Mathematics analysis of the effect of public health educational campaigns, screening and therapy on HIV/AIDS transmission

Marsudi*, Trisilowati, A Suryanto and I Darti

Department of Mathematics, Brawijaya University, 65145 Malang, Indonesia

*Corresponding Author : marsudi61@ub.ac.id

Abstract. In this paper, a deterministic mathematical models are formulated and analyzed to assess the effect of the public health educational campaigns, screening, and therapy on the dynamics of HIV/AIDS. The information causes a change in behavior resulting in three susceptible classes. We calculate the effective reproduction number using the next-generation matrix method. It has been shown that the disease-free equilibrium point is locally asymptotically stable when the effective reproduction number is less than unity. Sensitivity analysis of effective reproduction number with respect to the model parameters were carried out. The most sensitive parameter on the effective reproduction number is the contact rate of susceptible to unaware HIV infective followed by the screening rate and the least sensitive parameter is the progression rate of screened infective to full-blown AIDS. Numerical simulations and sensitivity analysis are carried out to support the analytical results and to determine the parameters influencing the dynamics of the disease.

1. Introduction

HIV (Human Immunodeficiency Virus) is a virus that attacks and destroys the human immune system. The immune system is a body's defense system that naturally fights all kinds of infections and diseases. AIDS (Acquired Immune Deficiency Syndrome) is a condition in people with HIV who experience serious illness because their immune system can no longer function effectively against disease. AIDS sufferers lose so many white blood cells or CD4 + cells (Cluster Designation 4). If CD4 + cells are available ±350 cells/mm3 of blood, the body is not sufficiently protected so that the body loses endurance and is susceptible to various diseases including tuberculosis, diarrhoea, skin aches, and others.

Today, mathematical modelling for epidemiological problems has become very important in the management and control of an epidemic of infectious diseases such as HIV/AIDS. Mathematical models based on the mechanism of the spread of HIV/AIDS can help medical or scientists understand and anticipate the spread of the epidemic and evaluate the potential effectiveness of different approaches to keep the epidemic under control. Tripathi at al [1] have examined the effect of screening on unaware infective in the spread of HIV infection and Safiel et al [2] examined the effects of screening and treatment on transmission of HIV/AIDS infection in a population. Marsudi et al [3] present a mathematical model that refers to Safiel et al [2] with the assumption that only screened infective were treated to investigate the effects of screening and treatment of HIV on the HIV/AIDS infection dynamics in a population. This model does not consider (neglect) the progression of full blown AIDS to treated infections because it is assumed that transmission of HIV disease only through sexual contact (sexually transmitted diseases (STD)) and individuals in the full blown AIDS group

undergo treatment. Then the model presented by Marsudi et al [4] expand the model in [5] by examining the impact of condom education, screening and therapy on the spread of HIV infection. Marsudi et al [6] developed a mathematical model for assess effect of educational campaign on susceptible and antiretroviral therapy on pre-AIDS infections.

In this paper, the model in [4] is extended by developing susceptible individuals into two groups which refers to Joshi et al [7], namely groups that get an education and behave AB (Abstinence, Be faithful) or behave C (Condom) with different levels of infectivity.

2. Mathematical Model

In this section, we introduce a HIV/AIDS model with public health educational campaigns, screening, and therapy. The total population , denoted by *N,* was classified into seven disjoint subpopulations, namely, susceptible individuals ($S(t)$), educated susceptible and the group following abstinence and fathful or AB behavior group ($S_1(t)$), educated susceptible and the group using condom or C behavior group (S_2 (t)), unaware infectives (I_1 (t)), aware (screened) infectives (I_2 (t)), screened infectives receiving therapy ($T(t)$), and full-blown AIDS class ($A(t)$).

We assumed that transmission rate (λ) proportional to the susceptible, and the ratio between the number of I_1, I_2 and T and the total population; unaware infective, aware infectives, and treated infectives can infect susceptible at different rates β_1, β_2 dan β_3 respectively ($\beta_3 < \beta_2 < \beta_1$); unaware infectives can be screened infectives at rate θ; only screened infectives can be therapy infectives at rate δ; unaware infectives, screened infectives and treated infectives move to full-blown AIDS at different rates σ_1, σ_2 and σ_3 respectively ($\sigma_3 < \sigma_2 < \sigma_1$); the AIDS-related dead rate γ, the natural mortality rate μ and recruitment into susceptible at a rate Λ. Because of the interactions of individuals in class S with the education E, a proportion of the susceptibles leave S and move to S_1 and S_2 at the rate α_1 and α_2 is $\alpha_i ES_i, i = 1, 2.$ The public health educational campaigns at S_1 and S_2 has the effect of reducing the infection rate $(1-\psi_1)\lambda$ and $(1-\psi_1)\lambda$ respectively. Parameters ψ_i ($0 \le \psi_i \le 1, i = 1, 2$) measures the efficacy of public health educational campaigns.

The transfer diagram are shown in Figure 1.

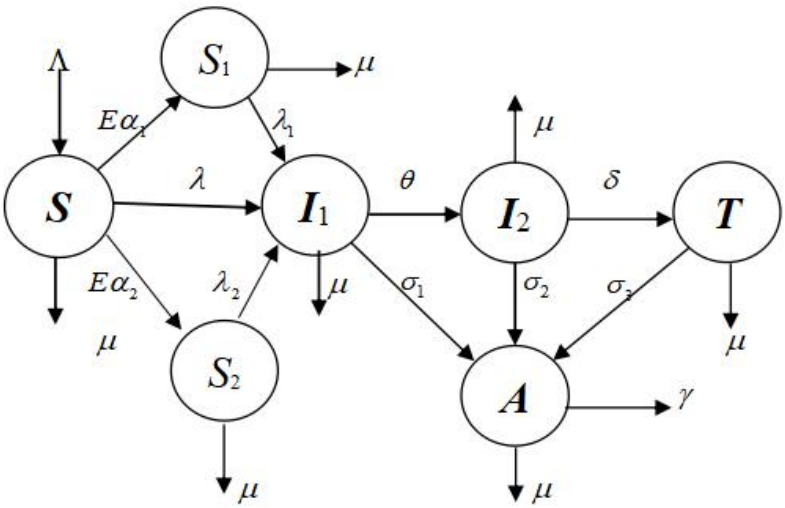

Figure 1. Transfer diagram of the model (1)

The transfer diagram leads to the following system of ordinary differential equations (1).

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \lambda S - E\alpha_1 S - E\alpha_2 S - \mu S \\
\frac{dS_1}{dt} &= E\alpha_1 S - \lambda_1 S_1 - \mu S_1 \\
\frac{dS_2}{dt} &= E\alpha_2 S - \lambda_2 S_2 - \mu S_2 \\
\frac{dI_1}{dt} &= \lambda S + \lambda_1 S_1 + \lambda_2 S_2 - (\theta + \sigma_1 + \mu) I_1 \\
\frac{dI_2}{dt} &= \theta I_1 - (\delta + \sigma_2 + \mu) I_2 \\
\frac{dT}{dt} &= \delta I_2 - (\sigma_3 + \mu) T \\
\frac{dA}{dt} &= \sigma_1 I_1 + \sigma_2 I_2 + \sigma_3 T - (\gamma + \mu) A
\end{aligned} \tag{1}$$

where $\lambda = \frac{\beta_1 c_1 I_1 + \beta_2 c_2 I_2 + \beta_3 c_3 T}{N}$, $\lambda_1 = (1-\psi_1)\lambda, \lambda_2 = (1-\psi_2)\lambda, N = S + S_1 + S_2 + I_1 + I_2 + T + A$ with initial conditions

$$S(0) = S_0, S_1(0) = S_{10}, S_2(0) = S_{20}, I_1(0) = I_{10}, I_2(0) = I_{20}, T(0) = T_0, A(0) = A_0. \tag{2}$$

3. Model Analysis

3.1. Invariant Region

Since the model system of equation (1) monitors changes in the human population, the variables and the parameters of model are assumed to be positive for all $t \geq 0$. The model will be analyzed in a suitable feasible region where all state variables are positive.

Lemma 1. The solutions of the system (1) are feasible for all $t \geq 0$ if they enter the invariant region Γ.

Proof. Let $\Omega = (S, S_1, S_2, I_1, I_2, T, A) \in R_+^7$ be a solution of the system (1) with non-negative initial conditions. The rate of change of the total populations is obtained by adding the equations of the system (1) to give

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t) - \gamma A \leq \Lambda - \mu N(t). \tag{3}$$

Using Birkhoff and Rota's theorem [8] on differential inequalities on (3) we obtain

$$N(t) \leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t}. \tag{4}$$

where $N(0)$ represents the initial values of the respective variables. As $t \to 0$ in (4), $N(t) \leq N(0)$ and as $t \to \infty$, $N(t) \leq \frac{\Lambda}{\mu}$ which implies that $0 \leq N(t) \leq \frac{\Lambda}{\mu}$. Thus, $\frac{\Lambda}{\mu}$ is an upper bound of N(t) provided $N(0) \leq \frac{\Lambda}{\mu}$. Hence, all feasible solution of the system (1) enters the region

$$\Gamma = \left\{ \Omega = (S, S_1, S_2, I_1, I_2, T, A) \in R_+^7 \mid S > 0, S_1 > 0, S_2 > 0, I_1 \geq 0, I_2 \geq 0, T \geq 0, A \geq 0, N \leq \frac{\Lambda}{\mu} \right\}. \quad (5)$$

In this case, whenever $N(t) > \frac{\Lambda}{\mu}$, then $\frac{dN(t)}{dt} < 0$ which means that the population decreases asymptotically to the carrying capacity and whenever $N(t) \leq \frac{\Lambda}{\mu}$, every solution with initial condition in Γ. Thus, the region is positively invariant (i.e. solutions remain positive for all times *t*). Thus, the system is biologically meaningful and mathematically well-posed in the domain of Γ. Hence, it is sufficient to study the dynamics of the basic model in Γ.

3.2. The Disease-free Equilibrium and *The Effective Reproductive Number*

In order to obtain the disease-free equilibrium point (DFE) of the model system (1) the right-hand sides of the model equations is set to zero. The disease-free equilibrium point are equalibrium state solutions where there is no disease (HIV/AIDS). The infective classes ($I_1, I_2, T,$ and A) are equal to zero. Thus, the disease-free equilibrium point of the basic model (1) is given

$$E_0 = (S^*, S_1^*, S_2^*, I_1^*, I_2^*, T^*, A^*) = \left(\frac{\Lambda}{E\alpha_1 + E\alpha_2 + \mu}, \frac{E\alpha_1 \Lambda}{\mu(E\alpha_1 + E\alpha_2 + \mu)}, \frac{E\alpha_2 \Lambda}{\mu(E\alpha_1 + E\alpha_2 + \mu)}, 0,0,0,0 \right) \quad (6)$$

The local stability of E_0 was established by using the next generation matrix method on the system (1). The second of equilibrium point of system (1) is the endemic equilibrium point, $E_1 = (S^{**}, S_1^{**}, S_2^{**}, I_1^{**}, I_2^{**}, T^{**}, A^{**})$ that depends on the force of infection λ and can be obtained if $I_1 \neq 0, I_2 \neq 0, T \neq 0, A \neq 0.$

The effective reproduction number R_e will be found by using the method of next generation matrix found in Van den Driessche [9]. Using the notations as in [9] for the system (1), let F_i is the rate of appearance of new infection in a compartment *i* and V_i is the transfer of individuals out of compartment *i* by any other means. The effective reproduction number R_e is the spectral radius (the largest eigen value) of the matrix FV^{-1},

$$F = \left[\frac{\partial F_i(E_0)}{\partial X_j} \right] = \begin{bmatrix} \frac{\beta_1 c_1 \mu + k_1 \beta_1 c_1 E\alpha_1 + k_2 \beta_1 c_1 E\alpha_2}{E\alpha_1 + E\alpha_2 + \mu} & \frac{\beta_2 c_2 \mu + k_1 \beta_2 c_2 E\alpha_1 + k_2 \beta_2 c_2 E\alpha_2}{E\alpha_1 + E\alpha_2 + \mu} & \frac{\beta_3 c_3 \mu + k_1 \beta_3 c_3 E\alpha_1 + k_2 \beta_3 c_3 E\alpha_2}{E\alpha_1 + E\alpha_2 + \mu} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad (7)$$

where $k_1 = (1 - \psi_1)$ and $k_2 = (1 - \psi_2)$,

$$V = \left[\frac{\partial V_i(E_0)}{\partial X_j} \right] = \begin{bmatrix} \theta + \sigma_1 + \mu & 0 & 0 & 0 \\ -\theta & \delta + \sigma_2 + \mu & 0 & 0 \\ 0 & -\delta & \sigma_3 + \mu & 0 \\ -\sigma_1 & -\sigma_2 & -\sigma_3 & \gamma + \mu \end{bmatrix}. \quad (8)$$

Hence, the effective reproduction number of the system (1) is given by

$$\begin{aligned} R_e = \rho(FV^{-1}) = & \frac{c_1\beta_1[(1-\psi_1)E\alpha_1 + (1-\psi_2)E\alpha_2 + \mu]}{(E\alpha_1 + E\alpha_2 + \mu)(\theta + \sigma_1 + \mu)} \\ & + \frac{c_2\beta_2\theta[(1-\psi_1)E\alpha_1 + (1-\psi_2)E\alpha_2 + \mu]}{(E\alpha_1 + E\alpha_2 + \mu)(\theta + \sigma_1 + \mu)(\delta + \sigma_2 + \mu)} \\ & + \frac{c_3\beta_3\theta\delta[(1-\psi_1)E\alpha_1 + (1-\psi_2)E\alpha_2 + \mu]}{(E\alpha_1 + E\alpha_2 + \mu)(\theta + \sigma_1 + \mu)(\delta + \sigma_2 + \mu)(\sigma_3 + \mu)}. \end{aligned} \tag{9}$$

The effective reproduction number R_e measures the average number of new infections caused by a single HIV infected individual in a population where education campaign, screening, and therapy are used to control strategies are in place.

Following Theorem 2 of [4], we have the following result on the local stability of E_0 of system (1).

Theorem 1. The disease-free equilibrium E_0 of the system (1) is locally asymptotically stable if $R_e < 1$ and unstable if $R_e > 1$.

Proof. The Jacobian matrix of the system (1) is calculated at the DFE is given by

$$J_0 = \begin{pmatrix} -(E\alpha_1 + E\alpha_2 + \mu) & 0 & 0 & -\frac{\beta_1 c_1 \mu}{E\alpha_1 + E\alpha_2 + \mu} & -\frac{\beta_2 c_2 \mu}{E\alpha_1 + E\alpha_2 + \mu} & -\frac{\beta_3 c_3 \mu}{E\alpha_1 + E\alpha_2 + \mu} & 0 \\ E\alpha_1 & -\mu & 0 & -\frac{k_1\beta_1 c_1 E\alpha_1}{E\alpha_1 + E\alpha_2 + \mu} & -\frac{k_1\beta_2 c_2 E\alpha_1}{E\alpha_1 + E\alpha_2 + \mu} & -\frac{k_1\beta_3 c_3 E\alpha_1}{E\alpha_1 + E\alpha_2 + \mu} & 0 \\ E\alpha_2 & 0 & -\mu & -\frac{k_2\beta_1 c_1 E\alpha_2}{E\alpha_1 + E\alpha_2 + \mu} & -\frac{k_2\beta_2 c_2 E\alpha_2}{E\alpha_1 + E\alpha_2 + \mu} & -\frac{k_2\beta_3 c_3 E\alpha_2}{E\alpha_1 + E\alpha_2 + \mu} & 0 \\ 0 & 0 & 0 & \frac{\beta_1 c_1 P}{E\alpha_1 + E\alpha_2 + \mu} - (\theta + \sigma_1 + \mu) & \frac{\beta_2 c_2 P}{E\alpha_1 + E\alpha_2 + \mu} & \frac{\beta_3 c_3 P}{E\alpha_1 + E\alpha_2 + \mu} & 0 \\ 0 & 0 & 0 & \theta & -(\delta + \sigma_2 + \mu) & 0 & 0 \\ 0 & 0 & 0 & 0 & \delta & -(\sigma_3 + \mu) & 0 \\ 0 & 0 & 0 & \sigma_1 & \sigma_2 & \sigma_3 & -(\gamma + \mu) \end{pmatrix}$$

The eigenvalues of the matrix J_0 are obtained by solving the characteristic equation $|\lambda I - J_0| = 0$ and obtained

$$(\lambda + Q)(\lambda + \mu)^2(\lambda + (\gamma + \mu)) \begin{vmatrix} \lambda - (\frac{\beta_1 c_1 P}{Q} - K) & -\frac{\beta_2 c_2 P}{Q} & -\frac{\beta_3 c_3 P}{Q} \\ -\theta & \lambda + L & 0 \\ 0 & -\delta & \lambda + M \end{vmatrix} = 0 \tag{10}$$

where $k_1 = 1 - \psi_1, k_2 = 1 - \psi_2, P = k_1 E\alpha_1 + k_1 E\alpha_1 + \mu, Q = E\alpha_1 + E\alpha_1 + \mu, K = \theta + \sigma_1 + \mu,$
$L = \delta + \sigma_2 + \mu, M = \sigma_3 + \mu.$

From equation (10) there are four root equations with negative values, i.e.

$$\lambda_1 = -Q, \lambda_2 = \lambda_3 = -\mu, \quad \lambda_4 = -(\gamma + \mu), \tag{11}$$

and eigen values obtained from polynomials

$$f(\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0 \tag{12}$$

where

$$a_1 = -\frac{\beta_1 c_1 P}{Q} + K + L + M,$$
$$a_2 = -\frac{\beta_1 c_1 P}{Q}(L+M) - \frac{\beta_2 c_2 \theta P}{Q} + KL + KM + LM$$
$$a_3 = -\frac{\beta_1 c_1 P}{Q}LM - \frac{\beta_2 c_2 \theta P}{Q}M - \frac{\beta_3 c_3 \delta\theta P}{Q} + KLM.$$

The root values of equation (12) can be solved using the Routh-Hurwitz criteria. According to the Routh-Hurwitz criteria, the necessary and sufficient condition for all eigenvalues of equation (12) has a negative real part is $a_1 > 0$, $a_3 > 0$, and $a_1 a_2 - a_3 > 0$.

Note that all parameters of the model are non-negative and from $R_e < 1$ caused

$$1 > \frac{\beta_1 c_1 P}{QK} + \frac{\beta_2 c_2 \theta P}{QKL} + \frac{\beta_3 c_3 \theta \delta P}{QKLM} > \frac{\beta_1 c_1 P}{QK}.$$

Then

$$a_1 = -\frac{\beta_1 c_1 P}{Q} + K + L + M = K\left(1 - \frac{\beta_1 c_1 P}{QK}\right) + L + M \quad > 0.$$

$$a_3 = -\frac{\beta_1 c_1 P}{Q}LM - \frac{\beta_2 c_2 \theta P}{Q}M - \frac{\beta_3 c_3 \delta\theta P}{Q} + KLM = KLM\left(1 - \frac{\beta_1 c_1 P}{QK} - \frac{\beta_2 c_2 \theta P}{QKL} - \frac{\beta_3 c_3 \delta\theta P}{QKLM}\right)$$
$$= KLM\,(1 - R_e) > 0 \quad \text{jika} \quad R_e < 1.$$

$$\begin{aligned} a_1 a_2 - a_3 &= LM^2 + L^2 M + KL^2\left(1 - \frac{\beta_1 c_1 P}{QK}\right) + KM^2\left(1 - \frac{\beta_1 c_1 P}{QK}\right) + K^2 L\left(1 - \frac{\beta_1 c_1 P}{QK}\right) \\ &\quad + K^2 M\left(1 - \frac{\beta_1 c_1 P}{QK}\right) + 2KLM\left(1 - \frac{\beta_1 c_1 P}{QK}\right) + \frac{\beta_3 c_3 \theta\,\delta P}{Q} + \frac{\beta_1 c_1 P^2 \beta_2 c_2 \theta}{Q^2} \\ &\quad + \frac{\beta_1^2 c_1^2 P^2 L}{Q^2} + \frac{\beta_1^2 c_1^2 P^2 M}{Q^2} - \frac{K\beta_1 c_1 PL}{Q} - \frac{K\beta_1 c_1 PM}{Q} \\ &> LM^2 + L^2 M + KL^2\left(1 - \frac{\beta_1 c_1 P}{QK}\right) + KM^2\left(1 - \frac{\beta_1 c_1 P}{QK}\right) + K^2 L\left(1 - \frac{\beta_1 c_1 P}{QK}\right) \\ &\quad + K^2 M\left(1 - \frac{\beta_1 c_1 P}{QK}\right) + 2KLM\left(1 - \frac{\beta_1 c_1 P}{QK}\right) + \frac{\beta_3 c_3 \theta\,\delta P}{Q} + \frac{\beta_1 c_1 P^2 \beta_2 c_2 \theta}{Q^2} \\ &\quad + \frac{\beta_1^2 c_1^2 P^2 L}{Q^2} + \frac{\beta_1^2 c_1^2 P^2 M}{Q^2} > 0. \end{aligned}$$

3.3. Sensitivity Analysis of model parameters

Sensitivity of each parameter in model is observed with respect to the effective reproduction number R_e. In this way, parameters that are more sensitive to the spread of infection will be known. Initial disease transmission is directly related to the effective reproduction number. In These indices tell us how crucial each parameter is to disease transmission and discover parameters that have a high impact on R_e that should be targeted by intervention strategies. The sensitivity index of the effective reproduction number R_e to the parameters in the model was calculated using the approach of [10]. The normalized forward sensitivity index of R_e with respect to parameter p is given by:

$$\mathbb{I}_p^{R_e} = \frac{\partial R_e}{\partial p} \times \frac{p}{R_e}. \tag{13}$$

Using the formula (13) and the parameter sets in Table 1, the sensitivity index of R_e with respect to parameters p are presented in Table 2.

Table 1. Parameters values used for sensitivity analysis

Parameter	Description	Values (year^{-1})	References
β_i , i = 1,2,3	Per capita contact rates for susceptible individuals with unaware infective, screened infective and treated individuals	0.2 0.005 0.001	Assumed Assumed Assumed
θ	Screening rate	0.6	Assumed
σ_i , i = 1,2,3	Progression rate from unaware infective, screened infective and therapy infective to full blown AIDS at different rates	0.2 0.01 0.001	[8] [8] [8]
μ	Natural death rate	0.01	Assumed
$\psi_i, i = 1,2$	The efficacy of public health educational measure	0.35; 0.45	Assumed
$c_i, i = 1, 2,3$	Average number of sexual partners per unit time for unaware infective, screened infective and treated individuals	3 2 1	Assumed [8] [8] [8]
E	Education rate	0.4	Assumed
$\alpha_i, i = 1,2$	Progression rate of from class S into class S_i	0.075 0.045	Assumed Assumed
δ	Treatment rate	0.99	Assumed
Λ	Recruitment rate	700	Assumed
γ	AIDS induced deaih rate	0.9	[8]

Table 2. Sensitivity indexes of the model parameters with respect to R_e

Parameter	Sensitivity Index
$\beta_1\ (c_1)$	+0.9099
θ	-0.8623
E	-0.0814
$\beta_3\ (c_3)$	+0.0811
ψ_1	-0.0811
ψ_2	-0.0626
α_2	-0.0591
σ_1	-0.0317
α_1	-0.0223
μ	-0.0091
$\beta_2\,(c_2)$	+0.0090
σ_3	-0.0074
δ	-0.0072
σ_2	-0.0009

Table 1 above consists of parameter values for the sensitivity analysis that is arranged from most sensitive to the least (in order of magnitude). The specific interpretation of each parameter from Table 1 shows that, the most sensitive parameter is the contact rate of susceptible to unaware HIV infective β_1 followed by the screening rate θ. Other important parameters include the education rate E and followed by the contact rate of susceptible with therapy infective β_3. Followed by the efficacy rate of education ψ_1 and than followed by the efficacy rate of education ψ_2. The sensitivity indexes for the other parameters are very small (- 0.0009 - 0.0090). the least sensitive parameter is the progression rate of screened infective to full-blown AIDS class σ_2. The sensitivity index of R_e with respect to the contact rate of susceptible to unaware HIV infective (β_1) is +0.9099 that means, increasing (or decreasing) the parameter value β_1 by 10% keeping other parameters constant, increases (or decreases) R_e by 9.099%, The sensitivity index of R_e with respect to the contact rate of susceptible to unaware HIV infective (β_1) is +0.7870 that means, increasing (or decreasing) the parameter value β_1 by 10% keeping other parameters constant, increases (or decreases) R_e by 7.87%. The sensitivity index of R_e with respect to the screening rate (θ) is -0.8623 that means, increasing (or decreasing) the parameter value the screening rate θ by 10% keeping other parameters constant, decreases (or increases) R_e by 8.623%.

4. Numerical Simulations

Simulated models (2) are carried out using the set of parameters values given in Table 1. Some parameter values were obtained from [2] and assumed. We simulate the model system by using ODE solver coded in Matlab program language by using the following initial conditions:

$$S(0)=20{,}000{,}000,\ S_1(0)=5{,}000{,}000,\ S_2(0)=50{,}000,\ I_1(0)=200{,}000,\ I_2(0)=25{,}000,\ T(0)=5{,}000 \text{ and } A(0)=2{,}000 \quad (14)$$

By using parameter values shown in Table 1 and equation (9) is obtained the effective reproduction numbers of the model system (2) is $R_e = 0.7110$.

4.1. Variation of a population under different screening rates

Figure 2 shows the variation of population of unaware infective, aware infective, treated and AIDS population for different values of θ. It is seen that as the screening rate (θ) increases, the number of screened and treated infectives increases rapidly after 5 years then reaches its equilibrium (Figure 2(b) and 2(c)). Figure 2(a) and 2(d) shows that the number of unaware infectives and the number of full-blown AIDS population initially decreases as θ increases and then starts to decreases reaches its equilibrium. Thus, the screening of unaware infective have the effect of reducing the transmission of HIV infections.

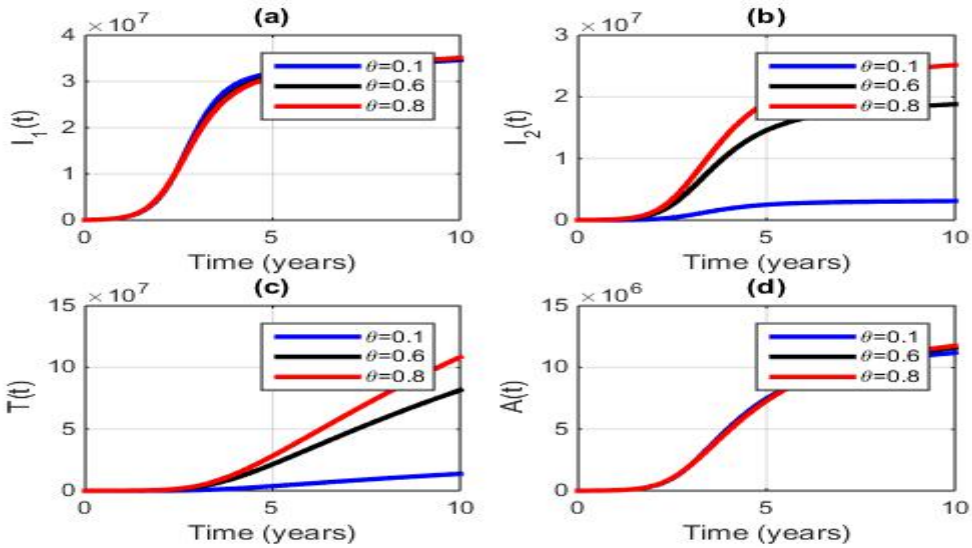

Figure 2. Variation of population for different values of θ

4.2. Variation of a population under different therapy rates

Figure 3 shows the variation of a population of infective individuals for different values of therapy rate (δ). We noted that in Figure 3(a), (b) and (d) the number of unaware, screened infectives and full-blown AIDS patient decreases when the value of δ increases. While, the number of treated population increases as δ increases (Figure 3(c)). This means the spread of HIV infection can only be reduced but not eliminated from the population. Thus, therapy programs at screened infective have the effect of reducing the transmission of HIV infections.

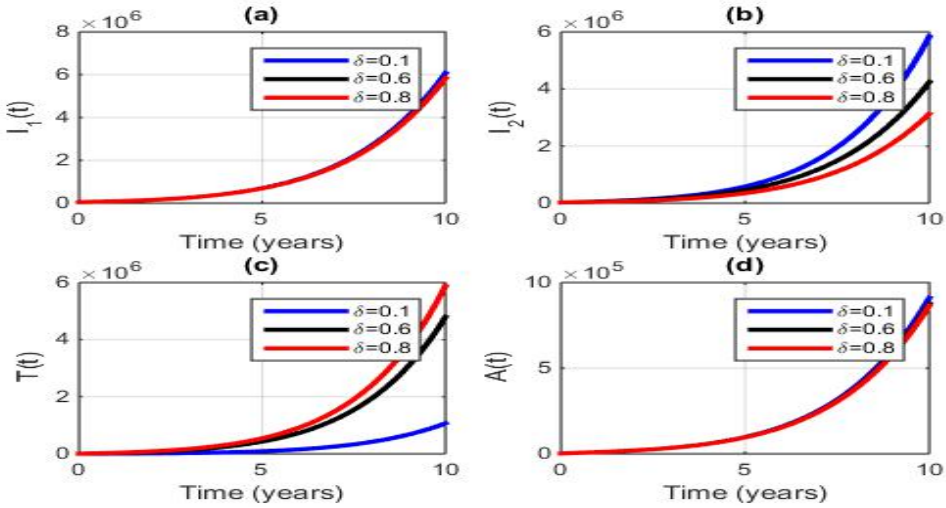

Figure 3. Variation of population for different values of δ

4.3. Variation of a population under public health educational campaigns rate

Figure 4 shows the variation of a population of infective population for different values of the public health education campaign (E). It is seen that as the education campaign (E) increases, the number of unaware infective, screened infective, treated infectives, and full-blown AIDS class initially decreases as E increases and then after about 6 years starts to increases reaches its equilibrium. This means the spread of HIV infection can only be reduced but not eliminated from the population. Thus, the public health educational campaigns programs at susceptible have the effect of reducing the transmission of HIV infections.

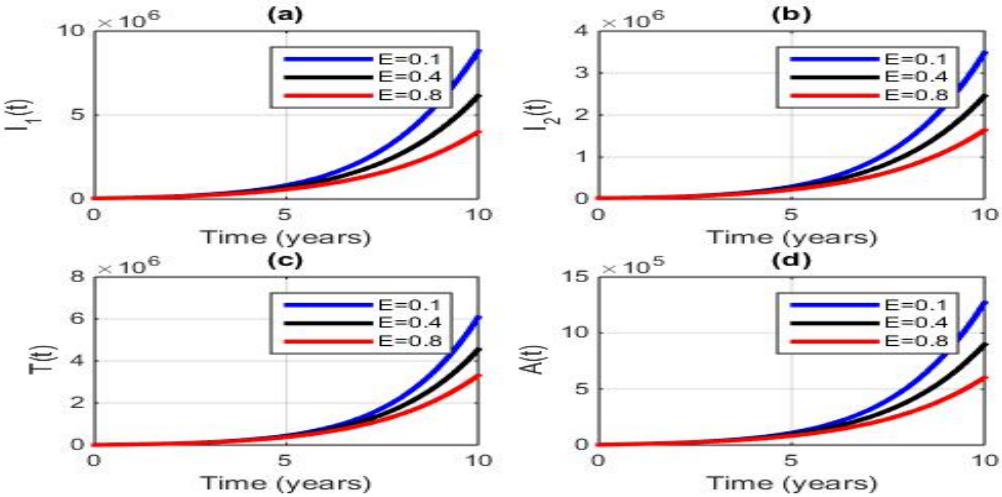

Figure 4. Variation of population for different values of E

5. Conclusion

We presented a deterministic model for assessing the effect of the education campaign on susceptible, screening on unaware infectives, and therapy on aware infectives in the spread of HIV in the population. The analysis shows that the education of susceptible, the screening of unaware HIV infective and therapy of screened HIV infective have the effect of reducing the transmission of the disease. A sensitivity analysis shows that the contact rate of susceptible to unaware HIV infective is the most sensitive parameter on the effective reproduction number R_e followed by the screening rate and the least sensitive parameter is the progression rate of screened infective to full-blown AIDS. It is observed that when education campaign, the screened infective, and therapy infective participate in the transmission of the infection, the AIDS population is significantly reduced in comparison to the case where there is no education campaign, screening and therapy.

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