Jñānābha, **Vol. 50(2) (2020), 75-83**
(Dedicated to Honor Dr. R. C. Singh Chandel on His 75th Birth Anniversary Celebrations)

DYNAMICS OF AN SIRS EPIDEMIC MODEL WITH PARTICULAR NON-LINEAR INCIDENCE RATE AND MEDIA EFFECTS

By
Garima Saxena
Institute of Engineering and Technology
Devi Ahilya Vishwavidyalaya
Indore-452020, Madhya Pradesh, India
Email:rimasa.1907@gmail.com
R. K. Sharma
Department of Mathematics, Government Holkar Science College
Devi Ahilya Vishwavidyalaya
Indore-452001, Madhya Pradesh, India
Email:raj_rma@yahoo.co.in
Ankit Agrawal
Department of Mathematics, Acropolis Institute of Technology and Research, Indore-453771, Madhya Pradesh, India
Email:ankit_agrawal458@yahoo.com

(*Received : May* 24, 2020*; Revised : September* 26, 2020*; Final Form : December* 18, 2020)

DOI: https://doi.org/10.58250/jnanabha.2020.50209

Abstract

This paper deals with the nonlinear dynamics of Susceptible, Infected, Recovered epidemic model with a particular non linear incidence rate and the consequences of media awareness program. The model analysis shows that the spread of an infectious disease can be controlled by using awareness programs. Different equilibrium points and their stability are discussed. The basic reproduction number R_0 is obtained. We also apply Lasalle's invariance principle to show that the disease-free equilibrium is globally asymptotically stable if $R_0 < 1$ and we use a geometric approach to find out the global stability of the endemic equilibrium. In addition, to our analytical results, several numerical simulations are also illustrated.

2010 Mathematics Subject Classifications: 34D20, 34D23, 92B05, 92D05.
Keywords and phrases: social distancing, wearing protective masks, geometric approach, stability, media, COVID-19.

1 Introduction

Mathematical models of infectious diseases are proven to be very important in better understanding of epidemiological patterns and disease control in human populations. In the study of the epidemiological models, incidence rate play a very important role while controlling the transmission of infectious diseases. Epidemic models with different types of incidence rate have been studied and developed by many authors. In order to model this disease transmission process many authors use the incidence functions: The earliest one is the bilinear incidence rate λSI used by Kermack and Mckendrick [7] in 1927. In 1978, Capasso and Serio [3] introduced a saturated incidence rate $\frac{\lambda I}{1+\alpha I}$ by research of the Cholera epidemic spread in Bari. Also in 1978, May and Anderson [1] proposed the saturated incidence rate $\frac{\lambda SI}{1+\alpha S}$. The general incidence rate $\frac{\lambda I^p S}{1+\alpha I^q}$ was proposed by Liu et. al. [10, 11] in 1986-87, Derick and Ven Den Driessche [4] in 1993, etc. Ruan and Wang [17] studied an epidemic model with a specific nonlinear incidence rate $\frac{\lambda I^2 S}{1+\alpha I^2}$ and presented a detailed quantitative analysis and bifurcation analysis and Bogdanov-Takens bifurcation for the model in 2003. Xiao and Zhou [20] considered the non-monotone incidence rate $\frac{\lambda IS}{1+\beta I+\alpha I^2}$ in 2006. To model the effects of psychological factor, protection measures and intervention policies when a serious disease emerges, Xiao and Ruan [21] proposed the specific incidence rate $\frac{\lambda IS}{1+\alpha I^2}$ in 2007.

Controlling infectious diseases has been an increasingly more and more complex issue in recent years. In the field of epidemiology, treatment, vaccination, isolation, media awareness program and many more play a crucial role in controlling the disease spread. Media is can be helpful to develop the awareness among in common people regarding the rich nature of the disease, make people knowledgeable about the disease to take precautions such as social distancing, wearing protective masks, vaccination etc., to reduce their probabilities of being infected and some other impacts. It is observed that, media coverage gives rise to healthy behaviour among the population. Few research works on media coverage can be found in [8, 9, 12, 16, 18]. However, mathematical models to study the disease transmission dynamics together with media effect is still largely remain unexplored.

In the present research, we intend to study the influence of media coverage to control and eradicate the disease with a particular non-linear incidence function $U(S,I) = \frac{\lambda S I}{1+\alpha_1 S+\alpha_2 I^2}$ used by [2], which has the property of being saturated with infectives as well as with susceptible individuals. We can see that

$$\frac{\partial U(S,I)}{\partial I} = \frac{\lambda S(1+\alpha_1 S-\alpha_2 I^2)}{(1+\alpha_1 S+\alpha_2 I^2)^2},$$

which is positive when $I^2 < \frac{(1+\alpha_1 S)}{\alpha_2}$ and negative when $I^2 > \frac{(1+\alpha_1 S)}{\alpha_2}$. Hence, $U(S,I)$ is a non-monotonic function with respect to I, since it increases when the number of infectives is relatively small but decreases as the number of infectives becomes larger. On the other hand

$$\frac{\partial U(S,I)}{\partial S} = \frac{\lambda I(1+\alpha_2 I^2)}{(1+\alpha_1 S+\alpha_2 I^2)^2} > 0,$$

so $U(S,I)$ grows monotonically with respect to susceptibles.

This kind of non-linear and non-monotonic incidence function models the idea that, at the beginning of the infection, the population has little awareness of preventive measures, so the contact rate increases rapidly. As time advances, media reporting on early stage symptoms of the disease, the population becomes more aware of the risk and takes measures to control or eradicate the disease, so the number of infectious contacts decreases.

This manuscript is organized as follows: In ***Sect.*2**, $SIRS$ model is presented. In ***Sect.*3**, basic properties of solutions are discussed. In ***Sect.*4**, we calculate the basic reproduction number then in ***Sect.*5**, we determine all possible equilibria of model. In ***Sect.*6**, we discuss and analyze the local stability of the equilibriums. In ***Sect.*7**, we discuss and analyze the global stability of the equilibriums. We present in ***Sect.*8**, some numerical examples of the dynamics of the model. Finally, in ***Sect.*9**, swe discussed the conclusion.

2 Model Formulation

In this section, deterministic nonlinear $SIRS$ model is considered by taking media awareness and particular incidence rate into account. The variables and parameters of the model are described in ***Table* 2.1** and ***Table* 2.2** respectively.

***Table* 2.1:** Description of the model state variables.

State variables	Description
$S(t)$	Number of susceptible individuals at time t
$I(t)$	Number of infected individuals at time t
$R(t)$	Number of recovered individuals at time t
$N(t)$	The total population size at time t

To model the situation considered a region with total population $N(t)$ at any instant of time t. By taking into account the aforementioned considerations, the system of equations that capture the dynamics of the infectious disease is designed and the ordinary differential equations of the system (2.1) is as follows.

$$\text{(2.1)}\quad \begin{cases} \dfrac{dS}{dt} = a - dS - \dfrac{\lambda S I}{1+\alpha_1 S+\alpha_2 I^2} + \beta R - pSM \\ \dfrac{dI}{dt} = \dfrac{\lambda S I}{1+\alpha_1 S+\alpha_2 I^2} - (d+\delta)I \\ \dfrac{dR}{dt} = \delta I - (d+\beta)R + pSM, \end{cases}$$

whose state space is the first quadrant $R_3^+ = \{(S,\ I,\ R) : S \geq 0,\ I \geq 0,\ R \geq 0\}$ and subject to the initial conditions $S(0) = S_0 \geq 0$, $I(0) = I_0 \geq 0$, $R(0) = R_0 \geq 0$. It is assumed that all the parameters are positive.

***Table* 2.2:** Description of the model parameters.

State parameters	Description
a	Recruitment rate of the population
d	The natural death rate of the population
λ	The effective contact rate
$\alpha_1 \& \alpha_2$	The parameter measures of the psychological or inhibitory effect
β	The rate at which recovered individuals lose immunity and return to susceptible class
δ	The natural recovery rate of infection
p	The dissemination rate of awareness among unaware susceptible due to which they form a different class
M	The media control parameter (fixed)

3 Basic Properties of the Model

Summing up the four equations of model (2.1) and denoting

$$N(t) = S(t) + I(t) + R(t),$$

having

$$N'(t) = a - dN.$$

If disease is not present, then $N'(t) = a - dN$. This shows that population size $N \to \frac{a}{d}$ as $t \to \infty$. It follows that the solutions of model (2.1) exists in the region defined by

$$\Omega = \{(S,\ I,\ R) \in R_4^+ : S,\ I,\ R \geq 0,\ S + I + R \leq a/d\}. \tag{3.1}$$

This gives the following lemma which shows that the solutions of model (2.1) are bounded, continuous for all positive time and lie in a compact set.

***Lemma* 3.1** *The set* Ω *defined in* (3.1) *is a positively invariant region for model* (2.1)*. Moreover, every trajectory of model* (2.1) *is eventually staying in a compact subset of* Ω*.*

4 Basic Reproductive Number

The basic reproduction number sometimes called basic reproductive rate or basic reproductive ratio is one of the most useful threshold parameters which characterize mathematical problems concerning infectious diseases. This metric is useful because it helps determine whether or not an infectious disease will spread through a population. In this section, we will calculate the basic reproduction number R_0 of system (2.1) by using the next-generation matrix method described in [19]. For that, we rewrite model (2.1) as

$$\frac{dx}{dt} = F(x) - \mathfrak{V}(x),$$

where $x = (I, R, S)$,

$$F(x) = \begin{pmatrix} \frac{\lambda S I}{1+\alpha_1 S+\alpha_2 I^2} \\ 0 \\ 0 \end{pmatrix} \text{ and } \mathfrak{V}(x) = \begin{pmatrix} (\gamma + \delta + d + d_1)I \\ -\delta I + (\mu + d + d_2)Q \\ -\gamma I - \mu Q + dR \\ -A + dS + \frac{\beta S I}{(1+\alpha_1 S)(1+\alpha_2 I)} \end{pmatrix}.$$

We calculate the Jacobian matrices for $F(x)$ and $\mathfrak{V}(x)$ at the disease-free equilibrium $x_0 = (0, 0, a/d + pM)$.

$$F = \begin{pmatrix} \frac{\lambda a}{\alpha_1 a+d+pM} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},\ V = \begin{pmatrix} d+\delta & 0 & 0 \\ -\delta & d+\beta & -pM \\ \frac{\lambda a}{d+pM+\alpha_1 a} & -\beta & d+pM \end{pmatrix}.$$

FV^{-1} is the next generation matrix for model (2.1). It then follows that the spectral radius of matrix FV^{-1} is $\rho(FV^{-1}) = \frac{\lambda a}{(a\alpha_1+d+pM)(d+\delta)}$. Thus, the basic reproduction number of model (2.1) is

$$R_0 = \frac{\lambda a}{(a\alpha_1 + d + pM)(d + \delta)}.$$

5 Existence of Equilibria

In this section, we obtain the existence of the disease-free equilibrium E_0 and the endemic equilibrium E^* of model (2.1).

Set the right sides of model (2.1) equal zero, that is,

$$(5.1)\quad \begin{cases} a - dS - \dfrac{\lambda S I}{1 + \alpha_1 S + \alpha_2 I^2} + \beta R - pSM = 0 \\ \dfrac{\lambda S I}{1 + \alpha_1 S + \alpha_2 I^2} - (d+\delta)I = 0 \\ \delta I - (d+\beta)R + pSM = 0. \end{cases}$$

The model (2.1) always has the disease-free equilibrium point $E_0(\frac{a}{d+pM}, 0, 0)$. Solving (5.1) we also get a unique positive, endemic equilibrium point $E^*(S^*, I^*, R^*)$ of the model (2.1), where

$$S^* = \frac{(d+\delta)(1+\alpha_2 I^2)}{\lambda - \alpha_1(d+\delta)},$$
$$R^* = \frac{\delta I + pSM}{(d+\beta)},$$

and I^* is given as a root of the quadratic equation $\Omega_1 I^2 + \Omega_2 I + \Omega_3 = 0$,
where

$$\Omega_1 = -\alpha_2 d(d+\delta)(d+\beta+pM),$$
$$\Omega_2 = -(d^2 + d\beta + \delta d)(\lambda - \alpha_1 d - \alpha_1 \delta),$$
$$\Omega_3 = a(d+\beta)[\lambda - (d+\delta)\alpha_1] + (d+\delta)[-pMd - d(d+\beta)].$$

Now,

$$I^* = \frac{(d^2 + d\beta + \delta d)(\lambda - \alpha_1 d - \alpha_1\delta) + \sqrt{\Delta}}{-2\alpha_2 d(d+\delta)(d+\beta+pM)},$$

where,

$$\Delta^2 = [-(d^2 + d\beta + \delta d)(\lambda - \alpha_1 d - \alpha_1\delta)]^2 - 4[\{-\alpha_2 d(d+\delta)(d+\beta+pM\}a(d+\beta)\times$$
$$\{\lambda - (d+\delta)\alpha_1\} + (d+\delta)\{-pMd - d(d+\beta)\}].$$

6 Local Stability Analysis

In this section, we study the local stability of the disease-free equilibrium E_0 and the endemic equilibrium E^* of model (2.1).

***Theorem* 6.1** *If $R_0 < 1$, the disease-free equilibrium E_0 of model* (2.1) *is locally asymptotically stable. If $R_0 > 1$, the disease-free equilibrium E_0 is unstable.*

Proof. The Jacobian matrix of model (2.1) at the disease-free equilibrium E_0 is

$$J(E_0) = \begin{pmatrix} -d - pM & \dfrac{-\lambda a}{d + pM + \alpha_1 a} & \beta \\ 0 & \dfrac{\lambda a}{d + pM + \alpha_1 a} - (d+\delta) & 0 \\ pM & \delta & -(d+\beta) \end{pmatrix}.$$

The characteristic equation of $J(E_0)$ is

$$\left\{\{\frac{\lambda a}{d + pM + \alpha_1 a} - d - \delta - \mu\}\{\mu^2 + (2d + pM + \beta)\mu + (d^2 + d\beta + pMd)\}\right\} = 0.$$

Clearly, the one eigenvalue $\mu_1 = \frac{\lambda a}{d+pM+\alpha_1 a} - (d+\delta)$ and other two eigenvalues are given by the quadratic equation

$$\mu^2 + (2d + pM + \beta)\mu + (d^2 + d\beta + pMd) = 0$$

or

$$\mu^2 + \mu\psi_1 + \psi_2 = 0, A_0 \neq 0,$$

where $\psi_1 = 2d + pM + \beta$, $\psi_2 = d^2 + d\beta + pMd$.

By Routh-Hurwitz criteria, we know that the model is stable if $\psi_1 > 0$ and $\psi_2 > 0$, while $\mu_1 < 0$ for $R_0 < 1$ and $\mu_1 > 0$ for $R_0 > 1$.

Hence E_0 is locally asymptotically stable for $R_0 < 1$, while it is unstable for $R_0 > 1$.

***Theorem* 6.2** *If $R_0 > 1$, the endemic equilibrium E^* of model* (2.1) *is locally asymptotically stable.*

Proof. Consider

$$J(E^*) = \begin{pmatrix} -V_1 - d - pM & -V_2 & \beta \\ V_1 & V_2 - (d+\delta) & 0 \\ pM & \delta & -(d+\beta) \end{pmatrix},$$

where $V_1 = \frac{(1+\alpha_1 S^* + \alpha_2 I^{*2})\lambda I^* - \lambda S^* I^* \alpha_1}{(1+\alpha_1 S^* + \alpha_2 I^{*2})^2}$, $V_2 = \frac{(1+\alpha_1 S^* + \alpha_2 I^{*2})\lambda S^* - 2\lambda S^* I^{*2}\alpha_2}{(1+\alpha_1 S^* + \alpha_2 I^{*2})^2}$.

The characteristic equation of $J(E^*)$ is

$$\mu^3 + \mu^2 A_1 + \mu A_2 + A_3 = 0, A_0 \neq 0,$$

where

$$A_1 = (3d + \delta + \beta + pM + v_1 - v_2),$$

$$A_2 = (d^2 + \beta v_1 + pMv_2 + 2\beta pM + 2d\beta + \beta\delta - \delta v_1 - \beta v_2 - \delta pM),$$

$$A_3 = (d+\beta)(dv_2 + pMv_2 - d^2 - dpM - dv_1 - d\delta - \delta pM - \delta v_1) + \beta v_1 \delta + \beta pM(d + \delta - v_2).$$

We know that $A_1 > 0$ if $3d+\delta+\beta+pM+v_1 > v_2$ and $A_2 > 0$ if $(d^2+\beta v_1+pMv_2+2\beta pM+2d\beta+\beta\delta) > (\delta v_1+\beta v_2+\delta pM)$. By Routh-Hurwitz criteria, endemic equilibrium E^* of model (2.1) is locally asymptotically stable if and only if $A_1 > 0$, $A_2 > 0$ and $A_1 A_2 > A_0 A_3$.

7 Global Stability Analysis

In this section, we study the global stability of the disease-free equilibrium E_0 and the endemic equilibrium E^* of model (2.1).

***Theorem* 7.1** *If $R_0 < 1$, the disease-free equilibrium E_0 of model* (2.1) *is globally asymptotically stable.*

Proof. We prove the global stability of the model (2.1) at the equilibrium E_0 when $R_0 < 1$. Taking the Lyapunov function

$$V(S, I, R) = I(t).$$

Calculating the derivative of $V(t)$ along the positive solution of model (2.1), it follows that

$$\frac{dV}{dt} = \frac{dI}{dt} = \frac{\lambda S I}{(1 + \alpha_1 S + \alpha_2 I^2)} - (d+\delta)I.$$

Since the incidence function

$$\frac{\lambda S I}{(1 + \alpha_1 S + \alpha_2 I^2)} \leq \frac{\frac{\lambda I a}{d+pM}}{1 + \frac{\alpha_1 a}{d+pM} + \alpha_2 I^2}$$

for $0 \leq S \leq \frac{a}{d+pM}$,

$$\dot{V}(t) \leq \left[\frac{\lambda a}{(d + pM + \alpha_1 a)} - (d+\delta)\right] I$$

$$= (d+\delta)\,[R_0 - 1]\, I \leq 0.$$

Furthermore, $\dot{V} = 0$ only if $I = 0$, so the largest invariant set contained $\{(S,\ I,\ R) \in \Omega : \dot{V} = 0\}$ is the plane $I = 0$. By Lassalle's invariance principle [13], this implies that all solution in Ω approach the plane $I = 0$ as $t \to \infty$. On the other hand, solutions of (2.1) contained in such plane satisfy $\frac{dS}{dt} = a - dS + \beta R - pS\,M$, $\frac{dR}{dt} = -(d+\beta)R + pS\,M$, which implies that $S \to \frac{a}{d+pM}$ and $R \to 0$ as $t \to \infty$, that is, all of these solutions approach E_0 is globally asymptotically stable in Ω.

Next, we analysis the global stability of an endemic equilibrium E^* by using geometric approach method described by Li and Muldowney in [14]. For that, we need to consider a parameter

$$w = \max\left\{ \begin{array}{c} -pM - \frac{\lambda I}{1+\alpha_1 S+\alpha_2 I^2}(1 - \frac{S(\alpha_1 - 2\alpha_2 I)}{1+\alpha_1 S+\alpha_2 I^2}) + \beta, \delta(2-p) - \beta - \\ \frac{\lambda}{1+\alpha_1 S+\alpha_2 I^2}(S + I - \frac{SI\alpha_1}{1+\alpha_1 S+\alpha_2 I^2}), -\beta + \delta - \frac{-2\lambda S\alpha_2 I^2}{1+\alpha_1 S+\alpha_2 I^2} \end{array} \right\},$$

and we will make use of the following ***Theorem***.

***Theorem* 7.2** *(**Li Muldowney** [14]). Suppose that the system $x' = f(x)$, with $f : D \subset \mathbb{R}^n \to \mathbb{R}^n$, satisfies the following:*
(H1) D is a simply connected open set,
(H2) there is a compact absorbing set $K \subset D$,
(H3) x^* is the only equilibrium in D.
Then the equilibrium x^* is globally stable in D if there exists a Lozinskiĭ measure ηsuch that

$$\lim_{t\to\infty} \sup \sup_{x_0\in K} \frac{1}{t}\int_0^t \eta(B(x(s,x_0)))ds < 0, \tag{7.1}$$

$$B = P_f P^{-1} + PJ^{[2]}P^{-1} \tag{7.2}$$

and $Q \to Q(x)$ is an $\binom{n}{2} \times \binom{n}{2}$ matrix valued function.

In our case, model (2.1) can be written as $x' = f(x)$ with $f : D \subset \mathbb{R}^n \to \mathbb{R}^n$ and D being the interior of the feasible region Ω. The existence of a compact absorbing set $K \subset D$ is equivalent to proving that (2.1) is uniformly persistent (see [14, 5]) and the proof for this in the case when $R_0 > 1$ is similar to that of proposition 4.2 of [14]. Hence, $(H1)$ and $(H2)$ hold for system (2.1), and by assuming the uniqueness of the endemic equilibrium in D, we can prove its global stability with the aid of ***Theorem* 7.2**.

***Theorem* 7.3** *If $R_0 > 1$, $d < w$ and the endemic equilibrium E^* of system* (2.1) *is unique, then E^* is globally asymptotically stable in the feasible region Ω.*

Proof. Let J be the Jacobian matrix of the system (2.1). Then the second additive compound matrix [15] of J is given by

$$J^{[2]} = \begin{pmatrix} J_{11}+J_{22} & J_{23} & -J_{13} \\ J_{32} & J_{11}+J_{33} & J_{12} \\ -J_{31} & J_{21} & J_{22}+J_{33} \end{pmatrix},$$

$$J^{[2]} = \begin{pmatrix} -d-pM-v_1+v_2-(d+\delta) & 0 & -\beta \\ \delta & -d-pM-v_1+(-d-\beta) & -v_2 \\ -pM & v_1 & v_2-(d+\delta)-(d+\beta) \end{pmatrix},$$

where, $v_1 = \frac{(1+\alpha_1 S+\alpha_2 I^2)\lambda I - \lambda S I \alpha_1}{(1+\alpha_1 S+\alpha_2 I^2)^2}$, $v_2 = \frac{(1+\alpha_1 S+\alpha_2 I^2)\lambda S - 2\lambda S I^2 \alpha_2}{(1+\alpha_1 S+\alpha_2 I^2)^2}$.

Let P be the matrix-valued function defined by $P = P(S,I,R) = \mathrm{diag}(\frac{S}{I}, \frac{S}{I}, \frac{S}{I})$; then P is C^1 and non-singular in the interior of Ω, $P_f = \mathrm{diag}(\frac{S'I-SI'}{I^2}, \frac{S'I-SI'}{I^2}, \frac{S'I-SI'}{I^2})$ and $P^{-1} = \mathrm{diag}(\frac{I}{S}, \frac{I}{S}, \frac{I}{S})$, $P_f P^{-1} = \mathrm{diag}(\frac{S'}{S} - \frac{I'}{I}, \frac{S'}{S} - \frac{I'}{I}, \frac{S'}{S} - \frac{I'}{I})$ and $B = P_f P^{-1} + PJ^{[2]}P^{-1}$. Then B can be written in the block form

$$B = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix},$$

with $B_{11} = \frac{S'}{S} - \frac{I'}{I} - d - pM - v_1 + v_2 - (d+\delta)$, $B_{12} = (0, -\beta)$, $B_{21} = \begin{pmatrix} \delta \\ -pM \end{pmatrix}$ and

$$B_{22} = \begin{pmatrix} \frac{S'}{S} - \frac{I'}{I} - d - pM - v_1 + (-d-\beta) & -v_2 \\ v_1 & \frac{S'}{S} - \frac{I'}{I} + v_2 - (d+\delta) - (d+\beta) \end{pmatrix}.$$

Consider the vector norm in R^3 defined by $\|(u,v,w)\| = \max\{|u|, |v|+|w|\} \in R^3$ and let $\eta_1(B)$ be the *Lozinskiĭ* measure with respect to this norm. Then

$$\eta_1(B) \le \sup\{g_1, g_2\}, \tag{7.3}$$

where, $g_1 = (B_{11}) + |B_{12}|$, $g_2 = \mu(B_{22}) + |B_{21}|$, $|B_{12}|$ and $|B_{21}|$ denote the matrix norm with respect to l_1 vector norm in norm R^2 and η_1 is the *Lozinskiĭ* measure of B_{22} with respect to l_1 vector norm in R^2. We have $|B_{12}| = 0$, $|B_{21}| = \delta$, $\mu(B_{22}) = \frac{S'}{S} - \frac{I'}{I} - d + \max\{-pM - v_1 - d - \beta, v_2 - d - \delta - \beta\}$. From the second equation in the system (2.1), we have

$$\frac{I'}{I} = \frac{\beta S}{(1+\alpha_1 S)(1+\alpha_2 I)} - (\gamma + \delta + d + d_1 + q).$$

Therefore,

$$\mu(B_{22}) = g_2 = S'/S - d + \max\left\{\delta - pM - \beta - \frac{\lambda}{(1+\alpha_1 S+\alpha_2 I^2)}(S + I - \frac{SI\alpha_1}{(1+\alpha_1 S+\alpha_2 I^2)}\right.$$

$$-\beta - \frac{2\lambda S I^2 \alpha_2}{(1+\alpha_1 S+\alpha_2 I^2)^2}\Big\}.$$

Then

$$g_1 = S'/S - (d+pM) - \frac{\lambda I}{(1+\alpha_1 S+\alpha_2 I^2)}(1 - \frac{S[(\alpha_1 - 2\alpha_2 I)]}{[(1+\alpha_1 S+\alpha_2 I^2)]} + \beta,)$$

$$g_2 = S'/S - d + \max\Big\{\delta - pM - \beta - \frac{\lambda}{(1+\alpha_1 S+\alpha_2 I^2)}(S + I - \frac{S I\alpha_1}{(1+\alpha_1 S+\alpha_2 I^2)})$$

$$-\beta - \frac{2\lambda S I^2 \alpha_2}{(1+\alpha_1 S+\alpha_2 I^2)^2}\Big\} + \delta.$$

By (7.3), this implies that

$$\eta_1(B) \le S'/S - d + \max\left\{\begin{array}{c} -pM - \frac{\lambda I}{1+\alpha_1 S+\alpha_2 I^2}(1 - \frac{S(\alpha_1 - 2\alpha_2 I)}{1+\alpha_1 S+\alpha_2 I^2}) + \beta, \delta(2-p) - \beta - \\ \frac{\lambda}{1+\alpha_1 S+\alpha_2 I^2}(S + I - \frac{S I\alpha_1}{1+\alpha_1 S+\alpha_2 I^2}), -\beta + \delta - \frac{-2\lambda S\alpha_2 I^2}{1+\alpha_1 S+\alpha_2 I^2} \end{array}\right\}$$

$$= S'/S - (d - w).$$

By integrating both sides at the same time, we obtain

$$\frac{1}{t}\int_0^t \eta_1(B)\mathrm{ds} \le \tfrac{1}{t}\mathrm{In}\tfrac{S(t)}{S(0)} - (d-w).$$

Thus

$$\lim_{t\to\infty} \sup\sup \frac{1}{t}\int_0^t \eta_1(B)\mathrm{ds} \le -(d-w)$$

and therefore,

$$\lim_{t\to\infty} \sup\sup \frac{1}{t}\int_0^t \eta_1(B)\mathrm{ds} < 0,$$

provided $d > w$. Hence, E^* is globally asymptotically stable in Ω.

8 Numerical Simulations

In this section, we will give some numerical examples to illustrate our main results by using Milstein's Higher Order Method [6]. All simulations are done using the function ode45, which is *MATLAB's* standard solver for ordinary differential equations (*ODEs*).

As the present study is not a case study, no real data are available. Hence, the choice of parametric values is hypothetical with appropriate units and does not base on data. They are chosen only for illustrative purpose. Because the parametric values are not related to a specific disease, system (2.1) can be considered to be dimensionless. The interval of time is supposed to be [0, 50], while the various set of initial size of population are assumed to be $(S(0), I(0), R(0)) = (50, 40, 30)$. Here we present some numerical examples to discuss the effect of the choice of the parameters, nonlinear incidence rate and media awareness effect on the basic reproduction number R_0. For simulations, we take the set of parameters as shown in ***Table* 8.1** and ***Table* 8.2**.

***Table* 8.1:** Parameters used for simulation purpose when $R_0 = 0.09375 < 1$.

Symbol	a	β	d	δ	λ	M	p	α_1	α_2
Value	15	0.5	0.01	0.9	0.5	0.8	20	0.7	0.1

***Table* 8.2:** Parameters used for simulation purpose when $R_0 = 3.024194 > 1$.

Symbol	a	β	d	δ	λ	M	p	α_1	α_2
Value	1.0	0.1	0.1	0.4	2.0	0.8	0.78	0.1	0.7

For this simulation, we take the set of parameters as shown in ***Table* 8.1**. In this case, $S(t)$ approaches to its steady state value while $I(t)$, $Q(t)$ and $R(t)$ approaches to zero as $t \to \infty$. Hence the disease disappears and dies out. (***Fig.* 8.1**).

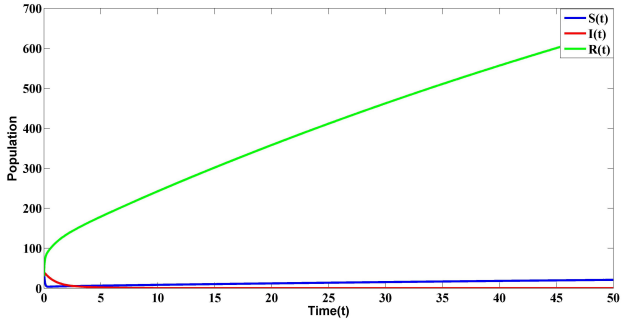

***Figure* 8.1:** The figure represents that the disease dies out.

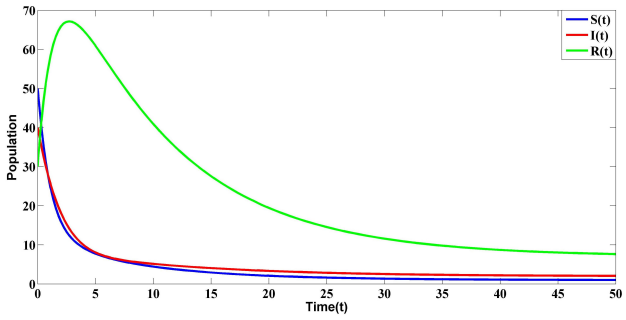

***Figure* 8.2:** The figure represents that the disease endemic.

For these simulations, we take the set of parameters as shown in ***Table* 8.2**. Here, ***Fig.* 8.2** present $S(t)$, $I(t)$ and $R(t)$ all approaches to their steady state values as $t \to \infty$. Hence the disease becomes endemic.

Fig.* 8.3** we present the variation of susceptible class when media effect is applied. The main importance of applying media effect can be observed in ***Fig.* 8.4**, where we draw the variations of infected individuals. It is observed that when media effect is applied optimally, the infected class population remains the least.Fig.* 8.5** represents the variation of recovered class of population. Thus the ***Figs.* 8.3-8.5** represent the behavioral change of all classes of population as time evolves. ***Fig.* 8.6**, represents the phase portrait in $SIRS$-space with different initial conditions. This phase diagram shows that $\lim_{t\to\infty}(S(t), I(t), R(t)) = (S^*, I^*, R^*)$ for $R_0 > 1$.

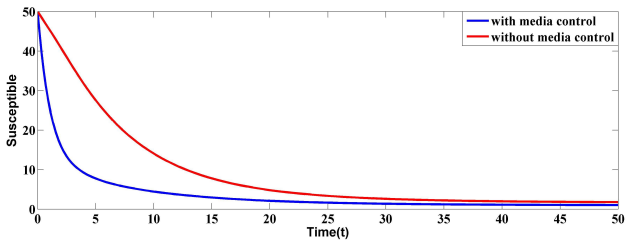

***Figure* 8.3:** Variation of the susceptible population for media control.

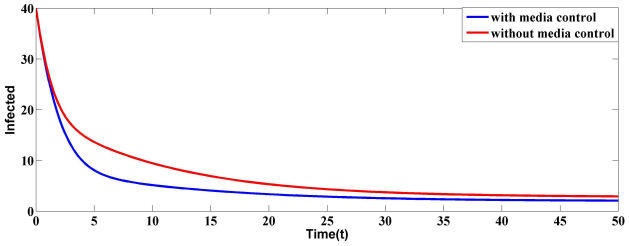

***Figure* 8.4:** Variation of the infected population for media control.

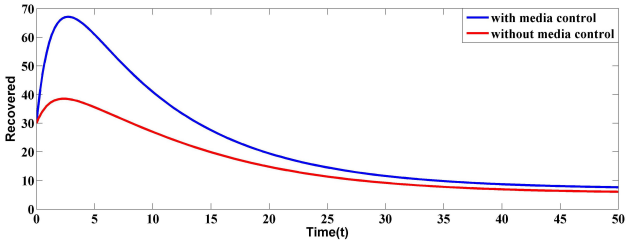

***Figure* 8.5:** Variation of the recovered population for media control.

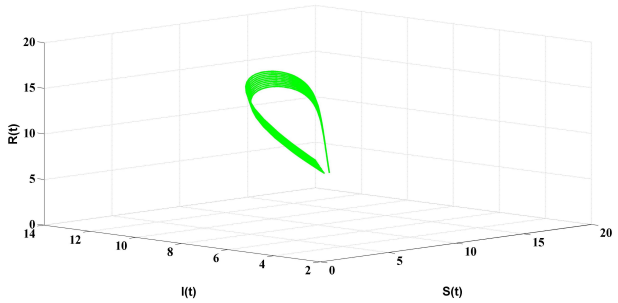

***Figure* 8.6:** The phase diagram at different initial values endemic equilibrium.

9 Discussions and Conclusions

This paper presented a mathematical study of $SIRS$ epidemiological model with a non-monotonic incidence rate and effect of awareness program through media coverage is considered as measure of disease control. The mathematical analysis shows that the basic reproduction number R_0 plays an important role to control the disease, we see that the basic reproduction number R_0 of our model contains the term $a\alpha_1$ in the denominator. Hence the saturation factor of epidemic control (α_1) can contribute to reducing R_0, whereas the inhibition factor with respect to infective (α_2) does not influence that value. We also show that the disease-free equilibrium E_0 is locally and globally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$ under certain conditions. Similarly, for the endemic equilibrium E^*, it has been obtained under certain conditions for locally as well as globally asymptotically stable. The phase diagram is demonstrated in ***Fig.* 8.6**, at different initial values to validate the global stability. The model analysis further shows

that awareness programs through the media campaigning are helpful in decreasing the spread of infectious diseases.

This kind of models can be particularly enlightening for the planning of public health policies for the control of diseases such as influenza, malaria, salmonella, cholera, whooping cough, and measles, COVID-19 since we can discover the many different behaviours the model can have as the parameters are varied.

Acknowledgement. We are very much thankful to the Editor and Reviewer for their valuable suggestions to bring the paper in the present form.

References

[1] R. M. Anderson and R. M. May, Regulation and Stability of host-parasite Population Interactions: I. Regulatory processes, *The Journal of Animal Ecology*, **47(1)** (1978), 219-247.

[2] A. Agrawal, A. Tenguria and G. Modi, Stability Analysis of an SIR Epidemic Model with Specific Non-liner Incidence Rate, *International Journal of Mathematical Theory and Modeling*, **6(1)** (2016), 45-51.

[3] V. Capasso and G. Serio, A Generalization of the KermackMckendrick Deterministic Epidemic Model, *Mathematical Biosciences*, **42(1)** (1978), 43-61.

[4] W. R. Derrick and P. Van Den Driessche, A Disease Transmission Model in a Non-constant Population, *Journal of Mathematical Biology*, **31(5)** (1993), 495-512.

[5] J. Hofbaue and J. W. H. So, Uniform Persistence and Repellors for Maps, *Proceedings of the American Mathematical Society*, **107** (1989), 1137-1142.

[6] D. J. Higham, An Algorithmic Introduction to Numerical Simulation of Stochastic Differential Equations, *Society for Industrial and Applied Mathematics*, **43** (2001), 525-546.

[7] W. O. Kermack and A. G. McKendrick, A Contribution to the Mathematical Theory of Epidemics, *Proceedings of the Royal Society London*, **115** (1927), 700-721.

[8] M. A. Khan, S. Islam and G. Zaman, Media Coverage Campaign in Hepatitis-b Transmission Model, *Applied Mathematics and Computation*, **331** (2018), 378-393.

[9] T. K. Kar, Swapan Kumar Nandi, Soovoojeet Janac and Manotosh Mandal, Stability and Bifurcation Analysis of an Epidemic Model with the Effect of Media, *Nonlinear Science and Non Equilibrium and Complex Phenomena*, **120** (2019), 188-199.

[10] W. M. Liu, S. A. Levin and Y. Iwasa, Influence of Nonlinear Incidence Rates upon the Behavior of Simple SIRS Epidemiological Models, *Journal of Mathematical Biology*, **23(2)** (1986), 187-204.

[11] W. M. Liu, H. W. Hethcote and S. A. Levin, Dynamical Behavior of Epidemiological Models with Nonlinear Contact Rate, *Journal of Mathematical Biology*, **25(4)** (1987), 359-380.

[12] Y. Liu and J. Cui, The Impact of Media Coverage on the Dynamics of Infectious Diseases, *International Journal of Biomathematics*, **1(1)** (2008), 65-74.

[13] J. P. Lasalle, The Stability of Dynamical System, *SIAM, Philadelphia, P A* , (1976).

[14] M. Y. Li and J. S. Muldowney, A Geometric Approach to Global Stability Problem, *SIAM Journal on Mathematical Analysis*, **27(4)** (1996), 1070-1083.

[15] M. Y. Li and L. Wang, A Criterion for Stability of Matrices, *Journal of Mathematical Analysis and Applications*, **225(1)** (1998), 249-264.

[16] A. K. Misra, A. Sharma and J. B. Shukla, Stability Analysis and Optimal Control of an Epidemic Model with Awareness Programs by Media, *Bio Systems*, **138** (2015), 53-62.

[17] S. Ruan and W. D. Wang, Dynamical Behavior of an Epidemic Model with a Nonlinear Incidence Rate, *Journal of Differential Equations*, **188** (2003), 135-163.

[18] S. Samanta, S. Rana, A. Sharma, A. K. Misra and J. Chattopadhyay, Effect of Awareness Programs by Media on the Epidemic Autbreaks: A Mathematical Model, *Applied Mathematics and Computation*, **219(12)** (2013), 6965-6977.

[19] P. Van den Driessche and J. Watmough, Reproduction Numbers and Sub-threshold Endemic Equilibria for Compartmental Models of Disease Transmission, *Mathematical Biosciences*, **180** (2002), 29-48.

[20] Xiao. D and Zhou. Y, Qualitative Analysis of an Epidemic Model, *Canadian Applied Mathematics Quarterly*, **14** (2006), 469-492.

[21] Xiao. D and Ruan. S, Global Analysis of an Epidemic Model with Non-monotone Incidence Rate, *Mathematical Biosciences*, **208** (2007), 419-429.