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GLOBAL ANALYSIS OF SIR EPIDEMIC MODEL WITH IMMIGRATION AND NON-MONOTONE INCIDENCE RATE

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ABSTRACT

In this paper we study an epidemic model with immigration and non-monotonic incidence rate, which describes the psychological effect of certain serious diseases on the community when the number of infective is getting larger. By carrying out a global analysis of the model and studying the stability of the disease-free equilibrium and the endemic equilibrium, we show that either the number of infective individuals tends to zero as time evolves or the disease persists.

KEYWORDS: Epidemic, Global Stability, Immigration, Non-Monotone Incidence Rate

1. INTRODUCTION

Let S(t) be the number of susceptible individuals, I(t) be the number of infective individuals, and R(t) be the number of removed individuals at time t, respectively. Let μ be the increase of susceptible at a constant rate. After studying the cholera epidemic spread in Bari in 1973, Capasso and Serio [2] introduced a saturated incidence rate g(I)S into epidemic models, where g(I) tends to a saturation level when I gets large,

$$\text{i.e., } g(I) = \frac{kI}{1+\alpha I} \tag{1}$$

where I measures the infection force of the disease and 1/(1+αI) measures the inhibition effect from the behavioral change of the susceptible individuals when their number increases or from the crowding effect of the infective individuals. This incidence rate seems more reasonable than the bilinear incidence rate

$$g(I)S = kIS \tag{2}$$

because it includes the behavioral change and crowding effect of the infective individuals and prevents the unboundedness of the contact rate by choosing suitable parameters. Wang W. and Ruan S. [10] studied an epidemic model with a specific nonlinear incidence rate

$$g(I)S = \frac{kI^2S}{1+\alpha I^2} \tag{3}$$

and presented a detailed qualitative and bifurcation analysis of the model. The general incidence rate

$$g(I)S = \frac{kI^pS}{1+\alpha I^q} \tag{4}$$

Was proposed by Liu, Levin and Iwasa [9] and used by a number of authors, see, for example, Derrick and Van Den Driessche, Hethcote and Van Den Driessche, Alexander and Moghadas, Liu W.M. and Hethcote H.W., Xiao D. and Ruan S. [3,6,1,8,11] etc.

If the function g(I) is non-monotone, that is, g(I) is increasing when I is small and decreasing when I is large, it can be used to 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. The recent epidemic outbreak of severe acute respiratory syndrome (SARS) had such psychological effects on the general public, aggressive measures and policies, such as border screening, mask wearing, quarantine, isolation, etc. have been proved to be very effective (Gumel et al. and Wang and Ruan [7]) in reducing the infective rate at the late stage of the SARS outbreak, even when the number of infective individuals were getting relatively larger. To model this phenomenon, we propose an incidence rate

$$g(I)S = \frac{\lambda IS}{1+\alpha I^2} \tag{5}$$

Where λI measures the infection force of the disease and $1/(1+\alpha I^2)$ describes the psychological or inhibitory effect from the behavioral change of the susceptible individuals when the number of infective individuals is very large. This is important because the number of effective contacts between infective individuals and susceptible individuals decreases at high infective levels due to the quarantine of infective individuals or due to the protection measures by the susceptible individuals. Notice that when $\alpha = 0$, the non-monotone incidence rate (5) becomes the bilinear incidence rate (2).

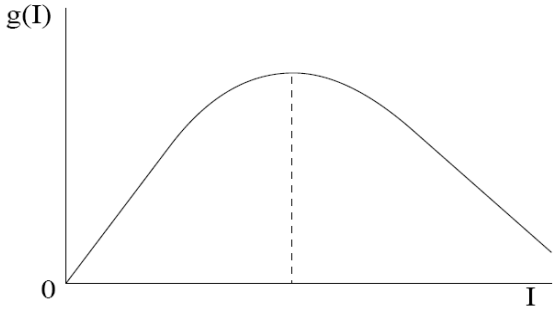

Figure 1: Non-Monotone Incidence Function g(I)

2. THE MODEL DESCRIPTION

The model to be studied takes the following form

$$\left.\begin{aligned} \frac{dS}{dt} &= a - dS - \frac{\lambda SI}{1+\alpha I^2} + \beta R + \mu \\ \frac{dI}{dt} &= \frac{\lambda IS}{1+\alpha I^2} - (d+m)I \\ \frac{dR}{dt} &= mI - (d+\beta)R \end{aligned}\right\} \tag{6}$$

Where S(t), I(t) and R(t) denote the numbers of susceptible, infective, and recovered individuals at time t, respectively. a is the recruitment rate of the population, d is the natural death rate of the population, λ is the proportionality constant, m is the natural recovery rate of the infective individuals, β is the rate at which recovered individuals lose immunity and return to the susceptible class, α is the parameter measures the psychological or inhibitory effect.

Because of the biological meaning of the components (S(t), I(t), R(t)), we focus on the model in the first octant of

R^3. We first consider the existence of equilibria of system (6). For any values of parameters, model (6) always has a disease-free equilibrium E_0 =(a+μ/d, 0, 0). To find the positive equilibria, set

$$a - dS - \frac{\lambda SI}{1+\alpha I^2} + \beta R + \mu = 0 \quad \text{(a)}$$

$$\frac{\lambda IS}{1+\alpha I^2} - (d+m)I = 0 \quad \text{(b)}$$

$$mI - (d+\beta)R = 0 \quad \text{(c)}$$

From (b) & (c) $R = \left[\frac{m}{d+\beta}\right] I$ and $S = \frac{(d+m)(1+\alpha I^2)}{\lambda}$

Substituting R and S in the equation (a), we get

$$[\alpha d (d+m)]I^2 + \left[\lambda\left(d+m-\frac{\beta m}{d+\beta}\right)\right] I - d(d+m) - \lambda(a+\mu) = 0$$

$$I = \frac{-\lambda\left[d+m-\frac{\beta m}{d+\beta}\right]}{2\alpha d(d+m)} + \sqrt{\frac{\lambda^2\left(d+m-\frac{\beta m}{d+\beta}\right)^2 - 4\alpha d^2(d+m)^2\left[1-\frac{\lambda(a+\mu)}{d(d+\beta)}\right]}{4\alpha^2 d^2 (d+m)^2}}$$

We define the basic reproduction number as follows

$$R_0 = \frac{\lambda(a+\mu)}{d(d+m)} \quad (7)$$

From equation (7), we see that

- If $R_0 \le 1$, then there is no positive equilibrium;
- If $R_0 > 1$, then there is a unique positive equilibrium $E^* = (S^*, I^*, R^*)$, called the

Endemic equilibrium and is given by

$$S^* = \frac{(d+m)(1+\alpha I^{*2})}{\lambda} \quad (8)$$

$$I^* = \frac{-\lambda\left(d+m-\frac{\beta m}{d+\beta}\right) + \sqrt{\Delta}}{2\alpha d(d+m)} \quad (9)$$

and $R^* = \frac{m}{d+\beta} I^*$ (10)

where $\Delta = \lambda^2\left(d+m-\frac{\beta m}{d+\beta}\right)^2 - 4\alpha d^2(d+m)^2[1-R_0]$ (11)

In the next section, we shall study the property of these equilibria and perform a global qualitative analysis of model (6).

3. MATHEMATICAL ANALYSIS

To study the dynamics of model (6), we first present a lemma.

Lemma 3.1: The plane S + I + R = $(a+\mu)$/d is an invariant manifold of system (6), which is attracting in the first octant.

Proof: Summing up the three equations in (6) and denoting N(t)=S(t)+I(t)+R(t), we have

$$\frac{dN}{dt} = a + \mu - dN \tag{12}$$

It is clear that N(t) = S(t)+I(t)+R(t) = $(a+\mu)$/d is a solution of equation (12) and for any $N(t_0) \geq 0$, the general solution of equation (12) is

$$N(t) = \frac{a+\mu}{d} - \frac{(a+\mu-dN(t_0))\, e^{-d(t-t_0)}}{d}$$

$$\Rightarrow \lim_{t\to\infty} N(t) = \frac{a+\mu}{d}$$

which implies the conclusion.

It is clear that the limit set of system (6) is on the plane S+I+R = $(a+\mu)$/d. Thus, we focus on the reduced system

$$\begin{aligned} \frac{dI}{dt} &= \frac{\lambda I}{1+\alpha I^2}\left(\frac{a+\mu}{d} - I - R\right) - (d+m)\, I = P(I, R) \\ \frac{dR}{dt} &= mI - (d+\beta)R = Q(I, R) \end{aligned} \tag{13}$$

We have the following result regarding the nonexistence of periodic orbits in system (13), which implies the non-existence of periodic orbits of system (6) by Lemma.

Theorem 3.2: System (13) does not have nontrivial periodic orbits.

Proof: Consider the system (13) for I > 0 and R > 0. Take a Dulac function

$$D(I,R) = \frac{1+\alpha I^2}{\lambda I}$$

We have

$$\frac{\partial(DP)}{\partial I} + \frac{\partial(DQ)}{\partial R} = -1 - \frac{2\alpha(d+m)}{\lambda} I - \frac{1+\alpha I^2}{\lambda I}(d+\beta) < 0$$

The conclusion follows.

In order to study the properties of the disease-free equilibrium E_0 and the endemic equilibrium E^*, we rescale (13) by

$x = \frac{\lambda}{d+\beta} I$, $y = \frac{\lambda}{d+\beta} R$, $T = (d+\beta)t$

Then we obtain

$$\begin{aligned} \frac{dx}{dT} &= \frac{x}{1+vx^2}(K - x - y) - ux \\ \frac{dy}{d\tau} &= wx - y \end{aligned} \tag{14}$$

where $v = \frac{\alpha(d+\beta)^2}{\lambda^2}$, $K = \frac{(a+\mu)\lambda}{d(d+\beta)}$, $u = \frac{d+m}{d+\beta}$, $w = \frac{m}{d+\beta}$

Note that the trivial equilibrium (0,0) of system (14) is the disease-free equilibrium E_0 of model (6) and the unique positive equilibrium (x^*, y^*) of system (14) is the endemic equilibrium E^* of model (6) if and only if $u - K < 0$, where

$$x^* = \frac{-(1+w) + \sqrt{(1+w)^2 - 4uv(u-K)}}{2uv} \text{ and } y^* = wx^*$$

We first determine the stability and topological type of (0, 0). The Jacobian matrix of system (14) at (0, 0) is

$$M_0 = \begin{bmatrix} K-u & 0 \\ w & -1 \end{bmatrix}$$

If $K - u = 0$, then there exists a small neighborhood N_0 of (0, 0) such that the dynamics of system (14) is equivalent to

$$\begin{aligned} \frac{dx}{dT} &= -x^2 - xy + O((x,y)^3) \\ \frac{dy}{dT} &= wx - y \end{aligned} \tag{15}$$

We know that (0, 0) is a saddle-node. Hence, we obtain the following result.

Theorem 3.3: The disease-free equilibrium (0,0) of system (14) is

- A stable hyperbolic node if $u - K > 0$,
- A saddle-node if $u - K = 0$,
- A hyperbolic saddle if $u - K < 0$.

When $u - K < 0$, we discuss the stability and topological type of the endemic equilibrium (x^*, y^*). The Jacobian matrix of (14) at (x^*, y^*) is

$$M_1 = \begin{bmatrix} \frac{x^*(vx^{*2} + 2vwx^{*2} - 2Kvx^* - 1}{(1+vx^{*2})^2} & \frac{-x^*}{(1+vx^{*2})^2} \\ w & -1 \end{bmatrix}$$

We have that

$$\det(M_1) = \frac{x^*(1+w+2Kvx^* - (1+w)vx^{*2})}{(1+vx^{*2})^2}$$

The sign of det (M_1) is determined by

$$P_1 = (1+w+2Kvx^* - (1+w)vx^{*2})$$

Note that $uvx^{*2} + (1+w)x^* + u - K = 0$. We have

$$uP_1 = (2Kuv + (1+w)^2)\left[x^* + \frac{(1+w)(2u-K)}{2Kuv+(1+w)^2}\right]$$

Now substituting $x^* = \frac{-(1+w)+\Delta_1}{2uv}$

where $\Delta_1 = \sqrt{(1+w)^2 - 4uv(u-K)}$,into P_1 and using a straightforward calculation, we've

$$P_1 = -\frac{\Delta_1}{2u^2v}\left[(1+w)\Delta_1 - [2Kuv+(1+w)^2]\right] = \frac{(1+w)\Delta_1}{2u^2v}\left[\left(1+w+\frac{2Kuv}{1+w}\right)-\Delta_1\right].$$

Since $\left(1+w+\frac{2Kuv}{1+w}\right)^2 - \Delta_1^2 = \frac{4u^2v^2K^2}{(1+w)^2} + 4u^2v > 0$, it follows that $P_1 > 0$. Hence, $\det(M_1) > 0$ and (x^*,y^*) is a node or a focus or a center. Furthermore, we have the following result on the stability of (x^*,y^*).

Theorem 3.4: Suppose $u-K < 0$, then there is a unique endemic equilibrium (x^*,y^*) of model (14), which is a stable node.

Proof: We know that the stability of (x^*,y^*) is determined by trace(M_1). We have

$$\text{trace}(M_1) = \frac{-v^2x^{*4} + (1+2w)vx^{*3} - 2(1+K)vx^{*2} - x^* - 1}{(1+vx^{*2})^2}$$

The sign of trace (M_1) is determined by

$$P_2 = -v^2x^{*4} + (1+2w)vx^{*3} - 2(1+K)vx^{*2} - x^* - 1$$

We claim that $P_2 \neq 0$. To see this, note that $uvx^{*2} + (1+w)x^* + u - K = 0$. Then we have

$u^3vP_2 = (B_1K + B_2)x^* + (B_3K + B_4)$,

where $B_1 = uv(2+3u+2w+4uw)$,

$B_2 = (1+w)[(1+w)^2+u(1+w)(1+2w)-2u^3v]$,

$B_3 = -(1+w)^2-u(1+w)(1+2w)+2u^3v$,

$B_4 = u[(1+w)^2+u(1+w)(1+2w)-v(1+2u)K^2]$.

When $u - K < 0$ we can see that $B_1K + B_2 > 0$:

Let $\xi = uv\,x^{*2} + (1+w)\,x^{*} + u - K$.

Similarly, we have

$(B_1K + B_2)^2\,\xi = u^3vpP_2 + P_3$, where p is a polynomial of x^* and

$S_3 = u^3v(1+K^2v+2w+w^2)$

$[(K+2Ku-2u^2)^2v+(1+K-u+w)(1+u+w+2uw)]$.

Assume that $P_2 = 0$. Since $\xi = 0$, it follows that $P_3 = 0$. However, when $u - K < 0$; we have $P_3 > 0$. Therefore, $P_2 \neq 0$ for any positive value of the parameters v, w and K, that is, trace $(M_1) \neq 0$. Thus, $u - K < 0$ implies that (x^*,y^*) does not change stability.

Take u = 1, K = 2, v = 1, w = 1. Then $x^* = -1 + \sqrt{2}$, $y^* = -1 + \sqrt{2}$,

Trace $(M_1) = -1.4645 < 0$.

By the continuity of trace (M_1) on the parameters, we know that trace $(M_1) < 0$ for

$u - K < 0$. This completes the proof.

Theorem 3.5: Let R_0 be defined by (8).

- If $R_0 < 1$, then model (6) has a unique disease-free equilibrium
- $E_0 = ((a+\mu)/d, 0, 0)$, which is a global attractor in the first octant.
- If $R_0 = 1$, then model (6) has a unique disease-free equilibrium
- $E_0 = ((a+\mu)/d, 0, 0)$, which attracts all orbits in the interior of the first octant.
- If $R_0 > 1$, then model (6) has two equilibria, a disease-free equilibrium

$E_0 = ((a+\mu)/d,0,0)$, and an endemic equilibrium $E^* = (S^*,I^*,R^*)$. The endemic equilibrium E^* is a global attractor in the interior of the first octant.

4. NUMERICAL SIMULATION

Let $\alpha = 4$ (constant), a = 1.0, d = 0.2, $\lambda = 0.2$, $\beta = 0.3$, m = 0.1, η varies from 0.1 to 1.5

Table 1

μ	a+μ	R_0	I^*	S^*	R^*
0.1	1.1	3.1421	0.6449	0.1935	4.6615
0.2	1.2	3.4285	0.6918	0.2075	5.1006
0.3	1.3	3.7142	0.7361	0.2208	5.5430
0.4	1.4	4.0000	0.7781	0.2334	5.9884
0.5	1.5	4.2857	0.8182	0.2454	6.4363
0.6	1.6	4.5714	0.8566	0.2569	6.8864
0.7	1.7	4.8570	0.8935	0.2680	7.3384
0.8	1.8	5.1428	0.9290	0.2787	7.7922
0.9	1.9	5.2485	0.9634	0.2890	8.2475

Table 1: Contd.,					
1.0	2.0	5.7142	0.9967	0.2990	8.7042
1.1	2.1	6.0000	1.0290	0.3087	9.1622
1.2	2.2	6.2857	1.0604	0.3181	9.6214
1.3	2.3	6.5714	1.0909	0.3273	10.0817
1.4	2.4	6.8751	1.1207	0.3362	10.5430
1.5	2.5	7.1428	1.1498	0.3449	11.0051

From this table we infer that, when α kept constant ($\alpha = 4$) and if μ varies from

0.1 to 1.5, we see that the endemic equilibrium $E^* = (S^*, I^*, R^*)$, monotonically increases for increasing values of α.

Annexure 2

Let $\mu = 0.5$ (constant), α varies from 1 to 15, $a = 1.0$, $d = 0.2$,

$\lambda = 0.2$, $\beta = 0.3$, $m = 0.15$, $\mu = 0.5$, $a + \mu = 1.5$ and $R_0 = 4.2857$

Table 2

α	I^*	R^*	S^*
1	1.4788	0.4436	5.5775
2	1.1094	0.3328	6.0577
3	0.9300	0.2790	6.2910
4	0.8182	0.2454	6.4363
5	0.7397	0.2219	6.5384
6	0.6806	0.2041	6.6152
7	0.6341	0.1902	6.6756
8	0.5961	0.1788	6.7250
9	0.5643	0.1693	6.7664
10	0.5372	0.1611	6.8016
11	0.5138	0.1541	6.8321
12	0.4932	0.1479	6.8588
13	0.4749	0.1424	6.8826
14	0.4586	0.1375	6.9038
15	0.4438	0.1331	6.9231

From this table we infer that, when α is raised ($\alpha = 1,2,3,\ldots,15$) and if μ is constant ($\mu = 0.5$), the endemic equilibrium I^* and R^* monotonically decreases for increasing values of α, while S^* monotonically increases for increasing values of α.

5. GRAPHICAL REPRESENTATION

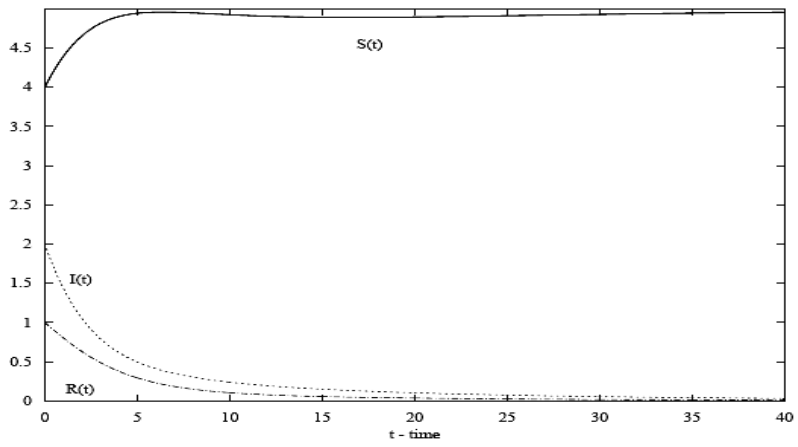

Figure 2

When $a+\mu = 1.0$, $d = 0.2$, $\lambda = 0.2$, $\alpha = 4.0$, $\beta = 0.3$, $m = 0.15$, $R_0 = 6/7 < 1$, S(t) approaches to its steady state value while I(t) and R(t) approach zero as time goes to infinity, the disease dies out. Here we proposed a non-monotone incidence rate of the form $\lambda IS = (1+\alpha I^2)$, which is increasing when I is small and decreasing when I is large. It can be used to interpret the "psychological" effect: the number of effective contacts between infective individuals and susceptible individual decreases at high infective levels due to the quarantine of infective individuals or the protection measures by the susceptible individuals. The recent epidemic outbreak of severe acute respiratory syndrome (SARS) had such psychological effects on the general public.

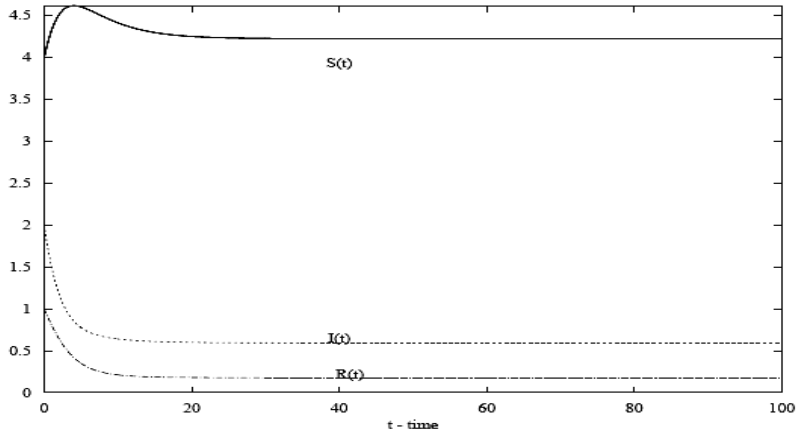

Figure 3

When $a+\mu = 1.0$, $d = 0.2$, $\lambda = 0.2$, $\alpha = 4.0$, $\beta = 0.3$, $m = 0.15$, $R_0 = 20/7 > 1$, all three components, S(t), I(t) and R(t), approach to their steady state values as time goes to infinity, the disease becomes endemic. We have carried out a global qualitative analysis of an SIR model with this non-monotone and nonlinear incidence rate and studied the existence and stability of the disease-free and endemic equilibria. It indicates that when $R_0 < 1$; the disease-free equilibrium is globally attractive (see Figure 2). When $R_0 > 1$; the endemic equilibrium exists and is globally stable (see Figure 3).

CONCLUDING REMARKS

SIR Epidemic model with non-monotone incidence rate describes the psychological effect of certain serious diseases on the community when the number of infective is getting larger. By carrying out a global analysis of the model and studying the stability of the disease-free equilibrium and the endemic equilibrium, we show that either the number of infective individuals tends to zero as time evolves or the disease persists.

From this model, the basic reproductive number R_0 has been introduced though the basic reproductive number R_0 does not depend on α explicitly, numerical simulations indicate that when the disease is endemic, I^* of the infective decreases as α increases. From (10) we see that I^* approaches zero as α tends to infinity.

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