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EPIDEMIC MODELS WITH NONLINEAR INFECTION FORCES

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Abstract. Epidemic models with behavior changes are studied to consider effects of protection measures and intervention policies. It is found that intervention strategies decrease endemic levels and tend to make the dynamical behavior of a disease evolution simpler. For a saturated infection force, the model may admit a stable disease-free equilibrium and a stable endemic equilibrium at the same time. If we vary a recovery rate, numerical simulations show that the boundaries of the region for the persistence of the disease undergo the changes from the separatrix of a saddle to an unstable limit cycle. If the inhibition effect from behavior changes is weak, we find two limit cycles and obtain bifurcations of the model as the population size changes. We also find that the disease may die out although there are two endemic equilibria.

1. **Introduction.** Many classical epidemic models admit threshold dynamics. If a basic reproduction number R_0 is below 1, a disease-free equilibrium is globally stable. If it is above 1, an endemic equilibrium is globally stable (see, for example, [2, 11, 16, 1, 18]). This means that the disease dies out if $R_0 < 1$ and persists if $R_0 > 1$. Further, the models do not have a limit cycle. However, Capasso and Wilson [4] pointed out that a bistable case is more likely to occur. Here, bistability means a disease-free equilibrium and an endemic equilibrium are stable at the same time. Indeed, for many diseases, long time behavior of disease transmission is related to initial positions. If the initial value of infective numbers is large, which means we have a large invasion of a disease, the disease will be persistent. If the initial value of infective numbers is small, which corresponds to a small invasion of a disease, the disease will be extinct. Furthermore, periodic oscillations have been observed in the incidence of many infectious diseases, including measles, mumps, rubella, chickenpox, and influenza. In some locations the incidence of some diseases, such as chickenpox, mumps, and poliomyelitis, goes up and down every year (see Hethcote and Levin [8]).

Why is it that classical epidemic models cannot explain these important phenomena? The first thing we should consider is the modification of incidence rates since the processes of disease transmissions are most directly described by these rates. In classical epidemic models, mass action incidence and standard incidence are frequently used. These incidences imply that contact rates and infection probability per contact are constant in time. In fact, the mobility of individuals is likely influenced by the number of infective individuals, because this number represents an infection risk. For instance, during the SARS outbreak in 2003, when

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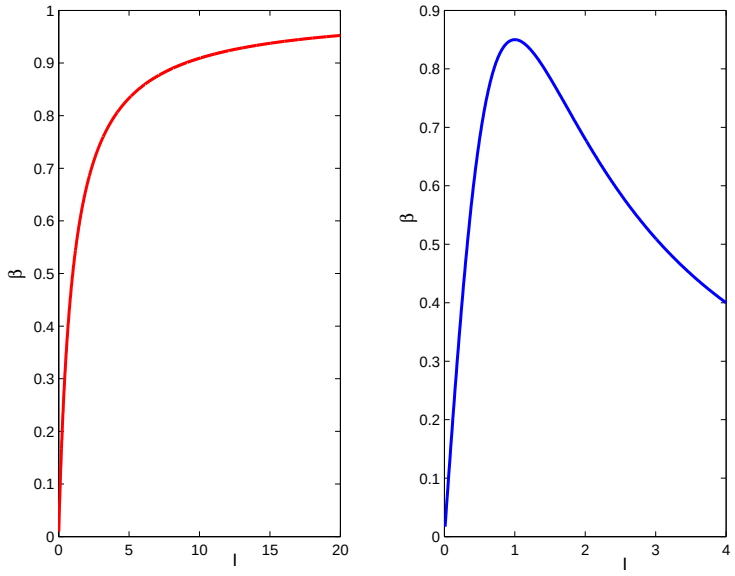

FIGURE 1. Two typical infection forces.

the number of SARS infectives was increasing, we took protection measures and control policies: closing schools, closing restaurants, postponing conferences, etc. So, contact numbers per unit time were greatly reduced, and therefore, the incidence was decreased (see Wang and Ruan [17]). This means that it is meaningful to consider the infection forces that include the adaptation of individuals to infection risks. Indeed, Hyman and Li [7] studied a sexually-transmitted-disease model in which people modify their behavior to reduce the probability of infection with individuals in highly infected groups through either reduced contacts, reduced partner formations, or the practice of safe sex. Furthermore, some epidemic diseases need multiple contacts to have a valid disease transmission. This may lead to an infection force which is a nonlinear function of infective numbers.

Liu, Levin, Hethcote and Iwasa [9, 10] proposed the incidence rate $\beta I^2 S$ and discovered that an SIR model admits bistable equilibria, saddle-node bifurcation and Hopf bifurcation if S and I are the densities of susceptible individuals and infectious individuals, respectively. Lizana and Rivero [12] studied the model further and found homoclinic bifurcations.

Capasso and Serio [3] proposed more general infection forces which are shown in Figure 1. The infection force on the left side, which is a saturated curve, describes "crowding effect" or "protection measures." Indeed, effective contacts between infectious individuals and susceptible individuals cannot grow quickly when there are many infectious individuals because of the crowding of infective individuals or because of protection measures by susceptible individuals. The infection force for the right side describes the effect of "intervention policy." When I is large, we perform intervention policies, for example, closing schools, restaurants and postponing conferences. So, the infection force decreases quickly at the high infection level. If the infection force is fixed as $aI^2/(b+I^2)$, which corresponds a saturated infection

force, rich dynamical behaviors were found in an $SIRS$ model by Ruan and Wang in [14].

Since intervention policies are important strategies to control epidemic diseases, we study the influence of a nonmonotonic infection force, i.e., the infection force on the right side in Figure 1, on a disease spread. Further, we show that the rich dynamical behavior of a disease evolution can be induced by a saturated infection force.

The remainder of this paper is organized as follows. In the next section, we consider an epidemic model with intervention strategies. Section 3 studies the model with a saturated infection force. In section 4, we analyze the model with a quadratic infection force. The paper concludes with a brief discussion in section 5.

2. **Infection Force under Intervention Policy.** Let us consider an epidemic disease of SIRS type. Denote the number of susceptible individuals by S, the number of infectious individuals by I, the number of recovered individuals by R, and the population size by N, which means $N = S + I + R$. We suppose that the dynamics of the disease transmission is governed by

$$\begin{aligned} \frac{dS}{dt} &= dN - dS - \beta(I)S + \nu R, \\ \frac{dI}{dt} &= \beta(I)S - (d+\gamma)I, \\ \frac{dR}{dt} &= \gamma I - (d+\nu)R, \end{aligned} \tag{2.1}$$

where d is the birth rate and death rate of the population, γ is the recovery rate of infective individuals, ν is the rate of removed individuals who lose immunity and return to susceptible class.

For simplicity in notations, we suppose that the infection force $\beta(I)$ can be factorized into $\lambda I/f(I)$, where $1/f(I)$ represents the effect of intervention policies on the reduction of valid contact coefficient λ. Then (2.1) becomes

$$\begin{aligned} \frac{dS}{dt} &= dN - dS - \frac{\lambda I}{f(I)}S + \nu R, \\ \frac{dI}{dt} &= \frac{\lambda I}{f(I)}S - (d+\gamma)I, \\ \frac{dR}{dt} &= \gamma I - (d+\nu)R. \end{aligned} \tag{2.2}$$

Since the population size is a constant C, it suffices to consider

$$\begin{aligned} \frac{dI}{dt} &= \frac{\lambda I}{f(I)}(C - I - R) - (d+\gamma)I, \\ \frac{dR}{dt} &= \gamma I - (d+\nu)R. \end{aligned} \tag{2.3}$$

To ensure a nonmonotonic infection force, we make the following assumptions

(H1) $f(0) > 0$ and $f'(I) > 0$ for $I > 0$;
(H2) there is a $\xi > 0$ such that $(I/f(I))' > 0$ for $0 < I < \xi$ and $(I/f(I))' < 0$ for $I > \xi$.

$E_0 = (0, 0)$ is a disease-free equilibrium of (2.3). By the formulae of [15], we can see that the basic reproduction number of (2.3) is $R_0 = C\lambda/(f(0)(d+\gamma))$. The

Jacobian matrix of (2.3) at E_0 is

$$\begin{bmatrix} \frac{\lambda C}{f(0)} - d - \gamma & 0 \\ \gamma & -d - \nu \end{bmatrix}.$$

It follows that E_0 is asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

An endemic equilibrium of (2.3) is a positive solution of the following system:

$$\begin{cases} R = C - I - \dfrac{d+\gamma}{\lambda} f(I), \\ R = \dfrac{\gamma}{d+\nu} I. \end{cases} \tag{2.4}$$

By (H1), it is easy to see that there is no endemic equilibrium in (2.3) if $R_0 < 1$ and there is a unique endemic equilibrium $E^* = (I^*, R^*)$ if $R_0 > 1$.

We now consider the stability of E^*.

THEOREM 2.1. *Let (H1) and (H2) hold. If $R_0 > 1$, system (2.3) admits a unique endemic equilibrium that is globally stable.*

Proof. The Jacobian matrix of (2.3) at E^* is

$$J = \begin{bmatrix} \dfrac{\lambda\,(C - 2I^* - R^*)}{f(I^*)} - d - \gamma - \dfrac{\lambda\, I^*\,(C - I^* - R^*)\, f^{'}(I^*)}{f^2(I^*)} & -\frac{\lambda\, I^*}{f(I^*)} \\ \gamma & -d - \nu \end{bmatrix}.$$

By the first equation of (2.4), we have

$$f(I^*) = \frac{\lambda\,(C - I^* - R^*)}{d+\gamma}. \tag{2.5}$$

As a result, we can obtain

$$\det(J) = \frac{I^*\left(f^{'}(I^*)\,(d\gamma + \gamma\,\nu + \,\nu\, d + \,d^2) + \lambda\,(d + \gamma + \nu)\right)(d+\gamma)}{\lambda\,(C - I^* - R^*)} > 0$$

and

$$\operatorname{trace}(J) = \frac{I^*(f^{'}(I^*)\,d^2 + \lambda\,\gamma + 2\,f^{'}(I^*)\,d\gamma + f^{'}(I^*)\,\gamma^2 - \lambda\,\nu) + \lambda\,(C - R^*)(d+\nu)}{\lambda\,(-C + I^* + R^*)}.$$

Using the second equation of (2.4), we see that the trace has the same sign as the following expression:

$$\lambda\, I^*\nu - \lambda\, C\nu - I^* f^{'}(I^*)\,d^2 - 2\, I^* f^{'}(I^*)\,d\gamma - I^* f^{'}(I^*)\,\gamma^2 - \lambda\, Cd,$$

which is negative because $I^* < C$ and $f^{'}(I^*) \geq 0$. Therefore, the eigenvalues of the Jacobian matrix J have negative real parts. This means that E^* is asymptotically stable.

Denote the right-hand side of (2.3) by (F_1, F_2) and choose a Dulac function as $Q = f(I)/I$. Then we have

$$\frac{\partial(QF_1)}{\partial I} + \frac{\partial(QF_2)}{\partial R} = -\lambda - (d+\gamma) f^{'}(I^*) - \frac{f(I)}{I}(d+\nu) < 0$$

when $I > 0$. Thus, system (2.3) does not have a limit cycle in the region $I > 0$. Then, it is easy to see that E^* is globally stable in the region $I > 0$. □

REMARK 1. *Without an intervention to the spread of the disease, i.e., $f(I) \equiv 1$, the basic reproduction number $R_0 = C\lambda/(d+\gamma)$. With the introduction of the intervention factor f, $R_0 = C\lambda/(f(0)(d+\gamma))$. Thus, if $f(0) = 1$, which essentially means that we perform intervention policies only at a suitable infection level, the basic reproduction number remains unchanged. But since f is an increasing function, it follows from (2.4) that the value of I^* is less than that in the absence of the intervention. Note that Theorem 2.1 shows that E^* is globally stable. Therefore, the intervention policy for controlling the disease decreases the endemic level and cannot induce any complicated dynamical behaviors for the model.*

3. **Saturated Infection Force.** In this section, we consider the effect of a saturated infection force. For simplicity in notations, we assume that the infection force $\beta(I)$ can be factorized as $Ig(I)$, where g is continuously differentiable. Then (2.1) becomes

$$\begin{aligned} \frac{dS}{dt} &= dN - dS - Ig(I)S + \nu R, \\ \frac{dI}{dt} &= Ig(I)S - (d+\gamma)I, \\ \frac{dR}{dt} &= \gamma I - (d+\nu)R. \end{aligned} \tag{3.1}$$

By similar arguments to those of the last section, it suffices to consider the following model:

$$\begin{aligned} \frac{dI}{dt} &= Ig(I)(C - I - R) - (d+\gamma)I, \\ \frac{dR}{dt} &= \gamma I - (d+\nu)R. \end{aligned} \tag{3.2}$$

We make the following assumptions for (3.2):

(H3) $g(I) \geq 0$ is bounded for $I \geq 0$; $Ig(I) \to k$ as $I \to \infty$;

(H4) $(Ig(I))' > 0$ for $I > 0$; there is an $\eta > 0$ such that $g'(I) > 0, I \in (0, \eta)$ and $g'(I) < 0, I \in (\eta, \infty)$, and such that $g''(I) \leq 0$ for $I \in (0, \eta)$.

We have assumed $\eta > 0$ in (H4). If $\eta = 0$, since $g(I)$ is a decreasing function, that is the case studied in the last section.

The basic reproduction number of (3.2) is $R_0 = g(0)C/(d+\gamma)$. $E_0 = (0,0)$ is a disease-free equilibrium of (3.2). The Jacobian matrix at E_0 is

$$\begin{bmatrix} g(0)C - d - \gamma & 0 \\ \gamma & -d - \nu \end{bmatrix}.$$

It follows that E_0 is asymptotically stable if $R_0 < 1$ and is unstable if $R_0 > 1$.

An endemic equilibrium of (3.2) is a positive solution of the following system:

$$\begin{cases} R = C - I - \dfrac{d+\gamma}{g(I)}, \\ R = \dfrac{\gamma}{d+\nu} I. \end{cases} \tag{3.3}$$

Thus,

$$F(I) := \frac{(d+\nu+\gamma)}{d+\nu} I + \frac{d+\gamma}{g(I)} = C. \tag{3.4}$$

Since

$$F'(I) = 1 + \frac{\gamma}{d+\nu} + \left(-\frac{d}{g^2(I)} - \frac{\gamma}{g^2(I)}\right) g'(I),$$
$$F''(I) = 2\,\frac{(d+\gamma)}{g^3(I)}(g'(I))^2 - \frac{(d+\gamma)}{g^2(I)} g''(I),$$

it follows from (H3) and (H4) that $F''(I) > 0$ for $I \in (0, \eta)$, which implies that $F(I)$ either is an increasing function or has an minimum at $\bar{I} \in (0, \eta]$. Thus, if $R_0 > 1$, (3.4) has a unique positive solution I^*, and therefore, (3.2) has a unique endemic equilibrium $E^* = (I^*, R^*)$ with $R^* = \gamma I^*/(d+\nu)$. Similarly, if $R_0 < 1$ and $F(\bar{I}) < C$, (3.4) has two positive roots $0 < I_1 < I_2$, which implies that (3.2) has two endemic equilibria: $E_1 = (I_1, R_1), E_2 = (I_2, R_2)$ with $R_i = \gamma I_i/(d+\nu)$; if $R_0 < 1$ and $F(\bar{I}) = C$, (3.2) has a unique endemic equilibrium $\bar{E}$; if $R_0 < 1$ and $F(\bar{I}) > C$, (3.2) has no endemic equilibrium.

Let us consider the stability of the endemic equilibrium E^*.

THEOREM 3.1. *Let (H3) and (H4) hold. If $R_0 > 1$, the endemic equilibrium E^* of (3.2) is stable when*

$$g^{'}(I^*) < \frac{C\nu - I^*\nu + Cd}{I^*\left(-C + I^* + R^*\right)^2}, \tag{3.5}$$

and unstable when

$$g^{'}(I^*) > \frac{C\nu - I^*\nu + Cd}{I^*\left(-C + I^* + R^*\right)^2}. \tag{3.6}$$

Proof. The Jacobian matrix of (3.2) at E^* is

$$J_1 = \begin{bmatrix} (C - I^* - R^*)\,(g(I^*) + I^* g^{'}(I^*)) - I^* g - d - \gamma & -I^* g \\ \gamma & -d - \nu \end{bmatrix}.$$

Then,

$$\det(J_1) = g^{'}(I^*) I^*\,(d+\nu)\,(-C + I^* + R^*) + D \tag{3.7}$$

where

$$D = (d+\nu)\,(d+\gamma) + g(I^*)(-Cd - C\nu + 2\,dI^* + 2\,I^*\nu + d\,R^* + R^*\nu + \gamma\,I^*).$$

By (3.3), we have

$$g(I^*) = \frac{d+\gamma}{C - I^* - R^*}. \tag{3.8}$$

It follows that

$$D = \frac{(d+\gamma)\,I^*\,(d+\nu+\gamma)}{C - I^* - R^*}. \tag{3.9}$$

By the analysis for the existence of E^*, we see that $F^{'}(I^*) > 0$. Thus,

$$g^{'}(I^*) < \frac{g^2(I^*)\,(d+\nu+\gamma)}{(d+\nu)\,(d+\gamma)} = \frac{(d+\gamma)\,(d+\nu+\gamma)}{\left(-C + I^* + R^*\right)^2 (d+\nu)}. \tag{3.10}$$

It follows from (3.7) and (3.9) that $\det(J_1) > 0$. This means that the stability of E^* is determined by the trace of J_1. Note that

$$\mathrm{trace}(J_1) = g^{'}(I^*)(I^*\,(C - I^* - R^*)) + (C - 2\,I^* - R^*)\,g(I^*) - 2\,d - \gamma - \nu.$$

Using (3.8), we have

$$\mathrm{trace}(J_1) = g^{'}(I^*) I^*\,(C - I^* - R^*) - \frac{\nu(C - I^*) + Cd}{C - I^* - R^*}.$$

It follows that trace$(J_1) < 0$ if (3.5) holds and trace$(J_1) > 0$ if (3.6) holds. Therefore, E^* is stable if (3.5) holds and unstable if (3.6) holds. □

We now consider the stability of E_1 and E_2. The analysis for the existence of E_1 and E_2 implies $F^{'}(I_1) < 0$ and $F^{'}(I_2) > 0$. If J_{21} and J_{22} are the Jacobian matrices of (3.2) at E_1 and E_2 respectively, then by similar discussions as those in the proof of Theorem 3.1, we see that trace$(J_{21}) < 0$ and trace$(J_{22}) > 0$. Hence, E_1 is a saddle and E_2 is a node or a focus. This means that the model may have a saddle node bifurcation.

Since it is not easy to perform global analysis for the model when two endemic equilibria occur, we will fix the function g and use computer simulations (by means of the packages PPLANE6 [13] and MatCont [6]) to find something interesting.

4. **Quadratic Infection Force with Saturation.** In this section, we fix the function g in system (3.2) as

$$g(I) = \lambda \frac{I}{1 + pI + qI^2}, \tag{4.1}$$

motivated by the work of Zhu, Campbell and Wolkowicz in [19] on a population model. If $p = 0$, the infection force becomes $\lambda I^2/(1 + qI^2)$, which was studied by Ruan and Wang in [14]. (3.2) with g given in (4.1) becomes

$$\begin{aligned} \frac{dI}{dt} &= \lambda \frac{I^2}{1 + pI + qI^2}(C - I - R) - (d + \gamma)I, \\ \frac{dR}{dt} &= \gamma I - (d + \nu)R. \end{aligned} \tag{4.2}$$

It is easy to see that (H3) and (H4) are satisfied with $\eta = 1/\sqrt{q}$. Further, by the formula for a basic reproduction number in [15], we obtain $R_0 = 0$. Then using the arguments of the last section, we see that there is no endemic equilibrium in (4.2) if

$$\lambda C \le p(d + \gamma) \tag{4.3}$$

or

$$\begin{cases} \lambda C > p(d + \gamma), \\ \lambda^2 (d + \nu) C^2 < 2\, p\lambda\, (d + \gamma)(d + \nu) C \\ \quad - (d + \gamma)\left(p^2 (d + \gamma)(d + \nu) - 4\, q (d + \gamma)(d + \nu) - 4\, \lambda\, (d + \gamma + \nu)\right); \end{cases} \tag{4.4}$$

there is only one endemic equilibrium in (4.2) if

$$\begin{cases} \lambda C > p(d + \gamma), \\ \lambda^2 (d + \nu) C^2 = 2\, p\lambda\, (d + \gamma)(d + \nu) C \\ \quad - (d + \gamma)\left(p^2 (d + \gamma)(d + \nu) - 4\, q (d + \gamma)(d + \nu) - 4\, \lambda\, (d + \gamma + \nu)\right); \end{cases} \tag{4.5}$$

and there are two endemic equilibria in (4.2) if

$$\begin{cases} \lambda C > p(d + \gamma), \\ \lambda^2 (d + \nu) C^2 > 2\, p\lambda\, (d + \gamma)(d + \nu) C \\ \quad - (d + \gamma)\left(p^2 (d + \gamma)(d + \nu) - 4\, q (d + \gamma)(d + \nu) - 4\, \lambda\, (d + \gamma + \nu)\right). \end{cases} \tag{4.6}$$

Motivated by the conditions for the existence of an endemic equilibrium, we define

$$\mathcal{R}_1 = \frac{\lambda C}{d + \gamma}.$$

By [5], $\mathcal{R}_1$ can be regarded as the contact numbers of one infective near the disease-free steady state during his or her infection period. Then the above discussions show that there are endemic equilibria if

$$\mathcal{R}_1 > p + 2\sqrt{q + \lambda\frac{d+\gamma+\nu}{(d+\gamma)(d+\nu)}}, \tag{4.7}$$

and there is no endemic equilibrium if

$$\mathcal{R}_1 < p + 2\sqrt{q + \lambda\frac{d+\gamma+\nu}{(d+\gamma)(d+\nu)}}. \tag{4.8}$$

Thus, the increase of parameters p, q and γ decreases the probability that endemic equilibria occur, but the increase of population size C lifts the possibility to admit endemic equilibria.

We now use computer simulations to study the asymptotic behavior of (4.2) when (4.7) is satisfied. We begin from the recovery rate γ. Fix $p = 0.1, q = 0.1, d = 0.4, C = 8, \nu = 0$ and $\lambda = 0.5$. Then (4.7) is satisfied when $0 < \gamma < 1.25$. The trace of the Jacobian matrix at E_2 is negative when $0 < \gamma < 1.14$ and is positive when $1.14 < \gamma < 1.25$ (see Figure 2). If we increase γ from 0, we obtain three

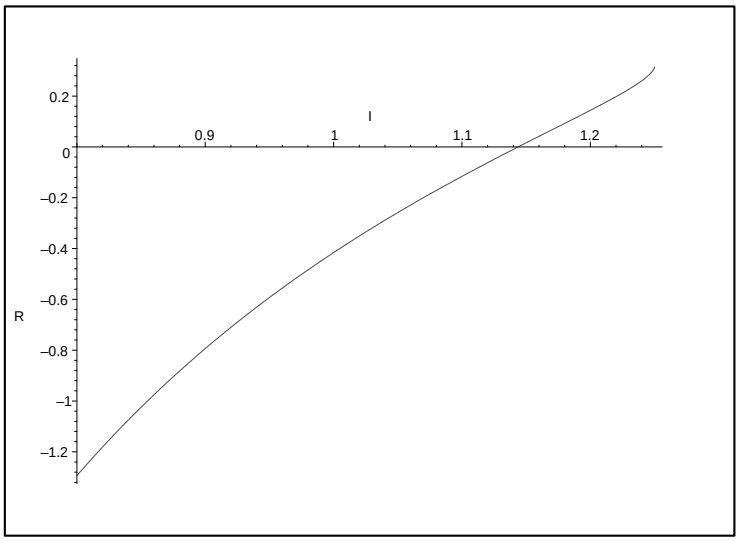

FIGURE 2. The trace changes sign.

types of dynamical behaviors. If $0 < \gamma < 1.111$, the stable manifolds of saddle E_1 separate the interior of the first quadrant of R^2 into two parts such that orbits initiating from the left part tend to the disease-free equilibrium $(0, 0)$, while orbits initiating from the right part tend to the endemic equilibrium E_2 (see Figure 3). If $1.111 < \gamma < 1.14$, there is an unstable limit cycle such that orbits initiating inside tend to the endemic equilibrium E_2, while orbits initiating outside tend to the disease-free equilibrium $(0, 0)$ except for the stable manifolds of E_1 (see Figure 4), and the limit cycle contracts as γ increases. If $1.14 < \gamma < 1.25$, the limit cycle disappears and all positive orbits except the two endemic equilibria and the two stable manifolds of the saddle tend to the disease-free equilibrium $(0, 0)$ (see Figure

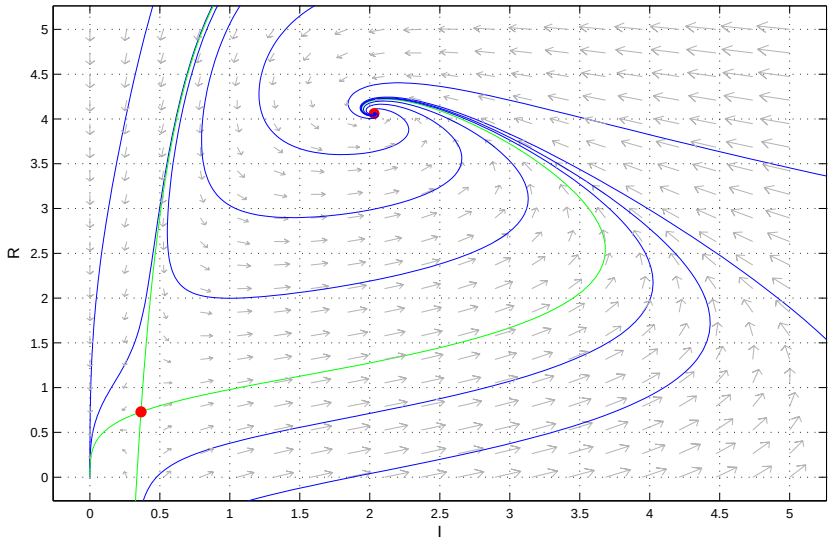

FIGURE 3. Bistable equilibria when $\gamma = 0.8$.

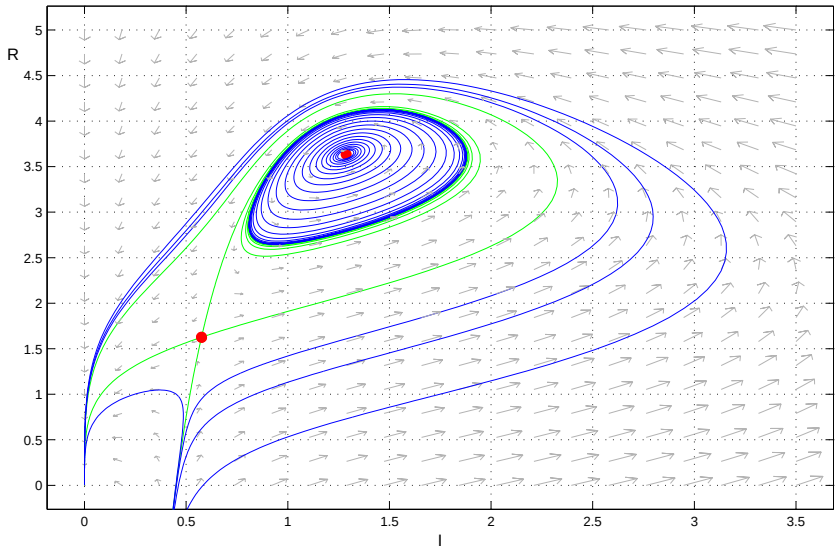

FIGURE 4. Bistable equilibria with an unstable limit cycle when $\gamma = 1.13$.

5). This means that the disease dies out although we have two endemic equilibria.

If p and q are small, i.e., if the inhibition effect from behavior changes is weak, we can find two limit cycles in (4.2). The larger one is unstable and the smaller one is stable such that orbits inside the large cycle (except the E_2) tend to the small cycle as time goes to infinity, and almost all orbits outside tend to the disease-free equilibrium $(0, 0)$ as time approaches infinity (see Figure 6). Indeed, if we fix $p = 0.005, q = 0.008, \nu = 0, \gamma = 4, d = 1, \lambda = 1$, we have a saddle node bifurcation of a nonhyperbolic periodic orbit at $C = 12.39579$ (see Figure 7). When

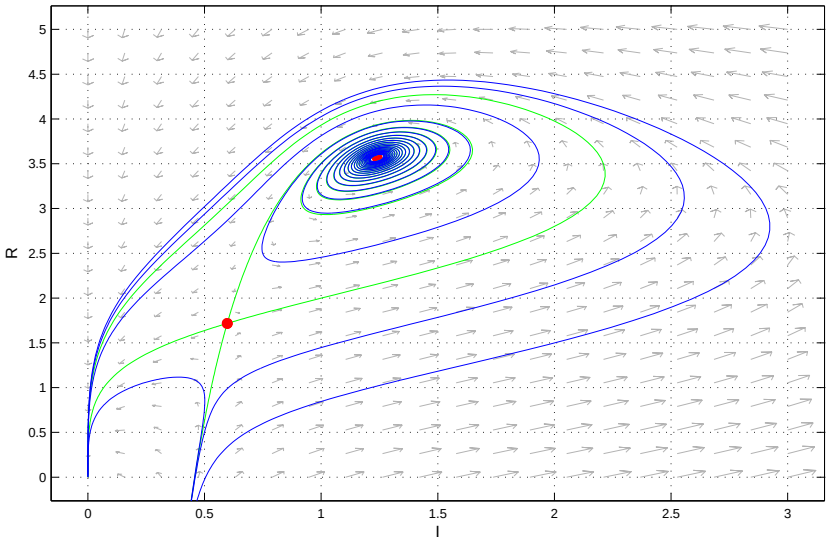

FIGURE 5. The disease dies out when $\gamma = 1.15$ although there are two endemic equilibria.

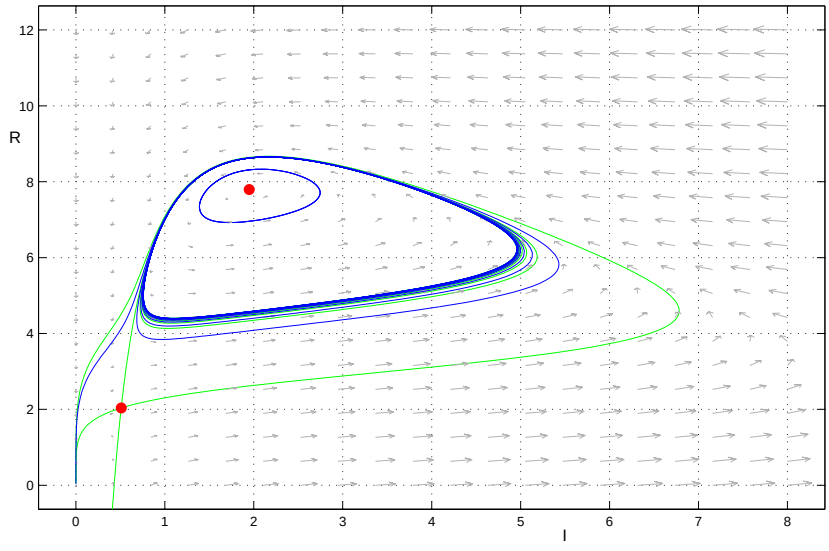

FIGURE 6. Unstable limit cycle and stable limit cycle.

$12.39579 < C < 12.421$, there are two limit cycles. The small cycle shrinks to the endemic equilibrium E_2 at 12.421. When $12.421 < C < 12.4297$, the larger cycle persists but the smaller one disappears. A homoclinic orbit occurs at $C = 12.4297$ (see Figure 8). If $C > 12.4297$, the homoclinic orbit is broken and the stable manifolds of the saddle E_1 split the feasible region into two parts such that one is the attraction region of E_2 and the other one the attraction region of $(0, 0)$. For

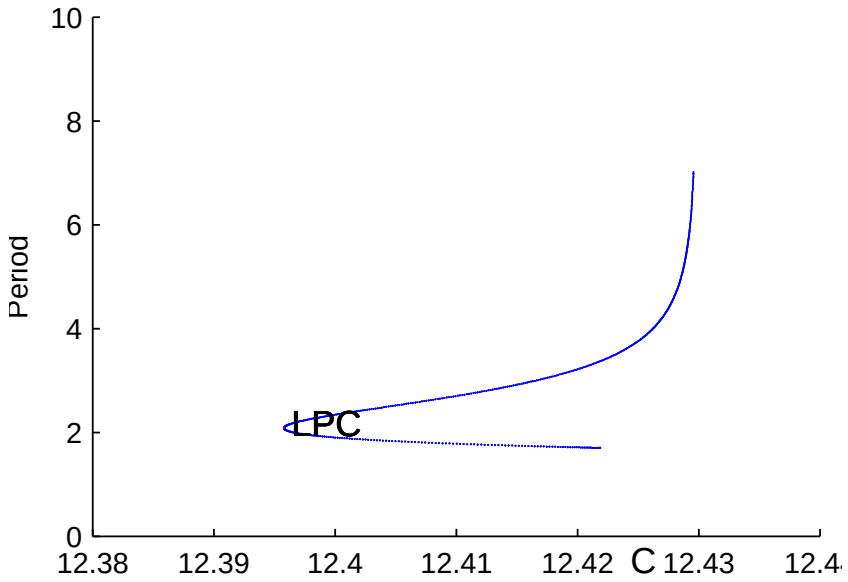

FIGURE 7. Bifurcation curve of period of cycles versus C.

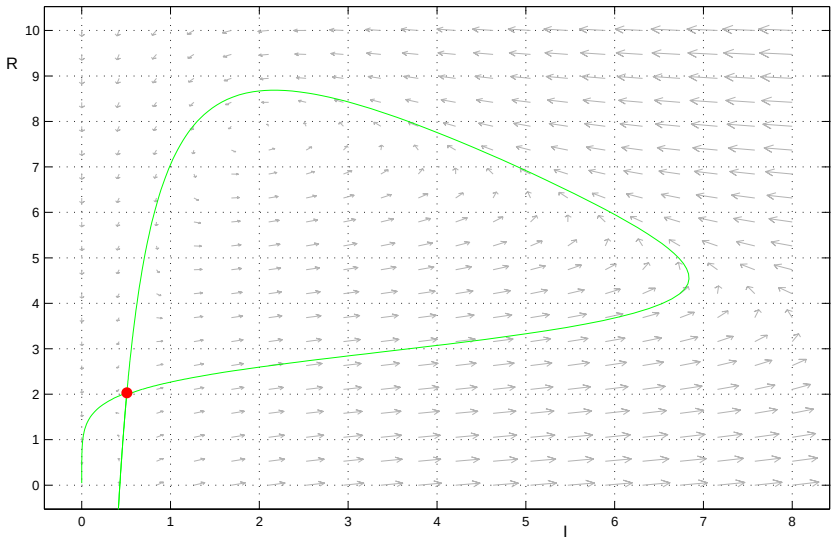

FIGURE 8. A homoclinic orbit at $C = 12.4297$.

the case where $10.065 < C < 12.39579$, the disease dies out although there are two endemic equilibria, which is similar to the behavior shown in Figure 5.

5. **Discussion.** In this paper, we have studied the epidemic models with nonlinear infection forces. For the model with intervention strategies, we have shown that the disease-free equilibrium is globally stable if the basic reproduction number is

less than 1, and the unique endemic equilibrium is globally stable if the basic reproduction number is greater than 1. By means of these results, we have verified that the intervention policy decreases endemic level. For the model with the saturated infection force, with the aid of computer simulations, we have shown that the model admits rich dynamical behaviors. In other words, it has saddle node bifurcations of endemic equilibria, Hopf bifurcations, the saddle node bifurcation of nonhyperbolic periodic orbit, which implies the existence of two limit cycles and homoclinic bifurcations. We have also found that the model admits bistable steady states such that the outcome of disease evolution depends upon initial positions. Two examples have indicated that the disease dies out although there are two endemic equilibria. This means that it is unnecessary to drive the control variable $\mathcal{R}_1$ below the quantity defined by the right side of the inequality (4.8) to eradicate the disease.

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