Rich dynamics of an SIR epidemic model

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Abstract. This paper aims to study an SIR epidemic model with an asymptotically homogeneous transmission function. The stability of the disease-free and the endemic equilibrium is addressed. Numerical simulations are carried out. Implications of our analytical and numerical findings are discussed critically.

1 Introduction

Over the past one hundred years, mathematics has been used to understand and predict the spread of diseases, relating important public-health questions to basic transmission parameters. From prehistory to the present day, diseases have been a source of fear and superstition. A comprehensive picture of disease dynamics requires a variety of mathematical tools, from model creation to solving differential equations to statistical analysis. Although mathematics has been so far done quite well in dealing with epidemiology but there is no denying that there are certain factors which still lack proper mathematization.

Almost all mathematical models of diseases start from the same basic premise: that the population can be subdivided into a set of distinct classes, dependent upon their experience with respect to the disease. One line of investigation classifies individuals as one of susceptible, infectious or recovered. Such a model is termed as an SIR model. The first SIR model, which computes the theoretical number of individuals infected with a contagious illness in a closed population over time, was proposed by Kermack and

McKendrick in the year 1927 [1]. The Kermack-McKendrick model is given by

$$
\frac{\mathrm{d}S}{\mathrm{d}t} = -rSI,
$$
$$
\frac{\mathrm{d}I}{\mathrm{d}t} = rSI - \lambda I, \tag{1}
$$
$$
\frac{\mathrm{d}R}{\mathrm{d}t} = \lambda I,
$$

where $S(t), R(t), I(t)$ represent the number of susceptible, infective, and recovered individuals at time t, respectively. The parameters r and λ are called transmission rate and recovery rate, respectively. The interpretation of this model is straightforward. The population of susceptible (healthy) individuals diminishes through their interaction with the infective ones, the number of which correspondingly increases through the mechanism. On the other hand, the population of infective individuals diminishes since some individuals are cured, and thus populate the class of recovered. A detailed history of mathematical epidemiology and basics of SIR epidemic models may be found in the classical books of Bailey [2], Murray [3], and Anderson and May [4]. After Kermack–McKendrick model, different epidemic models have been proposed and studied in the literature (see Capasso and Serio [5], Hethcote and Tudor [6], Liu et al. [7,8], Hethcote et al. [9], Hethcote and van den Driessche [10], Derrick and van den Driessche [11], Beretta and Takeuchi [12,13], Beretta et al. [14], Ma et al. [15,16], Ruan and Wang [17], Song and Ma [18], Song et al. [19], D'Onofrio et al. [20], Xiao and Ruan [21], and references cited there in).

Disease transmission is a dynamical process driven by the interaction between the susceptible and the infective. The behaviour of the SIR models are greatly affected by the way in which transmission between infected and susceptible individuals are modelled. Many models of epidemiology are based on the so called "mass action" assumption for transmission. During the last few decades, such assumptions have faced some questions (see McCallam et al. [22], and references there in) and consequently a number of realistic transmission functions have become the focus of considerable attention (Capasso and Serio [5], Liu et al. [7,8], Hethcote et al. [9], Hethcote and van den Driessche [10], Ruan and Wang [17], Xiao and Ruan [21]). Usually, if the degree of infectivity increases, sociological, psychological, or other mechanisms often come into the picture which have a saturation effect (Busenberg and Cooke [23]). In recent outbreaks of SARS, mask wearing, quarantine, isolation, et cetera have been proved to be effective (Gumel et al. [24], Wang and Ruan [25]). Actual epidemics differ considerably from the idealized model (as was shown by the SARS outbreak of 2002–2003) if public health responses are not taken into consideration (Brauer [26]). Isolating sick children and workers from the rest of the population, the spread of the measles can be checked. Quarantine and isolation are very well known protective measures for certain diseases. These are immensely useful if a small pox outbreak occurred (Wallace [27]). Plague prevention depends on the timely implementation of preventive measures, including public education, applying insecticides to kill fleas, using various personal protective measures (e.g., common insect repellents), and avoidance of sick or dead animals (see report of CDC on *prevention of plague* in

MMWR in the year 1996 [28]).

One fundamental parameter governs the spread of diseases, and is also related to the long term behaviours and the level of vaccination necessary for eradication. This parameter is called the "basic reproduction number", R_0. R_0 is defined by epidemiologists as "the average number of secondary cases caused by an infectious individual in a totally susceptible population". When $R_0 > 1$, the disease can enter a totally susceptible population and the number of cases will increase, whereas when $R_0 < 1$, the disease will always fail to spread. Therefore, in its simplest form R_0 tells us whether a population is at risk from a given disease. Nowadays, the results of many epidemiological research are presented in terms of basic reproduction number.

In this paper, we have considered an SIR epidemic model with an asymptotically homogeneous transmission function (i.e., for large population sizes the transmission rate will be approximately proportional to the fraction of infectives in the total population). The paper is organized as follows. In the next section, we present the model and derive the disease-free equilibrium and the endemic equilibrium. In the third section, we carry out a qualitative analysis of the model. Stability conditions for the disease-free equilibrium and the endemic equilibrium are derived. The fourth section presents different computer simulations of the system. In the last section, the biological significance of our analytical and numerical findings are discussed.

2 The basic mathematical model

The model we analyze in this paper is considered under the framework of the following nonlinear ordinary differential equations:

$$
\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= b - dS - \frac{kSI}{1 + \alpha S + \beta I} + \gamma R, \\
\frac{dI}{dt} &= \frac{kSI}{1 + \alpha S + \beta I} - (d + \mu)I, \\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \mu I - (d + \gamma)R,
\end{aligned}
\tag{2}
$$

where $S(t), R(t), I(t)$ represent the number of susceptible, infective, and recovered individuals at time t, respectively. b is the recruitment rate of the population, d is the natural death rate of the population, k is the proportionality constant, μ is the natural recovery rate of the infective individuals, γ is the rate at which recovered individuals lose immunity and return to the susceptible class, α and β are the parameters which measure the effects of sociological, psychological or other mechanisms.

The transmission rate $\phi = kI/(1 + \alpha S + \beta I)$ displays a saturation effect accounting for the fact that the number of contacts an individual reaches some maximal value due to spatial or social distribution of the population. The transmission function considered by Diekmann and Kretzschmar [29], and Zegeling and Kooij [30] is a particular case (when $\alpha = \beta$) of the transmission rate considered here.

Because of the biological meaning of the components $(S(t), R(t), I(t))$, we focus on the model in the first octant of $\mathbb{R}^3$. We first consider the existence of equilibria of system (2). For any values of parameters, model (2) always has a disease-free equilibrium $E_0 = (b/d, 0, 0)$. To find the positive equilibria, set

$$
\begin{aligned}
& b - dS - kSI[1 + \alpha S + \beta I] + \gamma R = 0, \\
& kSI[1 + \alpha S + \beta I] - (d + \mu)I = 0, \\
& \mu I - (d + \gamma)R = 0.
\end{aligned}
\tag{3}
$$

Define the basic reproduction number as follows:

$$
R_0 = \frac{bk - b\alpha(d + \mu)}{d(d + \mu)}.
\tag{4}
$$

Theorem 1. *From the system* (4) *it follows that*

(i) *if $R_0 \leq 1$, then there is no positive equilibrium;*

(ii) *if $R_0 > 1$, then there is a unique positive equilibrium $E^*(S^*, I^*, R^*)$ of the system* (2), *called the "endemic equilibrium", given by*

$$
\begin{aligned}
S^* &= \frac{(d + \mu)(1 + \beta I)}{k - \alpha(d + \mu)}, \\
I^* &= \frac{(d + \gamma)[bk - (d + \mu)(b\alpha + d)]}{\beta d(d + \mu)(d + \gamma) + d(d + \mu + \gamma)(k - \alpha(d + \mu))}, \\
R^* &= \frac{\mu I}{(d + \gamma)}.
\end{aligned}
$$

3 Mathematical analysis

Lemma 1. *The plane $S + I + R = b/d$ is a manifold of system* (2), *which is attracting in the first octant.*

Proof. Summing up the three equations in (2) and denoting $N(t) = S(t) + I(t) + R(t)$, we have

$$
\frac{\mathrm{d}N}{\mathrm{d}t} = b - dN.
\tag{5}
$$

It is clear that $N(t) = b/d$ is a solution of equation (5) and for any $N(t_0) \geq 0$, the general solution of equation (5) is

$$
N(t) = \frac{1}{d}\left[b - \left(b - dN(t_0)\right)e^{-d(t - t_0)}\right].
$$

Thus,

$$
\lim_{t \to \infty} N(t) = \frac{b}{d},
$$

which implies the conclusion. $\qquad\square$

It is clear that the limit set of system (2) is on the plane $S + I + R = b/d$. Thus, we focus on the reduced system

$$
\begin{aligned}
\frac{\mathrm{d}I}{\mathrm{d}t} &= \frac{dkI}{(d + \alpha b) + (\beta - \alpha)dI - \alpha dR} \left(\frac{b}{d} - I - R \right) - (d + \mu)I \equiv P(I, R), \\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \mu I - (d + \gamma)R \equiv Q(I, R).
\end{aligned}
\tag{6}
$$

Theorem 2. *System* (6) *does not have nontrivial periodic orbits if*

$$
(2d + \gamma + \mu)(\beta - \alpha) > \mu\alpha.
$$

Proof. Consider system (6) for $I > 0$ and $R > 0$. Take a Dulac function (Perko [31], Strogatz [32], Wiggins [33])

$$
D(I, R) = \frac{(d + \alpha b) + (\beta - \alpha)dI - \alpha dR}{dkI}.
$$

Notice that

$$
\frac{\partial(DP)}{\partial I} + \frac{\partial(DQ)}{\partial R} = -1 - \frac{(d + \gamma)(d + \alpha b)}{dkI} - \frac{1}{k}[(2d + \gamma + \mu)(\beta - \alpha) - \mu\alpha] < 0
$$

if

$$
(2d + \gamma + \mu)(\beta - \alpha) > \mu\alpha.
$$

Hence, the conclusion follows. $\square$

In order to study the properties of the disease-free equilibrium E_0 and the endemic equilibrium E^*, we rescale (6) by

$$
x = \frac{k}{d + \gamma}I, \quad y = \frac{k}{d + \gamma}R, \quad \tau = (d + \gamma)t.
$$

Then we obtain

$$
\begin{aligned}
\frac{\mathrm{d}x}{\mathrm{d}\tau} &= \frac{px}{1 + qx - ry}(A - x - y) - mx, \\
\frac{\mathrm{d}y}{\mathrm{d}\tau} &= sx - y,
\end{aligned}
\tag{7}
$$

where

$$
\begin{aligned}
p &= \frac{d}{d + \alpha b}, \quad q = \frac{(d + \gamma)d(\beta - \alpha)}{(d + \alpha b)k}, \quad r = \frac{\alpha(d + \gamma)d}{(d + \alpha b)k}, \\
A &= \frac{bk}{d(d + \gamma)}, \quad m = \frac{d + \mu}{d + \gamma}, \quad s = \frac{\mu}{d + \gamma}.
\end{aligned}
$$

The trivial equilibrium $(0,0)$ of system (7) is the disease-free equilibrium E_0 of model (2) and the unique positive equilibrium (x^*, y^*) of system (7) is the endemic equilibrium E^* of model (2) if and only if $Ap - m > 0$ and $q - rs > 0$, where

$$x^* = \frac{Ap - m}{p(1 + s) + m(q - rs)}, \quad y^* = sx^*.$$

We first determine the stability and topological type of $(0,0)$. The Jacobian matrix of system (7) at $(0,0)$ is

$$M_0 = \begin{bmatrix} Ap - m & 0 \\ s & -1 \end{bmatrix}.$$

If $Ap - m = 0$, then there exists a small neighbourhood N_0 of $(0,0)$ such that the dynamics of system (7) are equivalent to that of

$$\begin{aligned} \frac{\mathrm{d}x}{\mathrm{d}\tau} &= -px^2 + o((x,y)^2), \\ \frac{\mathrm{d}y}{\mathrm{d}\tau} &= sx - y. \end{aligned} \tag{8}$$

Theorem 3. *The disease-free equilibrium $(0,0)$ of system (7) is*

(i) *a stable hyperbolic node if $m - Ap > 0$;*

(ii) *a saddle-node if $m - Ap = 0$;*

(iii) *a hyperbolic saddle if $m - Ap < 0$.*

When $m - Ap < 0$, we discuss the stability and topological type of the endemic equilibrium (x^*, y^*). The Jacobian matrix of the system (7) at (x^*, y^*) is

$$M_1 = \begin{bmatrix} \frac{p[sx^*(r+q)-(1+Aq)]}{(1+qx^*-rsx^*)^2} & \frac{p[(Ar-1)-x^*(q+r)]}{(1+qx^*-rsx^*)^2} \\ s & -1 \end{bmatrix}.$$

We have that

$$\det(M_1) = \frac{p[1 + s + (q - rs)]}{(1 + qx^* - rsx^*)^2}.$$

Since $q > rs$, it follows that $\det(M_1) > 0$ 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 4. *Suppose $m - Ap < 0$, then there is a unique endemic equilibrium (x^*, y^*) of model (7), which is a stable node.*

Proof.

$$\text{tr}(M_1) = \frac{ps(r+q)x^* - p(1+Aq) - [x^*(rs-q)-1]^2}{(1+qx^* - rsx^*)^2}.$$

The sign of $\text{tr}(M_1)$ is determined by

$$S_1 = ps(r+q)x^* - p(1+Aq).$$

Substituting $x^* = \frac{Ap-m}{p(1+s)+m(q-rs)}$ into S_1 and using a straightforward calculation, we have

$$S_1 = \frac{p[-A(p+mq)(q-rs) - (mqs+mq+p+ps)]}{p(1+s)+m(q-rs)}.$$

Since $q > rs$, $[p(1+s)+m(q-rs)] > 0$ and $[-A(p+mq)(q-rs) - (mqs+mq+p+ps)] < 0$, hence, $S_1 < 0$. However, when $m - Ap < 0$, we have $\text{tr}(M_1) < 0$. This completes the proof. $\qquad\square$

In terms of the basic reproduction number, the above results on stability are summarized below.

Theorem 5. *Let R_0 be defined by (4).*

(i) *If $R_0 < 1$, then model (2) has a unique disease-free equilibrium $E_0 = (b/d, 0, 0)$, which is a global attractor in the first octant.*

(ii) *If $R_0 = 1$, then model (2) has a unique disease-free equilibrium $E_0 = (b/d, 0, 0)$, which attracts all orbits in the interior of the first octant.*

(iii) *If $R_0 > 1$, then model (2) has two equilibria, a disease-free equilibrium $E_0 = (b/d, 0, 0)$ and an endemic equilibrium $E^* = (S^*, I^*, R^*)$. The endemic equilibrium E^* is a global attractor in the interior.*

4 Numerical simulation

In this section we present computer simulation of some solutions of the system (2). From practical point of view, numerical solutions are very important beside analytical study.

We take the parameters of the system as $d = 2.29$, $\alpha = 3.1$, $\beta = 4.7$, $b = 3.1$, $\gamma = 1.5$, $k = 9$, $\mu = 0.19$, $(S(0), I(0), R(0)) = (4, 1, 1)$. Then $E_0 = (1.4104, 0, 0)$, $R_0 = 0.7162 < 1$. Therefore, by Theorem 1, E_0 is a global attractor in the first octant. Fig. 1 shows that $S(t)$ approaches to its steady-state value while $I(t)$ and $R(t)$ approach zero as time progresses, the disease dies out.

Now we take the parameters of the system as $d = 0.29$, $\alpha = 3.1$, $\beta = 4.7$, $b = 3.1$, $\gamma = 1.5$, $k = 6.5$, $\mu = 0.19$, $(S(0), I(0), R(0)) = (4, 1, 1)$. Then $E^*(S^*, I^*, R^*) = (3.1598, 6.8073, 0.72226)$ and $R_0 = 25.7167 > 1$. Therefore, by Theorem 1, the endemic equilibrium E^* is a global attractor in the interior of the first octant. Fig. 2 shows

that all three components, $S(t)$, $I(t)$ and $R(t)$ approach to their steady-state values, the disease becomes endemic.

Keeping other parameters fixed, if we change the value of β, it is seen that I^* decreases as β increases. It follows from Fig. 3.

Keeping other parameters fixed, if we change the value of α, it is seen that I^* increases as α dicreases. It follows from Fig. 4.
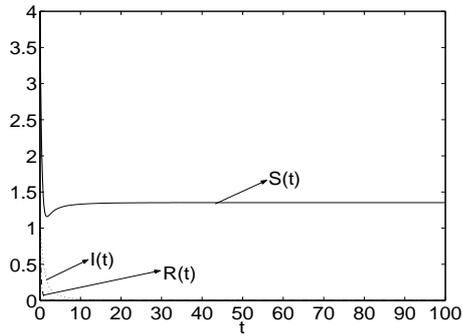
Fig. 1. Here $S(0) = 4$, $I(0) = 1$, $R(0) = 1$, $d = 2.29$, $\alpha = 3.1$, $\beta = 4.7$, $b = 3.1$, $\gamma = 1.5$, $k = 9$, $\mu = 0.19$, $R_0 = 0.7162 < 1$.
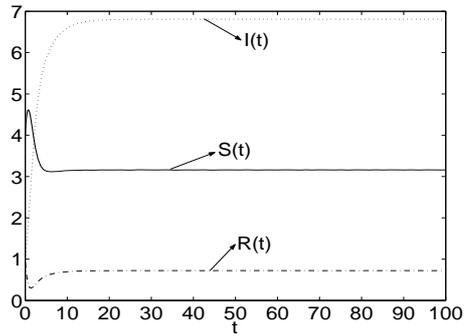
Fig. 2. Here $S(0) = 4$, $I(0) = 1$, $R(0) = 1$, $d = 0.29$, $\alpha = 3.1$, $\beta = 4.7$, $b = 3.1$, $\gamma = 1.5$, $k = 6.5$, $\mu = 0.19$, $R_0 = 25.7167 > 1$.
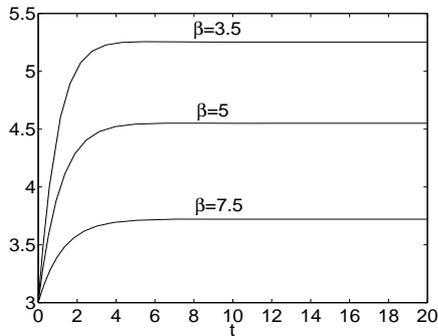
Fig. 3. The dependence of I^* on the parameter β keeping other parameters fixed.
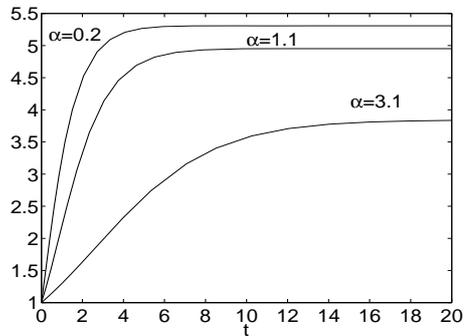
Fig. 4. The dependence of I^* on the parameter α keeping other parameters fixed.

5 Concluding remarks

At present, the entire globe is concerned about many infectious diseases which cause fearful tolls in different communities. Even today they are often attributed to evil spirits or

displeased gods. However, almost all the developing countries have increasingly realized the necessity of social consciousness in preventing the diseases. Also different protective measures against diseases are found to be effective.

One main goal of mathematical epidemiology is to understand how to control or eradicate diseases. Mathematical models are used extensively in the study of ecological and epidemiological phenomena. We all know that one of the main issue in the study of behaviour of epidemics is the analysis of steady states of the model and their stability. If the trivial or zero equilibrium is globally asymptotically stable, then, the disease does not persist, whatever the initial number of infectives in the population.

In this paper we have carried out the global qualitative analysis of a realistic SIR model. In terms of the basic reproduction number R_0 our main results indicate that when $R_0 < 1$, the disease-free equilibrium is globally attractive. When $R_0 > 1$, the endemic equilibrium exists and is globally stable. Though the basic reproduction number R_0 does not depend on β, numerical simulations indicate that when the disease is endemic, the steady state value I^* of the infectives decreases as β increases. This implies that the spread of disease decreases as the social or psychological protective measures for the infectives increases. From the steady state expression we can see that I^* approaches zero as β tends to infinity. These results are in good agreement with those of Xiao and Ruan [21]. From numerical simulations, we have also made another interesting observation that when the disease is endemic, I^* increases as α decreases. It implies that if the social consciousness about the disease decreases among the susceptibles, it might encourage to increase the infection rate and to spread the disease rapidly.

Acknowledgements

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