

Stability and Bifurcation of an SIS Epidemic Model with Saturated Incidence Rate and Treatment Function

Raid kamel Naji^a, Ashraf Adnan Thirthar^{b*}

^aDepartment of Mathematics, College of Science, University of Baghdad,
Baghdad, Iraq.

^bDirectorate-General for Education of Anbar, Ministry of Education, Anbar,
Iraq.

E-mail: rknaji@gmail.com

E-mail: a.a.thirthar@gmail.com

ABSTRACT. In this paper an *SIS* epidemic model with saturated incidence rate and treatment function is proposed and studied. The existence of all feasible equilibrium points is discussed. The local stability conditions of the disease free equilibrium point and endemic equilibrium point are established with the help of basic reproduction number. However the global stability conditions of these equilibrium points are established using Lyapunov method. The local bifurcation near the disease free equilibrium point is investigated. Hopf bifurcation condition, which may occurs around the endemic equilibrium point is obtained. The conditions of backward bifurcation and forward bifurcation near the disease free equilibrium point are also determined. Finally, numerical simulations are given to investigate the global dynamics of the system and confirm the obtained analytical results.

Keywords: Epidemic models, Local stability, Backward bifurcation, Hopf bifurcation.

2010 Mathematics Subject Classification: 37N25, 74H60.

*Corresponding Author

1. INTRODUCTION

The mathematical models, which describe the dynamics of human infectious diseases, have played an important role in the field of epidemiology. Researchers have proposed many epidemic models to grasp the mechanism of disease transmission, see [1-7]. In the mathematical modeling of epidemic transmission, there are several factors that substantially affect the dynamical behavior of the models. Incidence rate functions are seen as a major factor in producing the rich dynamics of epidemic models. There are many types of incidence rate functions, such as nonlinear incidence rate, standard incidence rate, and saturated incidence rate. Later on, there are many studies that have demonstrated the effect of nonlinear incidence rate, which is one of the key factors that induce periodic oscillations, on the epidemic models, see [8-13]. On the other hand, it is observed that in the classical epidemic models, the treatment function is an important factor to decrease the spread of the epidemiological diseases. Indeed most of the treatment functions of the infected individuals are considered to be proportional to the number of the infected individuals. However, Wang and Ruan [14] introduced a constant treatment function, while a piecewise linear treatment function was considered in [15]. Moreover, Eckalbar and Eckalbar [16] constructed an SIR epidemic model with a quadratic treatment function. Lately, saturated treatment function has been widely used in many epidemic models. In particular, Zhang and Liu [17] took a continuous and differentiable saturated treatment function, which is an extension to that used in [15].

It is well known that, the basic reproduction number (R_0) plays an important role in determine the stability of disease free equilibrium point and hence control the disease transmission. In fact, it is scientifically known that, a classical necessary condition for disease eradication is that the basic reproductive number satisfies that $R_0 < 1$ see [18]. However, the existence of some types of treatment functions in an epidemic models may cause the occurrence of some types of bifurcations, especially backward bifurcation [15]. The occurrence of backward bifurcation means that an endemic equilibrium point may also exists even $R_0 < 1$, which in turn leads to occurrence of bi-stable in the system and hence losing the control of disease when $R_0 < 1$. Many epidemic models were interested in study the existence of backward bifurcation may be found in the literature, for both generic and specific diseases, see [17 – 20]. The detection of the occurrence of backward bifurcation is done through the center manifold theory as its basis [21 – 22].

In this paper, an SIS epidemic model involving saturated incidence rate and treatment function is proposed and studied. This paper is organized as follows: in section (2) the hypotheses, which adopted to formulate the model is

presented. Section (3) deals with the stability analysis of the model. However section (4) studied the local bifurcation around disease free equilibrium point and Hopf bifurcation around the endemic equilibrium point. Moreover section (5) includes the backward and forward bifurcation analyses. Section (6) introduced a numerical solution of system (2.1) for a hypothetical set of parameters and discussed the effects of varying these parameters on the dynamical behavior. Finally section (7) included the main conclusion and discussion.

2. MODEL FORMULATION

An epidemic model with SIS type of disease is proposed and studied. Accordingly the total population is divided into two compartments namely $S(t)$ and $I(t)$, where $S(t)$ and $I(t)$ represent the number of susceptible and infected individuals at time t , respectively. In order to formulate the model mathematically the following hypotheses are adopted:

- (1) There is no vertical infection, that is mean all the newborn individuals are susceptible and exist within the recruitment rate of the population that given by $A > 0$.
- (2) The disease is transmitted by contact between the susceptible and infected individuals with probability rate $\lambda \in (0, 1)$, according to the saturated incidence rate $\frac{cSI}{(k+I)}$ that given in [23]. Here $c > 0$ represents the ratio of the contact rate to the saturation factor of the inhibitory effect, while $\frac{1}{k} > 0$ is stand for the saturation factor that measure the inhibitory effect of the disease due to the crowding of infected individuals.
- (3) The disease is disappear and the infected individuals become susceptible again with two strategies. The first is the natural recovery with a recovery rate $\epsilon > 0$ of the infected individuals and the second is due to treatment with treatment function $\frac{aI}{(b+I)}$, in which $a > 0$ represents the ratio of the maximum medical resource supplied per unit time to the saturation factor of the delayed in treatment, while $\frac{1}{b} > 0$ is stand for the saturation factor that measure the effect of the delay in treatment for the infected individuals.
- (4) The individuals in both compartments decay due to the natural death rate $\alpha > 0$. Moreover the existence of disease may cause death with the rate $\mu > 0$.

According to the above hypotheses the dynamics of SIS epidemic model with saturated incidence rate and treatment function can be represented by the following set of nonlinear differential equations:

$$\begin{cases} \dot{S} &= A - \alpha S - \lambda \frac{cSI}{(k+I)} + \epsilon I + \frac{aI}{(b+I)} \\ \dot{I} &= \lambda c \frac{SI}{(k+I)} - (\alpha + \epsilon + \mu)I - \frac{aI}{(b+I)} \end{cases} \quad (2.1)$$

with the initial condition given by $S(0) > 0$ and $I(0) \geq 0$. Further in the following theorem the feasible region of model (1) is established.

Theorem 2.1. The region $\Omega = \{(S, I) : S > 0, I \geq 0, 0 < S + I \leq \frac{A}{\alpha}\}$ is positively invariant for model (2.1).

Proof. Let $\beta = \lambda c \frac{I}{(k+I)}$, then from the first equation of (2.1) it is observed:

$$\dot{S} = A - \alpha S - \beta S + \epsilon \geq I + \frac{aI}{(b+I)} \geq -(\alpha + \beta)S$$

Hence we get that $S(t) \geq S(0)e^{-(\alpha+\beta)t} > 0$. Similarly the positivity of $I(t) \geq 0$, with equality occurs only when $I(0) = 0$, can be proved easily using the second equation of (2.1).

Now since $N(t) = S(t) + I(t)$, then direct computation gives $\frac{dN}{dt} < A - \alpha N$. Consequently, for $N(0) = N_0$, we get that $N(t) \leq \frac{A}{\alpha} - [\frac{1}{\alpha}(A - \alpha N_0)]e^{-\alpha t}$. Obviously as $t \rightarrow \infty$ then for any initial value in Ω , the population size N satisfy hat $0 < N < \frac{A}{\alpha}$. Thus all the solutions sets of (2.1) enter the region Ω and remain in it for all $t > 0$. Hence the region Ω is positively invariant. \square

3. STABILITY ANALYSIS OF MODEL (2.1)

In this section, all feasible equilibrium points along with the reproduction number are determined and then the local stability of each of them is discussed. Clearly the so called disease free equilibrium point that given by $E_0 = (\frac{A}{\alpha}, 0)$ always exists for system (2.1). On the other hand the basic reproduction number R_0 , which is representing the mean number of secondary infections caused by a single infective introduced into a susceptible population, can be determined using the next generation matrix, see [18], for system (2.1) at E_0 . It is well known that R_0 is a threshold value that governing the qualitative dynamics of system (2.1).

Let $x = (I, S)^T$ then the system (2.1) can be written as

$$\frac{dx}{dt} = \mathcal{F}(x) - \mathcal{V}(x)$$

where

$$\mathcal{F}(x) = \begin{pmatrix} \lambda c \frac{SI}{(k+I)} \\ 0 \end{pmatrix} \text{ and } \mathcal{V}(x) = \begin{pmatrix} (\alpha + \epsilon + \mu + \frac{a}{b+I})I \\ \alpha S + \lambda c \frac{SI}{k+I} - \epsilon I - \frac{aI}{b+I} - A \end{pmatrix}$$

Hence after some calculations and using E_0 , we obtain

$$F = (\lambda c \frac{A}{\alpha k}), \text{ and } V = (\alpha + \epsilon + \mu + \frac{a}{b})$$

Therefore, the reproduction number of system (2.1) is the spectral radius of the next generation matrix that given by FV^{-1} , which can be written as

$$R_0 = \rho(FV^{-1}) = \lambda c \frac{A}{\alpha k(\alpha + \epsilon + \mu + \frac{a}{b})} \quad (3.1)$$

Further the endemic equilibrium point of system (2.1) can be computed by solving the following algebraic system:

$$\begin{aligned} A - \alpha s - \lambda c \frac{SI}{k+I} + \epsilon I + \frac{aI}{b+I} &= 0 \\ \lambda c \frac{SI}{k+I} - (\alpha + \epsilon + \mu)I - \frac{aI}{b+I} &= 0 \end{aligned}$$

From the second equation we obtain that:

$$S(I) = \frac{[(\alpha + \epsilon + \mu)(b+I) + a](k+I)}{\lambda c(b+I)} \quad (3.2)$$

Substituting $S(I)$ in the first equation gives the following polynomials:-

$$a_0 I^2 + a_1 I + a_2 = 0 \quad (3.3)$$

where

$$\begin{aligned} a_0 &= -\alpha^2 - \alpha\epsilon - \alpha\mu - \alpha\lambda c - \mu\lambda c < 0 \\ a_1 &= A\lambda c - (\alpha + \epsilon + \mu)(\alpha b + \alpha k + \lambda cb) - \alpha a + \epsilon\lambda c \\ a_2 &= \alpha k b(\alpha + \epsilon + \mu + \frac{a}{b})(R_0 - 1) \end{aligned}$$

Now solving eq. (3.3) for I and substituting their roots in eq. (3.2) gives the endemic point or points. Clearly the number of positive roots of eq. (3.3) depends on the values of R_0 , a_1 and $\Delta = a_1^2 - 4a_0a_2$, and can be summarized in the following theorem. Recall that

$$\Delta = a_1^2 - 4a_0a_2 = 0 \Leftrightarrow R_0 = 1 + \frac{(a_1^2)}{(4a_0(\alpha k b(\alpha + \epsilon + \mu + \frac{a}{b})))} (\equiv R_0^c) \quad (3.4)$$

Moreover $\Delta < 0 \Leftrightarrow R_0 < R_0^c$; $\Delta > 0 \Leftrightarrow R_0 > R_0^c$.

Theorem 3.1. System (2.1) has

- (1) *Unique endemic equilibrium point whenever $R_0 > 1$.*
- (2) *Unique endemic equilibrium point when $R_0 = 1$ and $a_1 > 0$.*
- (3) *An endemic equilibrium point of multiplicity two when $R_0 = R_0^c$, $a_1 > 0$.*
- (4) *Two endemic equilibrium points, when $R_0^c < R_0 < 1$ and $a_1 > 0$.*
- (5) *No endemic equilibrium point whenever $R_0 < R_0^c$ and $a_1 < 0$ or $R_0 < 1$ and $a_1 < 0$.*

Proof. Straightforward using Descartes rule of sign and above discussion. \square

Note that Theorem (3.1) tell us that $R_0 = 1$ is a critical value at which the number of equilibrium point of system (2.1) changed. In fact $R_0 = 1$ represents a bifurcation value for the stability of the disease free equilibrium point as shown in the following theorem.

Theorem 3.2. The disease free equilibrium point is locally asymptotically stable point of system (2.1) for $R_0 < 1$ and unstable for $R_0 > 1$

Proof. The Jacobian matrix of system (2.1) at E_0 is given by:

$$J(E_0) = \begin{bmatrix} -\alpha & \frac{-\lambda c A}{\alpha k + \epsilon + \frac{a}{b}} \\ 0 & \frac{\lambda c A}{\alpha k} - (\alpha + \epsilon + \mu + \frac{a}{b}) \end{bmatrix} \quad (3.5)$$

Thus the eigenvalues of $J(E_0)$ are given by:

$$\gamma_1 = -\alpha \quad \text{and} \quad \gamma_2 = (\alpha + \epsilon + \mu + \frac{a}{b})(R_0 - 1)$$

Therefore all the eigenvalues are negative and hence E_0 is locally asymptotically stable provided that $R_0 < 1$, while its unstable saddle point for $R_0 > 1$ and the proof is complete. \square

Now the stability of the endemic equilibrium point is discussed in the following two theorems according to their existence conditions. Further, it is easy to verify that the Jacobian matrix at the endemic equilibrium point, say E , is given by

$$J(E) = \begin{pmatrix} -\alpha - \lambda c \frac{I^*}{k + I^*} & -\frac{k[(\alpha + \epsilon + \mu)(b + I^*) + a]}{(k + I^*)(b + I^*)} + \epsilon + \frac{ab}{(b + I^*)^2} \\ \lambda c \frac{I^*}{k + I^*} & \frac{k[(\alpha + \epsilon + \mu)(b + I^*) + a]}{(k + I^*)(b + I^*)} + (\alpha + \epsilon + \mu) - \frac{ab}{(b + I^*)^2} \end{pmatrix} \quad (3.6)$$

Theorem 3.3. Suppose there are two endemic equilibrium points, say E_i , $i = 1, 2$. Then E_i is locally asymptotically stable if the following conditions hold

$$b \geq k \quad (3.7a)$$

$$\frac{a}{b} \geq \frac{\alpha k}{\lambda c A} \quad (3.7b)$$

Proof. From theorem (3.1) we have that

$$R_0^c \leq R_0 < 1 \quad \text{and} \quad a_1 > 0$$

Hence straightforward computation gives that

$$Tr(J(E_i)) = -(2\alpha + \lambda c + \epsilon + \mu) \frac{I^*}{k + I^*} - \frac{a}{b + I^*} \left[\frac{b}{b + I^*} - \frac{k}{k + I^*} \right]$$

And

$$\begin{aligned} Det(J(E_i)) = & -\frac{(\alpha^2 k^2)}{(k + I^*)\lambda c A}(R_0 - 1) + \frac{\alpha k}{k + I^*} \left[\frac{a}{b} - \frac{\alpha k}{\lambda c A} \right] \\ & + \frac{\alpha a}{b + I^*} \left[\frac{b}{b + I^*} - \frac{k}{(k + I^*)} \right] + \alpha(\alpha + \epsilon + \mu) \\ & + \lambda c \frac{I^*}{(k + I^*)} [\alpha + \mu] \end{aligned}$$

Consequently, if the above sufficient conditions (3.7a) – (3.7b) hold we obtain $Tr(J(E_i)) < 0$ and $Det(J(E_i)) > 0$ Hence $J(E_i)$ has two eigenvalues with negative real parts, Therefore E_i is locally asymptotically stable and the proof is complete. \square

Theorem 3.4. Suppose that $R_0 > 1$, then the unique endemic equilibrium point E^* is locally asymptotically stable if the following condition holds:

$$\frac{k[(\alpha + \epsilon + \mu)(b + I^*) + a]}{(k + I^*)(b + I^*)} < (\alpha + \epsilon + \mu) + \frac{ab}{(b + I^*)^2} \quad (3.8)$$

Proof. From eq.(3.5) it is easy to verify that the trace and determinant of the Jacobian matrix at the endemic point can be rewritten as

$$\begin{aligned} Tr.(J(E^*)) = & -\alpha - \lambda c \frac{I^*}{(k + I^*)} + \frac{(k[(\alpha + \epsilon + \mu)(b + I^*) + a])}{(k + I^*)(b + I^*)} - (\alpha + \epsilon + \mu) \\ & - \frac{ab}{(b + I^*)^2} \\ Det.(J(E^*)) = & -\frac{(\alpha k[(\alpha + \epsilon + \mu)(b + I^*) + a])}{(k + I^*)(b + I^*)} + \alpha(\alpha + \epsilon + \mu) + \frac{\alpha ab}{(b + I^*)^2} \\ & + \frac{(\lambda c I^*)}{(k + I^*)} (\alpha + \mu) \end{aligned}$$

Consequently, condition (3.7) guarantee s that $Tr.(J(E^*)) < 0$ and $(J(E^*)) > 0$ Hence $J(E^*)$ has two eigenvalues with negative real parts. Therefore E^* is locally asymptotically stable and the proof is complete. Now the global stability of system (2.1) is investigated as shown in the following theorems. Recall that according to theorem (2.1) the region $\Omega = \{(S, I) : S > 0, I \geq 0, 0 < S + I \leq \frac{A}{\alpha}\}$ is positively invariant for system (2.1). Hence we obtain that $S(t) \leq \frac{A}{\alpha}$ and $I(t) \leq \frac{A}{\alpha}$. \square

Theorem 3.5. Assume that the disease free equilibrium point E_0 is locally asymptotically stable and let

$$\bar{R}_0 = \frac{\lambda c A}{\alpha k \left(\alpha + \epsilon + \mu + \frac{a}{(b + \frac{A}{\alpha})} \right)} < 1 \quad (3.9)$$

Then E_0 is a globally asymptotically stable.

Proof. Clearly if $\bar{R}_0 < 1$ then $R_0 < 1$. Consider the following Lyapunov function $V_1(S, I) = I$, then $V_1(S, I) = 0$ if and only if $I = 0$ and $V_1(S, I) > 0$ otherwise. Also

$$\dot{V}_1 = \left[\lambda c \frac{S}{(k+I)} - (\alpha + \epsilon + \mu) - \frac{a}{(b+I)} \right] I \leq \left[\lambda c \frac{S}{k} - (\alpha + \epsilon + \mu + \frac{a}{(b+I)}) \right] I$$

Since $S(t) \leq \frac{A}{\alpha}$ and $I(t) \leq \frac{A}{\alpha}$. Then $\dot{V}_1 \leq [\lambda c \frac{A}{\alpha} - (\alpha + \epsilon + \mu + \frac{a}{(b+\frac{A}{\alpha})})] I$

Clearly $\bar{R}_0 < 1$ guarantee s that $\dot{V}_1 = 0$ if and only if $I = 0$ and $\dot{V}_1 < 0$ otherwise. Hence E_0 is stable point. Since the largest compact invariant set in Ω with $\dot{V}_1 = 0$ is the singleton set E_0 . Thus by La Salle's Invariance Principle every solution initiate in the region Ω approaches to E_0 . Therefore $\{E_0\}$ is a globally asymptotically stable point. \square

Theorem 3.6. Assume that the unique endemic equilibrium point is locally asymptotically stable and let the following condition holds

$$(q_{12})^2 < 4q_{11}q_{22} \quad (3.10)$$

where q_{ij} are given in the proof, then the endemic equilibrium point is a globally asymptotically

Proof. Consider the function $V_2 = \frac{(S-S^*)^2}{2} + \frac{(I-I^*)^2}{2}$ Clearly, $V_2 : R^2 \rightarrow R$ with $V_2(S^*, I^*) = 0$ and $V_2(S, I) > 0; \forall (S, I) \in \Omega$. Then straightforward computation show that:

$$\begin{aligned} V_2' = & - \left[\alpha + \frac{\lambda c I}{(k+I)} \right] (S - S^*)^2 \\ & + \left[\epsilon + \frac{ab}{(b+I)(b+I^*)} + \frac{\lambda c I}{(k+I)} - \frac{(\lambda c k S^*)}{(k+I)(k+I^*)} \right] (S - S^*)(I - I^*) \\ & - \left[(\alpha + \epsilon + \mu) + \frac{ab}{(b+I)(b+I^*)} - \frac{(\lambda c k S^*)}{(k+I)(k+I^*)} \right] (I - I^*)^2 \end{aligned}$$

which gives

$$V_2' = -q_{11}(S - S^*)^2 + q_{12}(S - S^*)(I - I^*) - q_{22}(I - I^*)^2$$

here:

$$\begin{aligned} q_{11} &= \left[\alpha + \frac{\lambda c I}{(k+I)} \right] \\ q_{12} &= \left[\epsilon + \frac{ab}{(b+I)(b+I^*)} + \frac{\lambda c I}{(k+I)} - \frac{\lambda c k S^*}{(k+I)(k+I^*)} \right] \\ q_{22} &= \left[(\alpha + \epsilon + \mu) + \frac{ab}{(b+I)(b+I^*)} - \frac{(\lambda c k S^*)}{(k+I)(k+I^*)} \right] \end{aligned}$$

Now its easy to verify that condition (3.8) guarantee s that $q_2 > 0$, while condition (3.10) gives:

$$V_2' < -[\sqrt{q_{11}}(S - S^*) - \sqrt{q_{22}}(I - I^*)]^2$$

Hence $V_2' = 0$ if and only if $(S, I) = (S^*, I^*)$ and $V_2' < 0$ otherwise thus the endemic equilibrium is a globally asymptotically stable. \square

4. LOCAL BIFURCATION ANALYSIS

In this section, the local bifurcation analysis of system (2.1) is investigated as shown in the following theorems. Since the Jacobian matrix of system (2.1) at (S, I) can be rewritten as

$$Df = \begin{pmatrix} -\alpha - \lambda c \frac{I}{k+I} & -\lambda c S \frac{k}{(k+I)^2} + \epsilon + \frac{ab}{(b+I)^2} \\ \lambda c \frac{I}{k+I} & \lambda c S \frac{k}{(k+I)^2} - (\alpha + \epsilon + \mu) - \frac{ab}{(b+I)^2} \end{pmatrix} \quad (4.1a)$$

here $f = (f_1, f_2)^T$ with f_1 and f_2 are the interaction function in system (2.1), Then for any vector $U = (u_1, u_2)^T$, it is easy to verify that:

$$D^2 f.(U, U) = \begin{pmatrix} -2\lambda c \frac{k}{(k+I)^2} u_1 u_2 + 2\lambda c \frac{k}{(k+I)^3} u_2^2 - \frac{ab}{(b+I)^3} u_2^2 \\ 2\lambda c \frac{k}{(k+I)^2} u_1 u_2 - 2\lambda c \frac{k}{(k+I)^3} u_2^2 + \frac{ab}{(b+I)^3} u_2^2 \end{pmatrix} \quad (4.1b)$$

$$D^3 f.(U, U, U) = \begin{pmatrix} 4\lambda c \frac{k}{(k+I)^3} u_1 u_2^2 - 6\lambda c \frac{kS}{(k+I)^4} u_2^3 - \frac{6ab}{(b+I)^4} u_2^3 \\ -4\lambda c \frac{k}{(k+I)^3} u_1 u_2^2 + 6\lambda c \frac{kS}{(k+I)^4} u_2^3 + \frac{6ab}{(b+I)^4} u_2^3 \end{pmatrix} \quad (4.1c)$$

Moreover, it is well known that the necessary but not sufficient condition for bifurcation to occur is a non-hyperbolic property of the equilibrium point.

Theorem 4.1. The system (2.1) undergoes a transcritical bifurcation near the disease free equilibrium point, but saddle node bifurcations can not occur as the parameter λ passes through the bifurcation value

$$\lambda^* = \frac{\alpha k}{cA} (\alpha + \epsilon + \mu + \frac{a}{b})$$

provided that the following condition holds

$$(\alpha + \epsilon + \mu + \frac{a}{b}) (\frac{\alpha + \mu}{\alpha} + \frac{1}{k}) \neq \frac{a}{b^2} \quad (4.2a)$$

Otherwise, the system (2.1) undergoes a pitchfork bifurcation if the following condition holds

$$\frac{2}{Ak^2} (\alpha + \epsilon + \mu + \frac{a}{b}) [(\alpha + \mu) - \frac{(\alpha + \mu)^2}{\alpha} + 3A] \neq \frac{3a}{b^3} \quad (4.2b)$$

Proof. According to the Jacobian matrix of system (2.1) at E_0 that given in (3.5), it is clear that as the parameter λ passes through the value of λ^* then $R_0 = 1$ and the Jacobian matrix becomes:

$$Df(E_0, \lambda^*) = \begin{pmatrix} -\lambda & -(\alpha + \mu) \\ 0 & 0 \end{pmatrix}$$

Therefore the eigenvalues of $Df(E_0, \lambda^*)$ are $\lambda_1 = -\alpha, \lambda_2 = 0$ and hence E_0 becomes a non-hyperbolic point when $R_0 = 1$. Assume that $U = (u_1, u_2)^T$ is the eigenvector associated with the zero eigenvalue in $Df(E_0, \lambda^*)$ then we obtain that $u_1 = -\frac{\alpha + \mu}{\alpha}$ and $u_2 = 1$. Assume that $W = (w_1, w_2)^T$ be the eigenvector associated with the zero eigenvalue for $[Df(E_0, \lambda^*)]^T$, then we obtain $w_1 = 0$ and $w_2 = 1$. Now since

$$\frac{\partial f}{\partial \lambda} = f_\lambda = \begin{pmatrix} \frac{\partial f_1}{\partial \lambda} \\ \frac{\partial f_2}{\partial \lambda} \end{pmatrix} = \begin{pmatrix} \frac{-cSI}{k+I} \\ \frac{cSI}{k+I} \end{pmatrix}$$

Then

$$\begin{pmatrix} \frac{\partial f_1}{\partial \lambda} \\ \frac{\partial f_2}{\partial \lambda} \end{pmatrix} \big|_{(E_0, \lambda^*)} = \begin{pmatrix} 0 \\ 0 \end{pmatrix}$$

Therefore

$$W^T \cdot \frac{\partial f}{\partial \lambda} (E_0, \lambda^*) = zero$$

Further

$$Df_\lambda = \begin{pmatrix} -\frac{cI}{k+I} & -\frac{ckS}{(k+I)^2} \\ \frac{cI}{k+I} & \frac{ckS}{(k+I)^2} \end{pmatrix}$$

Then

$$Df_\lambda (E_0, \lambda^*) = \begin{pmatrix} 0 & -\frac{cA}{\alpha k} \\ 0 & \frac{cA}{\alpha k} \end{pmatrix}$$

Therefore $W^T [Df_\lambda (E_0, \lambda^*) U] = \frac{cA}{\alpha k} \neq 0$, hence saddle node bifurcation cannot occur.

Now by using (4.1b) with the eigenvectors U and W at the (E_0, λ^*) we obtain

that

$$W^T[D^2f(E_0, \lambda^*)(U, U)] = -2(\alpha + \varepsilon + \mu + \frac{a}{b})(\frac{\alpha + \mu}{\alpha} + \frac{1}{k}) + \frac{2a}{b^2}$$

Clearly condition (4.2a) guarantees that $W^T[D^2f(E_0, \lambda^*)(U, U)] \neq 0$ and hence transcritical bifurcation occurs. However, if the condition (4.2a) is violate while condition (4.2b) holds then by using (4.1c) with the eigenvectors U and W , it s obtained that

$$\begin{aligned} W^T[D^3f(E_0, \lambda^*)(U, U, U)] &= \frac{2}{(Ak^2)}(\alpha + \varepsilon + \mu + \frac{a}{b})[(\alpha + \mu) - \frac{(\alpha + \mu)^2}{\alpha} + 3A] \\ &\quad - 6\frac{a}{b^3} \neq 0 \end{aligned}$$

Hence pitchfork bifurcation occurs and the proof is complete. \square

Recall that, in order to system (2.1) undergoes a Hopf bifurcation around the endemic equilibrium point, the Jacobian matrix given by (3.6) should have complex conjugate eigenvalues $\beta_1(r)i\beta_2(r)$ such that $\beta_1(r^*) = 0$ and $\frac{d\beta_1}{dr}|_{(r=r^*)} \neq 0$ where r is a general parameter, see [24]. This is equivalent to that the Jacobin matrix given by (3.6) have $Tr.(r^*) = 0$, $Det.(r) > 0$ for all values of r and $\frac{(Tr.(.))}{dr}|_{(r=r^*)} \neq 0$, see [25].

Theorem 4.2. :*Suppose that $R_0 > 1$ with*

$$\begin{aligned} \alpha(2\alpha + \epsilon + \mu) + \frac{\alpha ab}{(b + I^*)^2} &< \frac{\alpha k[(\alpha + \epsilon + \mu)(b + I^*) + a]}{(k + I^*)(b + I^*)} \\ &< \alpha(\alpha + \epsilon + \mu) + \frac{\alpha ab}{(b + I^*)^2} + \frac{(\lambda c I^*)}{(k + I^*)}(\alpha + \mu). \end{aligned} \quad (4.3)$$

Then system (2.1) undergoes a Hopf bifurcation around the unique endemic point when the parameter λ passes through the parameter

$$\lambda^* = \frac{k[(\alpha + \epsilon + \mu)(b + I^*) + a](b + I^*) - [(2\alpha + \epsilon + \mu)(b + I^*)^2 + ab](k + I^*)}{cI^*(b + I^*)^2} \quad (4.4)$$

Proof. Recall that, it is easy to verify that under the condition (4.3) the determinant of the Jacobian matrix at the endemic point that given in theorem (3.4) satisfy the following: $Det.(J) > 0$ while the trace can be rewritten as

$$\begin{aligned} Tr.(J) &= \frac{1}{(k + I^*)(b + I^*)^2} \left(k[(\alpha + \epsilon + \mu)(b + I^*) + a](b + I^*) \right. \\ &\quad \left. - [(2\alpha + \epsilon + \mu)(b + I^*)^2 + ab](k + I^*) - \lambda c I \lambda^*(b + I^*)^2 \right). \end{aligned}$$

Consequently, for $\lambda = \lambda^*$ we have $Tr(J, \lambda^*) = 0$. However

$$\frac{(dTr(J))}{(d\lambda^*)}|_{(\lambda = \lambda^*)} = -\frac{(cI^*)}{(k + I^*)} \neq 0$$

Therefore all the necessary and sufficient conditions for occurrence of Hopf bifurcation are satisfied and hence the proof is complete. \square

5. BACKWARD AND FORWARD BIFURCATION

It is well known that the condition $R_0 < 1$ is a necessary and sufficient condition to eliminate the disease when the bifurcation is forward, while it is not enough when the bifurcation is backward. In fact, the backward bifurcation scenario involves the existence of a subcritical transcritical bifurcation at $R_0 = 1$ and of a saddle-node bifurcation at $R_0 < 1$, see [19]. Consequently, in the following we detect the type of the bifurcation in system (2.1) whether it is backward or forward by applying the theorem for backward and forward bifurcation that obtained in [26] and is based on the center manifold theory. Consider the dynamical system with a parameter ϕ :

$$\frac{dx}{dt} = f(x, \phi) \quad (5.1)$$

where $f : R^n \times R \rightarrow R$ and $f \in C^2(R^n \times R)$. Assume (without loss of generality) that the origin is an equilibrium point of system (5.1) for all values of ϕ . Moreover, suppose that the Jacobian matrix of system (5.1) at $(0, \phi) = (0, 0)$ has a simple zero eigenvalue with other eigenvalues have negative real parts. Let W be a nonnegative right eigenvector while V is a nonnegative left eigenvector correspond the zero eigenvalue and let the coefficients of the normal form that representing the system dynamics on the central manifold $\dot{C} = C(\hat{b}\phi + \frac{\hat{a}}{2}C)$, which defined in [26] as

$$\hat{a} = \sum_{(k,i,j=1)}^n v_k w_i w_j \frac{(\partial^2 f_k)}{(\partial x_i \partial x_j)}(0, 0); \hat{b} = \sum_{(k,i,j=1)}^n v_k w_i \frac{(\partial^2 f_k)}{(\partial x_i \partial \phi)}(0, 0) \quad (5.2)$$

where f_k, w_k and v_k represent the k^{th} component of f, W and V respectively. Then according to [26], if $\hat{a} < 0$ and $\hat{b} > 0$ system (5.1) has a forward bifurcation, while it has a backward bifurcation if a $\hat{a} > 0$ and $\hat{b} > 0$.

Now for system (2.1) the following theorem specify the type of bifurcation whether its backward or forward, which may occurs near the disease free point, and their sufficient conditions.

Theorem 5.1. Assume that $R_0 = 1$, then system (2.1) exhibits a backward bifurcation near the disease free equilibrium point if the following condition

holds.

$$\frac{\lambda^* c}{\alpha k} \left(\alpha + \mu + \frac{A}{ck} \right) < \frac{a}{b^2} \quad (5.3)$$

On the other hand if

$$\frac{\lambda^* c}{\alpha k} \left(\alpha + \mu + \frac{A}{ck} \right) > \frac{a}{b^2} \quad (5.4)$$

here λ^* is given in the proof . Then system (2.1) has a forward bifurcation.

Proof. According to Theorem 4.1 , $R_0 = 1$ is equivalent to $\lambda^* = \frac{\alpha k}{cA} (\alpha + \varepsilon + \mu + \frac{a}{b})$. Moreover the Jacobian matrix $Df(E_0, \lambda^*)$ has the following eigenvalues $\lambda_1 = -\alpha, \lambda_2 = 0$.

Let $W = (w_1, w_2)^T$ be the right eigenvector associated with the zero eigenvalue then we obtain that $W = [-\frac{\alpha+\mu}{\alpha}, 1]^T$. However the left eigenvector $V = (v_1, v_2)$, which satisfy that $vw = 1$, is determined as $V = [0, 1]$. Further, since

$$\begin{aligned} \frac{\partial^2 f_1}{\partial S \partial I} &= \frac{\partial^2 f_1}{\partial I \partial S} = \frac{-\lambda c}{k}; & \frac{\partial^2 f_1}{\partial I^2} &= 2 \frac{\lambda c A}{k^2 \alpha} - 2 \frac{a}{b^2} \\ \frac{\partial^2 f_1}{\partial I \partial \lambda} &= \frac{\partial^2 f_1}{\partial \lambda \partial I} = -\frac{cA}{k\alpha}; & \frac{(\partial^2 f_2)}{\partial I^2} &= -2 \frac{\lambda c A}{(k^2 \alpha)} + 2 \frac{a}{b^2} \\ \frac{\partial^2 f_2}{\partial S \partial I} &= \frac{\partial^2 f_2}{\partial I \partial S} = \frac{\lambda c}{k}; & \frac{\partial^2 f_2}{\partial I \partial \lambda} &= \frac{\partial^2 f_2}{\partial \lambda \partial I} = \frac{cA}{k\alpha} \end{aligned}$$

While all the other second order partial derivatives are zero. Thus by substituting these derivatives in (5.2) and evaluated at (E_0, λ^*) we get:

$$\begin{aligned} \hat{a} &= -2 \frac{\lambda^* c}{k\alpha} \left(\alpha + \mu + \frac{A}{k} \right) + 2 \frac{a}{b^2} \\ \hat{b} &= \frac{cA}{k\alpha} > 0 \end{aligned}$$

Since the value of \hat{b} is always positive, therefore the value of \hat{a} will determine the type of the bifurcation whether forward or backward. Thus under condition (5.3) system (2.1) exhibits a backward bifurcation, while system (2.1) exhibits a forward bifurcation under condition (5.4). \square

6. NUMERICAL SIMULATION

In this section we aim to verify our obtained theoretical results and specify the control set of parameters. Accordingly, system (2.1) is solved numerically for the following set of hypothetically feasible set of parameters using six-order Runge-Kuttamethod along with predictor-corrector method and then draw the resulting trajectories with the help of Matlab.

$$A = 20, \alpha = 0.1, \lambda = 0.5, \epsilon = 0.1, \mu = 0.1, a = 0.08, b = 10, k = 10, c = 0.9 \quad (6.1)$$

Obviously, for the data (6.1), system (2.1) approaches asymptotically to the endemic equilibrium point $E_1 = (55.13, 72.43)$ as shown in Figure (1). However for the data (6.1) with $\lambda = 0.001$, its observed that system (2.1) approaches asymptotically to the disease free equilibrium point $E_0 = (200, 0)$ as shown in Figure (2). Straightforward computation shows that for the data used in Figure (1) and Figure (2) the reproduction number is determine as $R_0 = 29.22 > 1$ and $R_0 = 0.0584 < 1$ respectively, which confirm our obtained analytical results regarding to stability conditions and the existence of bifurcation.

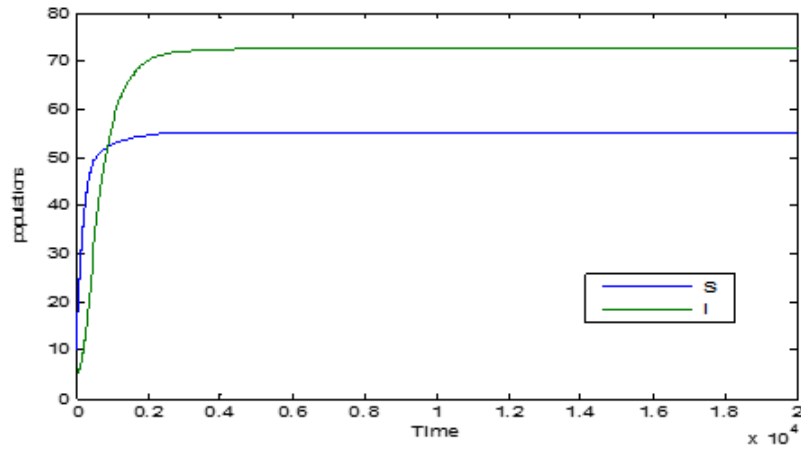


FIGURE 1. Trajectory of system (2.1) as a function of time for data (6.1), which approaches asymptotically to endemic equilibrium point.

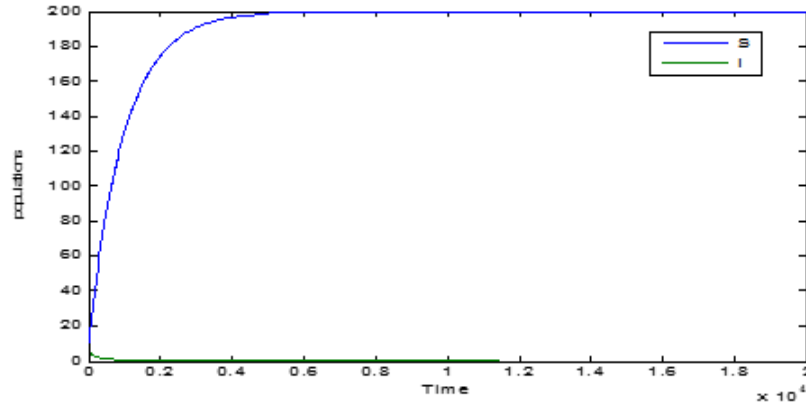


FIGURE 2. Trajectory of system (2.1) as a function of time for data (6.1), which approaches asymptotically to endemic equilibrium point.

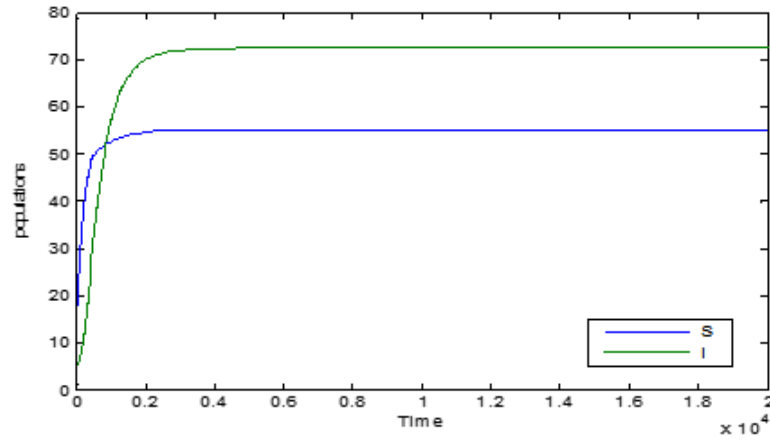


FIGURE 3. Trajectory of system (2.1) as a function of time for data (6.1) with $b = 0.0001$, which approaches asymptotically to the endemic point $E_1 = (55.15, 72.42)$.

Further analysis to the effect of other parameters on the dynamic of system (2.1) have been done and the results are summarized in the form of table below.

<i>Parameter</i>	R_0	<i>behavior</i>
$c > 0.0308$	$R_0 > 1$	E_1 is asymptotically stable
$c < 0.0308$	$R_0 < 1$	E_0 is asymptotically stable
$A > 0.6844$	$R_0 > 1$	E_1 is asymptotically stable
$A < 0.6844$	$R_0 < 1$	E_0 is asymptotically stable
$K > 292.207$	$R_0 < 1$	E_0 is asymptotically stable
$K < 292.20$	$R_0 > 1$	E_1 is asymptotically stable
ϵ	$R_0 > 1$	E_1 is asymptotically stable
μ	$R_0 > 1$	E_1 is asymptotically stable
a	$R_0 > 1$	E_1 is asymptotically stable

TABLE 1. Dynamical behavior of system (2.1) for the data (6.1) with varying a specific parameter

7. DISCUSSION

In this research paper, an epidemiological model of SIS type of disease is proposed and studied. It is assumed that the disease is transmitted according to the saturated incidence rate while the infected individuals recovered according to saturated treatment function. Local and global stability of the proposed system have been done using basic reproduction number and suitable Lyapunov functions. The sufficient conditions for occurrence of different types of bifurcations have been established. It is observed that, system (2.1) has always a disease free equilibrium point, while the existence and the number of the endemic equilibrium points depend on the value of basic reproduction number. On contrast to classical epidemic models, the system is rich in his dynamics due to existence of nonlinear term represented by the saturated treatment function. In fact the system approaches to an endemic equilibrium point under some conditions even when $R_0 < 1$. This is indicate to occurrence of backward bifurcation. Also the system undergoes other types of bifurcations including transcritical, pitchfork and Hopf, under certain conditions. Finally numerical simulation of system (2.1) is carried out using feasible hypothetical set of data to confirm our analytical results and specify the control set of parameters. It s observed that, the system undergoes different types of bifurcation when some parameters are varying, which confirm our obtained analytical results. Moreover, although the system don t have periodic dynamics for the data given by

(6.1), system (2.1) still has a possibility to have it for other set of data due to existence of Hopf bifurcation analytically.

REFERENCES

1. . R.M. Anderson, Population Biology of Infectious Diseases, *Springer-Verlag*. (1982) .
2. O. Diekmann, J.A.P. Heesterbeek, Mathematical Epidemiology of Infectious Diseases. Model Building, Analysis and Interpretation, *Chichester* , *John Wiley and Sons* (1971) .
3. M. Lizana, J. Rivero, Multiparametric bifurcations for a model in epidemiology *J. Math. Biol.* **35** . **35** (1996) 21–36.
4. S. Ruan, W. Wang, Dynamical behavior of an epidemic model with a nonlinear incidence rate, *J. Differential Equations* **188** (2003) 135–163.
5. Y. Takeuchi, X. Liu, J. Cui, Global dynamics of *SIS* models with transport-related infection, *J. Math. Anal. Appl* , **329** (2007) 1460 -1471.
6. W. Wang, Z. Ma, Global dynamics of an epidemic model with time delay, *Nonlinear Anal. , Real World Appl.* **3** (2002) 365 373.
7. R.K.Naji , R. M.Hussien , The Dynamics of Epidemic Model with Two Types of Infectious Diseases and Vertical Transmission , *Journal of Applied Mathematics* , *Volume* (2016).
8. M. E. Alexander and S. M. Moghadas, Periodicity in an epidemic model with a generalized non-linear incidence *Mathematical Biosciences Indiana Univ. Math. J.* **189** , no. 1, 75 96, (2004).
9. M. E. Alexander and S. M. Moghadas, Bifurcation analysis of SIRS epidemic model with generalized incidence, , *SIAM Journal on Applied Mathematics* **65**(5) (2005) 1794–1816.
10. Y. Jin , W. D. Wang, and S. W. Xiao, An SIRS model with a nonlinear incidence rate, *Chaos, Solitons Fractals* , **34**(5) (2007) 1482 -1497 .
11. G. H. Li and W. D. Wang, Bifurcation analysis of an epidemic model with nonlinear incidence, *Applied Mathematics and Computation*, **214** (2) 411- 423(2009) .
12. S. G. Ruan and W. D. Wang, Dynamical behavior of an epidemic model with a nonlinear incidence rate, *Journal of Differential Equations* **188** (1)135 163 (2003) .
13. W. M. Liu, S. A. Levin, and Y. Iwasa, Influence of nonlinear incidence rates upon the behavior of SIRS epidemiological models, *Journal of Mathematical Biology*, vol. 23, no. 2, pp. 187 204, 1986.
14. W. D. Wang and S. G. Ruan, Bifurcation in an epidemic model with constant removal rate of the infectives, *Journal of Mathematical Analysis and Applications*, **291**(2) (2004) 775 -793.
15. W. D.Wang, Backward bifurcation of an epidemic model with treatment, *Mathematical Biosciences*, **201** (1-2) ,(2006) 58 -71.
16. J. C. Eckalbar and W. L. Eckalbar, Dynamics of an epidemic model with quadratic treatment, *Nonlinear Analysis: Real World Applications* **12** (1) (2016) 320 -332.
17. X. Zhang and X. N. Liu, Backward bifurcation of an epidemic model with saturated treatment function, *Journal of Mathematical Analysis and Applications* , **348**(1) 433 - 443, 2008.
18. P. van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Math.Biosci.*, 180, pp. 29 -48, (2002).
19. B. Buonomo, D. Lacitignola, On the backward bifurcation of a vaccination model with nonlinear incidence , *Nonlinear Analysis: Modelling and Control* **16**(1) 30 -46, (2011).
20. J. Zhang, J. Jia, and X. Song, Analysis of an SEIR Epidemic Model with Saturated Incidence and Saturated Treatment Function *Article ID 910421* **2014** , 11 pages (2014).
21. J. Carr, Applications of Centre Manifold Theory *Springer, New York* (1981).

22. J. Guckenheimer, P. Holmes, Nonlinear Oscillations, Dynamical Systems and Bifurcations of Vector Fields, *Springer-Verlag Berlin* (1983).
23. V. Capasso, G. Serio, A generalization of the Kermack C. Mckendrick deterministic epidemic model *Math. Biosci.*, **42** 43-75 (1978) .
24. H. W.Hethcote, ,Y.Li,Z.Jing, Hopf Bifurcation in Models for Pertussis Epidemiology,*Mathematical and Computer Modelling* ,**30** (11-12), 29–45. (1999).
25. 25. M.Y. Li, J.S. Muldowney, P. van den Driessche, Global stability of SEIRS models in epidemiology,*Can. Appl. Math. Q.*,**7**,(1999). 409-425,
26. C. Castillo-Chavez, B. Song, Dynamical models of tuberculosis and their applications,*Math. Biosci. Eng.*, bf 1(2004) 361- 404,