Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.
The selection pressures induced non-smooth infectious disease model and bifurcation analysis

Wenjie Qin, Sanyi Tang

College of Mathematics and Information Science, Shaanxi Normal University, Xi'an 710062, PR China

A R T I C L E   I N F O

Article history:
Received 19 June 2014
Accepted 22 September 2014
Available online 22 October 2014

A B S T R A C T

Mathematical models can assist in the design strategies to control emerging infectious disease. This paper deduces a non-smooth infectious disease model induced by selection pressures. Analysis of this model reveals rich dynamics including local, global stability of equilibria and local sliding bifurcations. Model solutions ultimately stabilize at either one real equilibrium or the pseudo-equilibrium on the switching surface of the present model, depending on the threshold value determined by some related parameters. Our main results show that reducing the threshold value to an appropriate level could contribute to the efficacy on prevention and treatment of emerging infectious disease, which indicates that the selection pressures can be beneficial to prevent the emerging infectious disease under medical resource limitation.

1. Introduction

Emerging infectious disease, caused by nature or bioterrorism (such as Asiatic/Russian Flu [1], Spanish Flu [2], 2009 Flu pandemic [3,4]) is one of the most important disasters in the history of human and threatens public health, so the government of each country and peoples pay more and more attentions on the prevention and treatment for emerging infectious diseases. World Health Organization reports expenditure of more than $468 million in 2012–2013 to control the spread of infectious diseases [5], and funds for treatment and research emerging infectious diseases are huge and increasing. It is thus important to investigate effective control strategies that can prevent outbreaks or minimize the cost.

During the last decade, the outbreak of Severe Acute Respiratory Syndrome (SARS) in 2003 and avian influenza among humans (H5N1 in 2003, H1N1 in 2009, and H7N9 in 2013) emphasized the need to enhance the capacity to fight emerging infectious diseases. Since the first outbreaks in China in early 2003 SARS had spread rapidly across China into southeast Asia and even the world, and SARS was the first severe new disease of the 21st century, so we take SARS as an example of emerging infectious disease in this paper. In the early stages of the 2003 SARS outbreak, only a very few number of individuals infected by SARS, and the early symptoms were very similar to flu and could not be diagnosed by medical personnel.

Along with the rapid spread of SARS in some countries and regions, the numbers of SARS infected cases and flu were growing, those posed a grave threat to public health as its high morbidity and mortality. On the prevention and treatment for SARS, the effective and essential measures including heightened surveillance, early detection and treatment of cases, and infection control in all health facilities have been applied. Although the emergency ambulances including development of effective drugs and rapid tools for diagnosis provided a safe and reliable mobile medical treatment platform for tackling SARS, it would put pressure on limited medical resources such as doctors, hospital beds, isolation places and bring grave challenges to each country, especially in rural areas in many developing

http://dx.doi.org/10.1016/j.chaos.2014.09.014
0960-0779/© 2014 Elsevier Ltd. All rights reserved.
countries [6–9]. For example, it was a serious challenge for medical personnel to diagnose the patients who were infected by SARS or flu with the similar signs and symptoms such as coughs, colds, fevers and so on. Those required a lot of medical resources, which undoubtedly worse the already grave situation. Thus, medical resource limitation seriously restricted the prevention and treatment for SARS.

Therefore, faced with an increasing numbers of SARS infected cases and flu, the increasing serious situation caused the doctors have to focus their attentions on the SARS infected cases and put aside flu due to selection pressure. As such, emergency medical treatment services such as isolated treatment, personal protection, medical observation, sterilization and so on were only taken immediately for SARS. For the patients infected by flu, the doctors had to prescribe some medicines to them and advise them go home for home treatment as the selection pressure.

In order to describe the effects of limited medical resource and selection pressure, the number of the patients infected by SARS in a compartment has been chosen as an index for medical personnel to use decisions. In such a case, intervention is modeled and represented by using a piecewise function. This type of control strategy is so-called threshold policy [10,11], which is defined as follow: if the number of the patients infected by SARS is below the threshold level (denoted by \( L \)), there is no limited medical resource and selection pressure; above the threshold, due to the limited resource, and doctors treat SARS only. The threshold policy defined as above is also referred to as an on–off control which can be described by Filippov systems [12,13]. Recently, Filippov systems have been widely used in many fields of science and engineering [14–24].

The purpose of this study is to derive a novel non-smooth infectious disease model with threshold strategy to describe both medical resources limitation and selection pressures. This study investigates how the threshold value of the infected population and selection pressure affect the prevention and treatment for SARS under medical resources limitation. Furthermore, the key control parameters which are most significantly related to this threshold value are also investigated. In particular, mathematical and bifurcation analyses with regard to the local, global stability of equilibria and local sliding bifurcations are performed.

2. Filippov infectious disease model and preliminaries

2.1. Model formulation

The basic model we consider is based on the classical infectious disease model with limited capacity for treatment [25], i.e.,

\[
\begin{align*}
\dot{S}(t) & = A - \mu_S S - \beta SI, \\
\dot{I}(t) & = \beta SI - (\mu_I + \nu)I - \frac{c_1 I}{1 + c_1 I}, \\
\dot{R}(t) & = v I + \frac{c_1 I}{1 + c_1 I} - \mu_R R.
\end{align*}
\]  

The assumptions in model (1) are as follows:

- \( S(t), I(t) \) and \( R(t) \) denote the numbers of susceptible, infective and recovered individuals at time \( t \), respectively. \( A \) is the recruitment rate of susceptible individuals, \( \mu_S \) and \( \mu_I \) are the natural death rates of susceptible and recovered individuals, \( \mu_R \) is the death rate of the infected individuals which includes both the disease-related death and the natural death, hence \( \mu_R > \mu_S \).

- \( H(I) = cI/(1 + bI) \) represents the recovery rate from the infected compartment with hospital treatment, which is a saturated treatment function, where \( c \) represents the maximal recovery rate and \( b \) describes the effects of medical resource limitation on the treatment.

- \( \nu \) stands for the natural recovery rate of the infective individuals, obviously, \( \nu < c \), the incidence rate is assumed to be mass action incidence with bilinear interactions given by \( \beta SI \), and \( \beta \) is the transmission coefficient.

In order to describe the selection pressure for doctors faced with both SARS and flu cases occur simultaneously in the crowd, we extend model (1) as

\[
\begin{align*}
\dot{S}(t) & = A - \mu_S S - \beta SI - \beta_2 SI_2, \\
\dot{I}_1(t) & = \beta SI_1 - \mu_I I_1 - \nu_1 I_1 - \frac{p_1 c_1 I_1}{(1 + p_1 c_1 I_1 + p_2 c_2 I_2)}, \\
\dot{I}_2(t) & = \beta_2 SI_2 - \mu_I I_2 - \nu_2 I_2 - \frac{p_2 c_2 I_2}{(1 + p_1 c_1 I_1 + p_2 c_2 I_2)}, \\
\dot{R}(t) & = \nu_1 I_1 + \nu_2 I_2 + \frac{p_1 c_1 I_1 + p_2 c_2 I_2}{(1 + p_1 c_1 I_1 + p_2 c_2 I_2)} - \mu_R R.
\end{align*}
\]  

Here \( I_1 \) and \( I_2 \) denote the number of the patients infected by SARS and flu, respectively. \( \beta_i \) is called the basic transmission coefficient of \( I_i \), \( \mu_i \) is the death rate of \( I_i \) which include both the disease-related death and the natural death, hence \( \mu_1 > \mu_2 \). \( p_1 \) and \( p_2 \) denotes the probability that doctors will treat \( I_1, I_2 \) stands for the maximum recovery rate per unit time for \( I_1, I_2 \) describes effects of medical resource limitation on the treatment for \( I_1, I_2 \), \( \nu_i \) is the natural recovery rate for \( I_1, I_2 \), and \( \nu_1 < c_1, \nu_2 < c_2 \).

The question is how the doctors choose the patients who are either infected by SARS or flu. It is well known that both patients can be treated at the initial stage of SARS outbreak. Once the number of the patients infected by SARS increases and exceeds some threshold value, there is not enough medical resources. The doctors focus on their attentions on the patients by SARS. To determine the threshold value and consequently determine the key parameters, we consider the following function with respect to \( p_1 \) and \( p_2 \), i.e., we define

\[
\mathcal{R}(p_1, p_2) = \frac{p_1 c_1 I_1 + p_2 c_2 I_2}{(1 + p_1 c_1 I_1 + p_2 c_2 I_2)^2},
\]

and we consider \( \mathcal{R} \) as a fitness function which is maximized. Taking simple calculation, one yields

\[
\begin{align*}
\frac{\partial \mathcal{R}}{\partial p_1} & = \frac{(c_1 + p_2 c_2 I_2)(b_2 c_1 - b_1 c_2) I_1}{(1 + p_1 c_1 I_1 + p_2 c_2 I_2)^2}, \\
\frac{\partial \mathcal{R}}{\partial p_2} & = \frac{(c_2 - p_1 c_1 I_1)(b_2 c_1 - b_1 c_2) I_2}{(1 + p_1 c_1 I_1 + p_2 c_2 I_2)^2}.
\end{align*}
\]

In consideration of selection pressure, we assume \( b_2 c_1 - b_1 c_2 > 0 \), then \( \mathcal{R} \) is maximized at \( p_1 = 1 \), and it follows from \( \partial \mathcal{R}/\partial p_2 = 0 \) that the threshold value \( I_c = c_2/(b_2 c_1 - b_1 c_2) \).

All those show that \( \mathcal{R} \) is a monotonic increasing (or decreasing) function with respect to \( p_2 \) provided \( I_c < I_c \) (or
Thus, in order to obtain the maximum recovery rate, we choose \( p_2 = 1 \) for \( I_1 < I_c \), that is, if sufficient medical resources can treat those small number of infected patients at the early stage of SARS outbreak, the patients infected by flu can be treated simultaneously with SARS (i.e., \( p_1 = p_2 = 1 \)). However, along with the prevalence of SARS (i.e., \( I_1 > I_c \)), the limited medical resources cannot satisfy the growing trend towards SARS cases. Department of health or state has to cite the urgency of fighting SARS, adopts "green passage" policy that speeds for isolation and treatment for SARS. At this moment, the doctors have to prescribe medicines for the patients infected by flu and advise them go home for home treatment (i.e., \( p_1 = 1, p_2 = 0 \)). Meanwhile, this exposes the faultiness of state handling mechanism of paroxysmal public health events and infectious disease observation mechanism, especially in poor countries and areas. In this case, we choose \( p_2 = 0 \) for \( I_1 > I_c \).

Therefore, taking into account above facts, if the number of the patients infected by SARS is less than the threshold \( I_c \), then model (2) becomes

\[
\begin{align*}
\dot{S}(t) &= -\mu S - \beta_1 S I_1 - \beta_2 S I_2, \\
\dot{I}_1(t) &= \beta_1 S I_1 - \mu I_1 - v_1 I_1 - \frac{c_{11}}{1 + b_2 k}, \\
\dot{I}_2(t) &= \beta_2 S I_2 - \mu_2 I_2 - v_2 I_2 - \frac{c_{12}}{1 + b_2 k}, \\
\dot{R}(t) &= v_1 I_1 + v_2 I_2 + \frac{c_{11} + c_{12}}{1 + b_2 k} - \mu R.
\end{align*}
\]

If the number of the patients infected by SARS is large beyond the threshold \( I_c \), then model (2) becomes

\[
\begin{align*}
\dot{S}(t) &= -\mu S - \beta_1 S I_1 - \beta_2 S I_2, \\
\dot{I}_1(t) &= \beta_1 S I_1 - \mu I_1 - v_1 I_1 - \frac{c_{11}}{1 + b_2 k}, \\
\dot{I}_2(t) &= \beta_2 S I_2 - \mu_2 I_2 - v_2 I_2 - \frac{c_{12}}{1 + b_2 k}, \\
\dot{R}(t) &= v_1 I_1 + v_2 I_2 + \frac{c_{11} + c_{12}}{1 + b_2 k} - \mu R.
\end{align*}
\]

Without loss of generality, we consider the number of the patients infected by flu each year is a constant, i.e., \( I_2 = k \in Z^+ \). Meanwhile, due to the high risk of SARS, the patients infected by SARS will be taken care immediately no matter medical resource limitation, that is \( b_1 = 0 \).

Thus, models (3) and (4) can be rewritten as the following non-smooth dynamic system \[12,13\]

\[
\begin{align*}
\dot{S}(t) &= -\mu S - \beta_1 S I_1, \\
\dot{I}_1(t) &= \beta_1 S I_1 - \mu I_1 - v_1 I_1 - \frac{c_{11}}{1 + b_2 k},
\end{align*}
\]

with

\[
\varepsilon = \begin{cases} 1, & H(Z) < 0, \\ 0, & H(Z) > 0, \end{cases}
\]

where \( \mu = \mu_s + \beta_2 k, v = \mu_1 + v_1, I_c = c_2/(b_2 c_1) \) and \( H(Z) = I_1 - I_c \) with vector \( Z = (S, I_1)^T \). Model (5) with (6) is a description of the threshold policy, which is referred to as an on-off control, see \[10,11\] for more detailed introduction.

For convenience, we further denote

\[
F_{S_1}(Z) = \left( A - \mu S - \beta_1 S I_1, \beta_1 S I_1 - v_1 I_1 - \frac{c_{11}}{1 + b_2 k} \right)^T,
\]

\[
F_{S_2}(Z) = (A - \mu S - \beta_1 S I_1, \beta_1 S I_1 - v_1 I_1 - c_{11} I_1)^T.
\]

Then model (5) with (6) can be rewritten as the following Filippov system \[12,13\]

\[
\dot{Z}(t) = \begin{cases} F_{S_1}(Z), & Z \in S_1, \\ F_{S_2}(Z), & Z \in S_2, \end{cases}
\]

where \( S_1 = \{ Z \in \mathbb{R}_+^2 | H(Z) < 0 \} \), \( S_2 = \{ Z \in \mathbb{R}_+^2 | H(Z) > 0 \} \). Furthermore, the discontinuity boundary (or manifold) \( \Sigma \) separating two regions \( S_1 \) and \( S_2 \) is described as \( \Sigma = \{ Z \in \mathbb{R}_+^2 | H(Z) = 0 \} \), and \( H \) is a smooth scalar function with non-vanishing gradient \( H \) on \( \Sigma \). From now on, we call Filippov system (7) defined in region \( S_1 \) as system \( S_1 \), and defined in region \( S_2 \) as system \( S_2 \).

Let

\[
\sigma(Z) = \langle H(Z), F_{S_1}(Z) \rangle \cdot \langle H(Z), F_{S_2}(Z) \rangle = F_{S_1} H(Z) \cdot F_{S_2} H(Z),
\]

where \( \langle \cdot, \cdot \rangle \) denotes the standard scalar product, and \( F_{S_1} H(Z) = F_{S_2} \cdot \text{grad} H(Z) \) is the Lie derivative \[26\] of \( H \) with respect to the vector field \( F_{S_2} \) at \( Z \) for \( i = 1, 2 \), then the sliding domain can be defined as \( \Sigma_s = \{ Z \in \Sigma | \sigma(Z) = 0 \} \).

The following definitions on all types of equilibria of Filippov system \[27,28\] are necessary throughout the paper.

**Definition 2.1.** A point \( Z \), is called a real equilibrium of Filippov system (7) if \( F_{S_1}(Z) = 0, H(Z) < 0 \), or \( F_{S_2}(Z) = 0, H(Z) > 0 \). Similarly, a point \( Z \), is called a virtual equilibrium if \( F_{S_1}(Z) = 0, H(Z) = 0 \), or \( F_{S_2}(Z) = 0, H(Z) = 0 \). Both the real and virtual equilibria are called regular equilibria.

**Definition 2.2.** A point \( Z \), is called a pseudo-equilibrium if it is an equilibrium of the sliding mode of system (7), i.e., \( \lambda F_{S_1}(Z) + (1 - \lambda) F_{S_2}(Z) = 0, H(Z) = 0 \) and \( 0 < \lambda < 1 \), where \( \lambda = F_{S_1} H(Z)/[(F_{S_2} - F_{S_1}) H(Z)] \).

**Definition 2.3.** A point \( Z \), is called a boundary equilibrium of Filippov system (7) if \( F_{S_1}(Z) = 0, H(Z) = 0 \), or \( F_{S_2}(Z) = 0, H(Z) = 0 \).

Further, we say the boundary equilibrium bifurcation occurs at \( Z \) if \( F_{S_1}(Z) \) is invertible (or equivalently the eigenvalues of \( \text{det} F_{S_1}(Z) \) have real part different from zero and \( F_{S_2} H(Z) \neq 0, i, j = 1, 2, i \neq j \)). These bifurcations are classified as boundary focus, boundary node and boundary saddle in \[29\].

**Definition 2.4.** A point \( Z \), is called a tangency point of Filippov system (7) if \( Z \in \Sigma_s \) and \( F_{S_1} H(Z) = 0 \) or \( F_{S_2} H(Z) = 0 \).

2.2. Qualitative analysis of subsystems

For subsystem \( S_1 \), it has the disease-free equilibrium \( E_0 \) and the endemic equilibrium \( E_1 \), and

\[
E_0 = \left( \frac{A}{\mu}, 0 \right), \quad E_1 = (S_1^*, I_1^*) = \left( \frac{1}{\beta_1} \left( v + \frac{c_1}{1 + b_2 k} \right), \frac{\mu}{\beta_1} (R_{01} - 1) \right).
\]
where
\[ R_{01} = \frac{1}{A} \frac{\beta_1}{v + \frac{\mu}{\gamma} + \frac{1}{\gamma}} \]
is the basic reproduction number of subsystem \( S^1 \).

For the global stabilities of \( E_0 \) and \( E_1 \), we can choose Lyapunov functions
\[
V_0(t) = I_1(t),
\]
\[
V_1(S, I_1) = \frac{1}{2S} (S - S_1)^2 + \left( I_1 - I_1^0 - I_1^0 \ln \frac{I_1}{I_1^0} \right)
\]
for two equilibria, and using Lasalle invariant set principle, we get the global stabilities of \( E_0 \) and \( E_1 \) easily provided \( R_{01} < 1 \) and \( R_{01} > 1 \), respectively.

Analogously, for subsystem \( S^2 \), it has the disease-free equilibrium \( E_0 = (A, 0, 0) \) which is globally asymptotically stable if \( R_{02} < 1 \), and the endemic equilibrium
\[
E_2 = (S^2, I_2) = \left( \frac{1}{\beta_1} (v + c_1), \frac{\mu}{\beta_1} (R_{02} - 1) \right)
\]
is globally asymptotically stable if \( R_{02} > 1 \). Here
\[ R_{02} = \frac{\beta_1 A}{v + c_1} \]
is the basic reproduction number of subsystem \( S^2 \).

Meanwhile, the characteristic polynomial of subsystem \( S^2 \) about the endemic equilibrium \( E_1 = (S^1, I_1^0) \) is
\[
\lambda^2 + \frac{A}{S^1} \lambda + \beta_1 (A - \mu S_1) = 0
\]
and \( E_1 \) could be a node or focus point which depends on the sign of
\[
\Delta_i = \frac{A^2}{S^1} - 4 \beta_1 (A - \mu S_1), \quad i = 1, 2.
\]

Further, noting that \( R_{02} < R_{01} \). Thus, if \( R_{01} < 1 \), both free system \( S^1 \) and control system \( S^2 \) stabilize at its disease-free equilibrium; If \( R_{02} > 1 \), these two subsystems \( S^1 \) and \( S^2 \) have their own endemic states.

3. Basic properties of Filippov system (7)

3.1. Existence of sliding domain

It follows from the definition of function \( \sigma(Z) \) that we have
\[
\sigma(Z) = \begin{cases} I_1^0 \left( \beta_1 S - v - \frac{c_1}{1 + \frac{\mu}{\gamma}} \right) (\beta_1 S - v - c_1), & Z \in \Sigma; \end{cases}
\]
which is equivalent to check if the components of vector \( Z = (S, I_1) \) are transversal to \( \Sigma \). That is \( I_1 \) evaluated for \( I_1 = I_1^0 \), with the second equation in both subsystems \( S^1 \) and \( S^2 \) can be of opposite sign.

Therefore, the sliding domain \( \Sigma_e \) can be obtained as
\[
\Sigma_e = \left\{ Z \in \Sigma \left| \frac{1}{\beta_1} \left( v + \frac{c_1}{1 + \frac{\mu}{\gamma}} \right) \leq S \leq \frac{1}{\beta_1} (v + c_1), I_1 = I_1^0 \right. \right\},
\]
that is,
\[
\Sigma_e = \left\{ Z \in \Sigma \left| S^1 \leq S \leq S^2, I_1 = I_1^0 \right. \right\}.
\]

3.2. Sliding mode dynamics

Here we employ Utkin’s equivalent control method introduced in [13] to obtain the differential equation for sliding dynamics defined in the region \( \Sigma_e \). It follows from \( H = 0 \) that
\[
\frac{dH}{dt} = I_1 = I_1 S_1 - v I_1 - \frac{c_1 I_1^0}{1 + \frac{\mu}{\gamma}} E_2 = 0,
\]
solving the above equation with respect to \( \varepsilon \) yields
\[
\dot{\varepsilon} = \frac{c_1 + v - \beta_1 S}{b_2 k (\beta_1 S - v)}.
\]

According to Utkin’s equivalent control method the dynamics on the sliding domain \( \Sigma_e \) can be determined by the following scalar differential equation
\[
\dot{S}(t) = A - \mu S - \beta_1 S_1 e,
\]
where \( S \in (S^1, S^2) \). Obviously, the sliding mode (9) exists a unique pseudo-equilibrium \( E_P = (S^1, I_1^0) \) provided \( S_P \in S_d \), where \( S_P = A(\mu + \beta_1 I_1^0) \), and \( S_P \in S_d \) is equivalent to
\[
\frac{\mu}{\beta_1} (R_{01} - 1) < I_c < \frac{\mu}{\beta_1} (R_{01} - 1).
\]

Noting that
\[
I_c = \frac{c_2}{b_2 c_1}, \quad I_1^0 = \frac{\mu}{\beta_1} (R_{01} - 1), \quad I^2 = \frac{\mu}{\beta_1} (R_{02} - 1),
\]
we can rewrite the above inequality as
\[
H_1 = b_2 c_1 I_1^0 < c_2 < b_2 c_1 I^2 \triangleq H_2.
\]

For the scalar equation (9), it is easy to show that the pseudo-equilibrium \( E_P \) is locally asymptotically stable on the sliding domain \( \Sigma_e \).

4. Sliding bifurcation analysis

4.1. Bifurcation sets of equilibria and sliding modes

In this subsection, we will address the richness of all possible equilibria and sliding mode that Filippov system (7) can exhibit. To do this, parameters \( k \) and \( c_2 \) are chosen to build the bifurcation diagram and all other parameters are chosen as those in Fig. 1.

Note that \( R_{01} > R_{02} \) and \( I_1^0 > I_1^2 \). Therefore, if \( c_2 > H_2 \) (i.e., \( \hat{\Omega}_1 \cup \hat{\Omega}_2 \cup \hat{\Omega}_3 \) in Fig. 1), then the equilibria \( E_1 \) and \( E_2 \) are real and virtual (denoted as \( E_1^0 \) and \( E_2^0 \), respectively. If \( H_1 < c_2 < H_2 \) (i.e., \( \hat{\Omega}_4 \cup \hat{\Omega}_5 \) in Fig. 1), then both equilibria \( E_1 \) and \( E_2 \) are virtual (denoted as \( E_1^0 \) and \( E_2^0 \)). In this case the pseudo-equilibrium \( E_P \) is a locally asymptotically stable. If \( c_2 < H_1 \) (i.e., \( \hat{\Omega}_6 \) in Fig. 1), then the equilibria \( E_1 \) and \( E_2 \) are real and virtual (denoted as \( E_1^0 \) and \( E_2^0 \), respectively.

To investigate the global stability and the long-term dynamics of Filippov system (7), we initially explore the
relationship between the sliding domain \( \Sigma_s \) and the invariance region of Filippov system (7)
\[
\Omega \triangleq \left\{ S + I_1 \in \mathbb{R}_+^2 \mid 0 < S + I_1 \leq d = \frac{A}{\min(\mu_5, \mu_1, \mu_2)}, S \geq 0, I_1 \geq 0 \right\}.
\]

The sliding domain \( \Sigma_s \) lies in the invariance region \( \Omega \) if \( S_i^2 < d - I_1 \), i.e.,
\[
c_2 < b_2 c_1 (d - S_i^2) \triangleq H_3
\]
and \( \Sigma_s \) is out of \( \Omega \) if \( S_i^2 > d - I_1 \), i.e.,
\[
c_2 > b_2 c_1 (d - S_i^2) \triangleq H_4.
\]

4.2. Boundary equilibrium bifurcation

Boundary equilibrium bifurcations in Filippov system are characterized by the collision of pseudo-equilibrium, tangent point, and real equilibrium (or tangent point and real equilibrium) at the discontinuity surface when one parameter passes through a critical value. Throughout this section, we will investigate the boundary equilibrium bifurcation of Filippov system (7), and we first discuss the tangent point and boundary equilibrium as following.

Tangent point of Filippov system (7) satisfies
\[
\beta_1 S_1 - v_1 - \frac{c_1 I_1}{1 + b_2 k} = 0, \quad I_1 = I_c,
\]
solving the above equations with respect to \( S \) yields \( E^1_b = (S^1_b, I_c) \) or \( E^2_b = (S^2_b, I_c) \). Note that \( E^1_b \) and \( E^2_b \) are the endpoints of the sliding segment \( \Sigma_s \).

Boundary equilibrium of Filippov system (7) satisfies
\[
A - \mu S - \beta_1 S_1 = 0, \quad \beta_1 S_1 - v_1 - \frac{c_1 I_1}{1 + b_2 k} = 0, \quad I_1 = I_c,
\]
which indicate that if
\[
\frac{A}{\mu + \beta_1 I_c} = \frac{1}{\beta_1} \left( v + \frac{c_1}{1 + b_2 k} \right),
\]
then we have the boundary equilibrium \( E^1_b = (S^1_b, I_c) \) or \( E^2_b = (S^2_b, I_c) \).

Further, we have
\[
F_{S_1} H(E^1_b) = \left( \beta_1 S^1_b - v - \frac{c_1}{1 + b_2 k} \right) I^2 = \frac{c_1 b_2 k (1 - R_{01})}{\beta_1 (1 + b_2 k)} < 0,
\]
and from the expression of \( \Delta_i \) in Section 2.2, we know that
\[
\frac{det(F_{S_1} (E^1_b))}{(2 S_i^2)} \text{ possesses complex eigenvalues with nonzero real part } -A/(2 S_i^2) \text{ or nonzero real eigenvalues } \left( -A \pm \sqrt{\Delta_i S_i^2} \right)/(2 S_i^2) \text{ depends on the sign of } \Delta_i, i = 1, 2.
\]

According to Definition 2.3, a boundary equilibrium bifurcation occurs at \( E^3_b \). That is, the existence of a boundary equilibrium indicates the existence of a boundary equilibrium bifurcation.

In summary, we get the following conclusion.

**Theorem 4.1.** A boundary focus (node) bifurcation occurs at \( E^3_b \) if \( \Delta_i < 0 (\Delta_i > 0), i = 1, 2 \).

Especially, from Fig. 2, we can see that the stable focus \( E^3_b \) and a tangent point \( E^2_b \) collide together as the parameter \( c_2 \) passes through the critical value \( c_2 = 0.2631 \) (in this case, \( I_c = 0.2631, \Delta_2 = -0.5825 < 0 \)), the boundary focus bifurcation occurs at \( E^3_b \). A stable focus \( E^2_b \) and a tangent point \( E^2_b \) coexist, as shown in Fig. 2(A) when \( c_2 < 0.2631 \).

\[\text{(A)}\]
\[\text{(B)}\]
\[\text{(C)}\]

**Fig. 2.** Boundary focus bifurcation for Filippov system (7). Here we choose \( c_2 \) as a bifurcation parameter and fix all other parameters as follows: \( A = 0.7, \beta_1 = 1, \beta_2 = 0.01, \mu_1 = 0.39, \mu_2 = 0.5, \mu_4 = 0.38, v_1 = 0.01, b_2 = 2, \quad c_1 = 0.5, \quad k = 4 \), and (A) \( c_2 = 0.1 \); (B) \( c_2 = 0.2631 \); (C) \( c_2 = 0.6 \).
They collide at \( c_2 = 0.2631 \) (see Fig. 2) and are substituted by a pseudo-equilibrium \( E_p \) and a tangent point \( E_T^2 \) as \( c_2 > 0.2631 \), see Fig. 2(C) for more details.

Similarly, a boundary node bifurcation of Filippov system (7) occurs at \( E_1^1 \) as \( c_2 = 0.6477 \) (in such case, \( I_c = 0.6477, \Delta_1 = 0.0667 > 0 \), see Fig. 3. A stable node \( E_T^1 \) and a tangent point \( E_T^1 \) coexist, as shown in Fig. 3(A) when \( c_2 > 0.6477 \). They collide at \( c_2 = 0.6477 \) (see Fig. 3) and are substituted by a pseudo-equilibrium \( E_p \) and a tangent point \( E_T^1 \) when \( c_2 < 0.6477 \), as shown in Fig. 3(C).

5. Global behavior

In this section, we focus on the asymptotical behaviors of all the possible equilibria including two real equilibria \( E_i, i = 1, 2 \) and pseudo-equilibrium \( E_p \) for Filippov system (7) provided \( R_{\omega} > 1 \). To do so, we need to rule out the existence of limit cycle. For convenience, we summarize three types of limit cycles in \( \Omega \) for Filippov system (7) as following.

I. Limit cycle composed only by the orbit of the vector field \( F_{S_i}(Z) \) or \( F_{S_2}(Z) \), as shown in Fig. 4(A).

II. Crossing cycle tangents to the sliding segment \( \Sigma_i \) (see Fig. 4(B)) or contains part of the sliding segment \( \Sigma_i \) (see Fig. 4(C)).

III. Crossing cycle surrounds the sliding segment \( \Sigma_i \), as shown in Fig. 4(D).

5.1. Non-existence of limit cycle

In order to prove the global stability of the equilibrium of system (7), we need to rule out the existence of limit cycles listed above. We initially preclude the existence of the first type of limit cycle, i.e., limit cycle totally in region \( S_1 \) or \( S_2 \). Denote the right-hand side of system \( S_i \) by \( f^{(i)}(Z) \), where \( f^{(i)}(Z) = (f_1^{(i)}(Z), f_2^{(i)}(Z)), i = 1, 2 \).

**Fig. 3.** Boundary node bifurcation for Filippov system (7). Here we choose \( c_2 \) as a bifurcation parameter, (A) \( c_2 = 0.75 \); (B) \( c_2 = 0.6477 \); (C) \( c_2 = 0.4 \). The other parameters are identical to those in Fig. 2.

**Fig. 4.** Phase plane \( S-I_1 \) of Filippov system (7) to illustrate all the types of possible limit cycles in the invariance region \( \Omega \).

**Lemma 5.1.** There exists no limit cycle totally composed by the orbit of the vector field \( F_{S_1}(Z), i = 1, 2 \).

**Proof.** Let the Dulac function be \( B(S, I_1) = 1/(S I_1) \) for subsystem \( S_1 \), we have
\[
\frac{\partial (B f_1^{(i)})}{\partial S} + \frac{\partial (B f_2^{(i)})}{\partial I_1} = -\frac{A}{S^2 I_1} < 0,
\]
so there is no limit cycle totally in region \( S_i \) for \( i = 1, 2 \).

Therefore, there exists no limit cycle totally composed by the orbit of the vector field \( F_{S_1}(Z) \) or \( F_{S_2}(Z) \). This completes the proof of Lemma 5.1.

Next, we preclude the existence of the second type of limit cycles.

**Lemma 5.2.** There exists no limit cycle surrounding a sliding segment, which contains a tangent point only or part of a sliding segment.

**Proof.** In order to proof Lemma 5.2, we consider the following three cases.

**Case i:** Assume that \( I_2^2 < I_c < I_1^1 \), i.e., \( H_1 < c_2 < H_2 \).

It follows that there exists a pseudo-equilibrium \( E_p \) which is locally asymptotically stable in the sliding domain \( \Sigma_i \). The local stability of \( E_p \) on the sliding domain indicates that the conclusion in Lemma 5.2 follows.

**Case ii:** Assume that \( I_c < I_2^1 \), i.e., \( c_2 < H_1 \).

In the sliding domain \( \Sigma_i \) (here the segment \( E_T^1 \) in Fig. 5), we have
\[
\dot{S}(t) = A - \mu S - \beta_i S I_c > 0,
\]
which shows that the trajectory moves from the left to the right on \( \Sigma_i \).

We should show that the orbit \( I_1 \) initiating at \( E_T^1 \) will not hit the sliding domain \( \Sigma_i \) again. Note that the orbit \( I_1 \)
starting at $E_1^2$ either tends to the stable equilibrium $E_2^0$ directly or spirally since $E_2^0$ could be a stable node or focus in region $S_2$. If the latter happens, then the orbit $l_1$ intersects with the horizontal isocline $g_1^2$ as the point $M_1$ first, and $N_1$ second, where $N_1$ is on the segment $E_2^0E_1^2$. Obviously, the two points $M_1$ and $N_1$ are above the point $E_1^2$. Hence, $l_1$ starting at $E_1^2$ cannot form a cycle, as shown in Fig. 5.

**Case iii:** Assume that $l_1 > l_1^*$, i.e., $c_2 > H_2$. We can use a similar process as Case ii to prove the conclusion. Therefore, the combination of Cases i–iii, there exists no limit cycle contains a tangent point only or part of a sliding segment. This completes the proof of Lemma 5.2. □

In order to preclude the existence of the third type of limit cycle, we give the following lemma and detailed proof.

**Lemma 5.3.** There exists no limit cycle surrounding the whole sliding segment.

**Proof.** We supposed that Filippov system (7) has a limit cycle $\Gamma$ in $\Omega$, and $\Gamma$ surrounds the sliding segment $T_1T_2$. As shown in Fig. 6, $\Gamma$ is divided into two parts $\Gamma_1$ and $\Gamma_2$ by the manifold $\Sigma$, we denote the intersection points by $H_1$ and $H_2$. Meanwhile, the intersection points between $\Gamma$ and the auxiliary line $l_1 = l_1 - \epsilon$ (or $l_1 = l_1 + \epsilon$) are $A_1, A_2$ (or $A_3, A_4$), where $\epsilon > 0$ is sufficiently small. The region bounded by $\Gamma_1$ (or $\Gamma_2$) and segment $A_1A_2$ (or $A_3A_4$) is denoted with $G_1$ (or $G_2$), and we denote the boundary of $G_1$ (or $G_2$) by $L_1$ (or $L_2$), respectively, and the directions indicated in Fig. 6. Let the Dulac function be $B = 1/(S_1)$, it follows from Green’s theorem that

$$\int_{\Gamma_1} \left[ \frac{\partial B_1^{(1)}}{\partial S} + \frac{\partial B_2^{(1)}}{\partial l_1} \right] dS dl_1 = B \int_{\Gamma_1} [f_1^{(1)} dl_1 - f_2^{(1)} ds]$$

Similarly, we have

$$\int_{\Gamma_2} \left[ \frac{\partial B_1^{(2)}}{\partial S} + \frac{\partial B_2^{(2)}}{\partial l_1} \right] dS dl_1 = -\int_{A_2A_1} B_2^{(2)} ds.$$ 

If $G_0 \subset G_1$, we have

$$\xi \triangleq \int_{G_0} \left[ \frac{\partial B_1^{(1)}}{\partial S} + \frac{\partial B_2^{(1)}}{\partial l_1} \right] dS dl_1 < 0,$$

then we obtain

$$0 > \xi > \int_{G_0} \left[ \frac{\partial B_1^{(1)}}{\partial S} + \frac{\partial B_2^{(1)}}{\partial l_1} \right] dS dl_1 + \int_{G_0} \left[ \frac{\partial B_1^{(2)}}{\partial S} + \frac{\partial B_2^{(2)}}{\partial l_1} \right] dS dl_1$$

$$= -\int_{A_2A_1} B_2^{(2)} ds - \int_{A_2A_4} B_2^{(2)} ds.$$ 

For the sake of simplicity of computation, we denote the abscissas of the points $H_1, H_2, A_1, A_2, A_3, A_4$ by $x_1, x_2, x_3, h_1(\epsilon), x_2 - h_2(\epsilon), x_1 + h_1(\epsilon), x_2 - h_4(\epsilon)$, where $h_i(\epsilon) > 0$ is continuous and satisfies $\lim_{\epsilon \to 0} h_i(\epsilon) = 0$ for $i = 1, 2, 3, 4$. Thus, we have

$$\lim_{\epsilon \to 0} \left[ -\int_{A_2A_1} B_2^{(2)} ds \right] = \lim_{\epsilon \to 0} \int_{x_2 - h_2(\epsilon)}^{x_1 + h_1(\epsilon)} \left( \beta_1 - \frac{v + c_1}{x_1 + h_1(\epsilon)} \right) ds$$

$$= \lim_{\epsilon \to 0} \left[ \beta(x_2 - h_2(\epsilon) - x_1 + h_1(\epsilon)) - \left( v + \frac{c_1}{1 + b_2 k} \right) \ln \frac{x_2 - h_2(\epsilon)}{x_1 + h_1(\epsilon)} \right]$$

$$= \beta(x_2 - x_1) - \left( v + \frac{c_1}{1 + b_2 k} \right) \ln \frac{x_2}{x_1}.$$ 

Analogously, we get

$$\lim_{\epsilon \to 0} \left[ -\int_{A_2A_4} B_2^{(2)} ds \right] = \beta(x_1 - x_2) + (v + c_1) \ln \frac{x_2}{x_1}.$$
Therefore,
\[
\lim_{\epsilon \to 0} \left[ -\int_{A_2A_1} B_{12}^{(1)} \, ds - \int_{A_3A_4} B_{12}^{(2)} \, ds \right] = \frac{c_1 b_2 k}{1 + b_2 k} \ln \frac{x_2}{x_1} > 0
\]
which contradicts with (13). This precludes the existence of the limit cycle. This completes the proof of Lemma 5.3.

5.2. Global stability of Filippov system (7)

To establish all possible behaviors that Filippov system (7) can exhibit, we choose the corresponding parameter values such that the dynamics in all regions are presented in Fig. 1. We initially show that $E^l_k$ is globally asymptotically stable as following.

**Theorem 5.4.** For Filippov system (7), the endemic equilibrium $E^l_k$ is globally asymptotically stable if $c_2 > H_2$ (i.e., $I_c > I^*_1$).

**Proof.** By calculation, the two endemic equilibria $E^l_k$ and $E^v_k$ for two structures lie on the same side of $\Sigma$ as $c_2 > H_2$. Although there exists the sliding domain $\Sigma$, no pseudo-equilibrium exists in $\Sigma$. The endemic equilibrium $E^l_k$ is locally asymptotically stable in $S_1$ since $R_{01} > 1$. It follows from the sliding domain $\Sigma$ that
\[
\dot{S}(t) = A - \mu S - \beta_1 S I_c < 0,
\]
which indicates that the trajectory moves from the right to the left on the sliding segment $E^l_kE^v_k$ (see Fig. 7).

Noting that $H_1 < H_4$, $H_1 < H_2$, $H_1 < H_3$, $H_2 < H_4$. Then, either $H_3 < H_2$ or $H_3 > H_2$ may hold true. For different sets of values of the parameters, the sliding segment may be exclusively, partly, or totally in the invariance region $\Omega$, so we consider the following three cases.

**Case i.** If $c_2 > H_4$ (the region $\Omega_1$ shown in Fig. 1), the sliding segment $E^l_kE^v_k$ lies in $\Omega$, as shown in Fig. 7(A).

According to Lemmas 5.1,5.2,5.3, we can preclude the existence of three types limit cycles listed above. Hence, the endemic equilibrium $E^l_k$ is globally asymptotically stable.

**Case ii.** If $\max\{H_2, H_3\} < c_2 < H_4$ (the region $\Omega_2$ shown in Fig. 1), the part of the sliding segment $E^l_kE^v_k$ lies in $\Omega$, as shown in Fig. 7(B). Note that again the direction of the vector field in region $S_2 \cap \Omega$ points downward. Moreover, according to Lemma 5.2, we know that there exists no closed orbit containing part of the sliding segment $E^l_kE^v_k$. Hence, the endemic equilibrium $E^l_k$ is globally asymptotically stable.

**Case iii.** If $c_2 < \max\{H_2, H_3\}$ (the region $\Omega_3$ shown in Fig. 1), the sliding segment is out of $\Omega$, as shown in Fig. 7(C).

The two endemic equilibria $E^l_k$ and $E^v_k$ lie in $S_1 \cap \Omega$, and the direction of the vector field in $S_2 \cap \Omega$ points downward. Hence, there exists no limit cycle in $\Omega$ which is partly in $S_1$ and partly in $S_2$. In addition, by Lemma 5.1, there is no limit cycle totally in $S_1$. Therefore, the endemic equilibrium $E^l_k$ is globally asymptotically stable.

![Fig. 7. Global stability of the endemic equilibrium $E^l_k$. Parameters are:](image)

$A = 0.6, \beta_1 = 2, \beta_2 = 0.5, \mu_0 = 0.39, \mu_1 = 0.5, \mu_2 = 0.38, \nu_1 = 0.01, b_2 = 1, k = 2$

and (A) $c_1 = 0.9, c_2 = 0.7$; (B) $c_1 = 0.9, c_2 = 0.85$; (C) $c_1 = 0.8, c_2 = 1$. 

W. Qin, S. Tang / Chaos, Solitons & Fractals 69 (2014) 160–171
Note that both $E_1^1$ and $E_2^1$ lie in $S_1$, which indicate that all trajectories will attain the subregion of $\Omega$ below the manifold $\Sigma$. Moreover, they will remain in it and approach the endemic equilibrium $E_3$. Hence $E_2$ is globally asymptotically stable. This completes the proof of Theorem 5.4. \hfill \Box

Next, we will show that $E_P$ is globally asymptotically stable in the sliding domain $\Sigma_i$ as following.

**Theorem 5.5.** For Filippov system (7), the pseudo-equilibrium $E_P$ is globally asymptotically stable if $H_1 < c_2 < H_2$ (i.e., $I_2 < I_c < I_1^1$).

**Proof.** It is easy to see that both equilibria $E_1$ and $E_2$ are virtual (i.e., $E_1^1$ and $E_2^1$), and the pseudo-equilibrium $E_P$ exists in $\Sigma$ which is locally asymptotically stable. Note that the sliding segment $\Sigma_i$ may be partly or totally in $\Omega$. Then there are two possibilities to consider.

**Case i.** If $c_2 \geq \min\{H_2, H_3\}$ (the region $\Omega_4$ shown in Fig. 1), and part of the sliding segment $E_1^2$ lies in $\Omega$, as shown in Fig. 8(A), we can preclude the existence of limit cycles by using a similar method to Case ii in Theorem 5.4.

**Case ii.** If $c_2 < \min\{H_2, H_3\}$ (the region $\Omega_2$ shown in Fig. 1), and the whole sliding segment lies in $\Omega$, as shown in Fig. 8(B). We can rule out the existence of limit cycles by Lemma 5.3.

From what we have discussed above, there exists no limit cycle in $\Omega$. Hence, the pseudo-equilibrium $E_P$ is globally asymptotically stable. This completes the proof of Theorem 5.5. \hfill \Box

Finally, we show that $E_2^2$ is globally asymptotically stable as following.

**Theorem 5.6.** For Filippov system (7), the endemic equilibrium $E_2^2$ is globally asymptotically stable if $c_2 < H_1$ (i.e., $I_c < I_1^2$).

**Proof.** If $c_2 < H_1$ (the region $\Omega_4$ shown in Fig. 1), and the whole sliding segment lies in $\Omega$, as shown in Fig. 9.

Obviously, there exist two endemic equilibria $E_1^2$ and $E_2^2$ and no pseudo-equilibrium. The sliding mode $\Sigma_4$ exists, and the trajectory moves from left to right on $E_1^2 E_2^2$ in this case, as shown in Fig. 9. Using a similar method to Case i of Theorem 5.4, we can rule out the existence of limit cycle totally in $\Omega$. Hence, the endemic equilibrium $E_2^2$ is globally asymptotically stable. This completes the proof of Theorem 5.6. \hfill \Box

**Remark.** If $R_{01} < 1$, Filippov system (7) will stabilize at $E_0$ which is the disease-free equilibrium of free system $S^1$ (as shown in Fig. 10). In fact, if $R_{01} < 1$, Filippov system (7) does not have any regular equilibrium and sliding segment. According to Lemma 5.1, there exists no limit cycle for Filippov system (7). Consequently all the trajectories of Filippov system (7) will definitely hit the switching surface $\Sigma$, and finally stabilize at the boundary equilibrium $E_0$ of free system $S^1$.
5.3. Key parameters and biological significance

What we consider in the following is the effects of key parameters on the threshold values $R_{01}$ and $R_{02}$.

Although the threshold values $R_{01}$ and $R_{02}$ depend on all parameters of Filippov system (7), the most interesting parameter here is $c_1$, which is an important factor in controlling the spread of SARS. Obviously, $R_{01}$ and $R_{02}$ are monotonic decreasing functions with respect to $c_1$ as $\partial R_{01}/\partial c_1 < 0$ and $\partial R_{02}/\partial c_1 < 0$. Meanwhile, we can calculate the threshold

$$c_1 = \frac{(A\beta_1 - \mu)v(1 + b_2k)}{\mu},$$

such that $R_{01}(c_1) = 1$. So $R_{01}(c_1) < 1$ provided $c_1 > c_1$.

It follows from $I_c = c_2/(c_1b_2)$ that $I_c$ is a monotonic decreasing function with respect to $c_1$. Thus, based on the critical value $c_1$ the threshold value $I_c$ should be reduced as

$$I_c^* = \frac{c_2}{c_1b_2} = \frac{c_2\mu}{b_2(A\beta_1 - \mu)v(1 + b_2k)};$$

(14)

which could contribute to the efficacy on prevention and treatment of SARS. So the doctors choose the threshold hold $I_c$ no more than $I_c^*$ to implement selection treatment for SARS cases, as shown in Fig. 11. That is, at the outset of SARS outbreak (i.e., $I_1 < I_c^*$), SARS can be well-controlled by effective treatments, once the number of infected SARS cases reaches the threshold $I_c^*$ (i.e., $I_1 > I_c^*$), the limited medical resource cannot prevent SARS from spinning out of control (the dotted red line shows in Fig. 11), at this moment, we should take the highly selective treatment for SARS, and it will be brought under control (the solid blue line shows in Fig. 11). Therefore, the selection pressures can help us to prevent and treat SARS under limited medical resource.

Further, we investigate how the threshold value $I_c$ affects the spread of SARS. To do this, we let $c_1$ vary and fix all other parameters as those shown in Fig. 12. It is easy to calculate the threshold value $I_c^* \approx 0.29$ according to (14).

From Fig. 12, the blue line shows that SARS can be controlled as $I_c < I_c^*$, while the red and magenta lines show that SARS will be out of control as $I_c > I_c^*$, which indicates that it is very important to choose an appropriate threshold value $I_c$ to decide when the selective strategy should be implemented for prevention and treatment of SARS.

Therefore, in order to prevent and treat the patients infected by SARS, it points to the urgent need for improvements in medical facilities, access to a rapidly, accurate and efficient way for wild-ranged screening and early diagnosis for SARS. It is best to timely selective treatment for SARS infected cases so as not to miss the best timing of treatment. Meanwhile, it is essential for the doctors to develop more effective drugs for diseases prevalent. This
can greatly relieve the pressure of limited medical resource on the doctors or hospital.

6. Biological conclusion and discussion

In present work, we have proposed a non-smooth infectious disease model induced by selection pressures under medical resource limitation. In order to understand the effects of selection pressure on infectious disease transmission, by employing the qualitative theory and bifurcation techniques of non-smooth systems [13,27–29], we deliberately investigate the long-term dynamic behavior of the proposed Filippov model. In particular, the sliding mode dynamics, the sliding bifurcations and global dynamics of the proposed model have been addressed.

By using Utkin’s equivalent control method introduced in [13], we first obtain the differential equation for sliding dynamics of the Filippov system (7), and then the sliding mode dynamics and the local sliding bifurcations have been addressed by applying bifurcation theories [27–29], see Figs. 2 and 3. Meanwhile, the global dynamical behavior has been established by excluding the existence of limit cycles for system (7), as shown in Figs. 7–9.

Model (7) could stabilize at either one of the two equilibria \( E^1_p \) and \( E^2_p \) or the pseudo-equilibrium \( E_p \) on the switching surface, depending on the threshold level \( I_L \), which is determined by \( c_1, c_2, b_2 \) (i.e., \( I_L = c_2/(c_1b_2) \)). Especially, Fig. 8 indicates that the pseudo-equilibrium \( E_p \) is globally asymptotically stable, which indicates that the infected population can stabilize at a previously chosen level \( I_L \) once the threshold policy and some related parameters (i.e., \( c_1, c_2, b_2 \)) are chosen properly. Hence, it is very crucial to choose appropriate control parameters for making the decision to trigger the intervention on infectious disease transmission.

The main results also show that on the prevention and treatment of SARS, we should choose an appropriate threshold \( I_L \) (i.e., \( I_L = I^* \)) at which the selection treatment strategy should be implemented and decided. That is, if the number of people infected by SARS is large than the threshold \( I_L \), then we should take emergency medical treatment services (such as isolated treatment, personal protection, medical observation, sterilization and so on) immediately only for SARS cases due to medical resource limitation. Only in this way, SARS can be controlled as soon as possible. Those indicate that the results obtained here could be beneficial for accurately assessing the effect of selection pressure in the control and treatment of SARS (as shown in Figs. 11 and 12). In particular, the selection treatment strategy can help us to prevent the new emerging infectious disease.

However, this study is a special case that the patients infected by SARS will be taken care immediately no matter medical resource limit (i.e., \( b_1 = 0 \)). In fact, there exists limited medical resource in real world especially in rural areas for new emerging infectious disease. Therefore, if \( b_1 > 0 \), the dynamical behavior of both subsystems become much more complex and what we want to investigate is how the dynamic behavior of the model could be dramatically affected by the existence of medical resource limitation, and consequently influences the prevention and control for emerging infectious disease. Hence, we leave this work as our future study.

Acknowledgement

This work is supported by the National Natural Science Foundation of China (NSFCs: 11471201, 11171199, 11401360, 11371030, 11301320) and the Fundamental Research Funds for the Central Universities of China (GK201305010, GK201401004).

References

[1] Valleron AJ, Cori A, Valtat S, Meurisse S, Carrat F, Boelle PY. Transmissibility and geographic spread of the 1889 influenza pandemic. Proc Natl Acad Sci USA 2010;107:8778–81.
[2] Mills CE, Robins JM, Lipstitch M. Transmissibility of 1918 pandemic influenza. Nature 2004;432:904–6.
[3] Donaldson LJ, Rutter PD, Ellis BM, Greaves FE, Mytton OT, Pebbey RG, Yardley IE. Mortality from pandemic A/H1N1 2009 influenza in England: public health surveillance study. BMJ 2009;339:b5213.
[4] First global estimates of 2009 H1N1 pandemic mortality released by CDC-Led collaboration; 2012. <http://www.cdc.gov/flu/spotlights/pandemic-global-estimates.htm> [Online].
[5] World health organization programme budget for 2012–2013. <http://www.who.int/en/> [Online].
[6] Matrajt L, Halloran ME, Longini Jr JM. Optimal vaccine allocation for the early mitigation of pandemic influenza. PloS Comput Biol 2009;5:e1002964.
[7] Sullivan SP, Koutsonanoges DG, Martin MDP, Lee JW, Zarnitsyn V, Choi SO, Murthy N, Compans RW, Skountzou J, Prausnitz MR. Dissolving polymer microneedle patches for influenza vaccination. Nat Med 2013;19:19–20.
[8] Vaccine-delivery patch with dissolving microneedles eliminates sharps boosts protection; 2010. <http://www.sciencedaily.com/releases/2010/07/100718204733.htm> [Online].
[9] Qin WJ, Tang SY, Cheke RA. Nonlinear pulse vaccination in an SIR epidemic model with resource limitation. Abstr Appl Anal 2013:13 [Article ID 670263].
[10] Utkin VI. Sliding modes and their applications in variable structure systems. Moscow: Mir Publishers; 1978.
[11] Utkin VI. Sliding modes in control and optimization. Berlin: Springer-Verlag; 1992.
[12] Filippov AF. Differential equations with discontinuous righthand sides. Dordrecht: Kluwer Academic Publishers; 1988.
[13] Utkin VI, Guldner J, Shi JX. Sliding mode control in electromechanical systems. London: Taylor Francis Group; 2009.
[14] Xiao YN, Xu X, Tang SY. Sliding mode control of outbreaks of emerging infectious diseases. Bull Math Biol 2013;75:1743–66.
[15] Wang AL, Xiao YN. A Filippov system describing media effects on the pandemic of SARS. Abstr Appl Anal 2013:13 [Article ID 670263].
[16] Bernardo MD, Budd CJ, Champneys AR, Kowalczyk P, Nordmark AB, Tost GO, Piironen PT. Bifurcations in nonsmooth dynamical systems. SIAM Rev 2008;50:629–701.
[17] Brogliato B. Nonsmooth mechanics. New York: Springer-Verlag; 1999.
[18] Fonda MIS, Kaszkurewicz E, Bhaya A, Hsu L. Achieving global convergence to an equilibrium population in predator–prey systems by the use of a discontinuous harvesting policy. Ecol Model 2000;128:89–99.
[19] Dercole F, Gagnani A, Rinaldi S. Bifurcation analysis of piecewise smooth ecological models. Theor Popul Biol 2007;72:197–213.
[20] Doole SH, Hogan SJ. A piecewise linear suspension bridge model: nonlinear dynamics and orbit continuation. Dyn Syst 1996;11:19–29.
[21] Van de Vrande BL, Van Campen DH, De Kraker A. An approximate analysis of dryfriction-induced stick–slip vibration by a smoothing procedure. Nonlinear Dyn 1999;19:157–69.
[22] Tang SY, Liang JH, Xiao YN, Cheke RA. Sliding bifurcations of Filippov two stage pest control models with economic thresholds. SIAM J Appl Math 2012;72:1081–90.
[23] Tang SY, Liang JH. Global qualitative analysis of a non-smooth Gauss predator–prey model with a refuge. Nonlinear Anal TMA 2013;76:165–80.
[24] Tang GY, Qin WJ, Tang SY. Complex dynamics and switching transients in periodically forced Filippov prey–predator system. Chaos Solitons Fract 2014;61:13–23.

[25] Zhang X, Liu XN. Backward bifurcation of an epidemic model with saturated treatment function. J Math Anal Appl 2008;348:433–9.

[26] Ślebodziński W. Sur les équations de Hamilton. Bull Acad Roy Belg 1931;17:864–70.

[27] di Bernardo M, Budd CJ, Champneys AR, Kowalczyk P, Nordmark AB, Tost GO, Piiróinen PT. Bifurcations in nonsmooth dynamical systems. SIAM Rev 2008;50:629–701.

[28] Guardia M, Seara TM, Teixeira MA. Generic bifurcations of low codimension of planar Filippov systems. J Differ Equ 2011;250:1967–2023.

[29] Kuznetsov YuA, Rinaldi S, Gragnani A. One parameter bifurcations in planar Filippov systems. Int J Bifurcation Chaos 2003;13:2157–88.