Qualitative Analysis of Both Hyperbolic and Non-hyperbolic Equilibria of a SIRS Model with Logistic Growth Rate of Susceptibles and Inhibitory Effect in the Infection

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Abstract: This paper describes a SIRS model with the logistic growth rate of susceptible class. The effect of an inhibitory factor in the infection is also taken into consideration. We have analysed local as well as global stabilities of the equilibrium points (both hyperbolic and non-hyperbolic) of the system and investigated the Transcritical bifurcation at the disease free equilibrium point with respect to the inhibitory factor. The occurrence of Hopf bifurcation of the system is examined and it was observed that this Hopf bifurcation is either supercritical or subcritical depending on parameters. Some numerical simulations are carried out for the validity of theoretical results.

Key words: inhibitory factors, logistic growth, losses immunity, global stability, hopf bifurcation, transcritical bifurcation, centre manifold theory

I. INTRODUCTION

The dynamics of infectious diseases is an important research branch in mathematical epidemiology. Mathematical modelling in epidemiology provides understanding of the underlying mechanisms that influence the spread of infectious diseases and suggests its control strategies. One of the early triumphs of mathematical epidemiology was the formulation of a simple model by Kermack and McKendrick (1927) [1]. More developments and progresses have been particularly made during the past three decades. Massive mathematical models have been formulated to study the dynamical behaviour of various infectious diseases which shows rich nonlinear phenomena [2-6].

Researchers formulate compartmental models based on assumptions on the rates of flow between different classes of

members of the population [3,7,8]. To formulate the compartmental model two important factors play the crucial role: one is the growth rate of susceptible class and the other is the rate of infection [9-19]. The authors usually consider the constant growth rate [17], exponential growth rate and logistic growth rate [9-12, 26]. The logistic growth rate is considered in those models where food supply, space capacity or carrying capacities of the system are limited. On the other hand, the authors consider a different type of the incidence rate, i.e. the infection rate of susceptible individuals through their contacts with infected individuals. The first one is the bilinear incidence rate βSI [9, 10], where S and I are, respectively, the number of susceptible and infected individuals in the population and β is the transmission rate of infection. The other is the saturated incidence rate of the form $\frac{\beta SI}{1+\alpha S}$ or $\frac{\beta SI}{1+\alpha I}$ [12,15,17]. In the incidence rate $\frac{\beta SI}{1+\alpha S}$, α is defined as inhibition coefficient

and this incidence rate increases as the susceptible population increases and ultimately it tends to $\beta I/\alpha$ as $S \to \infty$ if I is finite. This type of infection is sometimes named as 'incidence rate with psychological effect', because the effect of α stems from epidemic control (taking appropriate preventive measures and awareness) and the rate of infection decreases as the inhibitory coefficient α increases.

To study disease dynamics of the SIR epidemic model, Enatsu et al in [9] and Wang et al in [10] considered the growth rate of the susceptible class as the logistic type and the rate of infection as bilinear type mass action. Wang et al in [11] studied the SIR model with the logistic type growth rate of susceptible class with the saturated type treatment rate. Kar and Mandal in [12] considered the SIR epidemic model with the logistic type growth rate of susceptible populations with the saturated type $\frac{\beta SI}{1+\alpha S}$ infection rate.

In this paper, we have considered a SIRS model with the logistic type growth rate of susceptible population and the rate of infection is affected by the inhibitory effect. We have also considered in our model that some recovered individuals lose immunity, [25], and so they become susceptible. SIRS models represent a class of airborne diseases, for example seasonal influenza, but in this paper we focus on a generic SIRS model. This model is the extension of the model considered in [12]. The main objective of this paper is to discuss the stability or instability of the both hyperbolic and non-hyperbolic equilibrium points and exhibition of Transcritical and Hopf bifurcation. The stability analysis of non-hyperbolic equilibrium points will be investigated here by using the Centre Manifold Theory and we will also analyse the role of awareness or inhibitory factors to control the infection of infectious diseases. The paper is organized as follows. In section 2 we have formulated the model, while in section 3 we discuss the existence of equilibria and we have obtained the basic reproduction number R_0 . Section 4 is devoted for local and global stability of equilibrium points, and in section 5 we have shown that the system experiences a Transcritical and Hopf bifurcation at the equilibrium points. Finally, a theoretical finding is justified using the numerical simulation in section 6 and boundedness and permanence of the system are shown in the appendix.

II. THE BASIC MATHEMATICAL MODEL

Here we assume that the susceptible class follows the logistic growth rate and the incidence rate is of saturated type that reflects the "psychological effect" or "inhibition effect" [20]. In the proposed model we also consider the loss of immunity of recovered class. Let S(t), I(t) and R(t) be the number of susceptible, infected and recovered individuals at time t, respectively. Incorporating all the assumptions described above, the governing differential equations of the model can be written as

$$\frac{dS}{dt} = rS\left(1 - \frac{S}{k}\right) - \frac{\beta SI}{1 + \alpha S} - dS + \mu R,$$

$$\frac{dI}{dt} = \frac{\beta SI}{1 + \alpha S} - (d + \gamma) I,$$

$$\frac{dR}{dt} = \gamma I - (d + \mu) R$$
 (1)

with nonnegative initial conditions. Parameters used in system (1) are nonnegative and listed in Table 1.

Since the exact solution of the nonlinear autonomous system (1) is impossible to find, we are analysing the qualitative behaviour of the solutions in the neighbourhood of the equilibrium points.

III. EXISTENCE OF EQUILIBRIA AND THE BASIC REPRODUCTION NUMBER

The system (1) has always trivial equilibrium point $E_0(0,0,0)$. The axial equilibrium $E_1(S_1,0,0)$ exists only when r > d. The system has only one endemic equilibrium point $E_2(S_2, I_2, R_2)$ if the thresholds: $\Delta_1 > 1$ and $\Delta_2 > 1$, where

$$S_{1} = k \left(1 - \frac{d}{r} \right),$$

$$S_{2} = \frac{1}{\alpha \left(\Delta_{1} - 1 \right)},$$

$$I_{2} = \frac{\left(d + \mu \right) \left(d + \frac{rS_{2}}{k} \right) S_{2} \left(\Delta_{2} - 1 \right)}{d \left(d + \mu + \gamma \right)},$$

$$R_{2} = \frac{\gamma I_{2}}{d + \mu},$$

$$\Delta_{1} = \frac{\beta}{\alpha \left(d + \gamma \right)} \text{ and}$$

$$\Delta_{2} = \frac{k \alpha r \left(\Delta_{1} - 1 \right)}{k \alpha d \left(\Delta_{1} - 1 \right) + r}.$$

So the disease free equilibrium point (DFE) E_1 will exist if the intrinsic growth rate is greater than the natural death rate. Again, from the expression of Δ_1 it is clear that Δ_1 will be large if β is high or α is low. Thus the rate of infection (β) and inhibitory parameter (α) play an important role for the existence of S_2 and I_2 . From the expression of I_2 it is clear that with the increase of Δ_2 (when β increases or α decreases) I_2 will increase, that is, the number of the infected will increase. Thus, to control the disease we have to increase inhibitory coefficient α .

Since the considered model has a DFE at which the population remains in the absence of disease, the model has a threshold parameter known as the basic reproduction number R_0 which plays an important role to control the disease.

Tab. 1. Model parameters and their descriptions

Parameters	Interpretations			
r	Birth rate (intrinsic growth rate) of the susceptible class			
k	Carrying capacity of the system			
β	Transmission rate of infection			
α	Inhibitory coefficient			
d	Natural death rate of the population			
μ	Rate at which the recovered class loses immunity and becomes susceptible			
γ	Rate at which the infected individuals recovered			

Lemma 1. The basic reproduction number for the model (1) and is

$$R_0 = \frac{\beta S_1}{\left(1 + \alpha S_1\right) \left(d + \gamma\right)}$$

Proof. Here is only one infected compartment, that is variable I and DFE is $E_1(S_1, 0, 0)$. The basic reproduction number R_0 is defined as the spectral radius of the next generation matrix FV^{-1} with small domain where

$$F = \left(\frac{\beta S_1}{1 + \alpha S_1}\right)_{1 \times 1}$$

$$V = (d + \gamma)_{1 \times 1}$$

[21]. Thus, R_0 for the model is $\frac{\beta S_1}{(1+\alpha S_1)(d+\gamma)}$.

IV. STABILITY OF EQUILIBRIA

In this section we investigate the local stability and global stability of the equilibrium points. Here the variational matrix corresponding to (1) is

$$J(S, I, R) = \begin{pmatrix} r - \frac{2rS}{k} - d - \frac{\beta I}{(1 + \alpha S)^2} & -\frac{\beta S}{1 + \alpha S} \\ \frac{\beta I}{(1 + \alpha S)^2} & \frac{\beta S}{1 + \alpha S} - (d + \gamma) \\ 0 & \gamma \end{pmatrix}$$

IV. 1. Local Stability of Equilibria

Now we establish the following theorem to show that E_0 , E_1 and E_2 are locally asymptotically stable equilibrium points under some conditions on parameters.

Theorem 1. The equilibrium point E_0 is stable if $r \le d$ and is unstable if r > d.

Proof. The eigenvalues of the variational matrix at the point $E_0(0,0,0)$ are -(d-r), $-(d+\gamma)$, $-(d+\mu)$. So E_0 is asymptotically stable for r < d and is unstable for r > d. When r = d the eigenvalues of the variational matrix are $0, -(d+\gamma), -(d+\mu)$. So in this case E_0 is a non-hyperbolic critical point. So we can apply the Centre Manifold Theory to determine its stability. The system (1) can be written as

$$\frac{dX}{dt} = AX + F\left(S, I, R\right),\tag{2}$$

where

$$X = \left(\begin{array}{c} S\\I\\R\end{array}\right),$$

$$\begin{array}{ccc} -\frac{\beta S}{1+\alpha S} & \mu \\ \frac{\beta S}{1+\alpha S} - (d+\gamma) & 0 \\ \gamma & -(d+\mu) \end{array} \right) \cdot \\ A = \left(\begin{array}{ccc} 0 & 0 & \mu \\ 0 & -(d+\gamma) & 0 \\ 0 & \gamma & -(d+\mu) \end{array} \right),$$

$$F(S,I,R) = \begin{pmatrix} -\frac{d}{k}S^2 - \beta SI \\ \beta SI \\ 0 \end{pmatrix}.$$

(Here we expand $(1 + \alpha S)^{-1}$ and we neglect the terms of order greater or equal to 3).

Clearly the matrix A is diagonalisable. Thus we can find a matrix

$$P = \left(\begin{array}{ccc} 1 & -\frac{\mu}{d+\gamma} & 1 \\ 0 & \frac{\mu-\gamma}{\gamma} & 0 \\ 0 & 1 & -\frac{d+\mu}{\mu} \end{array} \right)$$

so that

$$P^{-1}AP = D = \text{diag}(0, -(d + \gamma), -(d + \mu)).$$

By using the transformation X = PY, where

$$Y = \left(\begin{array}{c} S'\\ I'\\ R' \end{array}\right),$$

the system (2) can be put in the form (omitting the 'dash' sign)

$$\frac{dS}{dt} = 0 + g_{11} (S, I, R),
\frac{dI}{dt} = -(d + \gamma) I + g_{22} (S, I, R),
\frac{dR}{dt} = -(d + \mu) R + g_{33} (S, I, R), \text{ where}$$
(3)

$$g_{11} = -\frac{d}{k} \left(S - \frac{\mu}{d+\gamma} I + R \right)^2$$
$$+\beta \left\{ \frac{\mu\gamma}{(d+\mu)(d+\gamma)} - 1 \right\}$$
$$\times \left(S - \frac{\mu}{d+\gamma} I + R \right) \left(\frac{\mu-\gamma}{\gamma} \right) I$$
$$\equiv A_{11}S^2 + B_{11}I^2 + C_{11}R^2$$
$$+D_{11}SI + E_{11}SR + F_{11}IR,$$

$$g_{22} = \frac{\gamma\beta}{\mu - \gamma} \left(S - \frac{\mu}{d + \gamma} I + R \right) \left(\frac{\mu - \gamma}{\gamma} \right) I \equiv \\ \equiv B_{22}I^2 + D_{22}SI + F_{22}IR,$$

$$g_{33} = \frac{\mu\gamma\beta}{(\mu-\gamma)(d+\mu)} \left(S - \frac{\mu}{d+\gamma}I + R\right) \left(\frac{\mu-\gamma}{\gamma}\right)I \equiv \\ \equiv B_{33}I^2 + D_{33}SI + F_{33}IR.$$

(Where $A_{11} = -d/k$ and other components will be similarly determined).

By the Centre Manifold Theory there exists a centre manifold of the system (3) which can be expressed by $W^c(0) = \{(S, I, R) | I = h_1(S), R = h_2(S) \text{ for } S < \delta\}$, where $\delta (> 0)$ is some number, $h_1(0) = h_2(0) = 0$, $Dh_1(0) = Dh_2(0) = 0$.

To compute the centre manifold $W^c(0)$, we assume that $I = h_1(S) = h_{11}S^2 + h_{12}S^3 + \dots$ and $R = h_2(S) = h_{21}S^2 + h_{22}S^3 + \dots$

So from the Local Centre Manifold Theorem [23], we have the flow on the centre manifold $W^c(0)$ defined by the differential equation $\frac{dS}{dt} = A_{11}S^2$.

Since $A_{11} = -\frac{d}{k}$ is negative quantity, hence for r = d the system is locally asymptotically stable. Hence the theorem is proved.

Note: Other components B_{11} , C_{11} , D_{11} , E_{11} , F_{11} , B_{22} , D_{22} , F_{22} , B_{33} , D_{33} and F_{33} are not derived here as they are not in use.

Theorem 2. If r > d the disease free equilibrium point $E_1(S_1, 0, 0)$ is locally asymptotically stable for $R_0 < 1$ and is unstable for $R_0 > 1$.

Proof. The equilibrium point E_1 exists only when r > d and eigenvalues of the Jacobian matrix of the system (1) at E_1 are $-(r - d), -(d + \mu)$ and $(d + \gamma)(R_0 - 1)$. Thus E_1 is asymptotically stable for $R_0 < 1$ and is unstable for $R_0 > 1$. Hence the theorem is proved.

Theorem 3. If r > d and $R_0 = 1$ then $E_1(S_1, 0, 0)$ is an unstable equilibrium point.

Proof. For $R_0 = 1$, the eigenvalues of the variational matrix of (1) at E_1 are $0, -(r - d), -(d + \mu)$. Thus E_1 is a non-hyperbolic critical point and the Centre Manifold Theory will be applied to determine its stability.

We put $S' = S - S_1, I' = I, R' = R$ in (1) and we get (omitting the 'dash' sign)

$$\frac{dX}{dt} = AX + F\left(S, I, R\right),\tag{4}$$

where

$$\begin{split} X &= \begin{pmatrix} S \\ I \\ R \end{pmatrix}, \\ A &= \begin{pmatrix} (d-r) & -(d+\gamma) & \mu \\ 0 & 0 & 0 \\ 0 & \gamma & -(d+\mu) \end{pmatrix}, \\ F\left(S,I,R\right) &= \begin{pmatrix} -\frac{r}{k}S^2 - \frac{\beta}{(1+\alpha S_1)^2}IS \\ \frac{\beta}{(1+\alpha S_1)^2}IS \\ 0 \end{pmatrix}. \end{split}$$

Thus we can find a matrix

$$P = \begin{pmatrix} d(d+\gamma+\mu) & 1 & \mu \\ (d+\mu)(d-r) & 0 & 0 \\ \gamma(d-r) & 0 & r-2d-\mu \end{pmatrix}$$

so that

$$P^{-1}AP = \text{diag}(0, d - r, -(d + \mu)).$$

By using the transformation X = PY, where

$$Y = \begin{pmatrix} S' \\ I' \\ R' \end{pmatrix},$$

system (4) can be transformed into the form (omitting the 'dash' sign)

$$\frac{dS}{dt} = 0 + g_{11} (S, I, R),
\frac{dI}{dt} = -(r - d) I + g_{22} (S, I, R),
\frac{dR}{dt} = -(d + \mu) R + g_{33} (S, I, R), \text{ where}$$
(5)

$$g_{11} \equiv A_{11}S^2 + B_{11}IS + C_{11}RS,$$

$$g_{22} \equiv A_{22}S^2 + B_{22}I^2 + C_{22}R^2 + D_{22}SI + E_{22}SR + F_{22}IR,$$

$$g_{33} \equiv A_{33}S^2 + B_{33}SI + C_{33}SR$$
, where
 $A_{11} = \frac{\beta d (d + \gamma + \mu)}{(1 + \alpha S_1)^2}$

and other components will be similarly determined.

Now, we continue the process given in Theorem 1 and we get the flow on the centre manifold $W^{c}(0)$, which is defined by the differential equation

$$\frac{dS}{dt} = A_{11}S^2 + S \left\{ B_{11} \left(\frac{A_{22}}{r - d}S^2 + \dots \right) + C_{11} \left(\frac{A_{33}}{d + \mu}S^2 + \dots \right) \right\}.$$

Since $A_{11} > 0$, hence E_1 is unstable. Hence the theorem is proved.

Theorem 4. If $1 < \Delta_1 < 1 + \frac{2}{k\alpha}$, $\Delta_2 > 1$ and r > d, then the endemic equilibrium point $\tilde{E}_2^{\alpha}(S_2, I_2, R_2)$ will be locally asymptotically stable.

Proof. E_2 exists only when $\Delta_1 > 1$ and $\Delta_2 > 1$. The characteristic equation of the variational matrix of (1) at $E_2(S_2, I_2, R_2)$ is

$$\lambda^{3} + C_{1}\lambda^{2} + C_{2}\lambda + C_{3} = 0, (6)$$

where

$$C_1 = 2d + \mu + \frac{\beta I_2}{(1 + \alpha S_2)^2} - \frac{r}{\Delta_1 - 1} \left\{ \Delta_1 - (1 + \frac{2}{k\alpha}) \right\},\,$$

$$C_2 = \frac{\beta I_2 (2d + \gamma + \mu)}{(1 + \alpha S_2)^2} + (d + \mu) \left[d - \frac{r}{\Delta_1 - 1} \left\{ \Delta_1 - \left(1 + \frac{2}{k\alpha} \right) \right\} \right],$$
$$C_3 = \frac{\beta I_2 d (d + \gamma + \mu)}{(1 + \alpha S_2)^2}$$

and

$$C_{1}C_{2} - C_{3} = \frac{\beta^{2}I_{2}^{2}(2d + \gamma + \mu)}{(1 + \alpha S_{2})^{4}} + \frac{\beta I_{2}}{(1 + \alpha S_{2})^{2}} \left[-\frac{r}{\Delta_{1} - 1} \left\{ \Delta_{1} - \left(1 + \frac{2}{k\alpha}\right) \right\} \times (3d + 2\mu + \gamma) + (2d + \mu)^{2} + \gamma (d + \mu) \right] + (d + \mu) \left[\left(-r + \frac{2rS_{2}}{k} \right)^{2} - \frac{r}{\Delta_{1} - 1} \times \left\{ \Delta_{1} - \left(1 + \frac{2}{k\alpha}\right) \right\} (3d + \mu) + d (2d + \mu) \right].$$

Using the conditions stated in the theorem it can be easily shown that the Routh-Hurwitz criterion is satisfied. Hence the theorem is proved.

The stability conditions in Theorem 4 are only sufficient, but not necessary. Thus, if the conditions stated in Theorem 4 are satisfied then the disease will persist in the system. It can be noted that one can easily prove analytically that E_0 and E_1 are unstable whenever E_2 exists. Because E_2 exists if and only if $\Delta_1 > 1$, $\Delta_2 > 1$ and these two imply that r > d and basic reproduction number $R_0 > 1$. This is very significant from the biological point of view, because the analysis implies that the coexistence of two stable non-zero steady states is not possible. It means that there is no hysteresis loop. The existence conditions and stability criteria of three equilibrium points have been summarized in the following table.

IV. 2. Global Stability of Equilibria

Next, we shall obtain sufficient conditions on model parameters for the global stability of the disease free equilibrium point $E_1(S_1, 0, 0)$ and the endemic equilibrium point $E_2(S_2, I_2, R_2).$

Theorem 5. If r > d, $\Delta_1 > 1$ and $R_0 < 1$, then disease free equilibrium point $E_1(S_1, 0, 0)$ is globally asymptotically stable in the domain $D_1 = \{(S, I, R) \in R^3_+ : S < \frac{d+\gamma}{\beta - \alpha(d+\gamma)}\}.$ **Proof.** We define a Liapunov function L = I. Then $\frac{dL}{dt} = \{\frac{\beta S}{1+\alpha S} - (d+\gamma)\}I \le 0$, if $S < \frac{d+\gamma}{\beta - \alpha(d+\gamma)}$.

i.e. $\frac{dL}{dt} \leq 0$ in the domain D_1 . So for the positive definite function L, the derivative $\frac{dL}{dt}$ is negative semi definite in D_1 .

Now we consider the set where $\frac{dL}{dt} = 0$. Let $\Gamma = \{(S, I, R) \in D_1 : \frac{dL}{dt} = 0\} = \{(S, I, R) \in D_1 : dL \in I\}$ I = 0.

Let M be the largest invariant set in Γ . Then in Γ , we have

$$\frac{dS}{dt} = rS(1 - \frac{S}{k}) - dS + \mu R,$$
$$\frac{dR}{dt} = -(d + \mu)R.$$

From the second equation, we have $R \to 0$ as $t \to \infty$. Then from the first equation, we have

$$\frac{dS}{dt} = S\left(r - d - \frac{r}{k}S\right)$$

when $t \to \infty$. Since r > d, hence $S \to k(1 - \frac{d}{r}) = S_1$ as $t \to \infty$.

Hence M is singleton $\{(S_1, 0, 0)\}$. Then, it follows from the LaSalle-Liapunov theory, [22], that $E_1(S_1, 0, 0)$ is globally asymptotically stable in D_1 . Hence the theorem is proved.

Thus, we are able to establish the conditions for which the disease free equilibrium point is globally asymptotically stable. In the next theorem we devote our attention to discuss

Equilibrium points	Existence conditions	Stability conditions	
$E_0(0,0,0)$ Always exists		Stable if $r \leq d$, unstable if $r > d$	
$E_1(S_1, 0, 0)$	Exists if and only if $r > d$	Stable if $R_0 < 1$, unstable if $R_0 \ge 1$	
$\overline{E_2\left(S_2, I_2, R_2\right)}$	Exists if and only if $\Delta_1 > 1, \Delta_2 > 1$	Stable if $\Delta_1 < 1 + \frac{2}{k\alpha}$	

Tab. 2. Feasibility and local stability conditions of equilibrium points

the global stability of the endemic equilibrium point in the region D_2 .

Theorem 6. If

$$I_2 < \frac{r}{k\alpha\beta},$$

then the system is said to be globally asymptotically stable around the equilibrium point $E_2(S_2, I_2, R_2)$ in the region $D_2 = \{(S, I, R) \in R^3_+ : 1 < \frac{I_2}{I} < \frac{R_2}{R} < \frac{S_2}{S}\}$ when E_2 exists and also the system has no closed orbit in D_2 .

Proof. We consider a Liapunov function L defined as follows

$$L = \int_{S_2}^{S} \frac{S - S_2}{S} dS + \int_{I_2}^{I} \frac{I - I_2}{I} dI + \int_{R_2}^{R} \frac{R - R_2}{R} dR.$$

Then

$$\begin{split} \frac{dL}{dt} &= (\frac{S-S_2}{S})\frac{dS}{dt} + (\frac{I-I_2}{I})\frac{dI}{dt} + (\frac{R-R_2}{R})\frac{dR}{dt} \\ &= (S-S_2)\{r(1-\frac{S}{k}) - \frac{\beta I}{1+\alpha S} + \frac{\mu R}{S} - r(1-\frac{S_2}{k}) \\ &+ \frac{\beta I_2}{1+\alpha S_2} - \frac{\mu R_2}{S_2}\} + (I-I_2)\{\frac{\beta S}{1+\alpha S} - \frac{\beta S_2}{1+\alpha S_2}\} \\ &+ (R-R_2)\{\frac{\gamma I}{R} - \frac{\gamma I_2}{R_2}\} = -\frac{r}{k}(S_2-S)^2 \\ &+ \frac{\alpha \beta (S_2-S)(IS_2-SI_2)}{(1+\alpha S_2)(1+\alpha S)} - \frac{\gamma (R_2-R)(IR_2-I_2R)}{RR_2} \\ &- \frac{\mu (S_2-S)(RS_2-R_2S)}{SS_2}. \end{split}$$

Since $1 < \frac{I_2}{I} < \frac{R_2}{R} < \frac{S_2}{S}$, hence $\frac{S_2}{S} - \frac{I_2}{I} < \frac{S_2}{S} - 1$ i.e. $(IS_2 - SI_2) < I(S_2 - S)$. So

$$\begin{aligned} \frac{dL}{dt} &< -\frac{r}{k}(S_2 - S)^2 + \frac{\alpha\beta(S_2 - S)^2I}{(1 + \alpha S_2)(1 + \alpha S)} \\ &- \frac{\gamma(R_2 - R)(IR_2 - I_2R)}{RR_2} - \frac{\mu(S_2 - S)(RS_2 - R_2S)}{SS_2} < \\ &- \frac{r}{k}(S_2 - S)^2 + \alpha\beta(S_2 - S)^2I - \frac{\gamma(R_2 - R)(IR_2 - I_2R)}{RR_2} \\ &- \frac{\mu(S_2 - S)(RS_2 - R_2S)}{SS_2} < -(S_2 - S)^2\{\frac{r}{k} - \alpha\beta I_2\} \\ &- \frac{\gamma(R_2 - R)(IR_2 - I_2R)}{RR_2} - \frac{\mu(S_2 - S)(RS_2 - R_2S)}{SS_2} < 0 \end{aligned}$$

in the region D_2 , if $I_2 < \frac{r}{k\alpha\beta}$. Hence the theorem is proved.

Thus, there exists a region where the endemic equilibrium point is globally asymptotically stable. In Theorem 5 and Theorem 6 we obtain the conditions for which the equilibrium points are globally asymptotically stable. Biologically, globally asymptotically stable means whatever the initial number of S, I, R, the system finally will converge to the equilibrium point. From these two theorems one can conclude that despite the initial number of infected population, the system will converge to the corresponding equilibrium point when the conditions are satisfied.

Now we shall discuss the particular case in absence of the inhibitory factor. Then the reduced equilibrium point $E_2(S_2, I_2, R_2)$ is globally asymptotically stable in some region.

Theorem 7. For $\alpha = 0$ the system is said to be globally asymptotically stable around the equilibrium point $E_2(S_2, I_2, R_2)$ in the region

$$D_3 = \left\{ (S, I, R) \in R^3_+ : 1 < \frac{I_2}{I} < \frac{R_2}{R} < \frac{S_2}{S} \right\}$$

if E_2 exists.

Proof. We choose a Liapunov function L defined as follows

$$L = \int_{S_2}^{S} \frac{S - S_2}{S} dS + \int_{I_2}^{I} \frac{I - I_2}{I} dI + \int_{R_2}^{R} \frac{R - R_2}{R} dR.$$

Then

$$\begin{aligned} \frac{dL}{dt} &= \left(\frac{S-S_2}{S}\right) \frac{dS}{dt} + \left(\frac{I-I_2}{I}\right) \frac{dI}{dt} + \left(\frac{R-R_2}{R}\right) \frac{dR}{dt} \\ &= (S-S_2) \{r(1-\frac{S}{k}) - \beta I - d + \frac{\mu R}{S}\} \\ &+ (I-I_2) \{\beta S - (d+\gamma)\} \\ &+ (R-R_2) \{\frac{\gamma I}{R} - (d+\mu)\} \\ &= -\frac{r}{k} (S_2 - S)^2 - \frac{\gamma (R_2 - R)(IR_2 - I_2R)}{RR_2} \\ &- \frac{\mu (S_2 - S)(RS_2 - R_2S)}{SS_2} < 0 \end{aligned}$$

in the region D_3 . Hence the theorem is proved.

When the recovered class does not losse immunity and so does not become susceptible, then the reduced equilibrium point $E_2(S_2, I_2, R_2)$ will be globally asymptotically stable. **Lemma 2.** If $k\alpha < 1$, the system does not have a closed orbit in the first quadrant of (S, I) plane in the case $\mu = 0$.

Proof. If $\mu = 0$, then the system (1) is reduced to

$$\frac{dS}{dt} = rS\left(1 - \frac{S}{k}\right) - \frac{\beta SI}{1 + \alpha S} - dS \equiv F\left(S, I\right)$$
$$\frac{dI}{dt} = \frac{\beta SI}{1 + \alpha S} - \left(d + \gamma\right)I \equiv G\left(S, I\right).$$

Now, taking the Dulac function $B\left(S,I\right)=\frac{1+\alpha S}{SI},$ we obtain

$$\begin{aligned} &\frac{\partial}{\partial S} \left(BF \right) + \frac{\partial}{\partial I} \left(BG \right) = \\ &= \frac{r \left\{ \left(1 - \frac{S}{k} \right) \alpha - \left(1 + \alpha S \right) \frac{1}{k} \right\} - d\alpha}{I} < 0 \text{ if } k\alpha < 1. \end{aligned}$$

This proves the lemma.

Theorem 8. If $1 < \Delta_1 < 1 + \frac{2}{k\alpha}$, $\Delta_2 > 1$ and $k\alpha < 1$, then the endemic equilibrium point $E_2(S_2, I_2, R_2)$ will be globally asymptotically stable in the case $\mu = 0$.

Proof. In Theorem 4, it has been proved that E_2 is locally asymptotically stable if $1 < \Delta_1 < 1 + \frac{2}{k\alpha}$ and $\Delta_2 > 1$. Thus, it follows from Lemma 2 that E_2 is globally asymptotically stable.

V. BIFURCATION ANALYSIS OF THE MODEL

A bifurcation is a qualitative change in the behaviour of solutions as one or more parameters are varied. A bifurcation is called local bifurcation if the qualitative change occurs in the neighbourhood of an equilibrium point or periodic solution. Here we present two local bifurcations: one is Transcritical bifurcation and the other is Hopf bifurcation.

Theorem 9. If $\beta k (r - d) > r (d + \gamma)$, then the system (1) experiences Transcritical bifurcation at the disease free equilibrium point $E_1(S_1, 0, 0)$ with respect to the inhibitory factor α .

Proof. Let

$$f\left(S,I,R;\alpha\right) = \begin{pmatrix} rS\left(1-\frac{S}{k}\right) - \frac{\beta SI}{1+\alpha S} - dS + \mu R\\ \frac{\beta SI}{1+\alpha S} - (d+\gamma)I\\ \gamma I - (d+\mu)R \end{pmatrix},$$
$$\alpha_0 = \frac{\beta}{d+\gamma} - \frac{r}{k\left(r-d\right)}$$

and

$$A = Df(E_1, \alpha_0) = \begin{pmatrix} (d-r) & -\frac{\beta S_1}{1+\alpha S_1} & \mu \\ 0 & 0 & 0 \\ 0 & \gamma & -(d+\mu) \end{pmatrix}.$$

Clearly, $f(E_1, \alpha_0) = 0$ and A has a simple eigenvalue $\lambda = 0$. So we shall use Sotomayor theorem [23] to check the nature of solutions for $\alpha = \alpha_0$.

Now, the eigenvectors of A and A^T corresponding to the eigenvalue $\lambda = 0$ are, respectively,

$$V = \begin{pmatrix} \beta S_1 \left(d + \mu \right) - \mu \gamma \left(1 + \alpha S_1 \right) \\ \left(d + \mu \right) \left(1 + \alpha S_1 \right) \left(d - r \right) \\ \gamma \left(1 + \alpha S_1 \right) \left(d - r \right) \end{pmatrix}$$

and

$$W = \left(\begin{array}{c} 0 \\ 1 \\ 0 \end{array} \right).$$

Let f_{α} denote the vector of partial derivatives of the components of f with respect to α .

Thus

$$f_{\alpha} = \begin{pmatrix} \frac{\beta S^2 I}{(1+\alpha S)^2} \\ -\frac{\beta S^2 I}{(1+\alpha S)^2} \\ 0 \end{pmatrix}$$

and so

$$f_{\alpha}\left(E_{1},\alpha_{0}\right)=\left(\begin{array}{c}0\\0\\0\end{array}\right).$$

Therefore, $W^T f_{\alpha}(E_1, \alpha_0) = (0)$. Again,

$$W^{T} \left[Df_{\alpha} \left(E_{1}, \alpha_{0} \right) V \right] =$$

$$= (0, 1, 0) \left(\begin{array}{c} -\frac{(d+\gamma)(d+\mu)(r-d)^{2}k}{r} \\ \frac{(d+\gamma)(d+\mu)(r-d)^{2}k}{r} \\ 0 \end{array} \right) =$$

$$= \left(\frac{(d+\gamma)(d+\mu)(r-d)^{2}k}{r} \right) \neq (0).$$

Finally,

$$W^{T} \left[D^{2} f (E_{1}, \alpha_{0}) (V, V) \right] =$$

$$= \left(\frac{2\beta (d + \mu) (d - r) \{\beta S_{1} (d + \mu) - \mu \gamma (1 + \alpha_{0} S_{1})\}}{(1 + \alpha_{0} S_{1})} \right)$$

$$\neq (0).$$

Therefore, all the conditions for Transcritical bifurcation in Sotomayor theorem are satisfied. Hence, the system (1) experiences a Transcritical bifurcation at the equilibrium point $E_1(S_1, 0, 0)$ as the parameter α varies through the bifurcation value $\alpha = \alpha_0$. Hence the theorem is proved.

Now we consider the other possible scenario, where several limit cycles bifurcate from an equilibrium point. We shall vary r in the system to obtain a Hopf bifurcation around the endemic equilibrium point E_2 . In Theorem 4, we see that the characteristic equation of the Jacobian matrix of the system (1) at the equilibrium point E_2 is $\lambda^3 + C_1\lambda^2 + C_2\lambda + C_3 = 0$. Since the expressions of C_1, C_2, C_3 and $C_1C_2 - C_3$ depend

on intrinsic growth rate r, hence the sign of C_1, C_2, C_3 and $C_1C_2 - C_3$ can be controlled by changing the values of r. For Hopf bifurcation around E_2 at $r = r_c$, we must have $C_1(r_c) C_2(r_c) - C_3(r_c) = 0$ and C_1, C_2, C_3 are all positive. For $r \in (r_c - \varepsilon, r_c + \varepsilon)$, the roots are, in general, of the form

$$y_{1}(r) = \alpha_{0}(r) + i\beta_{0}(r) ,$$

$$y_{2}(r) = \alpha_{0}(r) - i\beta_{0}(r) ,$$

$$y_{3}(r) = -C_{1}(r) .$$

To apply the Hopf bifurcation theorem, we need to verify the transversality condition

$$\operatorname{Re}\left[\frac{dy_i}{dr}\right]_{r=r_c} \neq 0, \ i=1,2$$

which is equivalent to $[(C_1C'_2 + C'_1C_2 - C'_3)]_{r=r_c} \neq 0$. We choose the critical value of r, say r_c , in such a manner that $C_1C_2 - C_3 = 0$ and $[(C_1C'_2 + C'_1C_2 - C'_3)] \neq 0$ at $r = r_c$ which will hold if $\Delta_1 > 1 + \frac{2}{k\alpha}$ and $\Delta_2 > 1$. Thus we summarize the details in the following:

Theorem 10. If $\Delta_1 > 1 + \frac{2}{k\alpha}$ and $\Delta_2 > 1$, then the system may exhibit a Hopf bifurcation leading to a family of periodic solutions that bifurcates from E_2 for suitable values of intrinsic growth rate r of the susceptible class in a neighbourhood of r_c .

Next, we check the stability of the bifurcating periodic orbits. We know that the periodic solutions are stable for supercritical bifurcation, and they are unstable for subcritical bifurcation. We need to compute the index number Γ in the Hopf bifurcation theorem by using the Centre Manifold Theorem.

We first translate the equilibrium point $E_2(S_2, I_2, R_2)$ to the origin. So we put $S' = S - S_2$, $I' = I - I_2$ and $R' = R - R_2$ in (1) and we get (omitting the 'dash' sign)

$$\frac{dX}{dt} = BX + F(X).$$
⁽⁷⁾

Here
$$X = \begin{pmatrix} S \\ I \\ R \end{pmatrix}, B = \begin{pmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & 0 \\ 0 & a_{31} & a_{32} \end{pmatrix}$$
,

$$F(X) = \begin{pmatrix} a_{14}S^2 + a_{15}SI \\ a_{23}S^2 + a_{24}SI + a_{25}I^2 \\ 0 \end{pmatrix},$$

where

$$\begin{split} a_{11} &= r (1 - \frac{2S_2}{k}) - d - \frac{\beta I_2}{\left(1 + \alpha S_2\right)^2} \\ a_{12} &= -\frac{\beta S_2}{1 + \alpha S_2}, \\ a_{13} &= \mu, \\ a_{14} &= -\frac{r}{k} + \frac{\alpha \beta I_2}{\left(1 + \alpha S_2\right)^3}, \\ a_{15} &= -\frac{2\beta}{\left(1 + \alpha S_2\right)^2}, \\ a_{21} &= \frac{\beta I_2}{\left(1 + \alpha S_2\right)^2}, \\ a_{22} &= \frac{\beta S_2}{1 + \alpha S_2}, \\ a_{23} &= -\frac{\alpha \beta I_2}{\left(1 + \alpha S_2\right)^3}, \\ a_{24} &= \frac{\beta}{\left(1 + \alpha S_2\right)^2}, \\ a_{25} &= -\frac{\left(d + \gamma\right)}{2}, \\ a_{31} &= \gamma, \\ a_{32} &= -\left(d + \mu\right). \end{split}$$

We know that the eigenvalues of the matrix B are $\lambda = \pm \sqrt{C_2}i$, $-C_1$ at $r = r_c$. So an eigenvector of B corresponding to the eigenvalue

 $\lambda = \sqrt{C_2}i$

is

$$\left(\begin{array}{c} \alpha_1\\ \alpha_2\\ \alpha_3 \end{array}\right) + i \left(\begin{array}{c} \beta_1\\ \beta_2\\ \beta_3 \end{array}\right)$$

and corresponding to the eigenvalue $\lambda = -C_1$ is $\begin{pmatrix} \gamma_1 \\ \gamma_2 \\ \gamma_3 \end{pmatrix}$,

where

$$\begin{split} &\alpha_1 = -\frac{a_{22}}{a_{21}}, \ \alpha_2 = 1, \ \alpha_3 = -\frac{a_{31} \cdot a_{32}}{c_2 + a_{32}^2}, \\ &\beta_1 = \frac{\sqrt{c_2}}{a_{21}}, \ \beta_2 = 0, \ \beta_3 = -\frac{a_{31} \cdot \sqrt{c_2}}{c_2 + a_{32}^2}, \\ &\gamma_1 = -\frac{c_1 + a_{22}}{a_{21}}, \ \gamma_2 = 1, \ \gamma_3 = -\frac{a_{31}}{c_1 + a_{32}} \end{split}$$

Now, by the transformation X = PY, the system (7) can be written as

$$\frac{dY}{dt} = \left(P^{-1}BP\right)Y + P^{-1}F\left(PY\right),\tag{8}$$



Fig. 1. (a) Time series solutions and (b) phase portrait for the parametric values reported in Table 3 with r = 3.07, (c) time series solutions and (d) limit cycle for the parametric values reported in Table 3 with r = 3.136

where

$$X = \left(\begin{array}{c} S \\ I \\ R \end{array} \right),$$

$$Y = \left(\begin{array}{c} y_1\\y_2\\y_3\end{array}\right),$$

 $P = \begin{pmatrix} \beta_1 & \alpha_1 & \gamma_1 \\ \beta_2 & \alpha_2 & \gamma_2 \\ \beta_3 & \alpha_3 & \gamma_3 \end{pmatrix}$

and so

$$P^{-1} = \begin{pmatrix} p_{11} & p_{12} & p_{13} \\ p_{21} & p_{22} & p_{23} \\ p_{31} & p_{32} & p_{33} \end{pmatrix}$$

(where p_{ij} can be evaluated from the relation $PP^{-1} = P^{-1}P = I_3$).

We rewrite the system (8) as

$$\begin{pmatrix} \dot{y}_{1} \\ \dot{y}_{2} \\ \dot{y}_{3} \end{pmatrix} = \begin{pmatrix} 0 & -\sqrt{C_{2}} & 0 \\ \sqrt{C_{2}} & 0 & 0 \\ 0 & 0 & -C_{1} \end{pmatrix} \times \begin{pmatrix} y_{1} \\ y_{2} \\ y_{3} \end{pmatrix} + \begin{pmatrix} f_{1} \\ f_{2} \\ f_{3} \end{pmatrix}.$$
(9)

Here,

$$f_1(y_1, y_2, y_3) = p_{11}(a_{14}S^2 + a_{15}SI)$$

+ $p_{12}(a_{23}S^2 + a_{24}SI + a_{25}I^2),$

$$f_2(y_1, y_2, y_3) = p_{21}(a_{14}S^2)$$

+ $a_{15}SI$) + $p_{22}(a_{23}S^2 + a_{24}SI + a_{25}I^2)$,

$$f_3(y_1, y_2, y_3) = p_{31}(a_{14}S^2 + a_{15}SI) + p_{32}(a_{23}S^2 + a_{24}SI + a_{25}I^2);$$

where

$$S = \beta_1 y_1 + \alpha_1 y_2 + \gamma_1 y_3,$$
$$I = \beta_2 y_1 + \alpha_2 y_2 + \gamma_2 y_3,$$



Fig. 2. Stability region for the equilibrium point $E_1(S_1, 0, 0)$ in $\alpha - r$ plane and value of other parameters β, d, k, γ reported in Table 4. The red line in the boundary denotes the boundary curve

 $R = \beta_3 y_1 + \alpha_3 y_2 + \gamma_3 y_3.$

By the Centre Manifold Theory there exists a centre manifold of (9), which can be expressed by

$$W^{c}(0) = \{(y_1, y_2, y_3) | y_3 = h(y_1, y_2)\}$$

for

$$|y_1| < \delta, |y_2| < \delta\},\$$

where $\delta (> 0)$ is some number. Thus

$$y_3 = h(y_1, y_2) = k_1 y_1^2 + k_2 y_1 y_2 + k_3 y_2^2 + \cdots,$$

where the constants k_i (i = 1, 2, 3, ...) can be easily deter-

mined from the identity relation

$$(2k_1y_1 + k_2y_2 + \cdots) \left\{ -\sqrt{c_2}y_2 + f_1\left(y_1, y_2, k_1y_1^2 + k_2y_1y_2 + k_3y_2^2 + \cdots\right) \right\} + (k_2y_1 + 2k_3y_2 + \cdots) \left\{ \sqrt{c_2}y_1 + f_2\left(y_1, y_2, k_1y_1^2 + k_2y_1y_2 + k_3y_2^2 + \cdots\right) \right\} + c_1\left(k_1y_1^2 + k_2y_1y_2 + k_3y_2^2 + \cdots\right) - f_3\left(y_1, y_2, k_1y_1^2 + k_2y_1y_2 + k_3y_2^2 + \cdots\right) = 0.$$

Thus, the flow on the centre manifold has the form

$$\begin{pmatrix} \dot{y}_1 \\ \dot{y}_2 \end{pmatrix} = \begin{pmatrix} 0 & -\sqrt{C_2} \\ \sqrt{C_2} & 0 \end{pmatrix} \begin{pmatrix} y_1 \\ y_2 \end{pmatrix}$$
$$+ \begin{pmatrix} g^1(y_1, y_2) \\ g^2(y_1, y_2) \end{pmatrix},$$



Fig. 3. Stability region for the equilibrium point $E_1(S_1, 0, 0)$ in $\alpha - \beta$ plane and value of other parameters r, d, k, γ, μ reported in Table 4. The pink line in the boundary denotes the boundary curve



Fig. 4. Instability zone for the equilibrium point E_1 (S_1 , 0, 0) in $\alpha - k$ plane and value of other parameters r, β , d, γ reported in Table 4. The red line in the boundary denotes the boundary curve

where

$$g^{1}(y_{1}, y_{2}) = f_{1}(y_{1}, y_{2}, h(y_{1}, y_{2})),$$
$$g^{2}(y_{1}, y_{2}) = f_{2}(y_{1}, y_{2}, h(y_{1}, y_{2})).$$

Therefore, we can easily compute the index Γ , where

$$\begin{split} \Gamma = & \frac{1}{16} \left\{ g_{y_1y_1y_1}^1 + g_{y_1y_2y_2}^1 + g_{y_1y_1y_2}^2 + g_{y_2y_2y_2}^2 \right\} \\ & + \frac{1}{16\sqrt{C_2}} \left\{ g_{y_1y_2}^1 (g_{y_1y_1}^1 + g_{y_2y_2}^1) \\ & - g_{y_1y_2}^2 (g_{y_1y_1}^2 + g_{y_2y_2}^2) - g_{y_1y_1}^1 g_{y_1y_1}^2 + g_{y_2y_2}^1 g_{y_2y_2}^2 \right\} \end{split}$$

By using the Hopf bifurcation theorem, we obtain the following result about the periodic solutions bifurcated at E_2 .

Theorem 11. If $\Gamma < 0$, the periodic solution in the neighbourhood of the endemic equilibrium point $E_2(S_2, I_2, R_2)$ is stable, while $\Gamma > 0$ the periodic solution is unstable.

When $\Gamma < 0$, the system is said to be supercritical and the case $\Gamma > 0$ is refereed as the subcritical, where Γ is defined in the text.

VI. NUMERICAL SIMULATIONS

In this section, we present some numerical simulation results to exemplify the analytical studies. The values of the parameters are chosen arbitrarily.

Tab. 3. Values of the parameters

k	β	d	γ	μ	α
25	2.9	2.3	0.001	0.01	0.21

Figure 1(a)-(d) show the existence of Hopf bifurcation about the endemic equilibrium point E_2 with respect to the intrinsic growth rate r for the given set of parametric values. Figure 1(a)-(b) represent the stable solution for $r < r_c$ and in Figure 1(c)-(d), we see that periodic solutions arise for $r = r_c (\approx 3.136)$. Hence the system loses stability when rpasses through $r = r_c$ which suggests that Hopf bifurcation takes place at $r = r_c$.

Tab. 4. Values of the parameters

k	β	d	γ	μ	r
55	2.9	2.3	0.001	0.01	4

If r > d, then $E_1(S_1, 0, 0)$ is stable for $R_0 < 1$ and is unstable for $R_0 \ge 1$. So $R_0 = 1$ is the boundary curve for the Transcritical bifurcation at the point $E_1(S_1, 0, 0)$. Figure 2 and 3 represent the stability regions for E_1 in two parametric domain (α, r) and (α, β) , respectively. The unstable region for $E_1(S_1, 0, 0)$ is marked in two parametric domain (α, k) by Figure 4. We also have indicated the boundary curve in all three figures. The boundary lines in the Figures (2-4) represent the Transcritical bifurcation curves. When we analyse Figure 2, we can see that if we fix α in the range (0, 1.2) then the solution in the neighbourhood of disease free equilibrium point $E_1(S_1, 0, 0)$ is unstable for a low value of the intrinsic growth rate (r < 2.4 approx) but for r > 2.4 it is stable and if $\alpha > 1.2$ the solution is unstable for all r. Again, from Figure 3 we have seen that for the stability of the DFE, the value of inhibitory coefficient has to be high for high rate of infection, and Figure 4 shows that for very low carrying capacity of the system (k < 1.9 approx), the DFE is stable for $\alpha = 0$ but for k > 1.9, the inhibitory coefficient has to be increased for the stability of DFE.

Again, $E_2(S_2, I_2, R_2)$ is asymptotically stable if $C_1 > 0$,

 $C_3 > 0$ and $C_1C_2 - C_3 > 0$. For different values of the parameters, the two parametric stability regions are shown in Figure 5(a)-(c) for the endemic equilibrium point $E_2(S_2, I_2, R_2)$ in two parametric domain (k, r), (α, r) and (β, r) , respectively. Figure 5(a) shows that the endemic equilibrium point E_2 is stable if 2.39 < r < 3.136 for the parametric values given in Table 3. For these parametric values, E_2 does not exist if r < 2.39 and is unstable if r > 3.136. Again, if we fix the parameters k, β, d, γ and μ as listed in Table 3 then Figure 5(b) shows that for every values of r, E_2 cannot be stable if $\alpha > 1.26$. In fact, E_2 is not feasible for $\alpha > 1.26$. Moreover, for $\alpha < 1.26$ the region below the blue coloured region represents the infeasible region for E_2 and the region above the blue coloured region represents the unstable region for the endemic equilibrium point E_2 . Finally, Figure 5(c) indicates E_2 cannot be stable if $\beta < 0.5$ for the other parametric values given in Table 3. In fact, E_2 is infeasible for $\beta < 0.5.$ For $\beta > 0.5$ the regions below and above the stable

region represent the infeasible and unstable region for E_2 , respectively.

Now, we will present the one-parameter bifurcation diagrams to represent the behaviour of the solutions for different values of the bifurcation parameters r, α, β, k .

To illustrate the effect of intrinsic growth rate r, we have plotted all the populations S, I and R(cf. Figure 6) as the variation of the values of r. From the figure we observe that trivial equilibrium point E_0 is stable for $r \in (0, 2.3]$ and DFE E_1 is stable for $r \in (2.3, 2.39)$. Then all three populations exist at the positive state simultaneously and the populations I and R increase with increasing values of r and finally periodic solutions arise in the system for high values of r ($r \ge 3.136$) (note that for the given parametric values the eigenvalues associated with the Jacobian matrix at $E_2(0.95, 0.3, 0.00013)$ are $-2.31, 0 \pm 1.1722i$ when r = 3.136, which indicates existence of a periodic solution). From Figures 1 and 6 we can conclude that the system undergoes the supercritical Hopf



Fig. 5. Stability regions for the endemic equilibrium point $E_2(S_2, I_2, R_2)$ in (a)(k, r) parametric domain and other parametric values are reported in Table 3, (b) (α, r) parametric domain and other parametric values are reported in Table 3, (c) (β, r) parametric domain and other parametric values are reported in Table 3



Fig. 6. (a) Bifurcation diagram w.r.t. r and other parametric values reported in Table 3, (b) local amplification of the Figure (a) in the interval [2.2, 3.2]



Fig. 7. (a) Bifurcation diagram w.r.t. α and other parametric values reported in Table 3 with r = 4, (b) local amplification of the Figure (a) in the interval [0,0.14]



Fig. 8. Bifurcation diagram w.r.t. β and other parametric values reported in Table 3 with r = 4

bifurcation.

To illustrate the effect of inhibitory factor α , we plot the bifurcation diagram (cf. Figure 7) by taking α as a bifurcation parameter. This bifurcation diagram shows that the endemic equilibrium point E_2 is locally stable for lower values of α . The system changes from the stable steady state to periodic oscillation at $\alpha = 0.1125 (= \alpha_1)$ (note that for the given parametric values the eigenvalues associated with the Jacobian matrix at $E_2(0.87, 0.59, 0.00025)$ are $-2.31, \pm 1.8084i$ when $\alpha = \alpha_1$) and the solution changes from the periodic solution to the stable steady state at $\alpha = 1.053 (= \alpha_2)$ (note that for the given parametric values the eigenvalues associated with the Jacobian matrix at $E_2(4.82, 1.95, 0.00084)$ are $-2.31, \pm 0.5927i$ when $\alpha = \alpha_2$) and beyond the value of α_2 , no oscillation is observed. This means for $\alpha \in (\alpha_1, \alpha_2)E_2$ is unstable, and all the populations S, I, R co-exist in the oscillatory mode. Therefore, the system exhibits switching behaviour twice and the system undergoes two Hopf bifurcations as a function of the control parameter α . Figure 7 illustrates supercritical and subcritical Hopf bifurcations take place at $\alpha = \alpha_1$ and $\alpha = \alpha_2$, respectively. The bifurcation

diagram also shows that the infected population goes extinct with increasing values of the inhibitory factor. So, the inhibitory coefficient plays a crucial role in the changing of dynamics of the infectious disease.

Next, we have plotted all populations (cf. Figure 8) as the variation of bifurcation parameter β . It is clear from Figure 8 that there is a range (0, 0.69977) of β for which the DFE is stable, after that the susceptible population starts to decrease and other two populations increase and finally at $\beta = 1.27$ the system experiences the supercritical Hopf bifurcation (note that for the given parametric values the eigenvalues associated with the Jacobian matrix at $E_2(2.92, 1.57, 0.00068)$ are $-2.31, 0 \pm 1.325i$ when $\beta = 1.27$). Thus, for the low transmission rate of infection the disease is absent but for high transmission rate of infection the disease will persist in the system very strongly and, as it is periodic, the disease is difficult to control.

The role of carrying capacity of the system should not be neglected. The one-parametric bifurcation diagram (cf. Figure 9) demonstrates how the system behaves with the variation of the numerical values of k. It suggests that if we increase



Fig. 9. Bifurcation diagram w.r.t. k and other parametric values reported in Table 3 with r = 4

the value of k, the dynamics of the system moves from stability to oscillation through a point of the supercritical Hopf bifurcation at k = 15.7 (note that for the given parametric values the eigenvalues associated with the Jacobian matrix at $E_2(0.95, 0.6, 0.00026)$ are $-2.31, \pm 1.67i$ when k = 15.7). Periodicity of the solutions for high carrying capacity of the system indicates the disease will come back again in the system.

VII. CONCLUSIONS

This paper deals with a SIRS model where the logistic growth rate of susceptibles and the inhibitory factor in the incidence rate are considered. The local stability analysis of both hyperbolic and non-hyperbolic equilibrium points is analysed. We have seen that the dynamical system is globally asymptotically stable around the disease free equilibrium point E_1 (S_1 , 0, 0) and the endemic equilibrium point E_2 (S_2 , I_2 , R_2) in some domains, which means the solutions converge to the corresponding equilibrium point for all initial values of S, I and R within the specific domain. It is also observed from the obtained result that there exists a threshold value of inhibitory factor $\alpha = \alpha_0$, such that the disease free equilibrium point E_1 is stable for $\alpha > \alpha_0$ and is unstable for $\alpha \le \alpha_0$. This implies that the awareness factor plays an important role to control the disease. It was also found that

Bifurcation parameter	Parameter range	E_0	E_1	E_2
r	$0 < r \le 2.3$	Feasible, stable	Infeasible	Infeasible
	2.3 < r < 2.39	Feasible, unstable	Feasible, stable	Infeasible
	$2.39 \le r < 3.136$	Feasible, unstable	Feasible, unstable	Feasible, stable
	$r \ge 3.136$	Feasible, unstable	Feasible, unstable	Feasible, unstable
α	$0 < \alpha \le 0.1125$	Feasible, unstable	Feasible, unstable	Feasible, stable
	$0.1125 < \alpha \le 1.053$	Feasible, unstable	Feasible, unstable	Feasible, unstable
	$1.053 < \alpha \le 1.166$	Feasible, unstable	Feasible, unstable	Feasible, stable
	$\alpha > 1.166$	Feasible, unstable	Feasible, stable	Infeasible
β	$0<\beta<0.69977$	Feasible, unstable	Feasible, stable	Infeasible
	$0.69977 \leq \beta < 1.27$	Feasible, unstable	Feasible, unstable	Feasible, stable
	$\beta \ge 1.27$	Feasible, unstable	Feasible, unstable	Feasible, unstable
k	0 < k < 2.2402	Feasible, unstable	Feasible, stable	Infeasible
	$2.2402 \le k < 15.7$	Feasible, unstable	Feasible, unstable	Feasible, stable
	$k \ge 15.7$	Feasible, unstable	Feasible, unstable	Feasible, unstable

Tab. 5. Nature of equilibrium points in various ranges of bifurcation parameters, keeping other parameters fixed as in Table 3 with r = 4

the system undergoes a Hopf bifurcation leading to a fam- [10] J.J. Wang, J.Z. Zhang, Z. Jin, Analysis of an SIR model with ily of periodic solutions that bifurcates from E_2 for suitable values of the intrinsic growth rate of susceptible class. We have proved that there is no closed orbit in D_2 for the system (1) and the reduced system for $\mu = 0$ does not have any closed orbit if $k\alpha < 1$. It is also proved analytically that the trivial and disease free equilibrium points are unstable when endemic equilibrium point exists.

Finally, in section 6 we have drawn the stability or instability regions for the disease free and endemic equilibrium points. The bifurcation diagram is a very useful tool to describe the stability analysis and long-term behaviour of the system in a single figure. Therefore, we have plotted the bifurcation diagrams with respect to the parameters r, α , β and k. A detailed presentation of the relationship between the bifurcation parameters r, α, β, k and the nature of the equilibrium points is done in section 6 and we have summarized this in Table 5. The entire study of the paper is mainly based on the deterministic framework and our proposed model is valid for a large population only. The work is a theoretical modelling and it can be further justified using experimental results.

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References

- [1] W.Kermack, A. Mckendric, A contribution to mathematical theory of epidemics, Proc. Roy. Soc. Lond. A Mat. 115, 700-721 (1927).
- [2] W.Kermack, A. Mckendric, Contributions to the mathematical theory of epidemics-I, Bulletin of Mathematical Biology 53, 33-55 (1991).
- [3] O. Diekman, J.A.P. Heesterbeek, *Mathematical Epidemiology* of Infectious Disease, Wiley, New York, 2000.
- [4] N.T.J. Bailey, The Mathematical Theory of Infectious Diseases, Griffin, London, 1975.
- [5] J.D. Murray, Mathematical Biology, Springer, New York, 1993.
- [6] R.M. Anderson, R.M. May, Infectious Diseases of Humans: Dynamics and Control, Oxford University Press, 1998.
- [7] Z. Ma, J. Li (eds.), Dynamical Modelling and Analysis of Epidemics, World Scientific, 2009.
- [8] F. Brauer, C. Castillo-Chavez, Mathematical Models in Population Biology and Epidemiology, Springer, 2011.
- Y. Enatsu, E. Messina, Y. Muroya, Y. Nakata, E. Russo and A. [9] Vecchio, Stability analysis of delayed SIR epidemic models with a class of nonlinear incidence rates, Applied Mathematics and Computation 218, 5327-5336 (2012).

- bilinear incidence rate, Nonlinear Anal. RWA 11, 2390-2402 (2009).
- [11] J. Wang, S. Liu, B. Zhen and Y. Takeuchi, Qualitative and bifurcation analysis using an SIR model with a saturated treatment function, Mathematical and Computer Modelling 55, 710-722 (2012).
- [12] T.K. Kar, P. Mandal, Global dynamics and bifurcation in delayed SIR epidemic model, Nonlinear analysis: Real World Applications 12, 2058-2068 (2011).
- [13] W.M. Liu, S.A. Levin, Y. Iwasa, Influence of nonlinear incidence rates upon the behaviour of SIRS epidemiological models, J. Math. Biol 23, 187-204 (1986).
- [14] J.Z. Zhang, Z. Jin, Q.X. Liu, Z.Y. Zhang, Analysis of a delayed SIR model with non-linear incidence rate, Discrete Dynamics in Nature and Society, (2008).
- [15] L. Cai, S. Guo, X. Li, M. Ghosh, Global dynamics of a dengue epidemic mathematical model, Chaos Solitons Fractals 42, 2297-2304 (2009).
- [16] Z.X. Liu, S. Liu, H. Wang, Backward bifurcation of an epidemic model with standard incidence rate and treatment rate, Nonlinear Anal. RWA 348, 433-443 (2008).
- [17] L. Zhou, M. Fan, Dynamics of an SIR epidemic model with limited medical resources revisited, Nonlinear Anal. RWA 13, 312-324 (2012).
- [18] D. Xiao, S. Ruan, Global analysis of an epidemic model with non-monotone incidence rate, Math. Biosci. 208, 419-429 (2007).
- [19] X. Zhang, X.N. Liu, Backward bifurcation and global dynamics of an SIS epidemic model with general incidence rate and treatment, Nonlinear Anal. RWA 10, 565-575 (2009).
- [20] V. Capasso, G. Serio, A generalization of the Kermack-Mckendrick deterministic epidemic model, Math. Biosci. 42, 43-61 (1978).
- [21] P. Van den Driessche and J. Watmough, Reproduction Numbers and Sub-Threshold Endemic Equilibria for Compartmental Models of Disease Trans-mission, Mathematical Biosciences 180, 29-48 (2002).
- [22] J.K. Hale, Ordinary Differential Equations 2nd ed., Krieger, Basel, 1980.
- [23] L. Perko, Differential Equations and Dynamical Systems, Springer, New York, 2000.
- T.C.Gard, Persistence in Food Webs: Holling-Type Food Chains, [24] Math Biosci 49, 61-67 (1980).
- [25] M.B. Trawicki, Deterministic Seirs Epidemic Model for Modeling Vital Dynamics, Vaccinations, and Temporary Immunity, Mathematics 5(1), 7 (2017).
- [26] L. Wang, D. Zhou, Z. Liu, D. Xu, X. Zhang, Media alert in an SIS epidemic model with logistic growth, Journal of Biological Dynamics 11, 120-137 (2017).

Appendix: Boundedness and Permanence of the System

The concept of boundedness and permanence was introduced in population biology and has been studied extensively. This concept is very important in mathematical epidemiology as well. Boundedness of a system implies that the system is biologically well behaved and permanence implies that the disease will be maintained globally, irrespective of the initial composition.

Theorem A. The region

$$D = \left\{ (S, I, R) \in R^3_+ : S + I + R \le \frac{rk}{d} \right\}$$

is a positively invariant set for the system (1). **Proof.** Let N = S + I + R. Then

$$\frac{dN}{dt} = rS\left(1 - \frac{S}{k}\right) - dN =$$
$$-\frac{r}{k}(k - S)^2 - dN + rk - rS \le rk - dN.$$

It follows that $\limsup N(t) \leq \frac{rk}{d}$ as $t \to \infty$. Hence the theorem is proved.

Theorem B. If $r > d + \gamma$, the system (1) is persistent and permanent.

Proof. We make use of the function $\rho(S, I, R) = S^{r_1}I^{r_2}R^{r_3}$, where r_i 's are positive constants to be determined in the process of verifying that ρ is a persistence function for the system (1).

Here

$$\begin{split} \dot{\rho} =& \rho \Big[r_1 \left\{ r \left(1 - \frac{S}{k} \right) - \frac{\beta I}{1 + \alpha S} - d + \frac{\mu R}{S} \right\} \\ &+ r_2 \left\{ \frac{\beta S}{1 + \alpha S} - (d + \gamma) \right\} + r_3 \left\{ \frac{\gamma I}{R} - (d + \mu) \right\} \Big] \\ &\geq \rho \Big[\left\{ r_1 r - d - r_2 \left(d + \gamma \right) - r_3 \left(d + \mu \right) \right\} \\ &+ \frac{r_2 \beta S}{1 + \alpha S} - \frac{r_1 \beta I}{1 + \alpha S} - \frac{r_1 r S}{k} \Big]. \end{split}$$

We take r_1, r_2, r_3 such a manner that

$$rr_1 > d + r_2 (d + \gamma) + r_3 (d + \mu)$$

and

$$r_1 = r_2.$$

Then we have

$$\dot{\rho} \ge \rho \left[\left\{ (r - d - \gamma) r_1 - (d + \mu) r_3 - d \right\} - \frac{r_1 r S}{k} \right],$$

which is nonnegative for sufficiently small S. Thus ρ is a persistence function [24] for (1). Therefore, from Theorem A we conclude that the system (1) is also permanent.



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