

Modeling and Analysis of an SIRS Epidemic Model with Effect of Awareness Programs by Media

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Abstract—This paper proposes and analyzes an SIRS epidemic model incorporating the effects of the awareness programs driven by the media. Media and media driven awareness programs play a promising role in disseminating the information about outbreak of any disease across the globe. This motivates people to take precautionary measures and guides the infected individuals to get hospitalized. Timely hospitalization helps to reduce diagnostic delays and hence results in fast recovery of infected individuals. The aim of this study is to investigate the impact of the media on the spread and control of infectious diseases. This model is analyzed using stability theory of differential equations. The sensitivity of parameters has been discussed and it has been found that the awareness programs driven by the media have positive impact in reducing the infection prevalence of the infective population in the region under consideration.

Keywords—Infectious diseases, SIRS model, Media, Stability theory, Simulation.

I. INTRODUCTION

IN recent years, the role of media in controlling the transmission of epidemic is well accepted. It has great influence on the individual behaviors as well as on the construction and implementation of public health intervention and control policies [4], [5], [6]. The modern communication tools like internet/internet driven services including media enabled services, networking sites and free access to information via websites have made the information available to the human population almost in real time. These advanced technologies have strengthened the pro-active roles of the media, and now a days media is alert everywhere and has developed the capability to capture, monitor and report even minor incidences of interest from one part of the world to another part almost in contemporary times. Infectious diseases are considered as major barrier to the social and economic development of humankind and further to the society [1], [2]. The main aim of epidemiological modelling is to lower the rate of transmission and mortality caused by the diseases. There should be strong motivation and coordination between policymakers and health-care providers to accomplish the target to prepare society to fight against a pandemic and to reduce the transmission [11]. The target population should be given appropriate information about the risk factors and about the precautionary measures to escape from the disease as

the mode of transmission are different for different infectious disease which highly effect the rate of the transmission [8].

Media is an influential source in the knowledge transfer and dissemination process [9]. It plays an important role in gathering and reproducing information in the beginning of a epidemic and is considered as the most effective epidemic management program which can reduce the social burden of the disease [3]. American 'National Health Council' conducted a study in 1998, where it is was observed that 75% of the people receive health news via the media (40% from the TV, 35% - by magazines or journals, 16% from newspapers, and 2% through the internet) [10]. In fact it is well understood that media highly influence the people behavior towards the disease and helps in controlling the transmission of disease [7].

Although, the role of media in controlling the outbreak of SARS and H1N1 flu is well known, even then not much significant work has been done in the mathematical formulation of models emphasizing this particular parameter. Therefore it is imperative for researchers to study the impact of media on the transmission dynamics of infectious diseases. Recently, some researchers [6, 12-17], have studied the role of awareness through media on the spread of disease transmission. In [6] the authors have analyzed multiple outbreak of infectious diseases due to the psychological impact of the reported numbers of infectious and hospitalized individuals. In [15], impact of public health educational campaigns on the transmission dynamics of HIV/AIDS is studied. In [12], [13], authors have considered the impact of media awareness in the reduction of contact rate constant. Media coverage can help in reducing the burden of an epidemic and can shorten the duration of the disease outbreak [16], [17].

A deeper observation suggests that any awareness program related to disease can lead to the following three major advantageous points:

(i) If someone feels that he/she has the symptoms then he/she can approach to the doctor soon and can recover fast. So in addition to natural disease related recovery, there can be some infectives who will recover fast.

(ii) It can alert susceptible individuals and so some susceptible who comes across with media awareness campaign, will not interact with infectives.

(iii) Also due to media coverage some fraction of infectives can be isolated/hospitalized and remain under treatment so they cannot take part in transmission of disease.

We have formulated the mathematical model keeping the above mentioned points in focus. Our model follows the

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work proposed by Misra et al. [18], but there are significant differences in both the models. They have assumed that the media based awareness programs are influenced by the outbreak of diseases and depend upon the number of infectives. They have incorporated one separate equation for cumulative density of awareness programs. In the proposed model, including these facts we assume that there is also a constant input of media related awareness programs which increases with the increase in number of infectives. Although, technically it depends on infectives but it is never zero. Since there are diseases, TB, influenza, malaria, dengue etc., which are endemic in many part of the world and some sort of awareness programs are always being communicated on TV networks, newspapers etc. regularly; so our assumption is more realistic and close to the 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 do not care for getting infected with diseases and are not afraid of the disease and usually interact with infectives. This particular nature of some of the human beings, in our opinion, is erratic and not time dependent. Moreover, if someone is erratic then he/she will not listen to any of the media awareness programs and will interact with infectives in anyway. 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. Therefore, in our model we have kept such kind of individual in aware class only but we assume that they interact with infectives. This leads to the assumption that some fraction of total aware susceptible population is interacting with infectives.

II. THE MODEL

As we are considering an SIRS model, 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 ($S_m(t)$). 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:

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

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. The flow diagram describing population movements between the compartments is shown in Fig. 1.

All the solutions of (1) which initiate in Ω remain in the region Ω . This result can be summarized in the following theorem:

Theorem 1 For all time $t \geq 0$, all the solutions of the system (1) are eventually confined in the compact subset $\Omega = \{(S_0, I_0, R_0, S_{m0}) \in R_+^5 : S_0 > 0, I_0 \geq 0, R_0 \geq 0, S_{m0} \geq 0; N \leq \frac{A}{\mu}\}$

Proof Let $(S(t), I(t), R(t), S_m(t))$ be any solution with positive initial conditions (S_0, I_0, R_0, S_{m0}) with $N(S + I + R + S_m) = S(t) + I(t) + R(t) + S_m(t)$.

Notice that sum of first four compartments S, I, R and S_m in (1) is equal to total population size N , hence adding these equations we obtain the time derivative along the solution of (1) given as

$$\frac{dN}{dt} = A - \mu N - \alpha I \leq A - \mu N, \text{ i.e. } \frac{dN}{dt} + \mu N \leq A.$$

This follows that

$$0 \leq N(t) \leq \frac{A}{\mu}(1 - e^{-\mu t}) + N_0 e^{-\mu t}, \text{ and for } t \rightarrow \infty, \text{ we have}$$

$$\lim_{t \rightarrow \infty} \sup N \leq \frac{A}{\mu}$$

Thus, Ω is positively-invariant and all solutions are bounded in the interval $[0, \infty)$.

A. Basic Reproduction Number

The system (1) has a disease-free equilibrium (DFE) given by,

$$E_0 = \left(\frac{A\mu_0}{(\lambda\mu + d\mu_0)}, 0, 0, \frac{A\lambda\mu}{d(\lambda\mu + d\mu_0)}, \frac{\mu}{\mu_0} \right).$$

The basic reproduction number R_0 for this model is computed using the technique stated in [19]. Let $x = (I, R)^T$. Then for our model we have $\frac{dx_i}{dt} = \mathcal{F}_i(x) - [\mathcal{V}_i^-(x) - \mathcal{V}_i^+(x)] = \mathcal{F}_i(x) - \mathcal{V}_i(x)$, Here $\mathcal{F}_i(x)$ represents the rate of appearance of new infections

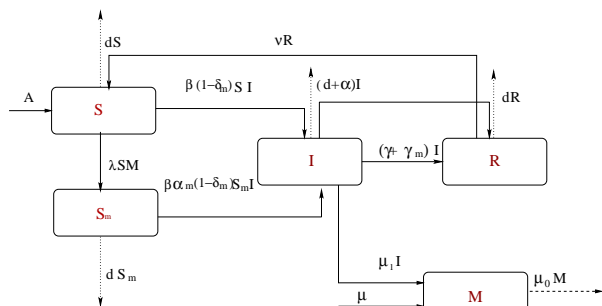


Fig. 1 Transfer diagram of the model system (1)

in compartment i , $\mathcal{V}_i^+(x)$ represents the rate of transfer of individuals into compartment i by all other means, and $\mathcal{V}_i^-(x)$ represents the rate of transfer of individuals out of compartment i .

Hence we can rewrite it as follows:

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

Using the the same notation as given in [19], the matrices \mathcal{F} and \mathcal{V} for the model system (1) are computed as follows.

$$\mathcal{F} = \begin{bmatrix} \beta S(1-\delta_m)I + \beta\alpha_m(1-\delta_m)S_mI \\ 0 \end{bmatrix},$$

$$\mathcal{V} = \begin{bmatrix} (\gamma + \gamma_m + \alpha + d)I \\ (\nu + d)R \end{bmatrix}$$

We get,

F = Jacobian of \mathcal{F} at DFE =

$$\begin{bmatrix} \beta S_0(1-\delta_m) + \beta\alpha_m(1-\delta_m)S_{m0} & 0 \\ 0 & 0 \end{bmatrix}$$

and

V = Jacobian of \mathcal{V} at DFE =

$$\begin{bmatrix} (\gamma + \gamma_m + \alpha + d) & 0 \\ 0 & (\nu + d) \end{bmatrix}$$

The basic reproduction number R_0 is given by the spectral radius (the dominant eigenvalue in magnitude) of the next generation matrix FV^{-1} . Hence $R_0 = \rho(FV^{-1})$, where,

$$FV^{-1} = \begin{bmatrix} \frac{\beta(1-\delta_m)(S_0 + \alpha_m S_{m0})}{(\gamma + \gamma_m + \alpha + d)} & 0 \\ 0 & 0 \end{bmatrix}$$

i.e.

$$R_0 = \frac{\beta(1-\delta_m)(S_0 + \alpha_m S_{m0})}{(\gamma + \gamma_m + \alpha + d)}$$

$$= \frac{\beta(1-\delta_m)A(d\mu_0 + \alpha_m \lambda \mu)}{d(\lambda \mu + d\mu_0)(\gamma + \gamma_m + \alpha + d)} \quad (2)$$

III. EQUILIBRIUM ANALYSIS

The system (1) has two equilibria, namely the disease-free equilibrium point $E_0(S_0, 0, 0, S_{m0}, M_0)$ and the endemic equilibrium point $E_1(S^*, I^*, R^*, S_m^*, M^*)$. For the disease

free equilibrium point, S_0, S_{m0} and M_0 are given by as follows:

$$S_0 = \frac{A\mu_0}{\lambda\mu + d\mu_0}, \quad S_{m0} = \frac{A\lambda\mu}{d(\lambda\mu + d\mu_0)},$$

$$M_0 = \frac{\mu}{\mu_0}, \quad I_0 = 0, \quad E_0 = 0.$$

The endemic equilibrium point $E_1(S^*, I^*, R^*, S_m^*, M^*)$ is obtained by putting the right hand sides of the system of equations (1) to zero. By solving these algebraic equations, we get following

$$M^* = \frac{\mu + \mu_1 I^*}{\mu_0},$$

$$R^* = \frac{(\gamma + \gamma_m)I^*}{\nu + d},$$

$$S_m^* = \frac{\lambda(\mu + \mu_1 I^*)S^*}{\mu_0[d + \beta\alpha_m(1-\delta_m)I^*]},$$

$$S^* = \frac{A + \frac{\nu(\gamma + \gamma_m)I^*}{(\nu + d)}}{\beta(1-\delta_m)I^* + \lambda\left(\frac{\mu + \mu_1 I^*}{\mu_0}\right) + d}$$

Also we get a relation between S^* and S_m^* as follows:

$$S^* + \alpha_m S_m^* = \frac{\gamma + \gamma_m + \alpha + d}{\beta(1-\delta_m)}. \quad (3)$$

Now using the above values of S^* and S_m^* in the equation (3), we get the following quadratic in I^* ,

$$D_1 I^{*2} + D_2 I^* + D_3 = 0, \quad (4)$$

where

$$D_1 = \beta(1-\delta_m)\alpha_m \{ \beta(1-\delta_m)\mu_0 + \lambda\mu_1 \}$$

$$\{ (\gamma + \gamma_m + \alpha + d)d + (\alpha + d)\nu \}$$

$$D_2 = (\nu + d) \{ \beta(1-\delta_m)\mu_0 + \lambda\mu_1 \}$$

$$\times \{ (\gamma + \gamma_m + \alpha + d)d - \beta(1-\delta_m)A\alpha_m \}$$

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

$$\times (\nu + d) - \nu(\gamma + \gamma_m)(\alpha_m \lambda \mu + \mu_0 d) \}$$

$$D_3 = (\gamma + \gamma_m + \alpha + d)(\nu + d)d(\lambda\mu + \mu_0 d)$$

$$- \beta(1-\delta_m)A(\nu + d)(\alpha_m \lambda \mu + \mu_0 d)$$

$$= -(\gamma + \gamma_m + \alpha + d)(\nu + d)d(\lambda\mu + \mu_0 d)(R_0 - 1)$$

Clearly, $D_1 > 0$ and $D_3 < 0$ under the condition on the reproduction number $R_0 > 1$. Hence the quadratic equation (4) has only one positive root irrespective of the sign of D_2 . This positive root, we name as I^* and then as all other variables are in terms of I^* , so can be calculated easily.

IV. 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 2 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

$$M_1 = \begin{pmatrix} m_{11} & m_{12} & \nu & 0 & -\lambda S_0 \\ 0 & m_{22} & 0 & 0 & 0 \\ 0 & m_{32} & m_{33} & 0 & 0 \\ m_{41} & m_{42} & 0 & -d & \lambda S_0 \\ 0 & \mu_1 & 0 & 0 & -\mu_0 \end{pmatrix}$$

here,

$$m_{11} = -(\lambda M_0 + d), m_{12} = -\beta(1 - \delta_m)S_0,$$

$$m_{22} = -(\gamma + \gamma_m + \alpha + d)(1 - R_0),$$

$$m_{32} = \gamma + \gamma_m, m_{33} = -(\nu + d), m_{41} = \lambda M_0,$$

$$m_{42} = -\beta\alpha_m(1 - \delta_m)S_{m_0}.$$

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

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

Theorem 3 The endemic equilibrium point $E_1(S^*, I^*, R^*, S_m^*, M^*)$ is locally asymptotically stable

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

$$\begin{vmatrix} a_4 & a_2 & a_0 & 0 \\ 1 & a_3 & a_1 & 0 \\ 0 & a_4 & a_2 & a_0 \\ 0 & 1 & a_3 & a_1 \end{vmatrix} > 0.$$

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 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 [20]. The rate of change of the variable representing the infected component of the system (1) can be rewritten as

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

where S_0 and S_{m_0} are same as in disease free equilibrium E_0 . However since $S \leq S_0$, & $S + S_m \leq S_0 + S_{m_0} = \frac{A}{d}$, so $S + \alpha_m S_m \leq S_0 + \alpha_m S_{m_0}$ as α_m lies between 0 and 1 and S, S_m, S_0, S_{m_0} are all positive. Hence

$$\frac{dI}{dt} \leq \{\beta(1 - \delta_m)(S_0 + \alpha_m S_{m_0}) - (\gamma + \gamma_m + \alpha + d)\} I \quad (5)$$

As for $R_0 < 1$ the bracketed term $\{\beta(1 - \delta_m)(S_0 + \alpha_m S_{m_0}) - (\gamma + \gamma_m + \alpha + d)\}$ of the inequality (5) is negative, thus it follows that $I \rightarrow 0$ as $t \rightarrow \infty$ by the comparison theorem in [20]. Also from the system (1) it is found that $S \rightarrow S_0, R \rightarrow 0, S_m \rightarrow S_{m_0}$ and $M \rightarrow M_0$ whenever $I = 0$. Thus for $R_0 < 1$, the disease

free equilibrium point $E_0(S_0, 0, 0, S_{m_0}, M_0)$ is globally asymptotically stable.

V. SIMULATION

The system (1) is simulated for various set of parameters using XPP [21]. The stability of disease free equilibrium point E_0 is shown in Fig. 2, where the reproduction number R_0 is equal to 0.799 which is less than one and parameter values are as follows:

$$A = 100, d = 0.01666, \beta = 0.000005, \alpha = 0.0002,$$

$$\alpha_m = 0.002, \delta_m = 0.2, \lambda = 0.0002, \nu = 0.03, \mu = 0.1,$$

$$\mu_0 = 0.03, \mu_1 = 0.001, \gamma_m = 0.01, \gamma = 0.002.$$

Figs. 3-6 are showing phase portraits in S-I, S-R, S- S_m and M-I planes respectively for the following set of parameter values:

$$A = 300, d = 0.01666, \beta = 0.000007, \alpha = 0.0002,$$

$$\alpha_m = 0.002, \delta_m = 0.2, \lambda = 0.0002, \nu = 0.03, \mu = 0.001,$$

$$\mu_0 = 0.03, \mu_1 = 0.001, \gamma_m = 0.01, \gamma = 0.002.$$

This corresponds to the stability of the endemic equilibrium point, for this set of parameter values the reproduction number R_0 is 3.4927.

Figs. 7-10 show the effects of parameters $\mu_1, \lambda, \gamma_m, \delta_m$ where increase in any of these parameter value gives rise to reduction in the equilibrium level of infective population. It has been observed that with the increase in any of these parameters except γ_m , the equilibrium level of recovered class decreases; where increase in γ_m increases the equilibrium level of recovered class as this parameter corresponds to the recovery of the infected individuals. Also it is found that with decrease in the parameter value of α_m , the equilibrium level of infective population as well as recovered population decreases i.e., when fraction of aware susceptibles interacting with infectives decreases then obviously this will cause the decrease in the equilibrium level of infectives. This fact is shown in Fig. 11 and it implies that media is able to convince people not to interact with infectives.

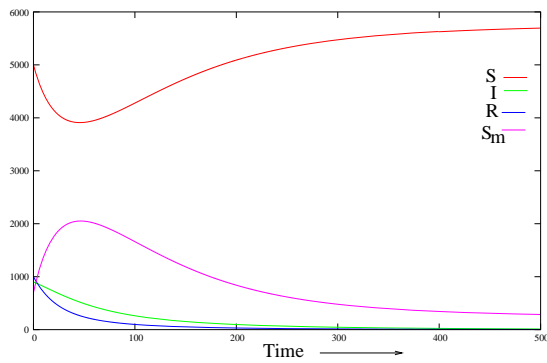


Fig. 2 Stability of the disease free equilibrium point E_0 for $R_0 = 0.799$.

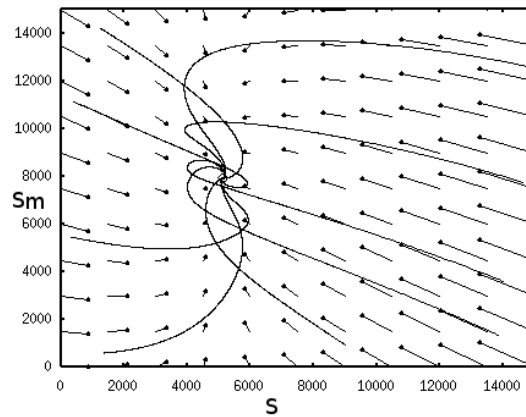


Fig. 5 Phase portrait corresponding to stability of endemic equilibrium point E_1 in $S - S_m$ plane.

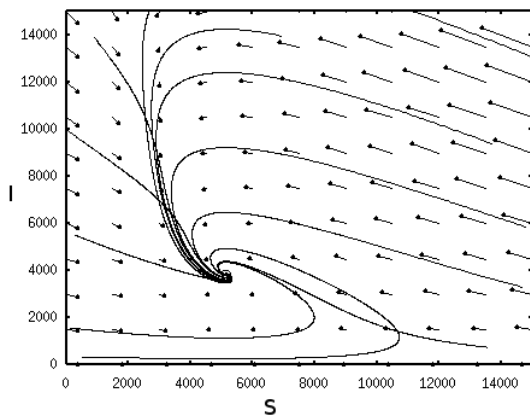


Fig. 3 Phase portrait corresponding to stability of endemic equilibrium point E_1 in $S-I$ plane.

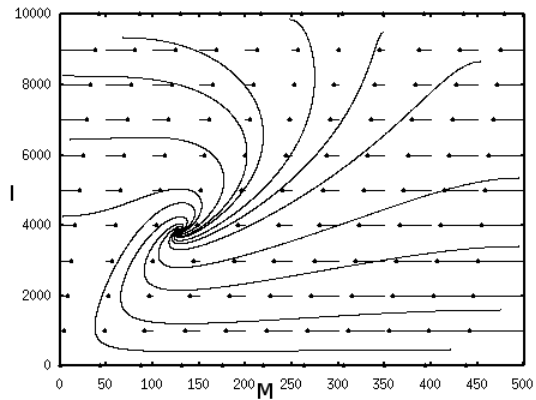


Fig. 6 Phase portrait corresponding to stability of endemic equilibrium point E_1 in $M-I$ plane.

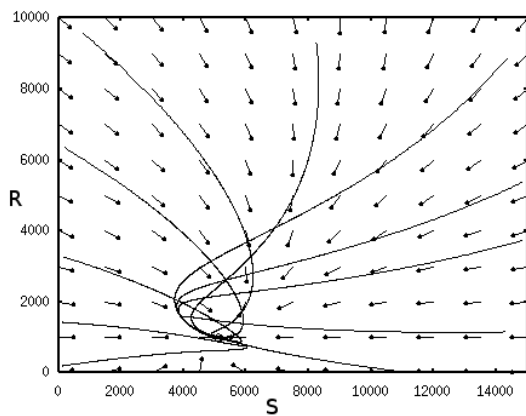


Fig. 4 Phase portrait corresponding to stability of endemic equilibrium point E_1 in $S-R$ plane.

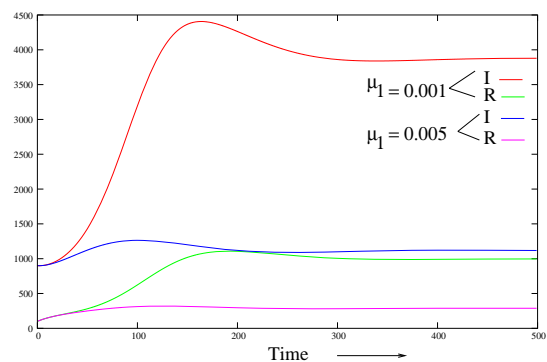


Fig. 7 Effect of μ_1 on the equilibrium levels of I and R classes.

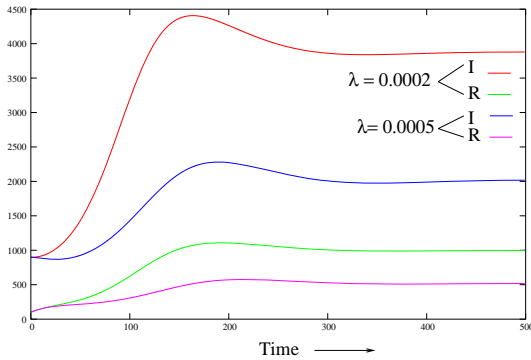


Fig. 8 Effect of λ on the equilibrium levels of I and R classes.

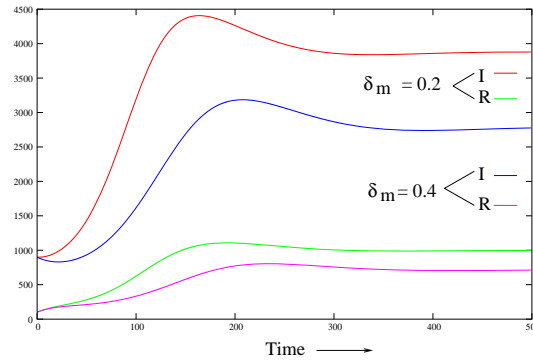


Fig. 10 Effect of δ_m on the equilibrium levels of I and R classes.

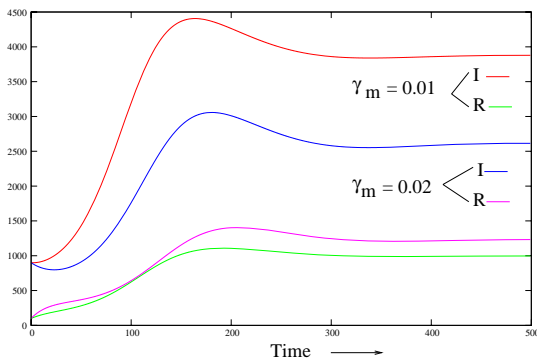


Fig. 9 Effect of γ_m on the equilibrium levels of I and R classes.

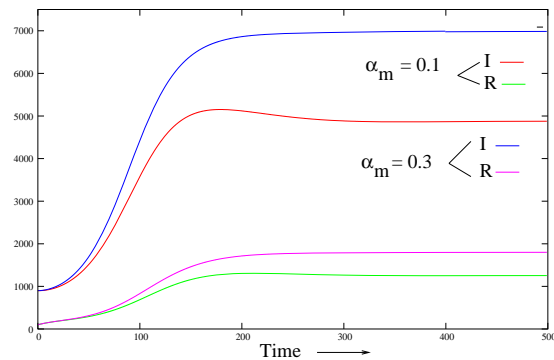


Fig. 11 Effect of α_m on the equilibrium levels of I and R classes.

VI. CONCLUSION

Our work provides an insight into the effects of media driven programs focusing on the transmission dynamics of infectious diseases. We have formulated and investigated a simple deterministic SIRS epidemic model incorporating the effect of media driven awareness programs on the transmission of diseases (such as influenza, tuberculosis, etc). This model has two equilibria: the disease free equilibrium point and the endemic equilibrium point. The disease free equilibrium point is locally as well as globally stable when the basic reproduction number $R_0 < 1$. The endemic equilibrium point exists only when $R_0 > 1$ and is locally asymptotically stable under some conditions on the parameter values. Finally, numerical simulation has been performed for varied set of parameters. The study suggests that with increase in the rate of implementation of awareness programs through media there is subsequent decline in the number of infectives in the targeted population. As the spread of infectious diseases depends on a variable number of factors, real time information dissemination about the disease and its risk factors through media have positive impact in controlling the transmission of infectious diseases as it changes people's perspective and behavior making them aware of the disease.

APPENDIX A: PROOF OF THEOREM 3

The variational matrix, M_2 corresponding to the system (1) at $E_1(S^*, I^*, R^*, S_m^*, M^*)$ is given by

$$M_1 = \begin{pmatrix} m_{11} & m_{12} & m_{13} & 0 & m_{15} \\ m_{21} & 0 & 0 & m_{24} & 0 \\ 0 & m_{32} & m_{33} & 0 & 0 \\ m_{41} & m_{42} & 0 & m_{44} & m_{45} \\ 0 & m_{52} & 0 & 0 & m_{55} \end{pmatrix}$$

where, $m_{11} = -\{\lambda M^* + d + \beta(1 - \delta_m)I^*\}$,
 $m_{12} = -\beta(1 - \delta_m)S^*$, $m_{13} = \nu$,
 $m_{15} = -\lambda S^*$, $m_{21} = \beta(1 - \delta_m)I^*$,
 $m_{24} = \beta(1 - \delta_m)\alpha_m I^*$, $m_{32} = \gamma + \gamma_m$,
 $m_{33} = -(\nu + d)$, $m_{41} = \lambda M^*$, $m_{42} = -\beta\alpha_m(1 - \delta_m)S^*$,
 $m_{44} = -\{d + \beta\alpha_m(1 - \delta_m)I^*\}$, $m_{45} = \lambda S^*$,
 $m_{52} = \mu_1$, $m_{55} = -\mu_0$

The characteristic polynomial corresponding to the variational matrix M_1 is given by

$$\psi^5 + a_4\psi^4 + a_3\psi^3 + a_2\psi^2 + a_1\psi + a_0 = 0,$$

where,

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

$$\begin{aligned}
a_3 &= m_{55}(m_{11} + m_{33}) + m_{11}m_{33} + (m_{52}m_{24} \\
&\quad - m_{42}m_{24}) - m_{21}m_{12} \\
a_2 &= -[(m_{11} + m_{33} + m_{55})(m_{52}m_{24} - m_{42}m_{24}) \\
&\quad + m_{11}m_{33}m_{55} + m_{21}(m_{32}m_{13} + m_{52}m_{15})] \\
&\quad + m_{12}m_{21}(m_{44} + m_{33} + m_{55}) - m_{12}m_{24}m_{41} \\
a_1 &= \{m_{55}(m_{11} + m_{33}) + m_{11}m_{33}\} \\
&\quad (m_{52}m_{24} - m_{42}m_{24}) + m_{11}m_{33}m_{44}m_{55} \\
&\quad + m_{21}(m_{32}m_{13}m_{55} + m_{52}m_{15}m_{33}) + (m_{32} \\
&\quad m_{13} + m_{52}m_{15})(m_{21}m_{44} - m_{41}m_{24}) \\
&\quad - m_{21}m_{12}\{m_{55}(m_{44} + m_{33}) + m_{44}m_{33}\} \\
&\quad + (m_{33} + m_{55})m_{12}m_{24}m_{41} \\
a_0 &= -m_{11}m_{33}m_{55}(m_{52}m_{24} - m_{42}m_{24}) - (m_{21} \\
&\quad m_{44} - m_{41}m_{24})(m_{32}m_{13}m_{55} + m_{52}m_{15}m_{33}) \\
&\quad - m_{12}m_{33}m_{55}(m_{24}m_{41} - m_{21}m_{44})
\end{aligned}$$

Here, clearly $a_4 > 0$, so E_1 will be locally asymptotically stable if the other three inequalities stated in Theorem 3 are also satisfied.

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REFERENCES

- [1] World Health Organization, Philadelphia: World Health Organization; 2005. WHO checklist for influenza pandemic preparedness planning. Available from: http://www.who.int/csr/resources/publications/influenza/WHO_CDS_CSR_GIP_2005_4/en/.
- [2] World Health Organization, Global Burden of disease: 2004 update, 2008. Available from: http://www.who.int/healthinfo/global_burden_disease/2004_report_update/en/index.html.
- [3] World Health Organization, Global Influenza Program. Philadelphia: World Health Organization; 2009. Pandemic Influenza Preparedness and Response. Available from: http://www.who.int/influenza/resources/documents/pandemic_guidance_04_2009/en/index.html.
- [4] C.R. Simpson, *Nature as News: Science Reporting in The New York Times 1898 to 1983*, The Int. J. Polit., Cult. Soc., 1 218-241, 1987.
- [5] M.D. Slater, K.A. Rasinski, *Media Exposure and Attention as Mediating Variables Influencing Social Risk Judgments*, J. Commun. 55 810-827, 2005.
- [6] R. Liu, J. Wu, H. Zhu, *Media/psychological impact on multiple outbreaks of emerging infectious diseases*, Comput. Math. Methods Med., 8 153-164, 2007.
- [7] J. Gholami, S.H. Hosseini, M. Ashoorkhani, R. Majdzadeh, *Lessons Learned from H1N1 Epidemic: The Role of Mass Media in Informing Physicians*, Int. J. Prev. Med., 2 32-37, 2011.
- [8] R.J. Blendon, J.M. Benson, C.M. DesRoches, E. Raleigh, K. Taylor-Clark, *The public's response to severe acute respiratory syndrome in Toronto and the United States*, Clin. Infect. Dis., 38 925, 2004.
- [9] A. Coulter, J. Ellins, *Patient-focused interventions: a review of the evidence*, The Health Foundation, London, 2006.
- [10] J. T. Shattuck, *Lecture medicine and the media*, N Engl J Med., 339 87-92, 1998.
- [11] A. Mensua, S. Mounier-Jack, R. Coker, *Pandemic influenza preparedness in Latin America: analysis of national strategic plans*, Health Policy Plan., 24 253-60, 2009.
- [12] J. Cui, Y. Sun, H. Zhu, *The Impact of Media on the Control of Infectious Diseases*, J. Dyn. Differ. Eqn., 20 31-53, 2007.
- [13] Y. Liu, J. Cui, *The Impact of Media Coverage on the Dynamics of Infectious disease*, Int. J. Biomath., 1 65-74, 2008.
- [14] Y. Li, J. Cui, *The effect of constant and pulse vaccination on SIS epidemic models incorporating media coverage*, Commun. Nonlinear Sci. Numer. Simul., 14 2353-2365, 2009.
- [15] Z. Mukandavire, W. Garira and J.M. Tchuente, *Modelling effects of public health educational campaigns on HIV/AIDS transmission dynamics*, Appl. Math. Model., 33 2084-2095, 2009.
- [16] C. Sun, W. Yang, J. Arino, K. Khan, *Effect of media-induced social distancing on disease transmission in a two patch setting*, Math. Biosci., Article in press, 2011.
- [17] J. M. Tchuente, N. Dube, C. P. Bhunu, R. J. Smith, C. T. Bauch, *The impact of media coverage on the transmission dynamics of human influenza*, BMC Public Health, 11(Suppl. 1)S5, 2011.
- [18] A. K. Misra, A. Sharma, J. B. Shukla, *Modeling and Analysis of effects of awareness programs by media on the spread of diseases*, Math. Comp. Model., 53 1221-1228, 2011.
- [19] P. Driessche, W. James, *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission*, Math. Biosci., 180 29-48, 2002.
- [20] V. Lakshmikantham, S. Leela, A.A. Martynuk, *Stability Analysis of Nonlinear Systems*, Marcel Dekker Inc., New York, 1989.
- [21] B. Ermentrout, *Simulating, Analyzing, and Animating Dynamical Systems: A Guide to XPPAUT for Researchers and Students (Software, Environments, Tools)*, 1st edition. SIAM, Philadelphia, PA, 2002.



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