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Dynamic behaviors of a modified SIR model with nonlinear incidence and recovery rates

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Abstract

A complex SIR epidemic dynamical model using nonlinear incidence rate and nonlinear recovery rate is established to consider the impact of available hospital beds and interventions reduction on the spread of infectious disease. Rigorous mathematical results have been established for the model from the point of view of stability and bifurcation. The model has two equilibrium points when the basic reproduction number \( R_0 \) > 1; a disease-free equilibrium \( E_0 \) and a disease endemic equilibrium \( E_1 \). We use LaSalle’s invariance principle and Lyapunov’s direct method to prove that \( E_0 \) is globally asymptotically stable if the basic reproduction number \( R_0 \) < 1, and \( E_1 \) is globally asymptotically stable if \( R_0 \) > 1, under some conditions on the model parameters. The existence and nonexistence of limit cycles are investigated under certain conditions on model parameters. The model exhibits Hopf bifurcation near the disease endemic equilibrium. We further show the occurring of backward bifurcation for the model when there is limited number of hospital beds. Finally, some numerical results are represented to validate the analytical results.

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1. Introduction

In infectious disease modeling, the function that describes the incidence rate is a main factor that determines the dynamics of the model. Standard bilinear incidence rate \( \beta IS \) have been often used in epidemiological models \([1,2,4,11,12,15,26]\), where the dynamics are simply determined by \( R_0 \); the disease will disappear and all population will become susceptible if \( R_0 < 1 \) and will persist otherwise. Those bilinear incidence rates do not consider in their formation the influence of intervention strategies (preventive measures), such as self-isolation, quarantine and mask-wearing, which play critical roles in control of the disease spread of an infectious diseases. Hence, it is crucial to consider the influence of intervention strategies as well as intervention gradual reduction on the spread of infectious diseases. The coronavirus which is a big problem for the humans society in the world currently, having a lot of infected people with limited resources in many countries such as the availability of the hospital beds, ventilators and many more. Some mathematical model that addressed the corona infection through mathematical models are considered in \([27–31]\).
Several authors considered different types of incidence rate in their work. For example, in 1978, Anderson and May [1] introduced the saturated incidence rate \( \frac{\beta IS}{1 + IS} \) which saturates due to crowding of infective individuals at high levels of infection. Another nonlinear incidence rates is \( \frac{\beta IS}{1 + IS} \) which was independently introduced by Beddington [5] and DeAngelis [7]. Gui-Hua Li and Yong-Xin Zhang [25] modified this incidence rate and used \( \frac{\beta IS}{1 + IS} \) with nonlinear recovery rate.

Gradual reduction in intervention strategies making the response of the incidence rate more slowly compared to the standard bilinear term \( \beta IS \). In contrast, if little intervention strategies are took place, then a faster rate of increase can be seen than \( \beta IS \). As a result of pandemic such as COVID-19, government interventions have huge negative impact on economic, including increasing rates of unemployment and business bankruptcies which led governments to reduce the interventions [3,6,9,10,13,22]. Here in the present investigations we extend the SIR model by considering the nonlinear Monod equation type of incidence rate to study the effect of intervention reduction on the transmission of infectious diseases. The nonlinear Monod equation is a ratio expressed as

\[
\frac{\beta IS}{k + I}
\]

where \( \beta IS \) represents the disease infection force, \( k \) denote a positive constant that represents the intervention levels and determines the shape of the incidence rate as a function of the infected sub-population \( I \). Monod equation type of incidence rate implies that, for low number of infected sub-population the incidence rate is low due to strict intervention and it increases as the number of infected individuals increase till it becomes independent of the number of infected sub-population as witnessed throughout the world in response to COVID-19 pandemic.

Resource availability to the public is estimated as hospital bed-population ratio (HBPR) by the World Health Organization (WHO). Shan and Zhu [14] considered the impact of resource availability and considering the following nonlinear recovery rate

\[
\alpha(h, I) = z_0 + (z_1 - z_0) \frac{b}{b + I},
\]

where \( z_0 \) and \( z_1 \) \((0 < z_0 < z_1)\) denote the respectively the minimum and maximum per capita recovery rates, due to the sufficiency of the health care resource and the number of infected sub-population. The positive constant \( b \) is biologically significant as it represents the impact of the number of hospital beds on the transmission of infectious diseases. Shan and Zhu [14] found that this nonlinear recovery term can lead to complex dynamic behaviors such as backward and forward bifurcations. Some recent research where the authors used different type of incidence rate to investigate the dynamics of infectious diseases [16–21]. For instant the authors considered an age structured model for tuberculosis dynamics using nonlinear incidence rate [16]. The dynamics of tuberculosis infection in adults and children using nonlinear incidence rate together fractional derivative has been studied in [17]. An SEIR model has been studied in [18] where the authors used a general nonlinear incidence rate an presented the dynamics of the model. The authors considered an SIR model using fractional derivative using nonlinear generalized incidence rate in [19]. The dynamics of influenza using half saturated incidence rate has been analyzed in [20]. The use of saturated treatment function in the modeling of vector host disease has been studied in [21].

The present study proposes an SIR model with a Monod type equation with incidence rate and the recovery rate in nonlinear form described by Shan and Zhu [14] and explained above. The organization of this paper is as following: The mathematical model establishment is presented in Section 2. Basic analytical results, including stability of the disease-free and the disease endemic equilibria, are explored in Section 3. Bifurcation analysis is done in Section 4. Numerical results are given in Section 5 to confirm some of the analytical results obtained in Section 3. At the end, we summarizing the fining of our work in Section 6.

2. Model formulation

The total population \( N(t) \) will be divided into three categories: susceptible \( S(t) \), infected \( I(t) \) and recovered \( R(t) \) individuals (SIR), where \( N(t) = S(t) + I(t) + R(t) \) (as shown in Fig. 1). Individuals move from compartment \( S \) to \( I \) with transmission rates \( \frac{\beta IS}{1 + IS} \) (see the plot of the incidence rate in Fig. 2).

Once an individual becomes infected, that individual will either recover at the rate \( \alpha(I, b)I \) (see the plot of the recovery rate in Fig. 3), or dies at the rate \( \gamma I \). We have three sub-populations, therefore a nonlinear dynamical system consisting of three nonlinear differential equations established is as follows:

\[
\begin{align*}
\frac{dS}{dt} &= A - \frac{\beta IS}{1 + IS} - \mu S, \\
\frac{dI}{dt} &= \frac{\beta IS}{1 + IS} - \left(z_0 + \frac{b}{b + I}ight)I - \gamma I - \mu I, \\
\frac{dR}{dt} &= \left(z_0 + \frac{b}{b + I}ight)I - \mu R,
\end{align*}
\]

where \( A \) is a positive constant representing the birth rate, \( \mu \) denotes the natural death rate at each compartment while \( \gamma \) denotes the death due to disease.

3. Stability analysis

In the first two equations of system (1), \( R(t) \) does not appear, therefore it is sufficient to only consider the first two equations and \( R(t) = N(t) - S(t) - I(t) \). Thus, the focus in the discussions will be on the following reduced model

\[
\begin{align*}
\frac{dS}{dt} &= A - \frac{\beta IS}{1 + IS} - \mu S, \\
\frac{dI}{dt} &= \frac{\beta IS}{1 + IS} - \left(z_0 + \frac{b}{b + I}ight)I - \gamma I - \mu I.
\end{align*}
\]

The equilibria is found by solving the following system:

\[
\begin{align*}
A - \frac{\beta IS}{1 + IS} - \mu S &= 0, \\
\frac{\beta IS}{1 + IS} - \left(z_0 + \frac{b}{b + I}ight)I - \gamma I - \mu I &= 0.
\end{align*}
\]

The disease-free equilibrium (DFE) can be determined by solving the above system to be \( E_0 = \left( \frac{A}{\mu}, 0 \right) \). When the system reaches the disease-free equilibrium point \( E_0 \), the disease disappears in the system and all population are susceptible.
3.1. Positive and boundedness of the model

Theorem 1. \( \Omega = \left\{ (S(t), I(t)) \in R^2_+ : S(t) + I(t) \leq \frac{\mu}{\beta}, t \geq 0 \right\} \) is for system (2) the positive invariant region.

Proof. Consider \( N = S + I \), then adding up the equations of system (2) gives
\[
\frac{dN}{dt} = A - \mu N(t) - \alpha S(t)I(t) - \gamma I(t).
\]
It is obvious that
\[
N(t) \leq N(0)e^{-\mu t} + \frac{A}{\mu}(1 - e^{-\mu t}).
\]
Thus, \( \lim_{t \to \infty} \sup N(t) \leq \frac{A}{\mu} \) and \( \dot{N} < 0 \) if \( N > \frac{A}{\mu} \). Hence solutions of (2) are bounded and \( \Omega \) is positively invariant.

3.2. Basic reproduction number \( R_0 \)

The next generation matrix method will be used to calculate \( R_0 \), more details about the method are in [8]. At \( E_0 \) we have \( \alpha(S, I) = \alpha_1 \). The system (2) is written as follows
\[
\frac{dw}{dt} = \Phi(w) - \Psi(w),
\]
where \( \Phi := (F_1, F_2)^T \) and \( \Psi := (V_1, V_2)^T \), or more explicitly
\[
\begin{pmatrix}
\dot{S} \\
\dot{I}
\end{pmatrix} =
\begin{pmatrix}
\frac{\beta SI}{k+I} \\
(\alpha + (\alpha - \alpha_0) \frac{b}{k+I})I
\end{pmatrix} -
\begin{pmatrix}
(\alpha_1 + \gamma + \mu)I \\
-A - \mu S + \frac{\beta SI}{k+I}
\end{pmatrix}.
\]
Now, let \( M' = \left(0, \frac{A}{\mu} \right)^T \) and \( F \) and \( V \) be the following submatrices of the Jacobian of the above system. We have
\[
F = \left( \frac{\partial \Phi}{\partial w} \right)_{M'} = \frac{\beta A}{k\mu},
\]
and
\[
V = \left( \frac{\partial \Psi}{\partial w} \right)_{M'} = \alpha_1 + \gamma + \mu.
\]
The spectral radius of \( FV^{-1} \) is equal to \( R_0 \). Thus
\[
R_0 = \frac{\beta A}{k\mu(\alpha_1 + \gamma + \mu)}.
\]
3.3. Existence of endemic equilibrium $E_3(S^*, I^*)$

To obtain the endemic equilibrium we solve the following algebraic equations

$$A - \frac{\beta I S}{k + I} - \mu S = 0, \quad A I + \left(2 I + \frac{b}{k + I} \right) I - \gamma = 0.$$

Solving the second equation for $S^*$, we get $S^*$ as a function of $I^*$ as follows:

$$S^* = \frac{\left(\frac{\beta}{b + I} \mu + \gamma + z_0\right)}{b(b + I)} \left(\frac{k + I}{k + I} \right).$$

Substituting (4) into the first equation of (2) we get

$$A I^2 + A_2 I + A_3 = 0,$$

where

$$A_1 = \left(\frac{\beta}{b + I} \right) \left(\mu + \gamma + z_0\right), \quad A_2 = \left(\frac{\beta}{b + I} \right) \left(\mu + \gamma + z_1\right), \quad A_3 = \left(\frac{\beta}{b + I} \right) \left(\mu + \gamma + z_1\right).$$

The solution of Eq. (5) is given by

$$I_{1,2} = -A_2 \pm \sqrt{A_2^2 - 4 A_1 A_3} \over 2 A_1,$$

where

$$A_2^2 - 4 A_1 A_3 = \mu^2 \left(\mu + \gamma + z_1\right)^2 - 2b \left(\frac{\beta}{b + I} \right) \left(\mu + \gamma + z_0\right) \left(\mu + \gamma + z_1\right) + \left[\left(\frac{\beta}{b + I} \right) \left(\mu + \gamma + z_0\right) \left(\mu + \gamma + z_1\right)\right]^2.$$

- **Case 1.** $\mathcal{R}_0 < 1$. We have that $A_1 > 0$ and $A_2 > 0$. Clearly Eq. (5) has no positive root if $A_2 \geq 0$, which occurs when $b \geq \frac{\mu(z_1 - z_0)}{(\mu + \gamma + z_0)(\mu + \gamma + z_1)}$. So, model (2) does not have endemic equilibrium whenever $b \geq \frac{\mu(z_1 - z_0)}{(\mu + \gamma + z_0)(\mu + \gamma + z_1)}$ then $A_2 < 0$, therefore, Eq. (5) has no positive root if $A_2^2 - 4A_1A_3 < 0$, one root if $A_2^2 - 4A_1A_3 = 0$, and two positive roots if $A_2^2 - 4A_1A_3 > 0$.

- **Case 2.** $\mathcal{R}_0 = 1$. We have $A_1 > 0, A_3 = 0$ and $A_2^2 - 4A_1A_3 = A_2^2 > 0$. Eq. (5) has no positive root if $A_2 \geq 0$ and a unique positive root if $A_2 < 0$, which occurs when $b < \frac{\mu(z_1 - z_0)}{(\mu + \gamma + z_0)(\mu + \gamma + z_1)}$.

- **Case 3.** $\mathcal{R}_0 > 1$. We have $A_1 > 0, A_3 < 0$ and $A_2^2 - 4A_1A_3 = A_2^2 > 0$. Eq. (5) has one unique positive root. That is, model (2) possess a unique endemic equilibrium point.

3.4. Local Stability

The following results are established to ensure the local stability results of the model considered.

**Theorem 2.** If $\mathcal{R}_0 < 1$, then the model (2) at the DFE case is locally asymptotically stable, otherwise unstable for $\mathcal{R}_0 > 1$.

**Proof.** At $E_0$, the Jacobian matrix $J(E_0)$ of the system (2) is given by

$$J(E_0) = \begin{pmatrix} -\mu & -\frac{\beta}{k + I} \\ 0 & \frac{\beta}{k + I} \left(\frac{k + I}{b + I}\right) \end{pmatrix}.$$  

Denoting the $2 \times 2$ identity matrix by $I$, the characteristic equation associated to $J(E_0)$ can be given as

$$\det(J(E_0) - \lambda I, \mathcal{J}) = \left[-\mu + \frac{\beta}{k + I} \left(\frac{k + I}{b + I}\right)\right] \lambda^2 - \mu \lambda + \frac{\beta}{k + I} \left(\frac{k + I}{b + I}\right) = 0.$$

Thus

$$\lambda_1 = -\mu, \quad \lambda_2 = \frac{\beta}{k + I} \left(\frac{k + I}{b + I}\right).$$

The matrix $J(E_0)$ has negative eigenvalues when $\mathcal{R}_0 = \frac{\beta}{k + I} \left(\frac{k + I}{b + I}\right) < 1$, therefore, according to the Routh-Hurwitz stability criterion $E_0$ is locally asymptotically stable. If $\mathcal{R}_0 > 1$, the eigenvalue $\lambda_2 > 0$, so $E_0$ is unstable. □

**Theorem 3.** The disease endemic equilibrium $E_3(S^*, I^*)$ is locally asymptotically stable if inequalities (6) and (7) are satisfied:

$$\frac{k \beta S^*}{(k + I)^2} + \frac{z_1 b^2}{(b + I)^2} < N_1,$$

and

$$\frac{z_1 \beta b I^*}{(k + I)^2} + \frac{k \mu b S^*}{(k + I)^2} + \frac{\mu z_2 b^2}{(b + I)^2} < N_2,$$

where

$$N_1 = \frac{\beta}{k + I} + \frac{z_1 b^2}{(b + I)^2} + 2 \mu + z_0 + \gamma,$$

and

$$N_2 = \frac{\beta}{k + I} + \frac{z_1 b^2}{(b + I)^2} + \frac{\mu z_2 b^2}{(b + I)^2} + \mu (z_0 + \gamma + \mu).$$

**Proof.** The Jacobian matrix $J(E_1)$ of the system (2) is given by

$$J(E_1) = \begin{pmatrix} -\mu & -\frac{\beta}{k + I} & \frac{-\mu x_0}{\beta} + \frac{x_0 \beta}{k + I} \\ \frac{\beta}{k + I} & -\frac{\mu x_0}{\beta} & \frac{-\mu x_0}{\beta} + \frac{x_0 \beta}{k + I} \\ \frac{\beta}{k + I} & \frac{x_0 \beta}{\beta} & \mu (x_0 + \gamma + \mu) \end{pmatrix}.$$  

The characteristic equation associated to $J(E_1)$ can be given as

$$\det(J(E_1) - \lambda I, \mathcal{J}) = \lambda^3 + a_1 \lambda^2 + a_2 \lambda + 0,$$

where

$$a_1 = \frac{\beta}{k + I} + \frac{\mu x_0}{\beta}, \quad a_2 = 2 \mu + z_0 + \gamma + \frac{\mu}{\beta}.$$  

By the Routh-Hurwitz criterion, the Jacobian matrix above have eigenvalues with negative real parts if $a_1 > 0$ and $a_2 > 0$, therefore, $E_3(S^*, I^*)$ is locally asymptotically stable if inequalities (6) and (7) are satisfied.
Theorem 4. If
\[ \frac{x_1b\beta^2 T}{(k + T)(b + T)^2} + \frac{k\mu S^*}{(k + T)^2} + \frac{\mu x_1b^2}{(b + T)^2} > N_2, \]
holds true, then \( E_1(S^*, T) \) is a saddle point.

Theorem 5. If
\[ \frac{k\beta S^*}{(k + T)^2} + \frac{x_1b^2}{(b + T)^2} > N_1, \]
and inequality (7) are satisfied, then \( E_1(S^*, T) \) is unstable.

3.5. Global stability

Theorem 6. \( E_0 \) is globally asymptotically stable when
\[ k > \frac{\beta_4}{\mu_4 + \mu + \alpha_2}. \]

Proof. When \( k > \frac{\beta_4}{\mu_4 + \mu + \alpha_2}, \) it obvious that \( \mathcal{R}_0 < 1. \) Let us construct a Lyapunov function as follows:
\[ V(S, I) = \left( \frac{\mu}{A} S - \ln \left( \frac{\mu S}{A} \right) \right) + \frac{\mu}{A} I. \]

Differentiating \( V \) along system (2) and after simplification, we have
\[ \dot{V} = 2\mu - \frac{\mu^2}{A} S - A - \frac{\beta I}{k + T} + \frac{\mu}{A} (\gamma + \mu + \alpha(b, I)) I. \]

Since \( \alpha(b, I) > 0 \) for all \( I > 0, \) we obtain
\[ \dot{V} \leq 2\mu - \frac{\mu^2}{A} S - A + \frac{\beta}{k + T} (\gamma + \mu + \alpha_0) I - \frac{\mu}{k + T} (\gamma + \mu + \alpha_0) I. \]

It is clear that \( V(\dot{S}, I) < 0 \) if \( \mathcal{R}_0 \leq 1 \) and \( V(\dot{S}, I) = 0 \) if and only if \( (S, I) = E_0 \).

Theorem 7. \( E_1(S^*, I) \) is globally asymptotically stable.

Proof. Let \( h(S)g(I) = \frac{S}{k + T} \) and \( V(S, I) \) is a continuous function for all \( S, I > \epsilon. \)

\[ V(S, I) = S - \int_{S_0}^{S} \frac{h(S')}{h(S)} dS' + I - \int_{I_0}^{I} g(I') \frac{d}{g(I')} dI', \]

where \( \epsilon \) is an arbitrary constant, such that \( 0 < \epsilon \ll 1. \) The endemic equilibrium \( E_1(S^*, I) \) is the global minimum of the function \( V(S, I). \) Indeed,
\[ \frac{\partial V}{\partial S} = 1 - \frac{S'}{S}, \quad \frac{\partial V}{\partial I} = 1 - \frac{(k + T)I'}{(k + T)I}, \]

and hence the point \( E_1(S^*, I) \) is a stationary of the function \( V(S, I). \) The partial derivatives \( \frac{\partial V}{\partial S} \) and \( \frac{\partial V}{\partial I} \) are monotonically increasing because \( S \) and the function \( g(I) \) are monotonically increasing with respect to \( S \) and \( I. \)

Furthermore, since
\[ \frac{\partial^2 V}{\partial S^2} = \frac{S'}{S} > 0 \quad \text{and} \quad \frac{\partial^2 V}{\partial I^2} = \frac{k\beta S'I}{(k + T)(k + I)} > 0, \]
whereas
\[ \frac{\partial^2 V}{\partial S\partial I} = 0, \]
the point \( E_1(S^*, I) \) is the minimum \( (V(S, I) \geq V(S^*, I)). \) Thus \( V(S, I) \) is indeed a Lyapunov function.

For the equilibrium state \( E_1(S^*, I), \) the Lyapunov function (11) satisfies
\[ \frac{dV}{dt} = \frac{dS}{dt} + \frac{S'}{S} \frac{dI}{dt} + \frac{dI}{dt} = \frac{A - \frac{\beta S^*}{b + T} - \mu S^* - \frac{\beta}{b + T} A + \frac{\beta}{b + T} I^* + \frac{\mu}{b + T} I^* - (\alpha(b, I) + \gamma + \mu) I}{b + T} \]
\[ - \frac{\beta}{b + T} + (\alpha(b, I) + \gamma + \mu) I \frac{(b + T)I}{b + T}. \]

Using the equalities
\[ A = \frac{\beta S^*}{k + T} + \mu S^* \quad \text{and} \quad (\alpha(b, I) + \gamma + \mu) I = \frac{\beta S^* I}{k + T}, \]
we obtain
\[ \frac{dV}{dt} = \frac{\beta S^*}{b + T} + \mu S - \frac{\beta}{b + T} A + \frac{\beta}{b + T} I^* + (\alpha(b, I) + \gamma + \mu) \frac{(b + T)I}{b + T} \]
\[ - (\alpha(b, I) + \gamma + \mu) I - \frac{\beta}{b + T} A + (\alpha(b, I) + \gamma + \mu) \frac{(b + T)I}{b + T} \]
\[ = \mu S^* (\frac{b}{k + T} - 1) + \frac{\beta}{b + T} \left( \frac{(b + T)I}{b + T} - 1 \right) \]
\[ + \frac{\beta}{b + T} \left( \frac{(b + T)I}{b + T} - 1 \right) \frac{\beta}{b + T} \left( \frac{(b + T)I}{b + T} - 1 \right) \]
\[ - (\alpha(b, I) + \gamma + \mu) I \frac{(b + T)I}{b + T}. \]

Clearly
\[ \left( 1 - \frac{S}{S^*} \right) \left( 1 - \frac{S^*}{S} \right) \leq 0. \]

Let \( A(I) = (\alpha(b, I) + \gamma + \mu) I, \) we also have
\[ \frac{(k + T)I - A(I)}{(k + T)I} \left( 1 - \frac{(k + T)I}{k + T} \right) \]
\[ = \frac{g(I)}{g(I')} \left( \frac{A(I)}{A(I')} \right) \left( 1 - \frac{g(I)}{g(I')}, \right) \leq 0, \]
if
\[ \int_{I_0}^{I} g(I') \frac{dI'}{g(I')} < 0 \]
which hold because \( g(I) \) is a concave function (that is, \( \frac{d^2 g(I)}{dI^2} < 0 \) (see Fig. 4).

4. Bifurcation analysis

To establish bifurcation analysis of the model (2), we give the following result.
We can rewrite system (13), under above transformation as
\[
\frac{d^2}{dt^2} = \left( -\frac{bA}{\mu k^2} - \frac{\nu_1}{k^2} + \frac{\nu_2}{k^2} \right) V^2 + \frac{bA}{k} V + \mathcal{E} \left( (U, V) \right),
\]
\[
\frac{d}{dt} = -\mu U - \frac{b^2 V}{k} - \frac{b\nu_1^2}{k^2} \left( \frac{bA}{\mu k^2} + \frac{\nu_1}{k^2} + \frac{\nu_2}{k^2} \right) V + \mathcal{E} \left( (U, V) \right).
\]

This system in a diagonal form, so we can applied central manifold theorem, for which, \( U = H(V) \), \( H(V) = \alpha_0 V^2 + \mathcal{E}(V^3) \). For system (15), the center manifold \( W^0(0) \) can be approximated to the second order, by
\[
W^0 = \left\{ (U, V) : U = -\frac{A\beta(-b\beta \mu k + \mu k^2(z_0 - z_1) + A\beta b) V^2}{bk^2\mu^2} \right\}
\]
and the flow equation approximated by
\[
V' = \left( -\frac{bA}{\mu k^2} - \frac{\nu_1}{k^2} + \frac{\nu_2}{k^2} \right) V^2 + \mathcal{E}(V^3).
\]
Thus, the dynamic behaviors of the solution near \( \xi_0 = (0, 0) \) is given by quadratic term, when this term is not equal to zero, ie. \( b \neq \frac{\nu_1^2(z_0 - z_1)}{b\nu_2} \), hence, the system undergoes backward bifurcation if
\[
\frac{z_1 - z_0}{b} > \frac{bA}{\mu k^2} + \frac{A\beta^2}{k^2\mu^2},
\]
or
\[
b < \frac{k^2\mu(z_1 - z_0)}{\beta A(\beta + \mu)},
\]
and the system undergoes forward bifurcation if
\[
b > \frac{k^2\mu(z_1 - z_0)}{\beta A(\beta + \mu)}.
\]

**Theorem 9.** Assume that
\[
\frac{k\beta S^r}{(k + \Gamma)^2} + \frac{z_1b^2}{(b + \Gamma)^2} = N_1,
\]
and (7) are satisfied, then the system (2) shows Hopf bifurcation near \( E_1(S^r, \Gamma) \).

**Proof.** Eq. (16) leads to \( a_1 = 0 \) in Eq. (8), which ensures the possibility the pure imaginary roots. The results of Theorem 8 and 10 show that the parameter \( \beta \) passes through its critical value \( \beta = \beta_* \). \( E_1(S^r, \Gamma) \) changes its behavior from stable to unstable, where
\[
\beta_* = \frac{(k + \Gamma)^2}{k S^r - \Gamma (k + \Gamma)} \left( 2\mu + z_0 + \gamma + \frac{b^2(z_0 - z_1)}{(b + \Gamma)^2} \right),
\]
and
\[
k S^r \neq \Gamma (k + \Gamma).
\]
We have
\[
\frac{d}{dt} \left[ tr(J(E_1)) \right]_{\beta = \beta_*} = \frac{k S^r - \Gamma (k + \Gamma)}{(k + \Gamma)^2} = 0.
\]
Hence a Hopf bifurcation is exhibited for system (2) near \( E_1 \) when \( \beta = \beta_* \).
Theorem 10. In the interior of the positive quadrant of the plane $S - I$, system (2) have no periodic solution if $k > \frac{2a_0I}{a_1}$.

Proof. Let $G(S, I)$ is a real-valued function:

$$G(S, I) = \frac{k + I}{SI} > 0.$$  

Let us consider

$$g_1(S, I) = A - \frac{\beta S}{I + \gamma} - \mu S,$$

$$g_2(S, I) = \frac{\beta S}{I + \gamma} \left( x_0 + (x_1 - x_0) \frac{1}{I + \gamma} \right) I - (\gamma + \mu) I.$$  

Then we have

$$\frac{\partial}{\partial S} (Gg_1) + \frac{\partial}{\partial I} (Gg_2) = \frac{A(K + I)}{IS^2} - \frac{x_1b^2 + [(k + 2I)x_0 - kx_1]b + x_0f}{S(b + I)^2} - \frac{\gamma + \mu}{S} < 0,$$

if the inequality $k > \frac{2a_0I}{a_1}$ holds. Applying Dulac’s criterion, in the interior of the positive quadrant of the plane $S - I$, system (2) have no periodic solution.

Theorem 11. In the interior of the positive quadrant of the plane $S - I$, system (2) has at least one limit cycle if either (7) and (10) or (9) are satisfied. Infectious disease may reoccur in future if the positive equilibrium $E_1(S', I')$ is a saddle point or unstable.

5. Numerical results

In this section, we numerically show that $E_1(S', I')$ is locally and globally asymptotically stable using MATLAB solver ode45 which uses the Runge-Kutta methods to solve initial value problem. For parameters in Table 1, $R_0 = 4.2169$, the endemic equilibrium $E_1$ exists at $E_1(S', I') = (170.7414, 2.2070)$. The inequalities given in (6) and (7) in the result of Theorem 3 are held and, therefore, $E_1$ is locally asymptotically stable. Fig. 5 shows the solutions of $S$ and $I$ with the initial values of the variables $S(0) = 100$ and $I(0) = 50$ approach to $E_1(S', I') = (170.7414, 2.2070)$.

Further, in Fig. 6 we use parameters in Table 2 to show that the endemic equilibrium $E_1(209.9089, 2.9505)$ is globally asymptotically stable which means that the solutions of $S$ and $I$ converge to the same $E_1$ value regardless the initial values of $S$ and $I$.

| Parameter | Value | Dimension |
|-----------|-------|-----------|
| $A$       | 1.75  | Individual/Time |
| $\beta$   | 0.01  | (Individual $\times$ Time)$^{-1}$ |
| $k$       | 2     | Individual |
| $\mu$     | 0.005 | Time$^{-1}$ |
| $x_0$     | 0.2   | Time$^{-1}$ |
| $x_1$     | 0.21  | Time$^{-1}$ |
| $b$       | 0.2   | Individual |
| $\gamma$  | 0.2   | Time$^{-1}$ |

Fig. 5 Susceptible ($S$) and Infected ($I$) populations as functions of time.

Fig. 6 Stability of endemic equilibrium $E_1$ for different initial values.
We further simulate the influence of intervention levels on the spread of infectious disease using parameter in Table 3 and various values of \( k \). Fig. 7 shows different intervention levels, i.e., low, mild, moderate, and strict. The results as depicted in Fig. 7 is showing that moderate and strict interventions significantly reduce the number of infected individuals \( I(\theta) \).

6. Conclusion

We considered a new extended SIR model with Monod type of equation with nonlinear incidence and recovery rate. We examined with care the detailed mathematical results that possible for the considered model. Dynamics of this model has been investigated locally and globally. We investigated the stability of the model at disease-free equilibrium (DFE) and found it locally and globally asymptotically stable when \( R_0 < 1 \) and unstable when \( R_0 > 1 \). We found for \( R_0 > 1 \) that the disease endemic equilibrium \( E_1 \) exists and it is locally asymptotically stable under certain conditions given by the inequalities (6) and (7), and it is a saddle point if inequality (9) holds true, and unstable if inequalities (7) and (10) hold true. Using Lyapunov direct method we showed that \( E_1 \) is globally asymptotically stable. We further showed that when for limited hospital beds \( b < \frac{A_1}{k^2(a_0 + a_1)} \), the system undergoes backward bifurcation, and when \( b > \frac{k^2(a_0 + a_1)}{A_1} \) the system undergoes forward bifurcation. The system exhibits Hopf bifurcation near the disease endemic equilibrium \( E_1 \) when \( \beta = \beta^* \). The system does not have any periodic solution if \( k > \frac{A_1}{2a_0} \). If either the inequalities (7) and (10) or (9) hold true, at least one limit cycle at least exists for the model. The model shows a significant decrease for the infected compartment by increasing the value of the preventive measure \( k \).

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Declaration of Competing Interest

I declare no conflict of interest.

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Table 2 Parameters used to show global stability of \( E_1 \).

| Parameter | Value | Dimension |
|-----------|-------|-----------|
| \( A \)   | 2.3   | Individual/Time |
| \( \beta \) | 0.01  | (Individual × Time) \(^{-1} \) |
| \( k \)   | 2     | Individual    |
| \( \mu \) | 0.005 | Time \(^{-1} \) |
| \( x_0 \) | 0.2   | Time \(^{-1} \) |
| \( x_1 \) | 0.5   | Time \(^{-1} \) |
| \( b \)   | 0.2   | Individual    |
| \( \gamma \) | 0.2   | Time \(^{-1} \) |

Table 3 Parameters used to show the effect of \( k \) on \( I \).

| Parameter | Value | Dimension |
|-----------|-------|-----------|
| \( A \)   | 1.7   | Individual/Time |
| \( \beta \) | 0.01  | (Individual × Time) \(^{-1} \) |
| \( \mu \) | 0.005 | Time \(^{-1} \) |
| \( x_0 \) | 0.02  | Time \(^{-1} \) |
| \( x_1 \) | 0.06  | Time \(^{-1} \) |
| \( b \)   | 0.2   | Individual    |
| \( \gamma \) | 0.2   | Time \(^{-1} \) |

Fig. 7 Effect of \( k \) on the infected population \( I \).
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