On the Impact of Awareness Programs in HIV/AIDS Prevention: An SIR Model with Optimal Control

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ABSTRACT

In this work, a mathematical model for studying the impact of awareness programs on HIV/AIDS outbreak is proposed. The main idea is that people who are susceptible to infection can prevent it, if they are aware how the disease spreads and its consequences, and also the measures to control it. Various forms of communication media, educational, heath institutions and non-governmental organizations play a significant role to promote HIV/AIDS awareness amongst the most concerned people, namely couples and senior secondary school children. The developed HIV model is inspired from the classical SIR epidemic model where a control function is introduced to represent the effectiveness of an awareness program. The obtained optimal control, is characterized in terms of the optimality system, based on Pontryagin's maximum principle, and it is simulated using the Forward-Backward Sweep Method with a progressive-regressive Runge-Kutta fourth order scheme, which is adapted to solve a two-point boundary value problem.

General Terms

Awareness in the prevention of HIV/AIDS

Keywords

HIV/AIDS model, SIR model, Optimal control, Awareness program

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1. INTRODUCTION

Since their discovery at the beginning of the 1980s, human immunodeficiency virus infection and acquired immune deficiency syndrome (HIV/AIDS), have been reported by many researchers, as an epidemic representing a global threat to humans, particularly to people living with HIV in the sub-Saharan African regions that account for almost two thirds of the global total number of infected people with HIV worldwide, and the HIV/AIDS statistics are still staggering in those regions since that time [1]. In fact, based on a recent report of the Joint United Nations Programme on HIV/AIDS (UNAIDS) in 2014 [2], it is estimated that the number of people newly infected with HIV has decreased worldwide from the peak of 3.6 million in 2001 to an estimated 2.1 million in 2013. Similar conclusions about the newly infected with HIV among children have been observed by a decline of 58% since 2001. Such declines could be understood by the increased availability of the antiretroviral therapy for 13.6 million of people living with HIV in June 2014 after it was 12.9 million in 2013 [2]. However, fewer than half of people living in Africa, who are having the opportunity to receive treatment, which could unfortunately imply a stability or less important decrease regarding the number of infected people with HIV in the future next years [1]. In this context, UNAIDS aimed by its new program HIV/AIDS started in September 2014, to diagnose, facilitate access to antiretroviral therapy, and achieve viral suppression for 90% people infected with HIV by 2020 [3], but that will require about US\$ 32.8 billion to be invested by low- and middle-income countries [2]. Thus, HIV/AIDS epidemic alone, can cause a serious financial problem to governments of those countries for funding such UNAIDS programs. UNAIDS publishes annually other reports and provide some plans and guidelines for all health and political international communities to ensure the statistics used in the data analysis for their better employment and aiming to advance the HIV/AIDS prevention operations as soon as possible towards the elimination of new HIV infections [4, 5, 6].

HIV/AIDS does not only threaten the human global health and economy, but has also an impact on the sector of education participating in the decline of school enrolment in the sub-Saharan African countries due to AIDS at the beginning of the millennium and also to the illness and deaths of teachers by HIV [7]. It was observed that HIV/AIDS affects the normal life of students in rural schools more than in urban areas due to lack of basic needs and health services accessibility [8]. In spite of that, the relationship between education and HIV/AIDS is actually circular. More concretely, education can not be regarded only as a mean of passing information, but could equally play a major psychosocial role against HIV/AIDS spread [8, 9, 10, 11], based on different forms of awareness strategies, in an attempt to change attitudes and behaviors of the more concerned individuals such as senior secondary school children who will likewise warn their parents from the danger of HIV/AIDS. Heath institutions and some associations can also participate in HIV/AIDS prevention, explaining to their societies how the disease can spread and how they could control it.

Many published books focused on treating educational workshops and awareness programs as powerful plans and strategic actions in the war on HIV/AIDS in the middle east, and sub-Saharan and north African countries [12, 13]. In an other work [14], José Catálan et al. discussed some problems that meet some condoms and drugs users, and presented different alternatives forms of HIV/AIDS prevention based on reports of many papers where their authors highlighted the role of awareness programs that can be led by educational institutions and also the media, to intervene as psychosocial factors encouraging more actions and much efforts in the fight against HIV infection.

In order to show and prove the influence of awareness strategies on reducing the number of HIV/AIDS infectives, a threecompartmental SIR mathematical model is proposed with a control function representing the effectiveness of awareness programs on susceptible population when meeting infected people with HIV in an attempt to help them to recognize the danger that pursues them when they behave wrongly during their sexual life. In section 2., the different components of the suggested model are described, and the stability is studied when the control process function is only in the form of a constant parameter, and in section 3., the characterization of that control is sought for showing its impact on S,I and R functions as it is done in numerical simulations presented in section 4.

2. PRESENTATION OF THE MODEL

Consider an SIR model for HIV transmission in a population of individuals. The model sub-divides the total human population into three separate classes, S(t) susceptible individuals, I(t) infected individuals and R(t) individuals removed from the disease, i.e. people who are sexually inactive, or they take their precautions and they are no longer infecting susceptibles. It is assumed that susceptible individuals not yet infected with HIV but can be infected through the sexual contacts with infectives, or become aware and transfer to the removed class. Note that S, I, R > 0 because they represent numbers of people. It is also assumed that the recruited individuals (by birth and immigration), are constant and enter the susceptible compartment, that is, $\Gamma \succ 0$. Further, assuming that the number of people removed from each class due to natural causes such as death (not HIV or AIDS related), is proportional to the number of individuals in the compartmant, μS , μI and μR , where $\mu \succ 0$ will be called the natural death rate for historical reasons, which is constant. Additionally, the number of individuals removed from the infective class into the removed class (they are no longer sexually active by health conditions), is proportional to the number of individuals in the infective class, γI , where $\gamma \succ 0$ is the removal rate which is a constant. The proportion of infected individuals to sexually active individuals, $\frac{1}{S+I}$, and $\beta \succ 0$, is the infection rate, and which is a constant. Then, the disease transmission is modeled using standard incidence, given by

$$\frac{\beta I}{I+S}S$$

The following system of ODEs describes this SIR model

$$\frac{dS}{dt} = \Gamma - \frac{\beta I}{I+S}S - \mu S - \theta S \tag{1}$$

$$\frac{dI}{dt} = \frac{\beta I}{I+S}S - \gamma I - \mu I \tag{2}$$

$$\frac{dR}{dt} = \gamma I - \mu R + \theta S \tag{3}$$

Since R does not affect S or I, consider the equivalent system

$$\frac{dS}{dt} = \Gamma - \frac{\beta I}{I+S}S - \mu S - \theta S \tag{4}$$

$$\frac{dI}{dt} = \frac{\beta I}{I+S}S - \gamma I - \mu I \tag{5}$$

3. STEADY STATES

3.1 Disease-free equilibrium

Consider the case where there is no infection, then I = 0, thus by setting $\frac{dS}{dt} = 0$, it is obtained that

$$\Gamma - \frac{\beta I}{I+S}S - \mu S - \theta S = 0$$

It follows that

$$S = \frac{\Gamma}{\mu + \theta}$$

then the disease-free equilibrium here is

$$E_0 = \left(\frac{\Gamma}{\mu + \theta}, 0\right)$$

Thus, in the absence of infectives the susceptibles have an equilibrium value of $\frac{\Gamma}{\mu+\theta}$. To investigate the stability of this equilibrium, the reproductive number is defined by,

$$R_0 = \frac{\beta}{\mu + \gamma} \tag{6}$$

Proposition 2.1.1. The disease-free equilibrium $E_0 = \left(\frac{\Gamma}{\mu+\theta}, 0\right)$ is locally asymptotically stable if and only if $R_0 \prec 1$.

Proof. At E_0 , the Jacobien is

$$J(E_0) = \begin{pmatrix} -\mu - \theta & -\beta \\ 0 & -\gamma - \mu + \beta \end{pmatrix}$$

which has eigenvalues $\{-\mu - \theta, -\gamma - \mu + \beta\}$. All eigenvalues are strictly negative if and only if $-\gamma - \mu + \beta \prec 0$, which means that $R_0 \prec 1$.

Proposition 2.1.2. If $R_0 \prec 1$, then $I(t) \stackrel{t \to \infty}{\to} 0$.

Proof. $R_0 \prec 1$ implies that $\beta - (\gamma + \mu) \prec 0$. Then

$$\frac{dI}{dt} \prec \left(\beta - (\gamma + \mu)\right) I \prec 0$$

thus by positivity of I, $I(t) \stackrel{t \to \infty}{\to} 0$.

Theorem 2.1.1. The disease-free equilibrium E_0 is globally asymptotically stable if $R_0 \prec 1$.

Proof. From Proposition 2 it suffices to show that $S \to \frac{\Gamma}{\mu+\theta}$. Let $\epsilon \succ 0$, then there is $t_{\epsilon} \succ 0$ such that for $t \succ t_{\epsilon}$, $I(t) \leq \epsilon$. Then

$$\frac{dS}{dt} \ge \Gamma - \beta \epsilon S - (\mu + \theta) S$$

Let $S_{\epsilon}(t)$ be the solution of the differential equation

$$\frac{dS_{\epsilon}}{dt} = \Gamma - \beta \epsilon S_{\epsilon} - (\mu + \theta) S_{\epsilon}$$

, which has the steady state given by $S_{\epsilon}^* = \frac{\Gamma}{\beta \epsilon S + (\mu + \theta)}$. Since $S_{\epsilon}^{''} = -\beta \epsilon S - (\mu + \theta) \prec 0$ is strictly negative, then $S_{\epsilon} \rightarrow S_{\epsilon}^*$

and since $S_{\epsilon} \stackrel{\epsilon \to 0}{\to} S$, then

$$S \to \frac{\Gamma}{\mu + \theta}$$

which complete the proof.

3.2 The Endemic Equilibrium

Now consider the case where $R_0 > 1$ so that the system has an endemic infection. Then by proposition 1 E_0 is unstable. By setting

 $\frac{dS}{dt} = 0$ and $\frac{dI}{dt} = 0$

$$\frac{\beta I}{-\beta I}S = \Gamma - \mu S - \theta S$$

$$\frac{I+S}{I+S}S = (\gamma + \mu)I$$
(8)

or

then

$$\Lambda - \mu S - \theta S = (\gamma + \mu) I$$

$$S = \frac{\Gamma}{\mu + \theta} - \frac{I(\gamma + \mu)}{\mu + \theta}$$

And by (8)

$$\frac{\beta S}{I+S} = (\gamma + \mu)$$
$$\frac{\beta S}{\gamma + \mu} = I + S$$
$$I = \frac{\beta S}{\gamma + \mu} - S$$

thus

$$f = (R_0 - 1) S \tag{10}$$

Finally substituting (10) into (9) and solving for S it is obtained;

1

$$S^{*} = \frac{\Gamma}{\mu + \theta + (R_{0} - 1)(\gamma + \mu)}$$
(11)

$$I^{*} = \frac{(R_{0} - 1)\Gamma}{\mu + \theta + (R_{0} - 1)(\gamma + \mu)}$$
(12)

thus the endemic equilibrium point is

$$E_e = (S^*, I^*)$$
 (13)

Theorem 2.2.1. The endemic equilibrium $E_e = (S^*, I^*)$ is locally asymptotically stable if and only if $R_0 \succ 1$.



Fig. 1. The states of system (1-3) with (a) $E_0=(2439,0)$, $R_0=0.4$ (b) $E_e=(880,1594)$, $R_0=2.8114$

Proof. Finding the eigenvalues of the Jacobian matrix at E_e results in the characteristic equation

$$\lambda^{2} + (\theta + \beta - \gamma) \lambda + \frac{(-\beta + \gamma - \mu)(\gamma + \mu)(-\theta - \beta + \gamma)}{\beta} = 0$$

Since $R_0 \succ 1$, thus by (6) it follows that

$$\theta + \beta - \gamma \succ \theta + \mu \succ$$

so in order for the real part of both eigenvalues to be negative,

$$\frac{(-\beta + \gamma - \mu)(\gamma + \mu)(-\theta - \beta + \gamma)}{\beta} \succ 0 \tag{14}$$

0

using the fact that $R_0 \succ 1$, then

$$-\beta + \gamma - \mu \prec 0$$
 and $-\theta - \beta + \gamma \prec 0$

then (14) holds. Thus, both eigenvalues of the Jacobian at E_e for $R_0 > 1$ have negative real part, so E_e is asymptotically stable. \Box

Figure 1 depicts the two equilibria of the model, where the left side of this figure (a), shows the disease-free equilibrium with $R_0 = 0.4$, while the right one (b) shows the endemic equilibrium with $R_0 = 2.8114$.

4. OPTIMAL AWARENESS PROGRAM

In this section, it is considered θ as a function of time t. Our main goal is to minimize the functional J given by

$$J(\theta) = \int_0^{t_f} \left(A_1 I(t) - A_2 R(t) + \frac{K}{2} \theta^2(t) \right) dt \qquad (15)$$

subject to

(7)

(9)

$$\frac{dS}{dt} = \Gamma - \frac{\beta I}{I+S}S - \mu S - \theta S \tag{16}$$

$$\frac{dI}{dt} = \frac{\beta I}{I+S}S - \gamma I - \mu I \tag{17}$$

$$\frac{dR}{dt} = \gamma I - \mu R + \theta S \tag{18}$$

The first terms represent the crucial goal of the awareness program, that is of reducing the number of the infected people, and increasing the removed ones. The other term is systemic cost of the awareness program. The positive constants A_1 , A_2 and K balance the size of terms. Also the reason behind considering a finite time horizon is that such control program is usually restricted to a limited time window.

An optimal control θ^* is sought such that

$$J(\theta^*) = \min \left\{ J(\theta) \, | \, \theta \in \Theta \right\}$$

Where

$$\Theta = \{\theta \text{ measurable}, 0 \le \theta(t) \le 1, t \in [0, t_f] \}$$

The necessary conditions that an optimal control problem must satisfy come from Pontryagin's maximum principle [15]. This principle converts (15)–(16-18) into a problem of minimizing pointwise a Hamiltonian H, with respect to θ

$$H = A_1 I(t) - A_2 R(t) + \frac{\kappa}{2} \theta^2(t)$$

+ $\lambda_1(t) \left[\Gamma - \frac{\beta I(i)}{I(i) + S(i)} S(i) - \mu S(i) - \theta(i) S(i) \right]$
+ $\lambda_2(t) \left[\frac{\beta I(i)}{I(i) + S(i)} S(i) - \gamma I(i) - \mu I(i) \right]$
+ $\lambda_3(t) \left[\gamma I(i) - \mu R(i) + \theta(i) S(i) \right]$

By applying Pontryagin's Maximum Principle [15] and the existence result for the optimal control from [16], The following theorem is obtained:

Theorem 3.1. There exists an optimal control θ^* and corresponding solution, S^* , I^* and R^* that minimizes $J(\theta)$ over Θ Furthermore, there exists adjoint functions, $\lambda_1(t)$, $\lambda_2(t)$ and $\lambda_3(t)$, such that

$$\dot{\lambda}_{1} = -\left[\frac{\beta I^{*^{2}}}{\left(I^{*} + S^{*}\right)^{2}} \left(\lambda_{2} - \lambda_{1}\right) - \left(\mu + \theta^{*}\right)\lambda_{1} + \theta\lambda_{3}\right]$$
(19)

$$\dot{\lambda}_{2} = -[A_{1} + \frac{\beta S^{*2}}{(I^{*} + S^{*})^{2}} (\lambda_{2} - \lambda_{1}) - (\gamma + \mu) \lambda_{2} + \gamma \lambda_{2}]$$
(20)

$$\dot{\lambda}_{3} = -[-A_{2} - \mu\lambda_{3}] \tag{20}$$

with transversality conditions

$$\lambda_i(t_f) = 0, \, i = 1, 2, 3 \tag{22}$$

The following characterization holds

$$\theta^*(t) = max\left\{min\left\{\left(\lambda_1(t) - \lambda_3(t)\right)\frac{S^*(t)}{K}, 1\right\}, 0\right\} (23)$$

Proof. Corollary 4.1 of [16] gives the existence of an optimal control due to the convexity of integrand of J with respect to θ , a priori boundedness of the state solutions, and the Lipschitz property of the state system with respect to the state variables. Applying Pontryagin's Maximum Principle, the following adjoint system is obtained

$$\begin{split} \dot{\lambda}_1 &= -\frac{dH}{dS} \\ &= -[\frac{\beta I^{*^2}}{\left(I^* + S^*\right)^2} \left(\lambda_2 - \lambda_1\right) - \left(\mu + \theta^*\right) \lambda_1 + \theta \lambda_3] \\ \lambda_1(t_f) &= 0, \\ \dot{\lambda}_2 &= -\frac{dH}{dI} \\ &= -[A_1 + \frac{\beta S^{*^2}}{\left(I^* + S^*\right)^2} \left(\lambda_2 - \lambda_1\right) \\ &- \left(\gamma + \mu\right) \lambda_2 + \gamma \lambda_3], \\ \lambda_2(t_f) &= 0, \\ \dot{\lambda}_3 &= -\frac{dH}{I_2} \end{split}$$

$$\dot{\lambda}_3 = -\frac{dH}{dR} \\ = -[-A_2 - \mu\lambda_3],$$



Fig. 2. The optimal control of the awareness strategy with $K = 1 \times 10^6$, $A_1 = A_2 = 1 S(0) = 1000$, I(0) = 200 and R(0) = 0 (a) States of system (16-18) without control (b) States of system (16-18) with control (c) Control $\theta(t)$ as a function of time.

$$\lambda_3(t_f) = 0,$$

evaluated at the optimal control θ^* and corresponding states S^* , I^* and R^* , which results in the stated adjoint system (19-21) and (22), [17]. By considering the optimality conditions,

$$\frac{dH}{d\theta} = 0$$

and solving for θ^* , it follows

$$\frac{dH}{d\theta} = K\theta - \lambda_1 S + \lambda_3 S = 0$$

Taking into account the bounds on θ^* in Θ , it is deduced that

$$\theta^*(t) = max \left\{ min \left\{ \left(\lambda_1(t) - \lambda_3(t)\right) \frac{S(t)}{K}, 1 \right\}, 0 \right\}$$

Due to the priori boundedness of the state and adjoint functions and the resulting Lipschitz structure of the ODEs, the uniqueness of the optimal control is obtained for small t_f . The uniqueness of the optimal control follows from the uniqueness of the optimality system, which consists of (16-18) and (19-21), (22) with characterizations (23). There is a restriction on the length of the time interval in order to guarantee the uniqueness of the optimality system. This smallness restriction on the length on the time interval is due to the opposite time orientations of (16-18) and (19-21), (22), the state problem has initial values and the adjoint problem has final values. This restriction is very common in control problems [18, 19, 20, 21, 22, 23, 24].

5. NUMERICAL RESULTS AND DISCUSSIONS

In this section, the numerical solutions of the optimality system and the corresponding optimal control, the parameter choices, and the interpretations from various cases are discussed. The optimal



9 1000 500 0 200 400 600 800 1000 1200 time (in days)



program strategy is obtained by solving the optimality system. An iterative method is used for solving the optimality system. The resolution of the state equations is started with a guess for the controls over the simulated time using a forward fourth order Runge-Kutta scheme. Because of the transversality conditions (22), the adjoint equations are solved by a backward fourth order Runge-Kutta scheme using the current iteration solution of the state equations. Then, the controls are updated by using a convex combination of the previous controls and the value from the characterizations (23). This process is repeated and iteration is stopped if the values of variables at the previous iteration are very close to the ones at the present iteration.

Figure 1 depicts the numerical simulations of the differential system (1)-(3) where it can be seen that for a reproductive number $R_0 = 0.4$ and a disease-free equilibrium $E_0 = (2439, 0)$ (case a),



Fig. 5. The control $\theta(t)$ is plotted as a function of time for different values of β with $K = 1 \times 10^4$



Fig. 6. The control $\theta(t)$ is plotted as a function of time for the 4 different values of S(0) with I(0) = 200, R(0) = 0 and $K = 1 \times 10^5$



Fig. 7. The control $\theta(t)$ is plotted as a function of time for the 4 different values of I(0) with S(0) = 100000, R(0) = 0 and $K = 2 \times 10^3$

it results a very small number of infected and removed people, and a big number of susceptible people because there is yet no infection and no strategy is needed in this case to fight against any disease (case b),. In contrast, for a reproductive number $R_0 = 2.8114$ and a disease-free equilibrium $E_e = (880, 1594)$ (case b), it is observed that the number of infected people is increasing towards $E_{e_{y}} = 1594$. By the introduction of the control $\theta(t)$ which represents the effectiveness of the awareness program, the number of infected people has decreased in the case (b) compared to the case (a) when there was yet no control. The impact of the control $\theta(t)$ on the number of removed people can be understood by the increase of the number of removed people for about 800 days once the control $\theta(t)$ is introduced to the differential system (16)-(18) until it takes values around 0.1, and when it begins closer to zero between 800 to 1200 days (see Figure 2), R variable decreases and I variable increases simultaneously between that period.

It should also be noted that the final time is taken enough large (about 3 years) because an awareness program aiming to prevent HIV from spread, could often be observed successful only after a long period. Figure 3 shows more clearly the relationship between the control function and the variables S, I and R, and finally it can be deduced that the followed control strategy succeed to reduce

the number of infected people by HIV and increase the number of the removed people. By taking other values of the control severity weight K in Figure 4, it can be concluded that the more K is big, the more it is obtained lesser number of infected people and larger number of removed people, because the more K is small, the control $\theta(t)$ is big (see the control characterization (23)). The reproductive number R_0 and the infection rate beta have both also an impact on the behavior of control $\theta(t)$ in Figure 5, and it is observed that the more R_0 and β are big, the more the values of the control $\theta(t)$ becomes important. In addition, for different values of the initial condition associated to the variable S(t) in Figure 6, the values of the control $\theta(t)$ do not change, but change only and become more important whenever the initial condition associated to the variable I(t) are taken bigger as it is observed in Figure 7.

6. CONCLUSION

Education could help international societies to gain more time in the fight against epidemics, particularly HIV/AIDS here, and because it is believed that collaborations between health institutions are needed more today to face the danger of that disease, awareness programs can be seen as optimal strategies or preferable plans in this subject rather than treatments. A mathematical model for studying the impact of awareness programs on HIV outbreak was therefore proposed and analyzed. A control function was introduced to the SIR model to represent the effectiveness of the awareness programs. Optimal control strategies were identified for several values of the control severity weights as well as the infection rate and initial conditions of susceptibles and infectives, to show the importance and the effectiveness of the approach in controlling the infection spread. Control programs that follow these strategies can effectively reduce the number of infected cases and increase the number of people who are mindful of the danger of HIV. Thus, people can change their sexual behaviors and/or take their precautions by using for instance condom or by having regular blood tests.

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