

# Modeling the role of media awareness programs on the spread of HIV/AIDS

Agraj Tripathi<sup>1\*</sup>, Ram Naresh<sup>2</sup>

<sup>1</sup> Department of Basic Science and Humanities, P S Institute of Technology, Kanpur, India

<sup>2</sup> School of Basic and Applied Sciences, Department of Mathematics, Harcourt Butler Technical University, Kanpur, India

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**Abstract.** Media plays a very crucial role in creating awareness to reduce the spread of any infectious disease in human population. It is a very important factor to change the behavior of people towards the epidemic. Awareness programs through media in the community change the pattern of spread of disease and reduce the infection rate. In this paper, we have proposed a nonlinear mathematical model to assess the impact of media campaigns on reducing the spread of HIV/AIDS. In the model, we have divided the total population into four sub-classes namely, susceptibles, HIV infecteds (assumed to be infectious), aware susceptibles and AIDS patients. A variable  $M$  present in the model represents the cumulative density of media awareness programs. The important mathematical features of the model including determination of equilibrium points and their stability are thoroughly investigated using stability theory of differential equations. The endemic equilibrium is found to be locally asymptotically stable as well as nonlinearly stable under certain conditions. The model analysis shows that the spread of HIV/AIDS can be significantly reduced when the media awareness programs are executed successfully targeting the susceptible population. Using Pontryagin's maximum principle optimal control theory was used on the system of differential equations to achieve the goal of minimizing the infected population and slow down the epidemic outbreak. Numerical simulations have also been performed to support the analytical findings.

**Keywords:** HIV/AIDS, media, reproduction number, equilibria, stability

## 1 Introduction

The Human Immunodeficiency Virus (HIV) infection which can cause Acquired Immunodeficiency Syndrome (AIDS), is one of the world's most serious cause of mortality. It has shown a high degree of prevalence in populations all over the world and has spread faster with more catastrophic long-term effects than any other disease. The advancement on biomedical front has only resulted in increasing the survival period of infected persons. Alternatively, the behavior change can be the effective prevention strategy to significantly curtail the further escalation of the disease. Unlike to other curable infectious diseases and the social taboos attached with HIV/AIDS in a population, the vast majority of people may opt for behavior change. The awareness programs through mass media campaigns can be immensely helpful to motivate the susceptible people modifying their behavior so that they may refrain from risky sexual behavior, lowering the prevalence of the disease.

Media coverage is an important factor regarding the transmission of infectious diseases. Since the epidemic prevalence strongly depends on the individual's social behavior in a population, the behavior change can alone contribute significantly to reducing the size of epidemic. The campaigns through mass media can induce to modify the human behavior towards disease, as they may create awareness among various high risk groups which in turn help in limiting the further spreading of disease. Many researchers have developed mathematical models for curable infectious diseases keeping this aspect in view<sup>[4, 5, 12-14, 16, 23-28, 30]</sup>. In some of the studies on the impact of awareness disseminated by mass media on epidemic outbreaks, it is assumed that media

\* Corresponding author. E-mail address: agrajtripathi@gmail.com

driven awareness programs help in modifying the contact rate between susceptibles and infectives<sup>[8, 9, 14, 16, 21]</sup>. In particular, Liu et al.<sup>[14]</sup> focused on the psychological impact on epidemic outbreaks. They have considered that an increase in infection level reduces the effective contacts but the factors of mandatory quarantine and isolation were not taken into account. Liu and Cui<sup>[16]</sup> studied the impact of media coverage on the dynamics of infectious diseases by considering the contact rate reduced proportionally to the Holling type-II functional response of infectives. Cui et al.<sup>[5]</sup> proposed a three dimensional compartmental model to investigate the impact of media coverage on the spread and control of infectious diseases (such as SARS) in a given region/area. They suggested that when  $R_0 > 1$  and the media impact is stronger enough, the model exhibits multiple positive equilibria which poses challenge to the prediction and control of the outbreaks of infectious diseases. Cui et al.<sup>[6]</sup> developed a model to explore the impact of media coverage on the control of spreading of re-emerging infectious diseases in a given population. They observed that the media coverage itself is not a determined fact to eradicate the infection of the diseases to a certain extent. Therefore, media coverage is critical for educating people in understanding the possibility of being infected by the disease.

In most of the above studies, the cumulative density of awareness programs is assumed to be constant but in real situation it is varying and should be considered in the modeling process. Misra et al.<sup>[17]</sup> proposed a model for the effect of awareness programs on the spread of infectious diseases assuming that the cumulative density of media awareness programs depends on the number of infectives present in the population and shown that the disease spread can be controlled by conducting awareness programs. Misra et al.<sup>[18]</sup> formulated a mathematical model to study the effect of awareness campaigns by media on the outbreak of an epidemic. They also identified an optimal implementation rate of awareness campaigns so that disease can be controlled with minimal possible expenditure on awareness campaigns, using optimal control theory. Dubey et al.<sup>[8]</sup> proposed a model to show the impact of awareness programs as well as treatment on an SIR model and observed that in absence of awareness an infection can not be eliminated in spite of adequate treatment.

It is noted here that almost all the above mathematical models, where the role of media campaigns have been incorporated, are for infectious diseases in which recovery is possible. But in case of HIV infection till date permanent recovery is not possible and also no vaccine is available. The mathematical frameworks for HIV/AIDS initially developed by Anderson et al.<sup>[1, 2]</sup> paved the way for further studies with specific issues and intervention strategies like contact tracing, medical screening of infectives, safe sexual interaction using condoms, quarantine, treatment, vaccination etc. incorporated in models for HIV/AIDS spread<sup>[3, 10, 15, 17-23, 29]</sup>. In particular, Tripathi et al.<sup>[31]</sup> proposed a model for HIV infection to study the effect of screening of unaware infectives in a homogeneous population and observed that the endemicity of the infection is reduced when people after becoming aware of their infection do not take part in sexual interaction. Naresh et al.<sup>[20]</sup> developed a model to study the effect of vaccination on the spread of HIV/AIDS in a homogeneously mixing population of variable size structure and found that if the vaccine efficacy is not high enough no amount of vaccination could lead to HIV eradication. These studies do not take into account the impact of media awareness as a preventive measure to stop spreading the HIV/AIDS.

In the absence of effective treatment and vaccination for HIV/AIDS, a large number of media driven awareness programs are required to be implemented to make people aware about the disease and its contributing factors. The aware people then may adopt preventive practices such as social distancing, avoidance of unsafe sexual interaction, insisting on HIV free blood samples etc. and try to minimize their chances of being infected<sup>[6, 11, 21, 22]</sup>. Del Valle et al.<sup>[7]</sup> studied the effects of education, vaccination and treatment on HIV transmission in a active homosexual population and suggested the awareness programs as an efficient option for reducing the disease spread. In a study by Rahman and Rahman<sup>[24]</sup>, it is suggested that education and media have a huge impact in preventing the spread of HIV/AIDS among married couples in Bangladesh. These studies indicate that the awareness through media campaigns can be substantially effective and helpful to curb the further spreading of disease in the human population.

Lack of knowledge, misunderstanding and social factors are the main reasons behind discrimination towards people living with HIV/AIDS (PLWHA) which in effect, resulted in accelerated growth of HIV epidemic. One of the most important element of the fight against AIDS is the prevention of new HIV infections. Thus, by reducing the contacts, the generation of new infections can be slowed down. For this, effective media campaigns and education are required to be implemented to make people aware of the disease so that they may opt

to avoid contacts with infective individuals. Media could act as a channel for communication and discussion and be instrumental in breaking the silence that envelops the disease as well as encouraging positive healthy behavior. If the media were to effectively live up to its role, then the current situation which sees fear lying at the heart of stigma and discrimination and underpinned by ignorance could be addressed and reversed. A very little attention has been paid on this aspect, while this could be one of the efficient and effective way to reduce the HIV infection in the absence of permanent cure.

In view of the above, in this paper, our objective is to model the control of HIV infection by implementing awareness programs in the community by media. Thus, a nonlinear mathematical model is proposed to study the impact of media awareness programs on the transmission of HIV in a population with constant inflow of susceptibles. The role of media is incorporated explicitly by assuming the cumulative density of awareness programs as a dynamical variable whose growth depends on the size of HIV infected individuals. A numerical study is also carried out to support the analytical results of the model system.

The paper is organized as follows, In Section 2, the model framework is put in place and it enables us to derive the model equations and also provide the region of biologically feasible solutions. Section 3 providing equilibrium existence together with proof (Section 3.1). The stability (local and nonlinear) analysis of the steady state is provided in Section 4. In Section 5, optimal control strategy is developed followed by numerical simulation in Section 6. Conclusions are given in Section 7.

## 2 Mathematical model

For model building, we have subdivided the total population  $N(t)$  at time  $t$ , with constant immigration rate  $A$ , into four compartments namely, susceptibles  $X(t)$ , HIV infectives  $Y_H(t)$ , aware susceptibles  $X_M(t)$  and AIDS population  $Y_A(t)$ . The variable  $M(t)$  represents the cumulative density of media awareness programs.

The growth rate of cumulative density of media programs is assumed to be proportional to the number of HIV infectives and AIDS population. The susceptible individuals  $X(t)$  become HIV infected upon contacts with HIV infectives at a rate  $\beta$ . It is assumed that anti-HIV treatment is not available in the community and therefore, all the HIV infectives are bound to develop full blown AIDS with a rate  $\delta$ . The susceptibles induced by media programs form a separate class and do not contact with HIV infectives and hence join the aware susceptible class  $X_M(t)$ , at a rate  $\lambda$ . Some of the aware susceptibles may loose awareness with time and become again susceptible with a rate  $\lambda_0$ .

Thus, the dynamics of the disease is assumed to be governed by the following system of nonlinear differential equations,

$$\frac{dX(t)}{dt} = A - \frac{\beta XY_H}{N} - dX - \lambda XM + \lambda_0 X_M, \quad (1)$$

$$\frac{dY_H(t)}{dt} = \frac{\beta XY_H}{N} - \delta Y_H - dY_H, \quad (2)$$

$$\frac{dX_M(t)}{dt} = \lambda XM - \lambda_0 X_M - dX_M, \quad (3)$$

$$\frac{dY_A(t)}{dt} = \delta Y_H - \alpha Y_A - dY_A, \quad (4)$$

$$\frac{dM(t)}{dt} = \mu(Y_H + Y_A) - \mu_0 M, \quad (5)$$

where  $d$  is the natural mortality rate constant,  $\alpha$  is disease-induced death rate constant. The constant  $\mu$  represents the rate by which awareness programs are being implemented and  $\mu_0$  represents the depletion rate of these programs due to ineffectiveness, social problems in the population, etc. It is assumed that all the dependent variables and parameters of the model system (1) are non-negative. Since  $N(t) = X(t) + Y_H(t) + X_M(t) + Y_A(t)$ , the above model system (1)-(5) can be rewritten as follows,

$$\frac{dN(t)}{dt} = A - dN - \alpha Y_A, \quad (6)$$

$$\frac{dY_H(t)}{dt} = \frac{\beta(N - Y_H - X_M - Y_A)Y_H}{N} - (\delta + d)Y_H, \quad (7)$$

$$\frac{dX_M(t)}{dt} = \lambda(N - Y_H - X_M - Y_A)M - (\lambda_0 + d)X_M, \quad (8)$$

$$\frac{dY_A(t)}{dt} = \delta Y_H - (\alpha + d)Y_A, \quad (9)$$

$$\frac{dM(t)}{dt} = \mu(Y_H + Y_A) - \mu_0 M, \quad (10)$$

continuity of the right hand side of the system of equations and its derivative imply that the model is well posed for  $N > 0$  and  $\beta > (\delta + d)$ . We define the threshold,  $\mathfrak{R}_0 = \frac{\beta}{\delta + d}$  as the basic reproductive number. The basic reproductive number defines the average number of new infections produced by one primary infected individual in a wholly susceptible population<sup>[2]</sup>. It is also noted that in the absence of infection the population size approaches the steady state  $\frac{A}{d}$  and in that case the role of media towards controlling the spread of disease is not required.

**Lemma 2.1.** Let  $(N(t), Y_H(t), X_M(t), Y_A(t), M(t))$  be the solution of model system (6)-(10), with initial conditions  $(N(0) > 0, Y_H(0) > 0, X_M(0) > 0, Y_A(0) > 0, M(0) \geq 0)$  and the compact set,

$$\Phi = ((N, Y_H, X_M, Y_A, M) \in R_+^5, 0 \leq (Y_H + X_M + Y_A) < N \leq \frac{A}{d}, 0 \leq M(t) \leq \frac{\mu A}{\mu_0 d}). \quad (11)$$

Under the flow described by model system (6)-(10),  $\Phi$  is a positively invariant set which attracts all solutions in  $R_+^5$ .

## 2.1 Positivity of solutions

In this section, we prove that all solutions of the system (6)-(10) with positive initial data will remain positive for all times  $t > 0$

**Lemma 2.2.** For initial data for all  $t$ , the solution  $(N(t), Y_H(t), X_M(t), Y_A(t), M(t))$  of the model remain positive for all time  $t > 0$ .

**Proof.** From Eq. (6), we have,

$$\begin{aligned} \frac{dN(t)}{dt} &= A - dN - \alpha Y_A, \\ \frac{dN(t)}{dt} &> -dN - \alpha Y_A, \\ \frac{dN(t)}{dt} &> -dN - \alpha N. \end{aligned}$$

From which we get,  $N(t) \geq c_1 e^{-(d+\alpha)t} > 0$ , where  $c_1$  is a constant of integration. A similar reasoning on the remaining equations shows that they are always positive for  $t > 0$ .

## 3 Existence of equilibria

The model system (6)-(10) has two non-negative equilibria. These equilibria are listed below,

(i) Disease-free equilibrium  $E_0(\frac{A}{d}, 0, 0, 0, 0)$

(ii) Endemic equilibrium  $E^*(N^*, Y_H^*, X_M^*, Y_A^*, M^*)$

The existence of  $E_0$  is obvious. In the following, we prove the existence of  $E^*$ .

### 3.1 Existence of $E^*(N^*, Y_H^*, X_M^*, Y_A^*, M^*)$

Here  $N^*$ ,  $Y_H^*$ ,  $X_M^*$ ,  $Y_A^*$  and  $M^*$  are positive solutions of the following system of algebraic equations,

$$\begin{aligned} A - dN - \alpha Y_A &= 0, \\ \frac{\beta(N - Y_H - X_M - Y_A)Y_H}{N} - (\delta + d)Y_H &= 0, \\ \lambda(N - Y_H - X_M - Y_A)M - (\lambda_0 + d)X_M &= 0, \\ \delta Y_H - (\alpha + d)Y_A &= 0, \\ \mu(Y_H + Y_A) - \mu_0 M &= 0. \end{aligned} \quad (12)$$

On solving simultaneous algebraic equations (12) we obtain,

$$\begin{aligned} X_M &= \frac{[\beta - (\delta + d)]N - \beta(\frac{\alpha + \delta + d}{\delta})(\frac{A - dN}{\alpha})}{\beta} = f(N), \\ Y_H &= (\frac{A - dN}{\alpha})(\frac{\alpha + d}{\delta}), \\ Y_A &= (\frac{A - dN}{\alpha}), \\ M &= (\frac{A - dN}{\alpha\mu_0})(\frac{\mu(\alpha + \delta + d)}{\delta}) = g(N). \end{aligned} \quad (13)$$

Substituting these values in third equation of algebraic Eq. (12) we get,

$$F(N) = \lambda[N - f(N) - (\frac{A - dN}{\alpha})(\frac{\alpha + \delta + d}{\delta})]g(N) - (\lambda_0 + d)f(N). \quad (14)$$

To show the existence of  $E^*$ , it would be sufficient to show that the eq. (14) has one and only one positive root between  $\frac{A}{\alpha + d}$  and  $\frac{A}{d}$ . To prove this, from eq. (14) we have,

$$F(\frac{A}{\alpha + d}) = \frac{A(\delta + d)}{(\alpha + d)\beta} [\frac{A\lambda}{(\alpha + d)\mu_0} (\frac{\mu(\alpha + \delta + d)}{\delta})] + A(\lambda_0 + d)[\frac{(\delta + d)}{(\alpha + d)\beta} + \frac{1}{\delta}] > 0. \quad (15)$$

$$F(\frac{A}{d}) = -(1 - \frac{1}{R_0})(\lambda_0 + d)\frac{A}{d} < 0. \quad (16)$$

Also,

$$F'(N) = \lambda(\frac{\delta + d}{\beta})(\frac{\mu(\alpha + \delta + d)}{\delta\alpha\mu_0})(A - 2dN). \quad (17)$$

If  $N > \frac{A}{2d}$ , then  $F'(N) < 0$ . It is easy to note by the intermediate property of calculus that if  $F'(N) < 0$ , then Eq. (14) has only one root (say  $N^*$ ) between  $\frac{A}{\alpha + d}$  and  $\frac{A}{d}$ . Using  $N^*$ , the values of  $Y_H^*$ ,  $X_M^*$ ,  $Y_A^*$  and  $M^*$  can be found easily.

### 3.2 Variation of HIV infectives with $\mu$ and $\mu_0$

From the Section 3.1 we have,

$$f_1 = \delta Y_H - (\alpha + d)(\frac{A - dN}{\alpha}) = 0. \quad (18)$$

$$f_2 = \frac{\mu\lambda}{\mu_0}[N - Y_H - f(N) - (\frac{A - dN}{\alpha})][Y_H + (\frac{A - dN}{\alpha})] - (\lambda_0 + d)f(N) = 0. \quad (19)$$

Therefore,

$$\frac{dY_H}{d\mu} = -\frac{\frac{\partial f_1}{\partial \mu} \frac{\partial f_2}{\partial N} - \frac{\partial f_1}{\partial N} \frac{\partial f_2}{\partial \mu}}{\frac{\partial f_1}{\partial Y_H} \frac{\partial f_2}{\partial N} - \frac{\partial f_1}{\partial N} \frac{\partial f_2}{\partial Y_H}}. \quad (20)$$

Now,

$$\frac{\partial f_1}{\partial \mu} \frac{\partial f_2}{\partial N} - \frac{\partial f_1}{\partial N} \frac{\partial f_2}{\partial \mu} = -\frac{d(\alpha + d)\lambda}{\alpha\mu_0} pq < 0, \quad (21)$$

and

$$\frac{\partial f_1}{\partial Y_H} \frac{\partial f_2}{\partial N} - \frac{\partial f_1}{\partial N} \frac{\partial f_2}{\partial Y_H} = -(\lambda_0 + d)\delta r - \frac{\lambda\mu d}{\mu_0\alpha}(\alpha + \delta + d)p < 0. \quad (22)$$

Here,  $p = [N - Y_H - f(N) - (\frac{A-dN}{\alpha})] > 0$ ,  $q = [Y_H + (\frac{A-dN}{\alpha})] > 0$  and  $r = [\frac{R_0-1}{R_0} + \frac{(\alpha+\delta+d)d}{\delta\alpha}] > 0$ .

On putting the values from Eqs. (21) and (22) in Eq. (20), we get  $\frac{dY_H}{d\mu} < 0$ . This shows that the number of HIV infectives decrease with increase in the implementation rate of awareness programs  $\mu$ . In a similar way we can also check that  $\frac{dY_H}{d\mu_0} > 0$ . This implies that the number of HIV infectives increase as the depletion rate of awareness programs ( $\mu_0$ ) increases.

## 4 Stability analysis

In this section, the stability results of the equilibrium points are stated.

### 4.1 Local stability of equilibria

**Theorem 4.1.** The disease-free equilibrium  $E_0$  is locally asymptotically stable if  $\mathfrak{R}_0 < 1$  and for  $\mathfrak{R}_0 > 1$  it is unstable. The endemic equilibrium  $E^*$  is locally asymptotically stable if following inequalities are satisfied,

$$(1 - \frac{1}{\mathfrak{R}_0})^2 Y_H^* < \frac{16dN^*}{9\beta}. \quad (23)$$

$$\frac{\lambda M^*}{(\lambda M^* + \lambda_0 + d)} < \frac{4}{3} \min. [\frac{dN^*}{\beta Y_H^*}, \frac{(\alpha + d)}{\delta}]. \quad (24)$$

$$\frac{\alpha^2}{d(\alpha + d)} < \frac{16\beta Y_H^*}{9\delta N^*} \quad (25)$$

$$\frac{9\lambda(N^* - Y_H^* - X_M^* - Y_A^*)^2}{4M^*(\lambda M^* + \lambda_0 + d)} < \frac{16\mu_0^2}{9\mu^2} \min. [\frac{(\alpha + d)}{\delta}, 1]. \quad (26)$$

For proof see Appendix A.

### 4.2 Nonlinear stability of endemic equilibrium

In order to establish nonlinear stability of  $E^*$ , we need the region of attraction as stated in Lemma 2.1 of Section 2.

**Theorem 4.2.** The equilibrium  $E^*$  is nonlinearly stable if the following inequalities are satisfied in the region  $\phi$ ,

$$(\frac{A}{d})^2 < \frac{16N^{*2}}{9\beta}. \quad (27)$$

$$\frac{\lambda M^*}{(\lambda M^* + \lambda_0 + d)} < \frac{4}{3} \min. [\frac{dN^*}{\beta}, \frac{(\alpha + d)}{\delta}]. \quad (28)$$

$$\frac{\alpha^2}{d(\alpha + d)} < \frac{16\beta}{9\delta N^*}. \quad (29)$$

$$\frac{9\lambda}{4M^*(\lambda M^* + \lambda_0 + d)} (\frac{A}{d})^2 < \frac{16\mu_0^2}{9\mu^2} \min. [\frac{(\alpha + d)}{\delta}, 1]. \quad (30)$$

For proof see Appendix B.

**Remarks:**

- (i) When  $\mathfrak{R}_0 = 1$ , then condition (23) is satisfied easily.
- (ii) When  $\lambda \rightarrow 0$  then conditions (24), (26), (28) and (30) are satisfied automatically.
- (iii) When contact rate  $\beta \rightarrow 0$  then condition (27) is satisfied.

**5 Optimal control system**

In this section, we consider the optimal control of the model system (6)-(10). In this we extend the initial model system to include two controls representing as function of time. The objective functional is defined as follows<sup>[29]</sup>,

$$J(\lambda_0, \mu_0) = \int_0^T [A_1 Y_H(t) + A_2 X_M(t) + A_3 \frac{\lambda_0^2}{2} + A_4 \frac{\mu_0^2}{2}] dt. \quad (31)$$

Here  $\lambda_0$ , the rate by which aware susceptibles again become susceptible, and depletion rate of the media program  $\mu_0$  are both functions of time. Our goal is to find the optimal control  $\lambda_0$  and  $\mu_0$  in order to minimize the objective functional given by  $J(\lambda_0, \mu_0)$ .

$$\min. J(\lambda_0, \mu_0) = J(\lambda_0^*, \mu_0^*), \quad (32)$$

where  $(\lambda_0, \mu_0) \in U$  and  $A_i (i = 1, 2, 3, 4)$  are the positive balancing constants. The control function  $\lambda_0$  represents the depletion of aware susceptibles, while  $\mu_0$  is the corresponding depletion rate of media. The control set  $U$  is,

$$U = [(\lambda_0, \mu_0) : 0 \leq a_1 \leq \lambda(t) \leq b_1 < 1, 0 \leq a_2 \leq \mu(t) \leq b_2 < 1, 0 \leq t \leq T], \quad (33)$$

$\lambda_0$  and  $\mu_0$  are Lebesgue measurable.

This system satisfies standard condition for the existence of an optimal control and now we derive the necessary conditions, using Pontryagin's maximum principle.

The Hamiltonian of the system is

$$\begin{aligned} H = & A_1 Y_H(t) + A_2 X_M(t) + A_3 \frac{\lambda_0^2(t)}{2} + A_4 \frac{\mu_0^2(t)}{2} + \lambda_1 [A - dN - \alpha Y_A] \\ & + \lambda_2 \left[ \frac{\beta(N - Y - X_M - Y_A)Y_H}{N} - (\delta + d)Y_H \right] \\ & + \lambda_3 [\lambda(N - Y - X_M - Y_A)M - (\lambda_0 + d)X_M] \\ & + \lambda_4 [\delta Y_H - (\alpha + d)M] + \lambda_5 [\mu(Y_H + Y_A) - \mu_0 M], \end{aligned} \quad (34)$$

where  $\lambda_{0i}(t) (i = 1, 2, 3, 4, 5)$  are the adjoint variables. In order to determine the transversality conditions and the adjoint equations, we use the Hamiltonian. The adjoint system results from Pontryagin's maximum principle.

$$\frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial N}, \quad \frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial Y_H}, \quad \frac{d\lambda_3}{dt} = -\frac{\partial H}{\partial X_M}, \quad \frac{d\lambda_4}{dt} = -\frac{\partial H}{\partial Y_A}, \quad \frac{d\lambda_5}{dt} = -\frac{\partial H}{\partial M}$$

with  $\lambda_i(T) = 0$ , for  $(i = 1, 2, 3, 4, 5)$ . In order to obtain the characterization of the control,



$$\lambda'_1 = -\frac{\partial H}{\partial N} = -[\lambda_1 d + \lambda_2 \frac{\beta(Y_H + X_M + Y_A)Y_H}{N^2} + \lambda_3 \lambda M]. \quad (35)$$

$$\lambda'_2 = -\frac{\partial H}{\partial Y_H} = -[A_1 + \lambda_2 [\frac{\beta(N - Y_H - X_M - Y_A)}{N} - \frac{\beta Y_H}{N} - (\delta + d)] - \lambda_3 \lambda M + \lambda_4 \delta + \lambda_5 \mu]. \quad (36)$$

$$\lambda'_3 = -\frac{\partial H}{\partial X_M} = -[A_2 - \lambda_2 \frac{\beta Y_H}{N} - \lambda_3 (\lambda M + \lambda_0 + d)]. \quad (37)$$

$$\lambda'_4 = -\frac{\partial H}{\partial Y_A} = -[\lambda_1 \alpha - \lambda_2 [\frac{\beta Y_H}{N} + (\delta + d)] - \lambda_3 \lambda M - \lambda_4 (\alpha + d) + \lambda_5 \mu]. \quad (38)$$

$$\lambda'_5 = -\frac{\partial H}{\partial M} = -[\lambda_3 \lambda (N - Y_H - X_M - Y_A) - \lambda_5 \mu_0]. \quad (39)$$

The optimality conditions on the interior of the control set of an optimal control pair  $\lambda_0^*$ ,  $\mu_0^*$  are  $\frac{\partial H}{\partial \lambda_0} = 0$ ,  $\frac{\partial H}{\partial \mu_0} = 0$ .

$$0 = \frac{\partial H}{\partial \lambda_0} = A_3 \lambda_0 - \lambda_3 X_M. \quad (40)$$

$$0 = \frac{\partial H}{\partial \mu_0} = A_4 \mu_0 - \lambda_5 M. \quad (41)$$

Hence using our control bounds,

$$\lambda_0^* = \min.[b_1, \max.(a_1, \lambda_3 \frac{X_M}{A_3})], \mu_0^* = \min.[b_2, \max.(a_2, \lambda_5 \frac{M}{A_4})].$$

## 6 Numerical simulation

We give here the numerical simulation of the model system (6)-(10) to show the existence of equilibrium values and to check the feasibility of stability conditions.

For this, we integrate the system (6)-(10) by fourth order Runge-Kutta method using MATLAB with following set of parameter values.

$$A = 1000, d = 0.02, \mu = 0.0005, \mu_0 = 0.06, \alpha = 1, \beta = 1.33, \delta = 0.1, \lambda_0 = 0.03, \lambda = 0.002$$

The equilibrium values of endemic equilibrium are computed as

$$N^* = 12692.12, Y_H^* = 7610.81, X_M^* = 3190.00, Y_A^* = 746.16, M^* = 69.64$$

The results of numerical simulation are displayed graphically in Figs. 1-6. In Figs. 1 and 2, the infective population and aware susceptible population respectively are plotted against the media class. We see from these figures that for any initial start, the solution curves tend to the equilibrium. Hence, we infer that the system (6)-(10) may be globally stable about this endemic equilibrium point  $E^*$ . In Fig. 3, the variation of aware susceptible population with time is shown for different values of interaction rate  $\lambda$  of susceptibles with media. It is seen that as this interaction rate increases, the aware susceptible population increases and consequently HIV infected population declines (see Fig. 4). It is also observed that if the interaction rate of susceptibles with media is negligibly small or tends to zero, the increase in the number of HIV infective population is quite significant due to rapid decline in the number of aware susceptible population. Fig. 5 depicts the variation of HIV infective population for different values of implementation rate of awareness programs by media. From this figure, it is observed that the HIV infective population declines continuously on increasing the rate of implementation programs by media. Fig. 6 shows the effect of depletion rate of awareness programs by media wherein it is found that the HIV infective population increases with increase in the depletion rate of awareness programs. Thus, if the awareness programs by media are implemented fully in the community with minimal depletion, the decrease in the HIV infective population is more predominant.

## 7 Conclusions

In this paper, a nonlinear mathematical model is proposed and analyzed to study the role of media awareness programs in reducing the spread of HIV/AIDS in a community. The cumulative density of media aware-



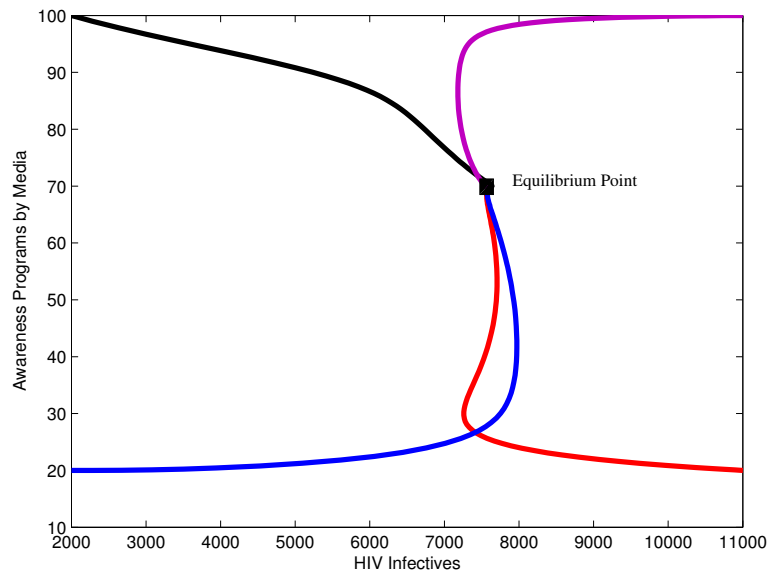


Fig. 1: Phase plot between  $Y_H^*$  and  $M^*$

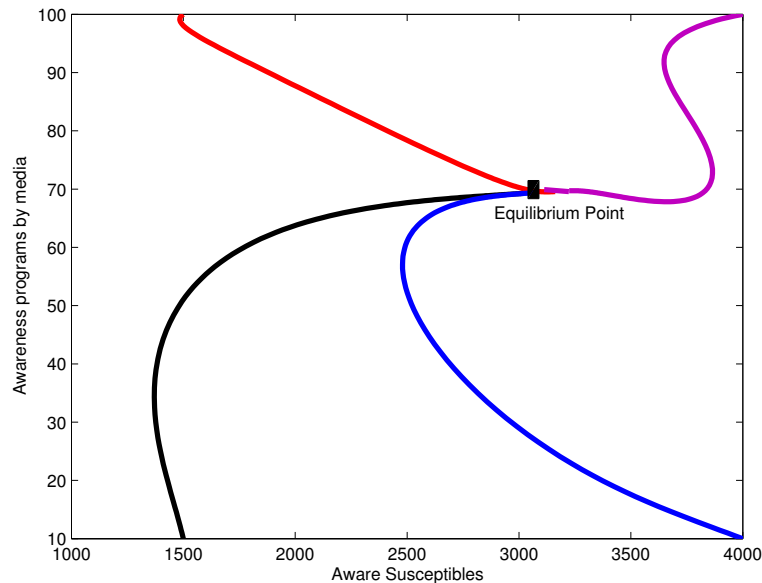


Fig. 2: Phase plot between  $X_M^*$  and  $M^*$

ness programs is incorporated in the modeling process as a dynamical variable and its growth is assumed to be dependent on HIV infected individuals. The proposed model has two equilibria namely, the disease-free equilibrium and the endemic equilibrium. The disease-free equilibrium is found to be locally asymptotically stable if the basic reproductive number  $\mathcal{R}_0 < 1$  and for  $\mathcal{R}_0 > 1$  it is unstable. The endemic equilibrium is locally as well as nonlinearly asymptotically stable under certain conditions.

We also studied the optimal control strategy for our model system via the Pontryagin's maximum principle. We considered time dependent controls to ensure the eradication of the disease in a finite time. Analysis of the model suggests that increasing the interaction rate of susceptibles with media programs results in increasing the aware susceptible population which, in turn, reduces the HIV infective population. Also increasing the implementation rate of media programs with minimal depletion further declines the HIV infective population. Thus, from the model analysis, it is observed that one of the effective way to substantially lower the prevalence

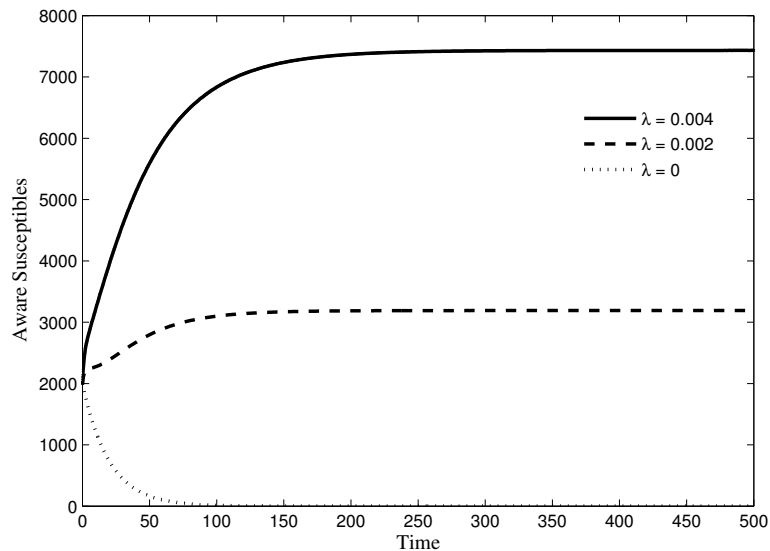


Fig. 3: Variation of aware susceptibles for different values of  $\lambda$

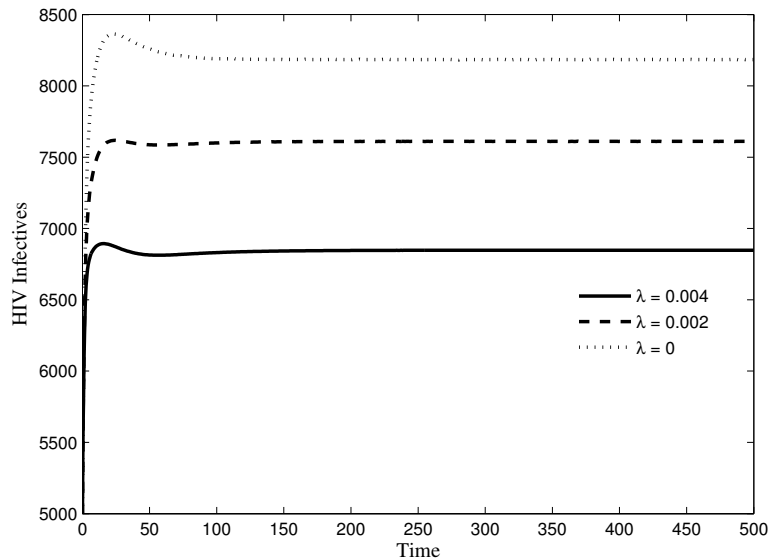


Fig. 4: Variation of HIV infectives for different values of  $\lambda$

level is to educate the population through media campaigns by making them aware of the consequences of HIV/AIDS spread and necessity of preventive measures against the infection. As a consequence of the impact of media campaigns and resulting behavior change after being aware of disease implications, the prevalence of disease can be controlled by isolation of aware susceptibles from infectives.

Since this disease can not be treated therefore, awareness programs is the most effective way to eradicate the disease from the community. We observed from the model that on increasing the quantity of media campaigns the disease transmission rate amongst the susceptible declines. It can be speculated that if the population presents the positive attitude towards disease prevention measures and adopt behavior change as a result of media awareness programs, the spread of HIV/AIDS can be significantly reduced in the population. Therefore, the media awareness programs must reach the community at all social levels to keep the prevalence level of HIV/AIDS under control.

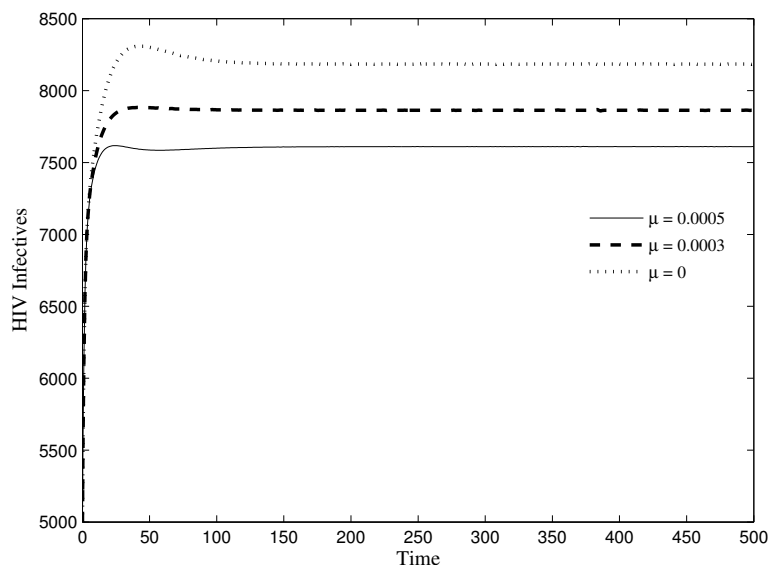


Fig. 5: Variation of HIV infectives for different values of  $\mu$

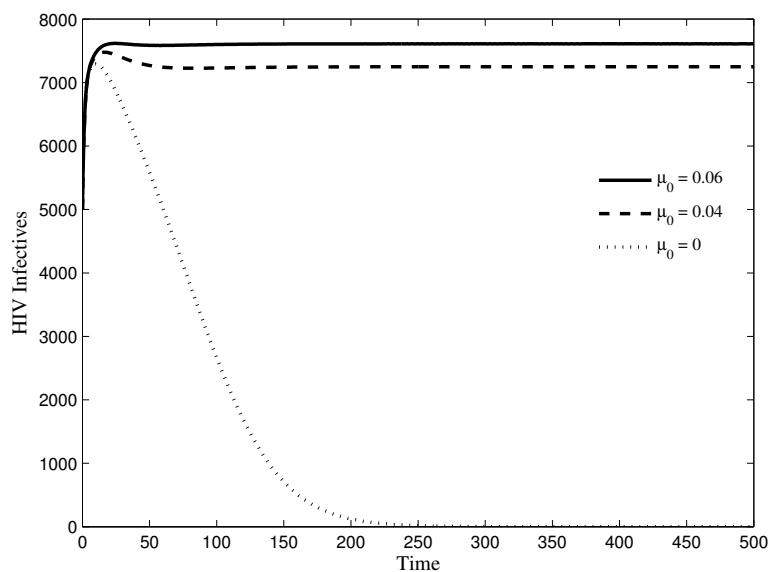


Fig. 6: Variation of HIV infectives for different values of  $\mu_0$

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## A Appendix

### Proof of Theorem 4.1.

The local stability of disease-free equilibrium can be checked easily by computing Jacobian matrix evaluated at  $E_0$ .

It is found that all the eigenvalues of the Jacobian matrix are negative if  $\beta < (\delta + d)$  i.e.  $\mathfrak{R}_0 < 1$ , then disease-free equilibrium  $E_0$  is locally asymptotically stable and will be unstable when  $\beta > (\delta + d)$  i.e.  $\mathfrak{R}_0 > 1$ .

Now to establish the local stability of the endemic equilibrium  $E^*$ , we linearize the system using small perturbations  $n, y_h, x_m, y_a$  and  $m$  as follows,

$$N = N^* + n, Y_H = Y_H^* + y_h, X_M = X_M^* + x_m, Y_A = Y_A^* + y_a, M = M^* + m.$$

Let us consider the following positive definite function,

$$U = p_0 \frac{1}{2} n^2 + p_1 \frac{1}{2} y_h^2 + p_2 \frac{1}{2} x_m^2 + p_3 \frac{1}{2} y_a^2 + p_4 \frac{1}{2} m^2. \quad (42)$$

On differentiating  $U$  with respect to  $t$

$$\frac{dU}{dt} = p_0 n \frac{dn}{dt} + p_1 y_h \frac{dy_h}{dt} + p_2 x_m \frac{dx_m}{dt} + p_3 y_a \frac{dy_a}{dt} + p_4 m \frac{dm}{dt}, \quad (43)$$

where  $p_0, p_1, p_2, p_3$  and  $p_4$  are positive constants to be chosen appropriately.

Now using the linearized system of (6)-(10) and after some algebraic manipulations, we get

$$\begin{aligned} \frac{dU}{dt} = & -p_0 dn^2 - p_1 \frac{\beta Y_H^*}{N^*} y_h^2 - p_2 (\lambda M^* + \lambda_0 + d) x_m^2 \\ & - p_3 (\alpha + d) y_a^2 - p_4 \mu_0 m^2 - p_0 \alpha n y_a \\ & + (p_3 \delta - p_1 \frac{\beta Y_H^*}{N^*}) y_h y_a + \frac{[\beta - (\delta + d)] Y_H^* p_1}{N^*} n y_h \\ & - (p_1 \frac{\beta Y_H^*}{N^*} + p_2 \lambda M^*) y_h x_m + p_2 \lambda M^* n x_m \\ & + p_2 \lambda (N^* - Y_H^* - X_M^* - Y_A^*) x_m m - p_2 \lambda M^* x_m y_a \\ & + p_4 \mu y_h m + p_4 \mu y_a m \end{aligned} \quad (44)$$

Choosing,  $p_3 = p_1 \frac{\beta Y_H^*}{\delta N^*}$ ,  $p_2 = p_1 \frac{\beta Y_H^*}{\lambda M^* N^*}$  and  $p_0 = p_1 = 1$ , we get  $\frac{dU}{dt}$  to be negative definite and the desired conditions are obtained. Here,  $\frac{9\beta Y_H^* \lambda (N^* - X_M^* - Y_H^* - Y_A^*)^2}{4N^* M^* \mu_0 (\lambda M^* + \lambda_0 + d)} < p_4 < \frac{16\mu_0 \beta Y_H^*}{9\mu^2 N^*} \min. [\frac{\alpha + d}{\delta}, 1]$ .

### Proof of Theorem 4.2.

To prove this theorem, we consider the following positive definite function,

$$\begin{aligned} V = & \frac{1}{2} k_0 (N - N^*)^2 + k_1 (Y_H - Y_H^* - Y_H^* \ln \frac{Y_H}{Y_H^*}) + \frac{1}{2} k_2 (X_M - X_M^*)^2 \\ & + \frac{1}{2} k_3 (Y_A - Y_A^*)^2 + \frac{1}{2} k_4 (M - M^*)^2. \end{aligned} \quad (45)$$

Differentiating  $V$  with respect to  $t$ , we get

$$\begin{aligned} \frac{dV}{dt} = & k_0 (N - N^*) \frac{dN}{dt} + k_1 \frac{(Y_H - Y_H^*)}{Y_H} \frac{dY_H}{dt} + k_2 (X_M - X_M^*) \frac{dX_M}{dt} \\ & + k_3 (Y_A - Y_A^*) \frac{dY_A}{dt} + k_4 (M - M^*) \frac{dM}{dt}, \end{aligned} \quad (46)$$

where  $k_i (i = 0, 1, 2, 3)$  are positive constants, to be chosen appropriately.

After some algebraic manipulations,  $\frac{dV}{dt}$  can be written as

$$\begin{aligned} \frac{dV}{dt} = & -k_0 d (N - N^*)^2 - k_1 \frac{\beta}{N^*} (Y_H - Y_H^*)^2 - k_2 (\lambda M^* + \lambda_0 + d) (X_M - X_M^*)^2 \\ & - k_3 (\alpha + d) (Y_A - Y_A^*)^2 - k_4 \mu_0 (M - M^*)^2 - k_0 \alpha (N - N^*) (Y_A - Y_A^*) \\ & + (k_3 \delta - \frac{\beta k_1}{N^*}) (Y_H - Y_H^*) (Y_A - Y_A^*) + k_1 \frac{\beta (Y_H + X_M + Y_A)}{N N^*} (N - N^*) (Y_H - Y_H^*) \\ & - \frac{\beta k_1}{N^*} (Y_H - Y_H^*) (X_M - X_M^*) - k_2 \lambda M^* (Y_H - Y_H^*) (X_M - X_M^*) \\ & + k_2 \lambda (N - Y_H - X_M - Y_A) (X_M - X_M^*) (M - M^*) \\ & + k_2 \lambda M^* (N - N^*) (X_M - X_M^*) - k_2 \lambda M^* (X_M - X_M^*) (Y_A - Y_A^*) \\ & + k_4 \mu (Y_H - Y_H^*) (M - M^*) + k_4 \mu (Y_A - Y_A^*) (M - M^*). \end{aligned} \quad (47)$$

On choosing,  $k_2 = \frac{\beta}{\lambda M^* N^*} k_1$ ,  $k_3 = \frac{\beta}{\delta N^*} k_1$  and  $k_0 = k_1 = 1$ , we get  $\frac{dV}{dt}$  negative definite and desired conditions. Here,

$$\frac{9\beta \lambda}{4N^* M^* \mu_0 (\lambda M^* + \lambda_0 + d)} (\frac{\beta}{\delta})^2 < k_4 < \frac{16\mu_0 \beta}{9\mu^2 N^*} \min. [\frac{\alpha + d}{\delta}, 1].$$