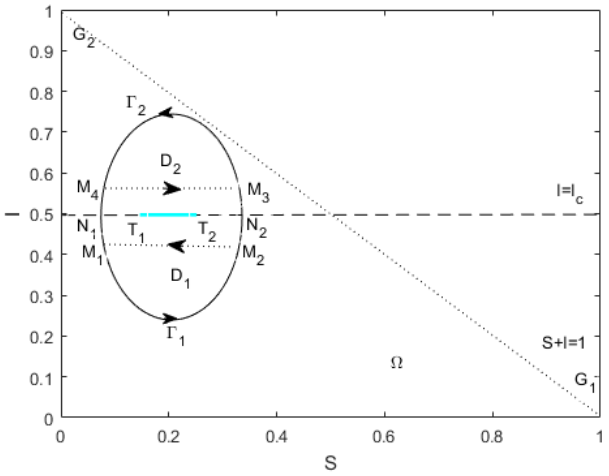

Figure 5. The possible closed orbit surrounding $\overline{\Sigma}_s$.

5.2. *Global dynamics*

In this section, we will prove the global stability of the system (2.2). First, we give the following Lemma.

Lemma 5.3. *(See [34]) If a half trajectory T^+ is bounded for the system (2.2), then its limit set $\Omega(T)$ contains either an equilibrium or a closed trajectory.*

In the following, according to the relationship among the basic reproduction number of the two subsystems R_0^1, R_0^2 and 1, the GAS of the system (2.2) can be divided into the following three cases.

Case 1: $R_0^2 < R_0^1 < 1$.

Theorem 5.1. *The disease-free equilibrium E_0 is GAS if $R_0^2 < R_0^1 < 1$.*

Proof. Since $R_0^2 < R_0^1 < 1$, the two endemic equilibria E_1 and E_2 are not feasible, and there ia a real disease-free equilibrium E_0 in $G_1 \bigcap \Omega$, then E_p does not exist. Furthermore, we obtain the solution of the system (2.2) are bounded and no closed orbit of any kind exists on the basis of Proposition 2.1, Theorems 3.1 and 3.2, Lemmas 5.1 and 5.2. Thus, the only real equilibrium E_0 is LAS. In this case the limit set $\Omega(T)$ of the system (2.2) is the unique real equilibrium E_0 by Lemma 5.3. So that any solution of the system (2.2) eventually stabilizes at the equilibrium E_0, i.e., E_0 is GAS (see Figure 6). □

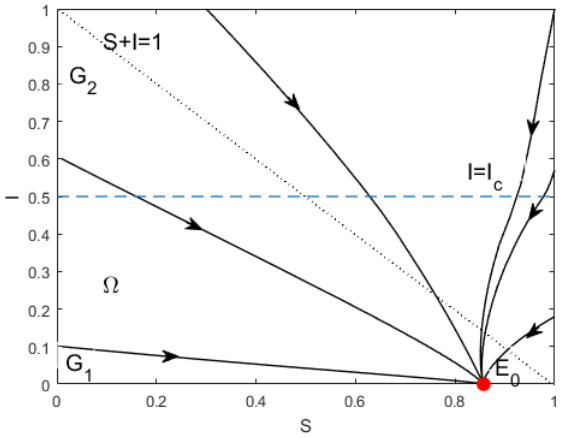

Figure 6. Case 1: $R_0^2 < R_0^1 < 1$. Global asymptotical stability of the system (2.2), where the parameters are chosen as follows: $\alpha = 0.1, \beta = 0.3, \mu = 0.3, \gamma = 0.5, \phi = 0.1, \theta = 0.3, I_c = 0.5, f = 0.3$.

Case 2: $R_0^2 < 1 < R_0^1$.

It is obvious that there exist two equilibria E_0 and E_1, where E_0 is a real saddle. However, we do not yet know whether E_1 is a real or virtual and whether E_p exists. Note that the threshold I_c may be greater than or less than I_1, so we discuss the following two subcases.

Theorem 5.2. *The endemic equilibrium E_1 is GAS if $R_0^2 < 1 < R_0^1$ and $I_c > I_1$.*

Proof. When $I_c > I_1$, we know E_1 real (denoted by E_1^r) and E_p does not exists. The trajectory moves from the right to the left on the sliding domain T_1T_2 by Lemma 5.1. Moreover, because there are no closed orbits in the region G_1, we obtain E_1 is LAS. According to the relationship between the sliding domain Σ_s and the invariant region Ω in Section 4, we can further divide it into the following three cases.

(i) If $I_c > I_c^1$, we can easy derive $S_1 + I_c > 1$, where S_1 is the left endpoint of the sliding domain. Therefore, the sliding domain Σ_s is totaly out the invariant region Ω, as shown in Figure 7(a).

(ii) If $\max\{I_c^2, I_1\} < I_c < I_c^1$, then $S_1 + I_c < 1$ and $S_2 + I_c > 1$, where S_1 and S_2 are the left and right endpoints of the sliding domain Σ_s respectively. There exists a point $T_* = (S_*, I_c)$ on T_1T_2, which satisfies $S_* + I_c = 1$. Thus part of the sliding segment T_1T_2 (i.e., T_1T_*) is in the invariant region Ω (see Figure 7(b)).

(iii) If $I_c < I_c^2$, then $S_2 + I_c < 1$, where S_2 is the left endpoint of the sliding domain. So Σ_s is totally in Ω, as shown in Figure 7(c).

Then the vector field in $G_2 \bigcap \Omega$ is pointing downwards. By Theorems 3.1 and 3.2, there is no close orbit totaly in the region G_1 and G_2. Furthermore, according to Lemmas 5.1 and 5.2, there is no close orbit that contains part of Σ_s and surrounding it. Therefore, for the subsystem (3.1), the equilibrium E_1 is GAS. Additionally, according to Lemma 5.3, it's not difficult to derive that the endemic equilibrium E_1 is GAS for the system (2.2). □

Theorem 5.3. *The pseudo-equilibrium E_p is GAS if $R_0^2 < 1 < R_0^1$ and $I_c < I_1$.*

Proof. When $R_0^2 < 1 < R_0^1$ and $I_c < I_1$, we know E_1 are virtual (denoted by E_1^v). Therefore, the rails in G_1 are oriented upward and those in G_2 are oriented downward, but they can not converge to their own equilibria and intersect with the sliding domain. The trajectory moves from the right to the left on the sliding domain E_pT_2, and the trajectory moves from the left to the right on the sliding domain T_1E_p by Theorem 3.1. Analogously, we discuss the relationship between Σ_s and Ω in the following two cases.

(i) If $I_c^2 < I_c < I_1$, we obtain part of Σ_s is in Ω similar to the proof of the Theorem 5.2 (ii) (see Figure 7(d)).

(ii) If $I_c < \min\{I_c^2, I_1\}$, according to the proof of the Theorem 5.2 (iii), we similarly conclude that Σ_s is totaly in Ω, as shown in Figure 7(e).

□

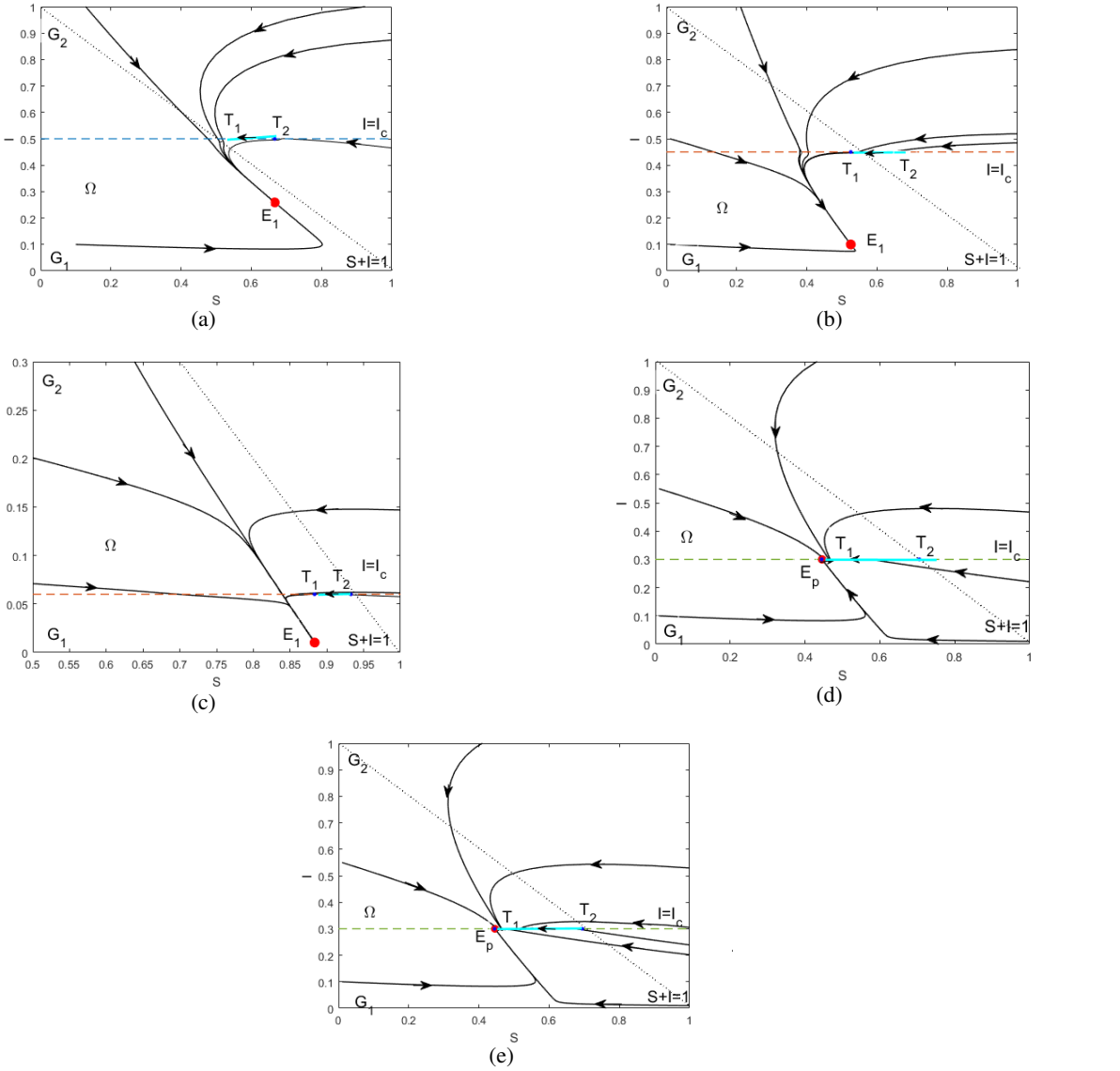

Figure 7. Case 2: Global asymptotical stability of the system (2.2) when $R_0^2 < 1 < R_0^1$, where the value of parameters are shown in Table 1.

According to the previous proof, the system (2.2) does not have any kind of closed orbits. Accordingly, by Lemma 5.3, the pseudo-equilibrium E_p is GAS for the system (2.2).

Case 3: $1 < R_0^2 < R_0^1$.

In this case, E_1 and E_2 are feasible, we now analyze the specific global stability of the system (2.2) in the following subcases similar to the Case 2.

Theorem 5.4. *The endemic equilibrium E_1 is GAS if $1 < R_0^2 < R_0^1$ and $I_c > I_1$.*

Proof. When $I_c > I_1$, we know E_1 real (denoted by E_1^r), E_2 is virtual (denoted by E_2^v) and E_p does not exists. The trajectory also moves from the right to the left on the sliding domain T_1T_2 by Theorem 3.1. Furthermore, we obtain E_1 is LAS. Analogously, there are three scenarios.

(i) If $I_c > I_c^1$, similar to the proof of the Theorem 5.2 (i), we obtain Σ_s is totaly out of Ω (see Figure 8(a)).

(ii) If $\max\{I_c^2, I_1\} < I_c < I_c^1$, similar to the proof of the Theorem 5.5 (ii), then part of Σ_s is in Ω (see Figure 8(b)).

(iii) If $I_c < I_c^2$, similar to the proof of the Theorem 5.2 (iii), we obtain Σ_s is totaly in Ω, as shown in Figure 8(c).

Then E_1^r and E_2^v are in the region $G_1 \bigcap \Omega$, and the vector field in $G_2 \bigcap \Omega$ is pointing downwards. By Theorems 3.1 and 3.2, there is no close orbit totaly in the region G_i $(i = 1, 2)$. Furthermore, according to Lemma 5.1, there is no close orbit that contains part of the sliding domain Σ_s. Therefore, the equilibrium E_1 is GAS for the subsystem (3.1). In addition, according to Lemma 5.3, we obtain E_1 is GAS for the system (2.2). □

Theorem 5.5. *The pseudo-equilibrium E_p is GAS if $1 < R_0^2 < R_0^1$ and $I_2 < I_c < I_1$.*

Proof. When $1 < R_0^2 < R_0^1$ and $I_2 < I_c < I_1$, E_1 and E_2 are virtual (denoted by E_1^v and E_2^v respectively), E_p does exist. Such that solutions of both subsystems can not converge to their own equilibria.

(i) If $I_c^2 < I_c < I_1$, according to the Theorem 5.3 (i), part of Σ_s is in Ω, as shown in Figure 8(d).

(ii) If $I_c < \min\{I_c^2, I_1\}$, similar to the Theorem 5.3 (ii), Σ_s is totaly in Ω, as shown in Figure 8(e).

Accordingly, due to the nonexistence of closed orbits in the invariant region Ω, in this case the pseudo-equilibrium E_p is GAS for the system (2.2). □

Theorem 5.6. *The endemic equilibrium E_2 is GAS if $1 < R_0^2 < R_0^1$ and $I_c < I_2$.*

Proof. When $1 < R_0^2 < R_0^1$ and $I_c < I_2$, E_1 is virtual (E_1^v) and E_2 is real (E_2^r), E_p does not exist. The trajectory moves from the left to the right on the sliding domain T_1T_2 by Theorem 3.1. Moreover, because there are no closed orbits in G_2, we obtain E_2 is LAS. In addition, by Lemmas 5.1 and 5.2, we obtain E_2 is GAS in the region G_2. Therefore, by Lemma 5.3, the ω-limit set of the system (2.2) is the unique real equilibrium E_2, which means E_2 is GAS. In addition, it is not difficult to derive Σ is totaly in Ω (see Figure 8(f)). □

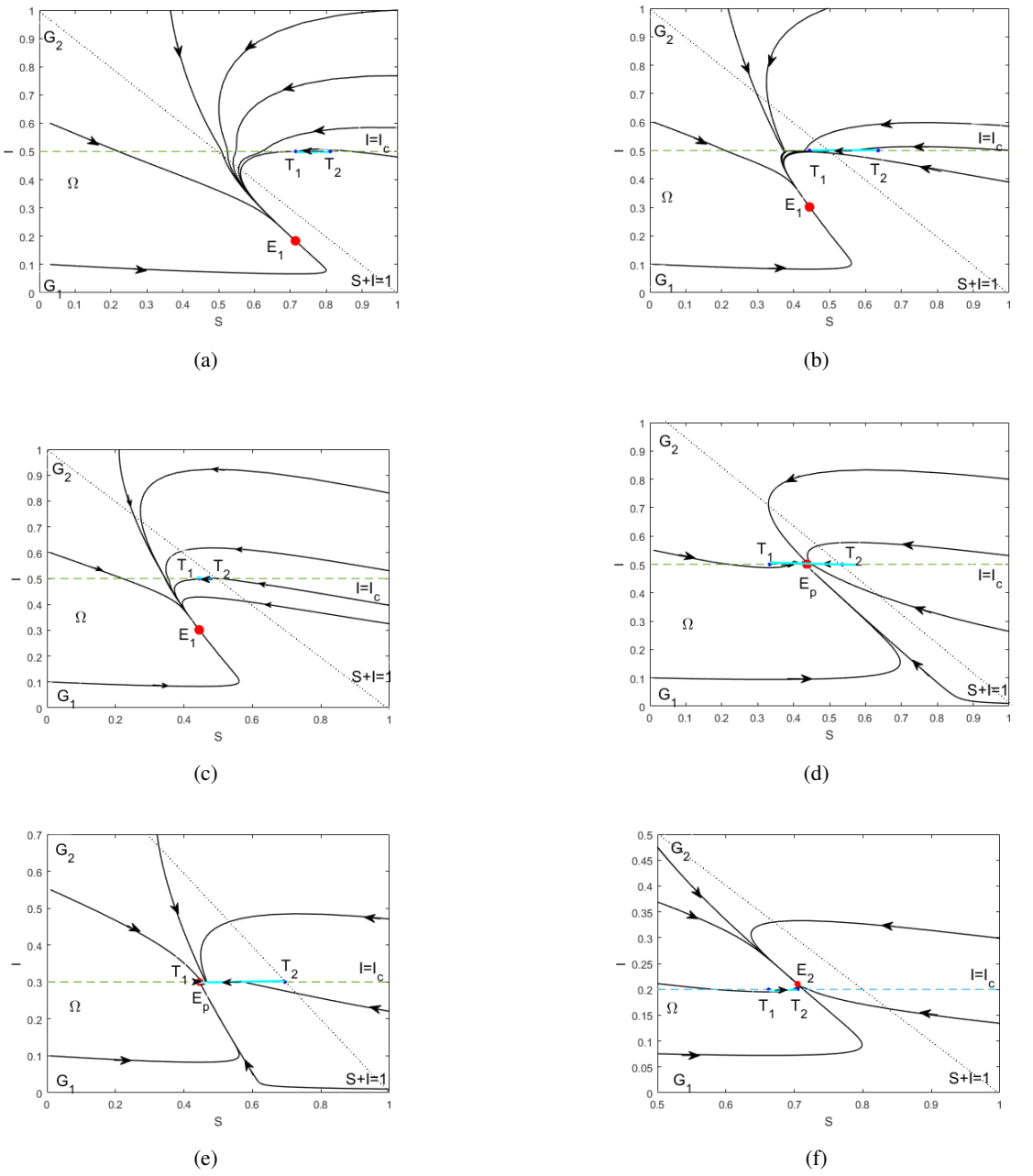

Figure 8. Case 3: Global asymptotical stability of the system (2.2) when $1 < R_0^2 < R_0^1$, where the value of parameters are shown in Table 1.

Remark 5.1. *The existence of pseudo-equilibrium E_p is one of the characteristics of a discontinuous system. Generally, the system could reach a new equilibrium state by controlling the threshold I_c, which is a new idea when infectious diseases could not be eradicated in a short time.*

Table 1. The parameters for Figures 7 and 8.

Case	α	β	μ	γ	ϕ	θ	I_c	f
Figure 7(a)	0.3	0.6	0.3	0.1	0.1	0.6	0.5	0.3
Figure 7(b)	0.4	0.8	0.4	0.02	0.5	0.3	0.45	0.4
Figure 7(c)	0.05	0.6	0.43	0.1	0.1	0.4	0.06	0.05
Figure 7(d)	0.3	0.9	0.3	0.1	0.4	0.4	0.3	0.31
Figure 7(e)	0.25	0.9	0.3	0.1	0.4	0.4	0.3	0.31
Figure 8(a)	0.3	0.7	0.3	0.2	0.1	0.4	0.5	0.1
Figure 8(b)	0.3	0.9	0.3	0.1	0.4	0.4	0.5	0.3
Figure 8(c)	0.05	0.9	0.3	0.1	0.4	0.4	0.5	0.05
Figure 8(d)	0.3	0.6	0.1	0.1	0.1	0.6	0.5	0.3
Figure 8(e)	0.3	0.9	0.3	0.1	0.4	0.4	0.3	0.3
Figure 8(f)	0.05	0.8	0.43	0.1	0.1	0.4	0.2	0.05

6. Biological implications and conclusions

In this paper, the media coverage and protective measures are considered in a discontinuous SIV system, in which the transmission rate influenced by both media coverage and protective measures is represented by a discontinuous function. Assuming that the total population is a constant, we transform the three-dimensional discontinuous differential equation system into a two-dimensional discontinuous differential equation system. For the two dimensional system, the first task is to prove the solutions with initial value are positive and bounded. After that, the system (2.2) is divided into two simple subsystems. For the subsystems, we calculate the basic reproduction number and the stability of the equilibria. Next, due to the specific switching characteristics of the Filippov system, the sliding mode domain and sliding mode equation are also the objects of our analysis. According to the analysis above, we study the global dynamics of the system (2.2), and obtain that the system (2.2) has no closed orbit of any kind. Detailed conclusions are shown in Table 2.

Table 2. Global dynamics of the system (2.2).

Case	Condition	Equilibria	Global dynamics
$R_0^2 < R_0^1 < 1$		E_0^r	E_0 is GAS (see Fig. 6)
$R_0^2 < 1 < R_0^1$	$I_c > I_1$	E_0^v, E_1^r	E_1 is GAS (see Fig. 7 (a), (b), (c))
$R_0^2 < 1 < R_0^1$	$I_c < I_1$	E_0^v, E_1^v, E_p	E_p is GAS (see Fig. 7 (d), (e))
$1 < R_0^2 < R_0^1$	$I_c > I_1$	E_1^r, E_2^v	E_1 is GAS (see Fig. 8 (a), (b), (c))
$1 < R_0^2 < R_0^1$	$I_2 < I_c < I_1$	E_1^v, E_2^v, E_p	E_p is GAS (see Fig. 8 (d), (e))
$1 < R_0^2 < R_0^1$	$I_c < I_2$	E_1^v, E_2^r	E_2 is GAS (see Fig. 8 (f))

Our ultimate goal is to reduce the scale of infection and control the spread of infectious diseases.

Modeling of discontinuous systems also provides a reference for government departments to formulate prevention and control measures, such as the timing and intensity of media coverage, and the intensity of protective measures. Different regulations on prevention and control measures have been introduced in light of the actual situation, including the scale of the epidemic, to make better use of public resources.

Firstly, we discuss the impact of infection threshold I_c on disease outbreak, which can be divided into three scenarios:

(a) $R_0^2 < R_0^1 < 1$

In this case, we hope the transmission rate β is sufficiently small, the vaccination rate of the susceptible population ϕ, and the recovery rate of infected individuals γ are sufficiently large. According to the Theorems 3.1 and 5.1, the number of infected people will extinct regardless of the number of I_c. After comparing $I_c = 0.1$, $I_c = 0.3$, and $I_c = 0.9$, we could see that taking an appropriate threshold value could control the spread of disease in a relatively short time. As indicated in Figure 9(a), when the scale of the disease $I > I_c$ decreases rapidly, the curve slope is steep. Once I is lower than the given threshold I_c and the prevention and control measures are loosened, there are still infected people, but their number will be small and slowly changing and the curve will decrease gently. At this point, if the threshold I_c is larger, i.e., more people remain infected when the prevention and control measures are lifted, the disease will die out more slowly. However, if I_c is very small, it is bound to waste large amounts of social resources and exert a long-term impact on humans' normal life. In conclusion, the selection of infection threshold I_c should suit the actual situation, being neither too large nor too small.

(b) $R_0^2 < 1 < R_0^1$

Similar to the scenario (*a*), to obtain $R_0^2 < 1 < R_0^1$, we hope the transmission rate β is not sufficiently small, the vaccination rate of the susceptible population ϕ or the cure rate of infected individuals γ are unsatisfactory, which causes the infectious disease to become endemic and pseudo-equilibrium states. According to Theorems 5.2 and 5.3, the solution of the system that satisfies any initial condition will eventually stabilize at the endemic equilibrium E_1 or pseudo-equilibrium E_p. Figure 9(b) also draws the above conclusion that the smaller the I_c is, the better it is to control the spread of the disease. In addition, if I_c is sufficiently small, we could balance I to a smaller value, i.e., in a pseudo-equilibrium state, thereby controlling the endemic range to a smaller value.

(c) $1 < R_0^2 < R_0^1$

As indicated in Figure 9(c), taking appropriate threshold leads to quicker control and fewer infections. There may be three equilibrium states, i.e., three possible endemic scales. The government should formulate appropriate measures and people should improve their self-protection awareness as far as possible to minimize the infection and achieve a pseudo equilibrium state.

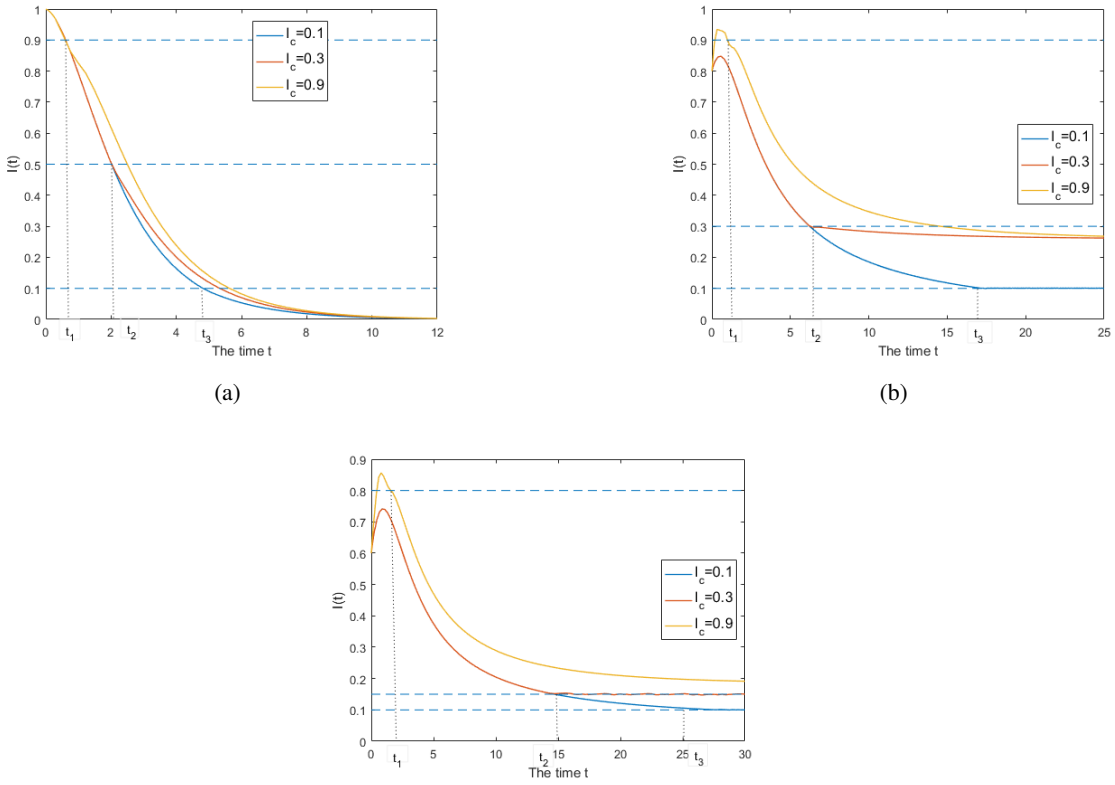

Figure 9. The graph of I with different threshold I_c while other parameters remain unchanged.

The transmission rate is a key factor in determining the dynamics of the disease and its ultimate outcome. Through the dynamic analysis, we discover that the transmission rate is greatly influenced by the intensity of protective measures f and the influence coefficient of media coverage α. Our next step is to investigate how the infectious disease influenced by the two parameters, and to show how to achieve our goal: to control the spread of infectious diseases.

Figure 10 presents the impact of protective measures f and the influence coefficient of media coverage α on the scale of the epidemic in four cases. As indicated in Figure 10(a), when $R_0^2 < R_0^1 < 1$, all curves eventually reach zero. As for epidemic prevention and control with the same intensity of media coverage, the stronger the protective measures, the better the effect. With the same protective measures, the more media coverage, the more effective. There is some overlap between media reports and protective measures and which method is more effective could be judged based on the following figures. According to the Theorems 5.2 and 5.3, when $R_0^2 < 1 < R_0^1$, the solution of the system (2.2) finally stabilizes at the endemic equilibrium E_1 or pseudo-equilibrium E_p, as indicated in Figure10(b), (c). Finally, in Figure 10(d), if $1 < R_0^2 < R_0^1$, the final scale of the disease has three scenarios which are stable at endemic equilibrium E_1, endemic equilibrium E_2, and pseudo-equilibrium E_p respectively, where $I_1 > I_2 > I_c$. Similar to Figure 10(a), Figure 10(b), (c), and (d) show that media coverage and protective measures have a positive impact on the control of infectious diseases, but protective measures are more effective than media coverage. Therefore, when an epidemic occurs,

government departments should formulate corresponding measures according to the actual stage of the spread of infectious diseases, select the intensity of protective measures and appropriate media reports, and prioritize protection supplemented by reporting to minimize the scale of infectious diseases as soon as possible by using the least social resources.

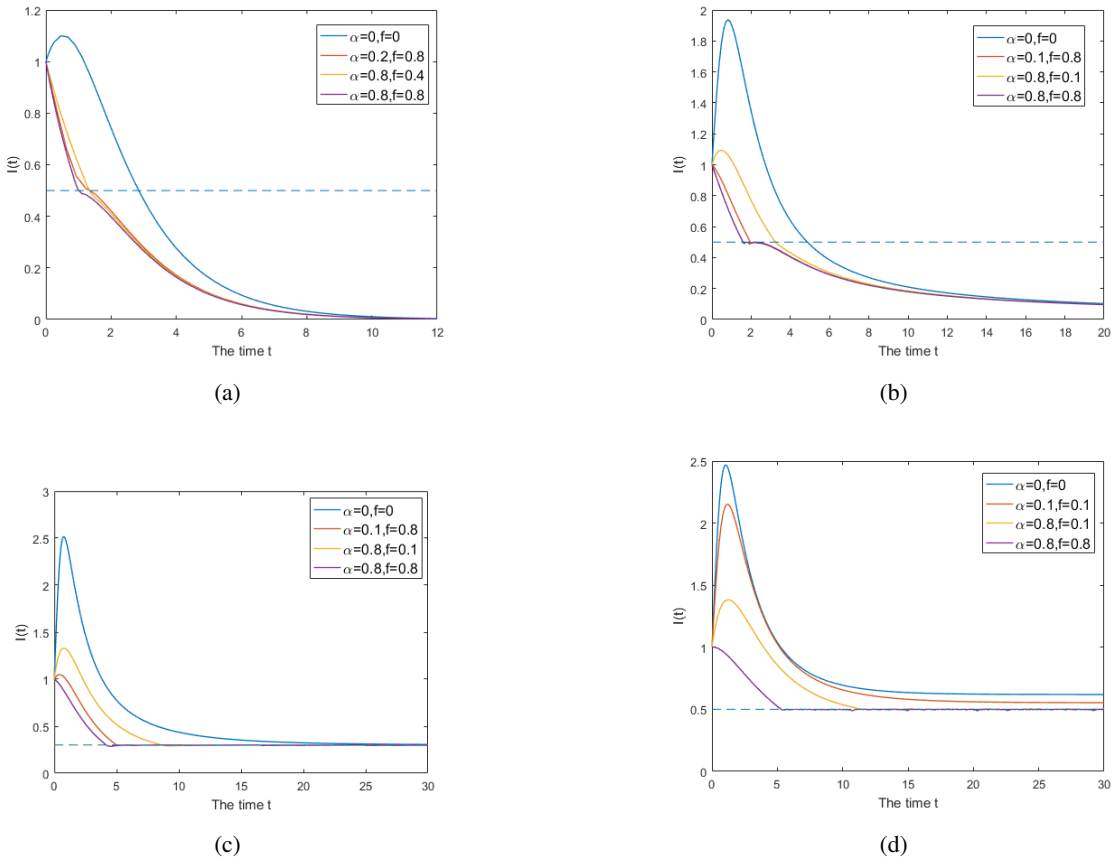

Figure 10. The graph of I with different intensity of protective measures f and influence coefficient of media coverage α while other parameters remain unchanged.

In conclusion, the theoretical and numerical simulation in this paper present that with reasonable control measures and appropriate threshold I_c, the spread of the disease can be taken under control in a shorter time and reduce the peak value. When an infectious disease breaks out over a period, we could make use of the characteristics of a discontinuous system to reduce its scale. If media reports were simply introduced into the original system, it would be difficult to analyze the dynamic behavior of the system. Therefore, we propose a more realistic model based on the existing work, and the results are similar. But we are focus on the biological significance of infectious diseases, we can take reasonable measures to control the disease below the threshold, rather than the outbreak of a large scale. In this paper, we only consider controlling media coverage and protection measures using the same threshold. In the future, we will separate the media coverage and protection measures, hoping to provide a more valuable reference.

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Conflict of interest

The authors declare no conflict of interest in this paper.

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