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Multiple Equilibria in a Non-smooth Epidemic Model with Medical-Resource Constraints

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Abstract

The issue of medical-resource constraints has the potential to dramatically affect disease management, especially in developing countries. We analyze a non-smooth epidemic model with nonlinear incidence rate and resource constraints, which defines a vaccination program with vaccination rate proportional to the number of susceptible individuals when this number is below the threshold level and constant otherwise. To better understand the impact of this non-smooth vaccination policy, we provide a comprehensive qualitative analysis of global dynamics for the whole parameter space. As the threshold value varies, the target model admits multistability of three regular equilibria, bistability of two regular equilibria, that of one disease-free equilibrium and one generalized endemic equilibria, and that of one disease-free equilibrium and one crossing cycle. The steady-state regimes include healthy, low epidemic and high epidemic. This suggests the key role of the threshold value, as well as the initial infection condition in disease control. Our findings demonstrate that the case number can be contained at a satisfactorily controllable level or range if eradicating it proves to be impossible.

Keywords Non-smooth epidemic model · Resources constraints · Nonlinear incidence · Generalized equilibrium · Crossing cycle · Multistability

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1 Introduction

Containing and mitigating infectious diseases are two of the challenging issues in our time due to the complex spreading patterns and increasing spread speed (Brockmann and Helbing 2013), despite notable success in prevention and control. Many efforts to curb a disease attempt to cut the transmission path or control it at its source. Important progress has been made by proposing mathematical models, which offer valuable information for decision making in global health (Heesterbeek et al. 2015; Xiao and Tang 2010). Modeling techniques help us to understand the observed epidemiological patterns and predict the consequences of the introduction of intervention measures to contain disease spread. However, one issue of practical concern, as a disease becomes more prevalent, is the limitations in the medical resources available to prevent those from being infected by a particular pathogenic agent or to treat those who have fallen ill. In the absence of sufficient vaccines and treatment, mathematical modeling can explore efficient control strategies (Wang and Ruan 2004; Zhang and Liu 2008; Wang 2006; Hansen and Day 2011; Zhou and Fan 2012; Shan and Zhu 2014; Wang et al. 2018; Böttcher et al. 2015; Qin et al. 2016; Abdelrazec et al. 2016; Shan et al. 2016; Wang and Xiao 2014).

One common aim when modeling medical-resource constraints is to describe how changes in intervention measures will affect the characteristics of the infection dynamics and consequently affect disease control. Thus, many control programs such as treatment and vaccination have been modeled. Classical epidemic models usually assume the vaccination rate is proportional to the size of the susceptible population, which has made key contributions to the vaccination program design (Xiao and Tang 2010; Rodrigues et al. 2014; Samsuzzoha et al. 2013). However, in many situations, we do not have enough resources to target all those being exposed in the contaminated environment. Faced with the limited capacity of medical resources, other more appropriate control measures have been proposed. Zhang and Liu (2008) adopted a saturated treatment function to characterize the limited medical resources. A similar treatment function is introduced in an SIR epidemic model to reveal the impact of varying the amount of medical resources on the transmission process (Zhou and Fan 2012). To clearly depict the impact of hospital settings on disease control, some more complicated saturated functions are defined to represent the treatment measure (Shan and Zhu 2014; Shan et al. 2016), which triggers rich dynamics including a saddle-node bifurcation, a Bogdanov–Takens bifurcation and a backward bifurcation. To determine a suitable capacity for treatment, Wang and Ruan first introduce a piecewise-defined function (Wang and Ruan 2004). This type of treatment function is further modified as following

$$T(I) = \begin{cases} rI, & \text{if } 0 \le I \le I_0 \\ rI_0 & \text{if } I \ge I_0, \end{cases}$$

which admits a backward bifurcation when the medical-resource capacity is small (Wang 2006).

The piecewise-defined treatment function is a good approximation for cases when the available medical resources cannot meet the demand of increasing infective cases.

It defines a control policy such that the maximum treatment rate is applied when the number of infectives is above some critical value; otherwise, the treatment ratio is proportional to the case number. However, these studies have focused mainly on the dynamics with fixed threshold levels, and little analysis has been done to explore the impact of threshold level on the dynamical behavior. In fact, the choice and positioning of the threshold is guided by the capacity of resources, so different threshold levels are available with different amounts of medical resources. In the present work, we adopt this type of piecewise-defined function to represent vaccination and explore how it affects the outcome of disease control. Our work extends that of Wang (2006) by examining the dynamic phenomena triggered by the variation of the threshold level.

Besides the medical-resource constraints, the incidence rate (i.e., the rate of new infections) is another key factor in modeling a communicable disease. Mass-action transmission has frequently been used in many classical disease-transmission models, which leads to a wide application of bilinear and standard incidence rates (Hethcote 2000; Brauer and Chavez 2001). Since Capasso and Serio have generalized the incidence rate to a saturated form (Capasso and Serio 1978), there is an increasing interest in describing the disease dynamics with nonlinear incidence rate (Xiao and Tang 2010; Liu et al. 1986, 1987; Xiao and Ruan 2007; Tripathi and Abbas 2016; Li et al. 2015; Li and Zhang 2017). To incorporate the impact of individual behavior changes on disease spread, Liu et al. chose a nonlinear incidence rate of the form $\beta S^q I^q$ with $p, q > 0$ (Liu et al. 1986, 1987), which is then used by Xiao in a vaccination model. In the latter case, a nonlinear incidence rate induces both forward and backward bifurcations (Xiao and Tang 2010).

The aim of this study is to use a vaccination model, including susceptible, infected and vaccinated classes, with a nonlinear incidence rate to gain insight into the complex dynamics of an epidemic when the vaccination of susceptible individuals depends on a threshold, which is built on the capacity of resources. The result is a non-smooth continuous model, which behaves differently from its continuous counterpart. The theoretic approach for non-smooth continuous systems has gained recent interest (Coll et al. 2001; Claudio et al. 2006; Han and Zhang 2010), but only a few applications have been found in disease control (Wang 2006; Wang et al. 2014). We emphasize that our goal is to explore how the variation of the threshold value gives rise to dynamic phenomena, especially the existence of multiple steady states, generalized endemic equilibria and a crossing cycle. We also seek to characterize how these novel phenomena in our targeted model affect disease control.

2 Model Formulation and Preliminaries

Nonlinear incidence rates are more reasonable in some circumstances than bilinear ones. We aim to explore how varying the threshold value, together with double exposure of susceptible individuals affects disease control. We adopt the nonlinear incidence rate βSI^2 to represent the effect of double explore of the susceptibles on disease spread. A more detailed explanation on this subject can be found in Xiao and Tang (2010). The model takes the form

$$\begin{aligned}\frac{\mathrm{d}S}{\mathrm{d}t} &= \Lambda - \beta SI^2 - \mu S - H(S),\\ \frac{\mathrm{d}I}{\mathrm{d}t} &= \beta SI^2 - \mu I - \epsilon I,\\ \frac{\mathrm{d}V}{\mathrm{d}t} &= H(S) - \mu V.\end{aligned} \tag{1}$$

where $S(t)$, $I(t)$, $V(t)$ are the numbers of susceptible, infected and immune individuals at time t and

$$H(S) = \begin{cases} rS, & S \le S_c \\ rS_c, & S > S_c. \end{cases} \tag{2}$$

In model (1), we have included a disease-induced death rate (μ), which is important for many long-term epidemic diseases, such as plague, AIDS and tuberculosis where population demography can overlap with the effect of the disease. The function $H(S)$ defines the following vaccination strategy: the vaccination rate is proportional to the number of susceptible individuals if the number of susceptible individuals is below the critical value S_c; otherwise, we take a constant vaccination strategy $k = rS_c$. All other parameters are positive constants, where Λ is recruitment rate, β denotes the basic transmission rate, μ represents natural death and ϵ represents the disease-induced death rate. We assume the maximum vaccination is less than the recruitment rate; i.e., $\Lambda > rS_c$. Model (1)–(2) represents a dynamical system subject to a threshold policy, which is a simple case of variable structure control in the literature.

Remarks 1. The function (2) describes the vaccination policy more accurately, from a biological perspective, when the medical resource is limited, which is the advantage of this type of vaccination function. As a result, model (1)–(2) is no longer smooth mathematically. A particular equilibrium called a generalized equilibrium is possible for the resulting non-smooth model, which is difficult to analyze, since the classical approach using the Jacobian for smooth vector fields cannot be applied.

2. To illustrate the main idea, we propose and analyze a simple SIV model without considering the amount of the medical resources as a separated compartment. Indeed, the variation of medical resources has important impacts on the process of disease control. An SIVM model of four dimension can be constructed by considering the resource class, which we will consider in future work.

Since the vaccination class does not affect the dynamics of the first two equations, we investigate the following equations:

$$\begin{aligned}\frac{\mathrm{d}S}{\mathrm{d}t} &= \Lambda - \beta SI^2 - \mu S - H(S),\\ \frac{\mathrm{d}I}{\mathrm{d}t} &= \beta SI^2 - \mu I - \epsilon I.\end{aligned} \tag{3}$$

Let $N = S + I + V$. We get

$$\frac{\mathrm{d}N}{\mathrm{d}t} = \Lambda - \mu N - \epsilon I.$$

It follows that

$$\Omega = \left\{(S, I) \in R_+^2 : 0 < S + I < \frac{\Lambda}{\mu}\right\}$$

is an attraction region of model (1)–(2). In fact, it follows from model (1) that

$$\begin{aligned}\left.\frac{\mathrm{d}S}{\mathrm{d}t}\right|_{S=0} &= \Lambda > 0, \quad \left.\frac{\mathrm{d}I}{\mathrm{d}t}\right|_{I=0} = 0, \quad \left.\frac{\mathrm{d}(S+I)}{\mathrm{d}t}\right|_{S+I=\Lambda/\mu} \\ &= \Lambda - \mu(S+I) - \epsilon I - H(S) < 0,\end{aligned}$$

which implies all solutions will enter into the region Ω and remain in it thereafter. Hence, Ω is an attraction region of model (1)–(2). Denote the switching boundary as

$$\Sigma = \left\{(S, I) \in R_+^2 : S = S_c\right\},$$

which splits R_+^2 into two parts; i.e.,

$$G_1 = \left\{(S, I) \in R_+^2 : S \le S_c\right\}, \qquad G_2 = \left\{(S, I) \in R_+^2 : S > S_c\right\}.$$

The subsystem defined on the subregion G_i $(i = 1, 2)$ is called system S_{G_i}. Denote

$$\begin{aligned}F_{G_1}(S, I) &= \left(\Lambda - \beta SI^2 - (\mu + r)S, \beta SI^2 - (\mu + \epsilon)I\right)^{\mathrm{T}} \doteq (F_{11}(S, I), F_{12}(S, I))^{\mathrm{T}} \\ F_{G_2}(S, I) &= \left(\Lambda - \beta SI^2 - \mu S - k, \beta SI^2 - (\mu + \epsilon)I\right)^{\mathrm{T}} \doteq (F_{21}(S, I), F_{22}(S, I))^{\mathrm{T}}.\end{aligned}$$

Thus, subsystem S_{G_i} $(i = 1, 2)$ is defined by F_{G_i} and system (3) can be written as

$$\frac{\mathrm{d}X}{\mathrm{d}t} = \begin{cases} F_{G_1}(S, I), & S \le S_c \\ F_{G_2}(S, I), & S > S_c, \end{cases}$$

with $X = (S, I)^{\mathrm{T}}$.

Note that the vector field defined by system (3) is continuous. We further claim that it is locally Lipschitz continuous. In fact, since $F_{G_i}(X)$ $(i = 1, 2)$ is continuous in G_i, it is sufficient to show that, for any closed rectangle $M \subset R_+^2$ centered at (S_c, I), there is a constant $L(M) > 0$ such that

$$\left|F_{G_1}(X_1) - F_{G_2}(X_2)\right| \le L|X_1 - X_2|$$

for $X_i \in M \cap G_i, i = 1, 2$. Denote $X_i = (S_i, I_i)$, $\tilde{f}_1 = \Lambda - \beta SI^2 - \mu S$ and $\tilde{f}_2 = \beta SI^2 - (\mu + \epsilon)I$. We then have

$$\begin{aligned}\left|F_{G_1}(X_1) - F_{G_2}(X_2)\right|^2 &= \left(\tilde{f}_1(X_1) - \tilde{f}_1(X_2) + r(S_1 - S_c)\right)^2 + \left(\tilde{f}_2(X_1) - \tilde{f}_2(X_2)\right)^2 \\ &\leq \left(\left|\tilde{f}_1(X_1) - \tilde{f}_1(X_2)\right| + r(S_2 - S_1)\right)^2 + \left(\tilde{f}_2(X_1) - \tilde{f}_2(X_2)\right)^2.\end{aligned}$$

Since $\tilde{f}_i(X)$ $(i = 1, 2)$ is smooth in M, there are constants $L_i > 0$ $(i = 1, 2)$ such that

$$\left|\tilde{f}_i(X_1) - \tilde{f}_i(X_2)\right|^2 \leq L_i |X_1 - X_2|^2.$$

Thus,

$$\begin{aligned}\left|F_{G_1}(X_1) - F_{G_2}(X_2)\right|^2 &\leq 2\left(L_1 + r^2\right)\left[|X_1 - X_2|^2 + (S_2 - S_1)^2\right] + L_2|X_1 - X_2|^2 \\ &\leq \left(2L_1 + 2r^2 + L_2\right)|X_1 - X_2|^2.\end{aligned}$$

Letting $L^2 = 2L_1 + 2r^2 + L_2$, it follows that

$$\left|F_{G_1}(X_1) - F_{G_2}(X_2)\right| \leq L|X_1 - X_2|.$$

Therefore, system (3) is locally Lipschitz continuous in R_+^2, and so the trajectory of (3) initiating from any point in R_+^2 exists and is unique.

It is worth noting that the equilibria of system (3) consist of all the equilibria of the two subsystems. Any equilibrium of system (3) could lie in the subregion where the corresponding subsystem is defined, the opposite one or on the switching boundary. The equilibrium in the first case can be the attractor, while the other two cases are special for non-smooth systems. Indeed, if the second case holds, the equilibrium is virtual and cannot act as the attractor of system (3). If the third case occurs, the equilibrium is the attractor of system (3) provided it is stable. To clearly address the dynamics of system (3), we list the definitions of the variable equilibria as follows. For convenience, we denote $\sigma(S) = S - S_c$.

Definition 1 Let $X^* = (S^*, I^*)^T$ be such that $F_{G_i}(X^*) = 0$ $(i = 1, 2)$. Then X^* is called a *real equilibrium* of system (3) if it belongs to G_i and a *virtual equilibrium* if it belongs to G_j, $j \neq i$. Both the real equilibrium and the virtual equilibrium are called *regular equilibria*.

Definition 2 A point $X^* \in \Sigma$ is called a *generalized singular point* of model (3) if $F_{G_1}\sigma(X^*)F_{G_2}\sigma(X^*) \leq 0$, where $F_{G_i}\sigma(X^*) = F_{G_i}(Z) \cdot \text{grad}\sigma(X^*)$ $(i = 1, 2)$ represents the Lie derivative of σ with respect of the vector field F_{G_i} at the point X^*. The generalized singular point is also called an *irregular singular point*.

If the trajectories near a generalized singular point turn around it, then it is called a *pseudo-focus point*. It behaves similarly to the focus points of a smooth system

and consists of four possible types: focus-focus type (FF), focus-parabolic type (FP), parabolic-focus type (PF) and parabolic-parabolic type (PP). We only concentrate on the pseudo-foci of FF type of system (3), which will be used in the rest of this paper. A pseudo-focus $X^* = (S_c, I^*)$ of FF type of system (3) refers to the one that is a focus for both systems S_{G_1} and S_{G_2}. Furthermore, a pseudo-focus of FF type is said to be elementary for (3) if it is elementary for both systems S_{G_1} and S_{G_2}. The detailed descriptions of other types of pseudo-foci can be found in Coll et al. (2001), Han and Zhang (2010).

3 Dynamics of the Subsystems

We concentrate on the dynamics of subsystems in this section. For subsystem S_{G_1}, we easily get the unique disease-free equilibrium $E_{10}(\Lambda/(\mu+r), 0) \doteq (S_{10}, 0)$, which always exists. The endemic equilibrium satisfies

$$\begin{cases} \beta(\mu+\epsilon)I^2 - \beta\Lambda I + (\mu+\epsilon)(\mu+r) = 0 \\ S = \dfrac{\mu+\epsilon}{\beta I}. \end{cases} \tag{4}$$

Denote

$$R_{c1} = \frac{\beta\Lambda^2}{4(\mu+r)(\mu+\epsilon)^2}.$$

It follows that:

- If $R_{c1} < 1$, there is no solution for (4) and so no endemic equilibrium exists for system (3).
- If $R_{c1} > 1$, two endemic equilibria $E_{11} = (S_{11}, I_{11})$ and $E_{12} = (S_{12}, I_{12})$ coexist, where

$$S_{1j} = \frac{\mu+\epsilon}{\beta I_{1j}}, \quad j = 1, 2, \quad I_{11} = \frac{\beta\Lambda + \sqrt{\Delta_1}}{2\beta(\mu+\epsilon)}, \quad I_{12} = \frac{\beta\Lambda - \sqrt{\Delta_1}}{2\beta(\mu+\epsilon)},$$
$$\Delta_1 = \beta^2\Lambda^2 - 4\beta(\mu+\epsilon)^2(\mu+r).$$

- If $R_{c1} = 1$, only one endemic equilibrium $E_1 = (S_1, I_1)$ exists, where

$$I_1 = \frac{\Lambda}{2(\mu+\epsilon)}, \quad S_1 = \frac{2(\mu+\epsilon)^2}{\beta\Lambda}.$$

To investigate the stability of the equilibria, we first calculate the Jacobian matrix of subsystem S_{G_1}:

$$J_1(E(S, I)) = \begin{pmatrix} -(\mu+r) - \beta I^2 & -2\beta SI \\ \beta I^2 & 2\beta SI - (\mu+\epsilon) \end{pmatrix}.$$

By checking the Jacobian matrix at E_{10}, we know the disease-free equilibrium is always locally asymptotically stable. Now let us examine the stability of the endemic equilibria. Calculating the determinant of J_1 at E_{12} yields

$$\det(J_1(E_{12})) = (\mu+\epsilon)[\beta I_{12}^2 - (\mu+r)] < 0,$$

which indicates E_{12} is a saddle and so it is always unstable. Since

$$\det(J_1(E_{11})) = (\mu+\epsilon)[\beta I_{11}^2 - (\mu+r)] > 0,$$

we easily know that E_{11} is an anti-saddle, whose stability is determined by the sign of

$$\text{tr}\,(J_1(E_{11})) = (\epsilon - r) - \beta I_{11}^2.$$

If $\epsilon \le r$, we easily get tr $(J_1(E_{11})) < 0$. Otherwise, it is necessary to evaluate

$$\text{sgn}\big((\epsilon - r) - \beta I_{11}^2\big) = \text{sgn}\Big(2(\mu+\epsilon)^3 - \beta\Lambda^2 - \Lambda\sqrt{\beta^2\Lambda^2 - 4\beta(\mu+\epsilon)^2(\mu+r)}\Big).$$

Direct calculation gives

$$\begin{aligned}
\beta\Lambda^2 \ge 2(\mu+\epsilon)^3 &\Longleftrightarrow R_{c1} \ge \frac{\mu+\epsilon}{2(\mu+r)},\\
\beta\Lambda^2(\epsilon - r) > (\mu+\epsilon)^4 &\Longleftrightarrow R_{c1} > \frac{(\mu+\epsilon)^2}{4(\mu+r)(\epsilon-r)},\\
\epsilon > \mu + 2r &\Longrightarrow \frac{\mu+\epsilon}{2(\mu+r)} > \frac{(\mu+\epsilon)^2}{4(\mu+r)(\epsilon-r)} > 1.
\end{aligned}$$

Then we get tr $(J_1(E_{11})) < 0$ if one of the following inequalities hold.

- $R_{c1} \ge \dfrac{\mu+\epsilon}{2(\mu+r)}$ or
- $\dfrac{(\mu+\epsilon)^2}{4(\mu+r)(\epsilon-r)} < R_{c1} < \dfrac{\mu+\epsilon}{2(\mu+r)}$, $\epsilon > \mu + 2r$.

We also know tr $(J_1(E_{11})) > 0$ if

$$R_{c1} < \frac{(\mu+\epsilon)^2}{4(\mu+r)(\epsilon-r)}.$$

Further discussion yields that E_{11} is a focus for $\eta_1 < 0$ and a node for $\eta_1 > 0$, where

$$\eta_1 = (\beta I_{11}^2 + r - \epsilon) - 4(\mu+\epsilon)[\beta I_{11}^2 - (\mu+r)].$$

The endemic equilibrium E_1 is a non-hyperbolic saddle node.

Concluding the above discussions yields the following result.

Theorem 3.1 *(i) The disease-free equilibrium E_{10} always exists for subsystem S_{G_1}. Two endemic equilibria E_{11} and E_{12} coexist when $R_{c1} > 1$, and they coincide with each other and are replaced by E_1 when $R_{c1} = 1$. There is no endemic equilibrium for $R_{c1} < 0$.*

(ii) E_{10} is always locally asymptotically stable, while E_{12} is unstable provided it is well defined. E_{11} is locally asymptotically stable if one of the following inequalities hold:

$$(H_1)\ R_{c1} > 1,\ \epsilon \le \mu + 2r;\ or$$
$$(H_2)\ R_{c1} > \frac{(\mu+\epsilon)^2}{4(\mu+r)(\epsilon-r)},\ \epsilon > \mu + 2r.$$

(iii) E_{11} is unstable if

$$(H_3)\ R_{c1} < \frac{(\mu+\epsilon)^2}{4(\mu+r)(\epsilon-r)},\ \epsilon > \mu + 2r.$$

By Theorem 3.1, whether the endemic equilibrium exists depends on the critical value R_{c1}. Two or one or no endemic equilibria exist for subsystem S_{G_1} if $R_{c1} > 1$ or $R_{c1} = 1$ or $R_{c1} < 1$. The nonexistence of endemic equilibria results in the global stability of the disease-free equilibrium.

Theorem 3.2 *The disease-free equilibrium E_{10} is globally asymptotically stable for subsystem S_{G_1} if $R_{c1} < 1$.*

Proof According to Sect. 2, Ω is an attraction region of subsystem S_{G_1}. Since there is no endemic equilibrium for subsystem S_{G_1} when $R_{c1} < 1$, the Poincaré–Bendixon Theorem leads to the nonexistence of periodic orbits in Ω for subsystem S_{G_1}. Note that Ω is bounded and positively invariant, while E_{10} is the unique equilibrium of subsystem S_{G_1}, so the local stability of E_{10} implies that E_{10} is the ω-limit set of any solution initiating inside Ω. Hence, the disease-free equilibrium E_{10} is globally asymptotically stable. This completes the proof. □

It follows from Theorem 3.2 that when $R_{c1} < 1$, the disease die out. However, when $R_{c1} > 1$, the endemic equilibria coexist with the disease-free equilibrium.

For subsystem S_{G_2}, the disease-free equilibrium $E_{20}((\Lambda - k)/\mu, 0) \doteq (S_{20}, 0)$ always exists. Define the critical value

$$R_{c2} = \frac{\beta(\Lambda - k)^2}{4\mu(\mu+\epsilon)^2}.$$

It follows that when $R_{c2} > 1$, two endemic equilibria $E_{21}(S_{21}, I_{21})$, $E_{22}(S_{22}, I_{22})$ exist, where

$$S_{2j} = \frac{\mu+\epsilon}{\beta I_{2j}},\quad j = 1, 2,\quad I_{21} = \frac{\beta(\Lambda-k) + \sqrt{\Delta_2}}{2\beta(\mu+\epsilon)},\quad I_{22} = \frac{\beta(\Lambda-k) - \sqrt{\Delta_2}}{2\beta(\mu+\epsilon)},$$
$$\Delta_2 = \beta^2(\Lambda-k)^2 - 4\beta\mu(\mu+\epsilon)^2.$$

When $R_{c2} = 1$, one endemic equilibrium $E_2(S_2, I_2)$ exists, where

$$I_2 = \frac{\Lambda - k}{2(\mu + \epsilon)}, \quad S_2 = \frac{2(\mu + \epsilon)^2}{\beta(\Lambda - k)}.$$

When $R_{c2} < 1$, no endemic equilibrium exists for subsystem S_{G_2}.

Next we examine the stability of equilibria for subsystem S_{G_2}. To this end, we first compute the Jacobian matrix of S_{G_2} as follows

$$J_2(E(S, I)) = \begin{pmatrix} -\mu - \beta I^2 & -2\beta SI \\ \beta I^2 & 2\beta SI - (\mu + \epsilon) \end{pmatrix}.$$

We easily get that the disease-free equilibrium E_{20} is always locally asymptotically stable, while the endemic equilibrium E_{22} is unstable. Evaluating the Jacobian matrix J_2 at E_{21} yields

$$\det(J_2(E_21)) = (\mu + \epsilon)(\beta I_{21}^2 - \mu) > 0, \quad \text{tr}\,(J_2(E_{21})) = \epsilon - \beta I_{21}^2,$$
$$\text{sgn}\big(\epsilon - \beta I_{21}^2\big) = \text{sgn}\Big(2(\mu + \epsilon)^3 - \beta(\Lambda - k)^2 - (\Lambda - k)\sqrt{\beta^2(\Lambda - k)^2 - 4\beta\mu(\mu + \epsilon)^2}\Big).$$

Since

$$\epsilon > \mu \Longrightarrow \frac{\mu + \epsilon}{2\mu} > \frac{(\mu + \epsilon)^2}{4\mu\epsilon} > 1,$$

we get tr $(J_2(E_{21})) < 0$ if one of the following inequalities hold.

- $R_{c2} \geq \dfrac{\mu + \epsilon}{2\mu}$; or
- $\dfrac{(\mu + \epsilon)^2}{4\mu\epsilon} < R_{c2} < \dfrac{\mu + \epsilon}{2\mu}$, $\epsilon > \mu$.

Furthermore, tr $(J_2(E_{21})) > 0$ if

$$R_{c2} < \frac{(\mu + \epsilon)^2}{4\mu\epsilon}, \ \epsilon > \mu.$$

Summarizing the above discussions gives the following conclusion.

Theorem 3.3 *(i) The disease-free equilibrium E_{20} exists for subsystem S_{G_2}. There exist two endemic equilibria E_{21} and E_{22} for $R_{c2} > 1$ and no endemic equilibrium for $R_{c2} < 1$. If $R_{c2} = 1$, the two endemic equilibria collide and are replaced by the equilibrium E_2.*

(ii) The disease-free equilibrium E_{20} is always locally asymptotically stable, while the endemic equilibrium E_{22} is unstable when it exists. The endemic equilibrium E_{21} is locally asymptotically stable if one of the following inequalities hold.

(H_4) $R_{c2} > 1$, $\epsilon \leq \mu$; or

(H_5) $R_{c2} > \frac{(\mu+\epsilon)^2}{4\mu\epsilon}$, $\epsilon > \mu$.

(iii) The endemic equilibrium E_{21} is unstable if

(H_6) $R_{c2} < \frac{(\mu+\epsilon)^2}{4\mu\epsilon}$, $\epsilon > \mu$.

Since the disease-free equilibrium E_{20} is globally asymptotically stable for $R_{c2} < 1$, the disease can theoretically be eradicated from the population. For $R_{c2} > 1$, the bistability phenomenon occurs due to the coexistence of the disease-free equilibrium E_{20} and the stable endemic equilibrium E_{21}. In such a scenario, the disease can be eradicated or become endemic, depending on the initial conditions.

4 Nature of Equilibria for Non-smooth System

The equilibria of the proposed non-smooth system (3) consist of all regular equilibria (i.e., equilibria of the subsystem $S_{G_j}(j = 1, 2)$) as well as the generalized equilibria. Every regular equilibrium may be real or virtual, and only the real ones can act as an attractor of system (3). We will initially examine the nature of the regular equilibria in the following.

Since

$$S_c > S_{10} \Longleftrightarrow S_c > \frac{\Lambda}{\mu + r} \Longleftrightarrow S_c > \frac{\Lambda - rS_c}{\mu} \Longleftrightarrow S_c > S_{20},$$

the disease-free equilibrium E_{10} is real (denoted by E_{10}^r) if and only if the disease-free equilibrium E_{20} is virtual (denoted by E_{20}^v). When $S_c = S_{10}$, we get that $S_c = S_{20}$, and so there exists a generalized disease-free equilibrium $E_0(\Lambda/(\mu + r), 0) \doteq (S_0, 0)$.

The endemic equilibrium E_{21} is real if and only if $S_c < S_{21}$, which is equivalent to

$$S_c\sqrt{\beta^2(\Lambda - rS_c)^2 - 4\beta\mu(\mu + \epsilon)^2} < 2(\mu + \epsilon)^2 - \beta(\Lambda - rS_c)S_c. \tag{5}$$

We need the following inequalities:

$$\begin{cases} 2(\mu + \epsilon)^2 - \beta S_c(\Lambda - rS_c) > 0 \\ \beta(\mu + r)S_c^2 - \beta\Lambda S_c + (\mu + \epsilon)^2 > 0 \end{cases} \tag{6}$$

to ensure (5). The first inequality of (6) can be rewritten as

$$\beta r S_c^2 - \beta\Lambda S_c + 2(\mu + \epsilon) > 0, \tag{7}$$

which is true if one of the following inequalities hold.

(a_1) $1 < R_{c1} < \dfrac{2r}{\mu + r}$;

(a_2) $R_{c1} > \dfrac{2r}{\mu + r}$, $S_c < \dfrac{\beta\Lambda - \sqrt{\beta^2\Lambda^2 - 8\beta r(\mu + \epsilon)^2}}{2\beta r} \equiv \xi_1^-$;

$$(a_3)\ \ R_{c1} > \frac{2r}{\mu + r}, S_c > \frac{\beta\Lambda + \sqrt{\beta^2\Lambda^2 - 8\beta r(\mu+\epsilon)^2}}{2\beta r} \equiv \xi_1^+.$$

The second inequality of (6) holds if $S_c < S_{11}$ or $S_c > S_{12}$, where $S_{1j}(j = 1, 2)$ is as defined in Sect. 3. As a result, the endemic equilibrium E_{21} is real if (a_i) $(i = 1, 2, 3)$ and $S_c < S_{11}$ or $S_c > S_{12}$ hold.

The endemic equilibrium E_{22} is real if and only if $S_c < S_{22}$, which is equivalent to

$$S_c\sqrt{\beta^2(\Lambda - rS_c)^2 - 4\beta\mu(\mu+\epsilon)^2} > \beta(\Lambda - rS_c)S_c - 2(\mu+\epsilon)^2. \tag{8}$$

The inequality (8) holds if

$$\beta(\Lambda - rS_c)S_c - 2(\mu+\epsilon)^2 \le 0 \tag{9}$$

or

$$\begin{cases} \beta S_c(\Lambda - rS_c) - 2(\mu+\epsilon)^2 > 0 \\ \beta(\mu+r)S_c^2 - \beta\Lambda S_c + (\mu+\epsilon)^2 < 0 \end{cases} \tag{10}$$

are true. The inequality (9) is true if the condition (a_1) or (a_2) or (a_3) is true. Solving (10) gives

$$R_{c1} \ge \frac{2r}{\mu + r}, \quad \max\left\{\xi_1^-, S_{11}\right\} < S_c < \min\left\{\xi_1^+, S_{12}\right\}. \tag{11}$$

Hence, the endemic equilibrium E_{22} is real if (a_i) $(i = 1, 2, 3)$ and (11) hold.

Similarly, we can derive conditions to ensure the equilibria E_{21} and E_{22} are virtual by reversing the inequalities (5) and (8), respectively; we omit them here.

The endemic equilibrium E_{11} is real when $S_c > S_{11}$, and the equilibrium E_{12} is real when $S_c > S_{12}$. Otherwise, both E_{11} and E_{12} are virtual equilibria if the last two inequalities are reversed.

We easily get that

$$S_{11} < \xi_1^- < \xi_1^+, \quad S_{12} > \xi_1^-.$$

Denote $\xi_1 = \Lambda/(2r)$, and we can address the nature of all the endemic equilibria E_{sl} $(s, l = 1, 2)$ in detail after some algebra. For clarity, we list the result in Table 1. It follows from Table 1 that one endemic equilibrium may be real or virtual for different sets of parameters. We always denote the real (or virtual) equilibrium $E_{ls}(l, s = 1, 2)$ as E_{ls}^r (or E_{ls}^v) in the following. More importantly, $S_c = S_{11}$ leads to $S_c = S_{21}$, so a generalized equilibrium $E_1(S_1, I_1)$ exists for (3) with

$$I_1 = \frac{\beta\Lambda + \sqrt{\beta^2\Lambda^2 - 4\beta(\mu+\epsilon)^2(\mu+r)}}{2\beta(\mu+\epsilon)}, S_1 = \frac{\beta\Lambda - \sqrt{\beta^2\Lambda^2 - 4\beta(\mu+\epsilon)^2(\mu+r)}}{2\beta(\mu+r)}.$$

The same method results in another generalized equilibrium $E_2(S_2, I_2)$ with

$$I_2 = \frac{\beta\Lambda - \sqrt{\beta^2\Lambda^2 - 4\beta(\mu+\epsilon)^2(\mu+r)}}{2\beta(\mu+\epsilon)}, S_2 = \frac{\beta\Lambda + \sqrt{\beta^2\Lambda^2 - 4\beta(\mu+\epsilon)^2(\mu+r)}}{2\beta(\mu+r)}.$$

Table 1 Nature of the endemic equilibria

Threshold value		E_{11}	E_{12}	E_{21}	E_{22}
$1 < R_{c1} < \frac{2r}{\mu+r}$	(Q_{11}) $S_c < S_{11}$	Virtual	Virtual	Real	Real
	(Q_{12}) $S_{11} < S_c < S_{12}$	Real	Virtual	Virtual	Real
	(Q_{13}) $S_c > S_{12}$	Real	Real	Real	Real
$R_{c1} > \frac{2r}{\mu+r}$	(Q_{21}) $\xi_1^- < S_c < \min\left\{\xi_1^+, S_{12}\right\}$	Real	Virtual	Virtual	Real
	(Q_{22}) $S_c < S_{11}$	Virtual	Virtual	Real	Real
	(Q_{23})§$_c > \max\left\{\xi_1^+, S_{12}\right\}$	Real	Real	Real	Real
	(Q_{24}) $S_{12} < S_c < \xi_1^+, S_{12} < \xi_1^+$	Real	Real	Virtual	Virtual
	(Q_{25}) $S_{11} < S_c < \xi_1^-$	Real	Virtual	Virtual	Real
	(Q_{26}) $\xi_1^+ \leq S_c < S_{12}, S_{12} > \xi_1$	Real	Virtual	Virtual	Real
	(Q_{27}) $S_c = \xi_1^-$	Real	Virtual	Virtual	Real
	(Q_{28}) $S_c = \xi_1^+, S_{12} < \xi_1^+$	Real	Real	Virtual	Real
$R_{c1} = \frac{2r}{\mu+r}$	(Q_{31}) $S_c < S_{11}$	Virtual	Virtual	Real	Real
	(Q_{32}) $S_c > S_{12}, S_c \neq \xi_1$	Real	Real	Real	Real
	(Q_{33}) $S_{11} < S_c < S_{12}$	Real	Virtual	Virtual	Real
	(Q_{34}) $S_c = \xi_1, S_{12} < \xi_1$	Real	Real	Virtual	Real

5 Dynamics of Non-smooth System (3)

In this section, we focus on the dynamic behavior of the non-smooth system (3). There are three possible disease-free equilibria and six endemic equilibria. Whether the endemic equilibrium exists depends on whether the threshold value R_{cj} $(j = 1, 2)$ is greater or less than 1. So we consider three cases in the following:

(A): $\max\{R_{c1}, R_{c2}\} < 1$; (B): $\max\{R_{c1}, R_{c2}\} > 1$, $\min\{R_{c1}, R_{c2}\} < 1$;
(C): $\min\{R_{c1}, R_{c2}\} > 1$.

Case (A) $\max\{R_{c1}, R_{c2}\} < 1$.

There are two disease-free equilibria (E_{10}, E_{20}) and no endemic equilibrium for system (3) in this scenario. Moreover, a possible generalized disease-free equilibrium E_0 exists. By the above discussion, the disease-free equilibrium E_{10} is real and E_{20} is virtual when $S_c > S_{10}$. For $S_c > S_{10}$, the equilibrium E_{10} is virtual, and E_{20} is real. The generalized equilibrium E_0 exists for system (3) if $S_c = S_{10}$. Moreover, we know that the regular disease-free equilibrium $E_{j0}(j = 1, 2)$ is locally asymptotically stable if it is real. For the local stability of the generalized disease-free equilibrium E_0, the classic approach of evaluating the Jacobian matrix for a C^1 vector field at this point is not available since the vector field defined by (3) is not smooth. Then we adopt the generalized Jacobian proposed by Clarke (Leine 2006; Clarke et al. 1998; Zhang and Wang 2012).

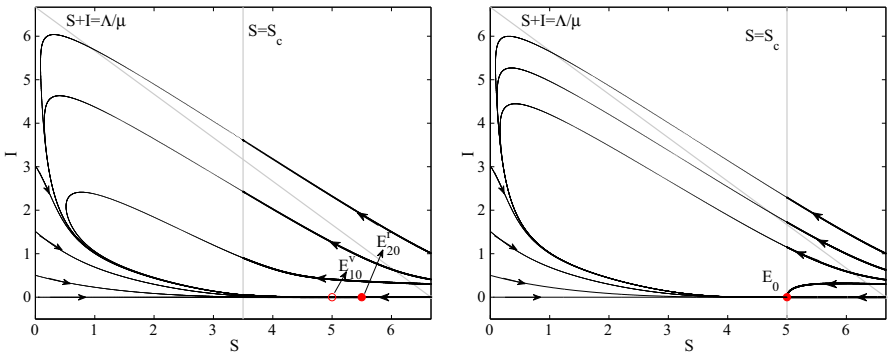

Fig. 1 Phase plane of the non-smooth epidemic model, showing the extinction of the disease with distinct attractors for different parameter sets. The parameter values are $\Lambda = 2$; $\beta = 0.8$; $\mu = 0.3$; $r = 0.1$; $\epsilon = 1.2$, $S_c = 3.5$ (**a**) and $S_c = 5$ (**b**)

Here, the generalized Jacobian matrix of system (3) takes the form

$$((1-p)J_1 + pJ_2)(X(S,I)) = \begin{pmatrix} -(\mu + rp) - \beta I^2 & -2\beta SI \\ \beta I^2 & 2\beta SI - (\mu + \epsilon) \end{pmatrix},$$

where $p \in [0, 1]$. Evaluating the above matrix at the generalized disease-free equilibrium E_0 gives

$$((1-p)J_1 + pJ_2)(E_0(S_0, 0)) = \begin{pmatrix} -(\mu + rp) & 0 \\ 0 & -(\mu + \epsilon) \end{pmatrix},$$

which suggests the local stability of E_0.

Summarizing the above discussion yields the following result.

Theorem 5.1 *The disease-free equilibrium E_{10} or E_{20} or E_0 is the attractor of system* (3) *when* $\max\{R_{c1}, R_{c2}\} < 1$.

In particular, the regular equilibrium E_{10} (or E_{20}) is locally asymptotically stable for $S_c > S_{10}$ (or $S_c < S_{10}$), as shown in Fig. 1a; the generalized disease-free equilibrium E_0 is locally asymptotically stable if $S_c = S_{10}$, as shown in Fig. 1b.

In Fig. 1, the circular points represent the disease-free equilibria, in which the solid ones are stable while the hollow ones are unstable (since they are virtual). The thick (or thin) lines are the trajectories of the subsystem S_{G_2} (or S_{G_1}). Subplot (a) shows the stability of the regular equilibrium E^r_{20}, and subplot (b) describes the stability of the generalized equilibrium E_0.

Case (B) $\max\{R_{c1}, R_{c2}\} > 1, \ \min\{R_{c1}, R_{c2}\} < 1$.

In this section, we will address the dynamic behavior of system (3) for the existence of endemic equilibria. To this end, we initially examine the nature of the endemic equilibria, which depends on the relationship between the threshold level and the abscissa of the steady state.

Direct calculation yields

$$\begin{aligned} R_{c1} - R_{c2} &= \frac{\beta}{4\mu(\mu+r)(\mu+\epsilon)^2}\big[-(\mu+r)rS_c^2 + 2\Lambda r(\mu+r)S_c - r\Lambda^2\big] \\ &= \frac{\beta}{4\mu(\mu+r)(\mu+\epsilon)^2} f(S_c), \end{aligned}$$

where

$$f(S_c) = -(\mu+r)rS_c^2 + 2\Lambda r(\mu+r)S_c - r\Lambda^2.$$

Solving $f(S_c) = 0$ gives

$$S_{c1}^- = \frac{\Lambda(\mu+r) - \Lambda\sqrt{\mu(\mu+r)}}{r(\mu+r)},\ S_{c1}^+ = \frac{\Lambda(\mu+r) + \Lambda\sqrt{\mu(\mu+r)}}{r(\mu+r)}.$$

It follows that

$$\begin{aligned} S_{c1}^- < S_c < S_{c1}^+ &\Rightarrow f(S_c) > 0 \text{ (i.e., } R_{c1} > R_{c2}) \\ S_c > S_{c1}^+ \text{ (or } S_c < S_{c1}^-) &\Rightarrow f(S_c) < 0 \text{ (i.e., } R_{c1} < R_{c2}) \\ S_c = S_{c1}^+ \text{ (or } S_c = S_{c1}^-) &\Rightarrow f(S_c) = 0 \text{ (i.e., } R_{c1} = R_{c2}). \end{aligned}$$

Since

$$R_{c2} < 1 \Longleftrightarrow \beta r S_c^2 - 2\beta r\Lambda S_c + [\beta\Lambda^2 - 4\mu(\mu+\epsilon)^2] < 0,$$

one gets

$$\frac{\beta\Lambda - 2(\mu+\epsilon)\sqrt{\beta\mu}}{\beta r} < S_c < \frac{\beta\Lambda + 2(\mu+\epsilon)\sqrt{\beta\mu}}{\beta r}.$$

Denote

$$S_{c2}^- = \frac{\beta\Lambda - 2(\mu+\epsilon)\sqrt{\beta\mu}}{\beta r},\quad S_{c2}^+ = \frac{\beta\Lambda + 2(\mu+\epsilon)\sqrt{\beta\mu}}{\beta r},$$

and we have $R_{c2} < 1$ for $S_{c2}^- < S_c < S_{c2}^+$, while $R_{c2} > 1$ if and only if $S_c > S_{c2}^+$ or $S_c < S_{c2}^-$. Direct calculation gives

$$\begin{aligned} \operatorname{sgn}\big(S_{c1}^- - S_{c2}^-\big) &= \operatorname{sgn}\Big(2(\mu+r)(\mu+\epsilon)\sqrt{\beta\mu} - \beta\Lambda\sqrt{\mu(\mu+r)}\Big) \\ &= \operatorname{sgn}\big(4(\mu+r)(\mu+\epsilon)^2 - \beta\Lambda^2\big). \end{aligned}$$

It follows that $S_{c1}^- > S_{c2}^-$ for $R_{c1} < 1$ and $S_{c1}^- < S_{c2}^-$ for $R_{c1} > 1$. Similar discussion yields that $S_{c1}^+ < S_{c2}^+$ for $R_{c1} < 1$ and $S_{c1}^+ > S_{c2}^+$ for $R_{c1} > 1$. Thus, we have

$$S_{c1}^- < S_{c2}^- < S_{c2}^+ < S_{c1}^+$$

for $R_{c1} > 1$ and

$$S_{c2}^- < S_{c1}^- < S_{c1}^+ < S_{c2}^+$$

for $R_{c1} < 1$. Note that S_{cj}^- and S_{cj}^+ $(j = 1, 2)$ are thresholds that make sense for $S_{cj}^- < \Lambda/\mu$, $S_{cj}^+ < \Lambda/\mu$. We have the following results:

- $S_{c1}^- < \Lambda/\mu$ always holds;
- $S_{c1}^+ \leq \Lambda/\mu$ holds for $r > \mu$ and $\mu^3 + 2r\mu^2 \leq r^3$;
- $S_{c2}^- < \Lambda/\mu$ holds if $r \geq \mu$ or $r < \mu$ and $\beta\Lambda^2(\mu - r)^2 < 4\mu^3(\mu + \epsilon)^2$;
- $S_{c2}^+ < \Lambda/\mu$ holds if $r > \mu$ or $r < \mu$ and $\beta\Lambda^2(\mu - r)^2 > 4\mu^3(\mu + \epsilon)^2$.

Note that we assume $rS_c < \Lambda$, whereas

$$S_c > S_{c1}^+ \Longrightarrow \Lambda < rS_c,$$

which is out of our consideration. We consider the following two possibilities: (B_1) $R_{c1} > 1 > R_{c2}$; (B_2) $R_{c2} > 1 > R_{c1}$.

It is worth mentioning that the disease cannot be eradicated from the population in most cases, so the aim of disease control is to contain the case number below some critical level. Thus, the equilibrium level of infected individuals in steady state plays an important role in disease control.

(B_1) $R_{c1} > 1 > R_{c2}$.

By the above discussion, the threshold value S_c satisfies $S_{c2}^- < S_c < S_{c2}^+$ in this scenario. Three possible disease-free equilibria (E_{10}, E_{20}, E_0) and two endemic equilibria (E_{11}, E_{12}) exist for system (3). It is worth mentioning that only the real equilibrium can act as the attractor, so we first address the nature of the equilibria. We have

$$\begin{aligned} S_{c1}^- - S_{11} &= \frac{\Lambda(\beta\Lambda + M_1)\left[(\mu + r) - \sqrt{\mu(\mu + r)}\right] - 2r(\mu + r)(\mu + \epsilon)^2}{r(\mu + r)(\beta\Lambda + M_1)} \\ &\geq \frac{\Lambda M_1\left[(\mu + r) - \sqrt{\mu(\mu + r)}\right] + \beta\Lambda^2\left[d + \frac{r}{2} - \sqrt{\mu(\mu + r)}\right]}{r(\mu + r)(\beta\Lambda + M_1)} \\ &> 0, \end{aligned}$$

where

$$M_1 = \sqrt{\beta^2\Lambda^2 - 4\beta(\mu + r)(\mu + \epsilon)^2}.$$

It follows that $S_{c1}^- > S_{11}$ and so E_{11} is real in this scenario. We easily get that $S_{c2}^- - S_{12} > 0$, so $S_c > S_{12}$, which suggests the endemic equilibrium E_{12} is also real.

According to Sect. 3, the endemic equilibrium E_{11} is stable if (H_1) or (H_2) is true, while it is unstable if (H_3) holds. For the former case, one of the disease-free equilibria E_{10} or E_{20} or E_0 competes with the stable endemic equilibrium E_{11}, which triggers

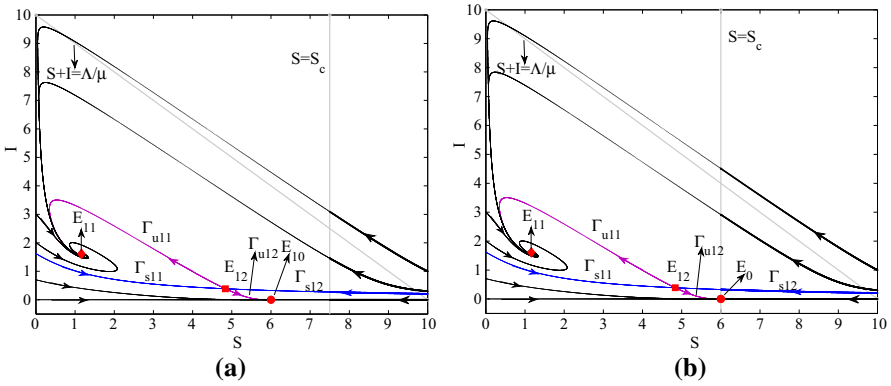

Fig. 2 Phase plane of the non-smooth model (3), showing the bistability of the system with distinct attractors for different parameter sets. The parameter values are $\Lambda = 3$; $\beta = 0.8$; $\mu = 0.3$; $r = 0.2$; $\epsilon = 1.2$, $S_c = 7.5$ (**a**), and $S_c = 6$ (**b**)

a bistability phenomenon. The two stable manifolds Γ_{s11} and Γ_{s12} of the saddle point E_{12} split the R^2_+ plane into two subregions, as shown in Fig. 2.

For simplicity, we denote the subregion above Γ_{s11} and Γ_{s12} as G_{e11} and the lower one as G_{e01}. Any trajectory initiating from the subregion G_{e11} ultimately tends to the endemic equilibrium E_{11}, while those initiating from the subregion G_{e01} reach one of the disease-free equilibria. This implies that G_{e11} is the endemic region while G_{e01} is the eradication region. This indicates the disease may die out or become endemic, depending on the initial infection, as shown in Fig. 2.

In Fig. 2, the diamond points and square points denote the endemic equilibria; the diamonds are anti-saddle points, while the squares are saddle points. The circular points represent the disease-free equilibria. Here we omit the virtual equilibria, and all the regular equilibria are real. The curves Γ_{s1j} and Γ_{u1j}($j = 1, 2$) are, respectively, the stable and unstable manifolds of the saddle point E_{12}.

If (H_3) is true, neither of the endemic equilibria is stable. If we further have $S_c > S_{10}$ or $S_c < S_{10}$ or $S_c = S_{10}$, the disease-free equilibrium E_{10} or E_{20} or E_0 acts as the attractor for system (3), as shown in Fig. 3. In Fig. 3, the endemic equilibrium E_{12} is a saddle with two stable manifolds Γ_{s11} and Γ_{s12}. The endemic equilibrium E_{11} is unstable, while the disease-free equilibrium E_{20} or E_0 is stable as shown in Fig. 3a, or b. So the case number approaches zero whenever the initial infection is not in the critical case (i.e., $(S_0, I_0) \in \Gamma_{s11} \cup \Gamma_{s12}$). This suggests that the disease can die out except for the critical cases, even though real endemic equilibria exist. It is interesting that $\max\{R_{c1}, R_{c2}\} < 1$ can lead to the eradication of the disease, but $\max\{R_{c1}, R_{c2}\} > 1$ may not trigger a disease outbreak. As the threshold value varies, the disease will be eradicated with different levels of susceptibles. In particular, the number of susceptible individuals tends to S_{10} for $S_c < S_{10}$ and S_{20} for $S_c > S_{10}$. For the critical level $S_c = S_0$, the number of susceptible individuals goes to S_0. This suggests that the disease can be eradicated and the final size of the susceptible population can be determined except for the critical case $(S_0, I_0) \in \Gamma_{s11} \cup \Gamma_{s12}$. The disease-free equilibrium E_{10} or E_{20} or E_0 is the attractor of system (3) in this case.

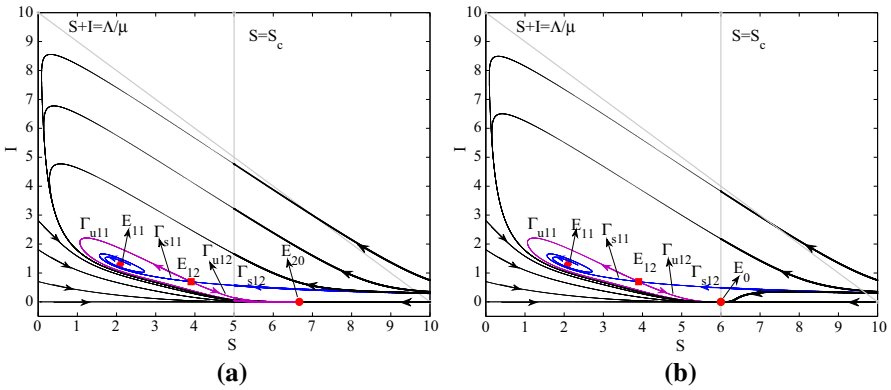

Fig. 3 Phase plane of the non-smooth model (3), showing disease extinction for most solutions. The parameter values are $\Lambda = 3; \beta = 0.55; \mu = 0.3; r = 0.2; \epsilon = 1.2, S_c = 5$ (**a**), and $S_c = 6$ (**b**)

In particular, the regular equilibrium E_{10} (or E_{20}) is locally asymptotically stable for $S_c > S_{10}$ (or $S_c < S_{10}$), as shown in Fig. 1a; the generalized disease-free equilibrium E_0 is locally asymptotically stable if $S_c = S_{10}$, as shown in Fig. 1b.

Let $X_0 = (S_0, I_0)$ be the initial point and E_{s0} be one of the disease-free equilibria. Thus, we can conclude the above discussion as follows.

Theorem 5.2 *If the inequalities* (H_1) *or* (H_2) *hold, then the solution of* (3) *will approach* E_{11} *for* $X_0 \in G_{e11}$, *while it will tend to* E_{s0} *for* $X_0 \in G_{e01}$. *If* (H_3) *holds, the solution of* (3) *ultimately goes to* E_{s0} *for* $X_0 \in R^2_+ \backslash \{\Gamma_{s11} \cup \Gamma_{s12}\}$.

(B_2) $R_{c2} > 1 > R_{c1}$.

Two endemic equilibria (E_{21}, E_{22}) and three possible disease-free equilibria coexist for system (3). According to the above discussion, we get that $S_c < S^-_{c2}$ in this scenario. Assume $S_{21} > S_c$, then we get

$$\beta\Lambda - \beta(r + 2\mu)S_c > \sqrt{\beta^2(\Lambda - rS_c)^2 - 4\beta\mu(\mu + \epsilon)^2}. \tag{12}$$

Direct calculation gives $S^-_{c2} < \Lambda/(r + 2\mu)$, so $\beta\Lambda - \beta(r + 2\mu)S_c > 0$. Inequality (12) is equivalent to

$$\beta(r + \mu)S_c^2 - \beta\Lambda S_c + (\mu + \epsilon)^2 > 0,$$

which is always true due to $R_{c1} < 1$. This implies that $S_{21} > S_c$ always holds and so $E_{2j}(j = 1, 2)$ is true in this case. Performing a similar analysis to case (B_1) gives that one of the disease-free equilibria E_{10}, E_{20}, E_0 competes with the endemic equilibrium E_{21}, and a bistable phenomenon occurs if the inequalities (H_4) or (H_5) are true. If the inequalities (H_6) are true, only the disease-free equilibrium (E_{10} or E_{20} or E_0) is stable. For the former case, the disease can die out or become endemic depending on the initial level of infection. If it becomes endemic, the case number ultimately

stabilizes at the level E_{21}. For the latter case, the disease can be eradicated except for the critical case.

Similarly, denote the stable manifolds of E_{22} as Γ_{s21} and Γ_{s22}. Let G_{e12} be the subregion above Γ_{s21} and Γ_{s23}, and let G_{e02} be the subregion below Γ_{s21} and Γ_{s22}. We get the following conclusion.

Theorem 5.3 *If the inequalities* (H_4) *or* (H_5) *hold, then the solution of* (3) *will approach* E_{21} *for* $X_0 \in G_{e12}$*, while it will tend to* E_{s0} *for* $X_0 \in G_{e02}$*. If the inequality* (H_6) *holds, the solution of* (3) *ultimately goes to* E_{s0} *for* $X_0 \in R^2_+ \backslash \{\Gamma_{s21} \cup \Gamma_{s22}\}$.

Case (C) $\min\{R_{c1}, R_{c2}\} > 1$.

We easily get that $S_c < S^-_{c2}$ or $S_c > S^+_{c2}$ holds in this case. Three possible disease-free equilibria and six possible endemic equilibria exist for system (3). According to the relationship between R_{c1} and R_{c2}, there are three possibilities to consider: (C_1) $R_{c1} > R_{c2} > 1$, (C_2) $R_{c2} > R_{c1} > 1$, (C_3) $R_{c1} = R_{c2} > 1$. As the threshold value S_c varies, the nature of the regular equilibria varies and the generalized equilibrium may exist or disappear.

(C_1) $R_{c1} > R_{c2} > 1$.

By the above discussion, we get that $S^-_{c1} < S_c < S^-_{c2}$ or $S^+_{c2} < S_c < S^+_{c1}$ in this scenario. The endemic equilibrium E_{11} is real, due to $S_{11} < S^-_{c1}$. Both inequalities $S_{12} < S^-_{c1}$ and $S_{12} > S^-_{c1}$ are possible with $S^-_{c2} > S_{12}$, so the equilibrium E_{12} may be real or virtual. The detailed nature of the endemic equilibrium E_{12} as well as that of E_{21} and E_{22} are listed in Table 1. By some algebra, we easily get that all cases except (Q_{11}), (Q_{22}), (Q_{31}) are possible in this scenario. Since one of the disease-free equilibria including E_{10}, E_{20} and E_0 is always stable, one of the endemic equilibria E_{11} and E_{21} or both compete with one of the disease-free equilibria. In particular, for cases (Q_{12}), (Q_{21}), (Q_{24}), (Q_{25}), (Q_{26}), (Q_{27}), (Q_{28}), (Q_{33}) and (Q_{34}), there exist two attractors (the endemic equilibrium E_{11} and one of the disease-free equilibria) for system (3) if E_{11} is stable; otherwise, only the disease-free equilibrium acts as the attractor if E_{11} is unstable.

For cases (Q_{13}), (Q_{23}) and (Q_{32}), stability of multiple equilibria may occur. In fact, there may be one or two or three attractors for system (3). When both endemic equilibria E_{11} and E_{21} are unstable, only the disease-free equilibrium is the attractor. The endemic equilibria E_{11} or E_{21} and one of the disease-free equilibria are the attractors if E_{11} or E_{21} is stable and the other one is unstable, as shown in Fig. 4. In Fig. 4, the endemic equilibrium E_{11} and the disease-free equilibrium E_{20} are locally asymptotically stable, while the endemic equilibrium E_{21} is unstable. Figure 4b shows the instability of the endemic equilibrium E_{21}, which is an augmentation of Fig. 4a. Any trajectory initiating from G_{e11} ultimately approaches the endemic equilibrium E_{11}, so the disease cannot die out and become endemic. Here G_{e11}, G_{e01}, G_{e12}, G_{e02} is as defined above. All trajectories starting from $G_{e01}\backslash\{\Gamma_{s21} \cup \Gamma_{s22}\}$ tend to the disease-free equilibrium E_{20}, which leads to the eradication of the disease.

If both the endemic equilibria E_{11} and E_{21} are stable for cases (Q_{13}), (Q_{23}) and (Q_{32}), three attractors (the endemic equilibria E_{11}, E_{21} and one of the disease-free equilibrium) coexist for system (3), as shown in Fig. 5. In Fig. 5, the two stable manifolds of E_{12} (i.e., curves Γ_{s11} and Γ_{s12}) and the two stable manifolds of E_{22} (i.e., curves Γ_{s21} and Γ_{s22}) divide the first quadrant into three parts. Denote the part above

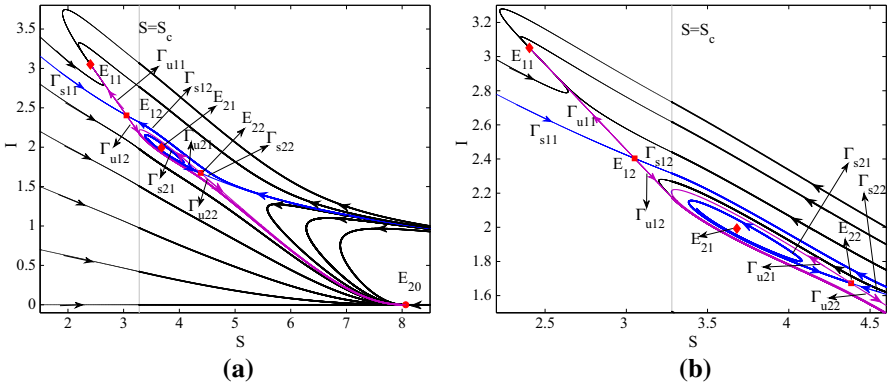

Fig. 4 Phase plane of the non-smooth model (3), showing the stability of multi-equilibria including E_{11} and E_{20}. The parameter values are $\Lambda = 6$; $\beta = 0.15$; $\mu = 0.5$; $r = 0.6$; $\epsilon = 0.5$, $S_c = 3.28$

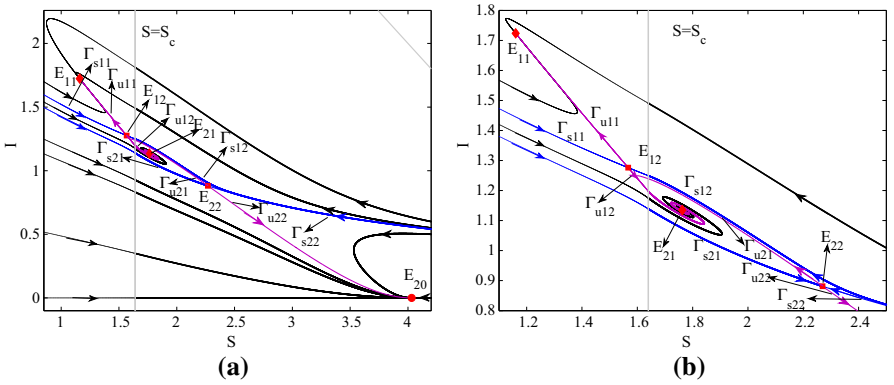

Fig. 5 Phase plane of the non-smooth model (3), showing the stability of multi-equilibria including E_{11}, E_{21} and E_{20}. The parameter values are $\Lambda = 3$; $\beta = 0.5$; $\mu = 0.5$; $r = 0.6$; $\epsilon = 0.5$, $S_c = 1.64$

Γ_{s11} and Γ_{s12} as G_{e13}, the one below Γ_{s21} and Γ_{s22} as G_{e03} and the part between $\Gamma_{s11}, \Gamma_{s12}$ and $\Gamma_{s21}, \Gamma_{s22}$ as G_{e23}. Subregions G_{e13}, G_{e23} and G_{e03} are shown in Fig. 6. Every trajectory initiating from G_{e13} tends to the endemic equilibrium E_{11}; those trajectories initiating from G_{e03} go to the disease-free equilibrium; all trajectories starting from G_{e23} approach the other endemic equilibrium E_{21}. This suggests that the area G_{e13} represents the high endemic region; the area G_{e23} stands for the low endemic region; the area G_{e03} represents the disease eradication region. Hence, the disease can die out by implementing the threshold policy if the initial infection lies in the disease eradication region. The disease can become endemic if the initial infection lies in either the higher endemic region or the lower endemic region. However, the size of the infected class in steady state is distinct for the last two cases, which implies that the ultimate number of infected individuals can be higher or lower depending on the initial situation if the disease cannot be eradicated from the population. Concluding the above discussion yields the following result.

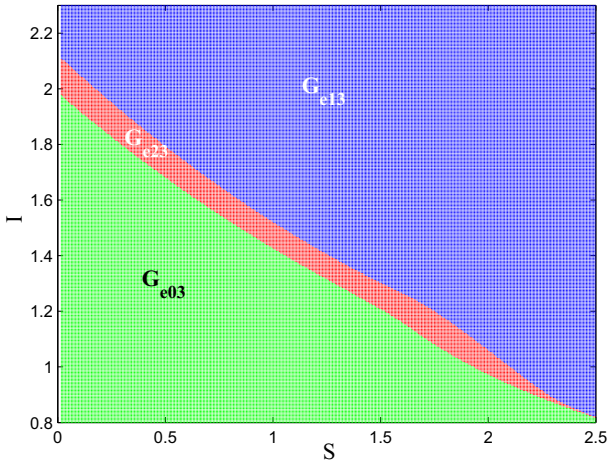

Fig. 6 The basin of attraction for the three attractors of system (3). Areas G_{e13}, G_{e23} and G_{e03} are the basins of attraction for the endemic equilibria E_{11}, E_{21} and the disease-free equilibrium E_{20}, respectively. The parameter values are $\Lambda = 3$; $\beta = 0.5$; $\mu = 0.5$; $r = 0.6$; $\epsilon = 0.5$, $S_c = 1.64$

Table 2 Main results of (3) for (C_1)

Values of R_{c1}, R_{c2}	Cases	Conditions	Attractors
$R_{c1} > R_{c2} > 1$	(Q_{12}),(Q_{13}),$(Q_{32})-(Q_{34})$, (Q_{21}),$(Q_{23})-(Q_{28})$,	(H_3) or (H_6)	E_{s0}
		(H_1) or (H_2)	E_{s0}, E_{11}
	(Q_{13}), (Q_{23}), (Q_{32})	(H_4) or (H_5)	E_{s0}, E_{21}
		(H_s) and $(H_l)(s = 1, 2, u = 4, 5)$	E_{s0}, E_{11}, E_{21}

Theorem 5.4 *(i) If the inequalities* (H_3) *and* (H_6) *hold, the solution of* (3) *ultimately goes to* E_{s0} *for most cases except Cases* (Q_{11}), (Q_{22}) *and* (Q_{31}) *and* $X_0 \in R^2_+ \backslash \{\Gamma_{s11} \cup \Gamma_{s12}\}$ *or* $X_0 \in R^2_+ \backslash \{\Gamma_{s21} \cup \Gamma_{s22}\}$ *or* $X_0 \in R^2_+ \backslash \{\Gamma_{s11} \cup \Gamma_{s12} \cup \Gamma_{s21} \cup \Gamma_{s22}\}$.

(ii) If the inequalities (H_1) *or* (H_2) *hold, then, for most cases except Cases* (Q_{11}), (Q_{22}) *and* (Q_{31}), *the solution of* (3) *will approach* E_{11} *for* $X_0 \in G_{e11}$ *while it will tend to* E_{s0} *for* $X_0 \in G_{e01}$ *or* $X_0 \in G_{e01} \backslash \{\Gamma_{s21} \cup \Gamma_{s22}\}$.

(iii) If the inequalities (H_4) *or* (H_5) *hold, then, for Cases* (Q_{13}), (Q_{23}) *and* (Q_{32}), *the solution of* (3) *will approach* E_{21} *for* $X_0 \in G_{e12}$ *or* $X_0 \in G_{e12} \backslash \{\Gamma_{s11} \cup \Gamma_{s12}\}$, *while it will tend to* E_{s0} *for* $X_0 \in G_{e02}$.

(iv) If the inequalities (H_s) $(s = 1, 2)$ *and* (H_l) $(l = 4, 5)$ *hold, then, for Cases* (Q_{13}), (Q_{23}) *and* (Q_{32}), *the solution of* (3) *will approach* E_{11} *for* $X_0 \in G_{e13}$, *solutions tend to* E_{21} *for* $X_0 \in G_{e23}$, *and will tend to* E_{s0} *for* $X_0 \in G_{e03}$.

For clarity, we list the result obtained in this case in Table 2.

(C_2) $R_{c2} > R_{c1} > 1$.

The threshold level satisfies $S_c < S^-_{c1}$ in this scenario. By implementing a similar analysis to (C_1), we get two possible attractors for Cases (Q_{12}), (Q_{21}), $(Q_{24})-(Q_{28})$,

(Q_{33}) and (Q_{34}); i.e., the endemic equilibrium E_{11} and one of the disease-free equilibria. Further discussion yields E_{11} and one of the disease-free equilibria are locally asymptotically stable if (H_1) or (H_2) hold, while only the unique disease-free equilibrium is locally asymptotically stable if (H_3) holds. This indicates that $R_{c2} > R_{c1} > 1$ will not always lead to prevalence of the disease. Similarly, if (H_4) or (H_5) hold, the endemic equilibrium E_{21} and one of the disease-free equilibria is locally asymptotically stable for Cases (Q_{11}), (Q_{22}) and (Q_{31}); if (H_6) holds, only one of the disease-free equilibria is locally asymptotically stable. For Cases (Q_{13}), (Q_{23}) and (Q_{32}), three possible attractors may coexist for system (3): the endemic equilibria E_{11}, E_{21} and one of the disease-free equilibria. In conclusion, both the endemic equilibria E_{11}, E_{21} and one of the disease-free equilibria are locally asymptotically stable if (H_l) $(l = 1, 2)$ and (H_k) $(k = 4, 5)$ hold; one of the endemic equilibria E_{j1} $(j = 1, 2)$ and one of the disease-free equilibria are locally asymptotically stable if (H_s) $(s = 1, 2, 4, 5)$ holds; only the unique disease-free equilibrium is locally asymptotically stable if (H_3) and (H_6) hold.

Theorem 5.5 *(i) If (H_3) and (H_6) hold, the solution of (3) ultimately goes to E_{s0} for $X_0 \in R^2_+ \backslash \{\Gamma_{s11} \cup \Gamma_{s12}\}$ or $X_0 \in R^2_+ \backslash \{\Gamma_{s21} \cup \Gamma_{s22}\}$ or $X_0 \in R^2_+ \backslash \{\Gamma_{s21} \cup \Gamma_{s22} \cup \Gamma_{s11} \cup \Gamma_{s12}\}$*

(ii) If (H_1) or (H_2) hold, then for cases $(Q_{12}), (Q_{13}), (Q_{21}), (Q_{23})-(Q_{28})$ and $(Q_{32})-(Q_{34})$, the solution of (3) will approach E_{11} for $X_0 \in G_{e11}$, while it will tend to E_{s0} for $X_0 \in G_{e01}$.

(iii) If (H_4) or (H_5) hold, then for cases $(Q_{11}), (Q_{13}), (Q_{22}), (Q_{23}), (Q_{31})$ and (Q_{32}), the solution of (3) will approach E_{21} for $X_0 \in G_{e21}$, while it will tend to E_{s0} for $X_0 \in G_{e02}$.

(iv) If (H_s) $(s = 1, 2)$ and (H_l) $(l = 4, 5)$ hold, then, for cases $(Q_{13}), (Q_{23})$ and (Q_{32}), the solution of (3) will approach E_{11} for $X_0 \in G_{e13}$, solutions tend to E_{21} for $X_0 \in G_{e23}$ and will tend to E_{s0} for $X_0 \in G_{e03}$.

Now we turn to examine the critical case $S_c = S_{11}$, when the threshold level S_c reaches exactly the level of susceptible people in steady state. Thus, a generalized equilibrium $E_*(S_*, I_*)$ occurs, where

$$S_* = \frac{\beta\Lambda - \sqrt{\beta^2\Lambda^2 - 4\beta(\mu+\epsilon)^2(\mu+r)}}{2\beta(\mu+r)}, \quad I_* = \frac{\beta\Lambda + \sqrt{\beta^2\Lambda^2 - 4\beta(\mu+\epsilon)^2(\mu+r)}}{2\beta(\mu+\epsilon)}.$$

Without loss of generality, let E_* be a focus point of subsystem S_{G_1} and S_{G_2}, which implies E_* is a parabolic-focus of FF type. It is worth emphasizing that system (3) is no longer smooth, which means the Jacobian matrix and the Lyapunov coefficients for smooth systems cannot determine local stability. In the following, we will examine its stability by using the theory developed in Clarke et al. (1998) and Leine (2006).

Evaluating the Jacobian matrix J_1 at E_* yields

$$\mathrm{tr}(J_1(E_*)) = (\epsilon - r) - \beta I_*^2, \quad \det(J_1(E_*)) = (\mu+\epsilon)(\beta I_*^2 - \mu - r).$$

Denote

$$\alpha_1 = \mathrm{tr}(J_1(E_*)), \quad \beta_1 = \sqrt{\alpha_1^2 - 4\det(J_1(E_*))}.$$

Similarly, by evaluating the Jacobian matrix J_2 of subsystem S_{G_2}, we get

$$\alpha_2 = \epsilon - \beta I_*^2, \quad \beta_2 = \sqrt{(\epsilon - \beta I_*^2)^2 - 4(\mu+\epsilon)(\beta I_*^2 - \mu)}.$$

The first-order generalized Lyapunov coefficient at E_* is

$$V_1 = v_1 v_2 - 1,$$

where

$$v_1 = \exp\left(\frac{\pi\alpha_1}{\beta_1}\right), \quad v_2 = \exp\left(\frac{\pi\alpha_2}{\beta_2}\right),$$

so $\mathrm{sgn}(V_1) = \mathrm{sgn}(\alpha_1\beta_2 + \alpha_2\beta_1) \doteq \mathrm{sgn}(\eta)$. To determine the sign of η, we will consider the following three possibilities: (a) $\epsilon - \beta I_*^2 \le 0$; (b) $\epsilon - r - \beta I_*^2 \ge 0$; (c) $\epsilon - \beta I_*^2 > 0,\ \epsilon - r - \beta I_*^2 < 0$ according to the sign of $\epsilon - \beta I_*^2$ and $\epsilon - r - \beta I_*^2$. We initially examine case (a).

(a) $\epsilon - \beta I_*^2 \le 0$.

In this scenario, we easily get $\eta < 0$, so $V_1 < 0$ and the generalized equilibrium E_* is locally asymptotically stable. In fact, $\epsilon - \beta I_*^2 \le 0$ is equivalent to

$$2(\mu+\epsilon)^2(\mu+\epsilon+r) - \beta\Lambda^2 \le \Lambda\sqrt{\beta^2\Lambda^2 - 4\beta(\mu+\epsilon)^2(\mu+r)}.$$

The last inequality is true if and only if

$$2(\mu+\epsilon)^2(\mu+\epsilon+r) - \beta\Lambda^2 \le 0 \tag{13}$$

or

$$\begin{cases} 2(\mu+\epsilon)^2(\mu+\epsilon+r) - \beta\Lambda^2 > 0 \\ (\mu+\epsilon)^2(\mu+\epsilon+r)^2 - \beta\epsilon\Lambda^2 \ge 0 \end{cases} \tag{14}$$

is true. Denote

$$M_1 = \frac{2(\mu+\epsilon)^2(\mu+\epsilon+r)}{\Lambda^2}, \quad M_2 = \frac{(\mu+\epsilon)^2(\mu+\epsilon+r)^2}{\epsilon\Lambda^2}.$$

We have $M_2 < M_1$ for $\epsilon > \mu + r$ and $M_2 > M_1$ for $\epsilon < \mu + r$. Solving (13) and (14) gives $\beta \ge M_1$ and $\epsilon > \mu + r$, $M_2 < \beta < M_1$, respectively. Hence, if the following inequalities

- $\epsilon \le \mu + r,\ \beta \ge M_1$ or
- $\epsilon > \mu + r,\ \beta > M_2$

hold, then $\eta < 0$, and so $V_1 < 0$, which results in the local stability of E_*.

(b) $\epsilon - r - \beta I_*^2 \geq 0$.

If this inequality is true, then $\eta > 0$, so $V_1 > 0$ and the generalized equilibrium E_* is unstable. In fact, the inequality $\epsilon - r - \beta I_*^2 > 0$ holds if and only if

$$2(\mu+\epsilon)^3 - \beta\Lambda^2 \geq \Lambda\sqrt{\beta^2\Lambda^2 - 4\beta(\mu+\epsilon)^2(\mu+r)},$$

which is equivalent to

$$\begin{cases} 2(\mu+\epsilon)^3 - \beta\Lambda^2 \geq 0, \\ \beta\Lambda^2(\epsilon - r) \leq (\mu+\epsilon)^4. \end{cases} \tag{15}$$

Solving (15) yields

$$\epsilon > r, \ \beta \leq \min\{M_3, M_4\},$$

where

$$M_3 = \frac{2(\mu+\epsilon)^3}{\Lambda^2}, \ M_4 = \frac{(\mu+\epsilon)^4}{\Lambda^2(\epsilon - r)}.$$

Direct calculation yields $M_3 < M_4$ for $\epsilon < \mu + 2r$ and $M_3 > M_4$ for $\epsilon > \mu + 2r$. As a conclusion, if one of the following inequalities

- $r < \epsilon \leq \mu + 2r, \ \beta \leq M_3$ or
- $\epsilon > \mu + 2r, \ \beta \leq M_4$

is true, the generalized equilibrium E_* is unstable.

(c) $\epsilon - \beta I_*^2 > 0, \ \epsilon - r - \beta I_*^2 < 0$.

For convenience, we refer to these two inequalities as inequality (c) in the following discussion. Different to (a) and (b), both a stable equilibrium E_* and an unstable equilibrium E_* are possible in this scenario. Performing a similar analysis to (a), we get the first inequality in this scenario; i.e., $\epsilon - \beta I_*^2 > 0$ holds if one of the following inequalities

- $\epsilon \geq \mu + r, \ \beta < M_2$ or
- $\epsilon < \mu + r, \ \beta < M_1$

is true. Similarly, the second inequality $\epsilon - r - \beta I_*^2 < 0$ is true if one of the following inequalities

- $\epsilon \leq \mu + 2r, \ \beta \geq M_3$ or
- $\epsilon > \mu + 2r, \ \beta > M_4$

are true. Direct calculation gives $M_3 < M_1$, so $M_4 < M_3 < M_1$ for $\epsilon > \mu + 2r$. Note that

$$\text{sgn}(M_3 - M_2) = \text{sgn}\{\epsilon^2 - 2r\epsilon - (\mu+r)^2\} = -1$$

if and only if

$$\epsilon < r + \sqrt{r^2 + (\mu + r)^2},$$

so the inequality $r + \sqrt{r^2 + (\mu + r)^2} > \mu + 2r$ leads to $M_3 < M_2$ for $\mu + r < \epsilon \leq \mu + 2r$. Similarly, we have

$$\operatorname{sgn}(M_4 - M_2) = \operatorname{sgn}\{-\epsilon^2 + r\epsilon + (\mu + r)^2\} = -1$$

if and only if

$$\epsilon > \frac{r + \sqrt{r^2 + 4(\mu + r)^2}}{2},$$

so $M_4 < M_2$ for $\epsilon > \mu + 2r$ due to $(r + \sqrt{r^2 + 4(\mu + r)^2})/2 < \mu + 2r$. Hence, inequality (c) is true if one of the following inequalities

$$\epsilon > \mu + 2r,\ M_4 < \beta < M_2$$

or

$$\mu + r < \epsilon \leq \mu + 2r,\ M_3 \leq \beta < M_2$$

or

$$\epsilon \leq \mu + r,\ M_3 \leq \beta < M_1$$

is true.

To derive the stability of E_*, examining $\eta < 0$ is necessary. Note that in this scenario,

$$\eta < 0 \Longleftrightarrow (\beta I_*^2)^2 + (r - 4\epsilon - 2\mu)\beta I_* + (\epsilon^2 + 2\epsilon\mu - \mu r) < 0. \tag{16}$$

Inequality (16) holds if and only if

$$N_{\min} < \beta I_*^2 < N_{\max},$$

where

$$N_{\max} = \frac{2\mu + 4\epsilon - r + \sqrt{12\epsilon^2 + 8\epsilon\mu + r^2 + 4\mu^2 - 8\epsilon r}}{2},$$

$$N_{\min} = \frac{2\mu + 4\epsilon - r - \sqrt{12\epsilon^2 + 8\epsilon\mu + r^2 + 4\mu^2 - 8\epsilon r}}{2}.$$

Denote

$$\begin{aligned}
\eta_1 &= (\mu+\epsilon)^2\big[4\mu+4\epsilon+r+\sqrt{12\epsilon^2+8\epsilon\mu+r^2+4\mu^2-8\epsilon r}\big]\\
\eta_2 &= (\mu+\epsilon)^2\big[4\mu+4\epsilon+r-\sqrt{12\epsilon^2+8\epsilon\mu+r^2+4\mu^2-8\epsilon r}\big]\\
\overline{\eta}_1 &= \frac{\eta_1^2}{2\Lambda^2(\mu+\epsilon)^2\big[2\mu+4\epsilon-r+\sqrt{12\epsilon^2+8\epsilon\mu+r^2+4\mu^2-8\epsilon r}\big]}\\
\overline{\eta}_2 &= \frac{\eta_2^2}{2\Lambda^2(\mu+\epsilon)^2\big[2\mu+4\epsilon-r-\sqrt{12\epsilon^2+8\epsilon\mu+r^2+4\mu^2-8\epsilon r}\big]}.
\end{aligned}$$

We have

$$\beta I_*^2 < N_{\max} \Longleftrightarrow \eta_1 - \beta\Lambda^2 > \Lambda\sqrt{\beta\Lambda^2 - \mu\beta(\mu+\epsilon)^2(\mu+r)}.$$

The last inequality is true if and only if

$$\begin{cases} \eta_1 - \beta\Lambda^2 > 0 \\ \eta_1^2 - 2\beta\eta_1\Lambda^2 + \beta^2\Lambda^4 > \Lambda^2\big[\beta\Lambda^2 - 4\beta(\mu+\epsilon)^2(\mu+r)\big]. \end{cases} \tag{17}$$

Solving (17) with respect to β gives

$$\beta < \min\left\{\frac{\eta_1}{\Lambda^2}, \overline{\eta}_1\right\}.$$

Similarly, we have

$$\beta I_*^2 > N_{\min} \Longleftrightarrow \eta_2 - \beta\Lambda^2 < \Lambda\sqrt{\beta\Lambda^2 - \mu\beta(\mu+\epsilon)^2(\mu+r)}.$$

The inequality on the right-hand side is true if and only if

$$\eta_2 - \beta\Lambda^2 \le 0 \tag{18}$$

or

$$\begin{cases} \eta_2 - \beta\Lambda^2 > 0 \\ \eta_2^2 - 2\beta\eta_2\Lambda^2 + \beta^2\Lambda^4 < \Lambda^2\big[\beta\Lambda^2 - 4\beta(\mu+\epsilon)^2(\mu+r)\big]. \end{cases} \tag{19}$$

Solving (18) and (19) with respect to β gives $\beta > \overline{\eta}_2$. Direct calculation yields

$$\overline{\eta}_1 < \frac{\eta_1}{\Lambda^2}, \quad \overline{\eta}_1 > \overline{\eta}_2,$$

so inequality (16) is true if and only if $\overline{\eta}_2 < \beta < \overline{\eta}_1$. Therefore, we conclude that the generalized equilibrium E_* is locally asymptotically stable if

$$\epsilon \le \mu + r, \ \min\{M_3, \overline{\eta}_2\} < \beta < \max\{M_1, \overline{\eta}_1\}$$

or

$$\mu + r < \epsilon \leq \mu + 2r,\ \min\{M_3, \overline{\eta}_2\} < \beta < \max\{M_2, \overline{\eta}_1\}$$

or

$$\epsilon > \mu + 2r,\ \min\{M_4, \overline{\eta}_2\} < \beta < \max\{M_2, \overline{\eta}_1\}$$

in this scenario.

A similar procedure gives the conditions for the unstable equilibrium E_*. Figures 7 and 8 illustrate the stability of the pseudo-equilibrium E_*. Here the diamond point E_* represents the pseudo-equilibrium and the thick cycle Γ_s stands for the crossing cycle. If we denote the subregion above the unstable manifolds of saddle E_{22} (the curves Γ_{s21} and Γ_{s22}) as G_{1p} and the subregion below them as G_{0p}, then summarizing (a)–(c) results in the following conclusion.

Remark 3. Here we use "crossing cycle" to denote the limit cycle composed of pieces of the orbit of subsystem S_{G_1} and pieces of the orbit of subsystem S_{G_2}.

Theorem 5.6 *(i) All solutions with $X_0 \in G_{1p}$ approach the generalized equilibrium E_* and those with $X_0 \in G_{0p}$ tend to the disease-free equilibrium E_{s0} (as shown in Fig. 7) if one of the following conditions hold:*

(C_{s1}) $\epsilon > \mu + 2r, \max\{M_4, \overline{\eta}_2\} < \beta < \min\{M_2, \overline{\eta}_1\}$ or $\beta > M_2$;
(C_{s2}) $\mu + r < \epsilon \leq \mu + 2r, \max\{M_3, \overline{\eta}_2\} < \beta < \min\{M_2, \overline{\eta}_1\}$ or $\beta > M_2$;
(C_{s3}) $\epsilon \leq \mu + r, \max\{M_3, \overline{\eta}_2\} < \beta < \min\{M_1, \overline{\eta}_1\}$ or $\beta > M_1$.

(ii) All solutions with $X_0 \in G_{1p} \backslash E_*$ approach the crossing cycle Γ_s, and those with $X_0 \in G_{0p}$ tend to the disease-free equilibrium E_{s0} (as shown in Fig. 8) if one of the following inequalities hold.

(C_{u1}) $\epsilon > \mu + 2r, \max\{M_4, \overline{\eta}_1\} < \beta < M_2$ or $M_4 < \beta < \min\{M_2, \overline{\eta}_2\}$ or $\beta < M_4$;
(C_{u2}) $\mu + r < \epsilon \leq \mu + 2r, \max\{M_3, \overline{\eta}_1\} < \beta < M_2$ or $M_3 < \beta < \min\{M_2, \overline{\eta}_2\}$ or $\beta < M_3$;
(C_{u3}) $r < \epsilon \leq \mu + r, \max\{M_3, \overline{\eta}_1\} < \beta < M_1$ or $M_3 < \beta < \min\{M_1, \overline{\eta}_2\}$ or $\beta < M_3$;
(C_{u4}) $\epsilon \leq r, \max\{M_3, \overline{\eta}_1\} < \beta < M_1$ or $M_3 < \beta < \min\{M_1, \overline{\eta}_2\}$.

It follows that the pseudo-equilibrium E_* competes with the disease-free equilibrium if the inequalities (C_{si}) $(i = 1, 2, 3)$ hold. In particular, the pseudo-equilibrium E_* is locally asymptotically stable for $X_0 \in G_{1p}$ and the disease-free equilibrium E_{20} is locally asymptotically stable for $X_0 \in G_{0p}$. This indicates that disease control is dependent on the initial sizes of the sub-populations. If the initial size of the sub-population satisfies $X_0 \in G_{0p}$, the disease becomes endemic, but the number of infected individuals can be contained at a previously given value I_*. However, if the conditions (C_{ui}) $(i = 1, 2, 3, 4)$ hold, the crossing cycle Γ_s coexists with the disease-free equilibrium. In this scenario, the crossing cycle Γ_s is locally asymptotically stable

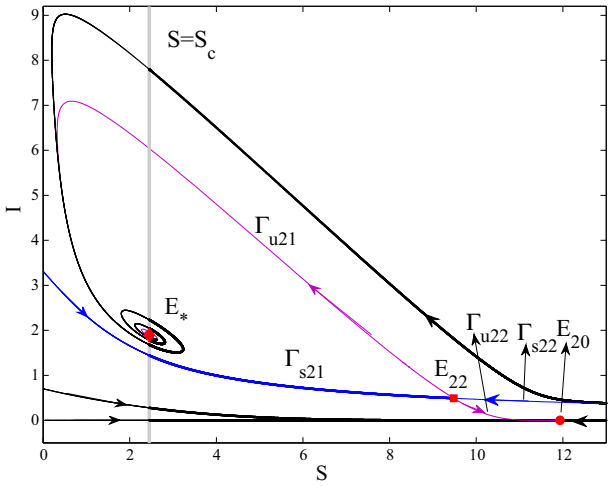

Fig. 7 Phase plane of the non-smooth model (3), showing the stability of FF-type generalized focus E_*. The parameter values are $\Lambda = 3$; $\beta = 0.215$; $\mu = 0.2$; $r = 0.25$; $\epsilon = 0.8$, $S_c = 2.8128$

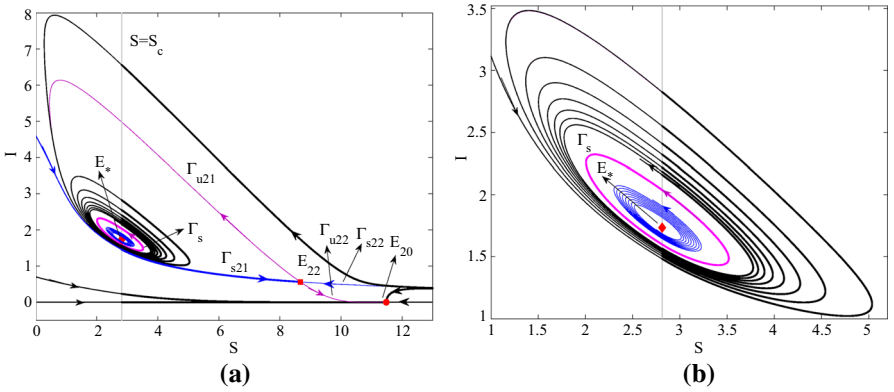

Fig. 8 Phase plane of the non-smooth model (3), showing the instability of the FF-type generalized focus and the stability of the crossing cycle and the disease-free equilibrium. The parameter values are $\Lambda = 3$; $\beta = 0.205$; $\mu = 0.2$; $r = 0.25$; $\epsilon = 0.8$, $S_c = 2.8128$

for $X_0 \in G_{1p}$ while the disease-free equilibrium is locally asymptotically stable for $X_0 \in G_{0p}$; i.e., G_{1p} and G_{0p} are also the endemic and eradication regions, respectively. Hence, the disease cannot be eradicated and the number of infected individuals varies periodically. In conclusion, if $S_c = S_{11}$ and $X_0 \in G_{1p}$, the case number can either be controlled at a previously given level or the solutions may vary periodically, but the disease cannot be eradicated.

For clarity, we list the results in this case in Table 3.

(C_3) $R_{c1} = R_{c2} > 1$.

We have that $S_c = S_{c1}^-$ in this scenario. Note that $S_{11} < S_{c1}^-$, so the endemic equilibrium E_{11} is always real. The nature of other endemic equilibria, especially that of the endemic equilibrium E_{21}, can be found in Table 1. Performing a similar

Table 3 Main results of (3) for (C_2)

Values of R_{c1}, R_{c2}	Cases	Conditions	Attractors
$R_{c2} > R_{c1} > 1$	All cases	(H_3) and (H_6)	E_{s0}
	$(Q_{12}), (Q_{13}), (Q_{21}),$ $(Q_{23})-(Q_{28}), (Q_{32})-(Q_{34})$	(H_1) or (H_2)	E_{s0}, E_{11}
	$(Q_{11}), (Q_{13}), (Q_{22}),$ $(Q_{23}), (Q_{31}), (Q_{32})$	(H_4) or (H_5)	E_{s0}, E_{21}
	$(Q_{13}), (Q_{23}), (Q_{32})$	(H_s) and $(H_l)(s \in \{1,2\}, u \in \{4,5\})$	E_{s0}, E_{11}, E_{21}
		$S_c = S_{11}$ and $(C_{sj})(j \in \{1,2,3\})$	E_{s0}, E_*
		$S_c = S_{11}$ and $(C_{uj})(j \in \{1,2,3\})$	E_{s0}, Γ_s

analysis as in Case (C_1), we get a series of interesting dynamical behavior including the stability of multiple equilibrium points (including two endemic equilibria and one disease-free equilibrium), bistability of the endemic equilibrium and a disease-free equilibrium, and the stability of the disease-free equilibrium. We omit the detail here.

Remarks 4. If we consider the continuous vaccination policy (i.e., $S_c = \infty$), then system (1) and (2) approaches system S_{G_1} in the limit. This illustrates the case when the medical resource is abundant and the vaccination rate is always proportional to the number of susceptible individuals. According to Theorems 3.1 and 3.2, the disease-free equilibrium E_{10} is globally asymptotically stable, and the disease can be eradicated for $R_{c1} < 1$; if $R_{c1} > 1$ and (H_3) is true, E_{10} is locally asymptotically stable, and the disease can be almost eradicated; otherwise, if the inequalities in (H_1) or (H_2) are true, both the endemic equilibrium E_{11} and the disease-free equilibrium E_{10} are locally asymptotically stable, which demonstrates that disease eradication depends on the initial infection.

5. However, the non-smooth system (1) and (2) is an epidemic system with a piecewise-defined vaccination policy. It follows from Theorems 5.1–5.6 that the dynamics are much richer than system S_{G_1}. In addition to the bistability of the endemic equilibrium E_{11} and one of the disease-free equilibria E_{s0}, coexistence of multiple equilibria may occur: the possibilities are three stable equilibria, E_{11}, E_{s0} and another endemic equilibrium E_{21}; coexistence of the generalized equilibria E_* and E_{s0}; and the coexistence of the crossing cycle Γ_s and E_{s0}. These outcomes are new and are qualitatively different from the dynamics for system S_{G_1}. Specifically, we have shown that different initial infections can trigger different control outcomes; a previously chosen level or region of the desired number of the infected individuals can be reached when the threshold level and other parameters are chosen properly; this may provide us options for disease control when medical resources are limited and the infectious disease cannot be eradicated.

6 Discussion and Conclusions

Medical-resource constraints are important in disease control (Wang and Ruan 2004; Zhang and Liu 2008; Wang 2006; Hansen and Day 2011; Zhou and Fan 2012; Shan

and Zhu 2014). To explore how they affect disease containment, many mathematical models with piecewise-defined treatment program have been proposed, resulting in non-smooth continuous models with threshold values that are qualitatively different from their smooth counterparts. However, to the best of our knowledge, there is little systematic analysis of the dynamical behavior as the threshold level varies. In this paper, we have analyzed a simple non-smooth model with vaccination, which is mathematically similar to the treatment policy in Wang (2006). Since we also take into account double exposure of the susceptibles on disease spread, a nonlinear incidence rate βSI^2 is incorporated in our model. Our main results show that the non-smooth vaccination measure induces the multistability of three regular equilibria, bistablity of two regular equilibria or one generalized equilibria and one disease-free equilibrium or one crossing cycle and one disease-free equilibrium.

We mainly focus on the interaction of the nonlinear incidence rate and non-smooth vaccination. To this end, we deliberately examine the dynamic behavior of the proposed model as the threshold value varies. We address the dynamics of the proposed model according to the relationship between the two advanced thresholds R_{c1} and R_{c2}, which depend on the threshold value S_c. If the threshold value S_c varies such that $\max\{R_{c1}, R_{c2}\} < 1$, no endemic equilibrium exists and the disease dies out, as shown in Fig. 1. If $R_{c1} > 1 > R_{c2}$, the disease also dies out except the case where the initial point $X_0 \in \Gamma_{s11} \cup \Gamma_{s12}$ for condition (H_3), as shown in Fig. 2. But for the conditions (H_1) and (H_2), the infection region G_{e11} exists next to the healthy region G_{e01}, so the disease can be eradicated from the population if the initial point $X_0 \in G_{e01}$, but it persists for $X_0 \in G_{e11}$, as shown in Fig. 3. Similar results can be obtained for the case $R_{c2} > 1 > R_{c1}$, where the disease almost dies out if (H_6) holds; it may die out or persist if (H_4) and (H_5) hold, depending on whether the initial condition lies in the healthy or infection region. It follows that the nonlinear incidence rate induces the local stability of the disease even with the existence of one or more steady states. It highlights the key role of the initial condition as well as the threshold value in the outcome.

If the threshold value S_c continuously varies such that $\min\{R_{c1}, R_{c2}\} > 1$, the model behaves dramatically different from its continuous counterpart. In this scenario, if we further have (H_s) $(s = 1, 2)$ and (H_l) $(l = 4, 5)$, three stable equilibria (two endemic equilibria and one disease-free equilibrium) coexist, so a high infection area G_{e13}, a low infection area G_{e23} and a healthy area G_{e03} coexist, as shown in Figs. 5 and 6 and Table 2. This demonstrates that the disease may die out or persist with a higher infection level or persist with a lower infection level depends on whether it originates in the healthy area, the high infection area or the low infection area. This shows that different initial infection levels lead to different long-term infection levels on the condition that the disease cannot be eradicated from the population. When the threshold value S_c passes through the critical level S_{11}, a generalized equilibrium E_* or crossing cycle Γ_s appears if other parameters satisfy conditions (C_{sj}) or (C_{uj}) with $j \in \{1, 2, 3\}$. As a consequence, the case numbers can be contained at a controllable level if eradicating it from the population proves to be impossible.

In summary, we have provided a dynamical analysis of a simple non-smooth epidemic model with the assistance of phase diagrams in the whole parameter space. We have found the steady-state regimes, including healthy, low epidemic and high

epidemic, under which the infected individuals approach zero or a relatively low level or a relatively high level. The main results obtained in this work give insight into the various consequences of implementing intervention measures. Our findings show that a proper threshold policy can assist in controlling and combating an emerging infectious disease, especially when the medical resources are constrained.

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