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Mathematical study of SEIR model with functional rates of incidence and treatment

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Abstract

In this paper, with nonlinear inhibitory effect and saturated treatment rate, an SEIR epidemic model is proposed. The basic reproduction number R_0, is calculated when determining the threshold value for the disease and the dynamics of the model. The criteria for the existence of all the points of equilibrium are established and we also found that the conditions depend on them. The stability of equilibrium is discussed in terms of local and global. All attempted were made to present the numerical simulations for the model we suggested. The theoretical findings are clearly predicted to be supported and evaluated.

Keywords

SEIR model, equilibrium point, stability analysis.

AMS Subject Classification

00A71.

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Contents

1. Introduction

A number of infectious diseases have been mapped, analyzed and applied to Different types of widespread models out, examined and applied to a variety of infectious diseases. All these have been carried out with qualitative and quantitative parameters. However, in the study of the widespread use and the control of infectious diseases, epidemic models are significant. In addition, these models are significant in policy making including optimizing multiple detection control-program evaluation. The bilinear incidence rate βSI is often implemented in many disease models [3,5,8,9]. Describe the fact that the over crowding of infected persons or susceptible persons saturates the amount of active encounters between infectious and susceptible persons.

A saturated incidence rate [1]

$$g(I)S = \frac{\beta IS}{1+\rho I}$$

where the positive constant ρ measures the inhibitory effect which explains the saturation effects or psychological effects [3,5,6] Further the treatment for a disease plays a key part in controlling or minimizing the spread of multiple of infectious disease forms. In general the treatment of a disease in any group or nation is limited. Therefore, the consideration of an effective treatment rate reflects a successful epidemic model. Wang and Ruan [13] were taken a constant removal rate into consideration in a classical SIR model. In this model they performed stability analysis and proposed several bifurcations. Subsequently Zhang and Suo [14] adapted the rate of treatment to Holling type II i.e.,

$$h(I) = \frac{\alpha I}{1+\delta I}$$

where $\alpha > 0, \delta \geq 0$, where α is the cure rate and δ measures the delay for treatment.

Although several literatures [1,2,4,5,6,7,8,10,13,14] have studied the roles of different types of epidemic models with a inhibitory and treatment rates, but less study has been done. The bilinear occurrence rate and treatment rate were considered by B. Dubey et al. [5] as type II and type III of Holling. The epidemic model under which both events occurred was considered by Zhang et al. [7,14]. Here we considered an epidemic model with Holling type II and III functional rates. Also assumpted that the immunity acquired by a person after recovery is permanent.

This paper is ordered in this manner: the mathematical model is formulated in the next section; the model's positivity and limits are evaluated. Section 3 addressed equilibrium and their existence. In addition, the very significant number in the disease modelling, i.e., the basic reproduction number is measured. In section 4 the local and global stability of equilibrium points was analysed. Section 5 is dedicated to promoting and complementing these numerical simulations.

2. The Mathematical model

The recovered individuals via treatment in the following considered SEIR model is assumed that they gained permanent immunity. As stated in the introduction the incidence rate and the treatment rate was taken to be Holling type–II and III respectively. To construct an SEIR model, we divide population into four subdivisions i.e. Susceptible (S), Exposed (E), Infectious (I) and Recovered (R). As follows the model to be analysed is:

$$\left.\begin{array}{ll} \frac{dS}{dt} & = b - \frac{\beta IS}{1+\rho I} - \mu S \\ \frac{dE}{dt} & = \frac{\beta IS}{1+\rho I} - (\sigma+\mu)E \\ \frac{dI}{dt} & = \sigma E - (\mu+\gamma)I - \frac{\alpha I^2}{1+\delta I^2} \\ \frac{dR}{dt} & = \gamma I - \mu R + \frac{\alpha I^2}{1+\delta I^2} \end{array}\right\} \tag{2.1}$$

Here b is the recruitment rate, ρ is inhibitory effect, β is the

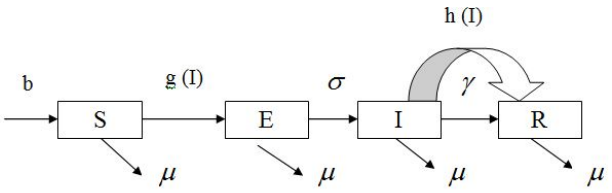

Figure 1. The flow chart of SEIR model

force of infection at which a susceptible is exposed and $\frac{1}{\beta}$ is the period of transmission. After an incubation period the exposed individual become infective and move into infective compartment. Let σ is the incubation period after which the exposed individuals to become infective individuals and the incubation period is $\frac{1}{\sigma}$. Let μ be the death rate and ρ be the inhibitory effect. Let γ be the rate of recovery of infective individuals and $\frac{1}{\gamma}$ is the infectious period. Let $h(I) = \frac{\alpha I^2}{1+\delta I^2}$ be the functional treatment rate through which the infective individuals can also be recovered. The parameters such as $b, \beta, \mu, \sigma, \gamma, \alpha$ are all positive and ρ and δ are nonnegative.

2.1 The boundedness of the model

Theorem 2.1. *The region*

$$\Omega = \left\{ (S,E,I,R) \in R_+^4 : N = S+E+I+R \leq \frac{b}{\mu} \right\}$$

is positively invariant for system (2.1).

Proof. Let $N(t) = S(t)+E(t)+I(t)+R(t)$, then

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt}.$$

From (2.1), $\frac{dN}{dt} \leq b - \mu N$. This implies that, $N(t) \leq \frac{b}{\mu}$, when $N(0) \leq \frac{b}{\mu}$. Thus Ω is positively invariant of the system (2.1). Further $N(0) > \frac{b}{\mu}$, then either of the solutions enters in to Ω in finite time or approaches to $\frac{b}{\mu}$ as $t \to \infty$. Hence the closed set Ω is captivating i.e., all solutions in R_+^4 eventually enters the region Ω and remains in Ω thereafter. □

3. Finding the equilibrium points and the basic reproduction number

This section discusses the conditions for existence of equilibrium points E_0 and E^* respectively. Furthermore, R_0 is calculated with the next generation matrix [4] method. The equilibrium points are obtained by solving the following system.

$$b - \frac{\beta IS}{1+\rho I} - \mu S = 0 \tag{3.1}$$

$$\frac{\beta IS}{1+\rho I} - (\sigma+\mu)E = 0 \tag{3.2}$$

$$\sigma E - (\mu+\gamma)I - \frac{\alpha I^2}{1+\delta I^2} = 0 \tag{3.3}$$

$$\gamma I - \mu R + \frac{\alpha I^2}{1+\delta I^2} = 0 \tag{3.4}$$

By solving (3.1), (3.2), (3.3) and (3.4), the disease-free equilibrium (DFE), $E_0 = \left(\frac{b}{\mu}, 0, 0, 0\right)$. With some algebra calculations $E^* = (S^*, E^*, I^*, R^*)$ becomes

$$S^* = \frac{b(1+\rho I^*)}{\mu + (\beta+\mu\rho)I^*} \tag{3.5}$$

$$E^* = \frac{\beta I^* b (1+\rho I^*)}{(\mu + (\beta+\mu\rho)I^*)(1+\rho I^*)(\mu+\sigma)} \tag{3.6}$$

$$R^* = \frac{I^*}{\mu}\left(\gamma + \frac{\alpha I^*}{1+\delta I^{*2}}\right) \tag{3.7}$$

where I^* is the positive solution of

$$AI^{*3}+BI^{*2}+CI^*+D=0, \tag{3.8}$$

here

$$\begin{aligned}A=&k\delta(\beta+\mu\rho)\\ B=&k\delta\mu+\alpha(\beta+\mu\rho)(\mu+\sigma)-\sigma\beta b\delta\\ C=&k(\beta+\mu\rho)+\mu\alpha(\mu+\sigma)\\ D=&k\mu-\sigma\beta b\end{aligned}$$

The solutions to (3.5) need to be real and positive. In order for the endemic equilibrium to exist. Here we first establish the basic reproduction number R_0, before going to find the existence of endemic equilibrium. Perhaps the most significant number in disease modeling is the basic reproduction number and it is described as the 'average number of cases directly arising from a typical primary case in an entirely susceptible population'.
The benefit of the method mentioned above to find R_0 is it involves DFE, which is simple to calculate. The DEF of the model (2.1) is $E_0=\left(\frac{b}{\mu},0,0,0\right)$. In (2.1) E and I are the diseased compartments. Therefore, the sub model of the model (2.1) contains the equations E and I, is

$$\frac{d\vec{x}}{dt}=F(\vec{x})-V(\vec{x}),\quad \vec{x}=\begin{bmatrix}E\\ I\end{bmatrix}$$

$$F(\vec{x})=\begin{bmatrix}\frac{\beta IS}{1+I}\\ 0\end{bmatrix},\quad V(\vec{x})=\begin{bmatrix}(\sigma+\mu)E\\ \left(\frac{\alpha I}{1+\delta I^2}+\gamma+\mu\right)I-\sigma E\end{bmatrix}$$

Now

$$J(F(E_0))=\begin{bmatrix}0&\frac{\beta B}{\mu}\\ 0&0\end{bmatrix},\quad J(V(E_0))=\begin{bmatrix}\sigma+\mu&0\\ -\sigma&\gamma+\mu\end{bmatrix}$$

$$V^{-1}=\begin{bmatrix}\frac{1}{\sigma+\mu}&0\\ \frac{\sigma}{(\sigma+\mu)(\gamma+\mu)}&\frac{1}{\gamma+\mu}\end{bmatrix}$$

$$FV^{-1}=\begin{bmatrix}\frac{\beta b\sigma}{\mu(\sigma+\mu)(\gamma+\mu)}&\frac{\beta b}{\mu(\gamma+\mu)}\\ 0&0\end{bmatrix}$$

The largest Eigen value of FV^{-1} is

$$\frac{\beta b\sigma}{\mu(\sigma+\mu)(\gamma+\mu)},$$

which is the spectral radius and it is equal to model's basic reproduction number. Consequently

$$R_0=\frac{\beta b\sigma}{\mu(\sigma+\mu)(\gamma+\mu)}.$$

4. Existence and stability analysis of equilibrium

Existence conditions of equilibrium points are discussed in this section by using R_0. It has also further discussed the global stability of this equilibrium.
The model (2.1) has unique DFE $E_0=\left(\frac{b}{\mu},0,0,0\right)$ which always exists. The solutions of (3.8) should be real and positive for $E^*=(S^*,E^*,I^*,R^*)$ to exist. We can determine the number of positive real roots of the cubic equation (3.8) according to Descartes rule of sign. We therefore have the findings that follow.

Theorem 4.1. *System (2.1) has endemic equilibrium points if the following results hold.*

1. *Unique endemic equilibrium when ever*

$$1<R_0<\frac{\alpha(\beta+\mu\rho)}{\mu\delta(\mu+\gamma)}.$$

2. *Two endemic equilibria whenever*

$$\frac{\alpha(\beta+\mu\rho)+\mu(\mu+\sigma)}{\mu\delta(\mu+\gamma)}<R_0<1.$$

3. *Three endemic equilibria when ever*

$$R_0>\frac{k\mu\delta+\alpha(\beta+\mu\rho)(\mu+\sigma)}{\sigma\beta b\delta}.$$

4. *No endemic equilibria when ever*

$$R_0>1+\frac{\alpha(\beta+\mu\rho)}{\mu\delta(\mu+\gamma)}\ \text{and}\ R_0<1.$$

Where,

$$R_0=\frac{\beta b\sigma}{\mu(\sigma+\mu)(\gamma+\mu)}$$

and $k=(\mu+\sigma)(\mu+\gamma)$.

Remark 4.2. *Here, even though the system has more than one endemic equilibrium, we concentrate on unique endemic equilibrium. The variational matrix of the system (2.1) is*

$$J=\begin{bmatrix}-\frac{\beta I}{1+\rho I}-\mu&0&-\frac{\beta S}{(1+\rho I)^2}&0\\ \frac{\beta I}{1+\rho I}&-\sigma-\mu&\frac{\beta S}{(1+\rho I)^2}&0\\ 0&\sigma&-\mu-\gamma-\frac{2\alpha I}{\left(1+\delta I^2\right)^2}&0\\ 0&0&\gamma+\frac{2I}{\left(1+\delta I^2\right)^2}&-\mu\end{bmatrix} \tag{4.1}$$

Theorem 4.3. *If* $R_0<1, E_0=\left(\frac{b}{\mu},0,0,0\right)$ *is asymptotically stable. Otherwise* E_0 *is unstable.*

Proof. The corresponding variational matrix at E_0

$$J(E_0)=\begin{bmatrix}-\mu&0&-\frac{\beta b}{\mu}&0\\ 0&-\sigma-\mu&\frac{\beta b}{\mu}&0\\ 0&\sigma&-\mu-\gamma&0\\ 0&0&\gamma&-\mu\end{bmatrix} \tag{4.2}$$

The characteristic equation of (4.2) is given by

$$(\lambda+\mu)^2\left(\lambda^2+K_1\lambda+K_2\right)=0, \tag{4.3}$$

where,

$$K_1=2\mu+\gamma+\sigma, K_2=(\mu+\gamma)(\mu+\sigma)-\frac{\beta b\sigma}{\mu}.$$

The characteristic values of matrix $J(E_0)$ are $\lambda_1=\lambda_2=-\mu$ and also the roots of quadratic equation By Descartes' rule of signs when, $(\mu+\gamma)(\mu+\sigma)>\frac{\beta b\sigma}{\mu}$ the quadratic equation has negative roots, This implies that,

$$R_0=\frac{\beta b\sigma}{\mu(\mu+\gamma)(\mu+\sigma)}<1.$$

□

Theorem 4.4. *When* $R_0<1, E_0=\left(\frac{b}{\mu},0,0,0\right)$ *is globally asymptotically stable.*

Proof. Consider the Lyapunov function on R^3_+

$$V(S,E,I)=l\left(S-S^*-S^*\ln\frac{S}{S^*}\right)+\frac{1}{(\mu+\sigma)}E+\frac{1}{\sigma}I. \tag{4.4}$$

Where $l>0$ is to be determined and $S^*=\frac{b}{\mu}$. Now

$$\begin{aligned}\frac{dV}{dt}=&l\left(1-\frac{S^*}{S}\right)\left(b-\frac{\beta IS}{1+\rho I}-\mu S\right)\\&+\frac{1}{(\mu+\sigma)}\left(\frac{\beta IS}{1+\rho I}-(\sigma+\mu)E\right)\\&+\frac{1}{\sigma}\left(\sigma E-(\mu+\gamma)I-\frac{\alpha I^2}{1+\delta I^2}\right)\end{aligned} \tag{4.5}$$

If $l=\frac{1}{\mu+\sigma}>0$, then (4.5) becomes,

$$\begin{aligned}\frac{dV}{dt}=&-lb\left[\frac{b}{\mu S}+\frac{\mu S}{b}-2\right]-\frac{\alpha I^2}{\sigma(1+\delta I^2)}\\&+\frac{\mu+\gamma}{\sigma}\left(\frac{R_0}{1+\rho I}-1\right)I\end{aligned} \tag{4.6}$$

$$\frac{dV}{dt}<0 \text{ for all } t\geq 0.$$

Therefore by Lyapunov stability theory, E_0 is globally asymptotically stable if $R_0<1$. Now,

$$J(E^*)=\begin{bmatrix}-\frac{\beta I^*}{1+\rho I^*}-\mu & 0 & -\frac{\beta S^*}{(1+\rho I^*)^2} & 0\\ \frac{\beta I^*}{1+\rho I^*} & -\sigma-\mu & \frac{\beta S^*}{(1+\rho I^*)^2} & 0\\ 0 & \sigma & -\mu-\gamma-\frac{2\alpha I^*}{(1+\delta I^{*2})^2} & 0\\ 0 & 0 & \gamma+\frac{2I^*}{(1+\delta I^{*2})^2} & -\mu\end{bmatrix} \tag{4.7}$$

$$(-\mu-\lambda)\left(a_1\lambda^3+a_2\lambda^2+a_3\right)=0, \tag{4.8}$$

which is the characteristic equation of (4.7). Where

$$\begin{aligned}a_1=&3\mu+\sigma+\gamma+\frac{2I^*\alpha}{(1+\delta I^{*2})^2}+\frac{\beta I^*}{1+\rho I^*}\\a_2=&(\sigma+\mu)\left(\mu+\gamma+\frac{2\alpha I^*}{(1+\delta I^{*2})^2}\right)\\&+\left(\mu+\frac{\beta I^*}{1+\rho I^*}\right)\left(2\mu+\sigma+\gamma+\frac{2I^*\alpha}{(1+\delta I^{*2})^2}\right)\\&-\frac{\beta S^*\sigma}{(1+\rho I^*)^2}\\a_3=&\left(\mu+\frac{\beta I^*}{1+\rho I^*}\right)(\sigma+\mu)\left(\mu+\gamma+\frac{2\alpha I^*}{(1+\delta I^{*2})^2}\right)\\&+\frac{\beta^2 S^*I^*}{(1+\rho I^*)^3}\\&-\left(\mu+\frac{\beta I^*}{1+\rho I^*}\right)\left(\frac{\beta S^*\sigma}{(1+\rho I^*)^2}\right)\end{aligned}$$

Clearly equation (4.8) has one eigenvalue i.e. $-\mu$ and if $a_1>0, a_3>0$ and $a_1a_2>a_3$. The eigenvalues of $a_1\lambda^3+a_2\lambda^2+a_3=0$ have negative real parts by Routh-Hurwitz criteria. □

Theorem 4.5. *If* $a_1>0, a_3>0$ *and* $a_1a_2>a_3$ *equilibrium point* $E^*=(S^*,E^*,I^*,R^*)$ *is locally asymptotically stable.*

Theorem 4.6. *When*

$$\frac{E}{I(1+\rho I^*)}<l_1<\frac{E}{S^*},$$

the stationary point E^* *is globally asymptotically stable.*

Proof. Considering the Lyapunov function on R^3_+

$$\begin{aligned}V(S,E,I)=&S-S^*-S^*\ln\left[\frac{S}{S^*}\right]+l_1\left\{E-E^*-E^*\ln\left[\frac{E}{E^*}\right]\right\}\\&+l_2\left\{I-I^*-I^*\ln\left[\frac{I}{I^*}\right]\right\}\end{aligned}$$

Where, $l_1, l_2>0$. Now

$$\frac{dV}{dt}=\left(\frac{S-S^*}{S}\right)\dot{S}+l_1\left(\frac{E-E^*}{E}\right)\dot{E}+l_2\left(\frac{I-I^*}{I}\right)\dot{I}. \tag{4.9}$$

By choosing

$$\frac{E}{I(1+\rho I^*)}<l_1<\frac{E}{S^*}$$

and

$$\begin{aligned}\frac{l_2}{l_1}<&\frac{IB}{\sigma EE^*(1+\rho I)(1+\rho I^*)}\\&[2(1+\rho I)I^*S^*-IE^*(1+\rho I^*)-E^*S^*].\end{aligned}$$

Then equation (4.9) becomes,

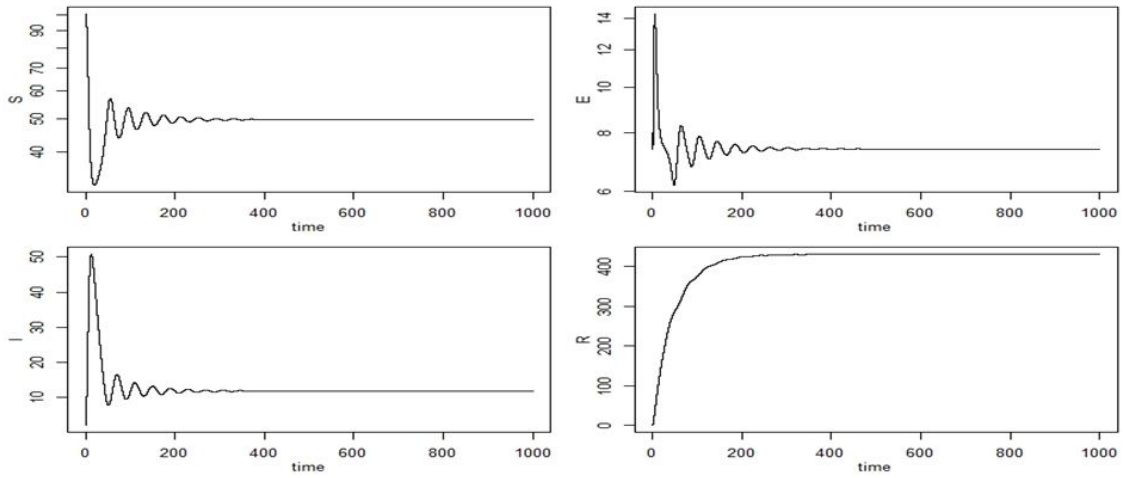

Figure 2. The population individuals approached to endemic equilibrium point $E^* = (49.64806, 7.382819, 11.71451, 431.2546)$

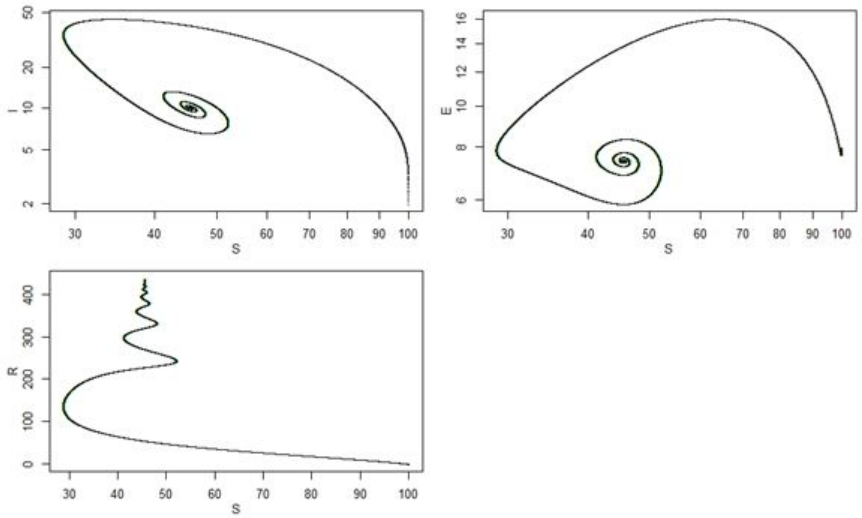

Figure 3. The phase portrait of Susceptible (S) vs. Invectives (I), Exposed (E) and Recovered (R) individuals

$$\begin{aligned}\frac{dV}{dt} &< -\left[\frac{b}{SS^*} - \frac{B}{2(1+\rho I)(1+\rho I^*)} + \frac{l_1 B(IE^* + \rho II^* E^*)}{2EE^*(1+\rho I)(1+\rho I^*)}\right](S-S^*)^2 \\ &- \left[\frac{l_1 B(IE^* + \rho II^* E^*)}{EE^*(1+\rho I)(1+\rho I^*)} - \frac{l_1 BE^* S^*}{2EE^*(1+\rho I)(1+\rho I^*)} - \frac{l_2\sigma}{2I} + \frac{l_1 B(I^* S^* + \rho II^* S^*)}{EE^*(1+\rho I)(1+\rho I^*)}\right](E-E^*)^2 \\ &+ \left[l_2\left(\frac{\sigma E^*}{II^*} + \frac{\alpha(1+\delta II^*)}{(1+\rho I^2)(1+\rho I^{*2})}\right) - \frac{l_1 BE^* S^*}{2EE^*(1+\rho I)(1+\rho I^*)} + \frac{B}{2(1+\rho I)(1+\rho I^*)} - \frac{l_2\sigma}{2I}\right](I-I^*)^2 \\ &< 0.\end{aligned}$$

Hence E^* is globally asymptotically stable. □

5. Numerical Illustrations

To explain theoretical findings outlined in this paper we will include some numerical simulations. The hypothetical set of parameter values is considered as follows for this reason.

$$\left.\begin{array}{ll} b & = 10, \beta = 0.03, \rho = 0.08, \mu = 0.02 \\ \sigma & = 1.2, \gamma = 0.004, \alpha = 0.5, \delta = 0.051 \\ S(0) & = 100, E(0) = 8, I(0) = 2, R(0) = 0. \end{array}\right\} \quad (5.1)$$

The above parameter values satisfies the first condition of theorem 4.1 hence unique E^* exists and it is

$$S^* = 49.64806, E^* = 7.382819, I^* = 11.71451, R^* = 431.2546.$$

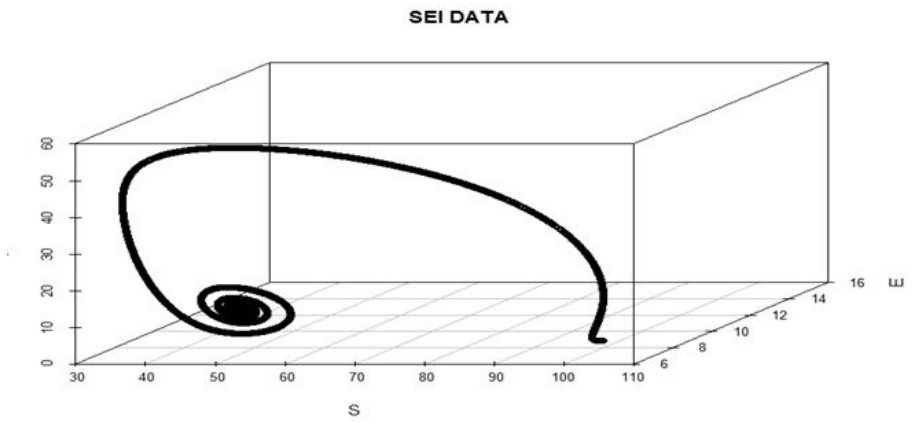

Figure 4. The phase portrait of Susceptible (S), Exposed (E), Invectives (I) individuals

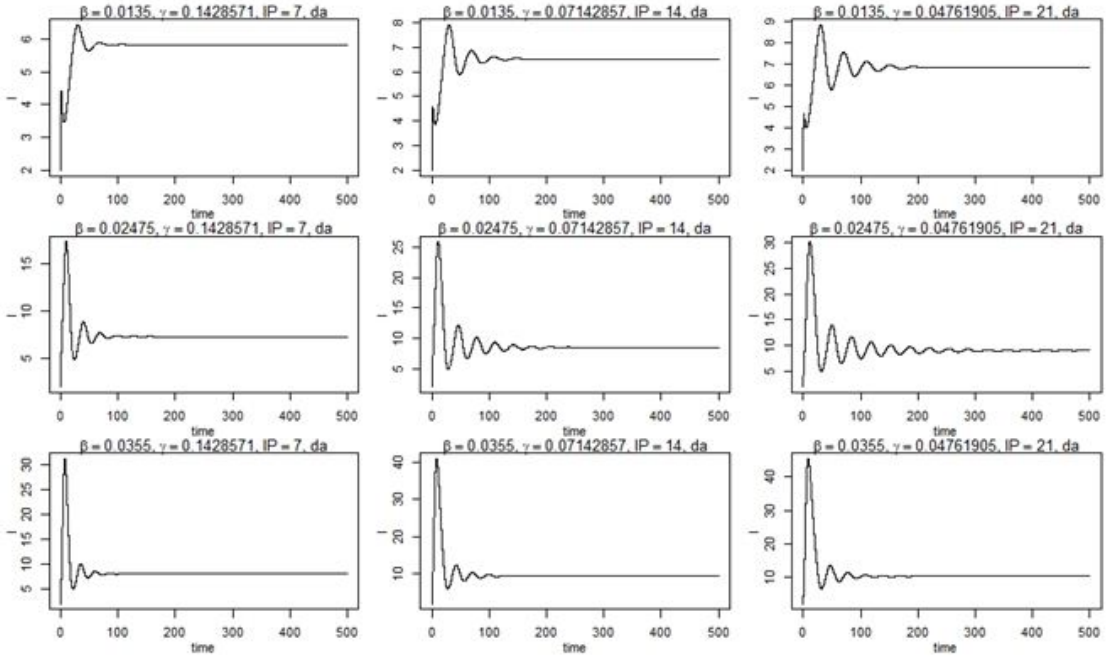

Figure 5. The effect of epidemic curve by varying the transmission rate β and the infection period $\frac{1}{\gamma}$

In addition, the above parameters values met the conditions in theorem 4.5. This reflects the endemic equilibrium point is locally asymptotically stable. The dynamics of the model is shown in the above Fig. 2. This is obtained from computer simulations using *R* programming [11] to do this.

6. Conclusion

The SEIR an epidemic model is considered in this paper, with functional rates of incidence and treatment as Holling type-II and III. The number R_0 is calculated. The point E_0 is asymptotiically locally and globally stable when $R_0 < 1$ and unstable if $R_0 > 1$. A unique E^* exits when $1 < R_0 < \frac{\alpha(\beta+\mu\rho)}{\mu\delta(\mu+\gamma)}$ and it is stable locally globally from theorems 4.5 and 4.6. The complex behavior of interacting populations is studied by using computer simulation. The transmission rate has been observed to increases, with disease become more endemic. The population should make an effort to reduce the rate of transmission in order to prevent the spread of the disease. It is noted in Fig 6 that the infectives are very high when the treatment is very poor and infectives decrease when the treatment is increasing. It is noticed that for increase of the susceptible and recovered individuals, by increase of saturation term for the infectives. This indicates that the idea of saturation has a beneficial impact on disease eradication.

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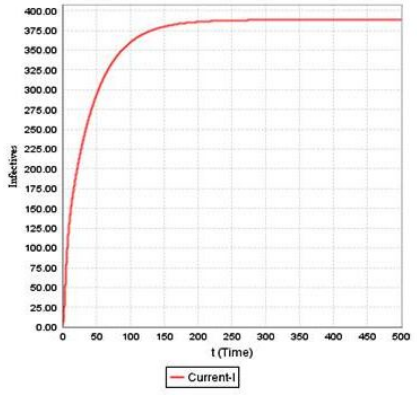
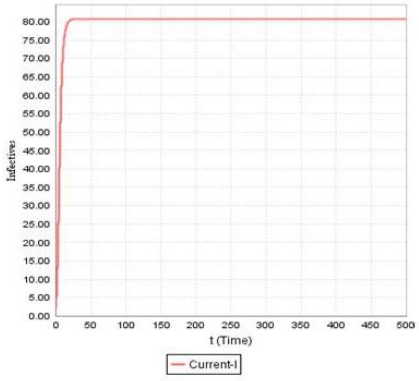


(a)



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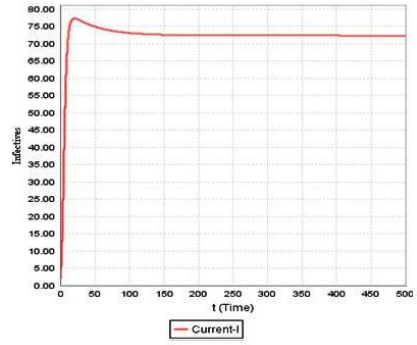
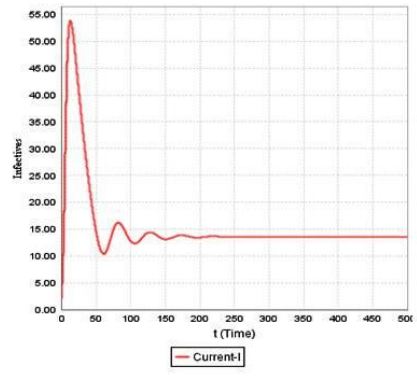
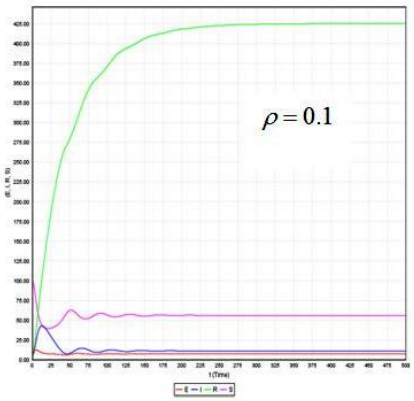


(c)

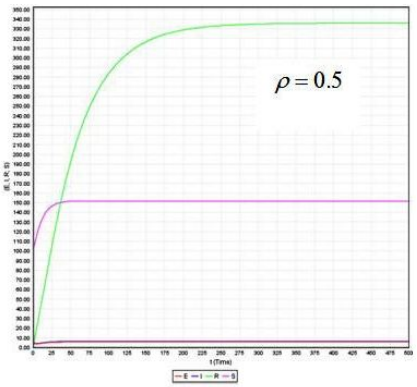


(d)

Figure 6. Behavior of invectives without medical resources (a) and with various levels of medical resources $((b),(c)$ and $(d))$.



(a)



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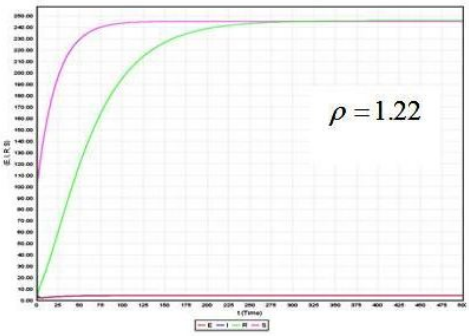

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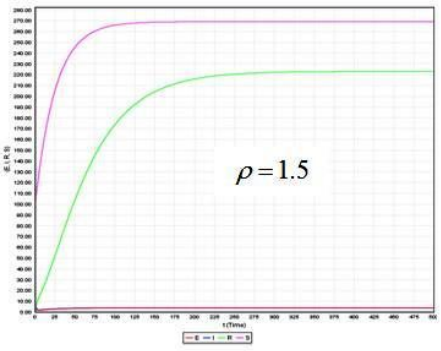

(d)

Figure 7. (a), (b), (c) and (d) show the inhibitory effect for the susceptible and infective individuals.

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