Psychological effect can lead to bistability in epidemics

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Abstract

In this paper, we study the psychological effect in a SIS epidemic model. The basic reproduction number is obtained. However, the disease free equilibrium is always asymptotically stable, which doesn’t depends on the basic reproduction number. The system has a saddle-node bifurcation appear and displays bistable behavior, which is a new phenomenon in epidemic dynamics and different from the backward bifurcation behavior.

Key words: SIS model, Psychological effect; Saddle-node bifurcation; Bistability behavior

1. Introduction

In classic disease transmission model, the incidence rate is bilinear in the infectious fraction \(I\) and the susceptible fraction \(S\). Recently nonlinear incidence functions in epidemic models attracted much attention [1–9, 11–14].

Capasso and Serio [1], Ruan and Wang [5] show the incidence function \(g(I)\) can interpret the “psychological” effect: for a very large number of infective individuals the infection force may decrease as the number of infective individuals increases, because in the presence of large number of infective the population may tend to reduce the number of contacts per unit time. Xiao and Ruan [8] studied an epidemic model with nonmonotonic incidence rate, which describes the psychological effect of certain serious diseases on the community when the number of infectives is getting larger. Lu et al. [9] provided a more reasonable incidence function, which first increases to a maximum when a new infectious disease emerges or an old infectious disease reemerges, then decreases due to psychological effect, and eventually tends to a saturation level due to crowding effect.

In this paper we will discuss the psychological effect in epidemics in a different way. The general SIS epidemic model takes the following form

\[
\begin{align*}
\frac{dS}{dt} &= b - dS - k_1 S g(I) + \gamma I, \\
\frac{dI}{dt} &= k_1 S g(I) - (d + \mu + \gamma) I, \\
\end{align*}
\]

where \(b\) is natural birth rate, \(d\) is natural decay rate, \(k_1\) is transmission rate for naive susceptible, \(\mu\) is disease related death rate, \(\gamma\) is the rate of infective individuals lose immunity and move into susceptible compartment. For the incidence rate \(g(I)\), we have following cases, some are based on the work of Andrews [10].

(I) If \(g(I) = I\), then system (1.1) is the classic SIS model;

(II) If we choose \(g(I)S\) as following

\[
g(I)S = \frac{S}{1 + \frac{S}{k_s + I}} = \frac{SI}{k_s + I},
\]

which is the saturated incidence rate in epidemic models [1, 11, 12]. Here \(k_s\) is the saturation constant of infected population concentration;

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(III) If $$g(I)S = \frac{S}{1 + \frac{S}{k} I} = \frac{kSI}{kI + k + I^2},$$
which is the nonmonotone incidence rate, where $$k_i$$ is the inhibition constant of infected population concentration. The special case is
$$g(I)S = \frac{S}{1 + \frac{S}{k} I} = \frac{kSI}{kI + k + I^2} = \frac{\frac{kSI}{kI + k + I^2}}{1 + \frac{S}{k} I},$$
which was studied by Xiao and Ruan [8].

(IV) If we choose $$g(I)S$$ as following
$$g(I)S = \frac{S}{1 + \frac{S}{k} I} = \frac{kSI^2}{kI + k + I^2},$$
which is the generalized nonmonotone and saturated incidence rate [9]. Especially, if
$$g(I)S = \frac{S}{1 + \frac{S}{k} I} = \frac{kSI^2}{kI + k + I^2} = \frac{\frac{kSI^2}{kI + k + I^2}}{1 + \frac{S}{k} I},$$
which was studied by Ruan and Wang [5], and Tang et al.[6].

(V) If we choose $$g(I)S$$ as following
$$g(I)S = \frac{S}{1 + \frac{S}{k} I} = \frac{kSI^p}{kI + k + I^q},$$
which is the general incidence rate. Here $$r, p, p'$$ and $$q = p + p'$$ are nonnegative. The special case is
$$g(I)S = \frac{S}{1 + \frac{S}{k} I} = \frac{kSI^p}{kI + k + I^q} = \frac{\frac{kSI^p}{kI + k + I^q}}{1 + \frac{S}{k} I},$$
which was studied by a number of authors [3, 4, 13, 14].

In this paper, we only consider the case $$p = 1, q = 2, r = 1$$. Denote $$k_1k_i = k, k_2k_i = \alpha, k_i = \beta$$, then system (1.1) can be written by following model
$$\begin{cases}
\frac{dS}{dt} = b - dS - \frac{\alpha SI}{\alpha + \beta I} + \gamma I, \\
\frac{dI}{dt} = \frac{\alpha SI}{\alpha + \beta I} - (\mu + \Delta)I.
\end{cases}$$
(1.2)

Here, $$\beta$$ is the inhibition psychological effect constant of infected population and $$\alpha, \beta$$ are positive.

2. Equilibria and thresholds

It can be verified that the nonnegative orthant $$R^+_2 = \{(S, I) : S \geq 0, I > 0\}$$ is positively invariant with respect to system (1.2) and the model is well posed.

Denote
$$R_0 = \frac{bk}{\beta d(\mu + \gamma + d)} = \frac{b}{\beta} \cdot k_1 \cdot \frac{1}{\mu + \gamma + d},$$
be the basic reproduction number, which determining whether or not the disease dies out in classical SIS epidemic models. We also denote
$$R_c = R_0 - \frac{2}{\beta} \sqrt{\alpha(1 + \frac{k(\mu + d)}{d(\mu + \gamma + d)})},$$
2
and
\[ R_{cc} = R_0 + \frac{2}{\beta} \sqrt{\alpha(1 + \frac{k(\mu + d)}{d(\mu + \gamma + d)})}. \]

It is easy to see that \( R_c < R_0 < R_{cc} \).

(i) System (1.2) always has a disease-free equilibrium \( E_0 = (\frac{b}{d}, 0) \).

(ii) To obtain the positive equilibria of system (1.2), we solve the following equations:
\[
\begin{align*}
  b - dS - \frac{kS \beta I}{a + \beta I + F} + \gamma I &= 0, \\
  \frac{kS I}{a + \beta I + F} - (\mu + \gamma + d) &= 0.
\end{align*}
\]

Solving the first equation of (2.1), we have
\[ S = \frac{b - (\mu + d)I}{d}. \]

substituting which into the second equation of (2.1) yields
\[ AI^2 + BI + a = 0, \quad (2.2) \]
where
\[ A = 1 + \frac{k(\mu + d)}{d(\mu + \gamma + d)}, \quad B = \beta(1 - R_0). \]

Denote \( \Delta = B^2 - 4Aa \). If \( \Delta > 0 \), then \( R_c < 1 \) or \( R_c > 1 \). If \( B < 0 \), then \( R_0 > 1 \). When \( R_c > 1 \), equation (2.2) has two positive roots:
\[ I^*_+ = \frac{-B \pm \sqrt{\Delta}}{2A} \]

Theorem 2.1 (i) System (1.2) always has a disease-free equilibrium \( E_0 \);

(ii) If \( R_c > 1 \), system (1.2) also has two positive equilibria \( E^*_+(S^*_+, I^*_+), E^*_-(S^*_-, I^*_-) \), where
\[
\begin{align*}
  S^*_+ &= \frac{b - (\mu + d)I^*_+}{d}, \quad I^*_+ = \frac{-B + \sqrt{\Delta}}{2A}, \\
  S^*_- &= \frac{b - (\mu + d)I^*_-}{d}, \quad I^*_- = \frac{-B - \sqrt{\Delta}}{2A}.
\end{align*}
\]

The existence of positive equilibria are summarized in Table 1.

|          | \( R_c < 1 \) | \( R_c > 1 \) |
|----------|--------------|--------------|
| \( E_0 \) | exist        | exist        |
| \( E^*_+ \) | —            | exist        |
| \( E^*_- \) | —            | exist        |

3. Stability analysis

Let \( \tilde{E} \) be any arbitrary equilibrium of system (1.2). The Jacobian matrix associated with system (1.2) is
\[
\mathcal{J}_E = \begin{bmatrix}
  -d & \frac{k\beta}{a + \beta I + F} \\
  \frac{k\beta}{a + \beta I + F} & -\frac{kS(\gamma + \beta I)}{(a + \beta I + F)^2} - (\mu + \gamma + d)
\end{bmatrix}.
\]
The characteristic equation of system (1.2) at $E$ is $|\lambda - \mathcal{J}_E| = 0$.

3.1. Stability analysis of the disease-free equilibrium

Theorem 3.1 The disease-free equilibrium $E_0$ of system (1.2) is always locally asymptotically stable.

Proof. The characteristic equation of system (1.2) at the disease-free equilibrium $E_0$ is obtained as

$$(\lambda + d)(\lambda + \mu + \gamma + d) = 0.$$ 

The characteristic polynomial has two roots $-d$, $-(-\mu + \gamma + d)$. Since the two roots are all negative, the disease-free equilibrium $E_0$ of system (1.2) is locally asymptotically stable.

3.2. Stability analysis of positive equilibria

Theorem 3.2 If $R_c > 1$, $a_1 > 0$, system (1.2) has two positive equilibria $E^*_+$ and $E^*_-$, where $E^*_+$ is a locally asymptotically stable and $E^*_-$ is unstable.

Proof. Denote an arbitrary positive equilibrium of system (1.2) as $E^*$. The characteristic equation of the system (1.2) at the arbitrary positive equilibrium $E^*$ is obtained as

$$\lambda^2 + a_1\lambda + a_2 = 0,$$

where

$$a_1 = \mu + \gamma + 2d + \frac{\mu + \gamma + \delta(2a_1 + \beta)}{a + \beta l (l + \gamma + \delta)},$$

$$a_2 = d(\mu + \gamma + d) + (\mu + \gamma + d)\left[\frac{\mu + \gamma + \delta(2a_1 + \beta)}{a + \beta l (l + \gamma + \delta)} - \gamma\frac{k(l^*)^2}{\alpha + \beta l (l + \gamma + \delta)}\right].$$

(i) For equilibrium $E^*_+$, we have

$$d(I^*_+)^2 + k(I^*_+)^2 - d\alpha - \gamma\frac{k(l^*_+)^2}{\alpha + \beta l (l + \gamma + \delta)} = 0,$$

$$d(1 + \frac{\mu + \gamma + \delta(2a_1 + \beta)}{a + \beta l (l + \gamma + \delta)})(I^*_+)^2 - d\alpha,$$

$$= d(1 + \frac{\mu + \gamma + \delta(2a_1 + \beta)}{\alpha + \beta l (l + \gamma + \delta)} + \frac{\mu + \gamma + \delta(2a_1 + \beta)}{\alpha + \beta l (l + \gamma + \delta)}) - d\alpha,$$

$$= d\left[(\beta(R_0 - 1) + \sqrt{\Delta})^2 - 4\Delta\right] - d\alpha,$$

It follows from

$$\frac{d(\beta(R_0 - 1) + \sqrt{\Delta})^2 - 4\Delta}{4\Delta} - d\alpha = \frac{d\Delta + d\beta(R_0 - 1)\sqrt{\Delta}}{2\Delta},$$

that $\frac{d(\beta(R_0 - 1) + \sqrt{\Delta})^2 - 4\Delta}{4\Delta} - d\alpha > 0$. Then,

$$\frac{d(\beta(R_0 - 1) + \sqrt{\Delta})^2 - 4\Delta}{4\Delta} - d\alpha > 0,$$

$$\iff d + \frac{k(l^*_+)^2}{\alpha + \beta l (l + \gamma + \delta)} = d\left[\frac{\mu + \gamma + \delta(2a_1 + \beta)}{\alpha + \beta l (l + \gamma + \delta)}\right] - \gamma\frac{k(l^*_+)^2}{\alpha + \beta l (l + \gamma + \delta)} > 0,$$

$$\iff a_2 > 0.$$ 

Clearly, $a_2 > 0$, and we also have $a_1 > 0$. By the Routh-Hurartz Criterion, we know that the positive equilibrium $E^*_+$ is a locally asymptotically stable node.

(ii) For equilibrium $E^*_-$, we have

$$d(I^*_-)^2 + k(I^*_-)^2 - d\alpha - \gamma\frac{k(l^*_-)^2}{\alpha + \beta l (l + \gamma + \delta)} = 0,$$

$$d(1 + \frac{\mu + \gamma + \delta(2a_1 + \beta)}{a + \beta l (l + \gamma + \delta)})(I^*_-)^2 - d\alpha,$$

$$= d(1 + \frac{\mu + \gamma + \delta(2a_1 + \beta)}{\alpha + \beta l (l + \gamma + \delta)} - \gamma\frac{k(l^*_-)^2}{\alpha + \beta l (l + \gamma + \delta)}) - d\alpha,$$

$$= d\left[\frac{\beta(R_0 - 1) - \sqrt{\Delta}}{4\Delta}\right] - d\alpha,$$

$$< d\left[\frac{\beta(R_0 - 1)^2 - 4\Delta}{4\Delta}\right] - d\alpha.$$

Thus, $a_2 < 0$. By the Routh-Hurartz Criterion, we know in this case the positive equilibrium $E^*_-$ is an unstable saddle.
Table 2: The stabilities of the equilibria and the behaviors of system (1.2).

| $E_0$ | $E_+^*$ | $E_-^*$ | System (1.2) |
|-------|---------|---------|--------------|
| $R_c < 1$ | LAS | — | — | Converges to $E_0$ |
| $R_c > 1$ | LAS | LAS | US | Bistable |

4. Saddle-node bifurcation

In this section, we discuss the bifurcation behavior of system (1.2). The conditions for saddle-node bifurcation are derived. If $R_c = 1$, system (1.2) undergoes a saddle-node bifurcation. The positive equilibrium $E_+^*$ and $E_-^*$ collide to each other and system (1.2) has a unique instantaneous positive equilibrium $E$. Also one of the eigenvalues of the Jacobian matrix at the instantaneous positive equilibrium $E = (S, I)$ is zero. Here $\tilde{S} = \frac{b - \mu + d}{d}$, $\tilde{I} = \frac{b (R_c - 1)}{2}$. If $R_c = 1$, system (1.2) undergoes a saddle-node bifurcation around instantaneous positive equilibrium $E = (S, I)$.

Proof. Let $R_0$ be the bifurcation parameter. We use the Sotomayor’s theorem to prove that system (1.2) undergoes a saddle-node bifurcation. The Jacobian matrix at the saddle-node must have a zero eigenvalue and two eigenvalues with negative real parts. Let $F = (f_1, f_2)^T$ with

$$
\begin{align*}
f_1 &= b - dS - \frac{kS^2}{\alpha + \beta} + \gamma I + d + \beta I, \\
f_2 &= \frac{kS^2}{\alpha + \beta} - (\mu + \gamma + d)I.
\end{align*}
$$

The Jacobian matrix of system (1.2) at $E$ is given by

$$
J_E = \begin{pmatrix}
-d - \frac{kS^2}{\alpha + \beta} & \gamma - \frac{(\mu + \gamma + d)(2a + \beta)}{\alpha + \beta} \\
\frac{kS^2}{\alpha + \beta} & \gamma - (\mu + \gamma + d)I
\end{pmatrix}.
$$

The matrix has a simple zero eigenvalue, which requires that $\text{det}(J_E) = 0$ at $R_0 = R_0^{[n]}$. If $V$ and $W$ represent eigenvectors corresponding to the eigenvectors of $J_E$ and $J_E^T$ corresponding to the zero eigenvalue, respectively, then they are given by

$$
V = \begin{pmatrix}
v_1 \\
v_2
\end{pmatrix} = \begin{pmatrix}
\frac{-\mu + d}{1} \\
1
\end{pmatrix},
$$

$$
W = \begin{pmatrix}
w_1 \\
w_2
\end{pmatrix} = \begin{pmatrix}
1 + \frac{(\mu + 2d)(\alpha + \beta)}{k + (\alpha + \beta + d)} \\
\frac{1}{k + (\alpha + \beta + d)}
\end{pmatrix}.
$$

Thus we get

$$
F_{R_0}(E, R_0^{[n]}) = \begin{pmatrix}
\frac{\beta d (\mu + \gamma + d)E}{\alpha + \beta} - \frac{\beta d (\mu + \gamma + d)E}{\alpha + \beta} \\
\frac{\beta d (\mu + \gamma + d)E}{\alpha + \beta} - \frac{\beta d (\mu + \gamma + d)E}{\alpha + \beta}
\end{pmatrix},
$$

$$
D^2 F(E, R_0^{[n]})(V, W) = \begin{pmatrix}
\frac{(\mu + \gamma + d)(\alpha + \beta)}{\alpha + \beta} & \frac{\beta d (\mu + \gamma + d)E}{\alpha + \beta} \\
\frac{(\mu + \gamma + d)(\alpha + \beta)}{\alpha + \beta} & \frac{\beta d (\mu + \gamma + d)E}{\alpha + \beta}
\end{pmatrix} + \frac{2k (2a + \beta)}{\alpha + \beta + d} + \frac{\mu + d}{\alpha + \beta + d}.
$$

Clearly,

$$
W^T F_{R_0}(E, R_0^{[n]}) = \mu + 2d \frac{\mu + d}{k^2 + (\mu + \gamma + d)(\alpha - F)} \frac{\beta d (\mu + \gamma + d)E}{\alpha + \beta} \neq 0.
$$
\[ W^T D^2 F(\hat{E}, R_0^{[\infty]})(V, V) = -\left(\frac{\mu + \gamma + d(\beta - 1)}{\alpha + \beta + T} + 2k(\gamma + \beta^2)\right) \cdot \frac{\mu + 2d}{\alpha + \beta + T} \neq 0. \]

Therefore, from the Sotomayor’s theorem, system (1.2) undergoes a saddle-node bifurcation around instantaneous positive equilibrium \( \hat{E} = (\hat{S}, \hat{I}) \) at \( R_0 = R_0^{[\infty]} \). Hence, we can conclude that when the parameter a passes from one side of \( R_0 = R_0^{[\infty]} \) to the other side, the number of positive equilibria of system (1.2) changes from zero to two.

5. Numerical simulations and Discussion

To verify our analytical results, we carry out some numerical simulations. In the following, we fix the parameter values as follows[8, 15]:

\[ b = 1, d = 0.12, k = 0.2, \gamma = 0.05, \mu = 0.15, \alpha = 0.5. \quad (5.1) \]

If we choose \( \beta = 3 \), the thresholds \( R_0^{[\infty]} \approx 1.73 \) and \( R_0 \approx 1.005 \). In this case, we have a saddle-node bifurcation (Figure 1). When \( \beta = 2.8, R_0 = 1.86 \), two equilibria of the model \( E_\ast^1 \) and \( E_0 \) are stable (Figure 2). If we choose \( \beta = 3.5 \), such that \( R_0 = 1.49 \), then we have only one equilibrium \( E_0 \) which is stable (Figure 3);

![Figure 1: Bistability and saddle-node bifurcation diagram of system (1.2). In this case, \( R_0^{[\infty]} \approx 1.73 \). The system displays two stable equilibria \( E_0 \) (the blue solid line at the bottom) and \( E_\ast^1 \) (the above blue curve), indicating bistable behaviour. Here, \( \hat{E} \) is the saddle point, where the two equilibria converge and display saddle-node bifurcation. The point \( E_\ast^1 \) (dashed lines) on the bottom half of the curve is unstable, and the point \( E_\ast^1 \) (solid line) on the top half of the curve is stable. Here, \( \beta = 3 \) and other parameter values are listed in (5.1).](image)

In this paper, we consider a SIS model with psychological effect and performed mathematical studies. We found that the system displays bistable behaviors. System (1.2) admits a disease-free equilibrium \( E_0 \), and two positive equilibria \( E_\ast^1 \) and \( E_\ast^2 \). We obtain two thresholds, the basic reproduction number \( R_0 = \frac{bk}{\beta d(\mu + \gamma + d)} \) and \( R_c = \frac{bk}{\beta d(\mu + \gamma + d)} - 2 \frac{\beta}{\mu} \sqrt{\alpha(1 + \frac{\mu + \gamma + d}{\mu + \gamma + d})} \). We find that the system always admits a disease-free equilibrium \( E_0 \) which is always asymptotically stable, indicating that there is no infective in the system and all individuals are susceptible. Thus, if there is no disease, then the uninfected state will remain stable for a long time. When \( R_c > 1 \), both \( E_\ast^1 \) and \( E_\ast^2 \) exist, where \( E_\ast^1 \) is locally asymptotically stable and \( E_\ast^2 \) is unstable, which implies the coexistence of susceptible, infective individuals. Choosing \( R_0 \) as the branching parameter, our investigation implies that if \( R_c = 1 \) or \( R_0 = R_0^{[\infty]} \) system (1.2) undergoes a saddle-node bifurcation. The positive equilibria \( E_\ast^1 \) and \( E_\ast^2 \) collide to each other and system (1.2) has the unique instantaneous endemic equilibrium \( \hat{E} \). From the branch diagram in figure 1, we find that when \( R_0 > R_0^{[\infty]} \), the system has two stable equilibria \( E_\ast^1 \) and \( E_0 \) appear. The system displays bistable behavior. When \( R_0 < R_0^{[\infty]} \), the system has only one equilibrium point \( E_0 \), suggesting that infectious diseases will die out eventually.

Castillo-Chavez and Song [16] proposed the backward bifurcation to illustrate that even if the basic reproduction number \( R_0 < 1 \), disease outbreaks are still possible. The backward bifurcation indicates that the system displays bistable behavior when the bifurcation point \( R_c < R_0 < 1 \). However, when \( R_0 > 1 \), the system has only one positive equilibrium point, which is stable, and the disease-free equilibrium point is unstable.

In this paper, we investigated a SIS model with psychological effect. We find that (i) the disease-free equilibrium is always stable. (ii) When \( 1 < R_0 < R_0^{[\infty]} \), the model does not have positive equilibrium point. (iii)
When $R_0 > R_{0}^{[\text{inf}]}$, the system always display bistability behavior. Our investigation implies that psychological effect is a kind of self-protection behavior of human during the outbreak of a disease. Such self-protection behavior may lead to bistable behavior, i.e., there may or may not be a disease outbreak.

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