

Analysis of a Seirs Epidemic Model with Saturated Incidence Rate Considering the Initial State of the Diseases

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Abstract

In this work, a Susceptible-Exposed-Infected-Recovered-Susceptible (SEIR) epidemic model with saturated incidence rate is investigated. We established the disease-free equilibrium and endemic equilibrium states of the model and analyses for the local and global stabilities of the disease free equilibrium using matrix and Lyapunov function methods respectively when the basic reproduction number, $R_0 < 1$ were also studied. We proved that when $R_0 > 1$, the endemic state is locally asymptotically stable. The studied can be viewed as an extension of the work of Kuniya and Nakata to include saturated incidence rate. The effect of initial state of the disease was also studied. At the end, initial state of the disease knowledge play a vital role in disease eradication. Some numerical results were presented to compare our results with existing results'

Keywords: Reproductive Number, SEIRS, Saturated Incidence Rate

1.0 Introduction

The research in infectious diseases can be basically classified as descriptive, analytic, experimental and theoretic. Epidemic dynamics study is an important theoretic approach to investigate the transmission dynamics of infectious diseases. The mathematical models formulated are based on population dynamics, behaviour of disease transmissions, features of the infectious agents and the connections with other social and physiologic factors. Through quantitative and qualitative analysis, sensitivity analysis, and numeric simulations, mathematical models can give us good understanding of how infectious diseases spread, discover general principles governing the transmission dynamics of the diseases and identify more importance and sensitive parameters to make reliable predictions and provide useful control strategies and guidance. Several authors have worked in this area Liu [1] discussed dynamical behavior of epidemiological models with nonlinear incidence rates. Greenhalgh [2] considered SEIR models that incorporate density dependence in the death rate. Hethcote and Tudor [3] studied endemic infectious disease models for which infection conferred permanent immunity with no disease-related mortality but with vaccination. The infectious period had a general distribution

Cooke and van den Driessche [4] studied bifurcations in models of the SEIR type with density dependent contact rate and death rate with delays. Li and Muldowney [5] and Liu et al. [6] studied the global dynamics of the SEIR models with a non-linear incidence rate and with a standard incidence, respectively.

Li et al. [7] analyzed the global dynamics of a SEIR model with vertical transmission and a bilinear incidence. Zhang et al. [8] considered SEIR with saturating contact rate. In [9], Korobeinikov considers the global properties for SEIR and SEIS by means of Lyapunov functions. Greenhalgh [10], Li and Jin [11] considered the global stability of the SEI and SEIR model with infectious force in latent and infected period with non permanent immunity.

Hethcote [12] discussed disease transmission models with density-dependent demographics. They considered SIS and SIRS models with a standard incidence $\frac{\lambda SI}{N}$, where N is the total number of individuals. Greenhalgh [13] considered an SIR

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model with vaccination at birth and a transmission term $\frac{\beta(N)SI}{N}$. Greenhalgh [14] considered some SEIRS epidemiological models with vaccination and temporary immunity. He assumed that the average duration of immunity exceeded the infectious period and proved that there was a threshold parameter R_0 which determined the dynamics of system. Li, Graef et.al, [15] studied a SEIR model for the transmission of an infectious disease that spreads in a population through direct contact of the hosts. The force of infection is of proportionate mixing type.

For the case $\beta(N) - \beta$, they proved global stability results related to the results of Brauef and Driessche [16]. Classical epidemic models assume a transmission term of the form βSI . This implies that the contact rate for a single individual is βN , linearly proportional to the number of individuals in the population. An alternative assumption is to take a transmission term $\frac{\beta(N)SI}{N}$ which is nearer to models discussed by Anderson et. al, [17] for AIDS. This implies that the contact rate for a single individual is β , a constant, which is more suitable for sexually transmitted diseases.

In a recent paper, Kuniya and Nakata [18] studied the long time behavior of a nonautonomous SEIRS epidemic model. They obtained new sufficient conditions for the permanence (uniform persistence) and extinction of infectious population of the model. In this paper, we extend the work done by Kuniya and Nakata [18] to include saturated incidence rate. We present our result in form of basic reproduction number and theorems are used to prove the local and global stabilities of the disease free equilibrium. By numerical simulation, we study the effects of initial state of the diseases

2.0 Mathematical Formulation

A population of size $N(t)$ is partitioned into subclasses of individuals who are susceptible, exposed (infected but not yet infectious) infectious and recovered with sizes denoted by $S(t)$, $E(t)$, $I(t)$ and $R(t)$ respectively. The sum $E(t) + I(t)$ is the total infected population. It is assumed that all immigrant individuals are susceptible and vertical transmission can be assumed to acquire temporary immunity in which recovered individual goes back to the susceptible class again.

Kuniya and Nakata (2012) considered a non autonomous SEIRS epidemic model as below:

$$\left. \begin{aligned} \frac{dS(t)}{dt} &= \Lambda - \beta SI - \mu S + \delta R \\ \frac{dE(t)}{dt} &= \beta SI - (\mu + \varepsilon)E \\ \frac{dI(t)}{dt} &= \varepsilon E - (\mu + r)I \\ \frac{dR(t)}{dt} &= rI - (\mu + \delta)R \end{aligned} \right\} \tag{1}$$

The parameter $\Lambda(t) > 0$ is the birth rate, $\beta(t) > 0$ is the disease transmission coefficient, $\mu(t) > 0$ is the mortality/death rate, $\varepsilon(t) > 0$ is the rate of developing infectivity, $\gamma(t) > 0$ is the recovery rate, $\delta(t) > 0$ is the rate of losing immunity where $m > 0$ is the saturation is rate and $I_0(t) > 0$ is the initial infectious state of the disease. All other parameters are as defined in the model of Kuniya and Nakata (2012)

The following differential equations are solved based on the basis assumptions and we have our new model as stated below in equation (2)

$$\left. \begin{aligned} \frac{dS}{dt} &= \Lambda - \frac{\beta SI}{1 + mI_0} - \mu S + \delta R \\ \frac{dE}{dt} &= \frac{\beta SI}{1 + mI_0} - (\mu + \varepsilon)E \\ \frac{dI}{dt} &= \varepsilon E - (\mu + \gamma)I \\ \frac{dR}{dt} &= \gamma I - (\mu + \delta)R \end{aligned} \right\} \tag{2}$$

with initial value $S(0) > 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0$

3.0 Derivation of R_0 Using the Next Generation Matrix

Let G be a next generation matrix. It comprises of two parts F and V^{-1} where

$$F_i = \left[\frac{\partial f_i(x_0)}{\partial x_j} \right] \tag{3}$$

$$V_i = \left[\frac{\partial V_i(x_0)}{\partial x_j} \right] \tag{4}$$

F_i is the new infections, while the V_i transfers of infections from one compartment to another. X_0 is the disease free equilibrium state.

R_0 is the dominant Eigenvalue of the matrix

$$G = FV^{-1} \tag{5}$$

Though there are two disease states but only one way to create new infections. Hence, we are concerned with E and I compartment of the model. Thus;

$$\frac{dE}{dt} = \frac{\beta SI}{1 + mI_0} - (\mu + \varepsilon)E \tag{6}$$

$$\frac{dI}{dt} = \varepsilon E - (\mu + \gamma)I$$

From which we obtain:

$$G = FV^{-1} = \begin{pmatrix} \frac{\varepsilon\beta\Lambda}{\mu(\mu + \gamma)(\mu + \varepsilon)(1 + mI_0)} & \frac{\beta\Lambda}{\mu(\mu + \gamma)(1 + mI_0)} \\ 0 & 0 \end{pmatrix} \tag{7}$$

Clearly, It is easy to see that the dominant eigenvalue gives the R_0

$$\therefore R_0 = \frac{\varepsilon\beta\Lambda}{\mu(\mu + \gamma)(\mu + \varepsilon)(1 + mI_0)} \tag{8}$$

4.0 Method of Solution

Equilibrium / Critical Points: Let

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0 \dots \text{ to obtain the equations below} \tag{9}$$

$$\Lambda - \frac{\beta SI}{1 + mI_0} - \mu S + \delta R = 0$$

$$\frac{\beta SI}{1 + mI_0} - (\mu + \varepsilon)E = 0. \tag{10}$$

$$\varepsilon E - (\mu + \gamma)I = 0.$$

$$rI - (\mu + \delta)R = 0.$$

Solving equations (10) simultaneously for S, E,I,R respectively yields two equilibrium points namely

$$(S_1, E_1, I_1, R_1) = \left(\frac{\lambda}{\mu}, 0, 0, 0 \right) \text{ and}$$

$$S^* = \frac{\mu^2 + \gamma\mu + \mu^2 mI_0(t) + \mu mI_0(t)\gamma + \varepsilon\mu + \varepsilon\gamma + \varepsilon mI_0(t)\mu + \varepsilon mI_0(t)\gamma}{\beta\varepsilon},$$

$$E^* = -\frac{1}{(\varepsilon\gamma + \gamma\delta + \mu^2 + \gamma\mu + \varepsilon\mu + \mu\delta + \varepsilon\delta)} ((\mu^3 mI_0(t) + \varepsilon mI_0(t)\gamma\mu) / ((\varepsilon\gamma + \gamma\delta + \mu^2 + \gamma\mu + \varepsilon\mu + \mu\delta + \varepsilon\delta)\beta\mu\varepsilon)) \tag{11}$$

$$I^* = -\frac{1}{(\varepsilon\gamma + \gamma\delta + \mu^2 + \gamma\mu + \varepsilon\mu + \mu\delta + \varepsilon\delta)\beta\mu} ((\mu + \delta)(\mu^3 mI_0(t) / ((\varepsilon\gamma + \gamma\delta + \mu^2 + \gamma\mu + \varepsilon\mu + \mu\delta + \varepsilon\delta)\beta\mu))$$

$$R^* = -\frac{1}{(\varepsilon\gamma + \gamma\delta + \mu^2 + \gamma\mu + \varepsilon\mu + \mu\delta + \varepsilon\delta)\beta\mu} ((\mu^3 mI_0(t) + \varepsilon mI_0(t)\gamma\mu + \mu^2 mI_0(t)\gamma + \varepsilon mI_0(t)\mu^2 + \gamma\mu^2 - \wedge\beta\varepsilon + \varepsilon\gamma\mu + \mu^3 + \varepsilon\mu^2)\gamma))$$

where

(S_1, E_1, I_1, R_1) and (S^*, E^*, I^*, R^*) are the infection free and infection equilibrium respectively.

5.0 Local Stability of the Disease-Free Equilibrium

We shall now linearize the system of the equations in (2) as follows

Let $S - S_1 = x, E = E, I = I, R = R$

$$\therefore S = x + S_0 \Rightarrow \frac{dS}{dt} = \frac{dx}{dt}$$

So the system (2) can now be written as

$$\left(\begin{aligned} \frac{dx}{dt} &= \Lambda - \frac{\beta XI}{1 + mI_0} - \frac{\beta S_1 I}{1 + mI_0} - \mu x + \delta R \\ \frac{dE}{dt} &= \frac{\beta XI}{1 + mI_0} + \frac{\beta S_1 I}{1 + mI_0} - (\mu + \varepsilon)E \\ \frac{dI}{dt} &= \varepsilon E - (\mu + \gamma)I \\ \frac{dR}{dt} &= \gamma I - (\mu + \delta)R \end{aligned} \right) \tag{12}$$

By linearization, we have

$$\left(\begin{aligned} \frac{dx}{dt} &= -\frac{\beta S_1 I}{1 + mI_0} - \mu x + \delta R + \text{nonlinear terms} \\ \frac{dE}{dt} &= \frac{\beta S_1 I}{1 + mI_0} - (\mu + \varepsilon)E + \text{nonlinear terms} \\ \frac{dI}{dt} &= \varepsilon E - (\mu + \gamma)I \\ \frac{dR}{dt} &= \gamma I - (\mu + \delta)R \end{aligned} \right) \tag{13}$$

The resulting Jacobian matrix is

$$\begin{pmatrix} \dot{x} \\ \dot{E} \\ \dot{I} \\ \dot{R} \end{pmatrix} = \begin{pmatrix} -\mu & 0 & \frac{-\beta S_1}{1 + mI_0} & \delta \\ 0 & -(\mu + \varepsilon) & \frac{\beta S_1}{1 + mI_0} & 0 \\ 0 & \varepsilon & -(\mu + \gamma) & 0 \\ 0 & 0 & \gamma & -(\mu + \delta) \end{pmatrix} \begin{pmatrix} x \\ E \\ I \\ R \end{pmatrix} + \text{nonlinear terms} \tag{14}$$

Let

$$A = \begin{pmatrix} -\mu & 0 & \frac{-\beta\Lambda}{\mu(1+mI_0)} & \delta \\ 0 & -(\mu+\varepsilon) & \frac{\beta\Lambda}{\mu(1+mI_0)} & 0 \\ 0 & \varepsilon & -(\mu+\gamma) & 0 \\ 0 & 0 & \gamma & -(\mu+\delta) \end{pmatrix} \tag{15}$$

The characteristic equation is $|A - \lambda| = 0$ is written as

$$\begin{pmatrix} -\mu-\lambda & 0 & \frac{-\beta\Lambda}{\mu(1+mI_0)} & \delta \\ 0 & -(\mu+\varepsilon)-\lambda & \frac{\beta\Lambda}{\mu(1+mI_0)} & 0 \\ 0 & \varepsilon & -(\mu+\gamma)-\lambda & 0 \\ 0 & 0 & \gamma & -(\mu+\delta)-\lambda \end{pmatrix} = 0 \tag{16}$$

By solving the equation (16) we have,

$$\lambda_1 = -\mu, \lambda_2 = -(\mu+\delta), \lambda_3 = -\mu - \frac{\varepsilon}{2} - \frac{\gamma}{2} + \frac{1}{2}D, \lambda_4 = -\mu - \frac{\varepsilon}{2} - \frac{\gamma}{2} - \frac{1}{2}D \tag{17}$$

Where

$$D = \sqrt{\varepsilon^2 - 2\gamma\varepsilon + \gamma^2 + 4\mu\gamma^2R_0 + 4\mu\varepsilon R_0 + 4\gamma\mu R_0 + 4\gamma\varepsilon R_0} \tag{18}$$

Theorem 1: If $R_0 < 1$, the disease free equilibrium is locally asymptotically stable; if $R_0 = 1$, the disease free equilibrium is stable ; if $R_0 > 1$, the disease free is unstable.

Proof:

Now Since $\mu > 0, \varepsilon > 0, \gamma > 0, \delta > 0$, and if $R_0 < 1$, it follows that $\lambda_1, \lambda_2, \lambda_3$ and λ_4 are all negative, hence the disease – free equilibrium is asymptotically stable.

Equation (16) becomes

$$\begin{aligned} &(\mu + \lambda) ((\mu + \delta) + \lambda) [\lambda^2 + (2\mu + \varepsilon + \gamma)\lambda] = 0 \\ \Rightarrow &\lambda_1 = -\mu, \lambda_2 = -(\mu + \delta) \text{ and } \lambda^2 + (2\mu + \varepsilon + \gamma)\lambda = 0 \\ &\lambda_3 = 0, \text{ and } \lambda_4 = -(2\mu + \varepsilon + \gamma) \end{aligned} \tag{19}$$

Since $\lambda_3 = 0$, meaning all the eigenvalues are not all negative, the result follows immediately that the disease-free equilibrium is unstable when $R_0 = 1$

Also if $\mu > 0, \varepsilon > 0, \gamma > 0, \delta > 0$ and $R_0 > 1$, then from equation (19)

$$(\mu + \lambda) (\mu + \delta) + \lambda \left[\lambda^2 + (2\mu + \varepsilon + \gamma)\lambda + (\mu + \gamma)(\mu + \varepsilon)(1 - R_0) \right] = 0 \tag{20}$$

It follows that

$$\lambda_1 = -\mu, \lambda_2 = -(\mu + \delta)$$

We now apply Descartes' rule of signs $\lambda^2 + (2\mu + \varepsilon + \gamma)\lambda + (\mu + \gamma)(\mu + \varepsilon)(1 - R_0) = 0$

So we have:

$$\lambda^2 + (2\mu + \varepsilon + \gamma)\lambda + (\mu + \gamma)(\mu + \varepsilon)(1 - R_0) = 0 \tag{21}$$

If $\mu > 0, \varepsilon > 0, \gamma > 0$ and $R_0 > 1$

Clearly, there is only one sign change, which implies that we have at least one positive root. That is, not all eigenvalues are negative.

Also if we replace λ by $(-\lambda)$ and if $R_0 > 1, \mu > 0, \varepsilon > 0, \gamma > 0$ in (21) we have

$$\lambda^2 - (2\mu + \varepsilon + \gamma)\lambda - (\mu + \gamma)(\mu + \varepsilon)(R_0 - 1) = 0 \tag{22}$$

Again there is exactly one sign change, which implies that one of the Eigen values is positive and so not all Eigen values are negative, hence the disease free equilibrium $(S_1, E_1, I_1, R_1) = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$ is unstable.

6.0 Global Stability of the Disease Free Equilibrium

We consider the Lyapunov function defined thus

$$L = (\mu + \varepsilon)I + \varepsilon E \tag{23}$$

$$L^1 = (\mu + \varepsilon)I^1 + \varepsilon E^1$$

$$\begin{aligned} &= I \left(\frac{\varepsilon\beta S}{1+mI_0} - (\mu + \varepsilon)(\mu + \gamma) \right) \\ &= I(\mu + \gamma)(\mu + \varepsilon)[R_0 - 1] \end{aligned} \tag{24}$$

If $R_0 \leq 1$

$$L^1 \leq 0$$

Hence the disease free equilibrium is globally asymptotically stable.

7.0 Local Stability of the Disease Equilibrium

We let, $S - S_1^* = w, E - E_1^* = x, I - I_1^* = y, R - R_1^* = z$ (25)

$$\begin{aligned} \frac{dw}{dt} &= \Lambda - \frac{\beta wy}{1+mI_0} - \frac{\beta w I_1^*}{1+mI_0} - \frac{\beta S_1^* y}{1+mI_0} - \frac{\beta S_1^* I_1^*}{1+mI_0} - \mu w - \mu S_1^* + \delta z + \delta R_1^* \\ \frac{dx}{dt} &= \frac{\beta wy}{1+mI_0} + \frac{\beta w I_1^*}{1+mI_0} + \frac{\beta S_1^* I_1^*}{1+mI_0} + \frac{\beta S_1^* y}{1+mI_0} - (\mu + \varepsilon)x - (\mu + \varepsilon)E_1^* \\ \frac{dy}{dt} &= \varepsilon x + \varepsilon E_1^* - (\mu + \gamma)y - (\mu + \gamma)I_1^* \\ \frac{dz}{dt} &= \gamma y + \gamma I_1^* - (\mu + \delta)z - (\mu + \delta)R_1^* \end{aligned} \tag{26}$$

It follows by linearization that (26) can be written in term of its linear and non-linear parts as

$$\begin{pmatrix} \dot{w} \\ \dot{x} \\ \dot{y} \\ \dot{z} \end{pmatrix} = \begin{pmatrix} \frac{-\beta I_1^*}{1+mI_0} - \mu & 0 & \frac{-\beta S_1^*}{1+mI_0} & \delta \\ \frac{\beta I_1^*}{1+mI_0} & -(\mu + \varepsilon) & \frac{\beta S_1^*}{1+mI_0} & 0 \\ 0 & \varepsilon & -(\mu + \gamma) & 0 \\ 0 & 0 & \gamma & -(\mu + \delta) \end{pmatrix} \begin{pmatrix} w \\ x \\ y \\ z \end{pmatrix} + \text{Non-linear parts} \tag{27}$$

Where $\dot{w} = \frac{dw}{dt}, \dot{x} = \frac{dx}{dt}, \dot{y} = \frac{dy}{dt}, \dot{z} = \frac{dz}{dt}$ (28)

Let

$$A = \begin{pmatrix} \frac{-\beta I_1^*}{1+mI_0} - \mu & 0 & \frac{-\beta S_1^*}{1+mI_0} & \delta \\ \frac{\beta I_1^*}{1+mI_0} & -(\mu + \varepsilon) & \frac{\beta S_1^*}{1+mI_0} & 0 \\ 0 & \varepsilon & -(\mu + \gamma) & 0 \\ 0 & 0 & \gamma & -(\mu + \delta) \end{pmatrix} \tag{29}$$

The characteristics equations is given by

$$|A - \lambda| = 0$$

$$\begin{vmatrix} -\left(\frac{\beta I_1^*}{1+mI_0} + \mu\right) - \lambda & 0 & \frac{-\beta S_1^*}{1+mI_0} & \delta \\ \frac{\beta I_1^*}{1+mI_0} & -(\mu + \varepsilon) - \lambda & \frac{\beta S_1^*}{1+mI_0} & 0 \\ 0 & \varepsilon & -(\mu + \gamma) - \lambda & 0 \\ 0 & 0 & \gamma & -(\mu + \delta) - \lambda \end{vmatrix} = 0 \tag{30}$$

We now use theorem the stated theorem to analyze the nature of the roots of equation (30) as follows:

If $\gamma > 0, \mu > 0, \beta > 0, \varepsilon > 0, \delta > 0, I_0 > 0, I_1^* > 0, S_1^* > 0$ then there are no sign change in equation, which implies that there are no positive roots of (30) i.e all eigenvalues are negative. Also ,if we replace λ by $(-\lambda)$ in equation (30) we obtain

$$\begin{aligned} \lambda^4 - \left(\gamma + 2\varepsilon + 4\mu + \frac{\beta S_1^*}{1+mI_0}\right)\lambda^3 + \left(\frac{2\varepsilon\gamma + 6\mu\varepsilon + 3\mu\gamma + \frac{3\beta S_1^* \mu}{1+mI_0} + \frac{\beta S_1^* \gamma}{1+mI_0}}{1+mI_0} + \frac{2\beta S_1^* \varepsilon}{1+mI_0} + 6\mu^2 + \varepsilon^2 + \frac{\beta S_1^*}{1+mI_0} \right)\lambda^2 - \\ \left(\frac{3\beta S_1^* \mu^2}{1+mI_0} + 3\mu^2\gamma + 2\mu\varepsilon^2 + \varepsilon^2\gamma + 6\mu^2\varepsilon + \frac{4\beta S_1^* \mu\varepsilon}{1+mI_0} + \frac{2\beta S_1^* \mu\gamma}{1+mI_0} + \frac{2\varepsilon\beta S_1^*}{1+mI_0} + \frac{2\mu\beta S_1^*}{1+mI_0} + \frac{\beta S_1^* \varepsilon^2}{1+mI_0} + \frac{2\beta S_1^* \varepsilon\gamma}{1+mI_0} + 4\mu^3 + \frac{\beta^2 S_1^{*2}}{(1+mI_0)^2} + 4\mu\varepsilon\gamma \right)\lambda + \\ \left(\frac{2\beta S_1^* \varepsilon\mu^2}{1+mI_0} + \frac{\beta S_1^* \mu^2\gamma}{1+mI_0} + \mu\varepsilon^2\gamma + 2\mu^2\varepsilon\gamma + \frac{2\beta S_1^* \mu\varepsilon\gamma}{1+mI_0} + \frac{\beta S_1^* \varepsilon^2\mu}{1+mI_0} + \frac{2\mu\varepsilon\beta S_1^*}{1+mI_0} + \frac{\beta S_1^* \varepsilon^2\gamma}{1+mI_0} + \varepsilon^2\mu^2 + 2\varepsilon\mu^3 + \mu^3\gamma + \frac{\beta S_1^* \mu^3}{1+mI_0} + \frac{\beta^2 S_1^{*2}\varepsilon}{1+mI_0} + \frac{\beta^2 S_1^{*2}\mu}{1+mI_0} + \frac{\mu^2\beta S_1^*}{1+mI_0} - \varepsilon\delta + \mu^4 + \frac{\varepsilon\beta S_1^* \delta}{1+mI_0} \right) \end{aligned} = 0 \tag{31}$$

If $\gamma > 0, \mu > 0, \beta > 0, \varepsilon > 0, \delta > 0, I_0 > 0, I_1^* > 0, S_1^* > 0$, then equation (31) has 4 sign changes i.e. there are 4 negative roots or 2 negative roots. This implies that all eigenvalues are negatives, hence, the disease equilibrium is asymptotically stable.

8.0 Numerical Simulation

The result of the numerical simulations of the models are given below:

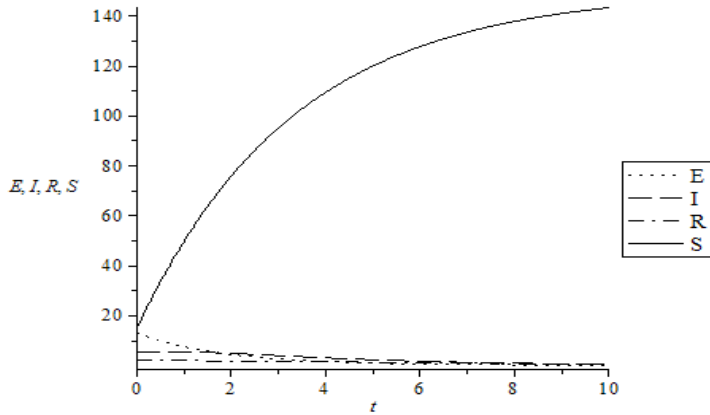


Figure 1: Simulation result when $\gamma=0.1, \beta=0.0005, \mu=0.3, \lambda=45, \delta=0.05, \epsilon=0.25, m=0.1, I_0=50$

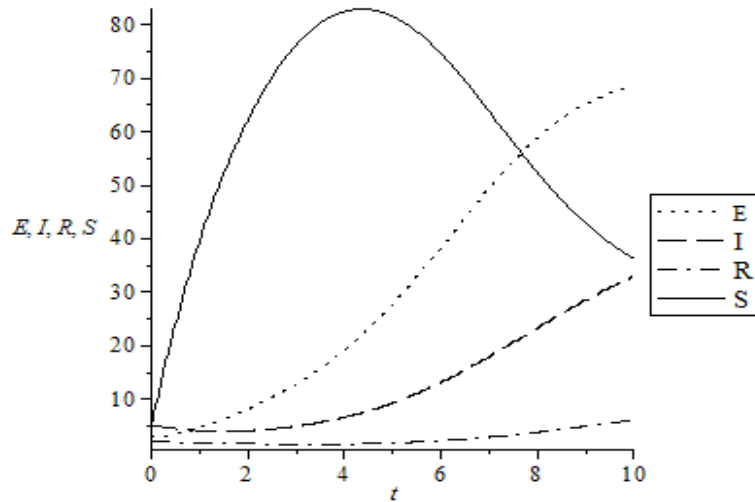


Figure 2: Simulation result when $\gamma=0.1, \beta=0.05, \mu=0.3, \lambda=45, \delta=0.05, \epsilon=0.25, m=0.1, I_0=5$

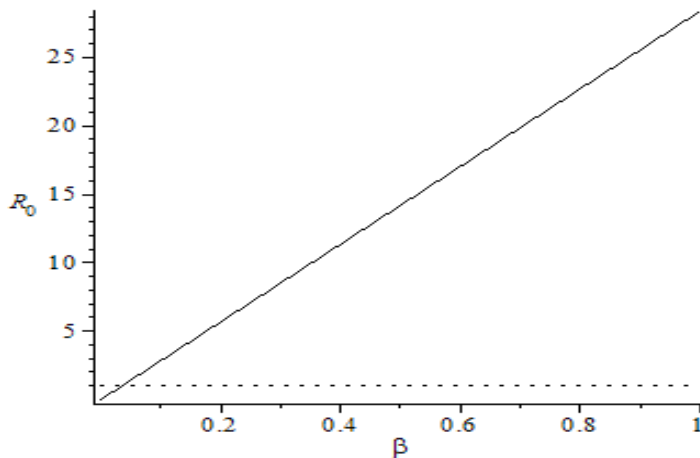


Figure 3: Plot of the basic reproduction number R_0 as a function of the transmission rate β with $\gamma=0.1, \mu=0.3, \lambda=45, \delta=0.05, \epsilon=0.25, m=0.1, I_0=50$

9.0 Conclusion

The simulation result displayed in Figure 1 with parameter set as shown in the Figure, reveals the asymptotic stability of the disease free equilibrium when $R_0 < 1$. It was also observed from that the susceptible class increases as the Exposed and

infected classes decreases which show the effect of initial state of the disease in the model and disease eradication.

Hence the higher I_0 (initial state of the disease), the better stability and at a point the disease will die out.

Figure 2. reveals the unstable nature of disease free equilibrium because, Susceptible class decreases as the Exposed and Infected classes increases. This shows the effect of initial state of the disease in the model and disease eradication

We also reveal that in Figure 3, transmission rate of the diseases β plays a vital role in the spread of the diseases i.e. if the transmission rate is increased as observed in fig 3, the spread of the disease is also increased. We observed that there exist a linear relationship between β and R_0 . We see that $R_0 < 1$ only when a smaller value of β is consider as show in Figure 3.

It is therefore recommended that medical practitioners need a very good knowledge on the initial state of a particular disease for better controlling and eradication.

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