A Modified Sir Epidemic Model with Immigration and Generalized Saturated Incidence Rate Function

D. Jasmine. E. C.¹, Henry Amirtharaj²

¹PG & Research Department of Mathematics Bishop Heber College (Autonomous), Trichy, India

²PG & Research Department of Mathematics Bishop Heber College (Autonomous), Trichy, India

Abstract: On account of the effect of limited treatment resources on the control of epidemic disease, a modified SIR epidemic model with generalized saturated incidence rate is incorporated. The stability analysis of the disease-free and the endemic equilibrium are discussed with a nonlinear incidence rate.

Keywords: Basic Reproduction Number Ro, Disease-Free Equilibrium, Epidemic, Endemic Equilibrium.

1. Introduction

Contacts between susceptible and infective may lead to infection, and infective may recover at different times after they become infective. This dynamics is stochastic in nature but for a large population, the statistical fluctuations may be ignored and the change in the size of each compartment becomes deterministic. Epidemic dynamics is an important method of studying the speared rules of infectious diseases qualitatively and also qualitatively. It is largely based on specific properties of population growth. Analysis through Mathematical Modeling requires transmission rules. i.e., rate of incidence. Incidence is an epidemiological model is the rate at which susceptible become infections. The behavior of the SIR models are greatly affected by the way in which transmission between infected and susceptible individual are modeled.

Mathematical Modeling is an important tool to understand and predict the spread of infectious diseases. In this process rate of incidence plays a crucial role. The incidence is an epidemiological model is the rate at which susceptible become infectious. The incidence rate has been frequently used in classical epidemic models. Capasso and Serio [3] introduced a saturated incidence rate into epidemic models. Mena Lorca and Hethcote [12] also analyzed an SIR model with the same saturation incidence. Ruan and Wang [15] studied an epidemic model with a specific non-linear incidence rate kI^2S / $(1+\alpha I^2)$ and presented a detailed qualitative and bifurcation analysis of the model. A more general incidence $\lambda I^p S / (1+\alpha I^q)$ was proposed by many other researchers [2, 5, 6, 7, 8, 9]. A very general form of nonlinear incidence rate was considered by Derrick and Driessche [4]. Simple mass action was introduced in classical Kermack-Mcendrick [10] model β SI, where β is transmission rate, S is susceptible population and I is infectious population. Another popularly used incidence rate is standard incidence $\beta SI/N$, where N is the total population and β is daily contact rate. An SIRS model with saturation incidence was proposed by Hethcote, Liu and Levin [7, 11] have proposed a non-linear incidence rate. Ankit Agrawal [1] have

proposed an incidence rate as $\frac{kI}{\rho + \beta I}$. In this paper we

consider a modified SIR model with the saturated incidence

function
$$\frac{\lambda SI}{\rho + \alpha_1 I + \alpha_2 I^2}$$
.

2. The Basic Mathematical Model

In this section we have considered an SIR epidemiological model with asymptotically homogeneous incidence rate function. Then our model under the frame work of the following form

$$\frac{dS}{dt} = a - dS - \phi + \beta R + \mu$$

$$\frac{dI}{dt} = \phi - (d+m)I$$

$$\frac{dR}{dt} = mI - (d+\beta)R$$

where S(t), I(t) and R(t) denote the number of susceptible, infective and recovered at time t respectively. a is the recruitment rate of population, d is the natural death rate of the population, m is the natural recovery rate of infective, β is the rate at which recovered individuals loss immunity and return to susceptible, μ is the increase of susceptible at the constant rate and ϕ is the transmission rate.

The transmission rate
$$\phi = \frac{\lambda SI}{\rho + \alpha_1 I + \alpha_2 I^2}$$
 displays a

saturation effect accounting for fact that the number of contacts an individual reaches some maximal value due to spatial distribution of the population, where λ is the proportionality constant ρ is the positive constant ≥ 1 , α is a positive parameter and λSI is the infection force of the disease.

3. Main Results

In this section we study an SIR epidemic model to obtain properties of the equilibrium points and analyze sufficient conditions under which the equilibrium points are unique or

International Journal of Science and Research (IJSR) ISSN (Online): 2319-7064

global. We rewrite the system (1) as

$$\dot{S} = a - dS - \phi + \beta R + \mu$$

$$\dot{I} = \phi - (d + m)I$$

$$\dot{R} = mI - (d + \beta)R$$
(2)

Because of the biological meaning of the components [S(t), I(t), R(t)], we focus on the model in the first octant of \mathbb{R}^3 .

We first consider the existence of equilibra of system (2). It is easy, by computations, to coincide that the system (2) has two equilibrium statuses. The disease free equilibrium state

$$E_0 = (\frac{a}{d}, 0, 0)$$
 which exists for all parameter values and

endemic equilibra (S^*, I^*, R^*). To find the endemic equilibra (S^*, I^*, R^*) of system (2) set $\dot{S} = \dot{I} = \dot{R} = 0$.

Then, we obtain

$$a - dS - \phi + \beta R + \mu = 0$$

$$\phi - (d+m)I = 0$$

$$mI - (d + \beta)R = 0$$

$$\Rightarrow R = \frac{mI}{d+\beta}$$

Then
$$R^* = \frac{mI^*}{d+\beta}$$

$$\phi - (d+m)I = 0$$

$$\Rightarrow \phi = (d+m)I$$

and
$$\frac{\lambda IS}{\rho + \alpha_1 I + \alpha_2 I^2} - (d + m)I = 0$$

$$S = \frac{(\rho + \alpha_1 I + \alpha_2 I^2)(d+m)I}{2I},$$

$$S^* = \frac{\phi(\rho + \alpha_1 I + \alpha_2 I^2)}{\lambda I^*}$$

We have

$$a - \frac{d(\rho + \alpha_1 I + \alpha_2 I^2)(d+m)}{\lambda}$$
$$-(d+m)I + \beta \frac{mI}{d+\beta} + \mu = 0$$

$$d\alpha_2(d+m)(d+\beta)I^2$$

$$+[d\alpha_1(d+m)(d+\beta)+(d+m)(d+\beta)\lambda-\beta\lambda m]I$$

$$+\rho d(d+m)(d+\beta) - \lambda(a+\mu)(d+\beta) = 0$$

$$\begin{split} I &= -\frac{\left[d\,\alpha_1(d+m)(d+\beta)\right]}{2\,d\,\alpha_2(d+m)(d+\beta)} \\ &+ \lambda(d+m)(d+\beta) - \beta\lambda m \\ &\frac{2\,d\,\alpha_2(d+m)(d+\beta)}{\end{split}$$

Paper ID: 020131483

$$\frac{\left[d\alpha_{1}(d+m)(d+\beta)+\lambda(d+m)(d+\beta)-\beta\lambda m\right]^{2}}{-4d\alpha_{2}(d+m)(d+\beta)^{2}\left[\rho d(d+m)-\lambda(a+\mu)\right]}$$

$$\pm \frac{-2d\alpha_{2}(d+m)(d+\beta)}{2d\alpha_{2}(d+m)(d+\beta)}$$

i.e.,

$$I^* = \frac{-[d\alpha_1(d+m)(d+\beta) + \lambda(d+m)(d+\beta) - \beta\lambda m]}{2d\alpha_2(d+m)(d+\beta)}$$

$$\frac{\int [d\alpha_{1}(d+m)(d+\beta) + \lambda(d+m)(d+\beta) - \beta\lambda m]^{2}}{\pm \frac{-4d\alpha_{2}(d+m)(d+\beta)^{2}[\rho d(d+m) - \lambda(a+\mu)]}{2d\alpha_{2}(d+m)(d+\beta)}}$$

which exists provided that the reproduction number

$$R_0 = \frac{\lambda(a+\mu)}{\rho d(d+m)} > 1 (3)$$

4. Mathematical Analysis

4.1 Lemma

The plane $S + I + R = \frac{a + \mu}{d}$ is a manifold of system (2)

which is attracting in the first octant.

Proof

Summing up the three equations in (2) and denoting N(t) = S(t) + I(t) + R(t), we have

$$\frac{dN}{dt} = (a + \mu) - dN$$
 (4)

It is clear that $N(t) = \frac{a + \mu}{d}$ is a solution of system (4) and

for any $N(t') \ge 0$, the general solution of system (4) is obtained by solving system (4). This is the linear differential equation of first order so the general solution of system (4) is

$$N = \frac{1}{d} \{ (a + \mu) - [(a + \mu) - dN(t')]e^{-d(t-t')} \}$$

When $t \rightarrow \infty$, we get

$$N(t) = \frac{a + \mu}{d}$$

This implies the conclusion.

It is clear that the limit set of system (2) is on the plane

$$S + I + R = \frac{a + \mu}{d}$$
. Thus, the reduced system is

$$\frac{dI}{dt} = \frac{\lambda I(\frac{a+\mu}{d} - I - R)}{\rho + \alpha_1 I + \alpha_2 I^2} - (d+m)I \cong P(I,R)$$

$$\begin{cases}
\frac{dR}{dt} = mI - (d+\beta)R \cong Q(I,R)
\end{cases}$$
(5)

4.2 Theorem

System (5) does not have non-trivial periodic orbit if α (2d + β + m) > 0.

Proof.

Consider system (5) for I > 0 and R > 0 . Take a Dulac function Wiggins [14],

$$D(I,R) = \phi^{-1}$$

i.e.,
$$D(I,R) = \frac{\rho + \alpha_1 I + \alpha_2 I^2}{\lambda IS}$$

Notice that

$$\begin{split} \frac{\partial (DP)}{\partial R} + \frac{\partial (DQ)}{\partial R} &= -\frac{d(\rho + \alpha_1 I + \alpha_2 I^2)}{\lambda (\frac{a + \mu}{d} - I - R)^2} \\ &- \frac{(\rho + \alpha_1 I + \alpha_2 I^2)(d + \beta)}{\lambda I (\frac{a + \mu}{d} - I - R)^2} [R - (\frac{a + \mu}{d} - I - R)] \\ \frac{\partial (DP)}{\partial R} + \frac{\partial (DQ)}{\partial R} &< 0 \end{split}$$

Hence the conclusion follows.

In order to study the properties of the disease-free equilibrium E_0 and the endemic equilibrium E^{\ast} , we rescale the system (5) by

$$x = \frac{\lambda I}{d + \beta}, \quad y = \frac{\lambda R}{d + \beta}, \quad \tau = (d + \beta)t$$

$$\frac{dx}{dt} = \frac{\lambda}{d + \beta} \left[\frac{\lambda I(\frac{a + \mu}{d} - I - R)}{\rho + \alpha_1 I + \alpha_2 I^2} - (d + m)I \right]$$

$$\frac{dx}{d\tau} = \frac{\lambda I}{d + \beta} \frac{1}{\rho + \alpha_1 I + \alpha_2 I^2} \left\{ \frac{\lambda (a + \mu)}{d (d + \beta)} - \frac{\lambda I}{d + \beta} - \frac{\lambda R}{d + \beta} \right\}$$

$$-\frac{\lambda I}{d + \beta} \frac{(d + \mu)}{(d + \beta)}$$

$$\frac{dx}{dt} = \frac{px}{1 + qx} (A - x - y) - Tx$$

$$\frac{dy}{dt} = sx - y$$

$$\{6\} \text{ where } p = \rho^{-1},$$

$$q = \frac{(\alpha_1 + \alpha_2 I)(d + \beta)}{\rho \lambda}, \ A = \frac{(a + \mu)\lambda}{d(d + \beta)}, \ T = \frac{d + m}{d + \beta}$$

and
$$s = \frac{m}{d + \beta}$$

The trivial equilibrium (0,0) of system (6) is the disease-free equilibrium of model (2) and the unique positive equilibrium (x*, y*) of system (6) is the endemic equilibrium E* of model (2) if and only if Ap-T>0 and q>0, where

$$x^* = \frac{Ap - T}{p(1+s) + Tq}, \ y^* = sx^*$$

We first determine the stability and topological type of (0, 0). The Jacobian matrix of system (6) at (0, 0) is

$$M_0 = \begin{pmatrix} Ap - T & 0 \\ s & -1 \end{pmatrix}$$

4.3 Theorem

If T - Ap > 0 the disease-free equilibrium (0,0) of system (6) is stable hyperbolic node, T - Ap = 0 then saddle node and T - Ap < 0 then hyperbolic saddle node.

When T - Ap < 0, we discuss the stability and topological type of the endemic equilibrium (x^*, y^*) .

The Jacobian matrix of the system (6) at (x^*, y^*) is

$$M_{1} = \begin{pmatrix} \frac{px^{*}[qsx^{*} - (Aq+1)]}{(1+qx^{*})^{2}} & \frac{px^{*}[-(1+qx^{*})]}{(1+qx^{*})^{2}} \\ s & -1 \end{pmatrix}$$

We have

$$\det(M_1) = \frac{px^*[Aq + (1+s)]}{(1+qx^*)^2}$$

Since q > 0, it follows that $det(M_1) > 0$ and (x^*, y^*) is a node or a focus or a center. Now we have the following result on the stability of (x^*, y^*) .

4.4 Theorem

Suppose T - Ap < 0, then there is a unique endemic equilibrium (x^*, y^*) of model (6) which is a saddle mode.

Proof.

The trace of the matrix M_1 is

$$Trace(M_1) = \frac{px^*[qsx^* - (Aq+1)] + (1+qx^*)^2}{(1+qx^*)^2}$$

The sign of $trace(M_1)$ is determined by

$$S_1 = px^*[qsx^* - (Aq+1)]$$

Substituting $x^* = \frac{Ap - T}{(1+s) + Tq}$ in S_1 and using a straight

forward calculation, we have

$$S_{1} = \frac{p(Ap - T)}{[p(1+s) + Tq]^{2}} [-Aq(p + Tq)$$

$$-(s+1)(Tq + p)]$$
Since $q > 0$, $[p(1+s) + Tq] > 0$
and $[-Aq(p + Tq) - (s+1)(Tq + p)] < 0$,
 $\Rightarrow S_{1} < 0$.

However, when T - Ap < 0, we have $trace(M_I) < 0$. This completes the proof.

5. Concluding Remarks

Modeling results are helpful to predict the developing tendency of disease. The model we have discussed provides learning about the transmission rate effects. In this paper, we consider a modified SIR model with immigration and generalized incidence rate ϕ . The basic reproduction number R_0 with the special kind of transmission rule. The global stability of the endemic equilibrium point

International Journal of Science and Research (IJSR) ISSN (Online): 2319-7064

 $E^* = (S^*, I^*, R^*)$ depends on the basic reproduction number R_0 . It plays an important role to control the disease. When $R_0 \leq 1$ the disease free equilibrium

 $E_0 = (\frac{a+\mu}{d}, 0, 0)$ is globally attractive in the first octant

and it is globally stable, that is the disease dies out. When $R_0 > 1$ the endemic equilibrium \boldsymbol{E}^* exists and is globally stable in the interior. Results and parametric conditions help to develop social consciousness about the disease among susceptible.

References

- [1] Ankit Agrawal and G. Saxena SIR Model with Generalized Standard Incidence Rate Function International Journal of Applied Mathematics & Statistical Sciences (IJAMSS) Vol. 2,Issue 5, Nov 2013, 75-82
- [2] Anderson, R. M. and May, R. M.: Infectious Diseases of Humans. Dynamics and Control, (Oxford University Press, Oxford. 1998).
- [3] Capasso V. and Serio G., (1978), A Generalization of the Kermack-Mckendrick Deterministic Epidemic model, Math. Biosci. 42, 43-61.
- [4] Derrick W.R. and P. Van Der Driessche (1993), A Disease Transmission Model in a Non-constant Population, J. Math. Biol., 31:495-512.
- [5] Gajendra Ujjainkar, V. K. Gupta, B.Singh, R. Khandelud., 2012. An Epidemic Model with Modified Non-monotonic Incidence Rate under Treatment, 6:1159-1171.
- [6] Hethcote, H. W. and Van Den Driessche, P.: Some Epidemiological Model with Nonlinear Incidence,
- [7] J. Math. Biol., 29, 271–287, (1991).
- [8] Hethcote, H. W.: The Mathematics of Infectious Disease, SIAM review 42, 599-653, (2000).
- [9] Jasmine D. and Henry Amirtharaj E.C., Modeling and Simulation of Modified SIR Epidemic Model with Immigration and Non-monotonic Incidence under Treatment, Indian Journal of Applied Research, 3(7), 43-44, 2013.
- [10] Jasmine D. and Henry Amirtharaj E.C., Global Analysis of SIR Epidemic Model with Immigration and Non-monotonic Incidence under Treatment, International Journal of Applied Mathematics and Statistical Research Vol.2(5), 83-92, 2013.
- [11] Kermack, A. McKendrick, Contributions to the mathematical theory of epidemics, Proc. R. Soc.
- [12] Lond., Ser. A, 115, pp. 700–721, 1927.

Paper ID: 020131483

- [13] Liu, W. M., Levin, S. A. and Iwasa, Y.: Influence of Non-linear Incidence Rates upon the Behavior of Simple SIRS Epidemiological Models, J. Math. Biol., 23, 187-204, (1986).
- [14] Mena Lorca J. and H. W. Hethcote., 1992. Dynamic Models of Infection Diseases as Regulators of Population Size, J. Math. Bio, 30:693-716.
- [15] Pathak, S., Maiti, A. and Samanta, G.P. Rich Dynamics of an SIR Epidemic Model, Nonlinear Analysis: Modeling and Control, 15, No. 1, 71–81, (2010).
- [16] Wiggins, S.: Introduction to Applied Nonlinear Dynamical Systems and Chaos, Texts in Applied

- Mathematics, Vol. 2, Springer-Verlag, New York, (2003).
- [17] Xiao D. and Ruan S., Global analysis of an Epidemic model with non monotone incidence rate, Math. Biosci. 208, (2007), 419-429.