

MATHEMATICAL ANALYSIS TO STUDY THE ROLE OF MASS-MEDIA IN THE TRANSMISSION DYNAMICS OF INFECTIOUS DISEASES

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ABSTRACT. Type the abstract here The aim of this paper is to investigate and analyze the transmission dynamics of infectious diseases in human population. We have formulated a non-linear SIRS model to study the role of awareness programs by mass-media in reducing the transmission of infectious diseases. The analysis shows that the awareness by mass-media plays a constructive role in the dissemination of knowledge/information and has positive impact in the reduction of disease transmission. Numerical simulations are performed to support and verify the analytical results.

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1. Introduction

The primary reason for studying infectious diseases is to analyze the control measures which would be helpful in control and eradication of the infection from the population. Mathematical modeling is the powerful tool in this approach that allow us to optimize the use of limited resources or specifically to target control measures more efficiently. Media have a key role to play in informing the public about infectious diseases such as TB, influenza and bringing key issues to the attention of policymakers. It is would be beneficial to increase the use of new media tools by government, minority, and other community partners to extend the reach of various awareness programs to communities at greatest risk. It also have an important role in informing people in the developed and developing world alike about the nature and extent of this disease, the shortcomings in current treatments and future possibilities for improved control.

Sir Ronald Ross, Kermack and McKendrick are considered as the pioneers, who made use of compartmental mathematical models for the study of epidemics (1-5). Recently some of the authors have analyzed few epidemic models (6-11) incorporating the effect

of awareness programs by media. Also in some epidemic compartmental models authors have assumed that the awareness through mass-media will significantly reduce the contact rate of susceptibles with infectives (10-11). The effect of public health educational campaigns on HIV/AIDS transmission dynamics has been analyzed in [15], the study effectively shows that the public health educational campaigns of HIV/AIDS are useful and can slow down the epidemic. They have concluded that campaigns affect peoples behavior and can divide the susceptibles class into subclasses with different infectivity rates. In [16] the authors have shown that the education efforts focused on several key risky behaviours pays a great impact to prevent and control the spread of an epidemic HIV.

From the analysis it has been observed that an awareness program related to diseases effectively help in the reduction of disease transmission as it will support one of the following behavioral change:

(i) When an individual feels the symptoms of an infection/disease then he/she can approach to the doctor on early stage and this can help them in fast recovery from the disease/infection. (ii) Awareness can act as an alert to the susceptible individuals which in result can be useful in reduction of their interaction with infectives. (iii) Moreover, because of media coverage some fraction of infectives can be isolated/hospitalized and remain under treatment and hence cannot take part in transmission of the disease.

In epidemic models, the transmission of the disease in the population is modelled by incidence terms. Among many possible forms of incidence terms in epidemic models, simple mass action and standard incidence terms are most commonly used. We formulate our mathematical model with standard incidence for infectious diseases keeping the above mentioned facts in focus. Our model follows a previous work by Misra et al. [12], where they have assumed that the awareness programs based on media are influenced by the outbreak of diseases and it depends upon the number of infectives. In [12], authors have incorporated one separate compartment for cumulative density of awareness programs that we have also considered in the present proposed model. However, we assume a constant input of media related awareness and educational programs and that increases with the increase in number of infectives which depends on the infectives but it can never be equal to zero. The diseases like TB, influenza and dengue etc. are endemic in many parts of the world and some sort of awareness programs are always being communicated on TV networks, newspapers, web-sites etc. on time to time. Hence, the assumption that we have considered is more realistic and close to real world conditions. Also in their formulation of the model, it is assumed that some of the aware susceptibles are going back to susceptible class as they interact with the infectives because they may not care for getting affected by the

disease and even are not afraid of the disease. This particular nature of some of the human beings is erratic and is independent of time, they might not listen to any of the mass-media awareness programs and will interact with infectives. Hence, sending them back to unaware susceptible class is not reasonable as once someone is aware he/she will remain aware and it is not that after some duration his/her awareness will vanish. In the present model, this group of individuals are kept in aware class only and it is assume that they will interact with infective individuals that means here we have considered that some fraction of total aware susceptible population is interacting with infectives.

2. The SIRS Model with Standard Incidence

In this paper, an SIRS with standard incidence is formulated and analyzed. The whole population under consideration is divided into four disjoint classes, namely susceptible class ($S(t)$), infective class ($I(t)$), recovered class ($R(t)$) and aware susceptible class ($S_m(t)$). $N(t)$ is the total population size. Let $M(t)$ be the cumulative density of the awareness programs driven by media in the region under consideration. It is assumed that susceptible individuals who come across with media campaign move to aware susceptible class and in general avoid contact with infectives. So only a small fraction (say α_m) of aware susceptible class interacts with infectives. Also due to the media awareness programs some of the infectives are identified in their early stage and they recover fast, so in addition to normal recovery rate we have added one more recovery rate constant γ_m which is driven by media awareness programs. As media also forces isolation/hospitalization of infectives, so let δ_m fraction of infectives are isolated and only $(1 - \delta_m)$ fraction of infectives are interacting with susceptibles. So based upon these facts we have formulated following model: So based upon these facts we have formulated following model:

$$\begin{aligned}
 \frac{dS}{dt} &= A - \frac{\beta S(1 - \delta_m)I}{N} - \lambda SM - dS + \nu R \\
 \frac{dI}{dt} &= \frac{\beta S(1 - \delta_m)I}{N} + \frac{\beta \alpha_m S_m(1 - \delta_m)I}{N} - (\gamma + \gamma_m + \alpha + d)I \\
 \frac{dR}{dt} &= (\gamma + \gamma_m)I - (\nu + d)R \\
 \frac{dS_m}{dt} &= \lambda SM - \frac{\beta \alpha_m S_m(1 - \delta_m)I}{N} - dS_m \\
 \frac{dM}{dt} &= \mu + \mu_1 I - \mu_0 M
 \end{aligned} \tag{2.1}$$

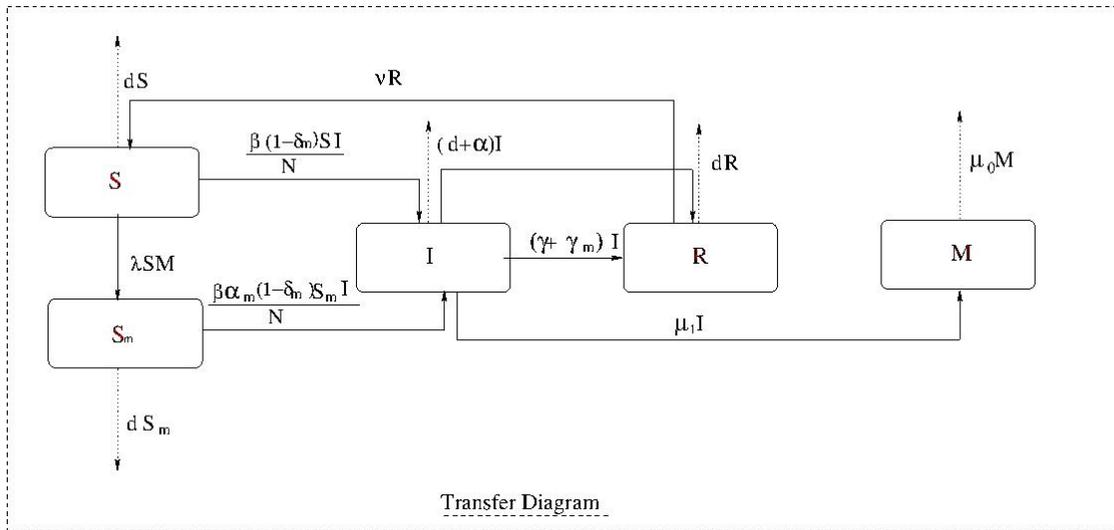


FIGURE 1. Transfer Diagram of the Model (2)

$$N = S + I + R + S_m, \text{ and}$$

$$\dot{N} = A - dN - \alpha I$$

Here, A is the recruitment rate constant; β is the transmission rate constant; λ is the dissemination rate of awareness among susceptibles due to media awareness programs; d is the natural death rate constant; ν is the rate at which individual from recovered class move to susceptible class again after loosing immunity; γ is the natural recovery rate constant; α is the disease related death rate constant; μ is the rate constant corresponding to regular media coverage, μ_1 is the rate constant influenced by number of infectives and μ_0 is the natural decay rate constant of media coverage/awareness programs. In Figure. 1, we describe the flow diagram of the disease dynamics considered in this paper.

The Basic reproduction number for the model is computed as

$$R_0 = \frac{\beta(1 - \delta_m)(\lambda\mu\alpha_m + d\mu_0)}{(\lambda\mu + d\mu_0)(\gamma + \gamma_m + \alpha + d)}$$

This gives the number of secondary infectious cases caused by an infectious individual in a completely susceptible population during his infectious period. For simplification we are re-writing the model using $N = S + I + R + S_m$

$$\begin{aligned} \frac{dN}{dt} &= A - dN - \alpha I \\ \frac{dI}{dt} &= \frac{\beta(N - I - R - S_m)(1 - \delta_m)I}{N} + \frac{\beta\alpha_m S_m(1 - \delta_m)I}{N} - (\gamma + \gamma_m + \alpha + d)I \\ \frac{dR}{dt} &= (\gamma + \gamma_m)I - (\nu + d)R \end{aligned} \tag{2.2}$$

$$\begin{aligned}\frac{dS_m}{dt} &= \lambda(N - I - R - S_m)M - \frac{\beta\alpha_m S_m(1 - \delta_m)I}{N} - dS_m \\ \frac{dM}{dt} &= \mu + \mu_1 I - \mu_0 M\end{aligned}$$

3. Equilibrium Analysis

The system (2.1) has two equilibria, namely the disease-free equilibrium point $E_0(S_0, 0, 0, S_{m_0}, M_0)$ and the endemic equilibrium point $E_1(S^*, I^*, R^*, S_m^*, M^*)$.

For the disease-free equilibrium point, S_0, S_{m_0} and M_0 are given by as follows:
 $N_0 = \frac{A}{d}$, $S_{m_0} = \frac{A\lambda\mu}{d(\lambda\mu + d\mu_0)}$, $M_0 = \frac{\mu}{\mu_0}$,
 Whereas, $I_0 = 0$ and $R_0 = 0$ for infection free equilibrium point.

The endemic equilibrium point $E_1(N^*, I^*, R^*, S_m^*, M^*)$ is obtained by putting the right hand sides of the system of equations (2.2) to zero i.e.

$$\begin{aligned}A - dN - \alpha I &= 0 \\ \frac{\beta(N - I - R - S_m)(1 - \delta_m)I}{N} + \frac{\beta\alpha_m S_m(1 - \delta_m)I}{N} - (\gamma + \gamma_m + \alpha + d)I &= 0 \\ (\gamma + \gamma_m)I - (\nu + d)R &= 0 \\ \lambda(N - I - R - S_m)M - \frac{\beta\alpha_m S_m(1 - \delta_m)I}{N} - dS_m &= 0 \\ \mu + \mu_1 I - \mu_0 M &= 0\end{aligned}$$

The endemic equilibrium points N^*, I^*, R^*, S_m^* and M^* are obtained by solving the following algebraic equations, Keeping $Y \neq 0$, we get following

$$N^* = \frac{A - \alpha I}{d}, \quad M^* = \frac{\mu + \mu_1 I}{\mu_0}, \quad R^* = \frac{(\gamma + \gamma_m)I}{\nu + d},$$

Now using equations for \dot{I} and \dot{S}_m in the model system (2.2) we have obtained two equations stating relation between I and S_m from which we have calculated S_m as,

$$S_m = \frac{-(d + \alpha + \gamma + \gamma_m)(\nu + d)d + \beta(1 - \delta_m)[(A - \alpha I) - d((\gamma + \gamma_m) - d(\nu + d))I]}{\beta(1 - \delta_m)(\nu + d)(1 - \alpha_m)d}, \quad (3.1)$$

and

$$S_m = \frac{\lambda[(A - \alpha I)(\nu + d) - (\gamma + \gamma_m)dI - d(\nu + d)I](\mu + \mu_1 I)(A - \alpha I)}{[\lambda(\mu + \mu_1 I)(A - \alpha I) + \beta\alpha_m(1 - \delta_m)\mu_0 dI + d(A - \alpha I)\mu_0](\nu + d)d} \quad (3.2)$$

Comparing eqn (3.1) and (3.2) we get a cubic $D_1I^3 + D_2I^2 + D_3I + D_4 = 0$, where

$$\begin{aligned}
 D_1 &= -\{\alpha\lambda\mu_1\{(d+\nu)(d+\alpha+\gamma+\gamma_m) - \beta(1-\delta_m)[d(d+\nu) + \alpha(d+\nu) + d(\gamma+\gamma_m)]\}\} \\
 &= -\alpha\lambda\mu_1\{[(d+\alpha+\gamma+\gamma_m) - \beta(1-\delta_m)(d+\alpha)] - \beta(1-\delta_m)d(\gamma+\gamma_m)\} \\
 D_2 &= -\alpha^2(d+\nu)(d+\alpha+\gamma+\gamma_m)(\lambda\mu+d\mu_0) \\
 &\quad -A\alpha\beta\lambda(d+\nu)(1-\delta_m)\alpha_m\mu_1 \\
 &\quad -[d(d+\nu) + \alpha(d+\nu) + d(\gamma+\gamma_m)]d\mu_0\alpha_m\beta^2(1-\delta_m)^2 \\
 &\quad -A[d(d+\nu) + \alpha(d+\nu) + d(\gamma+\gamma_m)]\lambda\alpha_m\mu_1\beta(1-\delta_m) \\
 &\quad +\alpha\beta(d+\nu)(d+\alpha+\gamma+\gamma_m)(1-\delta_m)\alpha_md\mu_0 \\
 &\quad +2A\alpha\lambda(d+\alpha+\gamma+\gamma_m)\mu_1 \\
 &\quad +[d(d+\nu) + \alpha(d+\nu) + d(\gamma+\gamma_m)](\lambda\mu\alpha_m+d\mu_0)\alpha\beta(1-\delta_m) \\
 D_3 &= 2A\alpha(d+\nu)(d+\alpha+\gamma+\gamma_m)(\lambda\mu+d\mu_0) \\
 &\quad +A^2d\beta^2(d+\nu)(1-\delta_m)^2\alpha_m\mu_0 \\
 &\quad +A^2\beta\lambda(d+\nu)(1-\delta_m)\alpha_m\mu_1 \\
 &\quad -A\alpha\beta\lambda\mu\alpha_m(d+\nu)(1-\delta_m) \\
 &\quad -A\beta\lambda\mu\alpha_m[d(d+\nu) + \alpha(d+\nu) + d(\gamma+\gamma_m)](1-\delta_m) \\
 &\quad -Ad\alpha\beta(d+\nu)(1-\delta_m)\mu_0 \\
 &\quad -Ad\beta(d+\nu)\alpha_m(d+\alpha+\gamma+\gamma_m)(1-\delta_m)\mu_0 \\
 &\quad -Ad\beta[d(d+\nu) + \alpha(d+\nu) + d(\gamma+\gamma_m)](1-\delta_m)\mu_0 \\
 &\quad -A^2\lambda(d+\nu)(d+\alpha+\gamma+\gamma_m)\mu_1 \\
 D_4 &= A^2(d+\nu)(d+\alpha+\gamma+\gamma_m)(d\mu_0+\lambda\mu)(R_0-1).
 \end{aligned}$$

again re-writing the coefficients of above cubic, we get $D_1 > 0$ and $D_4 < 0$

Using Descartes's rule of sign, following cases have been arose from above result,

Case(i) If $D_2 > 0$ and $D_3 > 0$; or $D_2 > 0$ and $D_3 < 0$; or $D_2 < 0$ and $D_3 < 0$

ensures the existence of atmost one positive real root.

Case(ii) If $D_2 < 0$ and $D_3 > 0$, then the system could have more than one endemic equilibrium for $R_0 > 1$.

Case(iii) If $D_2 < 0$ and $D_3 > 0$, then the system could have more than one endemic equilibrium for $R_0 < 1$.

The existence of more than one (multiple) endemic equilibria when $R_0 < 1$ suggests the possibility of backward bifurcation, where the stable disease-free equilibrium co-exists with a stable endemic equilibrium, when the $R_0 < 1$. real roots.

4. Stability Analysis

The local asymptotic stability of the disease-free equilibrium point E_0 is established using variational matrix method and stated in the following theorem.

Theorem 4.1 *If $R_0 < 1$, the disease-free equilibrium E_0 is locally asymptotically stable and is unstable for $R_0 > 1$.*

Proof: To study the stability of disease-free equilibrium the variational matrix M_1 of the system corresponding to disease-free equilibrium E_0 is obtained as

$$M_1 = \begin{pmatrix} -d & -\alpha & 0 & 0 & 0 \\ 0 & -(d + \alpha + \gamma + \gamma_m)(1 - R_0) & 0 & 0 & 0 \\ 0 & \gamma + \gamma_m & -(\nu + d) & 0 & 0 \\ \lambda M_0 & -\lambda M_0 - \frac{\beta \alpha_m (1 - \lambda_m) S_{m_0}}{N} & -\lambda M_0 & -(\lambda M_0 + d) & \lambda(N_0 - S_{m_0}) \\ 0 & \mu_1 & 0 & 0 & -\mu_0 \end{pmatrix}$$

The eigenvalues of this variational matrix are $-d, -(d + \alpha + \gamma + \gamma_m)(1 - R_0), -(\nu + d), -(\lambda M_0 + d)$ and $-\mu_0$. Clearly here one of the eigenvalue is positive for $R_0 > 1$ which implies instability of disease-free equilibrium E_0 . So the equilibrium is locally asymptotically stable provided $R_0 < 1$.

The local asymptotic stability of endemic equilibrium point E_1 can be established using variational matrix method and stated in the following theorem.

Theorem 4.2 *The endemic equilibrium point $E_1(N^*, I^*, R^*, S_m^*, M^*)$ is locally asymptotically stable provided*

$$\begin{pmatrix} a_4 & a_2 \\ 1 & a_3 \end{pmatrix} > 0, \begin{pmatrix} a_4 & a_2 & a_0 \\ 1 & a_3 & a_1 \\ 0 & a_4 & a_2 \end{pmatrix} > 0, \begin{pmatrix} a_4 & a_2 & a_0 & 0 \\ 1 & a_3 & a_1 & 0 \\ 0 & a_4 & a_2 & a_0 \\ 0 & 1 & a_3 & a_1 \end{pmatrix} > 0,$$

where $a_0, a_1, a_2, a_3,$ and a_4 are given in the proof of this theorem.

Proof: See Appendix A.

Theorem 4.3 *If $R_0 < 1$, the disease-free equilibrium E_0 is globally asymptotically stable and unstable if $R_0 > 1$.*

Proof: This theorem is proved using comparison theorem. The rate of change of the variable representing the infected component of the system (2.1) can be rewritten as

$$\frac{dI}{dt} = \left\{ \frac{\beta(1 - \delta_m)(S_0 + \alpha_m S_{m_0})}{N_0} - (\gamma + \gamma_m + \alpha + d) \right\} I - \beta(1 - \delta_m) \left\{ \left(\frac{S_0}{N_0} - \frac{S}{N} \right) + \alpha_m \left(\frac{S_{m_0}}{N_0} - \frac{S_m}{N} \right) \right\} I,$$

where N_0, S_0 and S_{m_0} are same as in disease-free equilibrium E_0 and $N = S + I + R + S_m$. Since $N < N_0, S \leq S_0$ and $S_m \leq S_{m_0}$, gives $\beta(1 - \delta_m) \left\{ \left(\frac{S_0}{N_0} - \frac{S}{N} \right) + \alpha_m \left(\frac{S_{m_0}}{N_0} - \frac{S_m}{N} \right) \right\} I$

is positive.

$$\begin{aligned} \frac{dI}{dt} &\leq \left\{ \frac{\beta(1 - \delta_m)(S_0 + \alpha_m S_{m_0})}{N_0} - (\gamma + \gamma_m + \alpha + d) \right\} I \\ \frac{dI}{dt} &\leq \left\{ \frac{\beta(1 - \delta_m)(d\mu_0 + \alpha_m \lambda \mu)}{(\gamma + \gamma_m + \alpha + d)} - 1 \right\} I, \\ \frac{dI}{dt} &= \{R_0 - 1\} I, \\ \frac{dI}{dt} &= -\{1 - R_0\} I, \end{aligned}$$

As for $R_0 < 1$ the bracketed term $\left\{ \frac{\beta(1 - \delta_m)(S_0 + \alpha_m S_{m_0})}{N_0} - (\gamma + \gamma_m + \alpha + d) \right\} I$ of the inequality (4.1) is negative, thus it follows that $I \rightarrow 0$ as $t \rightarrow \infty$ by the comparison theorem in [13]. Also from the system (2.1) it is found that $N \rightarrow N_0, S \rightarrow S_0, R \rightarrow 0, S_m \rightarrow S_{m_0}$ and $M \rightarrow M_0$ whenever $I = 0$. Thus for $R_0 < 1$, the disease-free equilibrium point $E_0(N_0, 0, 0, S_{m_0}, M_0)$ is globally asymptotically stable.

5. Bifurcation Analysis

Consider the transformed system

$$\begin{aligned} \dot{N} &= f_1 = A - dN - \alpha I \\ \dot{I} &= f_2 = \frac{\beta(N - I - R - S_m)(1 - \delta_m)I}{N} + \frac{\beta\alpha_m S_m(1 - \delta_m)I}{N} - (\gamma + \gamma_m + \alpha + d)I \\ \dot{R} &= f_3 = (\gamma + \gamma_m)I - (\nu + d)R \\ \dot{S}_m &= f_4 = \lambda(N - I - R - S_m)M - \frac{\beta\alpha_m S_m(1 - \delta_m)I}{N} - dS_m \\ \dot{M} &= f_5 = \mu + \mu_1 I - \mu_0 M \end{aligned} \tag{5.1}$$

The Jacobian of the system (2.1), (E_0) is given by

$$J(E_0) = \begin{pmatrix} -d & -\alpha & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma + \gamma_m & -(\nu + d) & 0 & 0 \\ \lambda M_0 & -\lambda M_0 - \frac{\beta\alpha_m(1 - \delta_m)S_{m_0}}{N} & -\lambda M_0 & -(\lambda M_0 + d) & \lambda(N_0 - S_{m_0}) \\ 0 & \mu_1 & 0 & 0 & -\mu_0 \end{pmatrix}$$

The associated reproduction number is given by

$$R_0 = \frac{\beta(1 - \delta_m)(\lambda\mu\alpha_m + d\mu_0)}{(\lambda\mu + d\mu_0)(\gamma + \gamma_m + \alpha + d)}$$

Consider the case when $R_0 = 1$. Suppose, β be chosen as the bifurcation parameter. Solving (2.1) for β gives $R_0 = 1$ when $\beta = \frac{(\lambda\mu + d\mu_0)(\gamma + \gamma_m + \alpha + d)}{(1 - \delta_m)(\lambda\mu\alpha_m + d\mu_0)}$

Note that the above linearized system, of transformed system (5.1) with $\beta = \beta^*$, has a zero eigenvalue which is simple and the other eigenvalues are real and negative. Hence, The center manifold theory [J Carr.] can be used to analyze the dynamic of (2.1) near $\beta = \beta^*$.

Eigenvectors of $J(E_0)|_{\beta=\beta^*}$

It can be shown that the Jacobian of (2.1) at $\beta = \beta^*$ (denoted by J_{β^*}) has a right eigenvector (associated with the zero eigenvalue) given by $w = [w_1, w_2, w_3, w_4, w_5]^T$, where

$$w_2 > 0, w_1 = -\frac{\alpha}{d}w_2, w_3 = \frac{(\gamma+\gamma_m)}{(\nu+d)}w_2,$$

$$w_5 = \frac{\mu_1}{\mu_0}w_2,$$

$$w_4 = \frac{1}{\lambda M_0 + d} [\lambda(N_0 - S_{m_0}) \frac{m\mu_1}{\mu_0} - \frac{\lambda M_0(\gamma+\gamma_m)}{(\nu+d)} - \frac{(\lambda M_0 + \beta\alpha_m(1-\delta_m)S_{m_0})}{N_0} - \frac{\lambda\alpha M_0}{d}] w_2$$

Further, J_{β^*} has a left eigenvector $v = [v_1, v_2, v_3, v_4, v_5]^T$ (associated with the zero eigenvalue), where

Here $-(\lambda M_0 + d)v_4 = 0$ gives $v_1 = 0 = v_3 = v_4 = v_5$ and $v_2 = v_2 > 0$ (arbitrary). For convenience, the theorem in [Song] is stated here,

Lemma 5.1 (Castillo-Chavez and Song) Consider the following general system of ordinary differential equations with a paramere ϕ

$$\frac{dx}{dt} = f(x, \phi), f : \mathbb{R}^n \times \mathbb{R} \rightarrow \mathbb{R} \text{ and } \mathbb{C}^2(\mathbb{R}^n \times \mathbb{R}),$$

where 0 is an equilibrium point of the system (that is, $f(0, \phi) \equiv 0$ for all ϕ and assume

A1 : $A = D_x f(0, 0) = (\frac{\partial f_i}{\partial x_j}(0, 0))$ is the liberalization matrix of the system (5.1) around the equilibrium 0 and ϕ evaluated at 0. Zero is a simple eigenvalue of A and other eigenvalues of A have negative real parts;

A2 : Matrix A has a right eigenvector w and a left eigenvector v (each corresponding to the zero eigenvalue);

Let f_k be the k^{th} component of f and

$$a = \sum_{k,j,i=1}^n v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0, 0), \quad b = \sum_{k,i=1}^n v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0, 0)$$

The local dynamics of the system around 0 is totally determined by the signs of a and b.

- i.** $a > 0, b > 0$. when $\phi < 0$ with $|\phi| \ll 1$, 0 is locally asymptotically stable and there exists a positive unstable equilibrium; when $0 < \phi \ll 1$, 0 is unstable and there exists a negative, locally asymptotically stable equilibrium;
- ii.** $a < 0, b < 0$. when $\phi < 0$ with $|\phi| \ll 1$, 0 is unstable; when $0 < \phi \ll 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium;
- iii.** $a > 0, b < 0$. when $\phi < 0$ with $|\phi| \ll 1$, 0 is unstable and there exists a locally asymptotically stable negative equilibrium; when $0 < \phi \ll 1$, 0 is stable, and a positive unstable equilibrium appears;

iv. $a < 0, b > 0$. when ϕ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.

Proof: Computations of a and b : For the system (5.1), the associated non-zero partial derivatives of F (at the DFE) are given by

$$\begin{aligned} \frac{\partial^2 f_2}{\partial x_1 \partial x_2} &= \frac{\beta(1-\delta_m)(1-\alpha_m)\lambda\mu d}{A(\lambda\mu+d\mu_0)} = \frac{\partial^2 f_2}{\partial x_2 \partial x_1}, \quad \frac{\partial^2 f_2}{\partial^2 x_2} = \frac{-2\beta(1-\delta_m)d}{A}, \\ \frac{\partial^2 f_2}{\partial x_2 \partial x_3} &= \frac{-\beta(1-\delta_m)d}{A} = \frac{\partial^2 f_2}{\partial x_3 \partial x_2} \\ \frac{\partial^2 f_2}{\partial x_2 \partial x_4} &= \frac{-d\beta(1-\delta_m)(1-\alpha_m)}{A} = \frac{\partial^2 f_2}{\partial x_4 \partial x_2}, \quad \frac{\partial^2 f_4}{\partial x_1 \partial x_2} = \frac{\beta(1-\delta_m)\alpha_m\lambda\mu d}{A(\lambda\mu+d\mu_0)} = \frac{\partial^2 f_4}{\partial x_2 \partial x_1}, \\ \frac{\partial^2 f_4}{\partial x_1 \partial x_5} &= \lambda = \frac{\partial^2 f_4}{\partial x_5 \partial x_1}, \quad \frac{\partial^2 f_4}{\partial x_2 \partial x_4} = \frac{-\beta\alpha_m(1-\delta_m)d}{A} = \frac{\partial^2 f_4}{\partial x_4 \partial x_2}, \\ \frac{\partial^2 f_4}{\partial x_3 \partial x_5} &= -\lambda = \frac{\partial^2 f_4}{\partial x_5 \partial x_3}, \quad \frac{\partial^2 f_4}{\partial x_4 \partial x_5} = -\lambda = \frac{\partial^2 f_4}{\partial x_5 \partial x_4}, \\ \frac{\partial^2 f_4}{\partial x_5 \partial x_2} &= -\lambda = \frac{\partial^2 f_4}{\partial x_2 \partial x_5}, \quad \frac{\partial^2 f_4}{\partial x_2 \partial \beta} = \frac{-\alpha_m(1-\delta_m)\lambda\mu}{\lambda\mu+d\mu_0} \\ \frac{\partial^2 f_2}{\partial x_2 \partial \beta} &= \frac{(1-\delta_m)d\mu_0}{(\lambda\mu+d\mu_0)} + \frac{\alpha_m(1-\delta_m)\lambda\mu}{(\lambda\mu+d\mu_0)} = \frac{(1-\delta_m)}{(\lambda\mu+d\mu_0)}(d\mu_0 + \lambda\mu\alpha_m). \end{aligned}$$

Thus,

$$\begin{aligned} a &= -2v_2w_2^2\beta(1-\delta_m)\left[\frac{\alpha(1-\alpha_m)\lambda\mu}{A(\lambda\mu+d\mu_0)} + \frac{2d}{A} + \frac{(\gamma+\gamma_m)d}{(\nu+d)A}\right] \\ &\quad - \frac{2\beta(1-\delta_m)\lambda\mu_0v_2w_2^2}{A(\lambda\mu+d\mu_0)}\left[\frac{A\lambda\mu_1}{(\lambda\mu+d\mu_0)} - \frac{\lambda\mu(\gamma+\gamma_m)}{(\nu+d)\mu_0} - \frac{\lambda\alpha\mu}{d\mu_0} - \frac{\lambda\mu[(\lambda\mu+d\mu_0)+\beta\alpha_m(1-\delta_m)\mu_0]}{\mu_0(\lambda\mu+d\mu_0)}\right] \\ b &= \frac{w_2v_2[d\mu_0 + \alpha_m\lambda\mu](1-\delta_m)}{(\lambda\mu+d\mu_0)} > 0 \end{aligned}$$

Here, $a < 0$ for $\frac{A\lambda\mu_1}{(\lambda\mu+d\mu_0)} > \frac{\lambda}{\mu_0}\left[\frac{\mu(\gamma+\gamma_m)}{(\nu+d)} + \frac{\alpha\mu}{d} + \frac{\mu[(\lambda\mu+d\mu_0)+\beta\alpha_m(1-\delta_m)\mu_0]}{(\lambda\mu+d\mu_0)}\right]$

Thus, if $a < 0$ and $b > 0$. Here we observe that the coefficient b is always positive so that, according to Lemma 5.1, it is the sign of the coefficient a which decides the local dynamics around the disease-free equilibrium for $\beta = \beta^*$. From this analysis we establish following theorem here.

Theorem 5.1 If $a > 0$, then the system (2.1) undergoes a backward bifurcation at $R_0 = 1$, otherwise if $a < 0$ then the endemic equilibrium is locally asymptotically stable for $R_0 > 1$ but close to one.

6. Simulation

The system (2.1) is simulated for various set of parameters using XPP [14]. The stability of disease-free equilibrium point E_0 is shown in Figure 2, where the reproduction number R_0 is equal to 0.409260 which is less than one and parameter values are as follows:

$$A = 100, d = 0.023, \beta = 0.5, \alpha = 0.001, \alpha_m = 0.202, \delta_m = 0.001,$$

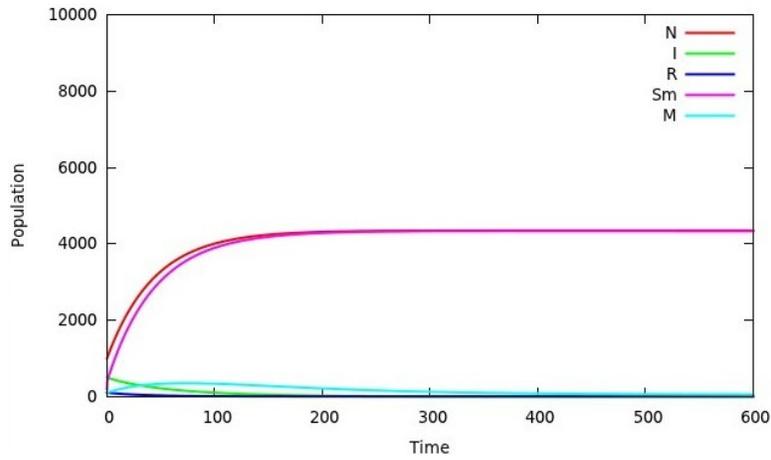


FIGURE 2. Stability of Disease-Free Equilibrium Point (E_0).

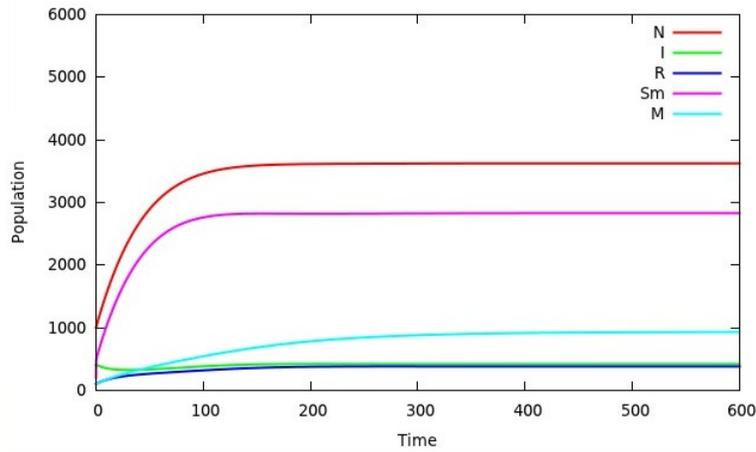


FIGURE 3. Stability of Endemic Equilibrium Point (E_1).

$$\lambda = 0.1, \nu = 0.01, \mu = 0.5, \mu_0 = 0.01, \mu_1 = 0.02, \gamma_m = 0.0001, \gamma = 0.001$$

The endemic equilibrium E_1 is shown in Figure 3, where reproduction number is equal to 1.302437, which is greater than one and the parameter values are:

$$A = 100, d = 0.023, \beta = 1.2, \alpha = 0.04, \alpha_m = 0.102, \delta_m = 0.03,$$

$$\lambda = 0.1, \nu = 0.01, \mu = 1, \mu_0 = 0.01, \mu_1 = 0.02, \gamma_m = 0.01, \gamma = 0.02$$

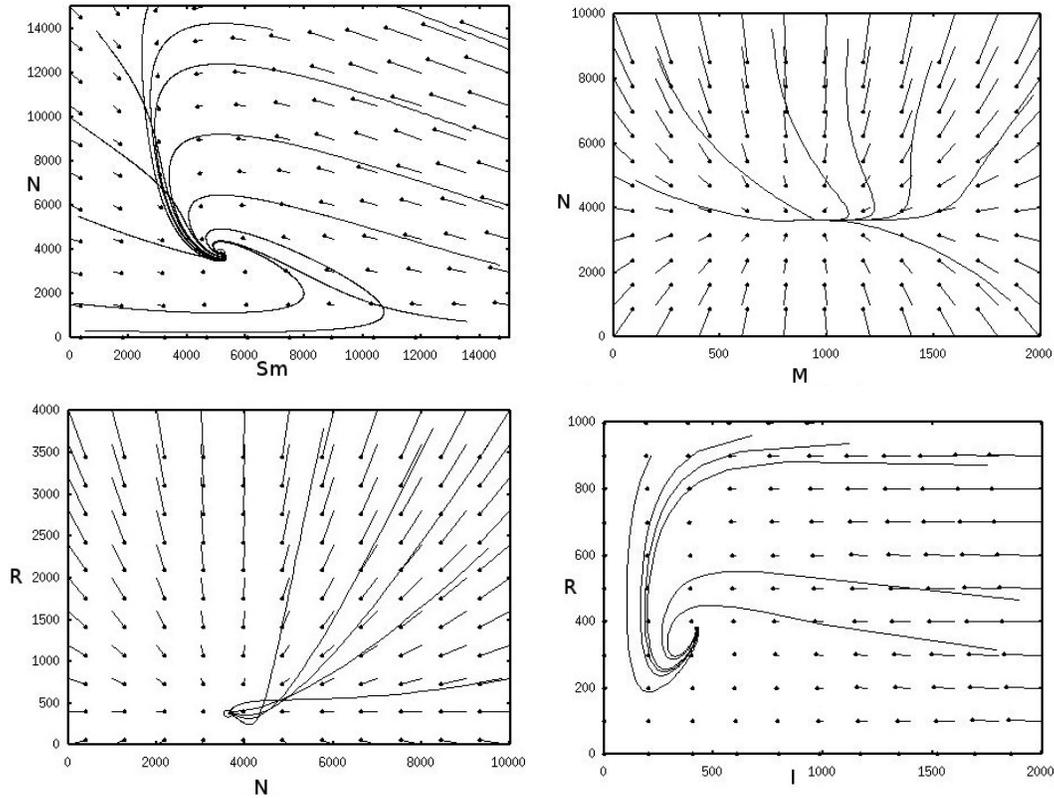


FIGURE 4. Phase portrait corresponding to stability of endemic equilibrium point in $S_m - N$, $M - N$, $N - R$ and $R - I$ plane.

In Figure 4., the phase portraits corresponding to stability of endemic equilibrium point in $S_m - N$, $M - N$, $N - R$ and $R - I$ plane are plotted, respectively for the following set of parameter values:

$$A = 300, d = 0.01666, \beta = 0.000007, \alpha = 0.0002, \alpha_m = 0.002, \delta_m = 0.2,$$

$$\lambda = 0.0002, \nu = 0.03, \mu = 0.001, \mu_0 = 0.03, \mu_1 = 0.001, \gamma_m = 0.01, \gamma = 0.002$$

7. Conclusion

In the present paper, we have formulated a nonlinear SIRS mathematical model to study the role of awareness in the transmission of infectious diseases. The model is analyzed using the stability theory of differential equations and it has been observed that the awareness has significant impact in the reduction of transmission of diseases. Hence, the use of preventative measures, effective medical treatment and awareness through education programmes should be promoted to reduce the spread of the disease. Numerical simulations are performed to support the analytical results.

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Appendix: A

$$M_1 = \begin{pmatrix} m_{11} & m_{12} & 0 & 0 & 0 \\ m_{21} & m_{22} & 0 & m_{24} & 0 \\ 0 & m_{32} & m_{33} & 0 & 0 \\ m_{41} & m_{42} & m_{43} & m_{44} & m_{45} \\ 0 & m_{52} & 0 & 0 & m_{55} \end{pmatrix}$$

where,

$$\begin{aligned} m_{11} &= -d, \quad m_{12} = -\alpha, \quad m_{21} = \beta(1 - \delta_m) \left(\frac{(I^* + R^*)I^* - (1 - \alpha_m)S_m^*I^*}{N^{*2}} \right), \\ m_{22} &= \beta(1 - \delta_m) \left(\frac{(N^* - R^*) - 2I^* - (1 - \alpha_m)S_m^*}{N^{*2}} \right) - (\gamma + \gamma_m + \alpha + \alpha_m), \\ m_{23} &= -\frac{\beta(1 - \delta_m)I^*}{N^*}, \quad m_{24} = -\frac{\beta(1 - \delta_m)(1 - \alpha_m)I^*}{N^*}, \quad m_{32} = (\gamma + \gamma_m), \\ m_{33} &= -(\nu + d), \quad m_{41} = \frac{\beta(1 - \delta_m)\alpha_m I^*}{N^*} + \lambda M^*, \quad m_{42} = -\frac{\beta(1 - \delta_m)\alpha_m I^*}{N^*} - \lambda M^*, \\ m_{43} &= -\lambda M^*, \quad m_{44} = -\frac{\beta(1 - \delta_m)\alpha_m I^*}{N^*} - \lambda M^* - d, \\ m_{45} &= \lambda(N - I - R - S_m), \quad m_{52} = \mu_1, \quad m_{55} = -\mu_0. \end{aligned}$$

The above defined variational matrix gives a fifth degree polynomial given by:

$$\lambda^5 + a_4\lambda^4 + a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0, \text{ here,}$$

$$a_4 = -(m_{11} + m_{22} + m_{33} + m_{44} + m_{55})$$

$$a_3 = -(m_{12}m_{21} - m_{11}m_{22} + m_{23}m_{32} - m_{11}m_{33} - m_{22}m_{33} + m_{24}m_{42} - m_{11}m_{44} - m_{22}m_{44} - m_{33}m_{44} - m_{11}m_{55} - m_{22}m_{55} - m_{33}m_{55} - m_{44}m_{55})$$

$$a_2 = -(-m_{11}m_{23}m_{32} - m_{12}m_{21}m_{33} + m_{11}m_{22}m_{33} + m_{12}m_{24}m_{41} - m_{11}m_{24}m_{42} - m_{24}m_{33}m_{42} + m_{24}m_{32}m_{43} - m_{12}m_{21}m_{44} + m_{11}m_{22}m_{44} - m_{23}m_{32}m_{44} + m_{11}m_{33}m_{44} + m_{22}m_{33}m_{44} + m_{24}m_{45}m_{52} - m_{12}m_{21}m_{55} + m_{11}m_{22}m_{55} - m_{23}m_{32}m_{55} + m_{11}m_{33}m_{55} + m_{22}m_{33}m_{55} - m_{24}m_{42}m_{55} + m_{11}m_{44}m_{55} + m_{22}m_{44}m_{55} + m_{33}m_{44}m_{55})$$

$$a_1 = -(-m_{12}m_{24}m_{33}m_{41} + m_{11}m_{24}m_{33}m_{42} - m_{11}m_{24}m_{32}m_{43} + m_{11}m_{23}m_{32}m_{44} + m_{12}m_{21}m_{33}m_{44} - m_{11}m_{22}m_{33}m_{44} - m_{11}m_{24}m_{45}m_{52} - m_{24}m_{33}m_{45}m_{52} + m_{11}m_{23}m_{32}m_{55} + m_{12}m_{21}m_{33}m_{55} - m_{11}m_{22}m_{33}m_{55} - m_{12}m_{24}m_{41}m_{55} + m_{11}m_{24}m_{42}m_{55} + m_{24}m_{33}m_{42}m_{55} - m_{24}m_{32}m_{43}m_{55} + m_{12}m_{21}m_{44}m_{55} - m_{11}m_{22}m_{44}m_{55} + m_{23}m_{32}m_{44}m_{55} - m_{11}m_{33}m_{44}m_{55} - m_{22}m_{33}m_{44}m_{55})$$

$$a_0 = -[m_{11}m_{24}m_{33}m_{45}m_{52} + m_{12}m_{24}m_{33}m_{41}m_{55} - m_{11}m_{24}m_{33}m_{42}m_{55} + m_{11}m_{24}m_{32}m_{43}m_{55} - (m_{11}m_{23}m_{32} + m_{12}m_{21}m_{33} - m_{11}m_{22}m_{33})m_{44}m_{55}].$$

Using *mathematica* we have analyzed that the coefficient of above fifth degree polynomial satisfies stability conditions of Routh-Hurwitz criteria.