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Modelling and Analysis of SEIR with Delay Differential Equation

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Abstract

Communicable diseases are generally referred to as those that spread from one person to another through contact with blood and body fluids, breathing in an airborne virus or being bitten by a virus carriers. We consider a communicable disease model in which transmission assume no immunity or permanent immunity. In this paper, a delay differential equation model is developed to give an account of the transmission dynamics of these diseases in a population. The stability of the equilibrium is analyzed with delay: the endemic equilibrium is locally stable without delay; and the endemic equilibrium is stable if the delay is under some condition.The basic reproductive number was established and analysed. The equilibrium points of the model was examined for local stability and its associated reproductive rate. It was found to be locally asymptotically stable whenever the reproductive number was less than one. Bifurcation analysis was conducted and it was noted that immunity duration is a sensitive parameter for dynamics of disease transmission. We performed numerical simulations of the system of equations of the model and compared the results with our theoretical analysis.

Key words: Reproduction number, Hopf bifurcation, delay differential equation, stability analysis, immunity duration.

AMS Subject classification: 92D30, 37M05.

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1. INTRODUCTION

Communicable diseases are those that spread from one person to another through a variety of ways that include; contact with blood and body fluids; breathing in an airborne virus or being bitten by a virus carriers. These diseases have claimed millions of lives in the world annually, especially in developing countries. Such diseases include Tuberculosis, malaria, influenza and Rabbles. Communicable disease modeling are basically employed to control such diseases and prevent outbreak. These models have significant biological implications as far as the investigation of the transmission dynamics of infectious diseases in host population are concerned [1, 2]

Different infectious agents display different traits and thus different dynamics arise, parameter that depend and the specific agent include the transmission rate, the recovery rate and finally immunity duration corresponding to the specific infection. Parameter that are independent of the infective agent typically include the natural birth and death rates of population relevant to an infective agents' dynamics is an incidences function, or a function that describe how infected and susceptible individual contact [3, 4].

Bifurcation theory ideally refers to the study of differences in qualitative structure in a given system. Such as integral curves of system of vector field and the solutions of coupled differential equations. This commonly applied to the study of dynamical system. Bifurcation is said to occur when small changes are made to the parameter values of a system, causes a sudden qualitative change in its behaviour. This can happen in both continuous systems and discrete systems [3, 5].

[6] formulated models by considering varying population size and considered SEIR model with varying population size. In their study, natural birth rate and death rate were incorporated. Additionally, death rate as a result disease were considered death in their models. Total population size might be varying with time. In their analysis, stability of their model was analysed with normalisation approach or method.

Alternative approach was presented in analysing or proving local and global stability of endemic equilibria. In the work done by [7], a review of work done in the field of malaria modeling was analysed. [8] created a model by considering immunity to this disease.

However, [9] initiated and formulated a model and analysed it for malaria infection when endemic. In the work of [10], they analysed a SEIR model with limited resources in the case of treatment. A general approach or method for analysing compartmental models in diseases was given by [1].

[11] also gave details approach and analyses on basic reproductive number, (R_0). The phenomenon of class or compartmental models is to ensure the division of population into groups of classes or compartments according to their epidemiological status.

Modeling of dynamic of infectious diseases are done through various models such as; Susceptible, Infection and Recovered, (SIR) which implies that susceptible people become infected recover and remain immune to any further infection. Susceptible, infectious, recovered and susceptible, $(SIRS)$, implying that susceptible people get infected recover and finally become susceptible again after immunity wears off [12, 13]. Susceptible, exposed infected, and recover, $(SEIR)$. Implying that susceptible people enter latent period called exposed state where the diseases is contagious meaning they carry the infectious agent but are not able to transmit. After a period of time expose people become infected and finally recover. Models are being assumed differently other assume that recovered person will not exhibit any sort of immunity where other models incorporated opened of immunity after recover [14, 15, 16].

2. MODEL DESCRIPTION AND FORMULATION

2.1 Model without Delay Differential Equation

The model without delay takes the form,

$$\left.\begin{aligned} \frac{dS}{dt} &= B-\beta SI-\mu S \\ \frac{dE}{dt} &= \beta SI-(\nu+\mu)E \\ \frac{dI}{dt} &= \nu E-(\rho+\mu+\delta)I \\ \frac{dR}{dt} &= \rho I-\mu R \end{aligned}\right\} \tag{2.1}$$

where $S>0$, $E\geq 0$,$I>0$, $R\geq 0$ and B is recruitment rate by birth. Since the epidemic occurs in a short time period, we ignore loss of temporary immunity.

2.2 Model with Delay Differential Equation

In the system dynamical behavior of the disease, the standard incidence rate is given as; $\frac{\beta s(t)I(t)}{N(t)}$ and the bi-linear incidence rate is$\beta s(t)I(t)$. When $I(t)$ reach the maximum number of effective contacts between the infectious and susceptible individuals, the susceptible may saturate at high infective levels due to crowding of infective individuals or due to the protection measures by the susceptible individuals.

Considering a delayed SIR model with the saturation incidence rate;

$$\frac{\beta S(t-\tau)I(t-\tau)e^{-\mu\tau}}{1+\alpha I(t-\tau)} \tag{2.2}$$

and exponential birth rate. We consider a delayed SIR model with the saturated incidence rate;

$$\frac{\beta S(t-\tau)I(t-\tau)e^{-\mu\tau}}{1+\alpha I(t-\tau)}. \tag{2.3}$$

When the parameter measure $\alpha = 0$, the saturation incidence rate will become a bi-linear incidence rate;

$$\beta S(t-\tau)I(t-\tau)e^{-\mu\tau}. \tag{2.4}$$

Considering the following SIR model with the saturation incidence rate;

$$\frac{\beta S(t-\tau)I(t-\tau)e^{-\mu\tau}}{1+\alpha I(t-\tau)} \tag{2.5}$$

and a time delay describing a latent period.

Where $S(t)$ denotes susceptible individuals, $I(t)$ denotes infective individuals, and $R(t)$ denotes recovered individuals. The following are the system of delay differential equations obtained from the model model:

$$\left.\begin{aligned}
\frac{dS(t)}{dt} &= b(S(t)+I(t)+R(t)) - \frac{\beta S(t-\tau)I(t-\tau)e^{-\mu\tau}}{1+\alpha I(t-\tau)} - \mu S(t) \\
\frac{dI(t)}{dt} &= \frac{\beta S(t-\tau)I(t-\tau)e^{-\mu\tau}}{1+\alpha I(t-\tau)} - (\mu+\rho+\delta)I(t) \\
\frac{dR(t)}{dt} &= \rho I(t) - \mu R(t)
\end{aligned}\right\} \tag{2.6}$$

Where natural birth rate, $b > 0$, natural death rate, $\mu > 0$ and birth rate is greater than natural death rate, $(b > \mu)$.

Where,$\delta > 0$ is the disease-related death rate, $\rho > 0$ is the rate of recovery, $\frac{1}{\tau}$ is the incubation period andα is the parameter that measure infections with the inhibitory effect.

3. THE POSITIVITY AND SOLUTION BOUNDEDNESS

In this section, we consider the following system of differential equations;

$$\left.\begin{aligned} \frac{dS}{dt} &= B-\beta SI-\mu S \\ \frac{dE}{dt} &= \beta SI-\nu E-\mu E \\ \frac{dI}{dt} &= \nu E-(\rho+\mu+\delta)I \end{aligned}\right\} \tag{3.1}$$

By summing above system of equations;

$$\frac{dS}{dt}+\frac{dE}{dt}+\frac{dI}{dt} = B-\beta SI-\mu S+\beta SI-\nu E-\mu E+\nu E-(\rho+\mu+\delta)I$$

$$\frac{d(S+E+I)}{dt} = (S+E+I) = B-\mu(S+E+I)-\delta I-\rho I \leq B-\mu(S+E+I)$$

This implies;

$$\limsup_{t\to\infty}(S+E+I) \leq \frac{B}{\mu} \tag{3.2}$$

So, the feasible region for (4) is

$$\Lambda = \{S,E,I) : S+E+I \leq \frac{B}{\mu}, S>0, E>0, I>0\} \tag{3.3}$$

4. EQUILIBRIUM POINTS

4.1 Disease free equilibrium

In this section, we compute the models endemic equilibrium points.The disease free equilibrium is obtained by setting the system of differential equations to zero. At disease free equilibrium, there are no infections and recovery. The disease free equilibrium is given by;

$$(S^*,E^*,I^*,R^*) = \left(\frac{B}{\mu},0,0,0\right) \tag{4.1}$$

4.2 Endemic equilibrium

In this section, we compute the models endemic equilibrium points.This is obtained by setting the system of differential equations to zero. The models endemic equilibrium

point is given by;
(S^*, I^*, R^*).

$$\left.\begin{array}{lll} S^* & , & \dfrac{(\mu+\rho+\delta)(I+\alpha I^*)}{\beta e^{-\mu\tau}} \\ I^* & , & \dfrac{\mu(b-\mu)(\mu+\rho+\delta)}{\beta e^{-\mu\tau}\{\mu(\mu+\rho+\delta)-b(\mu+\rho)\}-\alpha\mu(b-\mu+\delta)} \\ R^* & , & \dfrac{\rho I^*}{\mu} \end{array}\right\} \tag{4.2}$$

5. BASIC REPRODUCTIVE NUMBER

The basic reproduction number is defined as the number of secondary infection that one infected individual can produce in a completely susceptible population. This number or threshold value determines the spread of the infection. Using the next generation matrix approach in [15, 12, 1], we compute the basic reproduction number, (R_0).

The disease free equilibrium of the system is given by the relation;

$\xi_0 = [\frac{B}{\mu}, 0, 0]$

Therefore, I has to be less than its initial value I_0. Let $X^{'} = (E, I, S)^T$.

Therefore

$$X^{'} = \frac{dX}{dt} = F(X) - V(X) \tag{5.1}$$

Where $F(X)$ denotes the rate of appearance of new infections in the compartment and $V(X)$ gives the transfer of individuals.

$$F = \begin{pmatrix} \beta SI \\ \\ 0 \\ \\ 0 \end{pmatrix} \text{ and } V = \begin{bmatrix} (\nu+\mu)E \\ \\ -\nu E + (\rho+\mu+\delta)I \\ \\ -B + \beta SI + \mu S \end{bmatrix}$$

The partial derivatives of F and V at disease free equilibrium (ξ_0) are given by;

$F = \left[\dfrac{\partial F_i(X_0)}{\partial X_j}\right], V = \left[\dfrac{\partial V_i(X_0)}{\partial X_j}\right]$

Where $i = 1, 2; j = 1, 2$.

This gives;

$$F = \begin{bmatrix} 0 & \frac{\beta B}{\mu} \\ 0 & 0 \end{bmatrix} \tag{5.2}$$

And

$$V = \begin{pmatrix} \mu+\nu & 0 \\ -\mu & (\mu+\rho+\delta) \end{pmatrix} \tag{5.3}$$

Where;

$$FV^{-1} = \begin{pmatrix} \frac{\nu\beta B}{\mu(\mu+\nu)(\mu+\rho+\delta)} & \frac{\beta B}{\mu(\mu+\rho+\delta)} \\ 0 & 0 \end{pmatrix} \tag{5.4}$$

Hence, basic reproduction number, R_0 is given by;

$$R_0 = \frac{\nu\beta B}{\mu(\mu+\nu)(\mu+\rho+\delta)} \tag{5.5}$$

6. STABILITY

6.1 Stability of the disease free equilibrium

The disease free equilibrium, ξ_0 is locally asymptotically stable if all the eigenvalues of the matrix $D(\xi_0) = F(\xi_0) - V(\xi_0)$
have positive real parts.

Theorem 1. *Consider the disease transmission model given by with $X^{'}$.*
If ξ_0, is a disease free equilibrium of the model, then ξ_0 is locally asymptotically stable if $R_0 < 1$, but unstable if $R_0 > 1$, where R_0 is defined as;

$$R_0 = \frac{\nu\beta B}{\mu(\mu+\nu)(\mu+\rho+\delta)}. \tag{6.1}$$

Proof. Let $J = F - V$. Since V is a non-singular matrix and F is non-negative, $J = F - V$ has the Z sign pattern. Thus, □

$S(J) < 0 - J$ is non-singular matrix; $S(J)$ is spectral abscissa of J.

Since FV^{-1} is non-negative, $-JV^{-1} = 1 - FV^{-1}$also has the Z sign pattern.

Then;

Since, $FV^{-1}$is non-negative, all eigen values of FV^{-1}have magnitude less than or equal to ρFV^{-1}. Thus, $1 - FV^{-1}$is a non-singular matrix.

$\Longleftrightarrow \rho(FV^{-1}) < 1$

Hence,

$S(J) < 0$ if and only if $R_0 < 1$

Similarly it follows that $1 - FV^{-1}$ is a singular matrix

$\Longleftrightarrow \rho(FV^{-1}) = 1$

Hence,

$S(J) = 0$ if and only if $R_0 = 1$

It follows that;

$S(J) > 0$ if and only if $R_0 > 1$.

For;

$$R_0 = \frac{\nu\beta B}{\mu(\mu+\nu)(\mu+\rho+\delta)} \tag{6.2}$$

The disease free equilibrium ξ_0 is locally asymptotically stable if all the eigenvalues of the matrix;

$$D\xi_0\{= F(\xi_0) - V(\xi_0)\} \tag{6.3}$$

Have positive real parts.

6.2 Stability of the endemic equilibrium

Theorem 2. *If $R_0 < 1$, the solution of system of differential equation is*

$(S(t), I(t), R(t)) \to (\infty, 0, 0)$

with $t \to \infty$. If, system of differential equation has a unique endemic equilibrium, $E^* = (S^*, I^*, R^*)$.

Where the endemic equilibrium points are defined as;

$$\left.\begin{aligned} S^* &= \frac{(\mu+\rho+\delta)(I+\alpha I^*)}{\beta e^{-\mu\tau}} \\ I^* &= \frac{\mu(b-\mu)(\mu+\rho+\delta)}{\beta e^{-\mu\tau}\{\mu(\mu+\rho+\delta)-b(\mu+\rho)\}-\alpha\mu(b-\mu+\delta)} \\ R^* &= \frac{\rho I^*}{\mu} \end{aligned}\right\} \tag{6.4}$$

Proof. Considering the two cases: $I(t)=0$ and $I(t)>0$. If $I(t)=0$, it implies that $R(t)=0$, then it follows that $\frac{dS(t)}{dt}=(b-\mu)S(t)$. When $t\to\infty$, we have $S(t)\to\infty$. Then the solution of system of the system of differential equation; $(S(t),I(t),R(t))\to(\infty,0,0)$. If $I(t)>0$, then it implies that: $R^*=\frac{\rho I^*}{\mu}$ and from the endemic equilibrium point, $S^*=\frac{(\mu+\rho+\delta)I+\alpha I^*}{\beta e^{-\mu\tau}}$. □

Then substituting the above equations into (7) we get the unique root

$$I^*=\frac{\mu(b-\mu)(\mu+\rho+\delta)}{\beta e^{-\mu\tau}\{\mu(\mu+\rho+\delta)-b(\mu+\rho)\}-\alpha\mu(b-\mu)(\mu+\rho+\delta)} \tag{6.5}$$

If $I^*>0$, then we must have;

$\beta e^{-\mu\tau}\{\mu(\mu+\rho+\delta)-b(\mu+\rho)\}-\alpha\mu(b-\mu)(\mu+\rho+\delta)>0$

This means that $R_0>1$. Thus we get if $R_0>1$.

Now analysing the stability of the endemic equilibrium $E^*$with $R_0>1$. The characteristic equation at the endemic equilibrium, E^* is of the form;

$$f_1(\lambda)=(\lambda^3+p_1\lambda^2+p_2\lambda+p_3)+(q_1\lambda^2+q_2\lambda+q_3)e^{-\lambda\tau} \tag{6.6}$$

where;

$$\begin{aligned} P_1 &= (2\mu-b)+(\mu+\rho+\delta), \\ q_1 &= \frac{\beta I^* e^{-\mu\tau}}{I+\alpha I^*}-\frac{\beta S^* e^{-\mu\tau}}{(I+\alpha I^*)^2} \\ P_2 &= (2\mu-b)(\mu+\rho+\delta)-\mu(b-\mu) \end{aligned}$$

$$q_2 = (2\mu - b)\left(\frac{\beta I^* e^{-\mu\tau}}{I+\alpha I^*} - \frac{\beta S^* e^{-\mu\tau}}{(I+\alpha I^*)^2} + \frac{(\rho+\delta\beta I^* e^{-\mu\tau})}{I+\alpha I^*}\right)$$

$$P_3 = -\mu(b-\mu)(\mu+\rho+\delta)$$

$$q_3 = \mu(\mu+\rho+\delta) - b(\mu+\rho)\frac{\beta I^* e^{-\mu\tau}}{I+\alpha I^*} + \mu(b-\mu)\frac{\beta S^* e^{-\mu\tau}}{(I+\alpha I^*)^2}$$

Theorem 3. *If $R_0 > 1$, suppose $2\mu - b > 0$ and when $\tau = 0$, the endemic equilibrium E^* is stable, and when $\tau = 0$, it is unstable.*

Proof. Considering the case without $\tau = 0$, the characteristic equation would be given as: □

$$(\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0 \tag{6.7}$$

This implies that;

$$a_1 = p_1 + q_1 = (2\mu - b) + \frac{\alpha I^*(\mu+\rho+\delta)}{I+\alpha I^*} + \frac{\beta I^* e^{-\mu\tau}}{I+\alpha I^*}$$

$$a_2 = p_2 + q_2 = (2\mu - b)\frac{\alpha I^*(\mu+\rho+\delta)}{I+\alpha I^*} + \frac{\mu(b-\mu)(\mu+\rho+\delta)(\mu+\rho+\delta-b)}{\mu(\mu+\rho+\delta) - b(\mu+\rho)} + \frac{\mu^2 b(b-\mu) + b\mu\rho(b-\mu)}{\mu(\mu+\rho+\delta) - b(\mu+\rho)}$$

$$a_3 = p_3 + q_3 = \mu(b-\mu)(\mu+\rho+\delta)\frac{I}{I+\alpha I^*} > 0$$

By Ruth-Hurwitz criterion, when $\tau = 0$, the endemic equilibrium, $E^*$the system of differential equation is stable.

When $\tau = 0$, the system of differential equation has a purely imaginary root $\omega i(\omega > 0)$, then by separating real and imaginary parts, we have

$\omega^3 - \omega\rho_2 = \omega q_2 cos(\omega\tau) + (\omega^2 q_1 - q_3)sin(\omega\tau)$

$\omega^2\rho_1 - p_3 = \omega q_2 sin(\omega\tau) + (-\omega^2 q_1 + q_3)cos(\omega\tau)$

Hence,

$$\omega^6 + a_4\omega^4 + a_5\omega^2 + a_6 = 0 \tag{6.8}$$

where,

$$a_4 = p_1^2 - 2p^2 - q_1^2$$
$$a_5 = p_2^2 - 2p_1p_3 + 2q_1q_3 - q_2^2$$
$$a_6 = p_3^2 - q_3^2$$

Suppose,

$$f(\omega) = \omega^6 + a_4\omega^4 + a_5\omega^2 + a_6, \tag{6.9}$$

and let $\omega^2 = z$,

Then;

$$f(z) = z^3 + a_4z^2 + a_5z + a_6 \tag{6.10}$$

This implies that, $a_6 = p_3^2 - q_3^2 < 0$, then $f(0) < 0$ and $f(\infty) \to \infty$.

Thus, the equation, $f(z) = z^3 + a_4z^2 + a_5z + a_6$ has at least one positive root z_1.

Equation $\omega^6 + a_4\omega^4 + a_5\omega^2 + a_6 = 0$ has at least one positive root, denoted by;

$\omega_1 = \sqrt{z_1}$.

7. BIFURCATION ANALYSIS

By considering the delay, τ as a bifurcation parameter. Moreover, considering the equation;

$$f_1(\lambda) = (\lambda^3 + p_1\lambda^2 + p_2\lambda + p_3) + (q_1\lambda^2 + q_2\lambda + q_3)e^{-\lambda\tau} \tag{7.1}$$

as functions of the bifurcation parameter τ.

Let $\lambda(t) = y(\tau) + i\omega(\tau)$ be the eigen values of $f_1(\lambda) = (\lambda^3 + p_1\lambda^2 + p_2\lambda + p_3) + (q_1\lambda^2 + q_2\lambda + q_3)e^{-\lambda\tau}$.

Such that for some initial value of the bifurcation parameter τ_1, we have $y(\tau_1) = 0$, and $\omega(\tau_1) = \omega(\tau_1)$. Assuming $\omega_1 > 0$.

From;

$$\omega^3 - \omega\rho_2 = \omega q_2 cos(\omega\tau) + (\omega^2 q_1 - q_3)sin(\omega\tau) \tag{7.2}$$

and

$$\omega^2\rho_1 - p_3 = \omega q_2 sin(\omega\tau) + (-\omega^2 q_1 + q_3)cos(\omega\tau). \tag{7.3}$$

We have;

$$\tau_1 = \frac{1}{\omega_1} arcos\left(\frac{(p_1q_1 - q_2)\omega_1^4 + (p_2q_2 - p_3q_3)\omega_1^2}{q_2^2\omega_1^2 + (q_3 - q_1\omega_1^2)^2} + \frac{p_3q_3}{q_2^2\omega + (q_3 - q_1\omega_1^2)^2}\right) + 2j\pi.$$

Also, if $\frac{dRe\lambda(\tau)}{d(\tau)} > 0$. By continuity the real part of $\lambda(\tau)$ becomes positive when $\tau > \tau_1$ and the steady state becomes unstable.

A Hopf bifurcation occurs when τ passes through the critical value.

Considering the Delayed SIR Model when $\alpha = 0$, the standard incidence rate;

$$\beta S(t-\tau)I(t-\tau)e^{-\mu\tau}. \tag{7.4}$$

The model becomes:

$$\left.\begin{aligned} \frac{dS(t)}{dt} &= b(S(t)+I(t)+R(t)-\beta S(t-\tau)I(t-\tau)e^{-\mu\tau}-\mu S(t) \\ \frac{dI(t)}{dt} &= \beta S(t-\tau)I(t-\tau)e^{-\mu\tau}-(\mu+\rho+\delta)I(t), \\ \frac{dR(t)}{dt} &= \rho I(t)-\mu R(t) \end{aligned}\right\} \tag{7.5}$$

Theorem 4. *If, $R<1$, the solution of system (14) is $(S(t),I(t),R(t))\to(\infty,0,0)$ with $t\to\infty$. When $\bar{R}_0<1$, the system has a unique endemic equilibrium $P^*=(S^*,I^*,R^*)$.*

Where;

$$S^* = \frac{\mu(\mu+\rho+\delta)e^{-\mu\tau}}{\beta}$$

$$I^* = \frac{\mu(b-\mu)(\mu+\rho+\delta)e^{-\mu\tau}}{\beta\{\mu(\mu+\rho+\delta)-b(\mu+\rho)\}}$$

$$R^* = \frac{\rho(b-\mu)(\mu+\rho+\delta)e^{-\mu}}{\beta\{\mu(\mu+\rho+\delta)-b(\mu+\rho)\}}$$

Proof. When $t\to\infty$, consider the two cases: $I(t)=0$ and $I(t)>0$. If $I(t)=0$, it implies that $R(t)=0$.

From $\frac{dS(t)}{dt}=(b-\mu)S(t)$. When $t\to\infty$, we have $S(t)\to\infty$. Then, $(S(t),I(t),R(t))\to(\infty,0,0)$ with $t\to\infty$. If $I(t)=0$, from the system of differential equation,

$R^*=\frac{\rho I^*}{\mu}$.

Moreover, □

$$S^* = \frac{\mu(\mu+\rho+\delta)e^{-\mu\tau}}{\beta}$$

$$I^* = \frac{\mu(b-\mu)(\mu+\rho+\delta)e^{-\mu\tau}}{\beta\{\mu(\mu+\rho+\delta)-b(\mu+\rho)\}}$$

Then, we ensure that, $R^*\geq 0, I^*\geq 0, S^*\geq 0$.

We must have $\mu(\mu+\rho+\delta)-b(\mu+\rho)>0$.

This means there exists the endemic equilibrium with $R_0>1$.

Analysing the stability of the endemic equilibrium, P^* with $R_0>1$.

Theorem 5. *If $R_0>1$ and when $\tau=0$, the endemic equilibrium, P^* is stable, and when $\tau>0$, $P^*$is unstable.*

Proof. When $\tau=0$, the characteristic equation of system of differential equation becomes: □

$$\lambda^3+c_1\lambda^2+c_2\lambda+c_3=0 \tag{7.6}$$

This means, $c_1>0$,$c_2>0$ and $c_3>0$. By Ruth-Hurwitz criterion, the system is stable with $\tau=0$. When $\tau>0$, the system has a purely imaginary root $\omega i(\omega>0)$, then:

$$f(z)=z^3+c_4z^2+c_5z+c_6 \tag{7.7}$$

Where $c_6=p_3^2=q_3^2$. Then, $f^{'}(0)<0$ and $f^{'}(\infty)\to+\infty$.

Thus;

$$f(z)=z^3+c_4z^2+c_5z+c_6 \tag{7.8}$$

has at least one positive root z_2.

Also,

$$\frac{dRe\lambda(\tau)}{d\tau}>0 \tag{7.9}$$

A Hopf bifurcation occurs when τ passes through the critical value.

8. NUMERICAL SOLUTIONS

Numerical simulations was performed on the system of differential equation of the model parameters to see the dynamics of the population of susceptible, infectious and recovered in the system. This is done to see the how the population of the susceptible, infectious and the recovered change with time. The numerical simulations was done using Range-Kutta fourth order scheme. The following parameter values were taken from existing published data and others assumed for the numerical simulations;

8.1 Susceptible, Infectious and Recovered population

The numerical simulations the system in 1, support the claim in the theoretical or qualitative analysis of the model. The endemic equilibrium of the delayed epidemic model with the saturated incidence rate and the bi-linear incidence rate are locally asymptotically stable without delay.

Comparing the system of the standard incidence rate and that of the saturated incidence rate, it can be seen that the proportion of susceptible population is higher in the saturated incidence rate and the proportions of infectious and recovered are lower.

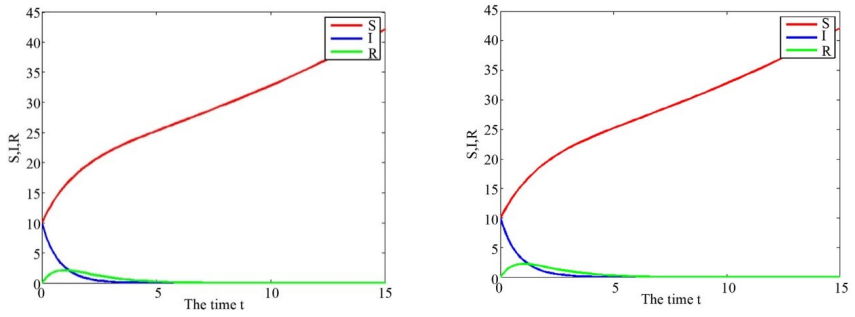

Figure 1: Population dynamics of Susceptible, Infectious and Recovery with time.

8.2 Population density of Susceptible, Infectious and Recovered at $\tau = 0$

The diagram in 2 of the delay model indicates that the endemic equilibrium point is locally asymptotically stable when $\tau = 0$ with both the saturated and standard incidence rate.

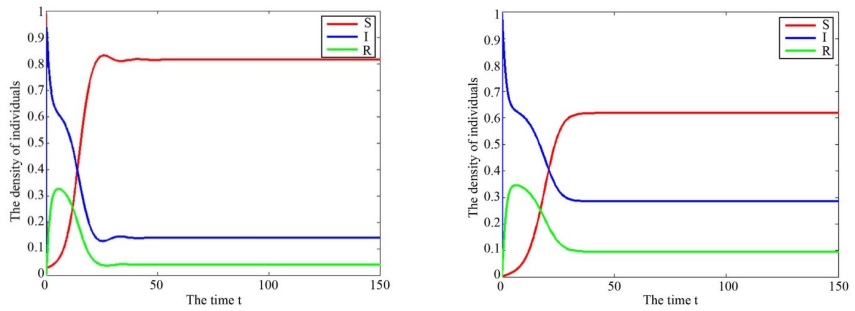

Figure 2: The endemic equilibrium is locally stable $\tau = 0$ with saturated and standard incidence rate.

8.3 Stable and unstable endemic equilibrium of SIR model

The diagram in 3showed that the endemic equilibrium of the delayed epidemic model is locally asymptotically stable when $\tau = 0.1$ and unstable when $\tau = 0.85$.

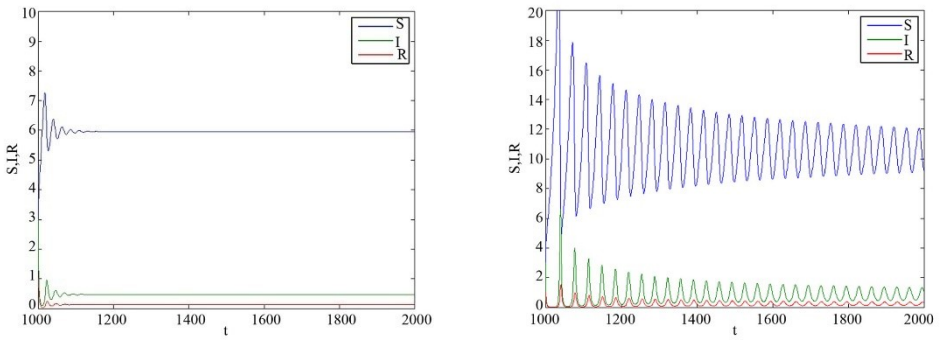

Figure 3: The endemic equilibrium is asymptotically stable with $\tau = 0.1$ and unstable with $\tau = 0.85$.

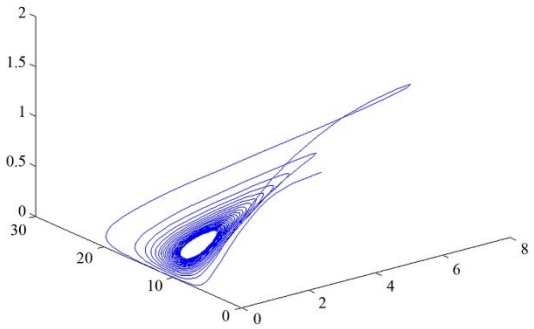

Figure 4: Endemic equilibrium stability of Susceptible, Infectious and Recovered.

8.4 Stable and unstable endemic equilibrium of the SIR model

Considering the diagram in 5, when $\tau = 0.1$, we can find some stability the dynamics of the susceptible, infectious and recovered populations. But looking at the second diagram in the same figure, we find that it is unstable when the value of $\tau = 0.85$. Moreover, the diagram in 6, the endemic equilibrium in the system of the standard incidence rate showed an existence of periodic solution.

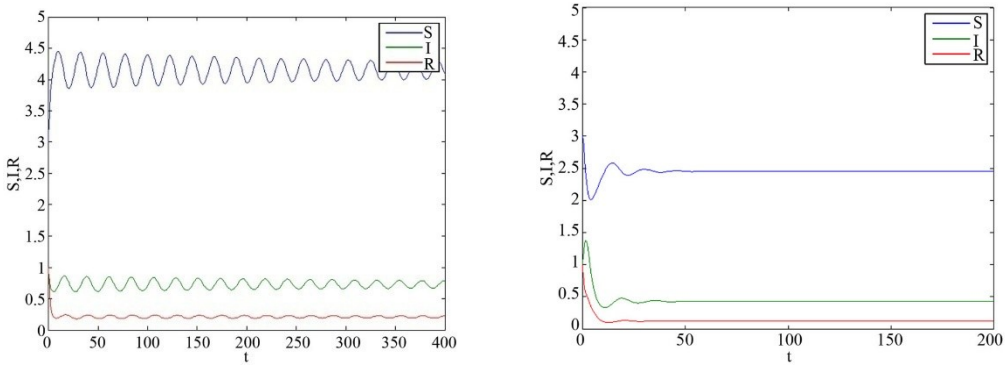

Figure 5: The endemic equilibrium is asymptotically stable with $\tau = 0.1$ and unstable with $\tau = 0.85$.

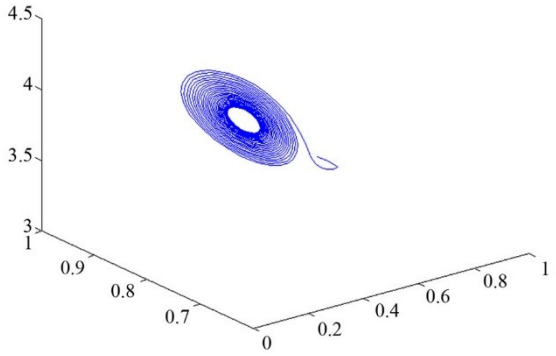

Figure 6: Existence of periodic solution at standard incidence rate.

9. CONCLUSION

The numerical simulations of the model showed that the endemic equilibrium point is locally asymptotically stable without time delay. The diagram 1in indicates that it is more effective to consider the inhibition effect of the population change of the infectious. This would reduce the infectious population. Moreover, the diagrams in 3 and 5 showed that the endemic equilibrium in system of the delay differential equation at saturated and standard incidence rate is locally asymptotically stable. It showed that endemic equilibrium of system is locally asymptotically stable whenever τ is suitably small. It also showed the existence of periodic solutions.

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