International J. of Math. Sci. & Engg. Appls. (IJMSEA)
ISSN 0973-9424, Vol. 10 No. III (December, 2016), pp. 47-55

MATHEMATICAL STUDY OF A DETERMINISTIC SIR EPIDEMIC MODEL WITH MODIFIED NON-LINEAR INCIDENCE RATE

MADHUSUDHAN REDDY K.[1], LAKSHMI NARAYAN K.[2] AND RAVINDRA REDDY B.[3]

[1] Department of Mathematics,
Vardhaman College of Engineering, Shamshabad, Hyderabad, India
[2] Department of Mathematics,
Vignan Institute of Technology & Sciences,
Deshmukhi, Hyderabad, India
[3] Department of Mathematics,
JNTUH College of Engineering, Jagityal, Karimnagar, India

Abstract

This paper makes a modest effort to explore an SIR epidemic model with modified non-linear incidence rate, which details the psychological impact of certain serious diseases on the community when the number of infections progressively registers an increase. The stability of the disease-free and endemic equilibrium is addressed. The global stability of the system was confirmed by deploying Dulac's criterion and applying Poincare-Bendixon theorem. Mat Lab has been applied to evaluate numerical results and it has been observed that the simulated results and analytical results coincide to a larger extent, implying the authenticity and validity of the model.

Key Words : *SIR epidemic model, Non-linear incidence rate, Global stability, Dulac's criterion, Poincare-Bendixon theorem.*

1. Introduction

It is evidently acknowledged by mathematicians and biologists alike that infectious diseases have been the root cause for spreading of infections, a direct effect of microorganisms acting on human body and weakening the immunity system. While the mode of transmission is usually from animal and birds to humans, one cannot rule out the imminent possibility of such infection spreading from humans to fellow humans. An interconnected series of events and factors may be attributed to the spread of such infectious diseases, among which poor hygiene, indiscriminate use of antibiotics, crowded human habitats, ease of travel that makes spread of diseases and several other factors. While bacteriologists and virologists make a point of studying about the occurrence of diseases and a cure for them; mathematicians grapple with the patterns discernible in such occurrences to ensure whether a mathematical model can predict the behavioral patterns of such microbes within the ambit of human understanding. Mathematical modeling meets this need admirably by formulating equations to account for, the spread and possible behavior of such infectious diseases.

The dynamics of infectious diseases is an important research area in mathematical epidemiology. Comprehending the manner of transmission of infectious diseases in communities, regions and countries which may result in better approaches for bringing down the transmission of these diseases. There are plenty of epidemic models in action that satisfactorily explain at what rate and when such diseases set in, and how they should be tackled. These models are defined in mathematical modeling form with respect to status of disease that normally consists of three components: susceptible (S) individuals who are abjectly prone to infection, infected (I) individuals who are in the grip of infection can disseminate the disease to susceptible individuals and recovered (R) individuals who have convalesced and thus out of danger, posing no threat with whom they come in contact with. Such models are known as SIR models. A detailed history of mathematical epidemiology and basics of SIR epidemic models may be found in the classical books of Bailey [2], Murray [3], and Anderson and May [4].The first deterministic SIR model for communicable diseases has been introduced by Kermack and McKendrick [1] in 1927.

Classical epidemic models hold that the incidence rate (rate at which susceptible become infectious) exercises its own role in determining the spread of any disease. Several

different incidence rates have been formulated and investigated by Hethcote [6], Ma et al. [7] and Xiao and Ruan [10], etc. Among the prominent ones, bilinear incidence rate is conspicuous and is denoted by βSI, where S and I stand for the number of the susceptible and infected individuals in the population, and β is a positive constant. Detailed investigation into cholera epidemic which spread in Bari in 1973 led Capasso and Serio [5] to inject the concept of saturated incidence rate of the form $\frac{\beta SI}{1+\alpha_1 I}$, where α_1 is a positive constant. This is significant that the number of the effective contacts between infected and susceptible individuals may saturate at higher infective levels owing to excessive accumulation of infected individuals or on account of preventive measures started off by the susceptible individuals. The suitably changed saturated incidence rate of the form $\frac{\beta SI}{1+\alpha_1 S+\alpha_2 I}$, where α_1, α_2 are positive constants, was proposed by Kaddar [8] and Pathak et al [9]. Finally, the specific nonlinear incidence rate of the form $\frac{\beta SI}{1+\alpha_1 I+\alpha_2 S+\alpha_3 SI}$ was proposed by Jihad Adnani et al [11].

One of the fundamental parameters denotes the spread of diseases, and is also identified with long term behaviors and the level of vaccination necessary for eradication. This parameter goes by the name of basic reproduction number, R_0, which is defined by epidemiologists as the average number of secondary cases caused by an individual infected in a totally susceptible population. When $R_0 > 1$, the disease can penetrate a susceptible population and the number of cases will increase, when $R_0 < 1$, the disease does not aggravate. Therefore, in its primitive form, R_0 throws sufficient evidence in the direction where in a population tends to lose from a specific disease. Nowadays, the result of epidemiological research is specified in terms of basic reproduction number.

In this paper, we have considered a deterministic SIR epidemic model with modified nonlinear incidence rate. The paper has been organized as follows. In section 2, we present the model and derive the disease-free equilibrium and the endemic equilibrium. In section 3, we have discussed stability analysis of the model. Numerical simulations of the system figure in section 4. In conclusion, the findings of the study are discussed in brief in section 5.

2. The Mathematical Model

In this paper, we consider the following SIR epidemic model with a modified nonlinear

incidence rate:

$$\begin{aligned} \frac{dS}{dt} &= b - \frac{\beta SI}{1+\alpha_1 I+\alpha_2 SI} - \mu S \\ \frac{dI}{dt} &= \frac{\beta SI}{1+\alpha_1 I+\alpha_2 SI} - (\mu+\gamma)I \\ \frac{dR}{dt} &= \gamma I - \mu R \end{aligned} \tag{1}$$

where $S(t)$, $I(t)$ and $R(t)$ denote the numbers of susceptible, infective, and recovered individuals at time t, respectively. b denotes the recruitment rate of the population, μ is the natural death rate of the population, γ is the recovery rate of infective individuals, β is the infection coefficient and α_1, α_2 are non negative constants.

The first two equations in system (1) do not depend on the third equation, and therefore this equation can be omitted without loss of generality. Hence, system (1) can be written as

$$\begin{aligned} \frac{dS}{dt} &= b - \frac{\beta SI}{1+\alpha_1 I+\alpha_2 SI} - \mu S \\ \frac{dI}{dt} &= \frac{\beta SI}{1+\alpha_1 I+\alpha_2 SI} - (\mu+\gamma)I. \end{aligned} \tag{2}$$

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

$$\frac{dN}{dt} = b - \mu N - \gamma I. \tag{3}$$

We shall show that the system (2) is uniformly bounded by the following theorem.

Theorem 1 : The solutions of (2) are eventually confined in the compact subset

$$\Gamma = \left\{ (S,I) \in R_+^2 : S \geq 0, S+I \leq \frac{b}{\mu} \right\}.$$

Proof : Let $S(t), I(t)$ be any solution of (2) with initial conditions $S(0) = S_0, I(0) = I_0$. From Eq. (3) it follows that $\frac{dN}{dt} \leq b - \mu N$.

Applying the theory of differential inequalities, we obtain

$$N(S,I) \leq \frac{b}{\mu}\left(1 - e^{-\mu t}\right) + N(S_0, I_0)e^{-\mu t},$$

and for $t \to \infty$, we have $\limsup_{t\to\infty} N \leq \frac{b}{\mu}$.

Hence all the solutions of (2) are eventually confined in Γ. This completes the proof.

System (2) always has a disease-free equilibrium $E_0(b/\mu, 0, 0)$.

To find the endemic equilibrium, set

$$\begin{aligned} b - \frac{\beta SI}{1+\alpha_1 I+\alpha_2 SI} - \mu S = 0 \\ \frac{\beta S}{1+\alpha_1 I+\alpha_2 SI} - (\mu+\gamma) = 0 \end{aligned} \tag{4}$$

This yields

$$\alpha_2(\mu+\gamma)^2 I^2 - (\mu+\gamma)(\mu\alpha_1 + b\alpha_2 + \beta)I + b\beta - \mu(\mu+\gamma) = 0. \tag{5}$$

Define the basic reproduction number as follows

$$R_0 = \frac{b\beta}{\mu(\mu+\gamma)}. \tag{6}$$

From Eq. (5), we can observe that

(i) if $R_0 \leq 1$, then there is no positive equilibrium;

(ii) if $R_0 > 1$, then there is a unique positive equilibrium (endemic equilibrium) $E^*(S^*, I^*)$, given by

$$S^* = \frac{b(1+\alpha_1 I^*)}{\mu R_0 - b\alpha_2 I^*}, \quad I^* = \frac{(\mu\alpha_1 + b\alpha_2 + \beta) + \sqrt{\Delta}}{2(\mu+\gamma)\alpha_2}$$

where $\Delta = (\mu\alpha_1 + b\alpha_2 + \beta)^2 + 4\alpha_2\mu(\mu+\gamma)(R_0 - 1)$.

3. Stability Analysis

Theorem 2 : The disease-free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$ and it is unstable if $R_0 > 1$.

Proof : The Jacobian matrix of system (2) is given by

$$J = \begin{pmatrix} -\frac{(1+\alpha_1 I)\beta I}{(1+\alpha_1 I+\alpha_2 SI)^2} - \mu & -\frac{\beta S}{(1+\alpha_1 I+\alpha_2 SI)^2} \\ \\ \frac{(1+\alpha_1 I)\beta I}{(1+\alpha_1 I+\alpha_2 SI)^2} & \frac{\beta S}{(1+\alpha_1 I+\alpha_2 SI)^2} - (\mu+\gamma) \end{pmatrix} \tag{7}$$

The Jacobian matrix at E_0 is

$$J(E_0) = \begin{pmatrix} -\mu & -\frac{b\beta}{\mu} \\ \\ 0 & \frac{b\beta}{\mu} - (\mu+\gamma) \end{pmatrix}.$$

At E_0 the characteristic equation is

$$(\lambda+\mu)\left[\lambda - \frac{b\beta}{\mu} - (\mu+\gamma)\right] = 0. \tag{8}$$

Obviously, (8) has two roots $\lambda_1 = -\mu < 0$ and $\lambda_2 = \frac{b\beta}{\mu} - (\mu+\gamma)$. Hence, E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Theorem 3 : The endemic equilibrium E^* is locally asymptotically stable if $R_0 > 1$.

Proof : The Jacobian matrix of system (2) at E^* is given by

$$J(E^*) = \begin{pmatrix} -\frac{(1+\alpha_1 I^*)\beta I^*}{(1+\alpha_1 I^*+\alpha_2 S^* I^*)^2} - \mu & -\frac{\beta S^*}{(1+\alpha_1 I^*+\alpha_2 S^* I^*)^2} \\ \\ \frac{(1+\alpha_1 I^*)\beta I^*}{(1+\alpha_1 I^*+\alpha_2 S^* I^*)^2} & \frac{\beta S^*}{(1+\alpha_1 I^*+\alpha_2 S^* I^*)^2} - (\mu+\gamma) \end{pmatrix}$$

that can be written as

$$J(E^*) = \begin{pmatrix} -\frac{(1+\alpha_1 I^*)\beta I^*}{(1+\alpha_1 I^*+\alpha_2 S^* I^*)^2} - \mu & -\frac{(\mu+\gamma)}{1+\alpha_1 I^*+\alpha_2 S^* I^*} \\ \\ \frac{(1+\alpha_1 I^*)\beta I^*}{(1+\alpha_1 I^*+\alpha_2 S^* I^*)^2} & -\frac{(\mu+\gamma)(\alpha_1 I^*+\alpha_2 S^* I^*)}{1+\alpha_1 I^*+\alpha_2 S^* I^*} \end{pmatrix}$$

when we take into account the following identity, which is obtained by the endemic equilibrium

$$\mu + \gamma = \frac{\beta S^*}{1 + \alpha_1 I^* + \alpha_2 S^* I^*}. \tag{9}$$

The trace of $J(E^*)$ is

$$tr(J(E^*)) = -\left[\mu + \frac{(1 + \alpha_1 I^*)\beta I^* + (\mu+\gamma)(\alpha_1 I^* + \alpha_2 S^* I^*)(1 + \alpha_1 I^* + \alpha_2 S^* I^*)}{(1 + \alpha_1 I^* + \alpha_2 S^* I^*)^2}\right] < 0.$$

Thus $tr(J(E^*)) < 0$.

The determinant of $J(E^*)$ is

$$det(J(E^*)) = \frac{(\mu+\gamma)[(1 + 2\alpha_1 I^* + \alpha_2 S^* I^*)\beta I^* + \mu(\alpha_1 I^* + \alpha_2 S^* I^*)(1 + \alpha_1 I^* + \alpha_2 S^* I^*)^2]}{(1 + \alpha_1 I^* + \alpha_2 S^* I^*)^3} > 0.$$

Thus $det(J(E^*)) > 0$. Here, the eigen values of the Jacobian matrix $J(E^*)$ have negative real parts. Hence the endemic equilibrium E^* is locally asymptotically stable if $R_0 > 1$.

Theorem 4 : If $R_0 > 1$, the unique endemic equilibrium $E^*(S^*, I^*)$ is globally asymptotically stable in the interior of Γ.

Proof : Let us denote

$$P(S, I) = b - \frac{\beta SI}{1 + \alpha_1 I + \alpha_2 SI} - \mu S; \quad Q(S, I) = \frac{\beta SI}{1 + \alpha_1 I + \alpha_2 SI} - (\mu + \gamma)I.$$

Take the Dulac function $D(S, I) = \frac{1+\alpha_1 I+\alpha_2 SI}{\beta SI}, S > 0, I > 0$. Then we have

$$\frac{\partial(DP)}{\partial S} + \frac{\partial(DQ)}{\partial I} = -\frac{b(1 + \alpha_1 I)}{\beta S^2 I} - \frac{(\mu+\gamma)\alpha_1}{\beta S} - \frac{(2\mu+\gamma)\alpha_2}{\beta} < 0.$$

Thus, system (2) does not have a limit cycle in the interior of Γ. From Theorem 3, if $R_0 > 1$ holds then E^* is locally asymptotically stable. A simple application of the classical Poincare-Bendixon theorem and Theorem 1, it suffices to show that the unique endemic equilibrium E^* is globally asymptotically stable in the interior of Γ. This completes Theorem 4.

4. Numerical Simulations

1. The parameters in the model (1) are taken as

$$b = 0.48; \mu = 0.023; \beta = 0.564; \alpha_1 = 0.056; \alpha_2 = 0.0032; \gamma = 0.965;$$

$$S^* = 1.841; I^* = 0.111; S(0) = 0.92; I(0) = 0.2; \text{ and } R_0 = 11.913 > 1 (Fig.1)$$

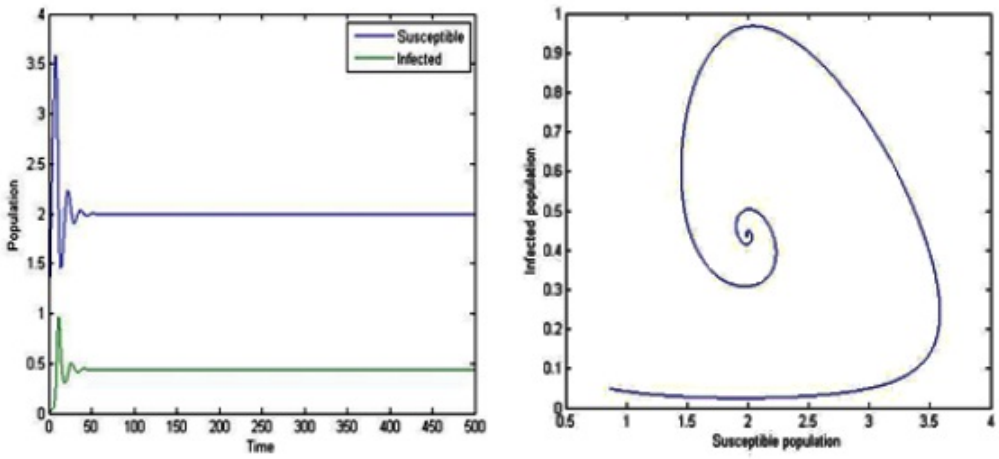

Fig.1: The deterministic trajectories and phase -portraits of system

2. The parameters in the model (1) are taken as

$$b = 0.02; \mu = 0.003; \beta = 0.564; \alpha_1 = 0.002; \alpha_2 = 0.006; \gamma = 0.965;$$

$$S^* = 1.717; I^* = 0.004; S(0) = 0.92; I(0) = 0.2; \text{ and } R_0 = 3.884 > 1 (Fig.2)$$

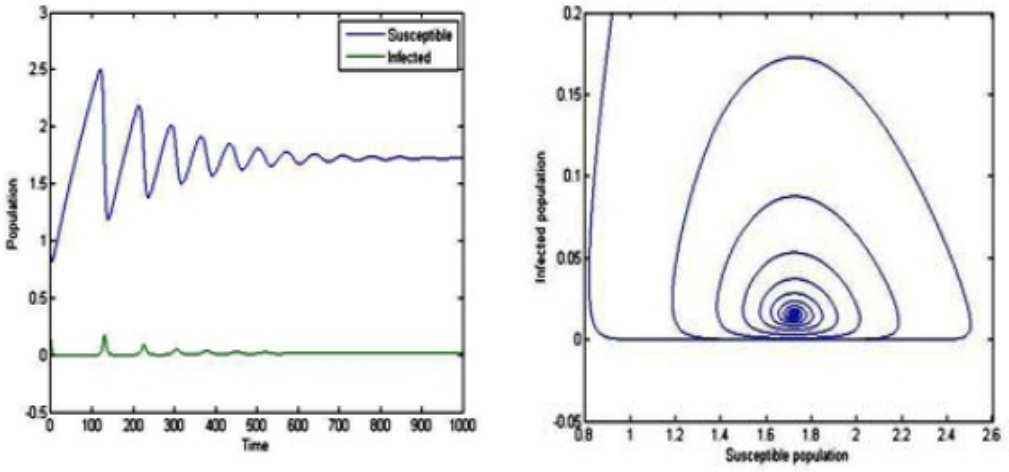

Fig.2: The deterministic trajectories and phase-portraits of system

5. Concluding Remarks

In this paper, we considered a deterministic SIR epidemic model with modified nonlinear incidence rate. Our analysis establishes that the global stability of the SIR epidemic model is totally determined by the basic reproduction number R_0. When $R_0 < 1$, the disease-free equilibrium is globally asymptotically stable in the feasible region. The proof is based on the construction of a Dulac function and applying Dulac's criterion and the Pioncare-Bendixson theorem as used by C. Vargas - De-Leon [12]. If $R_0 > 1$, a unique endemic equilibrium exists and is globally stable in the interior of the feasible region. Numerical results also support our analytical results.

References

[1] Kermack W. O. and McKendrick A. G., A contribution to mathematical theory of epidemics, Proceedings of Royal Society: Mathematical, Physical and Engineering Sciences, 115(772) (1927), 700-721.

[2] Bailey N. T. J., The Mathematical Theory of Infectious Diseases, Griffin, London (1975).

[3] Murray J. D., Mathematical Biology, Springer-Verlag, New York, (1975).

[4] Anderson R. M. and May R. M., Infectious Diseases of Humans: Dynamics and Control, Oxford University Press, Oxford, (1998).

[5] Capasso V. and Serio G., A generalization of the Kermack-McKendrick deterministic epidemic model, Mathematical Biosciences, 42 (1978), 43-61.

[6] Hethcote H. W., The mathematics of infectious disease, SIAM Review, 42(4) (2000), 599-653.

[7] Ma W., Song M. and Takeuchi Y., Global stability of an SIR epidemic model with time delay, Applied Mathematical Letters, 17 (2004), 1141-1145.

[8] Kaddar A., On the dynamics of a delayed SIR epidemic model with a modified saturated incidence rate, Electronic Journal of Differential Equations, 2009(133) (2009), 1-7.

[9] Pathak S., Maiti A. and Samanta G. P., Rich dynamics of an SIR epidemic model, Nonlinear Analysis: Modelling and Control, 15(1) (2010), 71-81.

[10] Xiao D. and Ruan S., Global analysis of an epidemic model with nonmonotone incidence rate, Mathematical Biosciences, 208 (2007), 419-429.

[11] Jihad Adnani, Khalid Hattaf and NouraYousfi., Stability analysis of a stochastic SIR epidemic model with specific nonlinear incidence rate, International Journal of Stochastic Analysis, 2013 (2013), 1-4.

[12] Vargas C-De-Leon., Stability analysis of a SIS epidemic model with standard incidence, Foro-Red-Mat: Revista Electronica de Contenido Matematico, 28(4) (2011), 1-11.

[13] Hale J. K., Ordinary Differential Equations, John Wiley & Sons, New York, (1969).