

IMPACT OF DISCONTINUOUS TREATMENTS ON DISEASE DYNAMICS IN AN SIR EPIDEMIC MODEL

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ABSTRACT. We consider an SIR epidemic model with discontinuous treatment strategies. Under some reasonable assumptions on the discontinuous treatment function, we are able to determine the basic reproduction number \mathcal{R}_0 , confirm the well-posedness of the model, describe the structure of possible equilibria as well as establish the stability/instability of the equilibria. Most interestingly, we find that in the case that an equilibrium is asymptotically stable, the convergence to the equilibrium can actually be achieved *in finite time*, and we can estimate this time in terms of the model parameters, initial sub-populations and the initial treatment strength. This suggests that from the view point of eliminating the disease from the host population, discontinuous treatment strategies would be superior to continuous ones. The methods we use to obtain the mathematical results are the generalized Lyapunov theory for discontinuous differential equations and some results on non-smooth analysis.

1. Introduction. Infectious diseases remain to be one of the main sources of deaths for the human beings. Advances of knowledge on the existing infectious diseases, including knowledge about the mechanisms of transmissions, can help reduce the spread and hence the deaths. One important approach to understand disease transmission mechanisms in the population level is mathematical modeling, and differential equations play a crucial role in this regards because such equations describe how the rate of changes of sub-populations in host population depends on the main parameters as well as on the sub-populations themselves. There have been many differential equations models and the books [4] and [11] provide good coverages of some basic and classic models.

When an infectious disease emerges in a host population, it is desirable to consider some control measures. A natural question arises: how would such a measure

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affect the disease dynamics? A natural way to answer this question is to incorporate the control measure into the existing mathematical model that has been well understood and investigate how the measure will affect the behavior of the solutions to the model. Note that a control measure here is in a general sense and can include vaccination, curing treatment, quarantining, and isolation etc. For effects of vaccination on disease dynamics in some models, see, e.g., [1, 2, 5, 9] and the references therein; for some models studying the impact of quarantine and/or isolation, see, e.g., [6, 15, 16, 19, 20, 22] and their references. For general treatment, Brauer [10] considered a model for a heterogeneous population with a treatment, and investigated the impact of mixing and treatment. Wang [21] added a treatment function to a classic SIR model and observed the backward bifurcation which would not exist in the absence of the treatment. Motivated by [21], Zhang [23] extended the study to an SIS model and also obtained backward bifurcation.

This work is motivated by [21] where the treatment function is assumed to be continuous. In reality, the treatment strategy usually is not smooth and even not continuous because, due to limited resources, usually there are some restrictions on treatment strengths. In this paper, we use the model in [21], but we adopt discontinuous treatment function. Since the resulting model is a discontinuous system, we need to make use of the theory for discontinuous differential equations and some results on non-smooth analysis from [7, 8, 13, 14, 17]. Under some reasonable assumptions on the discontinuous treatment function, we explore the well-posedness (Section 2), and structure as well as stability of equilibria (Section 3). The basic reproduction number to such a discontinuous epidemic model is also calculated and its threshold role is confirmed too. As the most novel part of this work, we find in Section 4 that the convergence to an equilibrium can be achieved *in finite time*. This is especially significant when the equilibrium is the disease free equilibrium since this provides possibility to drive a disease to extinction in finite time, a conclusion in strong contrast to the results for models with continuous treatment functions where convergence to an equilibrium is in the sense of “asymptotic” (i.e., as $t \rightarrow \infty$). Our results even give explicit formula for such a finite time, and this allows us to investigate how the model parameters, initial sub-populations and the initial treatment strength affect this time. This suggests that from the view point of eliminating the disease from the host population, discontinuous treatment strategies would be superior to continuous ones. We point out that the phenomenon of convergence to an equilibrium in finite time has been reported for discontinuous neural network models in [18] which stimulated our work on this topic.

2. Model description and preliminaries. Consider the following SIR model with treatment proposed in [21] :

$$\begin{cases} \frac{dS}{dt} = A - dS - \lambda SI, \\ \frac{dI}{dt} = \lambda SI - (d + \gamma + \epsilon)I - h(I), \\ \frac{dR}{dt} = \gamma I + h(I) - dR, \end{cases} \quad (1)$$

where state variable S , I and R denote the sub-populations of susceptible, infective and recovered classes respectively in a host population. Among the model parameters which are all positive constants, A is the recruitment rate of the population; d is the natural death rate of the population; γ is the natural recovery rate of infective individuals; ϵ is the disease-related death rate; λ is the infection coefficient. Function $h(I)$ represents the treatment rate, which was assumed to be continuous

in I in [21]. But here, for realistic considerations as mentioned in the introduction, we allow some possible *jump discontinuities* by assuming the following

(A1) $h(I) = \varphi(I)I$, where $\varphi : \mathbb{R}_+ \rightarrow \mathbb{R}_+$ is non-decreasing and has at most a finite number of jump discontinuities in every compact interval.

Remark 1. Without loss of generality, we can always assume that φ is continuous at 0, since otherwise, we can modify the value of φ at 0 to be $\varphi(0^+)$ and this will not change the fact of $h(0) = 0$, and will bring no change to (1) and (2).

Since the first two equations in model (1) are independent of the variable R , it is sufficient to consider the following sub-system:

$$\begin{cases} \frac{dS}{dt} = A - dS - \lambda SI, \\ \frac{dI}{dt} = \lambda SI - (d + \gamma + \epsilon)I - h(I). \end{cases} \quad (2)$$

Due to the discontinuity on the right hand side in (2), many results in the classical theory of ordinary differential equations can not be applied here. To proceed, firstly, we need to give an appropriate definition of solution for model (2). Here, we adopt the definition of solution in the sense of Filippov [17].

A vector function $(S(t), I(t))$ on $[0, T)$, $T \in (0, +\infty]$, is a solution of model (2) with the initial condition $S(0) = S_0 \geq 0$ and $I(0) = I_0 \geq 0$, if $(S(t), I(t))$ is absolutely continuous on any subinterval $[t_1, t_2]$ of $[0, T)$, $S(0) = S_0$, $I(0) = I_0$, and for almost all (a. a.) $t \in [0, T)$, $(S(t), I(t))$ satisfies the following differential inclusion:

$$\begin{cases} \frac{dS}{dt} = A - dS - \lambda SI, \\ \frac{dI}{dt} \in \lambda SI - (d + \gamma + \epsilon)I - \overline{\text{co}}[h(I)], \end{cases} \quad (3)$$

where $\overline{\text{co}}[h(I)] = [h(I^-), h(I^+)]$, $h(I^-), h(I^+)$ denote the left and right limits of function h at I , respectively.

Note that under (A1), $\overline{\text{co}}[h(I)]$ is an interval with non-empty interior when $h(I)$ is discontinuous at I , while $\overline{\text{co}}[h(I)] = h(I)$ is a singleton when $h(I)$ is continuous at I . It is easy to see that the map $(S, I) \mapsto (A - dS - \lambda SI, \lambda SI - (d + \gamma + \epsilon)I - \overline{\text{co}}[h(I)])$ is an upper semi-continuous set-valued map with non-empty compact convex values ([7], p.67, Lemma 1). By the Measurable Selection Theorem ([7], p.90, Thm.1), we know that if (S, I) is a solution of model (2) on $[0, T)$, where $T \in (0, +\infty]$, then, there exists a measurable function $m(t) \in \overline{\text{co}}[h(I(t))]$ such that

$$\begin{cases} \frac{dS}{dt} = A - dS - \lambda SI, \\ \frac{dI}{dt} = \lambda SI - (d + \gamma + \epsilon)I - m(t), \end{cases} \quad \text{for a. a. } t \in [0, T). \quad (4)$$

Remark 2. It is obvious that a) the measurable function $m(t)$ in (4) is uniquely determined by $(S(t), I(t))$ up to a set of measure zero in $[0, T)$; and b) $m(t)$ is continuous for all $t \in [0, T)$ if and only if $(S(t), I(t))$ is continuously differentiable for all $t \in [0, T)$.

For biological reason, we need to prove the positiveness and boundedness of solutions of model (2) with positive initial values.

Proposition 1. *Suppose that (A1) is satisfied. Let $(S(t), I(t))$ be a solution of model (2) with initial condition $S(0) = S_0 \geq 0$, $I(0) = I_0 \geq 0$ on $[0, T)$, where $T \in (0, +\infty]$. Then, $S(t) \geq 0$ and $I(t) \geq 0$ for $t \in [0, T)$.*

Proof. By the definition of solution of model (2) in Filippov sense, $(S(t), I(t))$ is a solution of differential inclusion (3). From the S equation in (3), we have

$$\left. \frac{dS}{dt} \right|_{S=0} = (A - dS - \lambda SI)|_{S=0} = A > 0.$$

This together with $S(0) = S_0 \geq 0$ shows that $S(t) \geq 0, t \in [0, T)$.

Note that (A1) implies that $\overline{\text{co}}[h(0)] = \{0\}$ and $h(I)$ is continuous at 0. By the continuity of φ at $I = 0$ (see Remark 1), there exists a positive constant δ such that when $|I| < \delta$, $\varphi(I)$ is continuous and the differential inclusion in (3) becomes the following differential equation with continuous right hand side:

$$\frac{dI}{dt} = \lambda SI - (d + \gamma + \epsilon)I - \varphi(I)I = I[\lambda S - (d + \gamma + \epsilon) - \varphi(I)]. \quad (5)$$

Now if $I_0 = 0$, it follows from (5) that $I(t) = 0$ for all $t \in [0, T)$. If $I_0 > 0$, we claim that $I(t) > 0$ for all $t \in [0, T)$. Otherwise, let $t_1 = \inf\{t : I(t) = 0\}$. Then, $t_1 > 0$ and $I(t_1) = 0$. It follows from the continuity of $I(t)$ on $[0, T)$ that there exists a positive constant θ such that $t_1 - \theta > 0$ and $0 < I(t) < \delta$ for $t \in [t_1 - \theta, t_1)$. Then, integrating (5) from $t_1 - \theta > 0$ to t_1 leads to

$$0 = I(t_1) = I(t_1 - \theta)e^{\int_{t_1 - \theta}^{t_1} [\lambda S(\xi) - (d + \gamma + \epsilon) - \varphi(\xi)] d\xi} > 0,$$

which is a contradiction. Therefore, $I(t) > 0$ for all $t \in [0, T)$. \square

The next result addresses the global existence and boundedness of solutions to the model (2).

Proposition 2. *Suppose that (A1) is satisfied. Then, for any $S_0 \geq 0$ and $I_0 \geq 0$ there is at least one solution $(S(t), I(t))$ to the model (2) satisfying $S(0) = S_0$ and $I(0) = I_0$. Furthermore, any such solution is bounded and exists for all $t \in [0, +\infty)$.*

Proof. Note that the map $(S, I) \mapsto (A - dS - \lambda SI, \lambda SI - (d + \gamma + \epsilon)I - \overline{\text{co}}[h(I)])$ is an upper semi-continuous set-valued map with nonempty compact convex values. By the existence theorem of solution of differential inclusion ([17], p.77, Thm.1), there exists a solution $(S(t), I(t))$ of model (2) on $[0, t_0)$ for some $t_0 > 0$ satisfying the initial condition $S(0) = S_0, I(0) = I_0$. By Proposition 1, we know that $S(t) \geq 0$ and $I(t) \geq 0$ for $t \in [0, t_0)$.

From (3), we have

$$\frac{d(S + I)}{dt} \in A - d(S + I) - (\gamma + \epsilon)I - \overline{\text{co}}[h(I)].$$

Choose any $v \in \overline{\text{co}}[h(I)]$. When $S + I > \frac{A}{d}$, we have that $A - d(S + I) - (\gamma + \epsilon)I - v < 0$. Therefore, $0 \leq S + I \leq \max\{S_0 + I_0, \frac{A}{d}\}$, that is, the solution $(S(t), I(t))$ is bounded on $[0, t_0)$. Using the boundedness and by virtue of the continuation theorem ([17], p.78, Thm.2), we concluded that the solution $(S(t), I(t))$ indeed exists on the time interval $[0, +\infty)$ and is bounded. The proof is completed. \square

3. Equilibria and their stability. By an equilibrium of model (2), we mean a constant solution of model (2), $(S(t), I(t)) = (S^*, I^*), t \in [0, +\infty)$. Clearly, (S^*, I^*) is an equilibrium of model (2) if and only if

$$\begin{cases} 0 = A - dS^* - \lambda S^* I^*, \\ 0 \in \lambda S^* I^* - (d + \gamma + \epsilon)I^* - \overline{\text{co}}[h(I^*)]. \end{cases} \quad (6)$$

Thus, if (S^*, I^*) is an equilibrium of model (2), then there exists a constant $\xi^* \in \overline{\text{co}}[h(I^*)]$ such that

$$\begin{cases} A - dS^* - \lambda S^* I^* = 0, \\ \lambda S^* I^* - (d + \gamma + \epsilon) I^* - \xi^* = 0. \end{cases} \quad (7)$$

Obviously, such a constant ξ^* is unique, being given by $\xi^* = S^* I^* - (d + \gamma + \epsilon) I^* \in \overline{\text{co}}[h(I^*)]$.

Suppose that (A1) holds. In order to obtain the equilibria of model (2), we need to solve the following inclusion:

$$\begin{cases} 0 = A - dS - \lambda SI, \\ 0 \in \lambda SI - (d + \gamma + \epsilon) I - \overline{\text{co}}[\varphi(I)]I. \end{cases} \quad (8)$$

Obviously, the disease free equilibrium $E_0 = (A/d, 0)$ always exists. An endemic equilibrium satisfies

$$\begin{cases} 0 = A - dS - \lambda SI, \\ 0 \in \lambda S - (d + \gamma + \epsilon) - \overline{\text{co}}[\varphi(I)]. \end{cases} \quad (9)$$

Solving the first equation of (9) for S in terms of I gives $S = A/(d + \lambda I)$. Substituting this into the second equation (inclusion), we have

$$\frac{A\lambda}{d + \lambda I} - (d + \gamma + \epsilon) \in \overline{\text{co}}[\varphi(I)] = [\varphi(I^-), \varphi(I^+)]. \quad (10)$$

Denote

$$g(I) = \frac{A\lambda}{d + \lambda I} - (d + \gamma + \epsilon),$$

and let

$$\mathcal{R}_0 = \frac{\lambda A}{d(d + \gamma + \epsilon + \varphi(0))}, \quad (11)$$

which is the basic reproduction number of the model (2). We will see in the sequel that the existence of endemic equilibrium is closely related to the size of \mathcal{R}_0 .

Lemma 1. *If $\mathcal{R}_0 > 1$, then inclusion (10) has an unique positive solution \tilde{I} satisfying*

$$\tilde{I} \leq \frac{A\lambda - d(d + \gamma + \epsilon)}{\lambda(d + \gamma + \epsilon)}.$$

Proof. We first show the existence of a positive solution \tilde{I} of inclusion (10). Note that $\mathcal{R}_0 > 1$ implies that $g(0) > \varphi(0) \geq 0$. Also note that $g(I)$ is decreasing in I and $\varphi(I)$ is non-decreasing in I . Moreover, $g(I) \leq 0$ when $I \geq [A\lambda - d(d + \gamma + \epsilon)]/[\lambda(d + \gamma + \epsilon)]$. Thus, the set $\{I : g(I) \geq \varphi(I^+), I > 0\}$ is bounded. Let

$$\tilde{I} = \sup\{I : g(I) \geq \varphi(I^+), I > 0\}.$$

Then, it is obvious that $g(\tilde{I}) \geq \varphi(\tilde{I}^-)$ and $0 < \tilde{I} \leq [A\lambda - d(d + \gamma + \epsilon)]/[\lambda(d + \gamma + \epsilon)]$.

We claim that $g(\tilde{I}) \in [\varphi(\tilde{I}^-), \varphi(\tilde{I}^+)]$. Otherwise, $g(\tilde{I}) > \varphi(\tilde{I}^+) = \lim_{I \rightarrow \tilde{I}^+} \varphi(I)$. By (A1), there exists a $\delta > 0$ such that $g(\tilde{I} + \delta) > \varphi(\tilde{I} + \delta) = \varphi((\tilde{I} + \delta)^+)$. This contradicts the definition of \tilde{I} . Therefore, $g(\tilde{I}) \in [\varphi(\tilde{I}^-), \varphi(\tilde{I}^+)]$, that is, \tilde{I} is a positive solution of the inclusion (10).

We next show that \tilde{I} is the unique positive solution of (10). Set $I_1^* = \tilde{I}$ and assume that $I_2^* \neq I_1^*$ is another positive solution of (10). Then, there exist $\eta_1^* \in \overline{\text{co}}[\varphi(I_1^*)]$ and $\eta_2^* \in \overline{\text{co}}[\varphi(I_2^*)]$ such that

$$\begin{cases} A\lambda = (d + \lambda I_1^*)(\eta_1^* + d + \gamma + \epsilon), \\ A\lambda = (d + \lambda I_2^*)(\eta_2^* + d + \gamma + \epsilon). \end{cases} \quad (12)$$

From the monotonicity of φ (see (A1)), it follows that

$$H = \frac{\eta_1^* - \eta_2^*}{I_1^* - I_2^*} \geq 0.$$

Subtraction of the two equations in (12) results in

$$\begin{aligned} 0 &= d(\eta_1^* - \eta_2^*) + \lambda(d + \gamma + \epsilon)(I_1^* - I_2^*) + \lambda(I_1^*\eta_1^* - I_2^*\eta_2^*) \\ &= d(\eta_1^* - \eta_2^*) + \lambda(d + \gamma + \epsilon)(I_1^* - I_2^*) + \lambda(I_1^*\eta_1^* - I_1^*\eta_2^* + I_1^*\eta_2^* - I_2^*\eta_2^*) \\ &= (d + \lambda I_1^*)(\eta_1^* - \eta_2^*) + \lambda(d + \gamma + \epsilon + \eta_2^*)(I_1^* - I_2^*) \\ &= (d + \lambda I_1^*)H(I_1^* - I_2^*) + \lambda(d + \gamma + \epsilon + \eta_2^*)(I_1^* - I_2^*) \\ &= [(d + \lambda I_1^*)H + \lambda(d + \gamma + \epsilon + \eta_2^*)](I_1^* - I_2^*). \end{aligned}$$

This further leads to $(d + \lambda I_1^*)H + \lambda(d + \gamma + \epsilon + \eta_2^*) = 0$. On the other hand, $I_1^* > 0$ and $\eta_2^* \geq 0$ imply $(d + \lambda I_1^*)H + \lambda(d + \gamma + \epsilon + \eta_2^*) > 0$, which is a contradiction. Therefore, (10) has the unique positive solution \tilde{I} , and the proof of the lemma is completed. \square

A direct consequence of Lemma 1 is the following uniqueness theorem for endemic equilibrium.

Theorem 1. *Suppose that (A1) holds. If $\mathcal{R}_0 > 1$, then model (2) has an unique endemic equilibrium $E^* = (S^*, I^*)$ with $I^* = \tilde{I}$ being the unique positive solution of the inclusion (10) as is shown in Lemma 1 and $S^* = A/(d + \lambda I^*)$.*

By (A1) and Remark 1, we can analyze the stability of the model at the disease free equilibrium E_0 by investigating the eigenvalues of the Jacobian matrix of (2) at E_0 . This matrix is calculated as

$$J_0 = \begin{bmatrix} -d & -\lambda \frac{A}{d} \\ 0 & \lambda \frac{A}{d} - (d + \gamma + \epsilon) - \varphi(0) \end{bmatrix}$$

It is clear that the stability of E_0 is fully determined by the sign of the term $\lambda A/d - (d + \gamma + \epsilon) - \varphi(0)$: E_0 is asymptotically stable if $\lambda A/d - (d + \gamma + \epsilon) - \varphi(0) < 0$; it is unstable if $\lambda A/d - (d + \gamma + \epsilon) - \varphi(0) > 0$. The above stability criteria can be stated in terms of \mathcal{R}_0 .

Theorem 2. *Assume that (A1) holds. Then the disease free equilibrium E_0 is asymptotically stable if $\mathcal{R}_0 < 1$ and it becomes unstable when $\mathcal{R}_0 > 1$.*

Now we turn to the stability of the unique endemic equilibrium $E^* = (S^*, I^*)$ which exists if $\mathcal{R}_0 > 1$. We can show that $\mathcal{R}_0 > 1$ is actually also a necessary condition for the existence of the endemic equilibrium $E^* = (S^*, I^*)$. Indeed, assuming that E^* exists, it follows from (9) that $\lambda A = (\eta^* + d + \gamma + \epsilon)(d + \lambda I^*)$, where $\eta^* \in \overline{\text{co}}[\varphi(I^*)]$. Thus, by the monotonicity of φ , we have

$$\begin{aligned} 0 < I^* &= \frac{d}{\lambda} \left(\frac{\lambda A}{d(d + \gamma + \epsilon + \eta^*)} - 1 \right) \\ &\leq \frac{d}{\lambda} \left(\frac{\lambda A}{d(d + \gamma + \epsilon + \varphi(0))} - 1 \right) \\ &= \frac{d}{\lambda} (\mathcal{R}_0 - 1), \end{aligned}$$

which implies that $\mathcal{R}_0 > 1$. Therefore $\mathcal{R}_0 > 1$ is a sufficient and necessary condition for the existence of the unique endemic equilibrium $E^* = (S^*, I^*)$.

Assume that $\mathcal{R}_0 > 1$ and that φ is differentiable at I^* . Then the Jacobian matrix of (2) at the endemic equilibrium E^* can be calculated as

$$J^* = \begin{bmatrix} -d - \lambda I^* & -\lambda S^* \\ \lambda I^* & -\varphi'(I^*)I^* \end{bmatrix}.$$

Note that

$$\begin{aligned} \text{tr}(J^*) &= -d - \lambda I^* - \varphi'(I^*)I^* < 0, \\ \det(J^*) &= (d + \lambda I^*)\varphi'(I^*)I^* + \lambda^2 S^* I^* > 0. \end{aligned}$$

Based on the above, we have the following theorem.

Theorem 3. *Suppose that (A1) holds. If $\mathcal{R}_0 > 1$ and that φ is differentiable at I^* , then the endemic equilibrium E^* is asymptotically stable.*

The above stability results are local. Moreover, for E^* it is assumed that φ is differentiable at I^* . In the sequel, we will show that E_0 is indeed globally asymptotically stable when $\mathcal{R}_0 \leq 1$; and E^* is globally asymptotically stable when $\mathcal{R}_0 > 1$, regardless of whether or not φ is differentiable at I^* . To this end, we need to apply the Lyapunov theory for discontinuous systems (see, e.g., [7, 8]). We begin by introducing a LaSalle-type invariance principle.

Consider a system described by the following differential inclusion

$$\dot{x}(t) \in F(x), \tag{13}$$

where F is an upper semi-continuous set-valued map from \mathbb{R}^n to \mathbb{R}^n with compact and convex values. We also assume $0 \in F(0)$, that is, 0 is an equilibrium of (13).

A Lyapunov function for (13) is a smooth function $V : \mathbb{R}^n \rightarrow \mathbb{R}$ satisfying the following conditions:

- (L1) Positive Definiteness: $V(x) > 0$ for all $x \neq 0$; in addition, $V(0) = 0$.
- (L2) Properness: the sublevel set $\{x \in \mathbb{R}^n : V(x) \leq a\}$ is bounded for every $a \geq 0$;
- (L3) Strong Infinitesimal Decrease:

$$\max_{v \in F(x)} \langle \nabla V(x), v \rangle \leq 0, \quad \forall x \neq 0.$$

A set W is said to be weakly invariant for (13) if for any $x_0 \in W$, there is at least one solution $x(t)$ satisfying $x(0) = x_0$ such that $x(t) \in W$ for all t at which $x(t)$ exists.

Let V be a Lyapunov function for (13). For any $l > 0$, by (L1) and the continuity of V , the level set $\{x \in \mathbb{R}^n : V(x) \leq l\}$ contains a neighborhood of 0. Denote by V_l the largest connected component of the level set $\{x \in \mathbb{R}^n : V(x) \leq l\}$ that contains 0. The following LaSalle-type invariance principle is from Theorem 3 in [8].

Lemma 2. *Assume that $V : \mathbb{R}^n \rightarrow \mathbb{R}$ is a Lyapunov function for (13) and let*

$$Z_V = \{x \in \mathbb{R}^n : \exists v \in F(x), \langle \nabla V(x), v \rangle = 0\}.$$

Denote by M the largest weakly invariant subset of $\overline{Z_V} \cap L_l$. Let $x_0 \in L_l$ and $x(t)$ be any solution with $x(0) = x_0$. Then $\text{dist}(x(t), M) \rightarrow 0$ as $t \rightarrow +\infty$. In particular, if $M = \{0\}$ and $l = +\infty$, then 0 is globally asymptotically stable for (13).

Now, we are in the position to state and prove the following global stability result for the disease free equilibrium.

Theorem 4. *Suppose that (A1) is satisfied. If $\mathcal{R}_0 \leq 1$, then the disease free equilibrium E_0 is globally asymptotically stable.*

Proof. In order to apply Lemma 2, we shift the disease free equilibrium E_0 to the origin by letting $x = S - \frac{A}{d}$. Then, (3) is transformed to the following form:

$$\begin{cases} \frac{dx}{dt} = -dx - \lambda xI - \frac{\lambda A}{d}I, \\ \frac{dI}{dt} \in \lambda xI + [\frac{\lambda A}{d} - (d + \gamma + \epsilon)]I - \overline{\text{co}}[\varphi(I)]I, \end{cases} \quad (14)$$

Let

$$V_1(x, I) = \frac{x^2}{2} + \frac{A}{d}I,$$

which is obviously a smooth function. It is easy to verify that (L1) and (L2) are satisfied for V_1 . Denote the right hand side of (14) by $G(x, I)$, that is,

$$G(x, I) = \begin{pmatrix} -dx - \lambda xI - \frac{\lambda A}{d}I \\ \lambda xI + [\frac{\lambda A}{d} - (d + \gamma + \epsilon)]I - \overline{\text{co}}[\varphi(I)]I \end{pmatrix}.$$

By (A1), it is easy to see that the map G is an upper semi-continuous set-valued map with non-empty compact convex values. For any $v = (v_1, v_2) \in G(x, I)$, there exists a corresponding function $\eta(t) \in \overline{\text{co}}[\varphi(I)]$ such that

$$v = \begin{pmatrix} -dx - \lambda xI - \frac{\lambda A}{d}I \\ \lambda xI + [\frac{\lambda A}{d} - (d + \gamma + \epsilon)]I - \eta(t)I \end{pmatrix}.$$

From this, we can calculate $\nabla V_1(x, I) \cdot v$ as below

$$\begin{aligned} \nabla V_1(x, I) \cdot v &= \left(x, \frac{A}{d}\right) \cdot \begin{pmatrix} -dx - \lambda xI - \frac{\lambda A}{d}I \\ \lambda xI + [\frac{\lambda A}{d} - (d + \gamma + \epsilon)]I - \eta(t)I \end{pmatrix} \\ &= -dx^2 - \lambda x^2I - \frac{A}{d}[d + \gamma + \epsilon + \eta(t) - \frac{\lambda A}{d}]I. \end{aligned}$$

When $\mathcal{R}_0 \leq 1$, by the monotonicity of φ , we have $d + \gamma + \epsilon + \eta(t) - \lambda A/d \geq d + \gamma + \epsilon + \varphi(0) - \lambda A/d \geq 0$ and hence $\nabla V_1(x, I) \cdot v \leq 0$. This verifies (L3), and hence, V_1 is a Lyapunov function for (14).

Furthermore, when $\mathcal{R}_0 < 1$, then

$$Z_{V_1} = \{(x, I) \in \mathbb{R}^2 : \exists v \in G(x, I), \langle \nabla V(x, I), v \rangle = 0\} = \{(0, 0)\}.$$

When $\mathcal{R}_0 = 1$, then $Z_{V_1} = \{(0, 0)\} \cup \{(0, I) : \eta(t) = \varphi(0), I \neq 0\}$. Note that if $x \equiv 0$, it follows from the first equation of (14) that $I = 0$. Therefore, for any $l > 0$ the largest weakly invariant subset of $\overline{Z_{V_1}} \cap L_l$ is the singleton $M = \{(0, 0)\}$. By Lemma 2, (0, 0) is globally asymptotically stable for (14) if $\mathcal{R}_0 \leq 1$; that is, E_0 is globally asymptotically stable for (2) if $\mathcal{R}_0 \leq 1$, and the proof is completed. \square

The following theorem deals with the global asymptotic stability of the endemic equilibrium E^* when $\mathcal{R}_0 > 1$.

Theorem 5. *Suppose that (A1) is satisfied. If $\mathcal{R}_0 > 1$, then model (2) has an unique endemic equilibrium $E^* = (S^*, I^*)$ which is globally asymptotically stable.*

Proof. The existence and uniqueness of E^* have been confirmed in Theorem 1. We now prove the globally asymptotic stability of E^* .

Let $x = S - S^*$ and $y = I - I^*$. Then, (3) is transformed to

$$\begin{cases} \frac{dx}{dt} = -dx - \lambda x(I^* + y) - (d + \lambda + \epsilon + \eta^*)y, \\ \frac{dy}{dt} \in \lambda x(I^* + y) + (\eta^* - \overline{\text{co}}[\varphi(I^* + y)])(I^* + y), \end{cases} \quad (15)$$

where $\eta^* = \lambda A / (d + \lambda I^*) - (d + \gamma + \epsilon) \in \overline{\text{co}}[\varphi(I^*)]$.

Consider the function

$$V_2(x, y) = \frac{x^2}{2} + \frac{d + \lambda + \epsilon + \eta^*}{\lambda} \left(y - I^* \ln \frac{I^* + y}{I^*} \right).$$

This is a smooth function with respect to (x, y) . It is easy to verify that (L1) and (L2) are satisfied.

Denote

$$H(x, y) = \left(\begin{array}{c} -dx - \lambda x(I^* + y) - (d + \lambda + \epsilon + \eta^*)y \\ \lambda x(I^* + y) + (\eta^* - \overline{c\phi}[\varphi(I^* + y)])(I^* + y) \end{array} \right).$$

It is easy to see that the map $H(x, y)$ is an upper semi-continuous set-valued map with nonempty compact convex values. For any $v = (v_1, v_2) \in H(x, y)$, there exists a corresponding function $\eta(t) \in \overline{c\phi}[\varphi(I^* + y)]$ such that

$$v = \left(\begin{array}{c} -dx - \lambda x(I^* + y) - (d + \lambda + \epsilon + \eta^*)y \\ \lambda x(I^* + y) + (\eta^* - \eta(t))(I^* + y) \end{array} \right).$$

The gradient of $V_2(x, y)$ is given by

$$\nabla V_2(x, y) = \left(x, \frac{d + \lambda + \epsilon + \eta^*}{\lambda} \frac{y}{I^* + y} \right).$$

Thus,

$$\begin{aligned} & \nabla V_2(x, y) \cdot v \\ &= \left(x, \frac{d + \lambda + \epsilon + \eta^*}{\lambda} \frac{y}{I^* + y} \right) \cdot \left(\begin{array}{c} -dx - \lambda x(I^* + y) - (d + \lambda + \epsilon + \eta^*)y \\ \lambda x(I^* + y) + (\eta^* - \eta(t))(I^* + y) \end{array} \right) \\ &= -dx^2 - \lambda x^2(I^* + y) - \frac{d + \lambda + \epsilon + \eta^*}{\lambda} (\eta(t) - \eta^*)y. \end{aligned}$$

The monotonicity of φ implies that $(\eta(t) - \eta^*)y \geq 0$, and hence,

$$dx^2 + \lambda x^2(I^* + y) + \frac{d + \lambda + \epsilon + \eta^*}{\lambda} (\eta(t) - \eta^*)y \geq 0.$$

That is $\nabla V_2(x, I) \cdot v \leq 0$ verifying (L3). Thus, V_2 is a Lyapunov function for (15).

Letting $\nabla V_2(x, y) \cdot v = 0$, we can obtain $Z_{V_2} = \{(0, 0)\} \cup \{(0, y) : \eta(t) = \eta^*, y \neq 0\}$. If $x \equiv 0$, then by the first equation of (15) we obtain $y = 0$. Therefore, for any $l > 0$ the largest weakly invariant subset of $\overline{Z_{V_2}} \cap L_l$ for (15) is $M = \{(0, 0)\}$. By Lemma 2, $(0, 0)$ is globally asymptotically stable for (15); that is, E^* is globally asymptotically stable for (2). The proof is completed. \square

4. Global convergence in finite time. One important feature of discontinuous ODE systems that a smooth ODE system can not have, is that *convergence to equilibrium in finite time* is possible under some conditions. This topic has been particularly explored recently by Forti et al [18] for discontinuous neural network models. Motivated by this, in the sequel, we investigate the possibility of convergence to equilibrium in finite time for our model (2). To this end, we need to apply non-smooth Lyapunov functions as was done in Forti et. al. [18], which requires a generalization of the notion of gradient.

A function $f : \mathbb{R}^n \rightarrow \mathbb{R}$ is said to be regular at x if the following hold: (i) it is locally Lipschitz near x ; (ii) for all direction $v \in \mathbb{R}^n$, there exists the usual one-sided directional derivative

$$f'(x, v) = \lim_{\rho \rightarrow 0^+} \frac{f(x + \rho v) - f(x)}{\rho}$$

and $f'(x, v) = f^o(x, v)$, where

$$f^o(x, v) = \limsup_{y \rightarrow x, \rho \rightarrow 0^+} \frac{f(y + \rho v) - f(y)}{\rho}$$

is the generalized directional derivative of f at x in the direction of v . A function f is said to be regular in \mathbb{R}^n , if it is regular at every $x \in \mathbb{R}^n$.

Let $f : \mathbb{R}^n \rightarrow \mathbb{R}$ be locally Lipschitz in \mathbb{R}^n . Then, by Rademacher Theorem, f is differentiable at almost all (a.a.) $x \in \mathbb{R}^n$ in the sense of Lebesgue measure (see, e.g., [14, 18]). For such a function, the Clarke Generalized Gradient (see [14]), denoted by ∂f , is defined by

$$\partial f(x) = \overline{\text{co}} \left\{ \lim_{i \rightarrow +\infty} \nabla f(x_i) : x_i \rightarrow x, x_i \notin \Omega_f \right\},$$

where Ω_f is the set of measure zero in which the gradient of f is not differentiable.

Lemma 3. (Chain Rule [13, 18]) *If $f(x) : \mathbb{R}^n \rightarrow \mathbb{R}$ is regular, and $x(t) : [0, +\infty) \rightarrow \mathbb{R}^n$ is absolutely continuous on any compact subinterval of $[0, +\infty)$, then $x(t)$ and $f(x(t)) : [0, +\infty) \rightarrow \mathbb{R}$ are differentiable for a.a $t \in [0, +\infty)$ and*

$$\frac{d}{dt} f(x(t)) = \langle \gamma(t), \dot{x}(t) \rangle, \quad \forall \gamma(t) \in \partial f(x(t)).$$

We start with the endemic equilibrium $E^* = (S^*, I^*)$. If $\varphi(I)$ is continuous at I^* , then the full model system (2) is continuous at the neighborhood of E^* , and hence, it is unlikely for the system to allow convergence to E^* in finite time. This observation motivates the following assumption:

(A2) Assume that $\mathcal{R}_0 > 1$ and that $\varphi(I)$ has an jump discontinuity at I^* where I^* is the unique positive solution of (10). Moreover, $\eta^* = \lambda S^* - (d + \gamma + \epsilon) \in (\varphi(I^{*-}), \varphi(I^{*+}))$ where $S^* = A/(d + \lambda I^*)$.

If ϕ does not have a jump discontinuity at I^* , then the solution can only reach equilibrium in infinite time, because once it gets sufficiently close to I^* , the discontinuities of ϕ will never again be encountered and so the subsequent evolution is effectively governed by a classical system.

Under (A2), $\delta := \min\{\varphi(I^{*+}) - \eta^*, \eta^* - \varphi(I^{*-})\} > 0$, and we have the following theorem confirming *global convergence to E^* in finite time*.

Theorem 6. *Suppose that (A1) and (A2) are satisfied. Then every solution of model (2) with initial condition $S(0) = S_0 \geq 0$ and $I(0) = I_0 > 0$ converges to E^* in finite time. More precisely, $(S(t), I(t)) = (S^*, I^*)$ for all*

$$t \geq t^* = \frac{\lambda^2 B(S_0, I_0)}{d\delta^2}, \quad (16)$$

where

$$\begin{aligned} B(S_0, I_0) &= \frac{(S_0 - S^*)^2}{2} + \frac{d + \lambda + \epsilon + \eta^*}{\lambda} (I_0 - I^* - I^* \ln \frac{I_0}{I^*}) \\ &\quad + 2d/\lambda^2 \int_0^{I_0 - I^*} \frac{\varphi(\rho + I^*) - \eta^*}{\rho + I^*} d\rho. \end{aligned}$$

Proof. Let $x(t) = S(t) - S^*$ and $y(t) = I(t) - I^*$. The by (15), we know that there exists a measurable function $\eta(t) \in \overline{\text{co}}[\varphi(I^* + y(t))]$ such that

$$\begin{cases} \frac{dx}{dt} = -dx - \lambda x(I^* + y) - (d + \lambda + \epsilon + \eta^*)y, \\ \frac{dy}{dt} = \lambda x(I^* + y) + (\eta^* - \eta(t))(I^* + y). \end{cases} \quad (17)$$

Construct the following Lyapunov function

$$V_3(x, y) = \frac{x^2}{2} + \frac{d + \lambda + \epsilon + \eta^*}{\lambda} \left(y - I^* \ln \frac{I^* + y}{I^*} \right) + \alpha \int_0^y \frac{\varphi(I^* + \rho) - \eta^*}{I^* + \rho} d\rho, \quad (18)$$

where α is a positive constant to be specified later. It can be easily verified that $V_3(x, y)$ is a regular function in (x, y) . Moreover, $V_3(x, y) > 0$ for $(x, y) \neq 0$, $V_3(0, 0) = 0$, and $V_3(x, y) \rightarrow +\infty$ as $x \rightarrow +\infty$ or $y \rightarrow +\infty$. Note that

$$\partial V_3(x, y) = x + \frac{d + \lambda + \epsilon + \eta^*}{\lambda} \frac{y}{I^* + y} + \alpha \frac{\overline{\text{co}}[\varphi(I^* + y)] - \eta^*}{I^* + y}.$$

By Lemma 3, we know that for a.a. $t \geq 0$,

$$\frac{dV_3(x(t), y(t))}{dt} = \langle \xi(t), (\dot{x}(t), \dot{y}(t)) \rangle, \quad \forall \xi(t) \in \partial V_3(x(t), y(t)).$$

In particular, for

$$\xi(t) = x(t) + \frac{d + \lambda + \epsilon + \eta^*}{\lambda} \frac{y(t)}{I^* + y(t)} + \alpha \frac{\eta(t) - \eta^*}{I^* + y(t)} \in \partial V_3(x(t), y(t)),$$

we obtain

$$\begin{aligned} \frac{dV_3(x(t), y(t))}{dt} &= -dx^2 - \lambda x^2 (I^* + y) - \frac{d + \lambda + \epsilon + \eta^*}{\lambda} (\eta(t) - \eta^*) y \\ &\quad + \alpha \lambda x (\eta(t) - \eta^*) - \alpha (\eta(t) - \eta^*)^2 \\ &\leq -dx^2 + \alpha \lambda x (\eta(t) - \eta^*) - \alpha (\eta(t) - \eta^*)^2 \\ &\leq -d \left(x - \frac{\alpha \lambda}{2d} (\eta(t) - \eta^*) \right)^2 - \frac{4d\alpha - \lambda^2 \alpha^2}{4d} (\eta(t) - \eta^*)^2 \\ &\leq -\frac{4d\alpha - \lambda^2 \alpha^2}{4d} (\eta(t) - \eta^*)^2. \end{aligned}$$

Choose $\alpha > 0$ sufficiently small such that $4d - \lambda^2 \alpha > 0$. It follows from (A2) that $[\eta(t) - \eta^*]^2 \geq \delta^2$ if $(x(t), y(t)) \neq (0, 0)$. Therefore, for almost all $t \in \{t : (x(t), y(t)) \neq (0, 0)\}$,

$$\frac{dV_3(x(t), y(t))}{dt} \leq -\delta^2 \frac{4d\alpha - \lambda^2 \alpha^2}{4d}.$$

Integrating both sides of the above inequality from 0 to t , we obtain

$$0 \leq V_3(x(t), y(t)) \leq V_3(x(0), y(0)) - \delta^2 \frac{4d\alpha - \lambda^2 \alpha^2}{4d} t.$$

This implies that $V_3(x(t), y(t))$ reaches 0 at $t = t^*$ where

$$t^* = \frac{4dV_3(S_0, I_0)}{\delta^2(4d\alpha - \lambda^2 \alpha^2)},$$

and retains 0 after t^* as well (see [18] for a detailed argument of this type). Thus we have proved that $(x(t), I(t)) = 0$ for $t \geq t^*$, or equivalently, $(S(t), I(t)) = (S^*, I^*)$ for $t \geq t^*$.

Note that the above argument is valid for all $\alpha \in (0, 4d/\lambda^2)$. Choosing $\alpha = 2d/\lambda^2$ at which the term $4d\alpha - \lambda^2 \alpha^2$ attains its maximum value $4d^2/\lambda^2$, one obtains the value

$$t^* = \frac{\lambda^2 V_3(S_0, I_0)}{d\delta^2} = \frac{\lambda^2 B(S_0, I_0)}{d\delta^2}.$$

The proof is completed. \square

Notice that if $\phi(I^{*+}) \rightarrow \phi(I^{*-})$, then $t^* \rightarrow \infty$, because $\delta = 0$ for a continuous ϕ and so we must have $\delta \rightarrow 0$ as the discontinuous jump is closed.

A more meaningful and desirable situation is the global convergence to the disease free equilibrium E_0 in finite time. Since (A1) assumes continuity of the treatment function $h(I)$ at $I = 0$, finite time convergence to E_0 is impossible under (A1). Thus, discontinuity is required for $h(I)$ at $I = 0$, as is stated in the following assumption:

(A3) $h : \mathbb{R}_+ \rightarrow \mathbb{R}_+$ is non-decreasing and has at most a finite number of jump discontinuities in every compact interval. Moreover, $h(0) = 0$ and $h(I)$ is discontinuous at $I = 0$.

A typical treatment function satisfying (A3) is the following

$$h(I) = \begin{cases} 0, & I = 0; \\ r, & I > 0. \end{cases}$$

This corresponds to an immediate response to the occurrence of a disease with the constant r being an effort strength.

Under (A3), by (7), we know that $(A/d, 0)$ is the disease free equilibrium of model (2). Let $x = S - A/d$. Then, (3) is transformed to

$$\begin{cases} \frac{dx}{dt} = -dx - \lambda x I - \frac{\lambda A}{d} I, \\ \frac{dI}{dt} \in \lambda x I + [\frac{\lambda A}{d} - (d + \gamma + \epsilon)]I - \overline{co}[h(I)], \end{cases} \quad (19)$$

From (4), there exists a measurable function $\eta \in \overline{co}[h(I)]$ corresponding to $(x(t), I(t))$ such that

$$\begin{cases} \frac{dx}{dt} = -dx - \lambda x I - \frac{\lambda A}{d} I, \\ \frac{dI}{dt} = \lambda x I + [\frac{\lambda A}{d} - (d + \gamma + \epsilon)]I - \eta(t), \end{cases} \quad \text{for a.a. } t \in [0, +\infty), \quad (20)$$

Theorem 7. *Suppose that (A3) holds. If $d + \gamma + \epsilon - \lambda A/d \geq 0$, then every solution of model (2) with initial condition $S(0) = S_0 \geq 0$ and $I(0) = I_0 \geq 0$ converges to E_0 in finite time, i.e. the disease dies out in finite time. More precisely, $(S(t), I(t)) = (A/d, 0)$ for*

$$t \geq t^* = \frac{dQ(S_0, I_0)}{Ah(0^+)}, \quad (21)$$

where

$$Q(S_0, I_0) = \frac{(S_0 - \frac{A}{d})^2}{2} + \frac{A}{d} I_0.$$

Proof. Let $V_1(x, I)$ be the same Lyapunov function as in the proof of Theorem 4. Then, evaluating $\dot{V}_1(x(t), I(t))$ along the system (19) gives

$$\begin{aligned} \frac{dV_1(x(t), I(t))}{dt} &= x(-dx - \lambda x I - \frac{\lambda A}{d} I) + \frac{A}{d}(\lambda x I + [\frac{\lambda A}{d} - (d + \gamma + \epsilon)]I - \eta(t)) \\ &\leq -dx^2 - \lambda x^2 I - \frac{A}{d}(d + \gamma + \epsilon - \frac{\lambda A}{d})I - \frac{A}{d}\eta(t). \end{aligned}$$

By (A3), we know that $\eta(t) \geq h(0^+)$. Note that $d + \gamma + \epsilon - \frac{\lambda A}{d} \geq 0$. Then, we have

$$\frac{dV_1(x(t), I(t))}{dt} \leq \frac{-Ah(0^+)}{d}.$$

Integrating both sides of the above inequality from 0 to t , we obtain

$$0 \leq V_1(x(t), I(t)) \leq V_1(x(0), I(0)) - \frac{Ah(0^+)}{d}t = Q(S_0, I_0) - \frac{Ah(0^+)}{d}t.$$

This implies that $V_1(x(t), I(t)) = 0$ for $t \geq t^*$ which means $(x(t), I(t)) = (0, 0)$ and hence $(S(t), I(t)) = (A/d, 0)$ for $t \geq t^*$. The theorem is proved. \square

Remark 3. Under (A3), the basic reproduction number of the model (2) is given by

$$\mathcal{R}_0 = \frac{\lambda A}{d(d + \gamma + \epsilon)}$$

and, thus condition $d + \gamma + \epsilon - \lambda A/d \geq 0$ is equivalent to $\mathcal{R}_0 \leq 1$.

5. Conclusion and discussion. We have revisited the SIR model with treatment considered by [21]. But unlike in [21] where treatment function is assumed to be continuous, our main concern here is the impact of the adoption of a discontinuous treatment function.

Our results on the disease free equilibrium E_0 show that when the basic reproduction number \mathcal{R}_0 of the model is less than one, as is expected, the disease free equilibrium is globally asymptotically stable. Note that under assumption (A1), \mathcal{R}_0 depends on $\varphi(0)$ by (11), which is decreasing function of $\varphi(0)$ (the initial treatment rate). Thus, a larger initial treatment rate will help eliminate the disease, and the formula (11) determines how large $\varphi(0)$ should be.

Under (A1) and when $\mathcal{R}_0 > 1$, the disease free equilibrium becomes unstable. However, the existence, uniqueness and global asymptotic stability of an endemic equilibrium all follow, regardless of whether I^* is a continuous or discontinuous point of $\varphi(I)$.

What we believe is most interesting and most novel in this paper are the results on the convergence to an equilibrium in *finite time*. This is impossible if a smooth treatment function is adopted. Therefore it presents a true advantage of discontinuous treatments. We are also able to establish an estimation of the precise time (finite) it takes for a solution to settle to the equilibrium. This is particularly important and useful for designing treatment strategies aiming to eliminate the disease in finite time. From the expressions (16) and (21), one may easily see how the model parameters as well as the initial values and the initial treatment strength will affect the (finite) time it takes to eradicate the disease. Taking (21) as an example, we find that t^* is increasing in the magnitude of the initial infectious population I_0 and decreasing in the initial treatment strength $h(0^+)$, which are all reasonable and natural. Since most, if not all, existing works on disease models with treatments assume continuous treatment functions, our results here on the finite time convergence suggest that it should be worthwhile to reconsider those models by incorporating discontinuous treatment functions.

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REFERENCES

- [1] M. E. Alexander, C. Bowman, S. M. Moghadas, R. Summers, A. B. Gumel and B. M. Sahai, *A vaccination model for transmission dynamics of influenza*, SIAM J. Appl. Dyn. Syst., **3** (2004), 503–524.
- [2] M. E. Alexander, S. M. Moghadas, P. Rohani and A. R. Summers, *Modelling the effect of a booster vaccination on disease epidemiology*, J. Math. Biol., **52** (2006), 290–306.

- [3] M. E. Alexander, S. M. Moghadas, G. Röst and J. Wu, *A delay differential model for pandemic influenza with antiviral treatment*, Bull. Math. Biol., **70** (2008), 382–397.
- [4] R. M. Anderson and R. M. May, “Infectious Diseases of Humans, Dynamics and Control,” Oxford University, Oxford, 1991.
- [5] J. Arino, C. McCluskey and P. van den Driessche, *Global results for an epidemic model with vaccination that exhibits backward bifurcation*, SIAM J. Appl. Math., **64** (2003), 260–276.
- [6] J. Arino, R. Jordan and P. van den Driessche, *Quarantine in a multi-species epidemic model with spatial dynamics*, Math. Biosci., **206** (2007), 46–60.
- [7] J.-P. Aubin and A. Cellina, “Differential Inclusions. Set-Valued Maps and Viability Theory,” Grundlehren der Mathematischen Wissenschaften [Fundamental Principles of Mathematical Sciences], **264**, Springer-Verlag, Berlin, 1984.
- [8] A. Baciotti and F. Ceragioli, *Stability and stabilization of discontinuous systems and non-smooth Lyapunov function*, ESAIM Control Optim. Calc. Var., **4** (1999), 361–376.
- [9] F. Brauer, *Backward bifurcations in simple vaccination models*, J. Math. Anal. Appl., **298** (2004), 418–431.
- [10] F. Brauer, *Epidemic models with heterogeneous mixing and treatment*, Bull. Math. Biol., **70** (2008), 1869–1885.
- [11] F. Brauer, P. van den Driessche and J. Wu, eds., “Mathematical Epidemiology,” Lecture Notes in Mathematics, **1945**, Mathematical Biosciences Subseries, Springer-Verlag, Berlin, 2008.
- [12] C. Castillo-Chavez and Z. Feng, *To treat or not to treat: The case of tuberculosis*, J. Math. Biol., **35** (1997), 629–656.
- [13] F. Ceragioli, “Discontinuous Ordinary Differential Equations and Stabilization,” Università di Firenze, 2000.
- [14] F. H. Clarke, “Optimization and Non-Smooth Analysis,” Wiley, New York, 1983.
- [15] Z. Feng, *Final and peak epidemic sizes for SEIR models with quarantine and isolation*, Math. Biosci. Eng., **4** (2007), 675–686.
- [16] Z. Feng and H. R. Thieme, *Recurrent outbreaks of childhood diseases revisited: The impact of isolation*, Math. Biosci., **128** (1995), 93–130.
- [17] A. F. Filippov, “Differential Equations with Discontinuous Righthand Sides,” Translated from the Russian, Mathematics and its Applications (Soviet Series), **18**, Kluwer Academic Publishers Group, Dordrecht, 1988.
- [18] M. Forti, M. Grazzini, P. Nistri and L. Pancioni, *Generalized Lyapunov approach for convergence of neural networks with discontinuous or non-Lipschitz activations*, Phys. D, **214** (2006), 88–99.
- [19] J. M. Hyman and J. Li, *Modeling the effectiveness of isolation strategies in preventing STD epidemics*, SIAM J. Appl. Math., **58** (1998), 912–925.
- [20] M. Nuño, Z. Feng, M. Martcheva and C. Castillo-Chavez, *Dynamics of two-strain influenza with isolation and partial cross-immunity*, SIAM J. Appl. Math., **65** (2005), 964–982.
- [21] W. Wang, *Backward bifurcation of an epidemic model with treatment*, Math. Biosci., **201** (2006), 58–71.
- [22] L. Wu and Z. Feng, *Homoclinic bifurcation in an SIQR model for childhood diseases*, J. Differ. Equat., **168** (2000), 150–167.
- [23] X. Zhang and X. Liu, *Backward bifurcation and global dynamics of an SIS epidemic model with general incidence rate and treatment*, Nonl. Anal. RWA, **10** (2009), 565–575.

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