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Sensitivity And Optimal Control Analysis Of HIV/AIDS Model*

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Abstract

In this study we revisit a theme of F. Nyabadza *et al.* [*Journal of Biological Systems* 18 No.2 (2010), 357–375], studying the optimal control of rolling out a public health education campaign and providing some further insights. We produce a Lyapunov function to prove global stability of the disease free equilibrium, and include a sensitivity analysis of the parameters and state variables. Finally, we present an optimal control problem on the implementation strategy of public health education, together with its solution. Various simulations are provided to illustrate the control problem.

1 Introduction

The HIV/AIDS epidemic has placed a large burden on public health resources globally, and especially in developing countries with limited resources. The importance of preventive measures in the fight against HIV, especially in resource-poor countries, cannot be over-emphasized. In the fight against HIV/AIDS, public health education plays an essential role. Public health education seeks to protect and improve the health of communities through education, promotion of healthy lifestyles, and awareness of measures for prevention of injury and disease. Public health professionals analyze the extent to which health is affected by various factors such as the environment, behavior, personal choice, genetics, etc., in order to develop programmes that benefit the health of families and communities.

Globally several initiatives on public health education have been launched. Different initiatives on education with regard to HIV have been launched by UNAIDS, to support

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the mainstreaming of HIV/AIDS in the Global Monitoring Report (GMR) (UNESCO and HIV/AIDS Training). In 2002, the UNAIDS Inter-Agency Task Team (IATT) on Education, convened by UNESCO, was established. Its aim is to improve and accelerate the roll-out of education on HIV and AIDS. Its specific objectives are to promote and support good practices in the education sector related to HIV/AIDS, and also to encourage alignment and harmonization within and across agencies to support global and country-level actions (UNESCO and HIV/AIDS Training).

In South Africa, the publication *HIV and AIDS Emergency Guidelines for Educators* from the Department of Education, sets out HIV facts and messages about preventing HIV. This deals with questions from educators about sexuality education, advises on universal precautions and how to build a school culture of non-discrimination (UNAIDS and Inter-Agency Task Team (IATT)). Some educational programmes on radio and television in South Africa, which enlighten children about HIV/AIDS were also supported by the government (UNAIDS and Inter-Agency Task Team (IATT), UNESCO and HIV/AIDS Training). A model, called *Schools as Centres of Care and Support* (SCCS), was also tested and supported by *Media in Education Trust* (MiET) in 2003/4. The model was later implemented by Swaziland and Zambia in 2005 and 2008 respectively.

Mathematical models have been an important tool as a means of informing control strategies, since they form the basis for short and long term prediction of HIV and AIDS prevalence. From the early models of May and Anderson (Anderson et al., 1986; Anderson, 1988; May and Anderson, 2011) several modifications of the modeling structure have been presented. Specific related issues have been researched by various authors. For instance, Cai et al. (2009) investigated an HIV/AIDS epidemic model with treatment, studying the stability of its equilibrium points. Naresh et al. (2009) used a mathematical model to analyze the spread of the HIV/AIDS epidemic with recruitment of infectives. Nyabadza et al. (2010) used a model incorporating condom use, sexual partner acquisition, behavior change and treatment to study the epidemic trends of HIV/AIDS in South Africa. The paper Hussaini et al. (2011) emphasizes and analyses the role of education in different countries. Recently, Nsuami and Witbooi (2018) propose a new model for the transmission of HIV/AIDS including ART and PrEP. The model was used to test the effects of ART and of the uptake of PrEP in a given population, and proved the global stability of the disease-free equilibrium and that of endemic equilibrium (Nsuami and Witbooi, 2018). Another study by Nyabadza et al. (2010), investigates the reduction in infection by observing the changes in sexual behavior through public health information campaigns, and self-withdrawal of individuals with AIDS from sexual activity. Their results showed that an increase in effective public health information campaigns together with AIDS individuals' withdrawing from sexual activity, will lead to a significant reduction in the spread of HIV/AIDS.

This paper makes a contribution to the theme of Nyabadza et al. (2010) by producing a Lyapunov function to prove global stability of the disease free equilibrium, analyzing the sensitivity of the basic reproduction number and the endemic equilibrium point, and investigating for an optimal control strategy for the roll-out of public health education to minimize the spread of HIV/AIDS. We determine the optimal levels of intensity of public health education effort, over time, for disease control using the Pontryagin's Maximum Principle. Both analytical and numerical studies of the

model are conducted to obtain the necessary information that could be useful towards reducing the spread of the disease. In Section 2 of this paper we present the HIV model proposed by Nyabadza et al. (2010) The stability discussion follows in Section 3 and the sensitivity analysis in Section 4. In Section 5 we present the optimal control problem together with its solution. Numerical results and analysis of the control problem appear in Section 6, while Section 7 offers some concluding remarks.

2 The Model and Its Equilibrium States

The model which is central to this paper is that of Nyabadza et al. (2010). The said model considers a sexually active population of size $N(t)$ at time t. The population is subdivided into the following subclasses (compartments): susceptibles $S(t)$, asymptomatic infectives $I_1(t)$ (infectious individuals who are yet to show symptoms of the disease), symptomatic infectives $I_2(t)$ (infectious individuals who show symptoms of the disease) and individuals with full blown AIDS, $A(t)$. We assume that the mode of transmission is via heterosexual contacts. Then the following equation holds.

$$N(t) = S(t) + I_1(t) + I_2(t) + A(t).$$

We also assume that any two susceptible individuals are equally likely to be infected by an infectious individual.

The normal mortality rate is μ and the disease induced mortality rate is denoted by σ. The recruitment rate of susceptible individuals is given by μb while the transfer rate from the asymptomatic compartment to the symptomatic compartment is given by σ and the removal rate of the symptomatic infectives as they develop to AIDS by ρ. Thus we have the following system.

$$\begin{cases} \frac{dS}{dt} = \mu b - \mu S - \lambda(\mathbf{I}, A)S, \\ \frac{dI_1}{dt} = \lambda(\mathbf{I}, A)S - (\mu + \sigma)I_1, \\ \frac{dI_2}{dt} = \sigma I_1 - (\mu + \rho)I_2, \\ \frac{dA}{dt} = \rho I_2 - (\mu + \delta)A, \end{cases} \tag{1}$$

where

$$\lambda(\mathbf{I}, A) = \frac{c\beta\{I_1 + \eta_1 I_2 + \eta_2(1-q)A\}}{1 + \alpha\{I_1 + \eta_1 I_2 + \eta_2(1-q)A\}}$$

η_1 and η_2 measure the contribution of I_2 and A relative to I_1, and $\mathbf{I} = (I_1, I_2)$.

The constant c is the mean number of sexual partners per given time and β denotes the probability of infection, while q represents the proportion of individuals who voluntarily withdraw from sexual activity as a result of knowing their HIV infection status, implying $(1-q)$ engage in sexual activities. By α we denote the effort on public health education, which we assume to be proportional to its effectiveness in reducing HIV transmission. In general we regard α as being a function of time. In fact, $\alpha(t)$ will serve as the control variable when we study the optimal control problem. When analyzing equilibria, then α is considered a time-independent constant parameter. Note

that all the parameters are positive. The initial state of the system (1) is given as

$$S(0) = S_0 > 0, \quad I_1(0) = I_{10} > 0, \quad I_2(0) = I_{20} > 0, \quad A(0) = A_0 > 0.$$

The proposed contact rate allows for flexibility, covering many special cases such as the popular standard forms (with $\alpha = 0$) while also approximating a saturation transmission rate when the term

$$\alpha\{I_1 + \eta_1 I_2 + \eta_2(1-q)A\}$$

is significantly bigger than 1. As declared in Nyabadza et al. (2010), in the latter case the transmission rate very closely reflects the availability of public health education in the population.

The equilibrium points have been discussed in Nyabadza et al. (2010). There is the possibility of an endemic equilibrium point E_1, on which we shall present a very brief sensitivity analysis in Section 3. The disease-free equilibrium point is denoted by E_0, and is given by $E_0 = (b, 0, 0, 0)$. It is known that for local asymptotic stability of E_0, a necessary condition is that the numerical value of the basic reproduction number does not exceed unity. Recall that the basic reproduction number, usually denoted by R_0, of a disease in a given population (see van den Driessche and Watmough (2002) for instance), is the expected number of secondary infections produced by one infective in a completely susceptible population. A method for calculating R_0 is developed in van den Driessche and Watmough (2002). For the model (1), the basic reproductive number is calculated in Nyabadza et al. (2010) and is given by

$$R_0 = \frac{\beta bc}{\mu + \sigma}\left[1 + \frac{\eta_1 \sigma}{\rho + \mu} + \frac{\eta_2 \rho \sigma (1-q)}{(\rho + \mu)(\delta + \mu)}\right].$$

We are interested in the global stability of E_0. If the disease-free equilibrium is globally asymptotically stable, then we can be sure that whenever there has been infection, over time the disease will vanish from the population provided there are no external disturbances on the population.

We prove the global stability of E_0 by exhibiting a Lyapunov function.

THEOREM 1. The disease-free equilibrium E_0 of the model (1) is globally asymptotically stable if $R_0 < 1$.

PROOF. Let us fix some constants a_1, a_2, a_3 and ξ as follows:

$$a_1 = (\mu + \rho)(\mu + \delta),$$

$$a_2 = c\beta b[\eta_1(\mu + \delta) + \rho\xi],$$

$$a_3 = \xi c\beta b(\mu + \rho) \quad \text{and} \quad \xi = \eta_2(1-q).$$

Now we define a function $V = V(I_1(t), I_2(t), A(t))$ which we shall prove to be a Lyapunov function at the point $(I_1, I_2, A) = (0, 0, 0)$.

$$V = a_1 I_1 + a_2 I_2 + a_3 A.$$

Taking the derivatives with respect to time, we obtain

$$\dot{V} = a_1\dot{I}_1 + a_2\dot{I}_2 + a_3\dot{A}.$$

Now noting that $\lambda < c\beta(I_1 + \eta_1 I_2 + \xi A)$ and $S(t) < b$ for all $t \geq 0$, it follows that we can write:

$$\dot{V} < Q_1 I_1 + Q_2 I_2 + Q_3 A,$$

where the coefficients Q_i are as follows:

$$\begin{aligned} Q_1 &= a_1[c\beta b - (\mu+\sigma)] + a_2\sigma, \\ Q_2 &= a_1\eta_1 c\beta b - a_2(\mu+\rho) + a_3\rho, \\ Q_3 &= a_1\xi c\beta b - a_3(\mu+\delta). \end{aligned}$$

Now we notice that substituting the values of a_1, a_2, a_3 and ξ, we obtain the following:

$$\begin{aligned} Q_2 &= a_1\eta_1 c\beta b - c\beta b[\eta_1(\mu+\delta) + \xi\rho](\mu+\rho) + a_3\rho \\ &= a_1\eta_1 c\beta b - a_1\eta_1 c\beta b - c\beta b\rho\xi(\mu+\rho) + c\beta b\xi(\mu+\rho)\rho \\ &= 0. \end{aligned}$$

Likewise for Q_3 we find:

$$\begin{aligned} Q_3 &= \xi c\beta b(\mu+\rho)(\mu+\delta) - \xi c\beta b(\mu+\rho)(\mu+\delta) \\ &= 0. \end{aligned}$$

Finally we turn to Q_1.

$$\begin{aligned} Q_1 &= a_1 c\beta b + a_2\sigma - a_1(\mu+\sigma) \\ &= a_1(\mu+\sigma)\left[\frac{c\beta b}{(\mu+\sigma)} + \frac{a_2\sigma}{a_1(\mu+\sigma)} - 1\right] \\ &= a_1(\mu+\sigma)\left[\frac{c\beta b}{(\mu+\sigma)} + \frac{c\beta b[\eta_1(\mu+\delta)+\rho\xi]\sigma}{(\mu+\rho)(\mu+\delta)(\mu+\sigma)} - 1\right] \\ &= a_1(\mu+\sigma)\left[\frac{c\beta b}{\mu+\sigma}\left\{1 + \frac{\eta_1\sigma}{\mu+\rho} + \frac{\rho\xi\sigma}{(\mu+\rho)(\mu+\delta)}\right\} - 1\right] \\ &= (\mu+\rho)(\mu+\delta)(\mu+\sigma)[R_0 - 1] \\ &< 0, \end{aligned}$$

since $R_0 < 1$. It follows that V is a Lyapunov function as asserted. This completes the proof.

3 Sensitivity Analysis of Model Parameters and State Variables

Knowledge of the sensitivity of parameters is quite useful for decision making and intervention purposes, because it helps in making recommendations more credible and

understandable. We proceed with such sensitivity analysis towards investigating the model robustness to parameter values. The basic reproduction number is pivotal in determining the stability of the disease free equilibrium. It is thus important for us to understand the behavior of R_0 with respect to the different parameters. We shall also study the sensitivity of the endemic equilibrium point. To carry out this analysis, we use the normalised forward sensitivity index of an invariant with respect to a parameter, as described in Makinde and Okosun (2011) for instance. The sensitivity of an invariant U with respect to a parameter ζ is given by:

$$\frac{\zeta}{U} \times \frac{\partial U}{\partial \zeta} \ .$$

It should also be noted that the same sensitivity index has another significance. We note that when applying a model to a real life situation, the parameters need to be estimated from data. Accuracy could be a problem, and the sensitivity index serves as an indicator of the effect of an error in the parameters' estimation.

3.1 Sensitivity Analysis of R_0

Table 1: Sensitivity indices of R_0

Parameter description	Parameter value	Sensitivity
Partner acquisition rate	c	+3.57
Probability of transmission	β	+3.57
Natural death rate	μ	-0.04
Rate of developing AIDS	ρ	-0.02
Rate of becoming symptomatic	σ	-0.003
Enhancement factor	η_1	0.002
Proportion of withdrawals by AIDS	q	-0.0007

Computation of the partial derivatives is quite routine and we skip the details. The parameter values as from Nyabadza et al. (2010) are shown in Table 3. In Table 1, the parameters are arranged from the most sensitive to the least for the given base values

of the parameters as in the list. The most sensitive parameters here are the partner acquisition rate c and probability of transmission β, followed by the natural death rate of individual μ. Other important parameters include the rate ρ of developing to AIDS with -0.02. The least sensitive parameter is the proportion of withdrawals by AIDS cases q.

The sensitivity index of R_0 with respect to the partner acquisition rate c is 3.5715, implying that decreasing (or increasing) c by 10% will result in R_0 decreasing (or increasing) by approximately 35.7%. We can similarly analyze the effect of incremental changes in other parameters.

3.2 Sensitivity Analysis of The Endemic Equilibrium State

Table 2: Sensitivity indices of state variables to model parameters.

	S^*	I_1^*	I_2^*	A^*
	β	-2.28	1.86	3.71
β	-2.28	1.86	3.71	6.19
ρ	0.48	-0.001	0.22	-1.85
μ	-1.98	-0.01	0.22	0.46
α	-0.70	1.00	2.00	0.33
η_1	0.05	0.0001	0.0002	0.00004

Here we derive the sensitivity of the endemic equilibrium values of the state variables to each of the parameters described in Table 2. In Table 2, the first row indicates that for the probability of transmission β, the class A^* is most sensitive with class I_1^* as the least. This means that an increase (or decrease) of β by 10% will increase (or decrease) A^* by 61.9% and I_1^* by 18.6%. In the table we have only listed the parameters that yield significantly high sensitivity indices, together with η_1, to show its relatively low sensitivity.

4 Optimal Control Analysis

In this section, we investigate for the optimal effort on public health education that would be needed to control HIV/AIDS, and we use optimal control theory. The control function is $\alpha(t)$ and the set of admissible controls is:

$$U = \{\alpha(\cdot)|\ \alpha(t)\text{is measurable and } 0 \leq \alpha \leq 1\}.$$

We propose an objective functional J through which we shall minimize the number of human infectives and AIDS individuals, balanced against the effort on public health education. The function J takes the form

$$J = \int_0^\tau (M_0 I_1 + M_1 I_2 + M_2 A + M_3 \alpha^2) dt$$

where M_0, M_1, M_2, M_3 are positive weights, for some $\tau > 0$. With the given objective function $J(\alpha)$, our goal here is to minimize in a weighted manner, the numbers $I_1(t), I_2(t)$ and $A(t)$, while also minimizing the cost of control $\alpha(t)$. We choose to use α^2 (i.e., quadratic rather than linear) to ensure that the Hamiltonian (see below) is convex in the control variable. This choice is commonly used in the literature on epidemic control. Solving the optimization problem requires the introduction of the Hamiltonian function $\mathcal{H}$, which for our problem is as follows:

$$\begin{aligned} \mathcal{H} &= M_0 I_1 + M_1 I_2 + M_2 A + M_3 \alpha^2 + \Phi_S[\mu b - \mu S - \lambda(\mathbf{I}, A) S] \\ &\quad + \Phi_{I_1}[\lambda(\mathbf{I}, A) S - (\mu + \sigma) I_1] + \Phi_{I_2}[\sigma I_1 - (\mu + \rho) I_2] \\ &\quad + \Phi_A[\rho I_2 - (\mu + \delta) A], \end{aligned} \tag{2}$$

where $\Phi_S, \Phi_{I_1}, \Phi_{I_2}$ and Φ_A are the adjoint variables. We now proceed towards solving the optimal control problem.

THEOREM 2. For optimality, the adjoint variables $\Phi_S, \Phi_{I_1}, \Phi_{I_2}$ and Φ_A satisfy the following differential equations:

$$\begin{cases} -\frac{d\Phi_S}{dt} = \mu \Phi_S + (\Phi_S - \Phi_{I_1})(I_1 + I_2 \eta_1 + (1-q) A) \psi, \\ -\frac{d\Phi_{I_1}}{dt} = -M_0 + (\mu + \sigma) \Phi_{I_1} - \sigma \Phi_{I_1} + (\Phi_S - \Phi_{I_1}) \psi, \\ -\frac{d\Phi_{I_2}}{dt} = -M_1 - \rho \Phi_A + (\mu + \rho) \Phi_{I_2} + (\Phi_S - \Phi_{I_1}) \eta_1 \psi, \\ -\frac{d\Phi_A}{dt} = -M_2 + (\delta + \mu) \Phi_A + (\Phi_S - \Phi_{I_1})(1-q) \eta_2 \psi, \end{cases} \tag{3}$$

where

$$\psi = \frac{c\,\beta}{\left(1 + \alpha\ (I_1 + I_2\,\eta_1 + A\ (1-q)\ \eta_2)\right)^2}$$

with transversality conditions

$$s(\tau) = \Phi_{I_1}(\tau) = \Phi_{I_2}(\tau) = \Phi_A(\tau). \tag{4}$$

The optimal control takes the form:

$$\alpha^* = \max\left\{0, \min\left(1, \frac{c\,S\,\beta\ (I_1 + I_2\,\eta_1 + A\ (1-q)\ \eta_2)^2\ \Phi_S}{2\,M_3\left(1 + \alpha\ (I_1 + I_2\,\eta_1 + A\ (1-q)\ \eta_2)^2\right)} (\Phi_S - \Phi_{I_1})\right)\right\}. \tag{5}$$

PROOF. An optimal control does exist due to the convexity of the integrand of J with respect to α, boundedness of the state solutions, and the Lipschitz property of

Table 3: Model parameters with their interpretations.

Parameter	Parameter description	Value	Source
c	Partner acquisition rate	1, 1.5	Nyabadza et al., 2010
μb	Recruitment rate	0.05	Nyabadza et al., 2010
q	Proportion of AIDS withdrawals	$0 \leq$ q ≤ 1	Estimate
σ	Rate of becoming symptomatic	0.14	Nyabadza et al., 2010
μ	Natural death rate	0.02	Nyabadza et al., 2010
α	Exposure rate to media campaigns	$0 \leq \alpha \leq 1$	Estimate
ρ	Rate of developing AIDS	0.05	Nyabadza et al., 2010
β	Probability of transmission	0.00000002	Nyabadza et al., 2010
(η_1, η_2)	Enhancement factor	1.6, 1.8	Nyabadza et al., 2010
δ	Disease-induced death rate	0.33	Nyabadza et al., 2010; Abiodun et al., 2013

the state system with respect to the variables, see Lenhart and Workman (2007) for instance. It suffices for us to check the first order conditions. Then firstly, the differential equations describing the adjoint variables are obtained by partial differentiation of the Hamiltonian function with respect to the state equations. This yields the system (6) of ode's. Secondly, the first order conditions require that α must minimize $\mathcal{H}$. So we proceed to calculating $\frac{\partial \mathcal{H}}{\partial \alpha}$. By a standard argument involving the latter partial derivative (set to zero) and the bounds on the controls, we get

$$\alpha^* = \begin{cases} 0 & \text{if } \zeta_2^* \leq 2, \\ \zeta_2^* & \text{if } 0 < \zeta_2^* < 1, \\ 1 & \text{if } \zeta_2^* \geq 1, \end{cases}$$

where

$$\zeta_2^* = \frac{c\,S\,\beta\,\left(I_1 + I_2\,\eta_1 + A\,(1-q)\,\eta_2\right)^2\,\Phi_S}{2\,M_3\left(1 + \alpha\,\left(I_1 + I_2\,\eta_1 + A\,(1-q)\,\eta_2\right)^2\right)}\left(\Phi_S - \Phi_{I_1}\right). \tag{6}$$

It follows that the optimal control $\alpha^*(t)$ does in fact take the form as asserted in the theorem. This completes the proof.

5 Numerical Results and Discussion

We generate some numerical solutions to our control problem using a fourth order Runge-Kutta scheme. This method is also tested for convergence. We iteratively solve for the state variables in the forward way given the initial values, and the adjoint variables in a backward way due to having terminal values. The method is the same as described in Lenhart and Workman (2007). A number of different numerical simulations are carried out for comparison in Figures 1, 2 and 3. The values of parameters used in the simulations are presented in Table 3 and some of these parameters are varied to test the response of the model. We illustrate the effects of public health education (α) on each of the classes. We also numerically solve for the optimal solution and test the effect of variation in the values of different parameters.

The values of the parameters M_0, M_1, M_2, M_3 are for public health management to decide (they are not dependent on the model). Here we choose a set of weight factors $M_1 = 920$, $M_2 = 25$, $M_3 = 80$ together with initial values (in millions) $S(0) = 0.5$, $I_1(0) = 0.7$, $I_2(0) = 0.6$, $A(0) = 0.06$.

5.1 The Optimal Solution Versus $\alpha = 0$.

Here the optimal effort on public health education (α^*) is compared with the case of $\alpha = 0$. In both cases the infected classes vanishes to zero. In Fig 1(a,b,c) we observe that the levels to which the ($\alpha = 0$)-curves have decreased at $t = 60$, will already have been attained at $t = 50$ in the case of $\alpha = \alpha^*$.

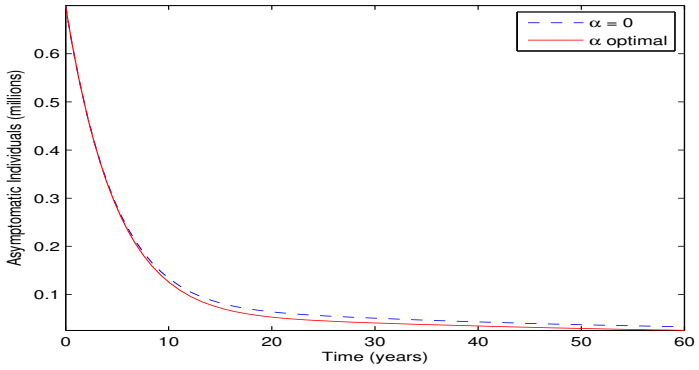
5.2 Effects of Infectives' Withdrawal From Sexual Activities.

In Fig 1(d) we observe the effect of infectives' withdrawal from sexual activity on each individual class by setting $q = 0.9$ and optimal control. Asymptomatic infectives I_1 can be seen to more or less vanishes at $t = 40$, symptomatic infectives I_2 first increases from initial 0.6 million to 0.7 million at the early stage but later decreases to zero at $t = 68$, while AIDS individuals A decreases to zero at $t = 70$.

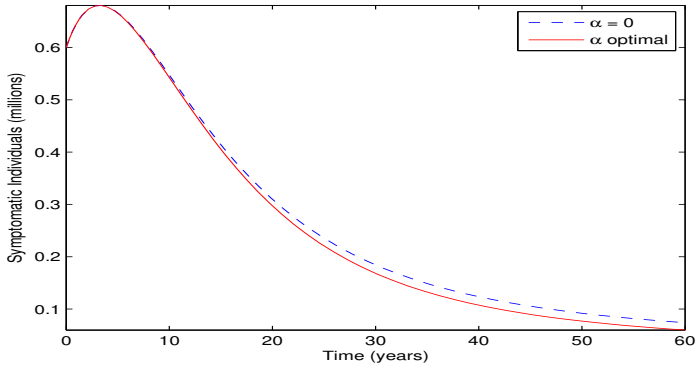
5.3 Effects of Infectives' Withdrawal From Sexual Activities on Asymptomatic Individuals

In Fig. 2, we use $\alpha = \alpha^*$ and observe the effect of infectives' self-withdrawal (q) from sexual activities on asymptomatic individuals. We first notice that if there is no withdrawal, i.e. when $q = 0$, then the number of asymptomatic infective individuals at time $t = 50$ is still at approximately 85000. For $q = 0.5$, the number of asymptomatic infective individuals decreases slightly faster, reaching approximately 75000 at $t = 50$ while the number gets much closer to vanishing at $t = 50$ when withdrawal is at the reasonably high level of $q = 0.9$. This implies that infectives' withdrawal from sexual activities has a serious impact in reducing the epidemic of HIV/AIDS.

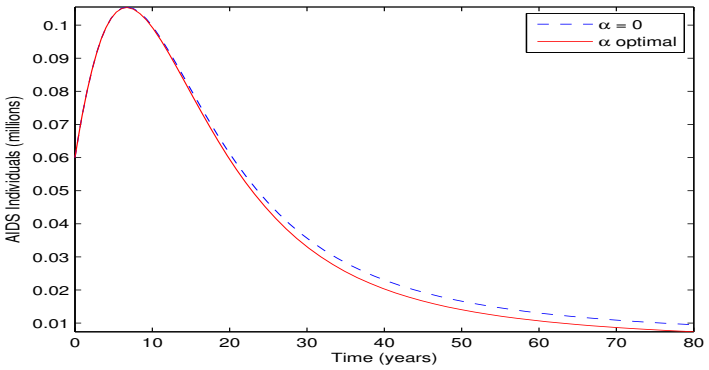
(a)



(b)



(c)



(d)

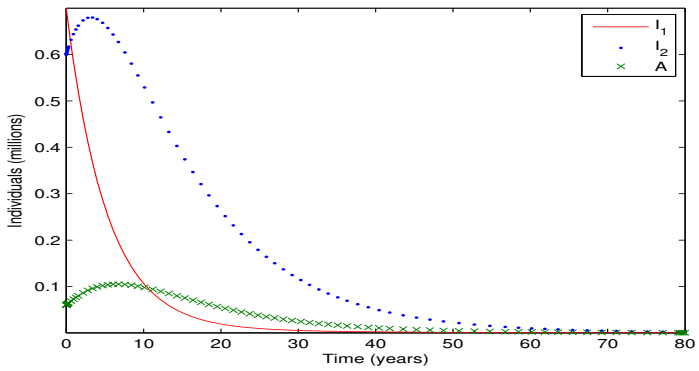


Figure 1: Simulations of the optimal solution.

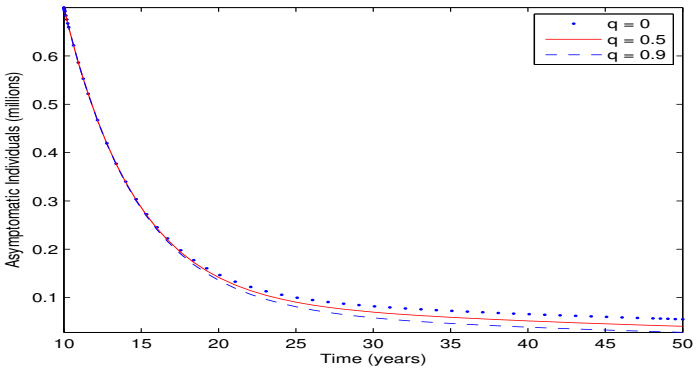


Figure 2: Simulations of the HIV/AIDS model showing the effect of withdrawing from sexual activities on asymptomatic individuals.

5.4 Effects of Transmission Probability and Partner Acquisition Rate on Asymptomatic Individuals

In these simulations again we use $\alpha = \alpha^*$. In Fig. 3(a), we show the effect of the transmission probability (β) on asymptomatic individuals. We observe that the higher the transmission probability, the longer the disease's prevalence in the population. Fig. 3(b) also explains that the disease persistence increases along with increase in partner acquisition rate.

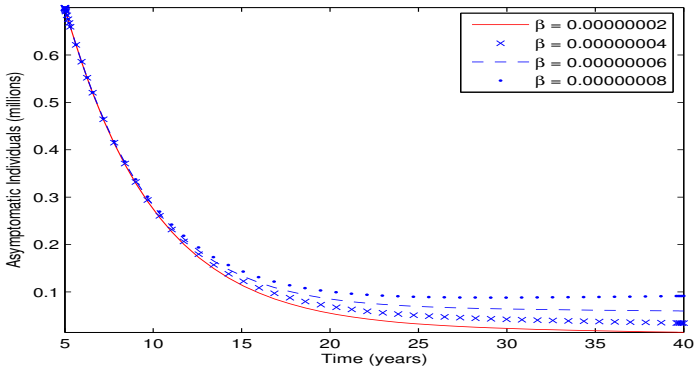
6 Conclusions

In this paper, we studied the control, stability and sensitivity of an HIV model with public health and infectives' withdrawal from sexual activity. We perform sensitivity analysis on the reproduction number and observed that the acquisition rate c and probability of transmission β are the most sensitive parameters. This analysis was also carried out on the endemic equilibrium point (when applicable) and the results show, for instance, that probability of transmission β is more sensitive on the A-class than on the I_1-class.

We observed the effect of public health campaigns and infectives withdrawal on the transmission of the disease by performing optimal control analysis on the model. Optimal control provides us with a means to calculate the levels of public health education effort, as a function of time, for the best results in controlling the spread of the disease. Our numerical results illustrate the responses of the model to variations in parameter values. So, for instance, we observed that infectives' self-withdrawal play a major role in reducing the HIV/AIDS scourge in a population.

In general this study significantly expands the work done in Nyabadza et al. (2010). In particular, it contributes to understanding HIV population dynamics, and informing optimal strategies for intervention with public health education. The model presented

(a)



(b)

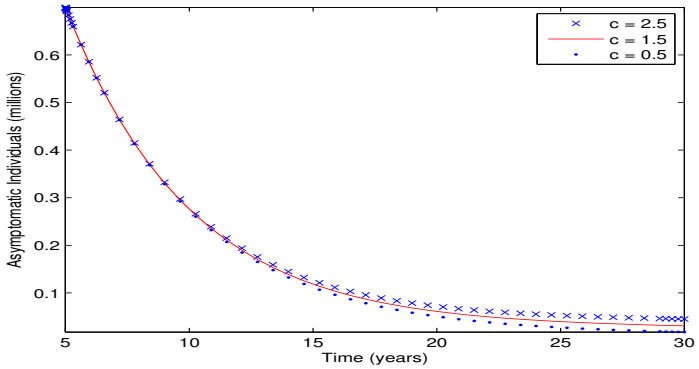


Figure 3: Simulations of the effects of transmission probability and partner acquisition rate on asymptomatic individuals

in this study did not consider the effects of therapeutic intervention strategies (such as the use of antiretroviral drugs) in the transmission dynamics of HIV/AIDS. Of course, there is a need to analyze models that are more complex, combining pharmaceutical treatment with preventive measures such as education and many other. The severity of the HIV problem necessitates the most serious global effort towards the control of the virus and protection of humans against it.

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