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 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
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 infectious. 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 (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 $\alpha_m$) of aware susceptible 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 $\gamma_m$ which is driven by media awareness programs. As media also forces isolation/hospitalization of infectives, so let $\delta_m$ 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:

$$\frac{dS}{dt} = A - \beta S(1-\delta_m)I - \lambda SM - dS + \nu R$$

$$\frac{dI}{dt} = \beta S(1-\delta_m)I + \beta \alpha_m S_m(1-\delta_m)I - (\gamma + \gamma_m + \alpha + d)I - \nu R$$

$$\frac{dM}{dt} = \lambda SM - \beta \alpha_m S_m(1-\delta_m)I - dS_m$$

$$\frac{dR}{dt} = (\gamma + \gamma_m)I - \nu R - dR$$

(1)

Here, $A$ is the recruitment rate constant; $\beta$ is the transmission rate constant; $\lambda$ is the dissemination rate of awareness among susceptibles due to media awareness programs; $d$ is the natural death rate constant; $\nu$ is the rate at which individual from recovered class move to susceptible class again after loosing immunity; $\gamma$ is the natural recovery rate constant; $\alpha$ is the disease related death rate constant; $\mu$ is the rate constant corresponding to regular media coverage, $\mu_1$ is the rate constant influenced by number of infectives and $\mu_0$ is the natural death 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 $\Omega$ remain in the region $\Omega$. 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 $\Omega = \{(S_0, I_0, R_0, S_m(t)) \in R_+^4 : S_0 > 0, I_0 \geq 0, R_0 \geq 0, S_m(0) \geq 0; N \leq \frac{\mu}{\lambda}\}$

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

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

This follows that

$$0 \leq N(t) \leq \frac{A}{\mu}(1 - e^{-\mu t}) + N_0e^{-\mu t}, \ and \ for \ t \to \infty, \ we \ have$$

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

Thus, $\Omega$ 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,

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

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

$$\frac{dx}{dt} = F(x) - \begin{bmatrix} V_i^- (x) - y_i^- (x) \end{bmatrix} = F_i(x) - y_i (x).$$

Here $F_i(x)$ represents the rate of appearance of new infections.
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

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

i.e.

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

### 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 \( E_1(S^*, I^*, R^*, S_{m0}^*, M^*) \). 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 \( E_1(S^*, I^*, R^*, S_{m0}^*, M^*) \) is obtained by putting the right hand sides of the system of equations (1) to zero. By solving these algebraic equations, we get following

\[
M^* = \frac{\mu + \mu_1 I^*}{\beta_0},
R^* = \frac{\nu + d}{(\gamma + \gamma_m)I^*} S^*,
S_{m}^* = \frac{\mu_0 d + \beta_{1m}(1-\delta_m)I^*}{(\nu + d)} A + \frac{\lambda \mu I^*}{\mu_0} + d
\]

Also we get a relation between \( S^* \) and \( S_{m}^* \) as follows:

\[
S^* + \alpha_m S_{m}^* = \frac{\gamma + \gamma_m + \alpha + d}{\beta(1-\delta_m)}.
\]

Now using the above values of \( S^* \) and \( S_{m}^* \) in the equation (3), we get the following quadratic in \( I^* \):

\[
D_1 I^{*2} + D_2 I^* + D_3 = 0,
\]

where

\[
D_1 = \beta(1-\delta_m)\alpha_m \{\beta(1-\delta_m)\mu_0 + \lambda \mu_1 \}
D_2 = (\nu + d) \{(\gamma + \gamma_m + \alpha + d) d + (\alpha + d)\nu\}
+ \beta(1-\delta_m)\alpha_m(\lambda \mu + \mu_0 d)(\gamma + \gamma_m + \alpha + d)
+ \beta(1-\delta_m)\alpha_m(\lambda \mu + \mu_0 d)(\gamma + \gamma_m + \alpha + d)
+ (\nu + d) \{(\gamma + \gamma_m + \alpha + d) d + (\alpha + d)\nu\}
- \beta(1-\delta_m)\alpha_m(\lambda \mu + \mu_0 d)(\gamma + \gamma_m + \alpha + d)
= -(\gamma + \gamma_m + \alpha + d)(\nu + d)\lambda \mu + \mu_0 d)R_0 - 1
\]

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
variation 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,

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

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

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

$m_{42} = -\beta\alpha m(1-\delta_m)S_m.$

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 provided $a_4 > 0$, $a_4 a_2 a_0 > 0$, $a_4 a_2 a_0 > 0$, and $a_4 a_2 a_0 > 0$.

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

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

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

$$
\frac{dI}{dt} \leq \left\{ \beta(1-\delta_m)(S_0 + \alpha_m S_m) - (\gamma + \gamma_m + \alpha + d) \right\} I
$$

(5)

As for $R_0 < 1$ the bracketed term $\left\{ \beta(1-\delta_m)(S_0 + \alpha_m S_m) - (\gamma + \gamma_m + \alpha + d) \right\}$ is negative, thus it follows that $I \to 0$ as $t \to \infty$ by the comparison theorem in [20]. Also from the system (1) it is found that $S \to S_0, R \to 0, S_m \to S_m$ and $M \to M_0$ whenever $I = 0$. Thus for $R_0 < 1$, the disease free equilibrium point $E_0(S_0, 0, 0, S_m, M_0)$ 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:

$$
A = 100, d = 0.01666, \beta = 0.000005, \alpha = 0.0002, \gamma_m = 0.002, \delta_m = 0.2, \lambda = 0.0002, \nu = 0.03, \mu = 0.1, \mu_0 = 0.03, \mu_1 = 0.001, \gamma_m = 0.01, \gamma = 0.002.
$$

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

$$
A = 300, d = 0.01666, \beta = 0.000007, \alpha = 0.0002, \gamma_m = 0.002, \delta_m = 0.2, \lambda = 0.0002, \nu = 0.03, \mu = 0.001, \mu_0 = 0.03, \mu_1 = 0.001, \gamma_m = 0.01, \gamma = 0.002.
$$

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 endemic equilibrium level of infective population. It has been observed that with the increase in any of these parameters except $\gamma_m$, the equilibrium level of recovered class decreases; where increase in $\gamma_m$ 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 $\alpha_m$, 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. 3 Phase portrait corresponding to stability of endemic equilibrium point $E_1$ in $S-I$ plane.

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

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

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

Fig. 7 Effect of $\mu_1$ on the equilibrium levels of $I$ and $R$ classes.
Fig. 8 Effect of $\lambda$ on the equilibrium levels of I and R classes.

Fig. 9 Effect of $\gamma_m$ on the equilibrium levels of I and R classes.

Fig. 10 Effect of $\delta_m$ on the equilibrium levels of I and R classes.

Fig. 11 Effect of $\alpha_m$ 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,

- $m_{11} = -\{\lambda M^* + d + \beta(1 - \delta_m)I^*\}$,
- $m_{12} = -\beta(1 - \delta_m)S^*$, $m_{13} = \nu$,
- $m_{15} = -\lambda S^*$, $m_{31} = \beta(1 - \delta_m)I^*$,
- $m_{24} = \beta(1 - \delta_m)I^*$, $m_{32} = \gamma + \gamma_m$,
- $m_{33} = -(\nu + d)$, $m_{41} = \lambda M^*$, $m_{42} = -\beta \alpha_m (1 - \delta_m) S^*$,
- $m_{44} = -(d + \beta \alpha_m (1 - \delta_m) I^*)$, $m_{45} = \lambda S^*$,
- $m_{52} = \mu_1$, $m_{55} = -\mu_0$.

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,

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