

Modeling and Analysis of an SEIRS Epidemic Model with Saturated Incidence

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Abstract

In this paper, an SEIRS epidemic model with nonlinear incidence rate is investigated. The model exhibits two equilibria namely, the disease-free equilibrium and the endemic equilibrium. It is shown that if the basic reproduction number, $R_0 < 1$ the disease free equilibrium is locally and globally asymptotically stable. Also, we show that $R_0 > 1$, the disease equilibrium is locally asymptotically stable and the disease is uniformly persisted. Some numerical simulations are given to illustrate the analytical results.

Keywords: Epidemic model, nonlinear incidence rate, basic reproduction number, local and global stability

I. Introduction

The spread of infectious disease has always been of concerns and a threat to public health. Epidemic models have been studied by many authors. Most of them are interested in the formulation of the incidence rate. Greenhalgh [14] considered SEIR models that incorporate density dependence in the death rate. Cooke and van den Driessche [16] introduced and studied SEIRS models with two delays. Recently, Greenhalgh [15] studied Hopf bifurcations in models of the SEIRS type with density dependent contact rate and death rate. Liu et al., [1] analyzed the dynamical behavior of SEIRS with nonlinear incidence rate. Rinaldi [2] analyzed epidemic models with latent period. He obtained global stability results for the non-trivial equilibrium for the model.

In order to model this transmission process, several authors employ the following incidence functions. The first one is the bilinear incidence rate βSI , where S and I are respectively the number of susceptible and infective individuals in the population, and β is a positive constant [6-10]. The second one is the saturated incidence rate of the form $\frac{\beta SI}{1 + \alpha_1 S}$, where α_1 is a positive constant. The effect of saturation factor α_1 stems from epidemic control taking appropriate percussive measures [11-14]. The third one is the saturated incidence rate of the form $\frac{\beta SI}{1 + \alpha_2 I}$, where α_2 is a positive constant. Here, the number of effective contacts between infective individuals or due to the protective measures by the susceptible individuals [7, 11, 16].

In this paper, SEIRS model with vital dynamics is considered along a saturated incidence rate of the form $\frac{\beta SI}{1 + \alpha I}$. Unlike [2], we assume that the disease does not give permanent immunity. The result is written in terms of basic reproduction number and stabilities of the equilibria are investigated.

II. Mathematical Model Formulation

Rinaldi in his paper [2] considered an SEIRS model with vital dynamics as follows:

$$\frac{dS}{dt} = -\lambda SI + \mu - \mu S + \delta R$$

$$\frac{dE}{dt} = \lambda SI - (\varepsilon + \mu)E \quad (2.1)$$

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

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

where individuals are susceptible (S), then Exposed (E), then infected (I), the recovered (R) with temporary immunity, becoming susceptible again where immunity is lost. μ is the birth rate which is equal to the rate of mortality, λ is the disease transmission coefficient, δ is the rate of losing immunity at time t, ε is the rate of developing

infectivity, γ is the recovery rate. In this paper, we extend equation (2.1) to include the saturated incidence rate $\frac{\beta SI}{1 + \alpha I}$ and we assume that the birth rate and death rate are not equal.

The Proposed Model

$$\frac{dS}{dt} = A - \frac{\beta SI}{1 + \alpha I} - \mu S + \delta R$$

$$\frac{dE}{dt} = \frac{\beta SI}{1 + \alpha I} - (\varepsilon + \mu)E \tag{2.2}$$

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

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

where A is the recruitment rate of the population and $\frac{\beta SI}{1 + \alpha I}$ represents the inhibition effect of the behavioural change of the susceptible individuals where there is an increase in the number of infective individuals. Other parameters are as defined in (2.1)

III. Local stability of the Disease Free Equilibrium (DFE)

The model has a disease-free equilibrium obtained by setting the right hand sides of (2.2) to zero.

$$A - \frac{\beta SI}{1 + \alpha I} - \mu S + \delta R = 0 \tag{3.1}$$

$$\frac{\beta SI}{1 + \alpha I} - (\varepsilon + \mu)E = 0 \tag{3.2}$$

$$\varepsilon E - (\gamma + \mu)I = 0 \tag{3.3}$$

$$\gamma I - (\delta + \mu)R = 0 \tag{3.4}$$

with I=0, this gives the DFE

$$P_0(S_0, E_0, I_0, R_0) = \left(\frac{A}{\mu}, 0, 0, 0\right)$$

Let

$$x = S - S_0, E = E, I = I, R = R$$

Equation (2.2) becomes

$$\left. \begin{aligned} \frac{dx}{dt} &= A - \left[\beta I \left(x + \frac{A}{\mu} \right) (1 + \alpha I)^{-1} \right] - \mu \left(x + \frac{A}{\mu} \right) + \gamma R \\ \frac{dE}{dt} &= \beta I \left(x + \frac{A}{\mu} \right) (1 + \alpha I)^{-1} - (\varepsilon + \mu)E \\ \frac{dI}{dt} &= \varepsilon E - (\gamma + \mu)I \\ \frac{dR}{dt} &= \gamma I - (\delta + \mu)R \end{aligned} \right\} \tag{3.5}$$

By linearizing (2.2), we have

$$\left. \begin{aligned} \frac{dS}{dt} &= -\frac{\beta A}{\mu} I - \mu S + \delta R + \text{non linear terms} \\ \frac{dE}{dt} &= \frac{\beta A}{\mu} I - (\varepsilon + \mu)E + \text{non linear terms} \\ \frac{dI}{dt} &= \varepsilon E - (\gamma + \mu)I \\ \frac{dR}{dt} &= \gamma I - (\delta + \mu)R \end{aligned} \right\} \tag{3.6}$$

which can be written in matrix form

$$\begin{pmatrix} \frac{dx}{dt} \\ \frac{dE}{dt} \\ \frac{dI}{dt} \\ \frac{dR}{dt} \end{pmatrix} = \begin{pmatrix} -\mu & 0 & -\frac{\beta A}{\mu} & \delta \\ 0 & -(\varepsilon + \mu) & \frac{\beta A}{\mu} & 0 \\ 0 & \varepsilon & -(\gamma + \mu) & 0 \\ 0 & 0 & \gamma & -(\delta + \mu) \end{pmatrix} \begin{pmatrix} x \\ E \\ I \\ R \end{pmatrix}$$

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

$$= \begin{vmatrix} -(\mu + \lambda) & 0 & -\frac{\beta A}{\mu} & \delta \\ 0 & -(\varepsilon + \mu + \lambda) & \frac{\beta A}{\mu} & 0 \\ 0 & \varepsilon & -(\gamma + \mu + \lambda) & 0 \\ 0 & 0 & \gamma & -(\delta + \mu + \lambda) \end{vmatrix} = 0$$

$$= -(\mu + \lambda)(\delta + \mu + \lambda) \left[-(\mu + \varepsilon + \lambda)(\gamma + \mu + \lambda) + \frac{\beta A}{\mu} \right] = 0$$

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

Then,

$$-(\mu + \varepsilon + \lambda)(\gamma + \mu + \lambda) + \frac{\beta A}{\mu} = 0$$

Therefore,

$$\lambda^2 + (2\mu + \gamma + \varepsilon)\lambda + \mu^2 + \mu\gamma + \varepsilon\gamma + \varepsilon\mu - \frac{\beta A}{\mu} = 0$$

For negative roots, we must have by Descartes' rule of signs

$$\mu^2 + \mu\gamma + \varepsilon\gamma + \varepsilon\mu - \frac{\beta A}{\mu} > 0$$

$$\mu^2 + \mu\gamma + \varepsilon\gamma + \varepsilon\mu < \frac{\beta A}{\mu}$$

$$(\mu + \varepsilon)(\mu + \gamma) > \frac{\beta A}{\mu}$$

$$1 > \frac{\beta A}{\mu(\mu + \varepsilon)(\mu + \gamma)}$$

$$\frac{\beta A}{\mu(\mu + \varepsilon)(\mu + \gamma)} < 1$$

$$\text{Let } R_0 = \frac{\beta A}{\mu(\mu + \varepsilon)(\mu + \gamma)}$$

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

Proof: We shall check the stability of the disease free equilibrium P_0 , from the model, then the linearization of disease-free equilibrium P_0 gives the following characteristic equation.

$$-(\mu + \lambda)(\gamma + \mu + \lambda) \left[-(\mu + \varepsilon + \lambda)(\gamma + \mu + \lambda) + \frac{\beta A}{\mu} \right] = 0$$

From equation (3.7), it can be seen that

$\lambda_1 = -\mu, \lambda_2 = -(\delta + \mu)$ are two of the eigenvalues and they are always negative. To obtain other eigenvalues of equation (3.7)

$$\lambda^2 + (2\mu + \gamma + \varepsilon)\lambda + \mu^2 + \mu\gamma + \varepsilon\gamma + \varepsilon\mu - \frac{\beta A}{\mu} = 0$$

$$\frac{dx}{dt} = A - \frac{\beta(x + S_*)(z + I_*)}{1 + \alpha(z + I_*)} - \mu(x + S_*) + \delta(q + R_*)$$

$$\frac{dE}{dt} = \frac{\beta(x + S_*)(z + I_*)}{1 + \alpha(z + I_*)} - (\varepsilon - \mu)(y + E_*)$$

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

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

From equation (3.7), we see that all roots have negative real parts if

$$\mu^2 + \mu\gamma + \varepsilon\gamma + \varepsilon\mu - \frac{\beta A}{\mu} > 0$$

That is, if $R_0 < 1$

The disease free equilibrium P_0 , is locally asymptotically stable, If $R_0 = 1$, one eigenvalue of equation (3.7) is zero and it is simple. Then P_0 is stable.

If $R_0 > 1$, one of the roots of equation (3.7) has a positive real part, then P_0 is unstable

IV. Global stability of the disease-free equilibrium

Define Lyapunov function:

$$L = \varepsilon E - (\varepsilon + \mu)I \tag{4.1}$$

By differentiating equation (2) we have

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

$$L^1 = \varepsilon \left[\frac{\beta SI}{1 + \alpha I} - (\varepsilon + \mu)E \right] - (\varepsilon + \mu) [\varepsilon E - (\gamma + \mu)I]$$

$$L^1 = \varepsilon \left[\frac{\beta SI}{1 + \alpha I} \right] - (\varepsilon + \mu)(\gamma + \mu)I$$

$$L^1 = \frac{\varepsilon \beta SI}{1 + \alpha I} - (\varepsilon + \mu)(\gamma + \mu)I$$

$$L^1 = I \left[\frac{R_0}{1 + \alpha I} - 1 \right]$$

If $I=0, L^1=0$ but if $I \neq 0$ and $R_0 < 1, L^1 < 0$

Therefore, the disease free equilibrium is globally asymptotically stable.

V. Local Stability of the Endemic Equilibrium

Let

$$x = S - S_*, \quad y = E - E_*, \quad z = I - I_*, \quad q = R - R_* \tag{5.1}$$

The resulting Jacobian matrix is

$$A = \begin{pmatrix} -(\beta I_* + \mu) & 0 & \beta S_* & \delta \\ \beta I_* & -(\varepsilon + \mu) & \beta S_* & 0 \\ 0 & \varepsilon & -(\gamma + \mu) & 0 \\ 0 & 0 & \gamma & -(\delta + \mu) \end{pmatrix}$$

$$|A - \lambda I| = \begin{pmatrix} -(\beta I_* + \mu + \lambda) & 0 & \beta S_* & \delta \\ \beta I_* & -(\varepsilon + \mu + \lambda) & \beta S_* & 0 \\ 0 & \varepsilon & -(\gamma + \mu + \lambda) & 0 \\ 0 & 0 & \gamma & -(\delta + \mu + \lambda) \end{pmatrix}$$

The resulting characteristic equation for the model is

I. $(\varepsilon + \mu + \lambda)(\gamma + \mu + \lambda) \left[\begin{matrix} \lambda^2 + \\ (\varepsilon + 2\mu + \gamma - \varepsilon\beta^2 S_* I_*)\lambda + \\ (\varepsilon + \mu)(\gamma + \mu) + \varepsilon\beta S_* \\ -\varepsilon\beta^2 S_* I_*(\mu + \delta) \end{matrix} \right] = 0$ $\delta = 0.0033, A = 0.02$

with different values of α

In figure 6.1, 6.2, 6.3, we use different values for α and we discovered that the higher the value of α , the more the susceptible class reaches steady state and the exposed, infected and recovered classes approach zero. This implies that the parameter has a part to play in the eradication of the disease in the population.

Numerical simulations

To see the dynamical behaviour of system (2.2), we solve the system by using maple using the parameters;

$\beta = 0.398, \gamma = 0.143, \mu = 0.04, \varepsilon = 1,$

From equation (5.3), it can be seen that $\lambda_1 = -(\varepsilon + \mu), \lambda_2 = -(\gamma + \mu)$ are two of the eigenvalues and are always negative. To

obtain the other eigenvalues of equation (5.3) we consider the equation

$$\lambda^2 + (\varepsilon + 2\mu + \gamma - \varepsilon\beta^2 S_* I_*)\lambda + (\varepsilon + \mu)(\gamma + \mu) + \varepsilon\beta S_* - \varepsilon\beta^2 S_* I_*(\mu + \delta) = 0$$

$$\varepsilon\gamma + \varepsilon\mu + \mu\gamma + \mu^2$$

If $\varepsilon + 2\mu + \gamma > \varepsilon\beta^2 S_* I_*$ and $\varepsilon\beta S_* > \mu\varepsilon\beta^2 S_* I_*$, all the roots are in the left-half plane.

$$+ \delta\varepsilon\beta^2 S_* I_* + \delta\varepsilon\beta\gamma I_*$$

Therefore, the endemic equilibrium is stable.

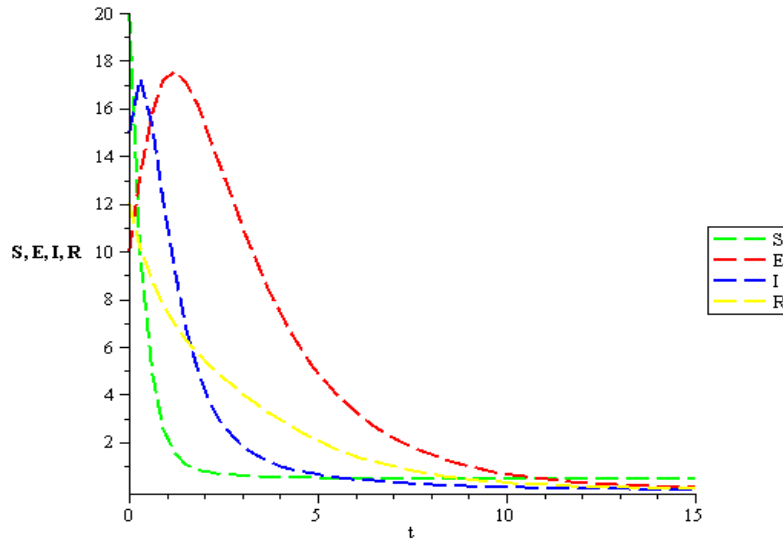


Fig. 6.1: Graph of S, E, I, R against time t, when $\beta = 0.398$, $\gamma = 0.143$, $\mu = 0.04$, $\varepsilon = 1$, $\delta = 0.0033$, $\alpha = 0.1$ and $A = 0.02$

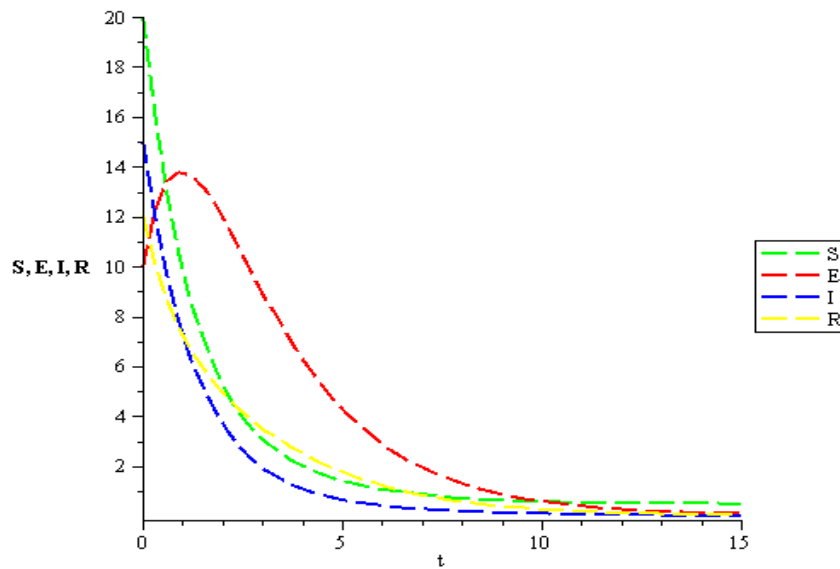


Fig. 6.2: Graph of S, E, I, R against time t, when $\beta = 0.398$, $\gamma = 0.143$, $\mu = 0.04$, $\varepsilon = 1$, $\delta = 0.0033$, $\alpha = 0.4$ and $A = 0.02$

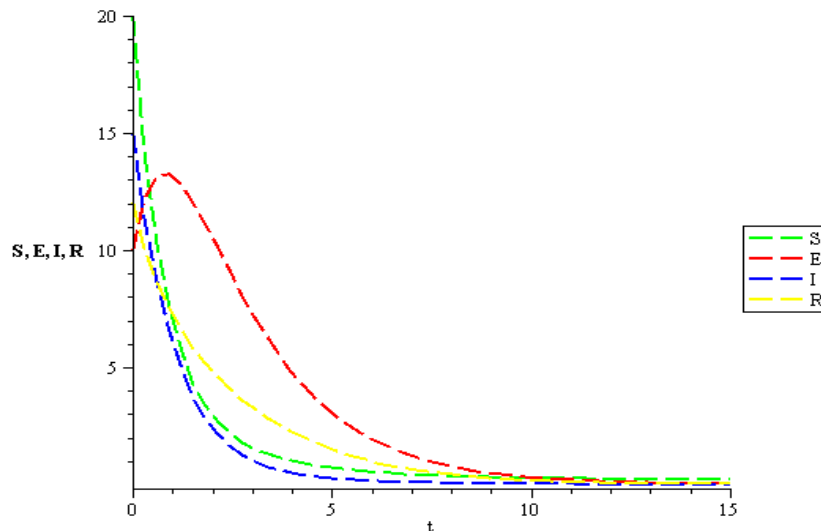


Fig. 6.3: Graph of S, E, I, R against time t, when $\beta = 0.398$, $\gamma = 0.143$, $\mu = 0.04$, $\varepsilon = 1$, $\delta = 0.0033$, $\alpha = 0.7$ and $A = 0.02$

II. Conclusions

In this paper, an SEIRS deterministic model with saturated incidence rate is formulated. Some of the main findings of this study are;

- (i) The model has locally and globally asymptotically stable disease-free equilibrium whenever the associated reproduction number is less than unity;
- (ii) The model has a unique endemic equilibrium and the endemic equilibrium is locally-asymptotically stable.
- (iii) Numerical simulations illustrate the importance of the parameter, α that measures the effects of sociological, psychological or other mechanisms of the disease.

References

- [1] Liu, W., Hethcote, H.W., and Levin, S. A. (1987): Dynamical behavior of epidemiological models with nonlinear incidence rates. *J. Math. Biol.* 25, 359-80.
- [2] Rinaldi, F. (1990): Global Stability Results for Epidemic models with Latent Period. *IMA Journal of Mathematics Applied in Medicine and Biology*, 7, 69-75.
- [3] M. Gabriela, M. Gomes, L. J. White, G. F. Medley (2005): The reinfection threshold. *J. Theor. Biol.*, 236, pp. 111-113.
- [4] Z. Jiang, J. Wei (2008): Stability and bifurcation analysis in a delayed SIR model: *Chaos Soliton. Tract.* 35, pp 609-619.
- [5] W. Wang, S. Ruan (2004): Bifurcation in epidemic model with constant removal rate infectives, *J. Math. Appl.*, 291 pp 775-793.
- [6] F. Zhang, Z.Z. Li and F. Zhang (2008): Global Stability of an SIR epidemic model with constant infectious period. *Appl. Math. Comput.* 199, pp 285-291.
- [7] Y. Zhou, H. Liu (2003): Stability of periodic solutions for an SIS model with pulse vaccination *Math. Comput. Model.* 38, pp 299-308.
- [8] R. M. Anderson and R. M. May (1978): Regulation and Stability of host-parasite population interactions in regulatory processes. *J. Anim. Ecol.* 47(1), pp 219-267.
- [9] L. S, Chen and J. Chen (1993): *Nonlinear biological dynamics system*, Scientific Press, China.
- [10] C. Wei and L. Chen (2008): A delayed epidemic model with pulse vaccination: *Discrete Dyn. Nat. Soc.*, doi:10.1155/2008/746951, pp. 1-12.
- [11] J. Z. Zhang, Z. Jin, Q. X. Liu and Z. Y. Zhang (2008) Analysis of a delayed SIR model with non-linear incidence rate. *Discrete Dyn. Nat. Soc.*, doi:10.1155/2008/636153, pp. 1-16.
- [12] V. Capasso and G. Serio (1978): A generalization of Kermack-Mekandrick deterministic epidemic model. *Math Biosci.* 42, pp. 41-61.
- [13] R. Xu and Z. Ma (2009): Stability of a delayed SIRS epidemic model with a nonlinear incidence rate. *Chaos, Soliton, Fract.* 419(5), pp. 2319-2325.
- [14] Greenhalgh, D. (1990): An epidemic model with a density-dependent death rate. *IMA J. Math. Appl. Med. Biol.* 7, pp 1-26.
- [15] Greenhalgh, D. (1992): Some threshold and stability results for epidemic models with a density dependent death rate. *Theor. Pop. Biol.* 42, pp. 130-157.
- [16] Cooke, K. and van den Driessche, P. (1996): Analysis of an SEIRS epidemic model with two delays. *J. Math Biol.* 35: 240-260.