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Dynamics of an SIR Model with Nonlinear Incidence and Treatment Rate

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Abstract

In this paper, global dynamics of an SIR model are investigated in which the incidence rate is being considered as Beddington-DeAngelis type and the treatment rate as Holling type II (saturated). Analytical study of the model shows that the model has two equilibrium points (disease-free equilibrium (DFE) and endemic equilibrium (EE)). The disease-free equilibrium (DFE) is locally asymptotically stable when reproduction number is less than one. Some conditions on the model parameters are obtained to show the existence as well as nonexistence of limit cycle. Some sufficient conditions for global stability of the endemic equilibrium using Lyapunov function are obtained. The existence of Hopf bifurcation of model is investigated by using Andronov-Hopf bifurcation theorem. Further, numerical simulations are done to exemplify the analytical studies.

Keywords: SIR model; Beddington-DeAngelis type nonlinear incidence rate; Limit cycle; Hopf bifurcation; Next generation matrix method; Central manifold theory

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1. Introduction

Epidemiological models have been recognized as valuable tools in analyzing the spread and control of infectious diseases. In epidemiological models, incidence rate as well as treatment rate play an important role while analyzing the transmission of diseases. The number of individuals who become infected per unit of time in epidemiology is called incidence rate. Incidence rate has been defined in multiple ways. Firstly, the bilinear incidence rate (Anderson and May (1992), Bailey (1975), Brauer and Castillo-Chavez (2001), Hethcote (2004), Kermack and McKendrick (1927), Shulgin et al. (1998), Zhang and Suo (2010), Ghosh et al. (2004), Shukla et al. (2011)) is based on the law of mass action (βSI, where β is infection rate and S and I denote the susceptible and infected individuals, respectively) which is unreasonable for large population. As we can infer from the term βSI that if the number of susceptibles increases, the number of individuals who become infected per unit of time increases, which is not realistic. So there is a need to modify the classical linear incidence rate to study the dynamics of infection among large population.

Several authors (Anderson and May (1978), Wei and Chen (2008), Zhang et al. (2008), Li et al. (2009), Li and Muldowney (1995), Korobeinikov and Maini (2005), Xu and Ma (2009), Capasso and Serio (1978)) suggested different types of nonlinear incidence rates. The saturated incidence rate $\frac{\alpha SI}{(1+\beta S)}$ was introduced by Anderson and May in 1978. The effect of saturation factor β stems from epidemical control. Further, many authors (Mondal and Kar (2013), Agarwal and Verma (2012), Wei and Chen (2008), Zhang et al. (2008)) incorporated this incidence rate into their models. Li et al. (2009) proposed an SIR model with nonlinear incidence rate given by $\frac{\alpha SI}{(1+\gamma I)}$. In this incidence rate the number of effective contacts between infective and susceptible individuals may saturate at high infective levels due to crowding of infective individuals. Beddington (1975) and DeAngelis (1975) independently introduced nonlinear incidence rate known as Beddington-DeAngelis type incidence rate $\left(\frac{\alpha SI}{1+\beta S+\gamma I}\right)$. Later, some authors (e.g. Kaddar (2009), Kaddar (2010), Huang et al. (2011), Elaiw and Azoz (2013)) used this incidence rate to describe epidemiological models.

We are aware of the fact that the treatment is an important method to reduce the spread of diseases. In classical epidemic models, the treatment rate of infected individuals is assumed to be either constant or proportional to the number of the infected individuals. However, we know that there are limited treatment resources available in community. Therefore, this is very important to choose a suitable treatment rate of a disease. In the absence of effective therapeutic treatments and vaccines, the epidemical control strategies are based on taking appropriate preventive measures. Wang and Ruan (2004) considered an SIR epidemic model with constant treatment rate (i.e., the recovery from infected subpopulation per unit time) as given below:

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

where r is a positive constant and I is the number of infected individuals. They studied stability analysis and showed that this model exhibits various bifurcations. Further, Zhou and Fan (2012)

modified the treatment rate to Holling type II

$$h(I) = \frac{\beta I}{(1+\gamma I)}, \quad I \geq 0, \quad \gamma \geq 0, \quad \beta \geq 0.$$

They have shown that, with varying amount of medical resources and their supply efficiency, the target model admits both backward bifurcation and Hopf bifurcation. Dubey et al. (2013) have also used Holling type II, III, and IV treatment rates to study their model.

To the best knowledge of the authors, an SIR model with Beddington-DeAngelis type incidence rate and the saturated treatment rate has not been considered. Taking these important facts into account and getting motivated from Kaddar's work (Kaddar (2009), Kaddar (2010)), we propose an SIR model with Beddington-DeAngelis type incidence rate and the saturated treatment rate.

This paper is organized as follows. After the abstract and introduction, Section 2 discusses the formulation of the mathematical model and well-posedness of the model. In Section 3, we discuss the equilibrium points of model (3), the stability of equilibrium points, and the existence of Hopf bifurcation. Further, in Section 4, numerical simulations are done to validate the analytical studies. Finally, Section 5 concludes this paper.

2. The Mathematical Model

We assume that the entire population is divided into three classes: susceptible individuals (S), infected individuals (I), and removed or recovered individuals (R). Susceptible individuals are those who are healthy and can contract disease under appropriate conditions. Infected individuals are the one who have contracted the disease and are now infected with it. These individuals are capable of transferring the disease to susceptible individuals via contacts. As time progresses, infected individuals lose infectivity and move to the removed or recovered compartment (by auto recovery due to immune response of the body or by treatment). These recovered individuals are immune to infectious microbes and thus do not acquire the disease again. The model is given by following differential equations:

$$\begin{cases} \frac{dS}{dt} = A - \delta_0 S - \frac{\alpha SI}{1+\beta S+\gamma I}, \\ \frac{dI}{dt} = \frac{\alpha SI}{1+\beta S+\gamma I} - \delta_0 I - \delta_1 I - \delta_2 I - \frac{aI}{1+bI}, \\ \frac{dR}{dt} = \delta_2 I - \delta_0 R + \frac{aI}{1+bI}, \end{cases} \tag{1}$$

where $S(0) > 0$, $I(0) \geq 0$, $R(0) \geq 0$.

Let the susceptibles be recruited at a constant rate A and δ_0 be the natural death rate of the population in each class. Let δ_1 be the death rate of infected individuals due to infection and δ_2 be the natural recovery rate of infected individuals due to immunity. In model (1), we take the incidence rate as Beddington-DeAngelis type:

$$f(S, I) = \frac{\alpha SI}{1+\beta S+\gamma I}. \tag{2}$$

Here α is the transmission rate, β is a measure of inhibition effect, such as preventive measure taken by susceptible individuals, and γ is a measure of inhibition effect such as treatment with respect to infectives. It is interesting to note that the following three types of incidence rates can be derived from the incidence rate proposed in this paper:

(1) If we set $\beta = \gamma = 0$, then $f(S, I) = \alpha SI$ which is bilinear incidence rate (Anderson and May (1992), Bailey (1975), Brauer and Castillo-Chavez (2001), Hethcote (2004), Kermack and McKendrick (1927), Shulgin et al. (1998), Zhang and Suo (2010)).
(2) If we set $\gamma = 0$, then $f(S, I) = \frac{\alpha SI}{(1+\beta S)}$, which is saturated incidence rate with the susceptible individuals. The inhibition effect due to the saturation factor β results due to the preventive measure to control the spread of epidemic (Korobeinikov and Maini (2005), Xu and Ma (2009), Capasso and Serio (1978)).
(3) If we set $\beta = 0$, then $f(S, I) = \frac{\alpha SI}{(1+\gamma I)}$, which is saturated incidence rate with the infected individuals. In such a case, the contact between infective and susceptible individuals may saturate at high infection level due to crowding of infective individuals or due to protection taken by susceptible individuals (Anderson and May (1978), Wei and Chen (2008), Zhang et al. (2008), Li et al. (2009), Mukhopadhyay and Bhattacharya (2008), Xue and Duan (2011), Liu et al. (1987), Li and Muldowney (1995)).

The term $h(I) = \frac{aI}{(1+bI)}$ in system (1) represents the treatment term, where a is a positive constant whereas b is a constant taking into account resource limitation (Zhang and Suo (2010), Zhou and Fan (2012)).

From the above system (1) we can infer that S and I are free from the effect of R. Thus it is enough to consider the following reduced system for the study:

$$\begin{cases} \frac{dS}{dt} = A - \delta_0 S - \frac{\alpha SI}{1+\beta S+\gamma I}, \\ \frac{dI}{dt} = \frac{\alpha SI}{1+\beta S+\gamma I} - \delta_3 I - \frac{aI}{1+bI}, \end{cases} \tag{3}$$

where $\delta_3 = \delta_0 + \delta_1 + \delta_2$ and $S(0) > 0$, $I(0) \geq 0$.

A. Positivity of the model

For the above system (3), we find a region of attraction which is given by Lemma 1.

Lemma 1.

The set $\Omega = \{(S, I) \in R^2_+ : 0 < S + I \leq \frac{A}{\delta_0}\}$ is a positively invariant region of system (3).

Proof:

Let $N = S + I$, then $\dot{N} = \dot{S} + \dot{I} = A - \delta_0 N - (\delta_1 + \delta_2)I - \frac{aI}{1+bI}$. Then,

$$N(t) \leq N(0)e^{-\delta_0 t} + \frac{A}{\delta_0}(1 - e^{-\delta_0 t}).$$

Thus, $\lim_{t\to\infty} \sup N(t) \leq \frac{A}{\delta_0}$. Furthermore, $\dot{N} < 0$ if $N > \frac{A}{\delta_0}$. This shows that solutions of system (3) point towards Ω. Hence Ω is positively invariant and solutions of (3) are bounded. □

The above lemma shows that all solutions of the model are non-negative and bounded. Thus the model is biologically well-behaved.

In the next section, first we find the equilibrium points of system (3), then discuss the existence and stability of equilibrium points of system (3).

3. Equilibrium points and their stability analysis

System (3) has only two equilibria: (i) the disease-free equilibrium (DFE) $E_0(S_0, I_0)$, i.e., there is no infection and (ii) the endemic equilibrium $E_1(S^*, I^*)$, i.e., infection persists. We can infer from system (3) that the disease-free equilibrium E_0 is trivial equilibrium point and given by $E_0(S_0, I_0) = E_0(\frac{A}{\delta_0}, 0)$.

To compute the basic reproduction number and to study the local stability of the DFE, we use the next generation matrix method (Diekmann et al. (1990), Van den Driessche and Watmough (2002)). Using the same notation as in Van den Driessche and Watmough (2002), we define $\dot{x} = F(x) - V(x)$, where $x = [I, S]^T$, $F(x)$ is the matrix of new infection terms, and $V(x)$ is the matrix of transfer terms into compartment and out of compartment. The Jacobian of matrices $F(x)$ and $V(x)$ at DFE $E_0(\frac{A}{\delta_0}, 0)$ is given by

$$F = \begin{bmatrix} \frac{\alpha A}{\delta_0 + A\beta} & 0 \\ 0 & 0 \end{bmatrix},$$

and

$$V = \begin{bmatrix} (\delta_3 + a) & 0 \\ \frac{\alpha A}{\delta_0 + A\beta} & \delta_0 \end{bmatrix}.$$

Then the spectral radius of new generation matrix (Van den Driessche and Watmough (2002)) (FV^{-1}) gives R_0 i.e.,

$$R_0 = \rho(FV^{-1}) = \frac{A\alpha}{(\delta_3 + a)(\delta_0 + A\beta)},$$

where R_0 is basic reproduction number, the number of newly infected individuals produced by a single infected person when introduced into a completely susceptible population. We conclude the following result using the above computation for R_0 and from Theorem 2 of the paper by Van den Driessche and Watmough (2002).

Theorem 1.

The disease-free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$ and is a saddle point with stable manifold locally in the S-direction and unstable manifold locally in the I-direction if $R_0 > 1$.

Epidemiologically, the above result depicts that small inflow of infected individuals will not be able to spread infection if $R_0 < 1$. In this case the spread of infection is dependent on initial sizes of sub-population. To ensure that the spread of infection is independent of initial sizes of sub-population, we study the global stability of the DFE in the next theorem.

Theorem 2.

(i) When $b = 0$, then the disease free equilibrium E_0 is globally asymptotically stable if $R_0 \leq 1$ and (ii) when $b \neq 0$, then the disease free equilibrium E_0 is globally asymptotically stable if $R_1 = \frac{\alpha A}{(\delta_0 + A\beta)\delta_3} \leq 1$.

Proof:

Let L be the Lyapunov function defined as

$$L = \frac{1}{1 + \beta S_0}\left(S - S_0 - S_0 ln\frac{S}{S_0}\right) + I, \quad \text{where} \quad S = \frac{A}{\delta_0}.$$

Differentiating L along the solutions of (3) and after simplification, we have

$$\dot{L}(t) = -\left[\frac{\delta_0(S - S_0)^2}{S(1 + \beta S_0)} + \frac{\alpha\gamma S_0 I^2}{(1 + \beta S + \gamma I)(1 + \beta S_0)}\right] + \frac{(\delta_3 + a)I}{(1 + \gamma I)}[R_0 - 1] + PI^2,$$

where $P = \frac{b}{1+bI}\left(\frac{\alpha S_0}{(1+\beta S_0)} - \delta_3\right)$.

Case I: $b = 0$
Then, clearly $P = 0$ and

$$\dot{L}(t) < 0 \text{ if } R_0 \leq 1 \text{ and } \dot{L}(t) = 0 \text{ iff } S = S_0 = \tfrac{A}{\delta_0} \text{ and } I = I_0 = 0.$$

Case II: $b \neq 0$
Then,

$$\dot{L}(t) < 0 \text{ if } P < 0 \text{ i.e., } \tfrac{\alpha A}{(\delta_0 + A\beta)} < \delta_3 \text{ and } \dot{L}(t) = 0 \text{ iff } S = S_0 = \tfrac{A}{\delta_0} \text{ and } I = I_0 = 0.$$

This implies that the largest compact invariant set in $\{(S, I) \in \Omega : \dot{L}(t) = 0\}$ is the singleton set $\{E_0\}$. From LaSalle's invariance principle (LaSalle (1976)) disease free equilibrium is globally asymptotically stable. □

Remark 1: (i) We observe that $R_0 < R_1$ (if $a > 0$) and $R_0 = R_1$ (if $a = 0$). (ii) When $R_1 \leq 1$, then $R_0 \leq 1$.

This implies that the threshold value for the disease eradication is less if there is no limitation on the medical resources availability in the community ($b = 0$). However, this threshold increases as the availability of the medical resources limits in the community ($b > 0$).

B. Analysis at $R_0 = 1$

In this section, we analyze the behavior of system (3) when the basic reproduction number is equal to one. We notice that the Jacobian matrix of system (3) evaluated at $R_0 = 1$ and $\alpha = \alpha^* = \frac{(\delta_3 + a)(\delta_0 + A\beta)}{A}$ has a simple zero eigenvalue and another eigenvalue with negative real part. Stability behaviour of equilibrium points at $R_0 = 1$ cannot be determined using linearization so we use Center manifold theory (Sastry (1999)). In order to apply center manifold theorem to

system (3), we made following assumptions: Let $S = x_1$ and $I = x_2$, then the system (3) can be rewritten as

$$\begin{cases} \frac{dx_1}{dt} = A - \delta_0 x_1 - \frac{\alpha x_1 x_2}{1+\beta x_1+\gamma x_2}, \\ \frac{dx_2}{dt} = \frac{\alpha x_1 x_2}{1+\beta x_1+\gamma x_2} - \delta_3 x_2 - \frac{a x_2}{1+b x_2}, \end{cases} \tag{4}$$

Let J be the Jacobian matrix at $R_0 = 1$ and $\alpha = \alpha^*$. Then

$$J = \begin{bmatrix} -\delta_0 & -\frac{\alpha^* A}{(\delta_0+A\beta)} \\ 0 & \frac{\alpha^* A}{(\delta_0+A\beta)} - \delta_3 - a \end{bmatrix}.$$

Let $w = [w_1, w_2]$ and $u = [u_1, u_2]^T$ be the left eigenvector and right eigenvector of J corresponding to the zero eigenvalue. Then we have

$$w_1 = 0, \quad w_2 = 1 \quad \text{and} \quad u_1 = -\frac{\alpha^* A}{(\delta_0 + A\beta)\delta_0}, \quad u_2 = 1.$$

The nonzero partial derivatives associated with the functions of the system (4) evaluated at $R_0 = 1$ and $\alpha = \alpha^*$ are

$$\left(\frac{\partial^2 f_2}{\partial x_1 \partial x_2}\right)_{E_0} = \frac{\alpha^*}{(1+\beta S_0)^2}, \quad \left(\frac{\partial^2 f_2}{\partial x_2^2}\right)_{E_0} = -\frac{2\alpha^*\gamma S_0}{(1+\beta S_0)^2}, \quad \left(\frac{\partial^2 f_2}{\partial x_2 \partial \alpha^*}\right)_{E_0} = \frac{S_0}{(1+\beta S_0)^2}.$$

Then from Theorem 4.1 of Castillo-Chavez and Song (2004), the bifurcation constants a_1 and b_1 are

$$\begin{aligned} a_1 &= \sum_{k,i,j=1}^{2} w_k u_i u_j \left(\frac{\partial^2 f_k}{\partial x_i \partial x_j}\right)_{E_0} \\ &= w_2 \left(u_1 u_2 \frac{\alpha^*}{(1+\beta S_0)^2} + u_2^2 \left(-\frac{2\alpha^*\gamma S_0}{(1+\beta S_0)^2}\right)\right) \\ &= -\frac{\alpha^*}{(1+\beta S_0)^2}\left(\frac{\alpha^* A}{(\delta_0 + A\beta)\delta_0} + 2\gamma S_0\right) < 0, \end{aligned}$$

and

$$b_1 = \sum_{k,i=1}^{2} w_k u_i \left(\frac{\partial^2 f_k}{\partial x_i \partial \alpha^*}\right)_{E_0} = w_2 \left(u_2 \frac{S_0}{(1+\beta S_0)^2}\right) = \frac{S_0}{(1+\beta S_0)^2} > 0.$$

Thus from Theorem 4.1(iv) of Castillo-Chavez and Song (2004), we conclude the following result.

Theorem 3.

The disease free equilibrium changes its stability from stable to unstable at $R_0 = 1$ and there exists a positive equilibrium as R_0 crosses one. Hence system (3) undergoes transcritical bifurcation at $R_0 = 1$.

C. The existence of endemic equilibrium $E_1(S^*, I^*)$

Equating the second equation of system (3) to zero, we have

$$\frac{\alpha S^* I^*}{1+\beta S^*+\gamma I^*} - \delta_3 I^* - \frac{aI^*}{1+bI^*} = 0. \tag{5}$$

After solving the above equation (5), we get S^* in terms of I^* as follows:

$$S^* = \frac{(\delta_3 + a + \delta_3 bI^*)(1+\gamma I^*)}{(\alpha - \delta_3\beta - a\beta) + (\alpha - \delta_3\beta)bI^*}. \tag{6}$$

S^* is positive if

$$\alpha > (\delta_3 + a)\beta. \tag{7}$$

Now equating the first equation of system (3) to zero and solving we get the following quadratic equation in S^*:

$$\delta_0\beta S^{*2} + (\delta_0 - A\beta + (\delta_0\gamma + \alpha)I^*)S^* - A(1+\gamma I^*) = 0. \tag{8}$$

Substituting the value of S^* from Equation (6) into Equation (8), we get the following cubic equation in I^*:

$$A_1 I^{*3} + A_2 I^{*2} + A_3 I^* + A_4 = 0, \tag{9}$$

where

$$A_1 = \delta_0\beta\gamma{\delta_3}^2 b^2 + \delta_3 b^2 pl,$$

$$A_2 = \delta_0\beta{\delta_3}^2 b^2 + 2\delta_0\delta_3(\delta_3 + a)\beta\gamma b + b\delta_3 ql + bp((\delta_3 + a)l + \delta_3 bm - Abp),$$

$$A_3 = 2\delta_0\delta_3(\delta_3 + a)\beta b + \delta_0\beta\gamma(\delta_3 + a)^2 + (\delta_3 + a)bmp + q((\delta_3 + a)l + \delta_3 bm - 2Abp),$$

$$A_4 = \delta_0\beta(\delta_3 + a)^2 + (\delta_3 + a)mq - Aq^2,$$

and

$$p = (\alpha - \delta_3\beta), \quad q = (\alpha - \delta_3\beta - a\beta), \quad l = (\delta_0\gamma + \alpha), \quad m = (\delta_0 - A\beta).$$

It may be noted that p, $q > 0$ under condition (7). Now using Descartes' rule of sign, the cubic equation (9) has unique positive real root I^* if any one of the following holds:
(i) $A_2 > 0$, $A_3 > 0$ and $A_4 < 0$,
(ii) $A_2 > 0$, $A_3 < 0$ and $A_4 < 0$,
(iii) $A_2 < 0$, $A_3 < 0$ and $A_4 < 0$.

We consider first two cases from which we have the following inequalities

$$(\delta_3 + a)l + \delta_0\delta_3 b > A\alpha b, \tag{10}$$

and

$$R_0 > 1. \tag{11}$$

After finding the value of I^*, we can find the value of S^* from equation (6). This implies that there exists a unique endemic equilibrium $E_1(S^*, I^*)$ if the inequalities (7), (10) and (11) are satisfied.

Theorem 4.

The endemic equilibrium $E_1(S^*, I^*)$ is locally asymptotically stable if and only if the following inequalities hold true:

$$\frac{\alpha S^*(1+\beta S^*)}{(1+\beta S^*+\gamma I^*)^2} < L_1, \tag{12}$$

$$\frac{\delta_0 \alpha S^*(1+\beta S^*)}{(1+\beta S^*+\gamma I^*)^2} < L_2, \tag{13}$$

where

$$L_1 = \delta_0 + \delta_3 + \frac{a}{(1+bI^*)^2} + \frac{\alpha I^*(1+\gamma I^*)}{(1+\beta S^*+\gamma I^*)^2},$$

$$L_2 = \left(\delta_3 + \frac{a}{(1+bI^*)^2}\right)\left(\delta_0 + \frac{\alpha I^*(1+\gamma I^*)}{(1+\beta S^*+\gamma I^*)^2}\right).$$

Proof:

The variational matrix corresponding to endemic equilibrium $E_1(S^*, I^*)$ is

$$M_{E_1} = \begin{bmatrix} -\delta_0 - \frac{\alpha I^*(1+\gamma I^*)}{(1+\beta S^*+\gamma I^*)^2} & -\frac{\alpha S^*(1+\beta S^*)}{(1+\beta S^*+\gamma I^*)^2} \\ \frac{\alpha I^*(1+\gamma I^*)}{(1+\beta S^*+\gamma I^*)^2} & \frac{\alpha S^*(1+\beta S^*)}{(1+\beta S^*+\gamma I^*)^2} - \delta_3 - \frac{a}{(1+bI^*)^2} \end{bmatrix}.$$

The characteristic polynomial of the above matrix is given by the following equation

$$\lambda^2 + a_1\lambda + a_2 = 0, \tag{14}$$

where

$$a_1 = \delta_0 + \frac{\alpha I^*(1+\gamma I^*)}{(1+\beta S^*+\gamma I^*)^2} - \frac{\alpha S^*(1+\beta S^*)}{(1+\beta S^*+\gamma I^*)^2} + \delta_3 + \frac{a}{(1+bI^*)^2},$$

$$a_2 = \left(\delta_3 + \frac{a}{(1+bI^*)^2}\right)\left(\delta_0 + \frac{\alpha I^*(1+\gamma I^*)}{(1+\beta S^*+\gamma I^*)^2}\right) - \frac{\delta_0 \alpha S^*(1+\beta S^*)}{(1+\beta S^*+\gamma I^*)^2}.$$

Using the Routh-Hurwitz criteria, it follows that eigenvalues of the above variational matrix have negative real parts if and only if $a_1 > 0$ and $a_2 > 0$. This implies that the endemic equilibrium $E_1(S^*, I^*)$ is locally asymptotically stable if and only if inequalities (12) and (13) hold true. □

Remark 2: If $\alpha = 0$, then conditions (12) and (13) are satisfied. This shows that if the transmission rate of infection is zero or very small, then E_1 is locally asymptotically stable.

Remark 3: If α is very large, then conditions (12) and (13) may not hold true. This implies that if the transmission rate of infection is large enough, then the endemic equilibrium may be unstable.

Remark 4: It may be noted that conditions (12) and (13) hold true if

$$\frac{\alpha S^*(1+\beta S^*)}{(1+\beta S^*+\gamma I^*)^2} < \delta_3 + \frac{a}{(1+bI^*)^2}.$$

From Equation (14), noting the sign of real parts of the eigenvalues λ, we can state the following two theorems, (5) and (6).

Theorem 5.

Let the following inequality hold true:

$$\frac{\delta_0 \alpha S^*(1+\beta S^*)}{(1+\beta S^*+\gamma I^*)^2} > L_2. \tag{15}$$

Then $E_1(S^*, I^*)$, whenever it exists, is a saddle point.

Theorem 6.

If inequality (13) and the following inequality hold true:

$$\frac{\alpha S^*(1+\beta S^*)}{(1+\beta S^*+\gamma I^*)^2} > L_1, \tag{16}$$

then $E_1(S^*, I^*)$, whenever it exists, is unstable.

In the following theorem, we are able to show the existence of a Hopf bifurcation under certain conditions.

Theorem 7.

Assume that:

$$\frac{\alpha S^*(1+\beta S^*)}{(1+\beta S^*+\gamma I^*)^2} = L_1, \tag{17}$$

and (13) hold true. Then system (3) exhibits Hopf bifurcation near $E_1(S^*, I^*)$.

Proof:

Condition (17) implies that $a_1 = 0$ in equation (14) and condition (13) implies that $a_2 > 0$. Thus, Equation (14) has purely imaginary roots. From Theorem 4 and Theorem 6, it follows that the positive equilibrium $E_1(S^*, I^*)$ changes its behavior from stability to instability as the parameter α passes through its critical value $\alpha = \alpha^*$, where

$$\alpha^* = \frac{(1+\beta S^*+\gamma I^*)^2}{S^*(1+\beta S^*) - I^*(1+\gamma I^*)} \left(\delta_0 + \delta_3 + \frac{a}{(1+bI^*)^2}\right).$$

Again we have

$$\begin{aligned}\frac{d}{d\alpha}[tr(M_{E_1})]_{\alpha=\alpha^*} &= \frac{S^*(1+\beta S^*) - I^*(1+\gamma I^*)}{(1+\beta S^*+\gamma I^*)^2} \\ &= \frac{1}{\alpha^*}\left(\delta_0 + \delta_3 + \frac{a}{(1+bI^*)^2}\right) \neq 0.\end{aligned}$$

Hence the system (3) shows a Hopf bifurcation near the positive equilibrium E_1 when $\alpha = \alpha^*$. □

In the following theorem, we show the nonexistence of limit cycle under certain condition.

Theorem 8.

If $b(1+\frac{\beta A}{\delta_0}) < \gamma$, then the model (3) does not have any periodic solution in the interior of the positive quadrant of the S-I plane.

Proof:

We define a real-valued function in the interior of positive quadrant of the S-I plane as follows:

$$H(S,I) = \frac{1+\beta S+\gamma I}{SI} > 0.$$

Let us consider

$$h_1(S,I) = A - \delta_0 S - \frac{\alpha SI}{1+\beta S+\gamma I},$$
$$h_2(S,I) = \frac{\alpha SI}{1+\beta S+\gamma I} - \delta_3 I - \frac{aI}{1+bI}.$$

Then we have

$$\begin{aligned} div(Hh_1, Hh_2) &= \frac{\partial}{\partial S}(Hh_1) + \frac{\partial}{\partial I}(Hh_2) \\ &= -\frac{A(1+\gamma I)}{IS^2} - \frac{\delta_0\beta}{I} - \frac{\delta_3\gamma}{S} - \frac{a(\gamma - b(1+\beta S))}{S(1+bI)^2}. \end{aligned}$$

We can see that the above expression is not equal to zero and this will not change sign in the positive quadrant of the S-I plane if the inequality $b(1+\frac{\beta A}{\delta_0}) < \gamma$ holds. Then from Dulac's criterion (Sastry (1999)), we can say that model (3) does not have any periodic solution in the interior of the positive quadrant of the S-I plane. □

Epidemiologically the above theorem implies that if the given inequality holds true then disease will not reoccur.

Since the set Ω defined in Lemma 1 is a positively invariant set, then the following theorem is a direct consequence of the Poincare-Bendixon theorem (Sastry (1999)) showing the existence of a limit cycle about the interior equilibrium E_1.

Theorem 9.

Assume that either (13) and (16) or (15) are satisfied, then the model (3) has at least one limit cycle in the interior of the positive quadrant of the S-I plane.

This theorem depicts that if the positive equilibrium point E_1 is a saddle point or unstable then disease may reoccur in future.

In the following theorem, we show that the endemic equilibrium $E_1(S^*, I^*)$ is globally asymptotically stable.

Theorem 10.

Let the following inequality holds in Ω:

$$\frac{\alpha^2\gamma S^* I^*(1+\gamma I^*)}{(1+\beta S^*+\gamma I^*)^2} < X_1 X_2, \tag{18}$$

where

$$X_1 = \delta_0 + \frac{\alpha I^*(1+\gamma I^*)\delta_0}{(\delta_0+(\beta+\gamma)A)(1+\beta S^*+\gamma I^*)},$$

$$X_2 = \frac{\alpha\gamma S^*\delta_0}{(\delta_0+(\beta+\gamma)A)(1+\beta S^*+\gamma I^*)} - \frac{ab}{1+bI^*}.$$

Then $E_1(S^*, I^*)$ is globally asymptotically stable with respect to all solutions in the interior of the positive quadrant Ω.

Proof:

We consider the following positive definite scalar function about E_1:

$$V = \frac{1}{2}(S-S^*)^2 + k\left(I - I^* - I^* ln\frac{I}{I^*}\right),$$

where k is a positive constant to be chosen suitably.

Now differentiating V with respect to time t along the solutions of model (3), we get

$$\frac{dV}{dt} = (S-S^*)\frac{dS}{dt} + k\frac{(I-I^*)}{I^*}\frac{dI}{dt}.$$

Substituting the values of $\frac{dS}{dt}$ and $\frac{dI}{dt}$ from model (3) into the above equation, we get

$$\frac{dV}{dt} = -a_{11}(S-S^*)^2 + a_{12}(S-S^*)(I-I^*) - a_{22}(I-I^*)^2,$$

where

$$a_{11} = \delta_0 + \frac{\alpha I^*(1+\gamma I^*)}{(1+\beta S+\gamma I)(1+\beta S^*+\gamma I^*)},$$

$$a_{12} = \frac{\alpha\gamma S^* I^*}{(1+\beta S+\gamma I)(1+\beta S^*+\gamma I^*)} - \frac{\alpha S}{(1+\beta S+\gamma I)} + \frac{k\alpha(1+\gamma I^*)}{(1+\beta S+\gamma I)(1+\beta S^*+\gamma I^*)},$$

$$a_{22} = \frac{k\alpha\gamma S^*}{(1+\beta S+\gamma I)(1+\beta S^*+\gamma I^*)} - \frac{kab}{(1+bI)(1+bI^*)}.$$

Sufficient conditions for $\frac{dV}{dt}$ to be negative definite are given as follows:

$$a_{11} > 0 \text{ and } a_{12}^2 < 4a_{11}a_{22}.$$

Here we can see that a_{11} is positive for all values of (S^*, I^*) and another condition for global stability $a_{12}^2 < 4a_{11}a_{22}$ is satisfied if inequality (18) holds true. Hence the theorem follows. □

4. Numerical Simulations

In this section, we present computer simulation results for system (3) by using MatLab 7.10.

We choose the set of parameters given in Table 1.

Table 1. List of parameters.

Parameter	Value	Unit
A	7	person $(\text{day})^{-1}$
δ_0	0.02	$(\text{day})^{-1}$
δ_1	0.05	$(\text{day})^{-1}$
δ_2	0.002	$(\text{day})^{-1}$
a	0.2	$(\text{day})^{-1}$
b	0.02	$(\text{person})^{-1}$
α	0.003	$(\text{person})^{-1}$ $(\text{day})^{-1}$
β	0.002	$(\text{person})^{-1}$
γ	0.5	$(\text{person})^{-1}$

For these values of parameters, conditions (7), (10), and (11) for the existence of $E_1(S^*, I^*)$ are satisfied and $E_1(S^*, I^*)$ is given by

$$S^* = 301.0107, \quad I^* = 3.7996.$$

We further note that inequalities (12) and (13) in Theorem 4 are satisfied for E_1 to be locally asymptotically stable. The trajectories of S and I with initial conditions $S(0) = 245$, $I(0) = 45$, approach to the endemic equilibrium $E_1(301.0107, 3.7996)$ as shown in Figure 1.

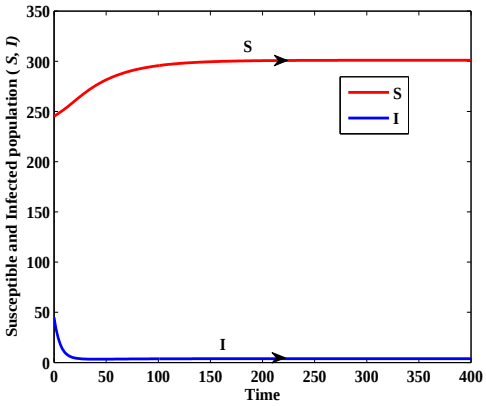

Figure 1: Susceptible (S) and Infected (I) population vs Time.

In Figure 1, the number of the infected population decreases with time due to treatment, and these individuals once recovered have become immunized to the infection and will not get reinfected in future. Furthermore, the susceptible population increases to attain a steady state. This increase may be due to decrease in the number of infected individuals because of treatment.

Further, we choose the set of parameters as given in Table 2.

Table 2. List of parameters.

Parameter	Value	Unit
A	1.97	person $(\text{day})^{-1}$
δ_0	0.2	$(\text{day})^{-1}$
δ_1	0.03	$(\text{day})^{-1}$
δ_2	0.03	$(\text{day})^{-1}$
a	0.02	$(\text{day})^{-1}$
b	0.02	$(\text{person})^{-1}$
α	0.05	$(\text{person})^{-1}$ $(\text{day})^{-1}$
β	0.01	$(\text{person})^{-1}$
γ	0.1	$(\text{person})^{-1}$

For these values of parameters given in Table 2, we see that the endemic equilibrium $E_1(7.0861, 1.9796)$ exists and all conditions of Theorem 4 and Theorem 5 are satisfied. From these simulations and following Figure 2, we conclude that the endemic equilibrium E_1 is globally asymptotically stable. This implies that for the given set of parameters the trajectories of S and I will converge to the same value (steady state) E_1 irrespective of the initial value of S and I. This implies that for the given set of parameters the disease will restrict itself to a given endemic zone, no matter what the magnitude of infection and susceptibility is.

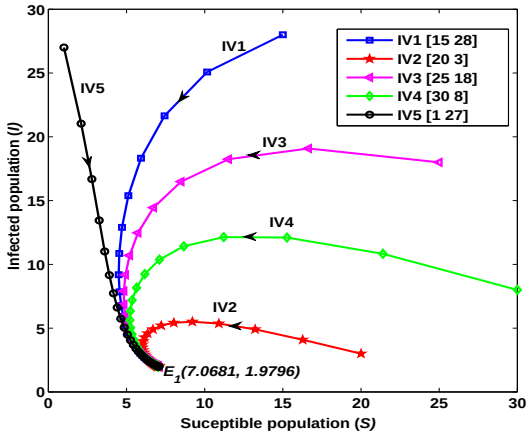

Figure 2: Global stability of endemic equilibrium point.

In Figure 2, we considered five different initial values of the susceptible and infected populations. All trajectories starting from different initial values approach to the endemic equilibrium $E_1(7.0861, 1.9796)$. All the details related to initial values (IV) are shown in the legend.

In Figures 3(a) and 3(b), we plotted the effect of incidence rate α on S and I population (respectively) for the set of parameters given in Table 1. In Figure 3(a) we see that as α increases, the susceptible population S shows sharp decline initially and after a threshold value of α (say $\alpha = 0.006$) S decreases slowly and settles to the its equilibrium point. From Figure 3(b), we note that when the incidence rate is high then more people will be infected and only the remaining

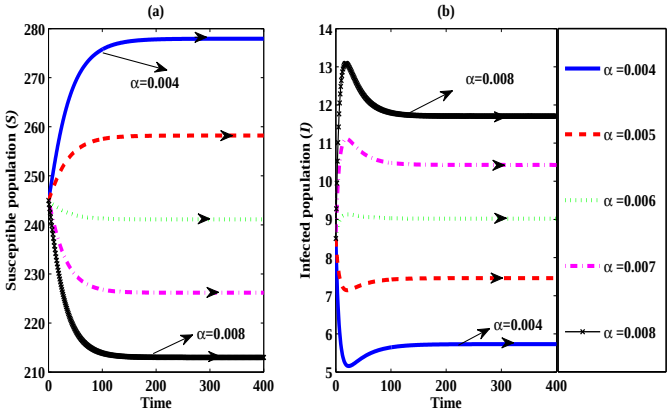

Figure 3: Effect of α on S and on I population respectively.

noninfected people will be susceptible. When the incidence rate is low, then less people are infected and the noninfected, i.e. susceptible population, is larger. We further note that for a larger incidence rate, the number of infected individuals increases initially, then decreases and finally settles down at its steady state. This decrease is possibly due to immunity and the treatments. When the incidence rate is below a threshold value, then the number of infected individuals first decreases, then increases and finally gets stabilized at its steady state. This increase may be due to the fact that the infection is not removed completely but will persist in the endemic zone due to inability of treatment to eradicate the infection. The details of different trajectories and different values of α used in Figures 3(a) and 3(b) are shown in the legend, which is same for both Figures.

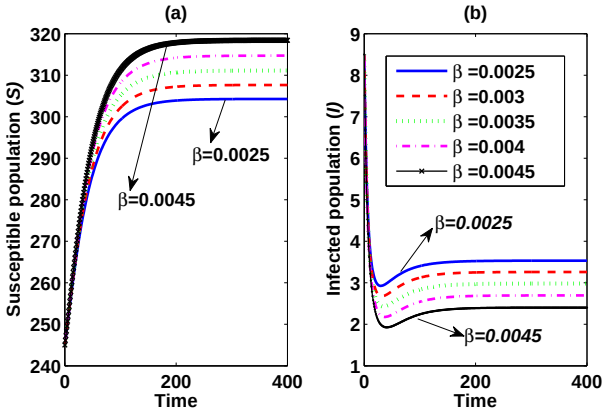

Figure 4: Effect of β on S on I population respectively.

In Figures 4(a) and 4(b), we plotted the effect measure of inhibition β (preventive measure taken by susceptible individuals) on the susceptible and infected populations respectively, with respect to time. From Figures 4 (a) and 4(b), we observe that the number of infected individuals decreases as β increases and consequently the susceptible population increases with increase in β. The trajectories of S and I settle down at their respective equilibrium levels. Figure 4(b) also

shows that initially the number of infected individuals decreases then increases for some time and finally obtains its equilibrium level. The initial decrease in number of infectives may be due to the prevention measures taken by susceptibles and the treatments received. However, these preventive measures and treatments may not be adequate, thus number of infectives increases slightly and gets stabilized at the steady state. This implies overall that when the inhibition is less then more people are infected and less people are susceptible whereas when the inhibition is more then more people are susceptible and less are infected.

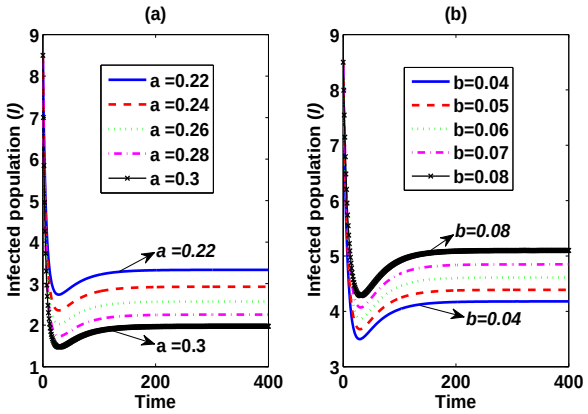

Figure 5: Effect of a and b on I population.

Figures 5(a) and 5(b) show the effect of treatment rate 'a' and limitation to treatment rate 'b' on infected population. Figure 5(a) shows a decrease in infected population as treatment rate a increases and it settles down at its steady state, but the disease is not getting totally eradicated as it will persist at a much lower level. Figure 5(b) shows an increase in infected population as b increases which is due to limited availability of resources in community.

Table 3. List of parameters.

Parameter	Value	Unit
A	7	person $(\text{day})^{-1}$
δ_0	0.002	$(\text{day})^{-1}$
δ_1	0.005	$(\text{day})^{-1}$
δ_2	0.01	$(\text{day})^{-1}$
a	2	$(\text{day})^{-1}$
b	0.02	$(\text{person})^{-1}$
β	0.02	$(\text{person})^{-1}$
γ	0.005	$(\text{person})^{-1}$

Now we choose another set of parameters for model (3) as given in Table 3. In addition to the values of parameters given in Table 3, we chose $\alpha = 0.15$ $(\text{person})^{-1}$ $(\text{day})^{-1}$. Then it is noted that all the conditions of Theorem 4are satisfied. Hence E_1 is locally asymptotically stable. For $\alpha = 0.06$ $(\text{person})^{-1}$ $(\text{day})^{-1}$ (keeping other values of parameters same as in Table 3, condition (16) in Theorem 6 is satisfied. Hence E_1 is unstable. Further, for $\alpha = \alpha^* = 0.08863$ $(\text{person})^{-1}$ $(\text{day})^{-1}$ and other values of parameters are same as in Table 3, all conditions in Theorem 7

are satisfied, which shows the existence of Hopf bifurcation near the interior equilibrium E_1. These three different behavior are shown in Figures 6(a) and 6(b) for susceptible and infected populations respectively.

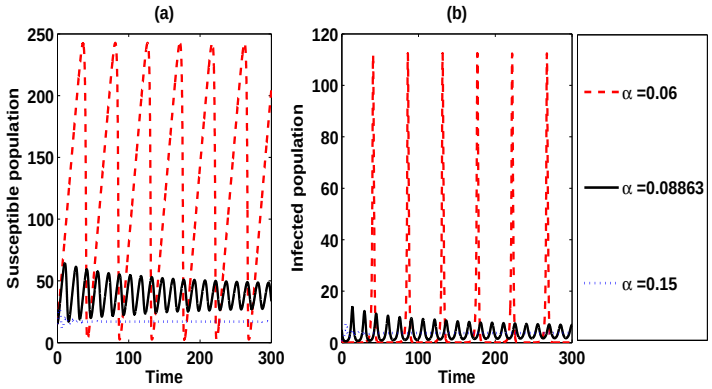

Figure 6: Plot of susceptible and infected population vs time for different values of α.

The phase plane analysis of susceptible and infected population are represented in Figure 7 and Figure 8, respectively. Figure 7 represents a stable limit cycle for $\alpha = 0.08863$ $(\text{person})^{-1}$ $(\text{day})^{-1}$ and other parameters are same as given in Table 3. In Figure 8, trajectories represent unstable endemic equilibrium for $\alpha = 0.06$ $(\text{person})^{-1}$ $(\text{day})^{-1}$ and other parameters are same as given in Table 3.

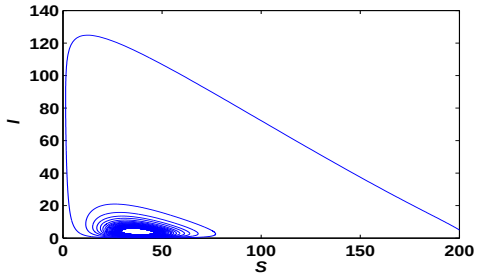

Figure 7: Limit cycle in $S - I$ plane.

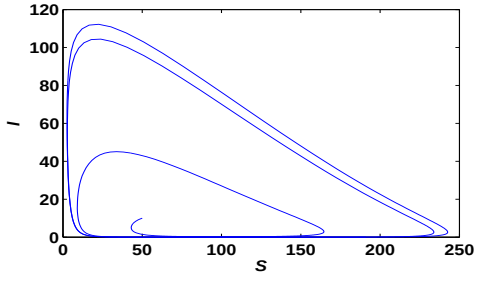

Figure 8: Phase portrait of model (3) in $S - I$ plane.

5. Conclusion

In this paper, we have introduced an SIR model with Beddington-DeAngelis type incidence rate and saturated treatment rate. The local and global dynamics of this model has been studied. The analysis of the proposed model shows that there exists only two non-negative equilibrium points; the disease-free equilibrium $E_0(\frac{A}{\delta_0}, 0)$, i.e. when there is no infection (as $I = 0$), and the endemic equilibrium $E_1(S^*, I^*)$, i.e. when infection is present in the community. The DFE is locally asymptotically stable when the basic reproductive number $R_0 < 1$ and globally asymptotically stable when $R_1 = \frac{\alpha A}{(\delta_0 + A\beta)} \leq 1$. It is also noted that the value of the threshold R_1 can be made less than or equal to one by decreasing the incidence rate (α) and by increasing the preventive measures (β) adopted by susceptibles. We have also shown that the system (3) undergoes transcritical bifurcation at $R_0 = 1$ and there exists an endemic equilibrium when R_0 exceeds one. Biologically this depicts that if the average number of newly infected individuals is more than one then infection will persist. The endemic equilibrium is locally asymptotically stable for $R_0 > 1$ and under conditions stated in Theorem 4. We observed that the system changes its stability behavior around the endemic equilibrium from stable to unstable as bifurcation parameter α changes and system (3) exhibits Hopf bifurcation near endemic equilibrium E_1 for $\alpha = \alpha^*$ (defined in the proof of Theorem -C). We have found that system (3) has periodic solution if inequalities as stated in Theorem -C hold true and there is no periodic solution if $b(1 + \frac{\beta A}{\delta_0}) < \gamma$ holds true. The existence of periodic solution shows that the infection may reoccur in the future.

The proposed model depicts the presence of endemic equilibrium point that is not only globally asymptotically stable but is also independent of the initial values of the susceptible and infected individuals. This indicates the restriction of the disease within endemic zone. This model shows a decrease in infected individuals with both decline in incidence rate α and an enhancement of inhibition rate (preventive measures) i.e., β. It has also been observed that the number of infected individuals decreases as the treatment rate (a) increases. However it increases as the limitation on resource (b) increases. This shows that for effective treatment the resource limitation should be minimized.

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