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GLOBAL ANALYSIS OF AN SEIRS EPIDEMIC MODEL WITH NEW MODULATED SATURATED INCIDENCE

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Abstract. In this paper, an SEIRS epidemic model with non-monotonic incidence rate is introduce and drive the necessary condition of the model are locally asymptotically stable, globally asymptotically stable or stable. The Global stability of the model is proved by constructing a Lyapunov function. Some numerical simulations are given to illustrate the analytical results.

Keywords: epidemic model; equilibrium; modulated incidence; reproduction number.

2010 AMS Subject Classification: 92D30, 93A30, 93D30, 34D23.

1. Introduction

Mathematical models are important tools in analyzing the spread and control of infectious diseases. The study of infectious disease by the means of mathematical modeling provides the behavior of the disease and helps to understand the planning of the eradication policy. The basic and important research subject in mathematical epidemiology is the global stability of the equilibrium states of the epidemic models. Generally, an epidemic model admits two types of equilibrium states. The first is the disease-free equilibrium whose global stability means biologically that the disease always dies out. The second is the endemic equilibrium, if is globally asymptotically stable, the disease persists at the endemic equilibrium level if it is initially present.

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Epidemic models have been studied by many authors. Most of them are interested in the formulation of the incidence rate. The form of the incidence rate that is used in the classical Kermack-Mckendrick model (1927) is the simple mass action βSI where β, S and I denote the transmission rate, the number of susceptible population and infectious population respectively. The standard incidence is $\beta SI/N$, where N is the total population size and is called β the daily contact rate. In epidemiology using a compartmental approach, one may assume that a susceptible individual first goes through a latent period (and is said to become exposed or in the class E) after infection, before becoming infectious. The resulting models are of SEIR or SEIRS types, respectively, depending on whether the acquired immunity is permanent or otherwise. These types of models have attracted the attention of many authors and a number of papers have been published in this area. For example, Greenhalgh [7] considered an SEIR model that incorporates density dependence in the death rate. Cooke and Driessche [3] introduced and analyzed the SEIRS model with two delays. Greenhalgh [8] studied Hopf bifurcations in the SEIRS type models with density dependent contact rate and death rate. Li and Muldowney [11] and Li et al. [13] studied the global dynamics of the SEIR models with a non-linear incidence rate as well as standard incidence rate. Li et al. [12] analyzed the global dynamics of the SEIR model with vertical transmission and a bilinear incidence. Rinalid [15] analyzed epidemic models with latent period. In 2003, Zhang and Ma [22] analyzed the global dynamics of the SEIR model with saturating contact rate. All the models discussed above are of SEIR-type epidemic models, which are described by a system of ordinary differential equations. Ruan and Wang [16] studied an epidemic SIR model with a specific nonlinear incident rate and presented a detailed qualitative and bifurcation analysis of the model and Kar and Batabyal [17] proposed an SIR model with non-monotonic incidence rate suggested by Xiao and Ruan [4] incorporating with a treatment function.

In recent year, many authors generalized new incidence rate function and applied in different epidemic models. For example, Kar and Batabyal [17] proposed an SIR model with non-monotonic incidence rate suggested by Xiao and Ruan [4] incorporating with a treatment function and G. Ujjainkar [5] generalized the model of Kar and Batabyal [17] with two inhibitory parameters and also I [1] analyzed an SIR model with new incidence saturating rate function. Here I presented an SEIRS model with a new modulated saturated incidence rate suggested by G. Ujjainkar [5] without any treatment function.

2. The Mathematical Model

In this mathematical model, the SEIRS model has been adopted and different analyses to test for the stability of disease free epidemic equilibrium of the model. Has been carried out the four compartmental models which consist of the Susceptible Individuals (S), Exposed individuals infected, but not infectious (E), Infected individuals (I) and Recovered individuals (R) with new modulated (non-monotonic) saturated incidence rate.

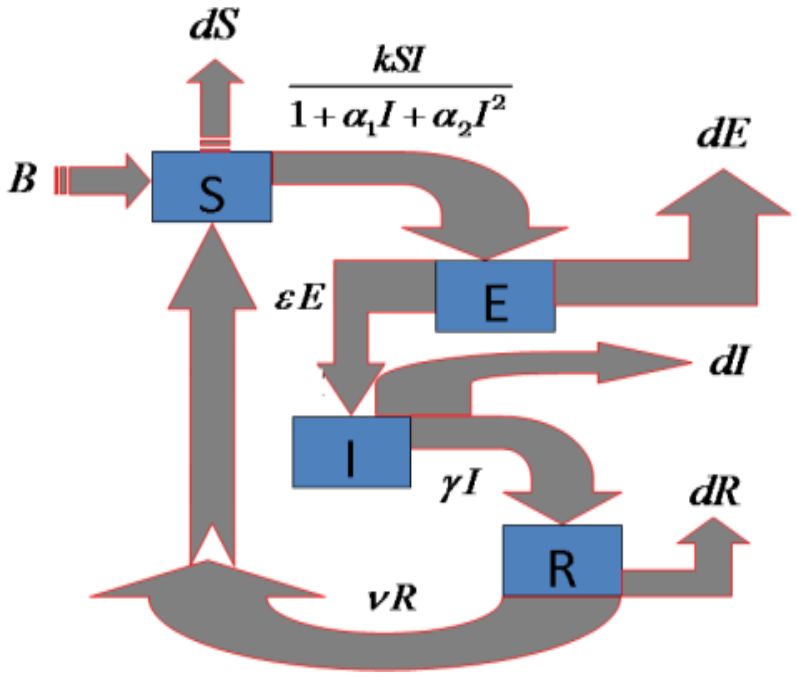

Figure-1. The Model Diagram

$$\left.\begin{aligned}
\frac{dS}{dt} &= B - dS - \frac{kSI}{1+\alpha_1 I+\alpha_2 I^2} + \nu R \\
\frac{dE}{dt} &= \frac{kSI}{1+\alpha_1 I+\alpha_2 I^2} - (\varepsilon + d)E \\
\frac{dI}{dt} &= \varepsilon E - (\gamma + d)I \\
\frac{dR}{dt} &= \gamma I - (\nu + d)R
\end{aligned}\right\} \qquad (1)$$

Where B is the recruitment rate of the population, ν is the rate of losing immunity at time t, ε is the rate of developing infectivity, γ is the recovery rate, d is the birth rate.

They considered a non-monotonic saturated incidence rate of the form $\frac{kSI}{1+\alpha_1 I+\alpha_2 I^2}$ .This represents the inhibition effect of the behavioral change of the susceptible individuals where there is an increase in the number of infective individuals, α_1 and α_2 are the parameter measures of the inhibitory effects. It assumes that the birth rate and death rate are not equal.

3. Equilibrium points

When the time derivatives are equal to zero, a disease free equilibrium (DEF) $P_0(S_0,E_0,I_0,R_0)=(\frac{B}{d},0,0,0)$ is achieved. For the endemic equilibrium $P^*(S^*,E^*,I^*,R^*)$ the following relations are mention below:

$S^*=\frac{(\varepsilon+d)(\gamma+d)(1+\alpha_1 I+\alpha_2 I^2)}{k\varepsilon}$, $E^*=\frac{(\gamma+d)I}{\varepsilon}$, $R^*=\frac{\gamma I}{(\nu+d)}$ and I is given as a root of the quadratic equation $aI^2+bI+c=0.$

Where, $a=[\alpha_2 d(\varepsilon+d)(\gamma+d)]$, $b=[\alpha_1 d(\varepsilon+d)(\gamma+d)+k(\varepsilon+d)(\gamma+d)+\frac{\nu\gamma\varepsilon}{\nu+d}]$ and

$c=[d(\varepsilon+d)(\gamma+d)-Bk\varepsilon]$.

Clearly, the above equation will have a positive root if $\Delta>0$ and $R_0>1$, where R_0 is basic reproduction number given as follows: $R_0=\frac{Bk\varepsilon}{d(\varepsilon+d)(\gamma+d)}$.

Now $I^*=\frac{-[\alpha_1 d(\varepsilon+d)(\gamma+d)+k(\varepsilon+d)(\gamma+d)+\frac{\nu\gamma\varepsilon}{\nu+d}]+\sqrt{\Delta}}{2[\alpha_2 d(\varepsilon+d)(\gamma+d)]}$

where, $\Delta=[\alpha_1 d(\varepsilon+d)(\gamma+d)+k(\varepsilon+d)(\gamma+d)+\frac{\nu\gamma\varepsilon}{\nu+d}]^2-4[\{\alpha_2 d(\varepsilon+d)(\gamma+d)\}\{R_0-1\}].$

Lemma 3.1. The system (1) has a disease-free equilibrium points if $N=\frac{B}{d}$.

Proof. Consider $N(t)=S(t)+E(t)+I(t)+R(t)$.

Then $\frac{dN}{dt}=B-dN(t)$ simplify and thus $\lim_{t\to\infty}N(t)=\frac{B}{d}$.

This implies the conclusion.

4. Stability

4.1. Local Stability of DEF

Let $x = S - S_0$, $E = E$, $I = I$ and $R = R$.

System (1) becomes,

$$\left.\begin{aligned} \frac{dx}{dt} &= B - [kI(x+\frac{B}{d})(1+\alpha_1 I+\alpha_2 I^2)^{-1} - d(x+\frac{B}{d}) + \nu R \\ \frac{dE}{dt} &= kI(x+\frac{B}{d})(1+\alpha_1 I+\alpha_2 I^2)^{-1} - (\varepsilon+d)E \\ \frac{dI}{dt} &= \varepsilon E - (\gamma+d)I \\ \frac{dR}{dt} &= \gamma I - (\nu+d)R \end{aligned}\right\} \tag{2}$$

By linearizing (1), we have

$$\left.\begin{aligned} \frac{dS}{dt} &= -\frac{kB}{d} I - dS + \nu R + \text{non linear terms} \\ \frac{dE}{dt} &= \frac{kB}{d} I - (\varepsilon+d)E + + \text{non linear terms} \\ \frac{dI}{dt} &= \varepsilon E - (\gamma+d)I \\ \frac{dR}{dt} &= \gamma I - (\nu+d)R \end{aligned}\right\} \tag{3}$$

This can be written in matrix from

$$\begin{pmatrix} \frac{dx}{dt} \\ \frac{dE}{dt} \\ \frac{dI}{dt} \\ \frac{dR}{dt} \end{pmatrix} = \begin{pmatrix} -d & 0 & \frac{-kB}{d} & \nu \\ 0 & -(\varepsilon+d) & \frac{kB}{d} & 0 \\ 0 & \varepsilon & -(\gamma+d) & 0 \\ 0 & 0 & \gamma & -(\gamma+d) \end{pmatrix} \begin{pmatrix} x \\ E \\ I \\ R \end{pmatrix}$$

$$= -(d+\lambda)(\nu+d+\lambda)[-(\varepsilon+d+\lambda)(\gamma+d+\lambda) + \frac{\varepsilon kB}{d}] = 0.$$

Lemma 4.1. If $R_0 < 1$ then the disease free equilibrium P_0 is locally asymptotically stable, P_0 is stable if $R_0 = 1$ and P_0 is unstable if $R_0 > 1$.

Proof. The characteristic equation of (3) at P_0 is

$$-(d+\lambda)(\nu+d+\lambda)[-(\varepsilon+d+\lambda)(\gamma+d+\lambda)+\frac{\varepsilon kB}{d}]=0.$$

It is that $\lambda_1=-d$, $\lambda_2=-(\nu+d)$ are two eigenvalues and they are always negative. To obtain other eigenvalues

$$-(\varepsilon+d+\lambda)(\gamma+d+\lambda)+\frac{\varepsilon kB}{d}=0.$$

Therefore, $d^2+\varepsilon d+\varepsilon\lambda+\gamma d-\frac{\varepsilon kB}{d}>0.$

Thus, the then the disease free equilibrium P_0 is locally asymptotically stable if $R_0<1$, P_0 is stable if $R_0=1$ and P_0 is unstable if $R_0>1$.

4.2. Global Stability of DEF

Define Lyapunov function:

$L=\varepsilon E-(\varepsilon+\mu)I$

By differentiating equation, we have

$L'=\varepsilon E'-(\varepsilon+\mu)I'$

$$L'=\varepsilon[\frac{kSI}{1+\alpha_1 I+\alpha_2 I^2}-(\varepsilon+d)E]-(\varepsilon+d)[\varepsilon E-(\gamma+d)I]$$

$$L'=[\frac{\varepsilon kSI}{1+\alpha_1 I+\alpha_2 I^2}-(\varepsilon+d)(\gamma+d)]I$$

$$L'=[\frac{R_0}{1+\alpha_1 I+\alpha_2 I^2}-1]I$$

If $I=0$, $L'=0$ but if $I\neq 0$ and $R_0<1$, $L'=0$ therefore, the disease free equilibrium is globally asymptotically stable.

4.3. Local Stability of Endemic Equilibrium

Let $x=S-S_*$, $y=E-E_*$, $z=I-I_*$, $q=R-R_*$

$$\frac{dx}{dt}=B-d(x+S_*)-\frac{k(x+S_*)(z+I_*)}{1+\alpha_1(z+I_*)+\alpha_2(z+I_*)^2}+\nu(q+R_*)$$

$$\frac{dE}{dt}=\frac{k(x+S_*)(z+I_*)}{1+\alpha_1(z+I_*)+\alpha_2(z+I_*)^2}-(\varepsilon+d)(y+E_*)$$

$$\frac{dI}{dt}=\varepsilon(y+E_*)-(\gamma+d)(z+I_*)$$

$$\frac{dR}{dt} = \gamma(z + I_*) - (\nu + d)(q + R_*)$$

The resulting Jacobin matrix is

$$A = \begin{pmatrix} -(kI_* + d) & 0 & kS_* & \nu \\ kI_* & -(\varepsilon + d) & kS_* & 0 \\ 0 & \varepsilon & -(\gamma + d) & 0 \\ 0 & 0 & \gamma & -(\nu + d) \end{pmatrix}$$

$$|A - \lambda I| = \begin{pmatrix} -(kI_* + d + \lambda) & 0 & kS_* & \nu \\ kI_* & -(\varepsilon + d + \lambda) & kS_* & 0 \\ 0 & \varepsilon & -(\gamma + d + \lambda) & 0 \\ 0 & 0 & \gamma & -(\nu + d + \lambda) \end{pmatrix} \quad (4)$$

The characteristic equation is

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

$$|A - \lambda I| = (\varepsilon + d + \lambda)(\gamma + d + \lambda)[\lambda^2 + (\varepsilon + 2d + \gamma - \varepsilon k^2 S_* I_*)\lambda + (\varepsilon + d)(\gamma + d) + kS_* - \varepsilon k^2 S_* I_* (d + \nu)] = 0.$$

From the characteristic equation of (4), it is found that $\lambda_1 = (\varepsilon + d)$ and $\lambda_2 = (\gamma + d)$ are two eigen values always negative. To obtain the other eigenvalues of (4) consider

$$\lambda^2 + (\varepsilon + 2d + \gamma - \varepsilon k^2 S_* I_*)\lambda + (\varepsilon + d)(\gamma + d) + kS_* - \varepsilon k^2 S_* I_* (d + \nu) = 0.$$

If $\varepsilon + 2d + \gamma > \varepsilon k^2 S_* I_*$ and $\varepsilon\gamma + \varepsilon d + d\gamma + d^2 + kS_* > d\varepsilon k^2 S_* I_* + \nu\varepsilon k^2 S_* I_*$ all the roots are in the left – half plane. Therefore, the endemic equilibrium is stable.

5. Numerical Simulations

To see the dynamical behavior of system (1), solve the system by using the parameters:

Case I. $B = 15$, $k = 0.398$, $\alpha_1 = 0.7$, $\alpha_2 = 0$, $d = 0.04$, $\nu = 0.0033$, $\varepsilon = 1$, $\gamma = 0.143$, then the basic reproduction number $R_0 > 1$. (figure 2).

Case II. Take $B = 3$, $k = 0.398$, $\alpha_1 = 0$, $\alpha_2 = 0.7$, $d = 0.04$, $\nu = 0.0033$, $\varepsilon = 1$, $\gamma = 0.143$, then the basic reproduction number $R_0 > 1$. (figure 3).

Case III. Take $B = 3$, $k = 0.398$, $\alpha_1 = \alpha_2 = 0.1$, $d = 0.04$, $\nu = 0.0033$, $\varepsilon = 1$, $\gamma = 0.143$, then the basic reproduction number $R_0 > 1$. (figure 4).

Case IV. $B=3,\ k=0.398,\ \alpha_1=\alpha_2=0.7,\ d=0.04,\ \nu=0.0033,\ \varepsilon=1,\ \gamma=0.143,$ then the basic reproduction number $R_0>1.$ (figure 5).

For the choice of parameters in above cases all the four component $S(t)$, $E(t)$, $I(t)$, $R(t)$ approach to their steady state values as time goes to infinity, the disease becomes endemic.

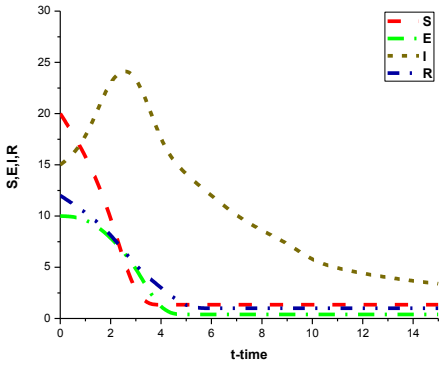

Figure-2

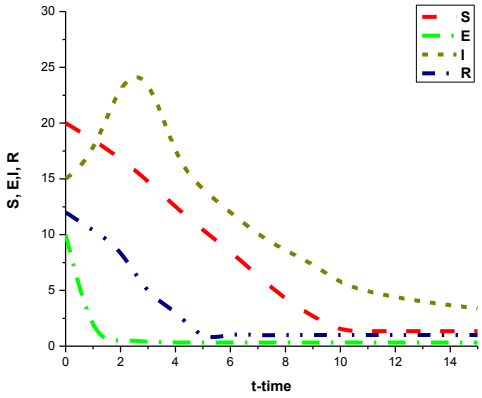

Figure-3

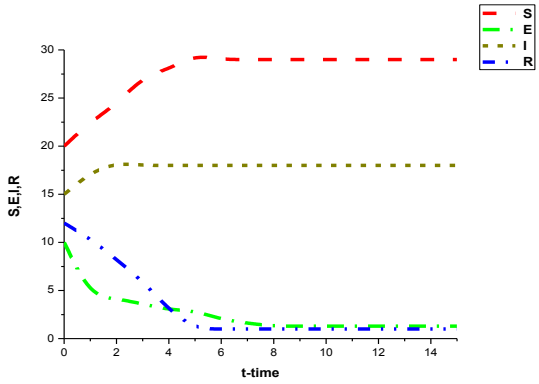

Figure-4

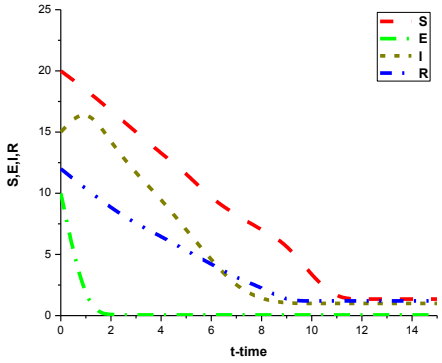

Figure-5

Case V. $B=0.01,\ k=0.398,\ \alpha_1=0.7,\ \alpha_2=0,\ d=0.04,\ \nu=0.0033,\ \varepsilon=1,\ \gamma=0.143.$ then the basic reproduction number $R_0<1.$ (figure 6).

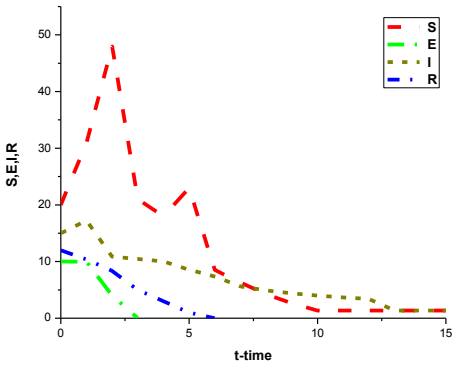

Figure-6

For the choice of parameters in Case V all the four component $S(t)$, $E(t)$, $I(t)$, $R(t)$ approach to unstable as time goes to infinity, there the disease becomes dies out.

Case VI. Taking all the parameters of Case V and interchange the value of α_1 and α_2 or both are equally probable (i.e. $\alpha_1 = \alpha_2 = 0.7$ or $\alpha_1 = \alpha_2 = 0.1$) or they have distinct values (i.e. $\alpha_1 = 0.1, \alpha_2 = 0.7$ or $\alpha_1 = 0.7, \alpha_2 = 0.1$) the all components are also unstable.

6. Conclusion

This paper investigates an SEIRS model with a non-monotone incidence function. The global behavior of the model is studied and basic reproduction number R_0 is defined. It has been noted that when $R_0 < 1$, the model has locally and globally asymptotically stable and when $R_0 > 1$ the disease is endemic. It is worth nothing that R_0 does not depend on the parameters α_1 and α_2 but numerical simulations illustrate the importance of the parameters α_1 and α_2.

Corollary 1. If $\alpha_2 = 0$ and $\alpha_1 = \alpha$, then the model coincides with that of Adebimpe. O. et. al. [14].

Corollary 2. If $\alpha_2 = \alpha$ and $\alpha_1 = 0$, then the model coincides with that of D. Xiao and S. Runa [4] into the three components of epidemic i.e. $S(t)$, $I(t)$, $R(t)$.

Conflict of Interests

The author declares that there is no conflict of interests.

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