

# Dynamical Study of an SIR Epidemic Model with Nonlinear Incidence Rate and Regress of Treatment

Saba Noori Majeed

snoori39@yahoo.com

Dept. of Computer/ College of Education for Women/ University of Baghdad

## Abstract

In this research, dynamical study of an SIR epidemical model with nonlinear direct incidence rate (Beddington-De Angelis) type, and regress of treatment investigated. An analytical study to the model shows that there are two equilibrium points appear, the discussed successfully with sufficient condition, the existence of local bifurcation and Hopf bifurcation was analyzed, finally numerical simulations are done to explain the analytic studies.

**Keywords:** SIR epidemic model, nonlinear incidence rate, regress treatment

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## 1. Introduction

Many theoretical mathematical study focused on SIR epidemical model, the classical model (SIR) usually has two equilibrium points, disease free equilibrium point DFE and at most one endemic equilibrium point EEP, see [1]. The basic reproduction number (threshold value)  $R_0$  is a very important tool to discuss stability of the equilibrium points [2]: when  $R_0 < 1$  then DFE exists and stable, while if  $R_0 > 1$  the DFE become unstable and the EEP exists and stable. The bifurcation happened when the DFE stability turns over from stable to unstable, and EEP occurs and stable, and this is called trans critical bifurcation. Newly many researchers have been very interested in nonlinear epidemical dynamic (such as saddle- node bifurcation and Hopf bifurcation), see [3,4,5,6]. For instance in [7] Dubey studied an SIR model with both nonlinear incidence and treatment rate, in [8] Li and Gosh proposed an SIR model with nonlinear incidence rate given by  $\frac{\alpha SI}{1+\gamma I}$  this incidence rate assumess that crowding of infective individuals caused effective contact between infective and susceptible. Beddington and De Angelis in [9] and [10] respectively, independently introduced nonlinear incidence rate known as Beddington-DeAngelis type incidence rate  $\frac{\alpha SI}{1+bs+cl}$  which is investigated in our research. The above researchers prove that the nonlinear dynamics of these epidemic models are very sensitive to the parameters included in the model and the outcomes may be highly affected depending on the population size, direct and indirect incidence rate and treatment rate.

In view of the previous studies we study the dynamic of an SIR nonlinear model with nonlinear direct incidence rate as (Beddington-DeAngelis type) incidence rate  $\frac{\alpha SI}{1+bs+cl}$ , and regress of treatment. The research is arranged into five sections; section 2 introduced the improvement of epidemic model study, section 3 demonstrates the existence of the equilibrium points and prove its local and global stabilities, in section 4 we investigate the Sotomayor theorem [11] to analyze the local bifurcation, to show the occurance of transcritical bifurcation however there in no saddle node and no pitch fork bifurcation, as well no occurance of Hopf bifurcation. Finally, in section 5 a numerical simulation is used to confirm our obtained analytical results and specify the control set of parameters.

## 2. Mathematical model

Consider a community of people  $N(t)$  at time  $t$ , whose population is distinguished into three compartments,  $S(t)$  represents susceptible people,  $I(t)$  represents infected people and  $R(t)$  is the recovered people at time  $t$ . Susceptible people  $S$  can become infected  $I$  with the disease only in case of direct contact with infected individuals and their chances of recovery are weak. The recovery  $R$  regression happened in many different ways such as the treatment is not available enough, or because lack of efficacy of treatment, or there is no actual treatment to cure the injury, leaving the infected person with the disease and the cause of transmission of the infection directly, and the effectiveness of treatment is limited to temporary suppression of symptoms of the disease ends if the patient stops taking medication, for instance hepatitis c, hepatitis b and AIDS...etc.. According to the previous description we assume the SIR epidemic model in our article development is based on model (1)

$$\begin{aligned} \frac{dS}{dt} &= A - \alpha SI - \alpha S \\ \frac{dI}{dt} &= \alpha SI + \beta R - \gamma(1 - \delta)I - (\alpha + k)I \end{aligned} \quad \dots\dots(1)$$

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$$\frac{dR}{dt} = \gamma(1 - \delta)I - (\alpha + \beta)R$$

model (1) represents an SIR nonlinear epidemic model with linear incidence rate  $a$  and failure treatment rate  $\delta$ . In this research we adopt the Beddington-DeAngelis incidence rate  $\frac{aSI}{1+bS+cl}$  see [7], this incidence rate indicates direct contact between the effective and susceptible individuals under crowded number of effective individuals, to get model (2) as follows:

$$\begin{aligned} \frac{dS}{dt} &= A - \frac{aSI}{1+bS+cl} - \alpha S \\ \frac{dI}{dt} &= \frac{aSI}{1+bS+cl} + \beta R - \gamma(1 - \delta)I - (\alpha + k)I \quad \dots\dots(2) \\ \frac{dR}{dt} &= \gamma(1 - \delta)I - (\alpha + \beta)R \end{aligned}$$

Model (2) is formulated with following hypotheses:

$A > 0$ , represents the natural birth rate of the population,  $b > 0$  is the measure rate of inhabitation effect such as preventive measure taken by susceptible,  $c > 0$  is the measure rate of inhabitation effect such as treatment with respect to infective,  $\alpha > 0$ , represents natural death rate,  $\beta > 0$  retreat rate with medication for individuals undergoing treatment,  $k > 0$  disease related death rate.  $\gamma > 0$  is the recovery rate due to success of treatment in some cases of infected individuals, finally  $0 \leq \delta \leq 1$ , is the failure treatment rate of infected individuals, when  $\delta = 0$  this means the treatment is very effective to treat the disease, however if  $\delta = 1$  then the treatment used is ineffective at all.

Therefore the dynamics of above described epidemic system can be described by the set of differential equations in model (2), with an initial condition in the region  $\mathbb{R}_+^3 = \{(S, I, R) \in \mathbb{R}_+^3; S > 0, I \geq 0, R \geq 0\}$ ; clearly the interaction functions given on the right hand side of model (2) are continuously differentiable. Therefore, the solution of model (2) with non-negative initial condition exists and is unique. Moreover, the solution is uniformly bounded as shown in the following theorem.

### Theorem 1

All the solutions of model (2), which initiate in the region  $\mathbb{R}_+^3$  are uniformly bounded at the set  $\Omega = \{(S, I, R) \in \mathbb{R}_+^3; R \geq 0, I \geq 0, S > 0 \text{ and } 0 < N(t) < \frac{A}{\alpha}\}$ .

### Proof

Let  $N(t) = S(t) + I(t) + R(t)$  represents the population size, then

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = A - \alpha N - kI, \text{ then we get}$$

$N(t) \leq N(0)e^{-\alpha t} + \frac{A}{\alpha}(1 - e^{-\alpha t})$ , and  $\lim_{t \rightarrow 0} N(t) < \frac{A}{\alpha}$ , so  $\frac{dN}{dt} < 0$  if  $N(t) > \frac{A}{\alpha}$ , and the solution of system (2) is uniformly bounded and defined at the set  $\Omega$ , prove complete. ■

## 3. Existence and Stability of Equilibrium Points.

In this section, we study the existence of equilibrium points of model (2), and their stability (local and global). It is clear model (2) has two equilibrium points, namely  $E_0$  and  $E_1$ , disease free equilibrium point DFE and endemic equilibrium point EEP respectively.  $E_0$  occurs when the population consists only of susceptible individuals, this means there are no

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infected individuals and no recovered individuals( i.e. disease does not exist), so  $I=0, R=0$  from model(2) we get  $E_0 = (S_0, 0, 0) = (\frac{A}{\alpha}, 0, 0)$ . However if the disease is present then infected and recovered individuals are present and the endemic equilibrium point EEP,  $E_1=(S_1, I_1, R_1)$  arises, where  $I_1 \neq 0, R_1 \neq 0$ , note that  $S_1, I_1,$  and  $R_1$  represent the positive solution of system (3):

$$A - \frac{aS_1I_1}{1+bS_1+cI_1} - \alpha S_1 = 0$$

$$\frac{aS_1I_1}{1+bS_1+cI_1} + \beta R_1 - \gamma(1 - \delta)I_1 - (\alpha + k)I_1 = 0 \quad \dots \dots \dots (3)$$

$$\gamma(1 - \delta)I_1 - (\alpha + \beta)R_1 = 0$$

Now from the third equation of system (3) we get  $R_1 = \frac{\gamma(1-\delta)}{(\alpha+\beta)} I_1 \quad \dots(4)$

substituting (4) in the second equation of system (3) we get  $S_1 = \frac{((\gamma(1-\delta)+(\alpha+k)) - (\frac{\beta\gamma(1-\delta)}{(\alpha+\beta)}))(1+cI_1)}{(a + \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)}b - \gamma(1-\delta) + (\alpha+k)b)} \dots(5)$ , While  $I_1$  is a positive root of the following equation

$$B_1I_1^2 + B_2I_1 + B_3 = 0 \quad \dots(6)$$

here:  $B_1 = - \left[ ac \left( (\gamma(1 - \delta) + (\alpha + k)) - \left( \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} \right) \right) \left( a + \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} b - \gamma(1 - \delta) + (\alpha + k)b \right) + abc^2 \left( (\gamma(1 - \delta) + (\alpha + k)) - \left( \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} \right) \right)^2 + \alpha c^2 \left( a + \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} b - (\gamma(1 - \delta) + (\alpha + k)b) \right) \right] \dots\dots\dots(6a)$

$$B_2 = \left[ cbA \left( (\gamma(1 - \delta) + (\alpha + k)) - \left( \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} \right) \right) \left( a + \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} b - (\gamma(1 - \delta) + (\alpha + k)b) \right) + Ac \left( a + \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} b - (\gamma(1 - \delta) + (\alpha + k)b) \right)^2 - a \left( (\gamma(1 - \delta) + (\alpha + k)) - \left( \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} \right) \right) \left( a + \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} b - \gamma(1 - \delta) + (\alpha + k)b \right) - ac \left( (\gamma(1 - \delta) + (\alpha + k)) - \left( \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} \right) \right) \left( a + \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} b - (\gamma(1 - \delta) + (\alpha + k)b) \right) - 2acb \left( (\gamma(1 - \delta) + (\alpha + k)) - \left( \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} \right) \right)^2 - ac \left( (\gamma(1 - \delta) + (\alpha + k)) - \left( \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} \right) \right) \right] \dots\dots\dots(b)$$

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$$\begin{aligned}
 B_3 &= Ab \left( (\gamma(1 - \delta) + (\alpha + k)) - \left( \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} \right) \right) \left( a + \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} b - (\gamma(1 - \delta) + \right. \\
 &(\alpha + k))b \Big) - \alpha \left( (\gamma(1 - \delta) + (\alpha + k)) - \left( \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} \right) \right) \left( a + \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} b - (\gamma(1 - \delta) + \right. \\
 &(\alpha + k))b \Big) - ab \left( (\gamma(1 - \delta) + (\alpha + k)) - \left( \frac{\beta\gamma(1-\delta)}{(\alpha+\beta)} \right) \right)^2 \\
 &\dots\dots\dots(6c)
 \end{aligned}$$

Clearly, by using Descarte’s rule [12] of signs then equation (6) has a unique positive root given by  $I_l$  if and only if one of the following conditions hold:  $B_2 < 0$  or  $B_3 > 0$ . In order to discuss stability of the above equilibrium points the basic reproduction number  $R_0$  [2], which represents the number of new infected people caused by a single infected patient when introduced into a completely susceptible population, is computed. It is well known that the value of this number plays a necessary role in the stability of the model (2). According to definition of basic reproduction number that given in [2], rewrite model (2) in the form  $x' = \mathcal{H}(x) - \mathcal{M}(x)$  .....(7)

where  $x = (I, S, R)^T$ ,  $\mathcal{H}(x)$  is the matrix of new infection individuals and  $\mathcal{M}(x)$  is the matrix of transferred individuals between the infected compartments and out of the infected compartments. Therefore

$$\mathcal{H}(x) = \begin{bmatrix} \frac{aSI}{1+bS+cl} \\ 0 \\ 0 \end{bmatrix}; \mathcal{M}(x) = \begin{bmatrix} -\beta R + \gamma(1 - \delta)I + (\alpha + k)I \\ \frac{aSI}{1+bS+cl} + \alpha S - A \\ (\alpha + \beta)R - \gamma(1 - \delta)I \end{bmatrix}$$

So the derivative of  $\mathcal{H}(x)$  and  $\mathcal{M}(x)$ , with respect to vector  $x$ , at the disease free equilibrium point  $E_0$  are computed as follows:

$$D\mathcal{H}(x) = \begin{bmatrix} \frac{aA}{\alpha+bA} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} = \begin{bmatrix} H & \mathbf{0} \\ \mathbf{0} & \mathbf{0} \end{bmatrix}; \mathcal{M}(x) = \begin{bmatrix} \gamma(1 - \delta) + (\alpha + k) & 0 & -\beta \\ \frac{aA}{\alpha+bA} & 0 & 0 \\ -\gamma(1 - \delta) & 0 & (\alpha + \beta) \end{bmatrix} =$$

$\begin{bmatrix} M & \mathbf{j}_1 \\ \mathbf{j}_2 & \mathbf{j}_3 \end{bmatrix}$ , where  $H = \left[ \frac{aA}{\alpha+bA} \right]$ ; and  $M = [\gamma(1 - \delta) + (\alpha + k)]$ . Consequently, according to definition of  $R_0$  that given in [2],  $R_0$  is equal to spectral radius of new generation matrix  $\mathcal{H}\mathcal{M}^{-1}$ . Therefore, the reproduction number of model (2) is given by

$$R_0 = \rho(\mathcal{H}\mathcal{M}^{-1}) = \frac{aA}{(\alpha+bA)(\gamma(1-\delta)+(\alpha+k))} \dots\dots\dots(8)$$

**Theorem 2:** The disease free equilibrium point DFE of model (2) is locally asymptotically stable provided that

$$R_0 < 1 \quad \dots(9a). \text{ While its unstable for } R_0 > 1 \quad \dots(9b)$$

**Proof**

The Jacobian matrix of model (2) at  $E_0$  can be written as:

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$$J(E_0) = [b_{ij}]_{3 \times 3} = \begin{bmatrix} -\alpha & \frac{aA}{\alpha+bA} & 0 \\ 0 & \frac{aA}{\alpha+bA} - (\gamma(1-\delta) - (\alpha+k)) & -\beta \\ 0 & \gamma(1-\delta) & -(\alpha+\beta) \end{bmatrix} \dots\dots(10a)$$

Then the characteristic equation of  $J(E_0)$  is given by

$$\lambda^3 + \Omega_1\lambda^2 + \Omega_2\lambda + \Omega_3 = 0 \dots\dots\dots(10b)$$

Where:

$$\Omega_1 = -(b_{11} + b_{22} + b_{33})$$

$$\Omega_2 = \{(b_{11}b_{22} - b_{12}b_{21}) + (b_{11}b_{33} - b_{13}b_{31}) + (b_{22}b_{33} - b_{23}b_{32})\}$$

$$\Omega_3 = -b_{11}b_{22}b_{33} + b_{11}b_{23}b_{32}$$

Further:  $\Delta = \Omega_1\Omega_2 - \Omega_3$

Now according to (Routh-Hurwitz) criterion [13],  $E_0$  is locally asymptotically stable provided that  $\Omega_1 > 0$  if  $b_{22} < 0$ ,  $\Omega_3 > 0$  if  $b_{22} < 0$ , and  $\Delta > 0$  if  $b_{22} < 0$ , i.e.  $R_0 < 1$ .

Clearly  $\Omega_1 > 0$ ,  $\Omega_3 > 0$ , and  $\Delta > 0$  holds in case condition (9a) satisfied.

However if condition (10a) happened then  $\Omega_1 < 0$ , and  $E_0$  in this case is unstable equilibrium point. Prove complete ■

**Theorem 3:** The endemic equilibrium point  $E_1 = (S_1, I_1, R_1)$  of model (2) is locally asymptotically stable, provided that

$$1 < R_0 < \frac{(1+bS_1+cI_1)^2}{1+bS_1} \dots\dots\dots(11)$$

**proof**

The Jacobian matrix of model (2) at the endemic equilibrium point  $E_1$  can be written as:

$$J(E_1) = [c_{ij}]_{3 \times 3} = \begin{bmatrix} \frac{-(aI_1+acI_1^2)}{(1+bS_1+cI_1)^2} - \alpha & \frac{-(aS_1+abS_1^2)}{(1+bS_1+cI_1)^2} & 0 \\ \frac{(aI_1+acI_1^2)}{(1+bS_1+cI_1)^2} & \frac{(aS_1+abS_1^2)}{(1+bS_1+cI_1)^2} - (\gamma(1-\delta) - (\alpha+k)) & \beta \\ 0 & \gamma(1-\delta) & -(\alpha+\beta) \end{bmatrix}$$

Then the characteristic equation of  $J(E_0)$  is given by

$$\lambda^3 + \Omega_1\lambda^2 + \Omega_2\lambda + \Omega_3 = 0 \dots\dots(10b), \text{ Where:}$$

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$$\Omega_1 = -(c_{11} + c_{22} + c_{33})$$

$$\Omega_2 = \{(c_{11}c_{22} - c_{12}c_{21}) + (c_{11}c_{33} - c_{13}c_{31}) + (c_{22}c_{33} - c_{23}c_{32})\}$$

$$\Omega_3 = -c_{11}c_{22}c_{33} + c_{33}c_{12}c_{21} + c_{11}c_{23}c_{32}$$

Further:  $\Delta = \Omega_1\Omega_2 - \Omega_3$

Now according to (Routh-Hurwitz) criterion [13],  $E_I$  is locally asymptotically stable provided that  $\Omega_1 > 0$ ,  $\Omega_3 > 0$ , and  $\Delta > 0$ . It is easy to verify that  $\Omega_1 > 0$ ,  $\Omega_3 > 0$ , and  $\Delta > 0$  holds in case condition (11) come true. Prove complete

■

#### Theorem 4 :

Assume that the disease free equilibrium point of model (2) is locally asymptotically stable then it's globally asymptotically stable for all:

$$S > 0, I \geq 0 \text{ and } R \geq 0 \quad \dots\dots(12)$$

#### Proof

Let  $\mathcal{W}_1 = \left(S - S_0 - S_0 \ln \frac{S}{S_0}\right) + I + R$ , clearly  $\mathcal{W}_1: \mathbb{R}_+^3 \rightarrow \mathbb{R}$  is continuously differential function such that  $\mathcal{W}_1(S_0, 0, 0) = 0$ , and  $\mathcal{W}_1(S, I, R) > 0; \forall (S, I, R) \neq (S_0, 0, 0)$ . Differentiating  $\mathcal{W}_1$  with respect to time and then after doing some algebraic computation,

we have: 
$$\frac{d\mathcal{W}_1}{dt} = -\alpha \frac{(S-S_0)^2}{S} - \alpha(R+I) - kI$$

Clearly condition (12) guarantee that  $\frac{d\mathcal{W}_1}{dt} < 0$ . Hence according to Lyapunov second theorem [14], of stability the free disease equilibrium point  $E_0$  is locally asymptotically stable and the proof is complete.

■

Next theorem establishes the global stability conditions for the endemic equilibrium point of model (2).

#### Theorem 5

Assume that the endemic equilibrium point  $E_1$ , of model (2) is locally asymptotically stable, then its globally asymptotically stable for all:

$$S > 0, I \geq 0 \text{ and } R \geq 0 \quad \dots\dots(13)$$

#### Proof

Consider the following function:  $\mathcal{W}_2 = \frac{(S-S_1)^2}{2} + \frac{(I-I_1)^2}{2} + \frac{(R-R_1)^2}{2}$ , Clearly  $\mathcal{W}_2 = \mathbb{R}_+^3 \rightarrow \mathbb{R}$  is continuously differential function with  $\mathcal{W}_2(S_1, I_1, R_1) = 0$ , while  $\mathcal{W}_2(S, I, R) > 0; \forall (S, I, R) \neq (S_1, I_1, R_1)$ . Moreover, straightforward computation gives that:  $\frac{d\mathcal{W}_2}{dt} = -(S - S_1)^2 q_{11} - (I - I_1)^2 q_{22} - (R - R_1)^2 q_{33}$ .

Where:

$$q_{11} = \frac{acI^2 + aI + \alpha(1+bS+cl)(1+bl+cS_1)}{(1+bS+cl)(1+bl+cS_1)}, \quad q_{22} = \frac{abS^2 + aS + \gamma(1-\delta) + (\alpha+k)(1+bS+cl)(1+bl+cS_1)}{(1+bS+cl)(1+bl+cS_1)} \text{ and}$$

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$$q_{33} = -(\beta + \gamma)$$

Thus according to the given conditions it's easy to verify that clearly condition (13) come true and  $\frac{dW_2}{dt} < 0$ , hence due to the second Lyapunov theorem [14], the endemic equilibrium point of model (2), is globally asymptotically stable. ■

#### 4. Local bifurcation analysis

In this section, the local bifurcation (such as saddle-node, transcritical and pitchfork) is studied to find out the influence of changing one of the parameters value, on the dynamical behavior of system (1) around the disease free equilibrium point  $E_0$ . An application of the Sotomayor's theorem, which presented in [10], for the local bifurcation is carried out in order to specify the type of bifurcation near the equilibrium point as shown in the following theorem.

##### Theorem 6

Assume that  $\delta = 1$ , then model (2) undergoes a transcritical bifurcation near the disease free equilibrium point  $E_0$ , but neither saddle-node bifurcation nor pitchfork bifurcation can occur, provided that

$$\frac{\alpha(1+bS_0^2)+2abS_0}{(1+bS_0^2)^2} + \frac{q}{(1+bS_0^2)^2} + \frac{2acS_0}{(1+bS_0^2)^2} \neq 0 \quad \dots (14), \text{ where } q \text{ is given in the proof.}$$

##### Proof

It is easy to verify that when  $\delta = 1$  then the Jacobian matrix given by (8a) would be :

$$J = Df(E_0, \alpha^*) = \begin{bmatrix} \alpha & \frac{\alpha^*A}{\alpha+bA} & 0 \\ 0 & 0 & \beta \\ 0 & 0 & -(\beta + \alpha) \end{bmatrix}; \text{ Clearly the second eigenvalue of}$$

$J$   $\lambda_I$  in the  $I$ -direction is zero ( $\lambda_I = 0$ ), while  $\lambda_s = \alpha$  and  $\lambda_R = -(\beta + \alpha)$ . Hence the disease free equilibrium point is a non-hyperbolic point for  $R_0 = 1$ .

Further the eigenvector, say  $K = (k_1, k_2, k_3)^T$ , that corresponds  $\lambda_I = 0$  is determined as:

$$K = (qk_2, k_2, 0)^T \quad (14a), \quad \text{where } q = \frac{\alpha^*A}{\alpha(\alpha+bA)} \text{ and } k_2 \text{ is a non-zero real}$$

number. Similarly the eigenvector  $W = (w_1 \ w_2 \ w_3)^T$  that corresponds  $\lambda_I = 0$  of  $J^T$  is determined as:

$W = (0, pw_3, w_3)^T$  (14b), where  $w_3$  is a non-zero real number. Now rewrite model (2) as vector form  $\frac{dX}{dt} = f(x)$ , where  $X = (S, I, R)^T$  and  $f = (f_1, f_2, f_3)^T$  be the interaction functions vector given in model (2). Then by determining  $\frac{df}{d\alpha} = f_\alpha$ , we get that:

$$f_\alpha = \begin{bmatrix} \frac{-SI}{1+bS+cI} \\ \frac{SI}{1+bS+cI} \\ 0 \end{bmatrix} \text{ then } f_\alpha(E_0, \alpha^*) = \begin{bmatrix} 0 \\ 0 \\ 0 \end{bmatrix} \text{ Therefore: } w^T \cdot f_\alpha(E_0, \alpha^*) = 0, \text{ Cosequently,}$$

according to Sotomayor theorem, model (2) has no saddle-node bifurcation near  $E_0$  at  $\alpha = \alpha^*$ . Now in order to investigate the other types of bifurcation, the derivative of  $f_\alpha$  with respect to vector  $X$  say  $Df_\alpha(E_0, \alpha^*)$  is computed:

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$$Df_{\alpha}(E_0, \alpha^*) = \begin{bmatrix} 0 & \frac{-S_0}{1+bS_0} & 0 \\ 0 & \frac{S_0}{1+bS_0} & 0 \\ 0 & 0 & 0 \end{bmatrix}, \text{ and } W^T[Df_{\alpha}(E_0, \alpha^*), K] = w_3 p k_2 \frac{S_0}{1+bS_0} \neq 0. \text{ Again,}$$

in view of Sotomayor theorem, if in addition to the above the following holds  $W^T[D^2f(E_0, \alpha^*), (K, K)] \neq 0$ , then model (2) possesses a transcritical bifurcation but no pitchfork bifurcation appear. Now since we get that:

$$D^2f(E_0, \alpha^*), (K, K) = \begin{bmatrix} [q^2 k_2^2 \frac{-a(1+bS_0^2)+2abS_0}{(1+bS_0^2)^2} - q k_2^2 \frac{q}{(1+bS_0^2)^2}] - k_2^2 \\ q^2 k_2^2 [\frac{a(1+bS_0^2)+2abS_0}{(1+bS_0^2)^2} + q k_2^2 \frac{q}{(1+bS_0^2)^2}] + k_2^2 \frac{2acS_0}{(1+bS_0^2)^2} \\ 0 \end{bmatrix}; \quad \text{Thus it}$$

follows that :  $W^T D^2f(E_0, \alpha^*), (K, K) \neq 0$ . Therefore model (2) has a trans critical bifurcation at  $E_0$  with  $\alpha = \alpha^*$  provided that condition (14) holds. Prove complete ■  
Now it is well known that, the three dimensional dynamical system undergoes a Hopf bifurcation if and only if there is a complex conjugate eigenvalue, say  $\lambda_i = \rho_1 \mp i\rho_2$ , with third eigenvalue is real and negative, so that [14]

$$\rho_1(\ell^*) = 0 \quad (15a), \text{ and } \frac{d\rho_1}{d\ell} |_{\ell=\ell^*} \neq 0 \quad (15b)$$

where  $\ell = \ell^*$  be a specific general parameter. This is equivalent, according to [14], to that model (2) undergoes a Hopf bifurcation around the endemic equilibrium point if and only if the coefficient's of the characteristic polynomial given by (10b) satisfy that  $\Omega_i > 0; \forall i = 1, 2, 3$  with  $\Omega_1(\ell^*)\Omega_2(\ell^*) = \Omega_3(\ell^*)$  (i.e.  $\Delta(\ell^*) = 0$ ) such that  $\frac{d\Delta}{d\ell} |_{\ell=\ell^*} \neq 0$ .

Since the condition (11) that guarantees  $\Omega_i > 0; \forall i = 1, 2, 3$  is the same condition which guarantees  $\Delta > 0$ , therefore there is no possibility to have a Hopf bifurcation for model (2).

## 5. Results and Numerical Simulation.

In this section, we will emphasize two main analytic results numerically according to the parameters hypotheses in table 1, we get:

1. Stability of equilibrium point  $E_0$  and  $E_I$  based on (basic reproduction number  $R_0$ ).

i. According to data in table 1,  $R_0 < 1 = 0.0769$  and the disease free equilibrium point of model (2),  $E_0 = (125, 0, 0)$  is locally asymptotically stable. We find that all trajectories  $p_1, p_2$ , and  $p_3$  are tends to  $E_0$ , which started from three different initial points:  $p_1 = (25, 15, 20)$ ,  $p_2 = (35, 25, 40)$ ,  $p_3 = (75, 35, 50)$ , as shown in (Figure1) achieve the result we get in theorem (2) at section 3., however when the incident rate  $a = 0.1$  preserving the rest data in table 1, and  $R_0 = 0.769 < 1$ , the disease free equilibrium point disappears and the endemic equilibrium point  $E_I = (78, 27, 5)$  shows up as asymptotically stable point as shown in (Figure2).

ii. According to data in table 1 when  $a = 0.2$  we have  $R_0 = 1.587 > 1$  the endemic equilibrium point of model (2)  $E_I = (33, 54, 10)$  is locally asymptotically stable and all

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solution tends to it started from three different initial points  $p_1 = (25, 15, 20)$ ,  $p_2 = (35, 25, 40)$ ,  $p_3 = (75, 35, 50)$  as shown in (Fig.5), achieve the result we get in theorem(3) at section 3.

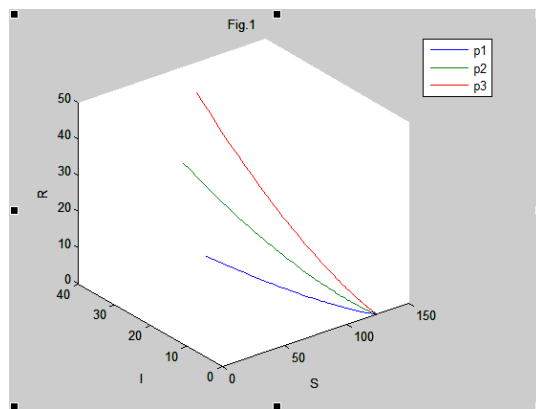
2. The effect of the parameter  $\delta$  that responsible on failure of treatment on model (2) shown as follows:

i. According to data in table 1, the time series trajectories in (Figure3) shows that when  $\delta = 0$  the susceptible individuals trajectory increase more than 50, while the infected decrease to 20 and recovered individuals is less than 10, this support model (2), when there is no failure of treatment(i.e.  $\delta = 0$ ), the population follows natural approach against the epidemic disease.

ii. While relatively to data in table 1, with  $a = 0.2$  (Figure4) shows that the susceptible individuals trajectories decrease less than 40, however the infected individuals increase to 70 and recovered individuals are exactly = 0, again this result support model (2), when the failure of treatment is full (i.e.  $\delta = 1$ ), the population is in complete decline with treatment. In addition a transcritical bifurcation happened (when  $\delta = 1$ ) for the disease free equilibrium point  $E_0$  and this demonstrates theorem 6.

**Table (1)**

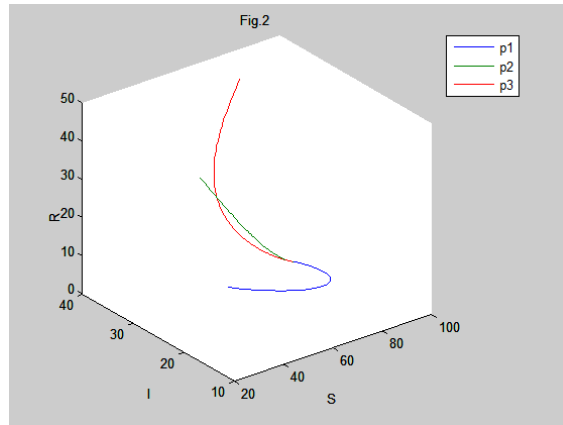
A	a	b	c	k	$\alpha$	$\beta$	$\gamma$	$\delta$	$E_0$	$E_1$	$R_0$
50	0.01	0.1	0.1	0.2	0.4	0.2	0.5	0.4	stable	Unstable	<1
								0	Stable	Unstable	<1
								1	Unstable	stable	>1
50	0.1	0.1	0.1	0.2	0.4	0.2	0.5	0.4	Unstable	Stable	$\approx 1$
50	0.2	0.1	0.1	0.2	0.4	0.2	0.5	1	unstable	stable	=1



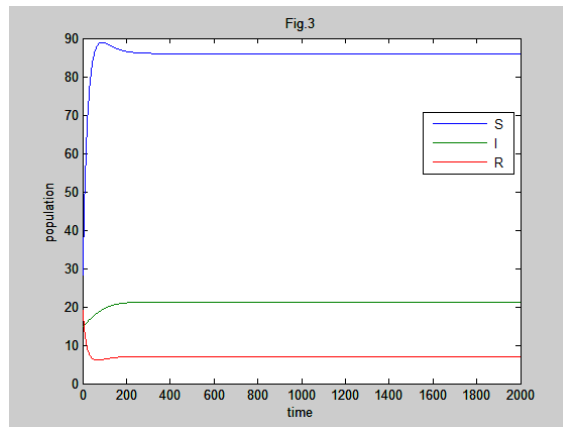
**Figure (1):** Stability of ( $E_0$ ) the trajectories  $p_1$ ,  $p_2$  and  $p_3$  are started from three different initial points with  $R_0 < 1$  and  $a=0.01$

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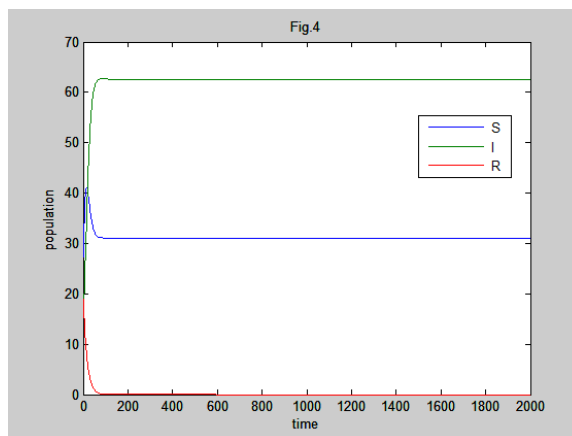
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**Figure (2):** Stability of  $(E_1)$  the trajectories started from three different initial points  $p_1, p_2$  and  $p_3$  and data in table 1, for  $R_0 < 1$  and  $a=0.1$



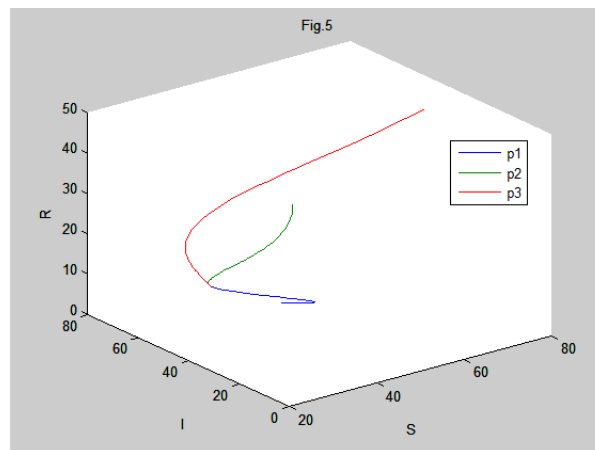
**Figure (3):** Time series of model (2) with respect to data in table 1, when  $\delta = 0$  and  $a=0.01$ .



**Figure (4):** Time series of model (2) with respect to data in table 1, when  $a = 0.2$ .

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**Figure (5):** Stability of  $(E_1)$  the trajectories started from three different initial points  $p_1$ ,  $p_2$  and  $p_3$ , for data in table 1 when  $R_0 > 1$  and  $a = 0.2$ .

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