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Role of media and treatment on an SIR model

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Abstract. In this paper, the impact of awareness programs as well as treatment on an SIR model has been investigated. We assume that the whole population is divided into four compartments, named as susceptible (S), infected (I), aware susceptible (S_a) and recovered (R). Analytical findings and numerical simulations of the model show that if the exposure to the awareness program is high and adequate treatment is available, then the infection can be eliminated. Analysis of the model also depicts that if treatment is not available, then infection is high even if enough awareness is present. But in absence of awareness an infection can not be eliminated inspite of adequate treatment. Effective treatment can led to a diminished level of infection. Stability analysis of the model is investigated by using stability theory of differential equations. Further, numerical simulations are carried out to validate the analytical results.

Keywords: awareness program, stability analysis, aware susceptible, SIR model.

1 Introduction

World Health Organization report [7] has shown that approximately 15 million people die each year due to infectious diseases. When an epidemic spreads in a society, there is a need for effective treatment to control the epidemic. Moreover, vaccination is a prophylactic measure to control the spread of the disease in susceptible individuals. Medical facilities and subsequent therapies may require some time to be developed and implemented. If individuals are familiar with the disease and have knowledge about the transmission modality of infection then they can take necessary preventive measures to avoid infection. The susceptible individuals may isolate themselves from infected individuals or they can take necessary prophylactic measures. Infection can be reduced by awareness among susceptibles but will not be eradicated. To control the spread of further infection and to

eradicate the infection from the society, there is a requirement of not only the awareness programs but also treatment.

Literature shows that several SIR models have been studied with different type of nonlinear incidence rates. Authors [2, 8, 15, 19] analyzed the dynamics of SIR models with different type of nonlinear incidence rates. Pathak et al. [15] proposed an SIR model with an asymptotic homogeneous transmission function and concluded that the spread of disease decreases as the social or psychological protection measures for the infection increases. Kaddar [8] studied the role of incubation period on the dynamics of an SIR model. Further, Xu [19] investigated the global stability dynamics of an SEIR epidemic model with disease relapse, a saturated incidence rate and a time delay describing the latent period of the disease. In [2], Buonomo et al. studied an SIR model with vaccination and treatment and obtained threshold on the basic reproduction number to control the spread of disease.

Surveys indicate that people who watch television [9, 16] or read article or magazine related to the public health on daily basis are more aware about the ways of spread of infection as compared to those who do not do so. Awareness programs run by media campaigns induce the behavioral changes in the susceptibles towards infection. These campaigns of awareness through media and education focus on individual's knowledge about the disease transmission and facilitate measures that can reduce the chances of being infected. Annual report of NACO [1] shows that the awareness campaigns about the HIV/AIDS driven by government are very helpful in controlling the epidemic. Many authors [4, 5, 11] have introduced models showing the effect of awareness programs run by media to control the spread of epidemic. Liu et al. [12] have studied the psychological impact on epidemic. They have postulated that an increase in infection level reduces the effective contacts but they did not take into account factors of mandatory quarantine and isolation. Further, Kiss et al. [10] proposed an SIS type compartmental model for Sexually Transmitted Infections with the assumption that the whole population is aware of risk but only a certain proportion chooses to respond by limiting their contact with infectives and seeking faster treatment. They have assumed that the total number of susceptibles remains relatively unchanged. The demographic factors such as natural birth rate, death rate, immigration were ignored. Funk et al. [6] studied the impact of awareness programs on the spread of epidemic using mathematical modeling and showed that awareness programs play a vital role in reducing the spread of epidemic. However, in all these studies, the density of awareness programs is considered to be a constant which need not be true in real life.

Misra et al. [13] proposed a non-linear mathematical model for the effects of awareness programs on the spread of infectious diseases, like flu. They have shown that awareness programs through the media campaigning are helpful in decreasing the spread of infectious diseases. This is done by isolating a fraction of susceptibles from infectives. Further, Misra et al. [14] proposed a non-linear mathematical model with delay to study the dynamics of the effects of awareness programs on prevalence of any epidemic. In this study, they have shown that though awareness programs cannot eradicate the infection but they can help in controlling disease prevalence. They have also shown that time delay in execution of awareness programs destabilizes the system and periodic solutions may arise

through Hopf-bifurcation. Recently, Samanta et al. [17] proposed a mathematical model to assess the effect of awareness programs by media on the prevalence of infectious diseases. They have shown that if the rate of implementation of awareness programs through the media increases, the number of individuals getting infected decline and the system remains stable up to a threshold value of implementation of awareness program. But, the system becomes unstable above that threshold. They have also observed that for moderate range of value of immigration rate the system shows unstable dynamics, but for lower and higher values the system becomes stable. Further, Cai et al. [3] studied the effect of treatment on HIV/AIDS epidemic in their model and showed that the disease may persist or die out depending on treatment parameter values. They have also considered time delay in their model to study the effect of time on dynamics of endemic equilibrium. Recently, Sharma and Misra [18] studied the impact of awareness program on the coverage of vaccination of hepatitis B. They showed that vaccination coverage can be increased and prevalence of the disease can be decreased by taking appropriate steps by media.

Since awareness programs alone can not eradicate the disease, treatment of the disease in infected population must go along with awareness programs for the susceptible. The effect of treatment has not been considered in the models studied by Misra et al. [13, 14, 17]. Keeping this aspect in view, we present here an SIR model to study the impact of two important parameters: (i) awareness programs (run by media), and (ii) treatment on the spread of an infectious disease.

2 Mathematical model

We assume that the whole population is first divided into three compartments, namely susceptible population (S), infected population (I) and recovered population (R). Next, we assume that a part of the susceptible population forms another class called susceptible aware population (S_a). This class develops due to awareness programs driven by social/electronic media of density M at any time t. When the media interacts with the susceptible population, it starts influencing them to take appropriate measures so that they should not be infected by the pathogens. This media influence is initially low and increases as the infection increases. But media can influence susceptible population only upto a certain level after that it gets saturated due to the resource limitation [12, 14]. Generally media sources do not deal with the same topic for a very long time. Their emphasis changes with changing social/political issues. Moreover, their impact may not reach to the entire population due to factors like time shortage, illiteracy and financial limitation of population as well as resources required to propagate information. Thus, neither media nor its impact can go on increasing forever and therefore attains saturation. Hence we assume that the impact of media on susceptible populations is governed by Holling type II functional response [12, 14]. It is also considered that the growth rate of the cumulative density of awareness programs driven by the media is proportional to the number of infectives present in the population. Further, the awareness about the disease will alert susceptibles to isolate themselves from infectives and avoid being infected by forming a separate class. The effect of depletion of awareness programs has also been

considered. We also consider the treatment rate as saturated treatment rate due to limited availability of resources in community. The model is given by following differential equations:

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= A - \delta_0 S - \alpha SI - \frac{\beta SM}{1+\gamma M} + \delta_3 S_a,\\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \alpha SI - \delta_0 I - \delta_1 I - \delta_2 I - \frac{aI}{1+bI},\\
\frac{\mathrm{d}S_a}{\mathrm{d}t} &= \frac{\beta SM}{1+\gamma M} - \delta_0 S_a - \delta_3 S_a,\\
\frac{\mathrm{d}M}{\mathrm{d}t} &= \mu I - \mu_0 M,\\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \delta_2 I - \delta_0 R + \frac{aI}{1+bI},\\
S(0) > 0, \qquad I(0) > 0, \qquad S_a(0) &\geqslant 0, \qquad M(0) \geqslant 0, \qquad R(0) \geqslant 0.
\end{aligned} \tag{1}$$

Let the susceptibles be recruited at a constant rate A and δ_0 be the natural death rate of the population in each class. δ_1 be the death rate of infected individuals due to infection and δ_2 be natural recovery rate of infected individuals due to immunity. Realistically speaking, the whole aware population may not keep themselves isolated. Due to negligence or loss of memory, a part of the aware population may become susceptible to the disease. Let δ_3 be the rate of transfer of aware individuals to susceptible class. μ be the implementation rate of awareness programs. The awareness programs may be slowed down due to treatment of the disease or due to the diversion caused by other hot topics of high priority coming in the media. Let μ_0 be the depletion rate of awareness programs, α is the incidence rate which is the number of persons getting infected per unit of time. For any outbreak of the disease, its treatment initially is slow due to non-availability of the treatment techniques and appropriate drugs. After some time, the treatment grows with the improvement in hospitals' conditions, effective drugs and skillful techniques. Thus, it is better to use saturated treatment rate which is given by

$$h(I) = \frac{aI}{1+bI},$$

where a and b are positive constants. In the above treatment function, a/b ($a/b = \lim_{I\to\infty} h(I)$) denotes the maximum supply of medical resources per unit time and $1/(1+bI)$ denotes the reverse effect of infected individuals being delayed for treatment [20].

Similar interpretation may be given for the term $f(M) = \beta M/(1+\gamma M)$, which denotes the effect of media coverage on susceptible population. β can be thought of as the dissemination rate of awareness programs among susceptibles and γ limits the effect of awareness programs on susceptibles, β/γ is the maximum effect that media can put on susceptibles.

From the above system (1) we can infer that S, I, S_a and M are free from the effect of R as we assume immunity in recovered individuals. Thus, it is enough to consider the

following sub-system:

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= A - \delta_0 S - \alpha SI - \frac{\beta SM}{1+\gamma M} + \delta_3 S_a,\\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \alpha SI - \delta_0 I - \delta_1 I - \delta_2 I - \frac{aI}{1+bI},\\
\frac{\mathrm{d}S_a}{\mathrm{d}t} &= \frac{\beta SM}{1+\gamma M} - \delta_0 S_a - \delta_3 S_a,\\
\frac{\mathrm{d}M}{\mathrm{d}t} &= \mu I - \mu_0 M.
\end{aligned} \tag{2}$$

Let $S + I + S_a = N$, then system (2) reduces to

$$\begin{aligned}
\frac{\mathrm{d}I}{\mathrm{d}t} &= \alpha(N - I - S_a)I - \delta I - \frac{aI}{1+bI},\\
\frac{\mathrm{d}S_a}{\mathrm{d}t} &= \frac{\beta(N - I - S_a)M}{1+\gamma M} - \delta_0 S_a - \delta_3 S_a,\\
\frac{\mathrm{d}N}{\mathrm{d}t} &= A - \delta_0 N - (\delta_1 + \delta_2)I - \frac{aI}{1+bI},\\
\frac{\mathrm{d}M}{\mathrm{d}t} &= \mu I - \mu_0 M,
\end{aligned} \tag{3}$$

where $\delta = \delta_0 + \delta_1 + \delta_2$.

For the above system (3), a region of attraction has been found and it is given in Lemma 1.

Lemma 1. *The set* $\Omega = \{(I, S_a, N, M) \in \Re^4_+ \colon 0 < I + S_a \leqslant N \leqslant A/\delta_0,\ 0 \leqslant M \leqslant \mu A/(\delta_0\mu_0)\}$ *is a positively invariant region of system* (3)*.*

Proof. Let $W(t) = (N(t), M(t))$, then

$$\frac{\mathrm{d}W}{\mathrm{d}t} = \left(\frac{\mathrm{d}N}{\mathrm{d}t}, \frac{\mathrm{d}M}{\mathrm{d}t}\right) = \left(A - \delta_0 N - (\delta_1 + \delta_2)I - \frac{aI}{1+bI},\ \mu I - \mu_0 M\right). \tag{4}$$

We note that $\mathrm{d}N/\mathrm{d}t \leqslant A - \delta_0 N \leqslant 0$ if $N \geqslant A/\delta_0$ and $\mathrm{d}M/\mathrm{d}t \leqslant \mu A/\delta_0 - \mu_0 M \leqslant 0$ if $M \geqslant \mu A/(\mu_0\delta_0)$.

From equation (4) $\mathrm{d}W/\mathrm{d}t \leqslant 0$ for $N \geqslant A/\delta_0$ and $M \geqslant \mu A/(\mu_0\delta_0)$. This shows that the set Ω is a positively invariant set. From the third equation of model (3) we have

$$\frac{\mathrm{d}N}{\mathrm{d}t} \leqslant A - \delta_0 N,$$

which implies that

$$\lim_{t\to\infty} \sup N(t) \leqslant \frac{A}{\delta_0}.$$

From the last equation of model (3) it follows that

$$\frac{\mathrm{d}M}{\mathrm{d}t} \leqslant \frac{\mu A}{\delta_0} - \mu_0 M,$$

and hence

$$\lim_{t\to\infty} \sup M(t) \leqslant \frac{\mu A}{\delta_0 \mu_0}.$$

This completes the proof of Lemma 1. □

The above lemma shows that all solutions of the model are non-negative and bounded. Thus, the model is biologically well-behaved.

In the next section, we discuss the existence of equilibrium points of system (3).

3 Equilibrium analysis

We see that system (3) has only two equilibria:

(i) the disease-free equilibrium (DFE) $E_0(0, 0, A/\delta_0, 0)$, and
(ii) the endemic equilibrium (EE) $E_1(I^*, {S_a}^*, N^*, M^*)$.

We can infer from system (3) that the disease-free equilibrium E_0 exists without any condition. Now we need to check the existence of endemic equilibrium E_1.

The existence of E_1. We note that I^*, ${S_a}^*$, N^*, and M^* are the positive solutions of the following system of algebraic equations:

$$\begin{aligned}
&\alpha(N^* - I^* - {S_a}^*) - \delta - \frac{a}{1+bI^*} = 0,\\
&\frac{\beta(N^* - I^* - {S_a}^*)M^*}{1+\gamma M^*} - \delta_0 {S_a}^* - \delta_3 {S_a}^* = 0,\\
&A - \delta_0 N^* - (\delta_1 + \delta_2)I^* - \frac{aI^*}{1+bI^*} = 0,\\
&\mu I^* - \mu_0 M^* = 0.
\end{aligned}$$

After solving the above equations, we get

$$\begin{aligned}
M^* &= \frac{\mu I^*}{\mu_0},\\
{S_a}^* &= \frac{\beta\mu(\delta + a + \delta b I^*)I^*}{\alpha(\delta_0+\delta_3)(1+bI^*)(\mu_0+\mu\gamma I^*)},\\
N^* &= I^* + {S_a}^* + \frac{(\delta + a + \delta b I^*)}{\alpha(1+bI^*)},\\
A_1 {I^*}^3 &+ A_2 {I^*}^2 + A_3 I^* + A_4 = 0,
\end{aligned} \tag{5}$$

where

$$\begin{aligned}
A_1 &= b\delta\alpha\mu\gamma(\delta_0+\delta_3),\\
A_2 &= \alpha(\delta_0+\delta_3)(\mu_0\delta b + \gamma\mu(\delta+a)) + \beta\mu\delta\delta_0 b\\
&\quad + \delta_0(\delta_0+\delta_3)\delta b\mu\gamma - \alpha A(\delta_0+\delta_3)b\mu\gamma,
\end{aligned}$$

$$A_3 = \alpha(\delta_0+\delta_3)\mu_0(\delta+a) + \beta\mu\delta_0(\delta+a) + \delta_0(\delta_0+\delta_3)(\delta b\mu_0 + (\delta+a)\mu\gamma) - \alpha A(\delta_0+\delta_3)(b\mu_0+\mu\gamma),$$
$$A_4 = \delta_0(\delta_0+\delta_3)(\delta+a)\mu_0 - \alpha A(\delta_0+\delta_3)\mu_0.$$

By Descartes' rule of sign, one can see that the cubic equation (5) has unique positive real root I^* if the following inequality hold:

$$1 < R_0 < \frac{\alpha}{b\delta_0}, \tag{6}$$

where $R_0 = A\alpha/((\delta+a)\delta_0)$ is the basic reproductive number.

After finding the value of I^*, we can find the values of N^*, ${S_a}^*$ and M^*. This implies that there exists a unique endemic equilibrium $E_1(I^*, {S_a}^*, N^*, M^*)$ if inequality (6) is satisfied.

Remark. Equation (6) shows a threshold on the basic reproduction number R_0 which depends upon α, δ_0 and b. We note that if the parameter b (delay in the treatment) is large, then equation (6) may not be satisfied and thus more than one positive equilibrium may exist.

4 Stability analysis

In this section, we discuss the local and global stability of the equilibrium points, $E_0(0,0,A/\delta_0,0)$ and $E_1(I^*, {S_a}^*, N^*, M^*)$. By calculating the Jacobian matrix at E_0, we note that three eigenvalues are always negative and the fourth one is negative if $R_0 < 1$ and positive if $R_0 > 1$. Thus, we state the following theorem.

Theorem 1. *The disease-free equilibrium $E_0(0,0,A/\delta_0,0)$ is locally asymptotically stable if $R_0 < 1$ and is unstable if $R_0 > 1$.*

Remark. If $R_0 = 1$, then one eigenvalue of the Jacobian matrix evaluated at E_0 is zero with multiplicity one (simple zero) and other three eigenvalues are $-(\delta_0+\delta_3), -\delta_0$ and $-\mu_0$, which are real and negative. Thus, E_0 is linearly locally stable. If $R_0 > 1$, then E_0 is unstable. This shows that transcritical bifurcation occurs at $R_0 = 1$, which is shown in Fig. 1.

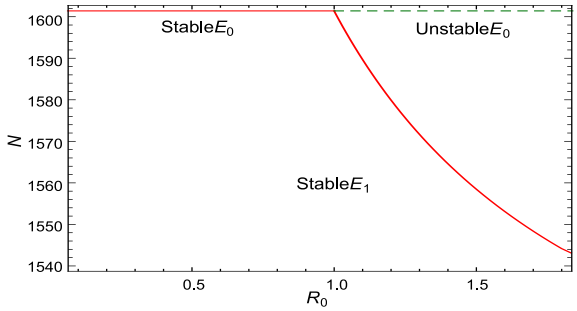


Figure 1. Plot of N vs R_0 showing the transcritical bifurcation at $R_0 = 1$.

In the following theorem, we have found some conditions for E_1 to be locally asymptotically stable.

Theorem 2. *The endemic equilibrium $E_1(I^*, {S_a}^*, N^*, M^*)$ is locally asymptotically stable if the following inequalities hold true*:

$$\alpha > \frac{ab}{(1+bI^*)^2}, \tag{7}$$

$$\left(\alpha + c_1\frac{\beta M^*}{1+\gamma M^*}\right)^2 < c_1\frac{4}{9}\left(\alpha - \frac{ab}{(1+bI^*)^2}\right)p, \tag{8}$$

where

$$c_1 < \min\left\{\frac{2}{9}\frac{(1+\gamma M^*)^4}{(\beta(N^*-I^*-{S_a}^*))^2}\frac{{\mu_0}^2}{\mu^2}\left(\alpha - \frac{ab}{(1+bI^*)^2}\right)p,\ \frac{2}{3}\frac{\alpha p}{q}\delta_0\frac{(1+\gamma M^*)^2}{(\beta M^*)^2}\right\}$$

and

$$p = \delta_0 + \delta_3 + \frac{\beta M^*}{1+\gamma M^*}, \qquad q = \delta_1 + \delta_2 + \frac{a}{(1+bI^*)^2}.$$

Proof. Let $x = I - I^*$, $y = S_a - {S_a}^*$, $n = N - N^*$, $m = M - M^*$ be the small perturbations about the endemic equilibrium E_1. Using the above new variables, we linearize model system (3) around the endemic equilibrium E_1. Then in the linear model, we consider the following positive definite function:

$$V_1 = \frac{1}{2I^*}x^2 + \frac{c_1}{2}y^2 + \frac{c_2}{2}n^2 + \frac{c_3}{2}m^2, \tag{9}$$

where c_1, c_2 and c_3 are positive constants to be chosen suitably.

Now differentiating V_1 with respect to time t along the solutions of model (3), a little algebraic manipulation yields

$$\begin{aligned}\frac{\mathrm{d}V_1}{\mathrm{d}t} = {}& -\frac{1}{2}a_{11}x^2 + a_{12}xy - \frac{1}{2}a_{22}y^2 \\ & - \frac{1}{2}a_{11}x^2 + a_{13}xn - \frac{1}{2}a_{33}n^2 - \frac{1}{2}a_{11}x^2 + a_{14}xm - \frac{1}{2}a_{44}m^2 \\ & - \frac{1}{2}a_{22}y^2 + a_{23}yn - \frac{1}{2}a_{33}n^2 - \frac{1}{2}a_{22}y^2 + a_{24}ym - \frac{1}{2}a_{44}m^2,\end{aligned}$$

where

$$\begin{aligned} a_{11} &= \frac{2}{3}\left(\alpha - \frac{ab}{(1+bI^*)^2}\right), \qquad a_{22} = \frac{2}{3}c_1p, \qquad a_{33} = c_2\delta_0, \qquad a_{44} = c_3\mu_0, \\ a_{12} &= -\left(\alpha + c_1\frac{\beta M^*}{1+\gamma M^*}\right), \qquad a_{13} = \alpha - c_2q, \qquad a_{14} = c_3\mu, \\ a_{23} &= c_1\frac{\beta M^*}{1+\gamma M^*}, \qquad a_{24} = c_1\frac{\beta(N^*-I^*-{S_a}^*)}{(1+\gamma M^*)^2}. \end{aligned}$$

Sufficient conditions for $\mathrm{d}V_1/\mathrm{d}t$ to be negative definite are given as follows:

$$a_{11} > 0, \tag{10}$$

$$a_{12}^2 < a_{11}a_{22}, \tag{11}$$

$$a_{13}^2 < a_{11}a_{33}, \tag{12}$$

$$a_{14}^2 < a_{11}a_{44}, \tag{13}$$

$$a_{23}^2 < a_{22}a_{33}, \tag{14}$$

$$a_{24}^2 < a_{22}a_{44}. \tag{15}$$

By choosing $c_2 = \alpha/q$ and $c_3 = \mu_0/(3\mu^2)(\alpha - ab/(1+bI^*)^2)$, we note that conditions (12) and (13) are satisfied. If we choose c_1 as given in Theorem 2, then conditions (14) and (15) are satisfied. Finally, we note that (7) implies (10) and (8) implies (11). Hence the theorem follows. □

In the following theorem, we show that the endemic equilibrium $E_1(I^*, {S_a}^*, N^*, M^*)$ is globally asymptotically stable.

Theorem 3. *Let the following inequalities hold in Ω*:

$$\alpha > \frac{ab}{(1+bI^*)}, \tag{16}$$

$$\left(\alpha + k_1\frac{\beta M^*}{1+\gamma M^*}\right)^2 < k_1\frac{4}{9}\left(\alpha - \frac{ab}{(1+bI^*)}\right)p, \tag{17}$$

where

$$k_1 < \min\left\{\frac{2}{9}\left(\frac{\delta_0(1+\gamma M^*)}{\beta A}\right)^2\frac{{\mu_0}^2}{\mu^2}\left(\alpha - \frac{ab}{(1+bI^*)}\right)p,\ \frac{2}{3}\frac{\alpha\delta_0 p}{r}\left(\frac{(1+\gamma M^*)}{\beta M^*}\right)^2\right\},$$

and

$$r = \delta_1 + \delta_2 + \frac{a\delta_0}{(1+bI^*)(\delta_0 + Ab)}.$$

Then $E_1(I^*, {S_a}^*, N^*, M^*)$ is globally asymptotically stable with respect to all solutions initiating in the interior of the positive octant Ω.

Proof. We consider the following positive definite function about E_1:

$$V_2 = I - I^* - I^*\ln\frac{I}{I^*} + \frac{k_1}{2}(S_a - {S_a}^*)^2 + \frac{k_2}{2}(N-N^*)^2 + \frac{k_3}{2}(M-M^*)^2,$$

where k_1, k_2 and k_3 are positive constants to be chosen suitably.

Now differentiating V_2 with respect to time t along the solutions of model (3), we get

$$\begin{aligned}\frac{\mathrm{d}V_2}{\mathrm{d}t} = &-\frac{1}{2}b_{11}(I-I^*)^2 + b_{12}(I-I^*)(S_a - {S_a}^*) - \frac{1}{2}b_{22}(S_a - {S_a}^*)^2\\ &-\frac{1}{2}b_{11}(I-I^*)^2 + b_{13}(I-I^*)(N-N^*) - \frac{1}{2}b_{33}(N-N^*)^2\end{aligned}$$

$$
\begin{aligned}
&-\frac{1}{2}b_{11}(I-I^*)^2+b_{14}(I-I^*)(M-M^*)-\frac{1}{2}b_{44}(M-M^*)^2\\
&-\frac{1}{2}b_{22}(S_a-{S_a}^*)^2+b_{23}(S_a-{S_a}^*)(N-N^*)-\frac{1}{2}b_{33}(N-N^*)^2\\
&-\frac{1}{2}b_{22}(S_a-{S_a}^*)^2+b_{24}(S_a-{S_a}^*)(M-M^*)-\frac{1}{2}b_{44}(M-M^*)^2,
\end{aligned}
$$

where

$$
\begin{gathered}
b_{11}=\frac{2}{3}\left(\alpha-\frac{ab}{(1+bI^*)(1+bI)}\right),\qquad b_{22}=\frac{2}{3}k_1p,\qquad b_{33}=k_2\delta_0,\qquad b_{44}=k_3\mu_0,\\
b_{12}=-\left(\alpha+k_1\frac{\beta M^*}{1+\gamma M^*}\right),\qquad b_{13}=\alpha-k_2\left(\delta_1+\delta_2+\frac{a}{(1+bI)(1+bI^*)}\right),\\
b_{14}=k_3\mu,\qquad b_{23}=k_1\frac{\beta M^*}{1+\gamma M^*},\qquad b_{24}=k_1\frac{\beta(N-I-S_a)}{(1+\gamma M^*)(1+\gamma M)}.
\end{gathered}
$$

Sufficient conditions for $\mathrm{d}V_2/\mathrm{d}t$ to be negative definite are given as follows:

$$
\begin{aligned}
&b_{11}>0, && (18)\\
&b_{12}^2<b_{11}b_{22}, && (19)\\
&b_{13}^2<b_{11}b_{33}, && (20)\\
&b_{14}^2<b_{11}b_{44}, && (21)\\
&b_{23}^2<b_{22}b_{33}, && (22)\\
&b_{24}^2<b_{22}b_{44}. && (23)
\end{aligned}
$$

For the given value of k_1 in Theorem 3, we note that conditions (22) and (23) are satisfied. Again (16) $\Rightarrow$ (18) and (17) $\Rightarrow$ (19). If we choose $k_2=\alpha/r$ and $k_3=\mu_0/(3\mu^2)(\alpha-ab/(1+bI^*))$, then (21) and (22) are satisfied. This implies that V_2 is a Liapunov's function with respect to the endemic equilibrium $E_1(I^*,{S_a}^*,N^*,M^*)$. Hence the theorem follows. □

5 Numerical simulations

In this section, we present computer simulation results for model system (3) by using MatLab 7.10. We choose the following set of values of parameters:

$$
\begin{aligned}
&A=250, && \delta_0=0.2, && \delta_1=0.005, && \delta_2=0.002,\\
&\delta_3=0.18, && a=0.2, && b=0.0003, && \alpha=0.0005, && (24)\\
&\beta=0.0022, && \gamma=0.008, && \mu=0.005, && \mu_0=0.24.
\end{aligned}
$$

For these values of parameters, we see that the endemic equilibrium $E_1(I^*,{S_a}^*,N^*,M^*)$ exists and $(I^*,{S_a}^*,N^*,M^*)$ are given as follows:

$$
I^*=223.42,\quad {S_a}^*=20.4951,\quad N^*=1032.8\quad\text{and}\quad M^*=4.6546.
$$

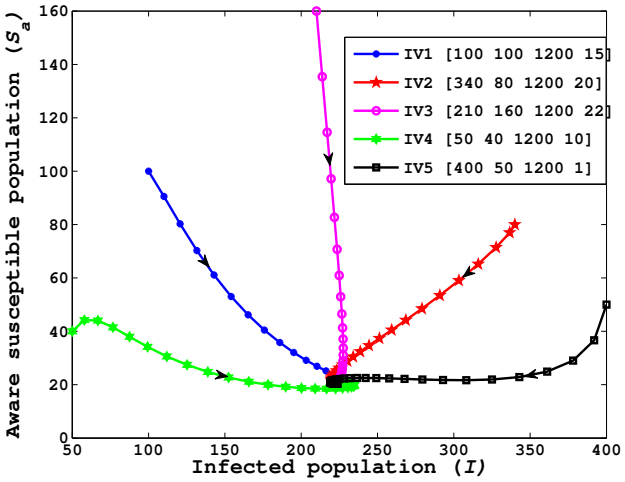


Figure 2. Global stability of $(I^*, {S_a}^*)$ in IS_a-plane.

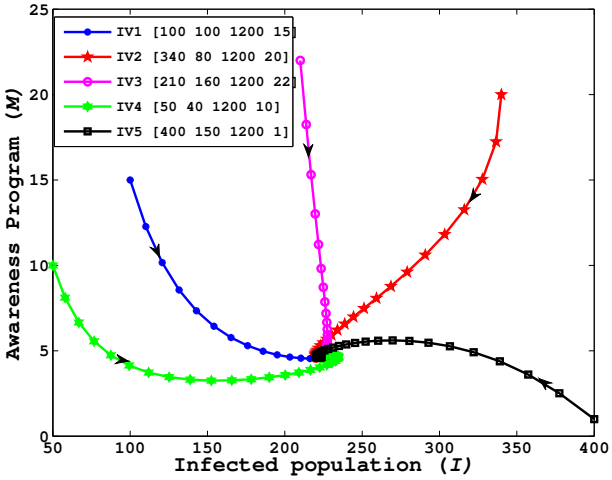
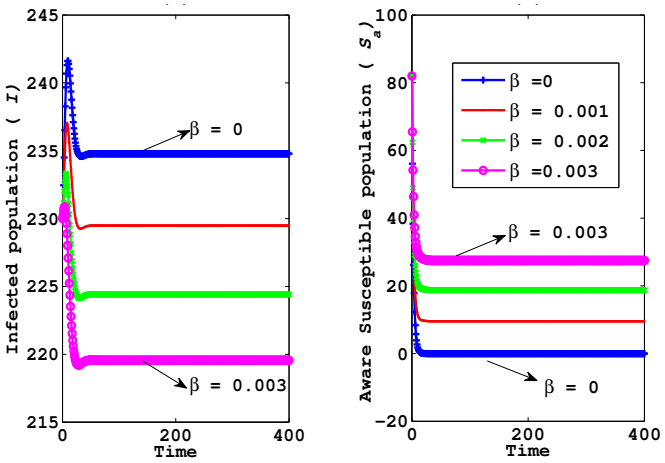


Figure 3. Global stability of (I^*, M^*) in IM-plane.

We also note that all conditions of Theorems 2 and 3 are satisfied. This implies that E_1 is locally as well as globally asymptotically stable for the set of values of parameters in equation (24).

In Fig. 2, we considered the five different initial values of the infected and aware susceptible populations. All trajectories starting from different initial values approach to $(I^*, {S_a}^*)$. This point is independent of the initial status. This shows that $(I^*, {S_a}^*)$ is globally asymptotically stable in the IS_a-plane. All the details related to initial values are shown in the legend of Fig. 2. Similarly in Fig. 3, we have shown that trajectories initi-



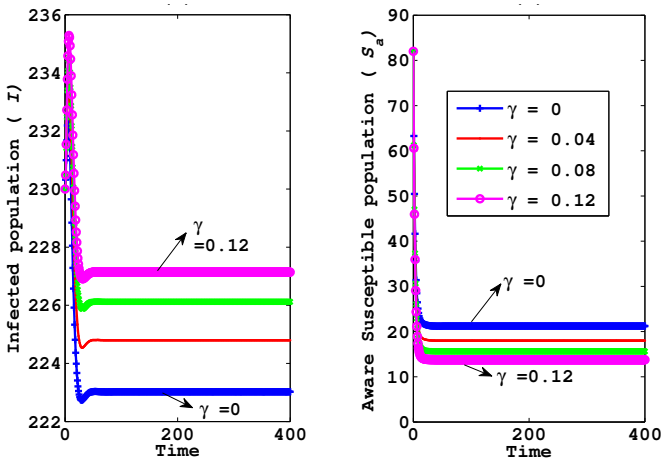
(a) (b)

Figure 4. Effect of β on infected population I and aware susceptible population S_a.

ating from different initial points converge to the same equilibrium point (I^*, M^*). This shows that (I^*, M^*) is globally asymptotically stable in the IM-plane. When awareness programs are delivered to susceptible population then too the stabilization of infected population takes place and it gets restricted to an equilibrium point which is again independent of the initial level of awareness or infection. All the details related to initial values are shown in the legend. This implies that for the given set of parameters the disease will restrict itself to a given endemic zone/population, irrespective of parameters like, the magnitude of infected population, aware susceptible population, total population and awareness programs.

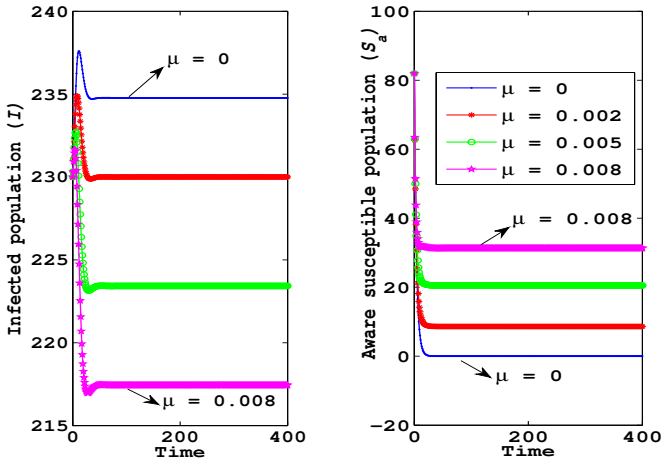
In Fig. 4, we have shown the effect of information dissemination rate (β) on (a) infected population (I) and (b) aware susceptible population (S_a), respectively. From Fig. 4 we observe that when there is no spread of awareness programs among susceptibles i.e. dissemination rate (β) is zero, then the number of infected population is high and the aware susceptible is zero. Further the infection decreases with increase in information dissemination rate (β). This shows that by increasing the dissemination rate of the awareness program, the number of infected individuals decreases but the number of aware susceptibles increases. Some of the susceptible individuals can keep themselves isolated and will not be infected.

Fig. 5 represent the effect of γ on (a) infected population and (b) aware susceptible population. We note from Fig. 5a that infected population increases as we increase γ, limitations on awareness program and is lowest when there is no limitation on dissemination of awareness ($\gamma = 0$). The transient kink settling down at a high infection level in former case can be explained by the behavioural slackness with time as information spreads. Whereas the kink when ($\gamma = 0$) can be explained by immunity. In Fig. 5b, we observe that the aware susceptible population decreases with an increase in γ (limitation to the dissemination of awareness). When there is no limitation to dissemination of awareness,



(a) (b)

Figure 5. Effect of γ on infected population I and aware susceptible population S_a.



(a) (b)

Figure 6. Effect of μ on infected population I and aware susceptible population S_a.

people are maximally aware but as limitations increase the aware susceptible population declines as more people remain ignorant of the disease.

In Fig. 6, we have shown the effect of implementation rate of awareness program on (a) infected population and (b) aware suceptible population. From Fig. 6 we can see that when there is no awareness program run by media then aware susceptible population is zero and infection is high. As we increase the implementation rate of awareness program (μ), aware susceptible population is increasing and infection is decreasing very rapidly. This implies that awareness programs may reduce the susceptibility to infection but will

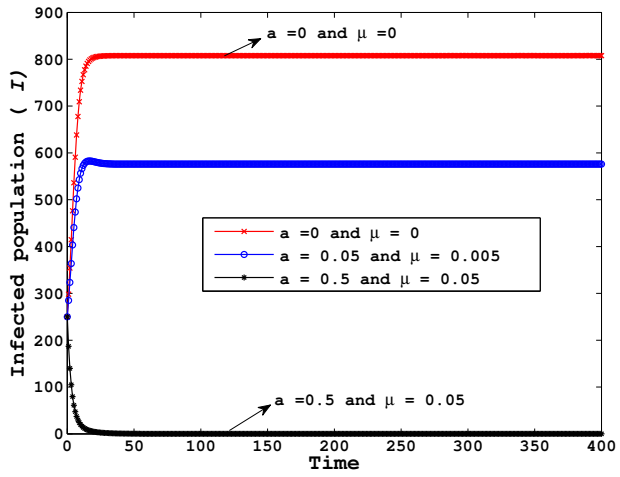


Figure 7. Effect of treatment rate a and μ on infected population.

not eradicate the infection. The transient increase in infectives at high infection level (as observed by the kink in graph) may be due to continued infection till adequate life style modification is made to prevent or overcome the infection. At a lower level of infection the downward kink may be explained by noncompliance or resistance to making long term life style modification inspite of information. When $\mu = 0$, then the same can be explained by virtue of immunity in the population.

In Fig. 7, we have shown the effect of treatment rate a and awareness program implementation rate μ on infected population simultaneously. We observe that when there is neither treatment nor an awareness program available then the rate of infection increases and gets saturated at high level. Further, if we increase the treatment and awareness then we observe the decrease in infection. And finally, when the treatment and awareness program both are high we observe the decrease in infected population and it settles down to zero level. This shows that the disease can be eradicated if treatment availability is high and if we are able to provide enough awareness to the susceptible population about the disease. Thus highlighting the fact that not only treatment but awareness too is a prerequisite of disease eradication.

6 Conclusions

In this paper, we introduced a mathematical model to study the effect of awareness programs (run by media) and treatment on infectious diseases. The global dynamics of this model has been studied. We have shown that there exists only two equilibrium points: the disease-free equilibrium $E_0(0, 0, A/\delta_0, 0)$ i.e. total elimination of infection (as $I = 0$) and the endemic equilibrium $E_1(I^*, {S_a}^*, N^*, M^*)$ i.e. disease will persist. The DFE is locally asymptotically stable for reproductive number $R_0 < 1$ and the endemic

equilibrium exists for $R_0 > 1$ and is globally asymptotically stable under the conditions stated in Theorem 3. When optimal treatment and awareness is provided then the former refers to total eradication of the infectious disease from the population whereas the later refers to the case when disease is localized to an endemic zone. We have also carried out numerical simulations to validate the analytical results. We have shown that the infected population decreases as we increase the information dissemination rate (β) as some of the susceptibles keep themselves isolated and did not get infected. Thus, the aware susceptibles increase with increase in dissemination rate (β). Further, we have shown that if awareness programs are not available in society then the infection is very high and if we introduce awareness programs run by media into society infection decreases and this can be further reduce by treatment. We have also shown that when there is enough awareness among the susceptibles and enough treatment is available then disease can be eradicated completely. But if either treatment or awareness is lacking then the disease can not be eradicated.

It may be noted that small pox is the only disease that has been totally eradicated but the eradication has taken place by virtue of vaccination and awareness since treatment of small pox was not available. Further diseases like Polio have been brought under control and largely eradicated from most of the countries in the world. This disease too can not be treated but vaccination and awareness regarding the disease has led to its near eradication. There are no known diseases yet which have been eradicated by only treatment. Thus sufficient emphasis must be given to awareness programs to ensure total eradication of treatable emerging infectious diseases.

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