Mathematical Analysis of a Model for the Transmission Dynamics of Trichomonas Vaginalis (TV) and HIV Co-infection

Salisu M. Garba[1] **and Chibale K. Mumba**

Department of Mathematics and Applied Mathematics, University of Pretoria, Pretoria 0002, South Africa.

Abstract

A deterministic model for the transmission dynamics of HIV and TV in a human population is designed and rigorously analysed. The model is shown to exhibit the phenomenon of backward bifurcation, where a stable disease-free equilibrium (DFE) co-exists with a stable endemic equilibrium whenever the associated reproduction number is less than unity. This phenomenon can be removed by assuming that the co-infection of individuals with HIV and TV is negligible. Furthermore, in the absence of co-infection, the DFE of the model is shown to be globally-asymptotically stable (GAS) whenever the associated reproduction number is less than unity. Numerical simulation of the model, using initial and demographic data, show that increased incidence of TV in a population increases HIV incidence in the population. It is further shown that control strategies, such as the treatment, condom-use and counselling of individuals with TV symptoms, can lead to the effective control or elimination of the HIV in the population if their effectiveness level is high enough. The time to disease elimination is reduced if more than one strategy (hybrid strategy) is considered.

Keywords: TV; HIV; equilibria; stability; reproduction number; backward bifurcation; control strategies.

1 Introduction

HIV/AIDS is one of the most severe health problems globally, with over 6.1 million cases (as of July 2015) [14]. In this study, we consider the co-infection of two sexually

[1]Corresponding author. Email: salisu.garba@up.ac.za

transmitted infection called Trichomonas Vaginalis (TV) and HIV. TV is an infection very common in both men and women, it is also the most prevalent non-viral STI globally [28] with more than 276 million people worldwide being annually affected [30]. Condoms are effective at reducing, but not fully preventing transmission of both HIV and TV. TV infection is treated and cured with metromidazole (7 days course) or tinidazole (2 days course) and can clear on its own after 3 months. HIV/AIDS on the other hand is treated using highly active antiretroviral therapy (HAART).
In women, TV is associated with vaginal itching, vaginal discharge, pain when urinating, e.t.c. [19]. Most males do not show any symptoms related to TV infection, although some experience swelling of the scrotum, urethral discharge and pain when urinating [19]. Further complications in women resulting from lack of treatment include pre-term delivery, low birth weight, and increased mortality, premature rapture of membranes, cervical cancer e.t.c. [8, 22, 26]. TV is of interest to this study because of its susceptibility to HIV, it increases the chances of an infected woman acquiring HIV if she has sexual contact with an infected individual [20, 21, 25, 29]. Also, a woman is more likely to transmit HIV to her sexual partners if she has TV [9, 15, 16, 22].
Although it is one of the most prevalent non-viral STI's, it receives the least public health attention [28]. Few papers have attempted a mathematical model on this topic [4, 7, 23]. TV requires more attention due to the damage it causes to the vaginal epithelium which increases a woman's susceptibility to HIV infection. In addition to inflammation, the parasite causes lysis of epithelial cells in the area, leading to more inflammation and disruption of the protective barrier usually provided by the epithelium.
The paper is organized as follows. The new model for the transmission dynamics of TV and HIV is formulated in Section 2. A sub-model of the full model is analysed for its dynamical properties in Section 3. Analysis of the full model is presented in Section 4. Numerical simulations as well as an assessment of control strategies of TV and HIV are presented in Section 5.

2 Model Formulation

A deterministic model for the transmission dynamics of HIV/AIDS and Trichomonas Vaginalis (TV) is considered and analysed. The total human population at time t given by $N(t)$ is divided into nine mutually exclusive compartments, namely, susceptible individuals ($S(t)$), newly infected individuals unaware of their TV status ($I_T^U(t)$), counselled individuals infected with TV ($I_T^C(t)$), infected individuals receiving treatment for TV ($T_T(t)$), individuals infected with HIV with no AIDS symptoms ($I_H(t)$), infected individuals with AIDS symptoms ($A_H(t)$), infected individuals (at both HIV and AIDS class) receiving treatment ($T_H(t)$), as well as individuals infected with both HIV and TV ($I_{TH}(t)$) and those with both infections who are receiving treatment for

TV ($T_{TH}(t)$), so that

$$N(t) = S(t) + I_T^U(t) + I_T^C(t) + T_T(t) + I_H(t) + A_H(t) + T_H(t) + I_{TH}(t) + T_{TH}(t).$$

The susceptible population is increased by the introduction of individuals into the sexually active population at a rate Π. These individuals either acquire TV or HIV infection, following effective contact with an infected individual in the $I_T^U(t)$, $I_T^C(t)$ and/or $T_T(t)$ class, or the $I_H(t)$, $A_H(t)$ and $T_H(t)$ classes at a rate λ_T^1 or λ_H, respectively. Therefore, the force of infection is given by

$$\lambda_1 = \lambda_T^1 + \lambda_H$$

where,

$$\lambda_T^1 = \frac{\beta_T(1-\epsilon\kappa)(I_T^U + \eta_1 I_T^C + \eta_2 T_T)}{N} \text{ and } \lambda_H = \frac{\beta_H(I_H + \eta_A A_H + \eta_T T_H)}{N}.$$

The parameters β_T and β_H are the effective contact rates (contact capable of leading to infection) for TV and HIV, respectively. The parameter $0 < \epsilon < 1$ is the condom efficacy, and $0 \leq \kappa < 1$ measures compliance in condom use. η_1 and η_2 are modification parameters accounting for the reduction in the transmissibility of counselled (I_T^C) and treated individuals (T_T), in relation to infected non-counselled individuals (I_T^U). Since treatment reduces infectiousness of treated individuals, it is plausible to set $0 < \eta_2 < 1$. Similarly it is assumed that counselled individuals modify their risky sexual behaviour positively, so that $0 < \eta_1 < 1$. The modification parameters $0 < \eta_A < 1$ and $0 < \eta_T < 1$ account for the relative risk of infectiousness of individuals with AIDS symptoms and treated individuals in comparison to those in I_H class, respectively. It is assumed that individuals in A_H and T_H classes are less infectious than those in I_H class because they change/reduce their risky sexual behaviour as they are sick and aware of their HIV status.

The population of susceptible individuals is further decreased by natural death (at a rate μ). It is assumed that natural deaths occur in all human compartments at the rate μ. The susceptible class is increased by the recovery of individuals in T_T class (at a rate ν). Thus the rate of change of the susceptible population is given by

$$\frac{dS}{dt} = \Pi + \nu T_T - (\lambda_H + \lambda_T)S - \mu S.$$

The population of infected individuals unaware of their TV status (non-councelled) $I_T^U(t)$ is generated by the infection of susceptible individuals (at the rate λ_T). This population is decreased by the counselling of infected individuals (at a rate ξ), infection with HIV (at the rate $\sigma\lambda_H$) and by natural death, so that

$$\frac{dI_T^U}{dt} = \lambda_T^1 S - \sigma\lambda_H I_T^U - (\xi + \mu)I_T^U.$$

The population of counselled infected individuals ($I_T^C(t)$) is increased by the counselling of individuals infected with TV (at the rate ξ). This population is decreased by treatment of infected counselled individuals (at a rate τ) and due to natural death, so that

$$\frac{dI_T^C}{dt} = \xi I_T^U - (\tau + \mu) I_T^C.$$

The population of treated individuals is increased by the treatment of counselled TV infected individuals (at the rate τ). The population is decreased by recovery (at the rate ν) and natural death. The recovered individuals return to the susceptible class. Thus

$$\frac{dT_T}{dt} = \tau I_T^C - (\nu + \mu) T_T.$$

Similarly, the population of individuals infected with HIV is generated by the infection of susceptible individuals (at the rate λ_H) and by recovery of individuals from TV in the T_{TH} class (at a rate ψ). This population is decreased due to the progression of HIV infected individuals to AIDS class (A_H) (at a rate α), treatment (at a rate θ) and natural death. This gives

$$\frac{dI_H}{dt} = \lambda_H S + \psi T_{TH} - (\alpha + \theta + \mu) I_H.$$

Individuals infected with HIV progress to AIDS class (A_H) (at the rate α). The population of individuals in A_H class reduces due to treatment (at a rate γ), natural death and disease-induced death (at a rate δ_1). So that

$$\frac{dA_H}{dt} = \alpha I_H - (\gamma + \mu + \delta_1) A_H.$$

The population of treated individuals (T_H) is increased by the treatment of individuals infected with HIV in the I_H and A_H classes (at the rates θ and γ), respectively. This population is decreased due to natural death and disease-induced death (at a rate δ_2). It is assumed that individuals infected with HIV do not fully recover. Thus,

$$\frac{dT_H}{dt} = \theta I_H + \gamma A_H - (\mu + \delta_2) T_H.$$

The populations of individuals infected with both TV and HIV is generated by co-infection of TV infected individuals with HIV (at the rate $\sigma\lambda_H$, where σ is the modification parameter that accounts for the increase in susceptibility to HIV of TV infected individual i.e $\sigma > 1$). It is reduced by treatment (at the rate τ) and natural death. So that

$$\frac{dI_{TH}}{dt} = \sigma\lambda_H I_T^U - (\tau + \mu) I_{TH}.$$

Finally, the population of treated individuals infected with TV and HIV is increased by the treatment of individuals in I_{TH} class (at the rate τ) and decreases due to recovery

from TV (and move to I_H class at the rate ψ) and natural death. This gives

$$\frac{dT_{TH}}{dt} = \tau I_{TH} - (\psi + \mu)T_{TH}.$$

Combining the aforementioned assumptions and derivations, it follows that the model for the transmission dynamics of TV and HIV co-infection is given by the following system of non-linear differential equations. (A flow chart for the model is illustrated in Figure 1 and a description of the parameters and variables is given in Tables 7 and 8).

$$\begin{aligned}
\frac{dS}{dt} &= \Pi + \nu T_T - (\lambda_H + \lambda_T^1)S - \mu S, \\
\frac{dI_T^U}{dt} &= \lambda_T^1 S - \sigma\lambda_H I_T^U - (\xi + \mu)I_T^U, \\
\frac{dI_T^C}{dt} &= \xi I_T^U - (\tau + \mu)I_T^C, \\
\frac{dT_T}{dt} &= \tau I_T^C - (\nu + \mu)T_T, \\
\frac{dI_H}{dt} &= \lambda_H S + \psi T_{TH} - (\alpha + \theta + \mu)I_H, \\
\frac{dA_H}{dt} &= \alpha I_H - (\gamma + \mu + \delta_1)A_H, \\
\frac{dT_H}{dt} &= \theta I_H + \gamma A_H - (\mu + \delta_2)T_H, \\
\frac{dI_{TH}}{dt} &= \sigma\lambda_H I_T^U - (\tau + \mu)I_{TH}, \\
\frac{dT_{TH}}{dt} &= \tau I_{TH} - (\psi + \mu)T_{TH}.
\end{aligned} \tag{1}$$

The Model (1) extends the model for the transmission dynamics of TV and HIV in [4], and is to the author's knowledge is the first to incorporate control strategies of TV and HIV co-infection in a population. In addition, it extends numerous TV models in the literature such as those in [3, 5] by (*inter alia*):

1. Allowing for TV transmission by treated individuals ($\eta \neq 0$), this is not considered in [4],

2. Sub-dividing the infected population with TV into counselled and non-counselled individuals, this is not considered in [4],

3. Assessing various control strategies for TV (counselling, treatment and condom use).

2.1 Qualitative Properties of the Model

The model (1) will now be rigorously analysed for it's dynamical features. This model monitors human population, therefore it is important that all the variables and parameters of the model are non-negative. This is proved below.

2.1.1 Positivity of solutions

Lemma 1 *Let the initial data* $S(0) > 0$, $I_T^U(0) > 0$, $I_T^C(0) > 0$, $T_T(0) > 0$, $I_H(0) > 0$, $A_H(0) > 0$, $T_H(0) > 0$, $I_{TH}(0) > 0$ *and* $T_{TH}(0) > 0$ *then the solutions* $S(t), I_T^U(t), I_T^C(t), T_T(t), I_H(t), A_H(t), T_H(t), I_{TH}(t), T_{TH}(t)$ *of the model* (1) *are positive for all* $t \geq 0$.

Proof. Suppose $S(t)$ is not positive, then there exists a first time, say $t^* > 0$, such that $S(t) > 0$ for $t \in [0, t^*)$ and $S(t^*) = 0$. By inspection of the equation of $I_T^U(t)$, we obtain that

$$\frac{dI_T^U(t)}{dt} \geq -(\sigma\lambda_H + \xi + \mu)I_T^U(t), \text{ for } t \in [0, t^*),$$

from which one can deduce that $I_T^U(t) > 0$ for $t \in [0, t^*)$. Thus it is clear from equation (1) that

$$\frac{dS(t)}{dt} \geq -(\lambda_T^1 + \lambda_H + \mu)S(t), \text{ for } t \in [0, t^*).$$

It follows that $S(t^*) > 0$, which contradicts that $S(t^*) = 0$. Therefore $S(t)$ is positive. Using a similar approach as that for $S(t)$ it is easy to show that $I_T^U(t) > 0$, $I_T^C(t) > 0$, $T_T(t) > 0$, $I_H(t) > 0$, $A_H(t) > 0$, $T_H(t) > 0$, $I_{TH}(t) > 0$ and $T_{TH}(t) > 0$ for all $t \geq 0$. ■

2.1.2 Feasible solution

Lemma 2 *: The biologically-feasible region given by*

$$\mathcal{D} = \left\{(S, I_T^U, I_T^C, T_T, I_H, A_H, T_H, I_{TH}, T_{TH}) \in \mathbf{R}_+^9 : S + I_T^U + I_T^C + T_T + I_H + A_H + T_H + I_{TH} + T_{TH} \leq \frac{\Pi}{\mu}\right\}$$

is positively-invariant.

Proof Adding all the differential equations in the model (1) gives

$$\frac{dN(t)}{dt} = \Pi - \mu N(t) - \delta_1 A_H(t) - \delta_2 T_H(t).$$

Thus

$$\frac{dN(t)}{dt} \leq \Pi - \mu N(t), \tag{2}$$

and so $\frac{dN(t)}{dt} < 0$ if $N(t) > \frac{\Pi}{\mu}$. It follows from (2), and Gronwall's inequality that

$$N(t) \leq N(0)e^{-\mu t} + \tfrac{\Pi}{\mu}(1 - e^{-\mu t}).$$

Hence, $N(t) \leq \frac{\Pi}{\mu}$ if $N(0) \leq \frac{\Pi}{\mu}$. Therefore, the region $\mathcal{D}$ is positively-invariant. Thus, in the region $\mathcal{D}$ the model (1) is well-posed epidemiologically and mathematically and it is sufficient to consider the dynamics of the flow generated by (1) in $\mathcal{D}$.

3 Analysis of the Sub-Model

In this section we consider a sub-model of Model (1) where we have one compartment for individuals infected with TV (obtained by combining the non-counselled and counselled individuals into one compartment) and in the absence of condom use (i.e. condom compliance $\kappa = 0$) given by the following deterministic non-linear differential equations

$$\begin{aligned}
\frac{dS}{dt} &= \Pi + \nu T_T - (\lambda_H + \lambda_T)S - \mu S,\\
\frac{dI_T}{dt} &= \lambda_T S - \sigma\lambda_H I_T - (\tau + \mu)I_T,\\
\frac{dT_T}{dt} &= \tau I_T - (\nu + \mu)T_T,\\
\frac{dI_H}{dt} &= \lambda_H S + \psi T_{TH} - (\alpha + \theta + \mu)I_H,\\
\frac{dA_H}{dt} &= \alpha I_H - (\gamma + \mu + \delta_1)A_H,\\
\frac{dT_H}{dt} &= \theta I_H + \gamma A_H - (\mu + \delta_2)T_H,\\
\frac{dI_{TH}}{dt} &= \sigma\lambda_H I_T - (\tau + \mu)I_{TH},\\
\frac{dT_{TH}}{dt} &= \tau I_{TH} - (\psi + \mu)T_{TH}.
\end{aligned} \tag{3}$$

where

$$\lambda_T = \frac{\beta_T(I_T + \eta T_T)}{N} \text{ and } \lambda_H = \frac{\beta_H(I_H + \eta_A A_H + \eta_T T_H)}{N},$$

and now

$$N(t) = S(t) + I_T(t) + T_T(t) + I_H(t) + A_H(t) + T_H(t) + I_{TH}(t) + T_{TH}(t).$$

3.1 Local Asymptotic Stability of the Disease-free Equilibrium (DFE)

The DFE of the model (3) is given by

$$\mathcal{E}_1 = (S^*, I_T^*, T_T^*, I_H^*, A_H^*, T_H^*, I_{TH}^*, T_{TH}^*) = \left(\frac{\Pi}{\mu}, 0, 0, 0, 0, 0, 0, 0\right).$$

The linear stability of the DFE, $\mathcal{E}_1$, can be established using the next generation operator method (Diekmann et al., 1990 [10]; van den Driessche and Watmough, 2002 [27]) on system (3). 2The basic reproduction number, denoted by $\mathcal{R}_0$ is then given by

$$\mathcal{R}_0 = \rho(FV^{-1}) = \max\{\mathcal{R}_1, \mathcal{R}_2\},$$

where $\mathcal{R}_1$ and $\mathcal{R}_2$ are the reproduction numbers associated with TV and HIV/AIDS, respectively, given by

$$\mathcal{R}_1 = \frac{\beta_T(Q_2 + \tau\eta)}{Q_1Q_2} \text{ and } \mathcal{R}_2 = \frac{\beta_H[Q_4(Q_5 + \theta\eta_T) + \alpha(\eta_A Q_5 + \gamma\eta_T)]}{Q_3Q_4Q_5}, \tag{4}$$

with $Q_1 = \tau + \mu,\ Q_2 = \nu + \mu,\ Q_3 = \theta + \alpha + \mu,\ Q_4 = \gamma + \mu + \delta_1, Q_5 = \mu + \delta_2$, and $Q_6 = \psi + \mu$. Thus, applying Theorem 2 of [27], the following result is established.

Lemma 3 *The DFE, $\mathcal{E}_1$, of the model (3) is LAS if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

The threshold quantity, $\mathcal{R}_0 = \max\{\mathcal{R}_1, \mathcal{R}_2\}$, is the basic reproduction number [1, 2, 13, 27]. It represents the average number of secondary cases generated by a typically TV or HIV infected individual in a completely susceptible population. The epidemiological implication of Lemma 3 is that when $\mathcal{R}_0$ is less than unity, a small influx of infected individuals into the community would not generate large outbreaks, and the disease dies out in time (since the DFE is LAS). However, we show in the next subsection that the disease may still persist even when $\mathcal{R}_0 < 1$ (this is owing to the existence of backward bifurcation).

3.2 Backward Bifurcation

Theorem 1 *The TV-HIV model (3) exhibits backward bifurcation whenever the coefficient a, given by (A.2) (in Appendix A) is positive.*

Proof. The proof, based on using the Center Manifold theorem [6], is given in Appendix A. ■

3.2.1 Non-existence of backward bifurcation for special case

The analysis in this section show that the backward bifurcation property of the TV-HIV model (3) is caused by the co-infection of HIV with TV in humans. Notice that, in the absence of co-infection (that is for the case when $\sigma = 0$), the bifurcation parameter $a < 0$. This rules out the existence of backward bifurcation when $\sigma = 0$. To further rule out the existence of backward bifurcation, a global stability analysis of the DFE

when $\sigma = 0$ is presented below.

First of all notice that by setting $\sigma = 0$ in (3) the equation of $\frac{dI_{TH}}{dt} \to 0$ as $t \to \infty$, thus $\frac{dT_{TH}}{dt} \to 0$ as $t \to \infty$. Hence, it follows that the system (3) is now reduced to

$$\begin{aligned}
\frac{dS}{dt} &= \Pi + \nu T_T - (\lambda_H + \lambda_T)S - \mu S, \\
\frac{dI_T}{dt} &= \lambda_T S - (\tau + \mu)I_T, \\
\frac{dT_T}{dt} &= \tau I_T - (\nu + \mu)T_T, \\
\frac{dI_H}{dt} &= \lambda_H S - (\alpha + \theta + \mu)I_H, \\
\frac{dA_H}{dt} &= \alpha I_H - (\gamma + \mu + \delta_1)A_H, \\
\frac{dT_H}{dt} &= \theta I_H + \gamma A_H - (\mu + \delta_2)T_H.
\end{aligned} \tag{5}$$

Next define the invariant region

$$\tilde{\mathcal{D}} = \{(S, I_T, T_T, I_H, A_H, T_H) \in \mathcal{D} : S \leq S^*\}.$$

We claim the following result.

Theorem 2 *The DFE, $\mathcal{E}_1$, of the model (3) with $\sigma = 0$ (or equivalently model(5)), is globally-asymptotically stable (GAS) in $\tilde{\mathcal{D}}$ whenever $\mathcal{R}_0 \leq 1$ and unstable if $\mathcal{R}_0 > 1$.*

Proof. Consider the following Lyapunov function

$$\mathcal{F} = \mathcal{R}_1 I_T + \frac{\beta_T \eta}{Q_2} T_T + \mathcal{R}_2 I_H + \frac{\beta_H(\eta_A Q_5 + \eta_T \gamma)}{Q_4 Q_5} A_H + \frac{\beta_H \eta_T}{Q_5} T_H$$

with Lyapunov derivative given by

$$\dot{\mathcal{F}} = \mathcal{R}_1\dot{I}_T + \frac{\beta_T\eta}{Q_2}\dot{T}_T + \mathcal{R}_2\dot{I}_H + \frac{\beta_H(\eta_A Q_5 + \eta_T\gamma)}{Q_4Q_5}\dot{A}_H + \frac{\beta_H\eta_T}{Q_5}\dot{T}_H,$$

$$= \mathcal{R}_1(\lambda_T S - Q_1 I_T) + \frac{\beta_T\eta}{Q_2}(\tau I_T - Q_2 T_T) + \mathcal{R}_2[\lambda_H S - Q_3 I_H]$$

$$+ \frac{\beta_H(\eta_A Q_5 + \eta_T\gamma)}{Q_4Q_5}[\alpha I_H - Q_4 A_H] + \frac{\beta_H\eta_T}{Q_5}[\theta I_H + \gamma A_H - Q_5 T_H],$$

$$= \mathcal{R}_1\lambda_T S - \beta_T(I_T + \eta T_T) + \mathcal{R}_2\lambda_H S - \beta_H(I_H + \eta_A A_H + \eta_T T_H), \tag{6}$$

$$= \mathcal{R}_1\lambda_T S - \lambda_T N + \mathcal{R}_2\lambda_H S - \lambda_H N,$$

$$= \lambda_T N\left[\frac{\mathcal{R}_1 S}{N} - 1\right] + \lambda_H N\left[\frac{\mathcal{R}_2 S}{N} - 1\right],$$

$$\leq \lambda_T N^*[\mathcal{R}_1 - 1] + \lambda_H N^*[\mathcal{R}_2 - 1], \quad \text{since } S \leq S^* \text{ in } \tilde{\mathcal{D}} \text{ and } N \leq N^*,$$

$$= \beta_T(I_T + \eta T_T)[\mathcal{R}_1 - 1] + \beta_H(I_H + \eta_A A_H + \eta_T T_H)[\mathcal{R}_2 - 1] \leq 0 \quad \text{when} \quad \mathcal{R}_0 \leq 1.$$

Since all the parameters and variables of the Model (5) are non-negative (Lemma 1), it follows that $\dot{\mathcal{F}} \leq 0$ for $\mathcal{R}_0 \leq 1$ (i.e. $\mathcal{R}_1 \leq 1$ and $\mathcal{R}_2 \leq 1$) with $\dot{\mathcal{F}} = 0$ if and only if $I_T = T_T = I_H = A_H = T_H = 0$. Hence $\dot{\mathcal{F}}$ is a Lyapunov function on $\tilde{\mathcal{D}}$. Thus, it follows by LaSalle's Invariance Principle [17], that $(I_T(t), T_T(t), I_H(t), A_H(t), T_H(t)) \to (0, 0, 0, 0, 0)$ as $t \to \infty$. Thus, it follows that every solution of the equations of the Model (5) with initial conditions in $\tilde{\mathcal{D}}$ approaches $\mathcal{E}_1$ as $t \to \infty$ (whenever $\mathcal{R}_0 \leq 1$). ■

The above result shows that in the absence of co-infection, the DFE of the model (3) is GAS. Therefore, HIV will be effectively controlled or eliminated from the population if $\mathcal{R}_0 \leq 1$. This result is depicted in Figures 2A and 2B, showing convergence to the DFE whenever $\mathcal{R}_0 \leq 1$.

3.2.2 Existence and stability analysis of endemic equilibrium

Existence We find an equilibrium where at least one of the infected components (I_T^{**}, T_T^{**}, I_H^{**}, A_H^{**}, T_H^{**}, I_{TH}^{**} and T_{TH}^{**}) is non zero. Let the EEP of the model (3) be denoted by

$$\mathcal{E}_{TH} = (S^{**}, I_T^{**}, T_T^{**}, I_H^{**}, A_H^{**}, T_H^{**}, I_{TH}^{**}, T_{TH}^{**}).$$

Solving the equations in (3) at endemic equilibrium point, in terms of the force of infection, by setting the right hand sides of the equations in (3) to zero, gives

$$S^{**} = \frac{Q_2(Q_1+\sigma\lambda_H^{**})\Pi}{\mu Q_1Q_2+\mu(\nu+Q_1)\lambda_T^{**}+Q_2[Q_1+\sigma(\lambda_T^{**}+\lambda_H^{**}+\mu)]\lambda_H^{**}},$$

$$I_T^{**} = \frac{Q_2\lambda_T^{**}\Pi}{\mu Q_1Q_2+\mu(\nu+Q_1)\lambda_T^{**}+Q_2[Q_1+\sigma(\lambda_T^{**}+\lambda_H^{**}+\mu)]\lambda_H^{**}},$$

$$T_T^{**} = \frac{\tau\lambda_T^{**}\Pi}{\mu Q_1Q_2+\mu(\nu+Q_1)\lambda_T^{**}+Q_2[Q_1+\sigma(\lambda_T^{**}+\lambda_H^{**}+\mu)]\lambda_H^{**}},$$

$$I_H^{**} = \frac{Q_2\lambda_H^{**}\Pi(Q_1^2Q_6+\psi\tau\sigma\lambda_T^{**}+Q_1Q_6\sigma\lambda_H^{**})}{Q_1Q_3Q_6[\mu Q_1Q_2+\mu(\nu+Q_1)\lambda_T^{**}+Q_2[Q_1+\sigma(\lambda_T^{**}+\lambda_H^{**}+\mu)]\lambda_H^{**}]},$$

$$A_H^{**} = \frac{\alpha\lambda_H^{**}\Pi(Q_1^2Q_6+\psi\tau\sigma\lambda_T^{**}+Q_1Q_6\sigma\lambda_H^{**})}{Q_1Q_3Q_4Q_6[\mu Q_1Q_2+\mu(\nu+Q_1)\lambda_T^{**}+Q_2[Q_1+\sigma(\lambda_T^{**}+\lambda_H^{**}+\mu)]\lambda_H^{**}]},$$

$$T_H^{**} = \frac{Q_2\lambda_H^{**}\Pi(\gamma\alpha+\theta Q_4)(Q_1^2Q_6+\psi\tau\sigma\lambda_T^{**}+Q_1Q_6\sigma\lambda_H^{**})}{Q_1Q_3Q_4Q_5Q_6[\mu Q_1Q_2+\mu(\nu+Q_1)\lambda_T^{**}+Q_2[Q_1+\sigma(\lambda_T^{**}+\lambda_H^{**}+\mu)]\lambda_H^{**}]},$$

$$I_{TH}^{**} = \frac{\sigma Q_2\lambda_T^{**}\lambda_H^{**}\Pi}{Q_1[\mu Q_1Q_2+\mu(\nu+Q_1)\lambda_T^{**}+Q_2[Q_1+\sigma(\lambda_T^{**}+\lambda_H^{**}+\mu)]\lambda_H^{**}]},$$

$$T_{TH}^{**} = \frac{\tau\sigma Q_2\lambda_T^{**}\lambda_H^{**}\Pi}{Q_1Q_6[\mu Q_1Q_2+\mu(\nu+Q_1)\lambda_T^{**}+Q_2[Q_1+\sigma(\lambda_T^{**}+\lambda_H^{**}+\mu)]\lambda_H^{**}]}. \tag{7}$$

Theorem 3 *The EEP of the Model (3), is GAS whenever* $\mathcal{R}_0 > 1$ *and* $\eta_T = \nu = \sigma = 0$.

The proof, based on using a non-linear Lyapunov function of Goh-Volterra type, is presented in Appendix B. Functions of such type have been used in mathematical epidemiology/ecology literature, see for instance [11, 12]. Simulations of the model showing convergence to EEP when $\mathcal{R}_0 > 1$ is depicted in Figures 3A and 3B.

4 Analysis of the Full-Model

The model (1) has a unique DFE obtained by setting the right-hand sides of the equations in the model (1) to zero, given by

$$\mathcal{E}_2 = (S^*, I_T^{U*}, I_T^{C*}, T_T^*, I_H^*, A_H^*, T_H^*, I_{TH}^*, T_{TH}^*) = \left(\frac{\Pi}{\mu}, 0, 0, 0, 0, 0, 0, 0, 0\right).$$

The linear stability of $\mathcal{E}_2$ can be established using the next generation operator method on system (1). Using the notation in [27], the matrices F (for the new infection terms) and V (for the transition terms) are given, respectively, by

$$F = \begin{pmatrix} \beta_T(1-\epsilon\kappa) & \beta_T\eta_1(1-\epsilon\kappa) & \beta_T\eta_2(1-\epsilon\kappa) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta_H & \beta_H\eta_A & \beta_H\eta_T & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} K_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\xi & K_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\tau & K_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & K_4 & 0 & 0 & 0 & -\psi \\ 0 & 0 & 0 & -\alpha & K_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\theta & -\gamma & K_6 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & K_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\tau & K_7 \end{pmatrix}.$$

where, $K_1 = \xi + \mu,\ K_2 = \tau + \mu,\ K_3 = \nu + \mu,\ K_4 = \theta + \alpha + \mu,\ K_5 = \gamma + \mu + \delta_1$, $K_6 = \mu + \delta_2$ and $K_7 = \psi + \mu$.

The associated reproduction number, denoted by $\mathcal{R}_T$ is then given by

$$\mathcal{R}_T = \rho(FV^{-1}) = \max\{\mathcal{R}_{01}, \mathcal{R}_{02}\},$$

where $\mathcal{R}_{01}$ and $\mathcal{R}_{02}$ are the associated reproduction numbers for TV and HIV/AIDS, respectively, given by

$$\mathcal{R}_{01} = \frac{\beta_T(1-\epsilon\kappa)(K_2K_3 + \eta_1\xi K_3 + \eta_2\tau\xi)}{K_1K_2K_3} \text{ and } \mathcal{R}_{02} = \frac{\beta_H[K_5(K_6 + \theta\eta_T) + \alpha(\eta_A K_6 + \gamma\eta_T)]}{K_4K_5K_6}.$$

Therefore, applying Theorem 2 of [27], the following result is established.

Lemma 4 *The DFE, $\mathcal{E}_2$, of the model* (1) *is LAS if $\mathcal{R}_T < 1$ and unstable if $\mathcal{R}_T > 1$.*

4.1 Backward Bifurcation

The TV-HIV model (1) exhibits backward bifurcation under certain conditions. This can be proved using a similar approach as in the proof of Theorem 1 (presented in Appendix A).

4.1.1 Non-existence of backward bifurcation for special case

The backward bifurcation property of the TV-HIV model (1) is caused by the co-infection of HIV with TV in humans. In the absence of co-infection (that is when $\sigma = 0$), the existence of backward bifurcation is ruled out. To further rule out the existence of backward bifurcation for the model (1), a global stability of the DFE when $\sigma = 0$ is proven in Appendix C.

4.1.2 Global asymptotic stability of the DFE

Theorem 4 *The DFE of the model* (1)*, is globally-asymptotically stable (GAS) in* $\tilde{\mathcal{D}}$*, whenever* $\mathcal{R}_T \leq 1$.

The proof is based on using the following Lyapunov function (see Appendix C for detailed calculations):

$$\mathcal{F} = \mathcal{R}_{01} I_T^U + \frac{\beta_T c(\eta_1 K_3 + \eta_2 \tau)}{K_2 K_3} I_T^C + \frac{\beta_T c \eta_2}{K_3} T_T + \mathcal{R}_{02} I_H + \frac{\beta_H(\eta_A K_6 + \eta_T \gamma)}{K_5 K_6} A_H + \frac{\beta_H \eta_T}{K_6} T_H.$$

5 Numerical Simulations

The model (1) is simulated using parameter values presented in Table 8 (unless otherwise stated).

5.1 Effect of TV on dynamics of HIV

The effect of TV on the transmission of HIV by individuals infected with TV is monitored by simulating the model (1), using varying values of the parameter for the increased likelihood of individuals infected with TV acquiring co-infection with HIV. The simulation results illustrated in Figure 4 show that the total number of new HIV infections decreases with decreasing values of σ. That is, a reduction in the TV incidence in the population reduces the HIV incidence in the same population. This could be due to a woman's increased susceptibility to HIV by two or three-fold by TV infection. Furthermore, the singular effect of treatment of individuals infected with HIV is assessed. The simulation results depicted in Figure 6 shows that an increase in treatment of individuals infected with HIV results in a decrease in the total number of individuals infected with HIV.

5.2 Assessment of Control Strategies of TV

In this section, the main control strategies for TV are considered. This is of interest to the study since infection with TV increases susceptibility to HIV. The control strategies considered include:

1. Using condoms by sexually active individuals (condom-only strategy);
2. Counselling of infected individuals (counselling-only strategy);
3. Treatment of infected individuals (treatment-only strategy);
4. Using condoms and receiving different levels of counselling for TV (condom and counselling strategy);
5. Counselling and treatment of infected individuals who have tested positive for TV at varying levels (counselling and treatment strategy);
6. Using condoms, receiving counselling and treatment for TV (condom, counselling and treatment strategy).

5.2.1 Condom-only strategy

Since not all the sexually active individuals in a population are expected to strictly comply to the use of condoms consistently and correctly during every sexual encounter, it is therefore informative to determine whether or not the use of condoms as a sole intervention strategy will offer a beneficial population-level impact. This is done by setting all the treatment and counselling related parameters and state variables of the model (1) to zero (i.e. $I_T^C = T_T = R = T_{TH} = \xi = \tau = \nu = \phi = \eta_1 = \eta_2 = 0$), which produces a reduced model with the following associated reproduction number,

$$\mathcal{R}_{c1} = \frac{\beta_T(1-\epsilon\kappa)}{\mu},$$

Firstly, the effect of condom use on TV transmission dynamics can be assessed qualitatively, by differentiating the expression for $\mathcal{R}_{c1}$ partially with respect to κ (condom compliance). This gives

$$\frac{\partial \mathcal{R}_{c1}}{\partial \kappa} = -\frac{\beta_T \epsilon}{\mu} < 0.$$

Since $\mathcal{R}_{c1}$ is a decreasing function of κ, an increase in condom compliance (κ) results in a decrease of $\mathcal{R}_{c1}$. This result is depicted in Table 1. Thus, the above analysis shows that condom use will always have a positive population-level impact (even for small efficacy and compliance level) by reducing the disease burden. In this study, condom efficacy is fixed at 0.8, unless otherwise stated.

Table 1: Reproduction numbers ($\mathcal{R}_{c1}$) of the model (1) for the condom-only strategy.

Compliance Level	$\mathcal{R}_{c1}$
Low ($\kappa = 0.25$)	31.7632
Moderate ($\kappa = 0.5$)	23.8824
High ($\kappa = 0.75$)	15.8816

A contour plot of the associated reproduction threshold $\mathcal{R}_{c1}$ (as a function of condom efficacy (ϵ) and compliance (κ)), is shown in Figure 7. This figure, generated by using $\beta_T = 0.045$ as well as the set of parameter values in Table 8 (where all parameters related to counselling and treatment are set to zero), shows a decrease in $\mathcal{R}_{c1}$ with increasing ϵ and κ. It is clear that significantly high condom efficacy and compliance is needed to effectively control TV, that is, to attain $\mathcal{R}_{c1} < 1$ so that TV/HIV can be eliminated as guaranteed by Lemma 4. In particular, even if the condom efficacy level is 80% ($\epsilon = 0.8$), at least 75% ($\kappa = 0.75$) of sexually-active individuals would still be required to use condoms consistently and correctly in order to effectively control the spread of TV in a population.

5.2.2 Counselling-only strategy

In this case, all the parameters and state variables of the model The model (1) is simulated using parameter values presented in Table 8 (unless otherwise stated)., related to condom-use and treatment, are set to zero. This gives a reduced model with the following associated reproduction number

$$\mathcal{R}_{c2} = \frac{\beta_T(1+\eta_1\xi)}{K_1},$$

where $K_1 = \xi + \mu$.
Differentiating $\mathcal{R}_{c2}$ partially with respect to the counselling rate ξ gives,

$$\frac{\partial \mathcal{R}_{c2}}{\partial \xi} = \frac{\beta_T \eta_1}{K_1} - \frac{\beta_T(1+\eta_1\xi)}{K_1^2} = \frac{\beta_T(\mu\eta_1 - 1)}{K_1^2}.$$

Thus, $\frac{\partial \mathcal{R}_{c2}}{\partial \xi} < 0$ since $0 < \mu < 1$ and $0 < \eta_1 < 1$.

Therefore, counselling individuals infected with TV will reduce the reproduction number $\mathcal{R}_{c2}$ and thus reduce the TV burden, if the relative risk of infectiousness of counselled infected individuals (η_1) does not exceed unity (that is, if counselled individuals infected with TV reduce their risky sexual behaviour).
Simulations are carried out to further assess the impact of counselling individuals infected with TV using the following levels of counselling:

I) Low counselling effectiveness: $\xi = 0.05$ (i.e., it takes 20 days on average to detect and counselled individual infected with TV);

II) Moderate counselling effectiveness: $\xi = 0.5$ (i.e., it takes 2 days on average to detect and counselled individual infected with TV);

III) High counselling effectiveness: $\xi = 5$ (i.e., it takes 1/5 day on average to detect and counselled individual infected with TV).

Table 2 shows that an increase in the level of effectiveness for counselling results in a decrease in the reproduction number. Thus, counselling individuals infected with TV results in a reduction of the burden of the disease. A high counselling effectiveness level is enough to get $\mathcal{R}_{c2} < 1$, which is enough to eradicate TV.

Table 2: Reproduction numbers ($\mathcal{R}_{c2}$) of the model (1) *for the counselling-only strategy.*

Level	$\mathcal{R}_{c2}$
Low ($\xi = 0.05$)	10.4484
Moderate ($\xi = 0.5$)	1.3691
High ($\xi = 5$)	0.1413

5.2.3 Treatment-only strategy

The singular effect of treatment of individuals with TV is assessed using the Model (3) (where counselling and condom-use related variables are not incorporated) by first of all differentiating the threshold quantity

$$\mathcal{R}_{c3} = \frac{\beta_T(Q_2 + \eta\tau)}{Q_1 Q_2},$$

(where $Q_1 = \tau + \mu$ and $Q_2 = \nu + \mu$), partially with respect to τ yields,

$$\frac{\partial \mathcal{R}_{c3}}{\partial \tau} = \frac{\beta_T \eta}{Q_1 Q_2} - \frac{\beta_T(Q_2 + \eta\tau)}{Q_1^2 Q_2} = \frac{\beta_T(\mu\eta - Q_2)}{Q_1^2 Q_2}.$$

It follows that $\frac{\partial \mathcal{R}_{c3}}{\partial \tau} < 0$ whenever $\eta < \eta_\tau$, where $\eta_\tau = \frac{Q_2}{\mu}$.
Therefore, the treatment of non-counselled individuals will reduce the reproduction number and therefore the TV burden if the relative infectiousness of treated individuals

(η) does not exceed the threshold quantity (η_τ). On the other hand, if $\eta > \eta_\tau$, then the use of treatment will increase the $\mathcal{R}_{c3}$ and as a result increase the burden of TV.

Lemma 5 *The treatment of infected individuals will have a positive population-level impact if* $\eta < \eta_\tau$.

Numerical simulations of the model are carried out to further assess the impact of the Treatment-only Strategy on TV in a population. The following arbitrarily chosen levels of treatment effectiveness are considered:

I) Low treatment effectiveness: $\tau = 0.5$;

II) Moderate treatment effectiveness: $\tau = 2$;

III) High treatment effectiveness: $\tau = 50$.

The simulation results depicted in Figure 5 shows that an increase in treatment of individuals infected with TV results in a decrease in the total number of individuals infected with TV. Furthermore, Table 3 shows that an increase in the level of effectiveness for treatment of individuals infected with TV results in a reduction of the reproduction number. Therefore, treating individuals infected with TV results in a decline of the burden of TV in the community.

Table 3: Reproduction numbers ($\mathcal{R}_{c3}$) of the model (3) for the treatment-only strategy.

Level	$\mathcal{R}_{c3}$
Low ($\tau = 0.5$)	7.4015
Moderate ($\tau = 2$)	2.1699
High ($\tau = 50$)	0.4367

5.2.4 Condom and counselling strategy

Here, we study the combined impact of condom use and counselling. The following counselling effectiveness levels will be used:

I) Low counselling effectiveness: $\xi = 0.05$ coupled with different condom compliance levels: low ($\kappa = 0.25$), moderate ($\kappa = 0.5$) and high ($\kappa = 0.75$);

II) Moderate counselling effectiveness: $\xi = 0.5$ coupled with different condom compliance levels: low ($\kappa = 0.25$), moderate ($\kappa = 0.5$) and high ($\kappa = 0.75$);

III) High counselling effectiveness:$\xi = 5$ coupled with different condom compliance levels: low ($\kappa = 0.25$), moderate ($\kappa = 0.5$) and high ($\kappa = 0.75$).

We first set all the treatment-related parameters and state variables of the Model (1) to zero, (i.e. $T_T = T_{TH} = \tau = \nu = \phi = \psi = 0$).
The associated reproduction number of the reduced model is given by

$$\mathcal{R}_{c4} = \beta_T(1 - \epsilon\kappa)\frac{\mu + \eta_1\xi}{\mu K_1}.$$

Table 4 shows a decrease in $\mathcal{R}_{c4}$ with increasing condom compliance and counselling effectiveness. This strategy reduces the burden of the disease (an increase in counselling and condom use reduces the reproduction number).

Table 4: Reproduction numbers ($\mathcal{R}_{c4}$) of the model (1) *for the condom use and counselling strategy.*

Condom compliance level	**Low counselling**	**Moderate counselling**	**High counselling**
Low ($\kappa = 0.25$)	8.3587	1.0983	0.1130
Moderate ($\kappa = 0.5$)	6.2691	0.8215	0.0848
High ($\kappa = 0.75$)	4.1794	0.5476	0.0565

5.2.5 Counselling and treatment strategy

Next, we study the combined impact of counselling and treating of individuals infected with TV.

The following levels of effectiveness will be used:

I) Low counselling and treatment effectiveness: $\xi = 0.05$ and $\tau = 0.5$;

II) Moderate counselling and treatment effectiveness: $\xi = 0.5$ and $\tau = 2$;

III) High counselling and treatment effectiveness: $\xi = 5$ and $\tau = 50$.

Setting the condom use related parameter values of model (1) to zero ($\kappa = \epsilon = 0$) gives a reduced model with the following reproduction number,

$$\mathcal{R}_{c5} = \frac{\beta_T(K_2K_3 + \eta_1\xi K_3 + \eta_2\tau\xi)}{K_1K_2K_3},$$

where, $K_1 = \xi + \mu,\ K_2 = \tau + \mu,\ K_3 = \nu + \mu.$

Table 5 shows that $\mathcal{R}_{c5}$ decreases with increasing counselling and treatment effectiveness. As expected, high levels of treatment and counselling reduces the burden of the disease in the population.

Table 5: Reproduction numbers ($\mathcal{R}_{c5}$) of the model (1) *for the counselling and treatment strategy.*

Treatment	**Counselling**		
	Low	**Moderate**	**High**
Low	10.7007	1.6997	0.4825
Moderate	10.5132	1.4541	0.2290
High	10.4511	1.3727	0.1450

5.2.6 Condom, counselling and treatment strategy

Finally, we explore the condom, counselling and treatment strategy. The following levels of effectiveness will be used:

I) Low counselling and treatment effectiveness: $\xi = 0.05$ and $\tau = 0.5$. Coupled with different condom compliance levels: low ($\kappa = 0.25$), moderate ($\kappa = 0.5$) and high ($\kappa = 0.75$);

II) Moderate counselling and treatment effectiveness: $\xi = 0.5$ and $\tau = 2$. Coupled with different condom compliance levels: low ($\kappa = 0.25$), moderate ($\kappa = 0.5$) and high ($\kappa = 0.75$);

III) High counselling and treatment effectiveness: $\xi = 5$ and $\tau = 50$. Coupled with different condom compliance levels: low ($\kappa = 0.25$), moderate ($\kappa = 0.5$) and high ($\kappa = 0.75$).

It follows from Table 6 that using a moderate effective counselling and treatment strategy, together with moderate and high condom compliance (subject to 80 % condom

efficacy) can reduce the burden of TV in the population. Table 6 also shows a decrease in the reproduction number $\mathcal{R}_{c6}$ with an increase in counselling and treatment, coupled with increasing values of condom compliance.

Table 6: Reproduction numbers ($\mathcal{R}_{c6}$) of the model (1) *for the condom, counselling and treatment strategy.*

Condom	**Counselling and Treatment**		
compliance level	**Low Level**	**Moderate Level**	**High Level**
Low ($\kappa = 0.25$)	8.5606	1.1632	0.1160
Moderate ($\kappa = 0.5$)	6.4204	0.8720	0.0870
High ($\kappa = 0.75$)	4.2803	0.5816	0.0580

It should also be noted that the best strategy for the eradication of TV in the population is the combined (counselling, treatment and condom use) strategy. Here, a low counselling and treatment effectiveness level coupled with high condom compliance level is enough to control the disease in the population.

6 Conclusion

A new model for the transmission dynamics of Trichomonas Vaginalis (TV) and HIV/AIDS co-infection is constructed and analysed. Some of the main theoretical findings of the study are summarized below:

i) Both the full-model and sub-model undergo phenomenon of backward bifurcation. It is established that co-infection of TV and HIV is the condition for the emergence of this phenomenon.

ii) In the absence of co-infection of TV with HIV/AIDS, the DFE of the model (and the sub-model) is shown to be globally-asymptotically stable whenever the associated reproduction number is less than unity.

iii) The endemic equilibrium of the sub-model is shown to be globally asymptotically stable for a special case.

iv) Numerical simulations of the models show the following.

- A reduction in the TV incidence in the population reduces HIV incidence in the same population.

- An increase in the treatment effectiveness level of individuals infected with TV and/or HIV results in a decline in the burden of TV and/or HIV in the community.

v) An assessment of control strategies of TV using the full model resulted in the following results:

- Condom-use has a positive population level impact on TV. This means that an increase in condom compliance will result in a decline in the burden of TV.
- Counselling has a positive population level impact on TV. Thus, an increase in counselling of individuals infected with TV will result in a decrease of TV in a population.
- An increase in the level of treatment effectiveness results in a decline of the associated reproduction number. The higher the level of treatment of TV in a population, the lower the burden of the disease.
- If the aforementioned control strategies are implemented at a high level of effectiveness, the burden of TV will be reduced in the population, thereby result in the reduction of HIV/AIDS incident in the population.

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Appendix A: Proof of Theorem 1

Proof. The existence of backward bifurcation will be explored using Centre Manifold Theory [6, 27]. To apply this theory we first carry out the following change of variables. Let $S = x_1, I_T = x_2, T_T = x_3, I_H = x_4, A_H = x_5, T_H = x_6, I_{TH} = x_7$, and $T_{TH} = x_8$ so that $N = x_1 + x_2 + x_2 + x_3 + x_4 + x_5 + x_6 + x_7 + x_8$. In addition, by using vector notation $X = (x_1, x_2, x_2, x_3, x_4, x_5, x_6, x_7, x_8)^T$, the TV-HIV model (3) can be written in the form $\frac{dX}{dt} = F(X)$, with $(f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8)^T$ as follows,

$$\begin{aligned}
\tfrac{dx_1}{dt} &= f_1 = \Pi + \nu x_3 - (\lambda_H + \lambda_T)x_1 - \mu x_1,\\
\tfrac{dx_2}{dt} &= f_2 = \lambda_T x_1 - \sigma\lambda_H x_2 - (\tau + \mu)x_2,\\
\tfrac{dx_3}{dt} &= f_3 = \tau x_2 - (\nu + \mu)x_3,\\
\tfrac{dx_4}{dt} &= f_4 = \lambda_H x_1 + \psi x_8 - (\alpha + \theta + \mu)x_4,\\
\tfrac{dx_5}{dt} &= f_5 = \alpha x_4 - (\gamma + \mu + \delta_1)x_5,\\
\tfrac{dx_6}{dt} &= f_6 = \theta x_4 + \gamma x_5 - (\mu + \delta_2)x_6,\\
\tfrac{dx_7}{dt} &= f_7 = \sigma\lambda_H x_2 - (\tau + \mu)x_7,\\
\tfrac{dx_8}{dt} &= f_8 = \tau x_7 - (\psi + \mu)x_8,
\end{aligned} \tag{A.1}$$

with the forces of infection given by

$$\lambda_T = \frac{\beta_T(x_2 + \eta x_3)}{x_1 + x_2 + x_2 + x_3 + x_4 + x_5 + x_6 + x_7 + x_8} \text{ and}$$

$$\lambda_H = \frac{\beta_H(x_4 + \eta_A x_5 + \eta_T x_6)}{x_1 + x_2 + x_2 + x_3 + x_4 + x_5 + x_6 + x_7 + x_8}.$$

Consider the case when $\mathcal{R}_0 = 1$ (that is $\max\{\mathcal{R}_1, \mathcal{R}_2\} = 1$). Also suppose that $\beta_H = \beta_H^*$ is chosen as the bifurcation parameter. Solving for $\beta_H = \beta_H^*$ from $\mathcal{R}_0 = 1$ in (4) gives

$$\beta_H^* = \frac{Q_3 Q_4 Q_5}{Q_4(Q_5 + \theta\eta_T) + \alpha(\eta_A Q_5 + \gamma\eta_T)}.$$

The Jacobian of the system (A.1) evaluated at the DFE $\mathcal{E}_3$, with $\beta_H = \beta_H^*$ and denoted by J^*, is given by

$$J^* = \begin{pmatrix} -\mu & -\beta_T & -\beta_T\eta + \nu & -\beta_H^* & -\beta_H^*\eta_A & -\beta_H^*\eta_T & 0 & 0 \\ 0 & \beta_T - Q_1 & \beta_T\eta & 0 & 0 & 0 & 0 & 0 \\ 0 & \tau & -Q_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta_H^* - Q_3 & \beta_H^*\eta_A & \beta_H^*\eta_T & 0 & \psi \\ 0 & 0 & 0 & \alpha & -Q_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & \theta & \gamma & -Q_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -Q_1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \tau & -Q_6 \end{pmatrix},$$

The Jacobian has a simple zero eigenvalue (with all other eigenvalues having negative real part), therefore the Centre Manifold Theory can be used to analyse the dynamics of the system (A.1).

Eigenvectors of $\mathbf{J}(\mathcal{E}_3)|_{\beta_H=\beta_H^*}$ For the case when $\mathcal{R}_0 = 1$, it can be shown that J has a left eigenvector (corresponding to the zero eigenvalue), given by $\bar{v} = [v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8]$, where

$$v_1 = 0, \qquad v_2 > 0, \qquad v_3 = \frac{\beta_T\eta}{Q_2}v_2, \qquad v_4 > 0, \qquad v_5 = \frac{\beta_H^*(\eta_A Q_5 + \gamma\eta_T)}{Q_4Q_5}v_4,$$

$$v_6 = \frac{\beta_H^*\eta_T}{Q_5}v_4, \qquad v_7 = \frac{\tau\psi}{Q_1Q_6}v_4, \qquad v_8 = \frac{\psi}{Q_6}v_4.$$

Similarly, the components of the right eigenvector of $J(\mathcal{E}_3)|_{\beta_H=\beta_H^*}$ (corresponding to the zero eigenvalue), denoted by $\bar{w} = [w_1, w_2, w_3, v, w_4, w_5, w_6, w_7, w_8]^T$ are

$$w_1 = -\frac{1}{\mu}\left[\left(\frac{\beta_T(Q_2 + \eta\tau) - \nu\tau}{Q_2}\right)w_2 + \left(\frac{\beta_H^*(Q_4Q_5 + \eta_A\alpha Q_5 + \eta_T\theta Q_4 + \eta_T\alpha\gamma)}{Q_4Q_5}\right)w_4\right],$$

$$w_2 > 0, \qquad w_3 = \frac{\tau}{Q_2}w_2, \qquad w_4 > 0, \qquad w_5 = \frac{\alpha}{Q_4}w_4, \qquad w_6 = \frac{(\theta Q_4 + \alpha\gamma)}{Q_4Q_5}w_4,$$

$$w_7 = 0, \qquad w_8 = 0.$$

It is worth noting that the free left eigenvalues v_2 and v_4, and right eigenvalues w_2 and w_4 are chosen to be

$$v_2 = v_4 = \frac{1}{2}, \qquad w_2 = \frac{Q_2^2}{Q_2^2 + \beta_T\eta\tau}, \quad \text{and}$$

$$w_4 = \frac{Q_4^2Q_5^2}{Q_4^2Q_5^2 + \beta_H[\alpha(\eta_A Q_5^2 + \eta_t\gamma Q_5 + \eta_T\gamma Q_4) + \eta_T\theta Q_4^2]}, \text{ so that}$$

the classical requirement of $\bar{v} \cdot \bar{w} = 1$ is satisfied (see for instance [6]). That is

$$\bar{v} \cdot \bar{w} = v_2 w_2 + v_3 w_3 + v_4 w_4 + v_5 w_5 + v_6 w_6$$

$$= \frac{Q_2^2 + \beta_T \eta \tau}{Q_2^2} v_2 w_2 + \frac{Q_4^2 Q_5^2 + \beta_H [\alpha(\eta_A Q_5^2 + \eta_t \gamma Q_5 + \eta_T \gamma Q_4) + \eta_T \theta Q_4^2]}{Q_4^2 Q_5^2} v_4 w_4 = 1.$$

Computation of a

For the transformed TV-HIV model (A.1), the associated non-zero partial derivatives of F (evaluated at the DFE) are given by

$$\frac{\partial^2 f_1}{\partial x_2 \partial x_2} = \frac{2\beta_T \mu}{\Pi}, \quad \frac{\partial^2 f_1}{\partial x_2 \partial x_3} = \frac{\mu(\beta_T + \beta_T \eta)}{\Pi}, \quad \frac{\partial^2 f_1}{\partial x_2 \partial x_4} = \frac{\mu(\beta_T + \beta_H)}{\Pi},$$

$$\frac{\partial^2 f_1}{\partial x_2 \partial x_5} = \frac{\mu(\beta_T + \beta_H \eta_A)}{\Pi}, \quad \frac{\partial^2 f_1}{\partial x_2 \partial x_6} = \frac{\mu(\beta_T + \beta_H \eta_T)}{\Pi}, \quad \frac{\partial^2 f_1}{\partial x_2 \partial x_7} = \frac{\partial^2 f_1}{\partial x_2 \partial x_8} = \frac{\beta_T \mu}{\Pi}$$

$$\frac{\partial^2 f_1}{\partial x_3 \partial x_3} = \frac{2\beta_T \eta \mu}{\Pi}, \quad \frac{\partial^2 f_1}{\partial x_3 \partial x_4} = \frac{\mu(\beta_T \eta + \beta_H)}{\Pi}, \quad \frac{\partial^2 f_1}{\partial x_3 \partial x_5} = \frac{\mu(\beta_T \eta + \beta_H \eta_A)}{\Pi},$$

$$\frac{\partial^2 f_1}{\partial x_3 \partial x_6} = \frac{\mu(\beta_T \eta + \beta_H \eta_T)}{\Pi}, \quad \frac{\partial^2 f_1}{\partial x_3 \partial x_7} = \frac{\partial^2 f_1}{\partial x_3 \partial x_8} = \frac{\beta_T \eta \mu}{\Pi}, \quad \frac{\partial^2 f_1}{\partial x_4 \partial x_4} = \frac{2\beta_H \mu}{\Pi},$$

$$\frac{\partial^2 f_1}{\partial x_4 \partial x_5} = \frac{\mu(\beta_H + \beta_H \eta_A)}{\Pi}, \quad \frac{\partial^2 f_1}{\partial x_4 \partial x_6} = \frac{\mu(\beta_H + \beta_H \eta_T)}{\Pi}, \quad \frac{\partial^2 f_1}{\partial x_4 \partial x_7} = \frac{\partial^2 f_1}{\partial x_4 \partial x_8} = \frac{\beta_H \mu}{\Pi},$$

$$\frac{\partial^2 f_1}{\partial x_5 \partial x_5} = \frac{2\beta_H \eta_A \mu}{\Pi}, \quad \frac{\partial^2 f_1}{\partial x_5 \partial x_6} = \frac{\mu(\beta_H \eta_A + \beta_H \eta_T)}{\Pi} \quad \frac{\partial^2 f_1}{\partial x_5 \partial x_7} = \frac{\partial^2 f_1}{\partial x_5 \partial x_8} = \frac{\beta_H \eta_A \mu}{\Pi},$$

$$\frac{\partial^2 f_1}{\partial x_6 \partial x_6} = \frac{2\beta_H \eta_T \mu}{\Pi}, \quad \frac{\partial^2 f_1}{\partial x_6 \partial x_7} = \frac{\partial^2 f_1}{\partial x_6 \partial x_8} = \frac{\beta_H \eta_T \mu}{\Pi},$$

$$\frac{\partial^2 f_2}{\partial x_2 \partial x_2} = -\frac{2\beta_T \mu}{\Pi}, \quad \frac{\partial^2 f_2}{\partial x_2 \partial x_3} = -\frac{\mu(\beta_T + \beta_T \eta)}{\Pi}, \quad \frac{\partial^2 f_2}{\partial x_2 \partial x_4} = -\frac{\mu(\beta_T + \sigma \beta_H)}{\Pi},$$

$$\frac{\partial^2 f_2}{\partial x_2 \partial x_5} = -\frac{\mu(\beta_T + \sigma \beta_H \eta_A)}{\Pi}, \quad \frac{\partial^2 f_2}{\partial x_2 \partial x_6} = -\frac{\mu(\beta_T + \sigma \beta_H \eta_T)}{\Pi}, \quad \frac{\partial^2 f_2}{\partial x_2 \partial x_7} = \frac{\partial^2 f_2}{\partial x_2 \partial x_8} = -\frac{\beta_T \mu}{\Pi},$$

$$\frac{\partial^2 f_2}{\partial x_3 \partial x_3} = -\frac{2\beta_T \eta \mu}{\Pi}, \quad \frac{\partial^2 f_2}{\partial x_3 \partial x_4} = \frac{\partial^2 f_2}{\partial x_3 \partial x_5} = \frac{\partial^2 f_2}{\partial x_3 \partial x_6} = \frac{\partial^2 f_2}{\partial x_3 \partial x_7} = \frac{\partial^2 f_1}{\partial x_3 \partial x_8} = -\frac{\beta_T \eta \mu}{\Pi},$$

$$\frac{\partial^2 f_4}{\partial x_2 \partial x_4} = \frac{\partial^2 f_4}{\partial x_3 \partial x_4} = \frac{\partial^2 f_4}{\partial x_4 \partial x_7} = \frac{\partial^2 f_4}{\partial x_4 \partial x_8} = -\frac{\beta_H \mu}{\Pi}, \quad \frac{\partial^2 f_4}{\partial x_4 \partial x_4} = -\frac{2\beta_H \mu}{\Pi},$$

$$\frac{\partial^2 f_4}{\partial x_2 \partial x_5} = \frac{\partial^2 f_4}{\partial x_3 \partial x_5} = \frac{\partial^2 f_4}{\partial x_5 \partial x_7} = \frac{\partial^2 f_4}{\partial x_5 \partial x_8} = -\frac{\beta_H \eta_A \mu}{\Pi}, \quad \frac{\partial^2 f_4}{\partial x_4 \partial x_5} = -\frac{\mu(\beta_H + \beta_H \eta_A)}{\Pi},$$

$$\frac{\partial^2 f_4}{\partial x_4 \partial x_6} = \frac{\partial^2 f_4}{\partial x_3 \partial x_6} = \frac{\partial^2 f_4}{\partial x_6 \partial x_7} = \frac{\partial^2 f_4}{\partial x_6 \partial x_8} = -\frac{\beta_H \eta_T \mu}{\Pi}, \quad \frac{\partial^2 f_4}{\partial x_4 \partial x_6} = -\frac{\mu(\beta_H + \beta_H \eta_T)}{\Pi},$$

$$\frac{\partial^2 f_4}{\partial x_5 \partial x_5} = -\frac{2\beta_H \eta_A \mu}{\Pi}, \quad \frac{\partial^2 f_4}{\partial x_6 \partial x_6} = -\frac{2\beta_H \eta_T \mu}{\Pi}, \quad \frac{\partial^2 f_4}{\partial x_5 \partial x_6} = -\frac{\mu(\beta_H \eta_A + \beta_H \eta_T)}{\Pi},$$

$$\frac{\partial^2 f_7}{\partial x_2 \partial x_4} = \frac{\sigma \beta_H \mu}{\Pi}, \quad \frac{\partial^2 f_7}{\partial x_2 \partial x_5} = \frac{\sigma \beta_H \eta_A \mu}{\Pi}, \quad \frac{\partial^2 f_7}{\partial x_2 \partial x_6} = \frac{\sigma \beta_H \eta_T \mu}{\Pi}.$$

Using the expressions above, it follows that

$$a = \sum_{k,i,j}^{8} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)$$

$$= \frac{2\mu}{\Pi}[v_7 w_2 \sigma \beta_H w_4 + v_7 w_2 \sigma \beta_H w_5 \eta_A + v_7 w_2 \sigma \beta_H w_6 \eta_T - v_2 w_2 \sigma \beta_H w_4 v_2 w_2 \sigma \beta_H - v_2 w_2 \sigma \beta_H w_5 \eta_A$$

$$- v_2 w_2 \sigma \beta_H w_6 \eta_T - \beta_T (v_2 w_2 w_2 + v_2 w_2 w_3 + v_2 w_2 w_4 + v_2 w_2 w_5 + v_2 w_2 w_6 + v_2 w_3 \eta w_2 + w_3 v_2 w_3 \eta)$$

$$- \beta_T (w_4 v_2 w_3 \eta w_5 + w_6 v_2 w_3 \eta) - \beta_H (w_2 v_4 w_4 + w_3 v_4 w_4 + w_4 v_4 w_4 + w_5 v_4 w_4 + w_6 v_4 w_4)$$

$$- \beta_H (w_2 v_4 w_5 \eta_A + w_3 v_4 w_5 \eta_A + w_4 v_4 w_5 \eta_A + w_5 v_4 w_5 \eta_A + w_6 v_4 w_5 \eta_A + w_2 v_4 w_6 \eta_T + w_3 v_4 w_6 \eta_T)$$

$$- \beta_H (w_4 v_4 w_6 \eta_T + w_5 v_4 w_6 \eta_T + w_6 v_4 w_6 \eta_T)].$$

Which can be simplified to the following,

$$
\begin{aligned}
a &= \sum_{k,i,j}^{8} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0) \\
&= \frac{2\mu}{\Pi}[v_7 w_2 \sigma \beta_H (w_4 + w_5\eta_A + w_6\eta_T) - v_2 w_2 \sigma \beta_H (w_4 + w_5\eta_A + w_6\eta_T) \\
&- \beta_T (v_2 w_2 + v_2 w_3 \eta)(w_2 + w_3 + w_4 + w_5 + w_6) \\
&- \beta_H (v_4 w_4 + v_4 w_5 \eta_A + v_4 w_6 \eta_T)(w_2 + w_3 + w_4 + w_5 + w_6)].
\end{aligned} \tag{A.2}
$$

Computation of b
Substituting the vectors $\bar{v}$ and $\bar{w}$ and the respective partial derivatives (evaluated at the DFE) into the expression of b yields,

$$
b = \sum_{k,i,j}^{8} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta_H}(0,0) = v_4 w_4 + v_4 w_5 \eta_A + v_4 w_6 \eta_T > 0.
$$

Since the coefficient b is automatically positive, it follows that the model (3) will undergo backward bifurcation if the coefficient a given by (A.2), is positive. ■

Appendix B: Proof of Theorem 3

Proof. Consider the following non-linear Lyapunov function

$$
\begin{aligned}
\mathcal{F} = S - S^{**} - S^{**} \ln \frac{S}{S^{**}} + I_T - I_T^{**} - I_T^{**} \ln \frac{I_T}{I_T^{**}} + \frac{\beta_T \eta S^{**}}{Q_2} \left[T_T - T_T^{**} - T_T^{**} \ln \frac{T_T}{T_T^{**}} \right] \\
+ I_H - I_H^{**} - I_H^{**} \ln \frac{I_H}{I_H^{**}} + \frac{\beta_H \eta_A S^{**}}{Q_4} \left[A_H - A_H^{**} - A_H^{**} \ln \frac{A_H}{A_H^{**}} \right],
\end{aligned}
$$

with Lyapunov derivative

$$\dot{\mathcal{F}} = \dot{S} - \frac{S^{**}}{S}\dot{S} + \dot{I}_T - \frac{I_T^{**}}{I_T}\dot{I}_T + \frac{\beta_T \eta S^{**}}{Q_2}\left[\dot{T}_T - \frac{T_T^{**}}{T_T}\dot{T}_T\right] + \dot{I}_H - \frac{I_H^{**}}{I_H}\dot{I}_H + \frac{\beta_H \eta_A S^{**}}{Q_4}\left[\dot{A}_H - \frac{A_H^{**}}{A_H}\dot{A}_H\right],$$

$$= \Pi - \lambda_H S - \lambda_T S - \mu S - \frac{S^{**}}{S}(\Pi - \lambda_H S - \lambda_T S - \mu S) + (\lambda_T S - Q_1 I_T) - \frac{I_T^{**}}{I_T}((\lambda_T S - Q_1 I_T)$$

$$+ \frac{\beta_T \eta S^{**}}{Q_2}\left[\tau I_T - Q_2 T_T - \frac{T_T^{**}}{T_T}(\tau I_T - Q_2 T_T)\right] + \lambda_H S - Q_3 I_H - \frac{I_H^{**}}{I_H}(\lambda_H S - Q_3 I_H)$$

$$+ \frac{\beta_H \eta_A S^{**}}{Q_4}\left[\alpha I_H - Q_4 A_H - \frac{A_H^{**}}{A_H}(\alpha I_H - Q_4 A_H)\right],$$

Let $\tilde{\beta} = \frac{\beta \mu}{\Pi}$. Applying this and simplifying yields

$$\dot{\mathcal{F}} = \Pi - \mu S - \Pi\frac{S^{**}}{S} + \tilde{\beta_T}(I_T + \eta T_T)S^{**} + \tilde{\beta_H}(I_H + \eta_A A_H)S^{**} + \mu S^{**} - Q_1 I_T$$

$$- \frac{I_T^{**}}{I_T}\tilde{\beta_T}(I_T + \eta T_T)S + Q_1 I_T^{**} + \frac{\tilde{\beta_T}\eta S^{**}}{Q_2}\tau I_T - \tilde{\beta_T}\eta S^{**} T_T - \frac{\tilde{\beta_T}\eta S^{**}}{Q_2}\frac{T_T^{**}}{T_T}\tau I_T + \tilde{\beta_T}\eta S^{**} T_T^{**}$$

$$- Q_3 I_H - \frac{I_H^{**}}{I_H}\tilde{\beta_H}(I_H + \eta_A A_H)S + Q_3 I_H^{**}$$

$$+ \frac{\tilde{\beta_H}\eta_A S^{**}}{Q_4}\alpha I_H - \tilde{\beta_H}\eta_A S^{**} A_H - \frac{\tilde{\beta_H}\eta_A S^{**}}{Q_4}\alpha I_H \frac{A_H^{**}}{A_H} + \tilde{\beta_H}\eta_A S^{**} A_H^{**}.$$

It can be shown from the model (3) that at endemic steady-state,

$$\Pi = \tilde{\beta_T} I_T^{**} S^{**} + \tilde{\beta_T}\eta T_T^{**} S^{**} + \tilde{\beta_H} I_H^{**} S^{**} + \tilde{\beta_H}\eta_A A_H^{**} S^{**} + \mu S^{**},$$

$$Q_1 I_T^{**} = \tilde{\beta_T} I_T^{**} S^{**} + \tilde{\beta_T}\eta T_T^{**} S^{**}, \qquad Q_2 T_T^{**} = \tau I_T^{**},$$

$$Q_3 I_H^{**} = \tilde{\beta_H} I_H^{**} S^{**} + \eta_A A_H^{**} S^{**}, \qquad Q_4 A_H^{**} = \alpha I_H^{**}.$$

Substituting the above relations gives,

$$\dot{\mathcal{F}} = \tilde{\beta_T} I_T^{**} S^{**} + \tilde{\beta_T} \eta T_T^{**} S^{**} + \tilde{\beta_H} I_H^{**} S^{**} + \tilde{\beta_H} \eta_A A_H^{**} S^{**} + \mu S^{**} - \mu S$$

$$- (\tilde{\beta_T} I_T^{**} S^{**} + \tilde{\beta_T} \eta T_T^{**} S^{**} + \tilde{\beta_H} I_H^{**} S^{**} + \tilde{\beta_H} \eta_A A_H^{**} S^{**} + \mu S^{**}) \frac{S^{**}}{S} + \mu S^{**} - \tilde{\beta_T} I_T^{**} S^{**} \frac{S}{S^{**}}$$

$$- \tilde{\beta_T} \eta T_T^{**} S^{**} \frac{S I_T^{**} T_T}{S^{**} I_T T_T^{**}} + \tilde{\beta_T} I_T^{**} S^{**} + \tilde{\beta_T} \eta T_T^{**} S^{**} - \tilde{\beta_T} \eta T_T^{**} S^{**} \frac{I_T T_T^{**}}{I_T^{**} T_T} + \tilde{\beta_T} \eta T_T^{**} S^{**}$$

$$- \tilde{\beta_H} I_H^{**} S^{**} \frac{S}{S^{**}} - \tilde{\beta_H} \eta_A A_H^{**} S^{**} \frac{S I_H^{**} A_H}{S^{**} I_H A_H^{**}} + \tilde{\beta_H} I_H^{**} S^{**} + \tilde{\beta_H} \eta_A A_H^{**} S^{**}$$

$$- \tilde{\beta_H} \eta_A A_H^{**} S^{**} \frac{I_H A_H^{**}}{I_H^{**} A_H} + \tilde{\beta} \eta_A A_H^{**} S^{**},$$

which can be simplified to

$$= \mu S^{**} \left[2 - \frac{S^{**}}{S} - \frac{S}{S^{**}} \right] + \tilde{\beta_T} I_T^{**} S^{**} \left[2 - \frac{S^{**}}{S} - \frac{S}{S^{**}} \right] + \tilde{\beta_H} I_H^{**} S^{**} \left[2 - \frac{S^{**}}{S} - \frac{S}{S^{**}} \right]$$

$$+ \tilde{\beta_T} \eta T_T^{**} S^{**} \left[3 - \frac{S^{**}}{S} - \frac{I_T T_T^{**}}{I_T^{**} T_T} - \frac{S I_T^{**} T_T}{S^{**} I_T T_T^{**}} \right] + \tilde{\beta_H} \eta_A A_H^{**} S^{**} \left[3 - \frac{S^{**}}{S} - \frac{I_H A_H^{**}}{I_H^{**} A_H} - \frac{S I_H^{**} A_H}{S^{**} I_H A_H^{**}} \right].$$

Since the arithmetic mean exceeds the geometric mean, the following inequalities hold

$$2 - \frac{S^{**}}{S} - \frac{S}{S^{**}} \leq 0,$$
$$3 - \frac{S^{**}}{S} - \frac{I_T T_T^{**}}{I_T^{**} T_T} - \frac{S I_T^{**} T_T}{S^{**} I_T T_T^{**}} \leq 0,$$
$$3 - \frac{S^{**}}{S} - \frac{I_H T_H^{**}}{I_H^{**} A_H} - \frac{S I_H^{**} A_H}{S^{**} I_H A_H^{**}} \leq 0.$$

Further, since all the model parameters are non-negative, it follows that $\dot{\mathcal{F}} \leq 0$ for $\mathcal{R}_0 > 1$ and $\mathcal{F}$ is a Lyapunov function on $\mathcal{D}$.
Now we have that,

$$\lim_{t \to \infty} S(t) = S^{**}, \ \lim_{t \to \infty} I_T(t) = I_T^{**}, \ \lim_{t \to \infty} T_T(t) = T_T^{**},$$

$$\lim_{t \to \infty} I_H(t) = I_H^{**} \text{ and } \lim_{t \to \infty} A_H(t) = A_H^{**}.$$

Furthermore, at endemic steady-state, as $t \to \infty$,

$$\lim_{t\to\infty} T_H(t) = \lim_{t\to\infty} \frac{\theta I_H(t) + \gamma A_H}{Q_5} = \frac{\theta I_H^{**}}{Q_5} + \frac{\gamma A_H^{**}}{Q_5} = T_H^{**},$$

$$\lim_{t\to\infty} I_{TH}(t) = \lim_{t\to\infty} \frac{\sigma\lambda_{TH} I_T(t)}{Q_1} = \frac{\sigma\lambda_{TH}^{**} I_T^{**}(t)}{Q_1} = I_{TH}^{**},$$

$$\lim_{t\to\infty} T_{TH}(t) = \lim_{t\to\infty} \frac{\tau I_{TH}(t)}{Q_6} = \frac{\tau I_{TH}^{**}(t)}{Q_6} = T_{TH}^{**}.$$

■

Appendix C: Proof of Theorem 4

First of all notice that by setting $\sigma = 0$ in (1) the equation of $\frac{dI_{TH}}{dt} \to 0$ as $t \to \infty$, thus $\frac{dT_{TH}}{dt} \to 0$ as $t \to \infty$. Hence, it follows that the system and/or HIV can be simplified to

$$\begin{aligned}
\frac{dS}{dt} &= \Pi + \nu T_T - (\lambda_H + \lambda_T)S - \mu S,\\
\frac{dI_T^U}{dt} &= \lambda_T^1 S - (\xi + \mu) I_T^U,\\
\frac{dI_T^C}{dt} &= \xi I_T^U - (\tau + \mu) I_T^C,\\
\frac{dT_T}{dt} &= \tau I_T - (\nu + \mu) T_T,\\
\frac{dI_H}{dt} &= \lambda_H S - (\alpha + \theta + \mu) I_H,\\
\frac{dA_H}{dt} &= \alpha I_H - (\gamma + \mu + \delta_1) A_H,\\
\frac{dT_H}{dt} &= \theta I_H + \gamma A_H - (\mu + \delta_2) T_H.
\end{aligned} \tag{C.1}$$

Next define the invariant region

$$\tilde{\mathcal{D}}_\prime = \{(S, I_T^U, I_T^C, T_T, I_H, A_H, T_H, I_{TH}, T_{TH}) \in \mathcal{D} : S \le S^*\}.$$

We claim the following result.

Theorem 5 *The DFE of the model* (1) *, is globally-asymptotically stable (GAS) in* $\tilde{\mathcal{D}}$*, whenever* $\mathcal{R}_T \leq 1$ *and unstable if* $\mathcal{R}_T > 1$*.*

Proof. Consider the following Lyapunov function

$$\mathcal{F} = \mathcal{R}_{01} I_T^U + \frac{\beta_T c(\eta_1 K_3 + \eta_2 \tau)}{K_2 K_3} I_T^C + \frac{\beta_T c \eta_2}{K_3} T_T + \mathcal{R}_{02} I_H + \frac{\beta_H(\eta_A K_6 + \eta_T \gamma)}{K_5 K_6} A_H + \frac{\beta_H \eta_T}{K_6} T_H$$

with Lyapunov derivative given by (where $c = 1 - \epsilon\kappa > 0$)

$$\begin{aligned}
\dot{\mathcal{F}} &= \mathcal{R}_{01} \dot{I}_T^U + \frac{\beta_T c(\eta_1 K_3 + \eta_2 \tau)}{K_2 K_3} \dot{I}_T^C + \frac{\beta_T c \eta_2}{K_3} \dot{T}_T + \mathcal{R}_{02} \dot{I}_H + \frac{\beta_H(\eta_A K_6 + \eta_T \gamma)}{K_5 K_6} \dot{A}_H + \frac{\beta_H \eta_T}{K_6} \dot{T}_H, \\
&= \mathcal{R}_{01}(\lambda_T^1 S - K_1 I_T^U) + \frac{\beta_T c(\eta_1 K_3 + \eta_2 \tau)}{K_2 K_3}(\xi I_T^U - K_2 I_T^C) + \frac{\beta_T c \eta_2}{K_3}(\tau I_T^C - K_3 T_T) \\
&+ \mathcal{R}_{02}[\lambda_H S - K_4 I_H] + \frac{\beta_H(\eta_A K_6 + \eta_T \gamma)}{K_5 K_6}[\alpha I_H - K_5 A_H] + \frac{\beta_H \eta_T}{K_6}[\theta I_H + \gamma A_H - K_6 T_H], \\
&= \mathcal{R}_{01} \lambda_T^1 S - \beta_T c(I_T^U + \eta_1 I_T^C + \eta_2 T_T) + \mathcal{R}_{02} \lambda_H S - \beta_H(I_H + \eta_A A_H + \eta_T T_H), \\
&= \mathcal{R}_{01} \lambda_T^1 S - \lambda_T^1 N + \mathcal{R}_{02} \lambda_H S - \lambda_H N, \\
&= \lambda_T^1 N \left[\frac{\mathcal{R}_{01} S}{N} - 1\right] + \lambda_H N \left[\frac{\mathcal{R}_{02} S}{N} - 1\right], \\
&\leq \lambda_T^1 N^*[\mathcal{R}_{01} - 1] + \lambda_H N^*[\mathcal{R}_{02} - 1], \quad \text{since } S \leq S^* \text{ in } \tilde{\mathcal{D}}, \text{ and } N \leq N^*, \\
&= \beta_T c(I_T^U + \eta_1 I_T^C + \eta_2 T_T)[\mathcal{R}_{01} - 1] + \beta_H(I_H + \eta_A A_H + \eta_T T_H)[\mathcal{R}_{02} - 1] \\
&\leq 0 \quad \text{whenever} \quad \mathcal{R}_T = \max\{\mathcal{R}_{01}, \mathcal{R}_{02}\} \leq 1.
\end{aligned} \tag{C.2}$$

Since all the parameters and variables of the Model (1) are non-negative (Lemma 1), it follows that $\dot{\mathcal{F}} \leq 0$ for $\mathcal{R}_T \leq 1$ (i.e. $\mathcal{R}_{01} \leq 1$ and $\mathcal{R}_{02} \leq 1$) with $\dot{\mathcal{F}} = 0$ if and only if $I_T^U = I_T^C = T_T = I_H = A_H = T_H = 0$. Hence $\dot{\mathcal{F}}$ is a Lyapunov function on $\tilde{\mathcal{D}}$. Thus, it follows by LaSalle's Invariance Principle [17], that $(I_T^U(t), I_T^C(t), T_T(t), I_H(t), A_H(t), T_H(t)) \to (0, 0, 0, 0, 0, 0)$ as $t \to \infty$. Thus, it follows that every solution of the equations of the Model (1) with initial conditions in $\tilde{\mathcal{D}}$ approaches $\mathcal{E}_2$ as $t \to \infty$ (when $\mathcal{R}_T \leq 1$). ■

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Table 7: Description of Variables and Parameters of the Model (1).

Variables	**Description**
N	Total population
S	Susceptible individuals
I_T	Population of individuals infected TV
T_T	Population of individuals receiving treatment for TV
I_H	Population of individuals infected with HIV
A_H	Population of infected individuals with AIDS symptoms
T_H	Population of individuals receiving treatment
I_{TH}	Population of individuals infected with TV and HIV
T_{TH}	Population of individuals receiving treatment for TV
λ_T	Force of infection for TV
λ_H	Force of infection for HIV

Parameters	**Description**
Π	Recruitment rate of humans
μ	Natural mortality rate of human
β_T	Effective contact rate for TV transmission
β_H	Effective contact rate for HIV transmission
η_1	Modification parameter accounting for the assumed reduction in infectiousness of counselled individuals, in comparison to infected individuals unaware of their TV status
η_2	Modification parameter accounting for the assumed reduction in infectiousness of treated individuals, in comparison to infected individuals unaware of their TV status
η_A	Modification parameter accounting for the assumed reduction in infectiousness of individuals at AIDS stage, in comparison to infected individuals with HIV
η_T	Modification parameter accounting for the assumed reduction in infectiousness of treated individuals, in comparison to infected individuals with HIV
ϵ	Condom efficacy
κ	Condom compliance
ξ	Counselling rate for TV infected individuals
σ	Co-infection parameter
τ	Treatment rate of individuals infected with TV
ν	Recovery rate of individuals infected with TV
ψ	Progression rate of individuals from T_{TH} class to I_H class
α	Progression rate of individual from I_H class to A_H class
θ	Treatment rate of infected individuals in I_H class
γ	Treatment rate of infected individuals in A_H class
δ_1	Disease-induced mortality rate of individuals in A_H class
δ_2	Disease-induced mortality rate of individuals in T_H class

Table 8: Ranges and baseline values for parameters of the model (1).

Parameter	**Range (day^{-1})**	**Baseline (day^{-1})**	**Reference**
Π	[1000, 3000]	2000	Assumed
μ	[0.00004, 0.00005]	0.000046	[11, 24]
β_T	[0.5, 0.9]	0.709	[5]
β_H	[0.45, 0.85]	0.65	Assumed
ϵ	[0.5, 1) (dimensionless)	0.8	[11]
κ	(0, 1) (dimensionless)	0.5	[11]
η_1	(0, 1) (dimensionless)	0.5	
η_2	(0, 1) (dimensionless)	0.5	
η_A	(0, 1) (dimensionless)	0.5	
η_T	(0, 1) (dimensionless)	0.5	
ξ	(0, 5] (dimensionless)	0.5	
σ	[1, 3]	2	Assumed
τ	(0, 50]	2	Assumed
ψ	[0.5, 0.9]	0.7	Assumed
α	[0.01, 0.1]	1/33	Assumed
θ	(0.5, 0.9]	0.8	Assumed
γ	[0.1, 0.8]	0.4	Assumed
δ	[0.009, 0.04]	0.01	Assumed
δ	[0.01, 0.07]	0.04	Assumed

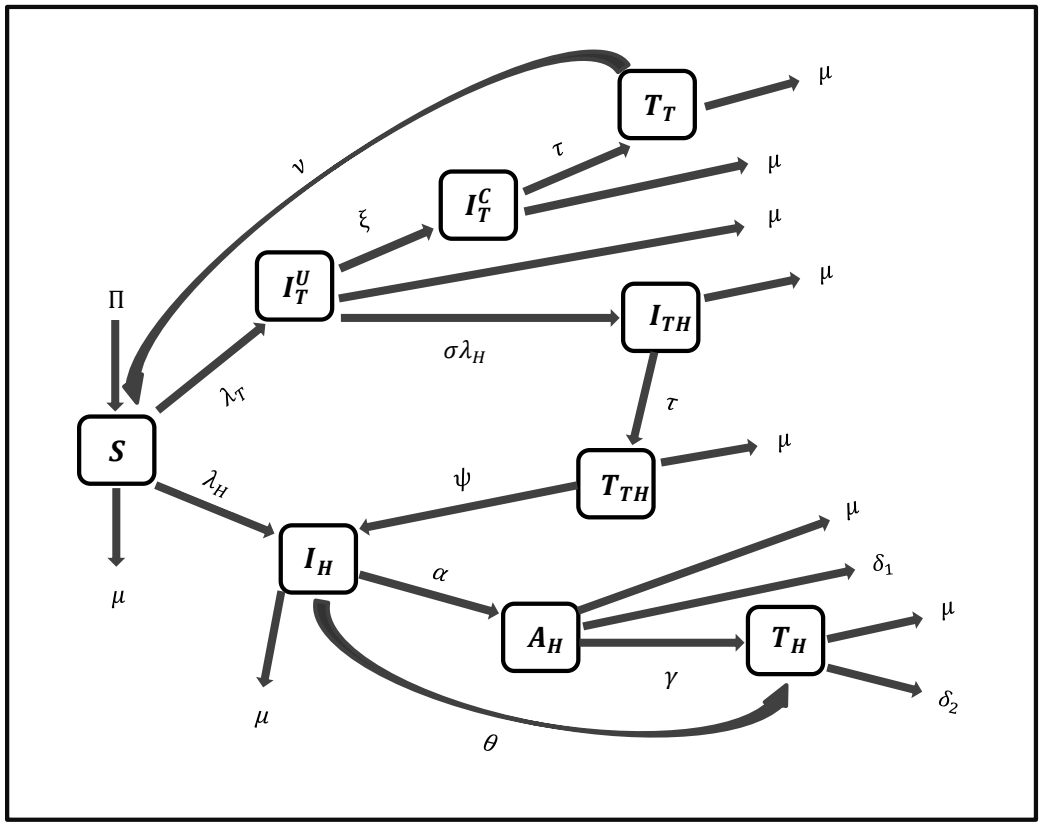

Figure 1: *Schematic Diagram of the Model* (1)

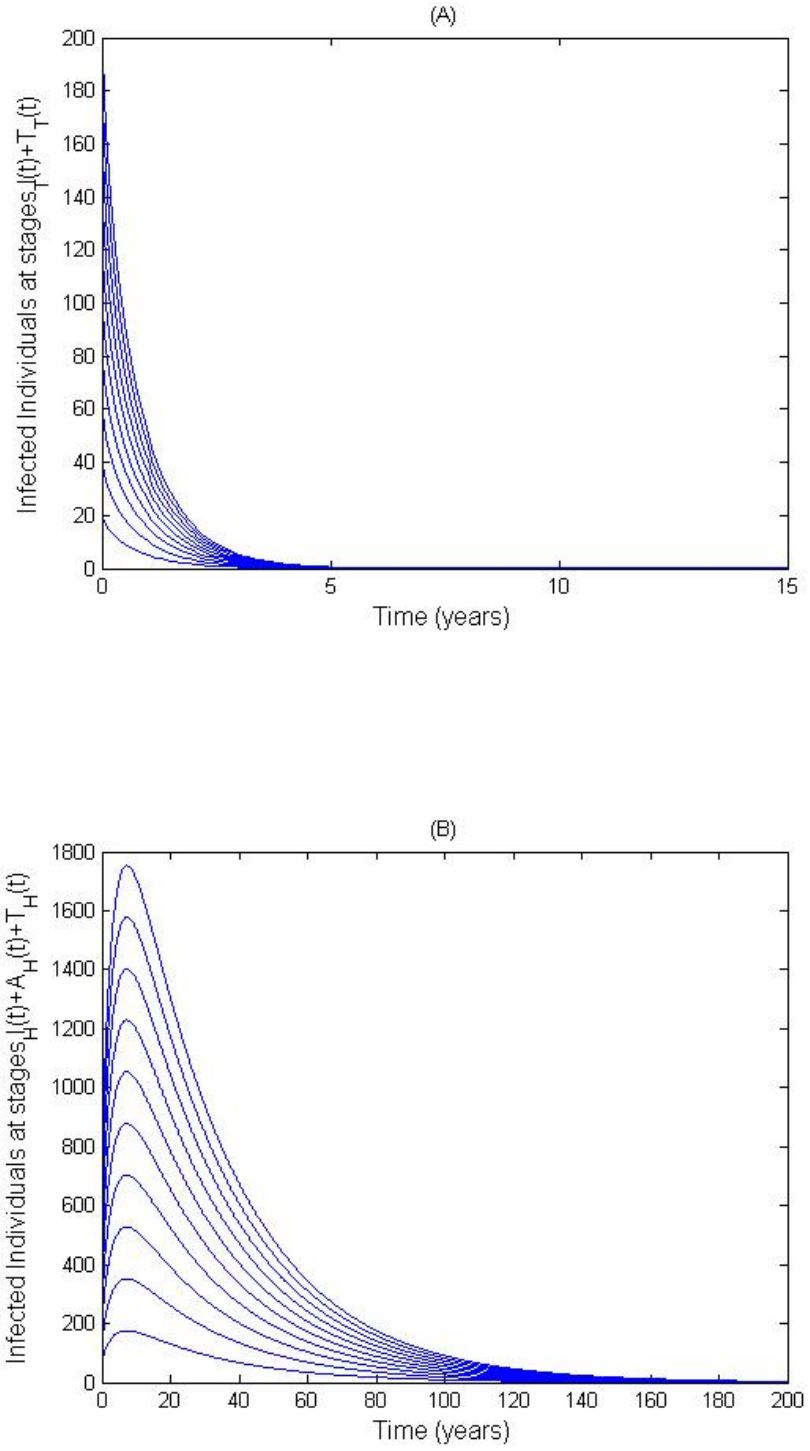

Figure 2: *Time series plot of the model (3) showing convergence to DFE. Parameter values used are given in Table 8 with* $\sigma = 0$, $\beta_T = 0.709$, $\beta_H = 0.65$ *so that* $\mathcal{R}_0 = 0.7298 < 1$.

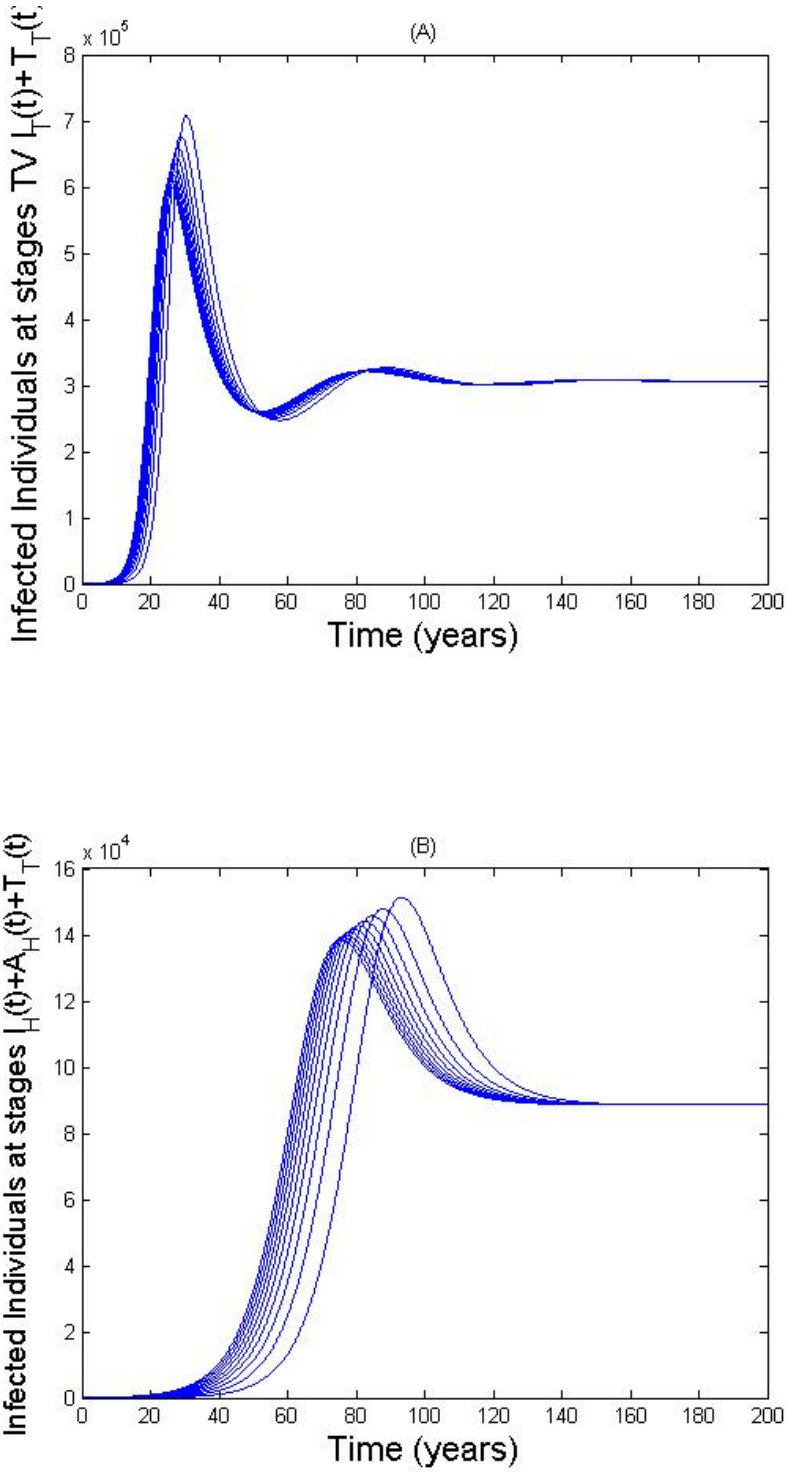

Figure 3: *Time series plot of the model (3) showing convergence to EEP. Parameter values used are given in Table 8 with* $\sigma = 0$, $\beta_T = 3.651$, $\beta_H = 1.65$ *so that* $\mathcal{R}_0 = 1.8526 > 1$.

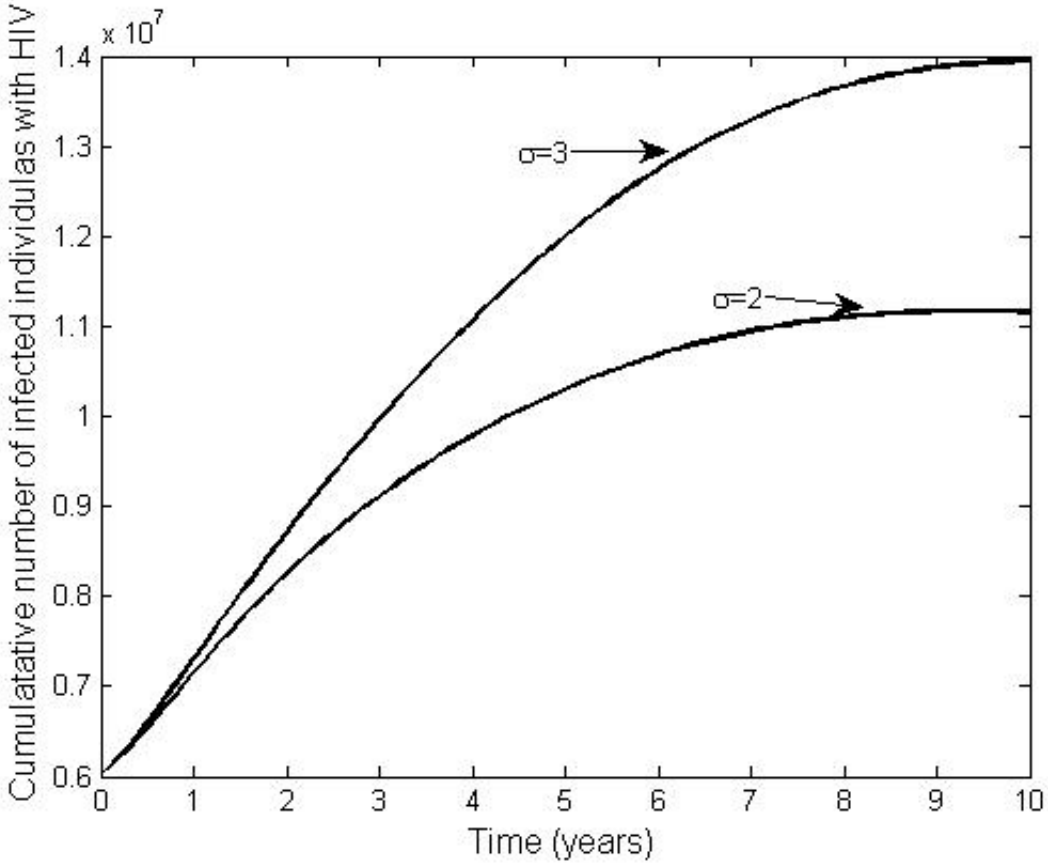

Figure 4: *Simulations of the model* (1) *showing the cumulative number of individuals infected with HIV/AIDS. Parameter values used are as given in Table 8 with varying values of* σ.

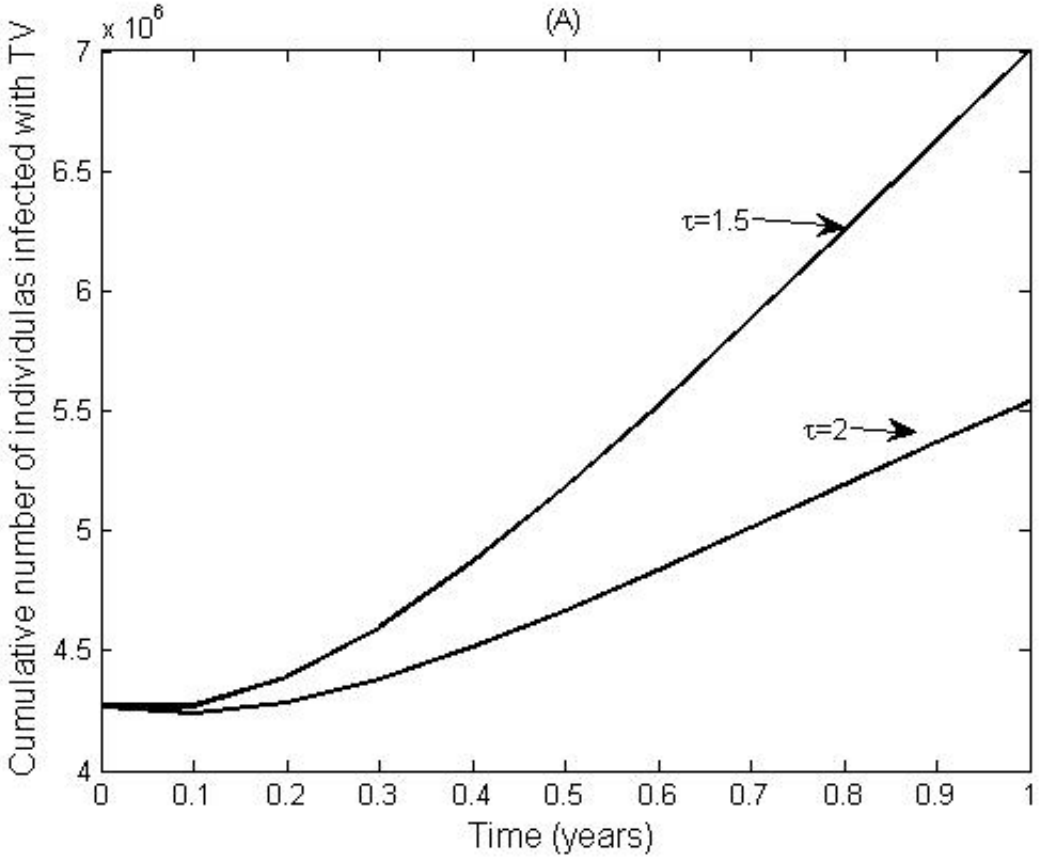

Figure 5: *Simulations of the model (3) showing the cumulative number of individuals infected with TV. Parameter values used are as given in Table 8 with varying levels of treatment* τ.

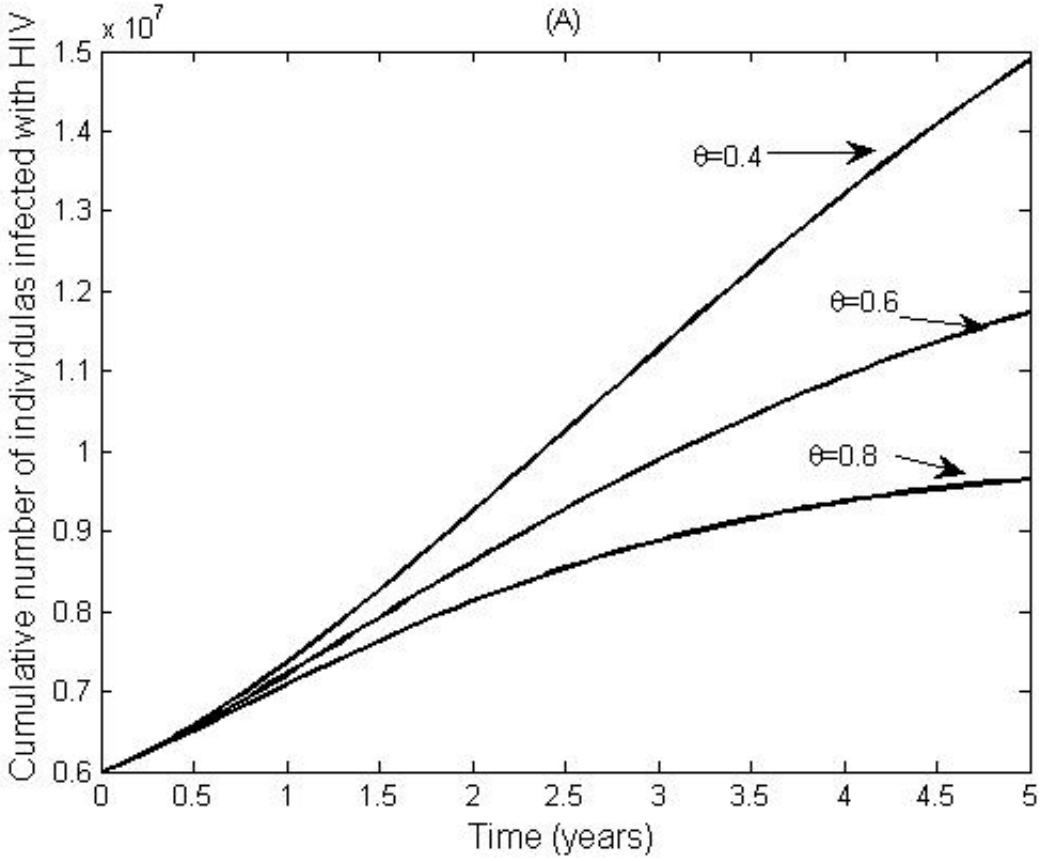

Figure 6: *Simulations of the model* (1) *showing the cumulative number of individuals infected with HIV/AIDS. Parameter values used are as given in Table 8 with varying values of* θ.

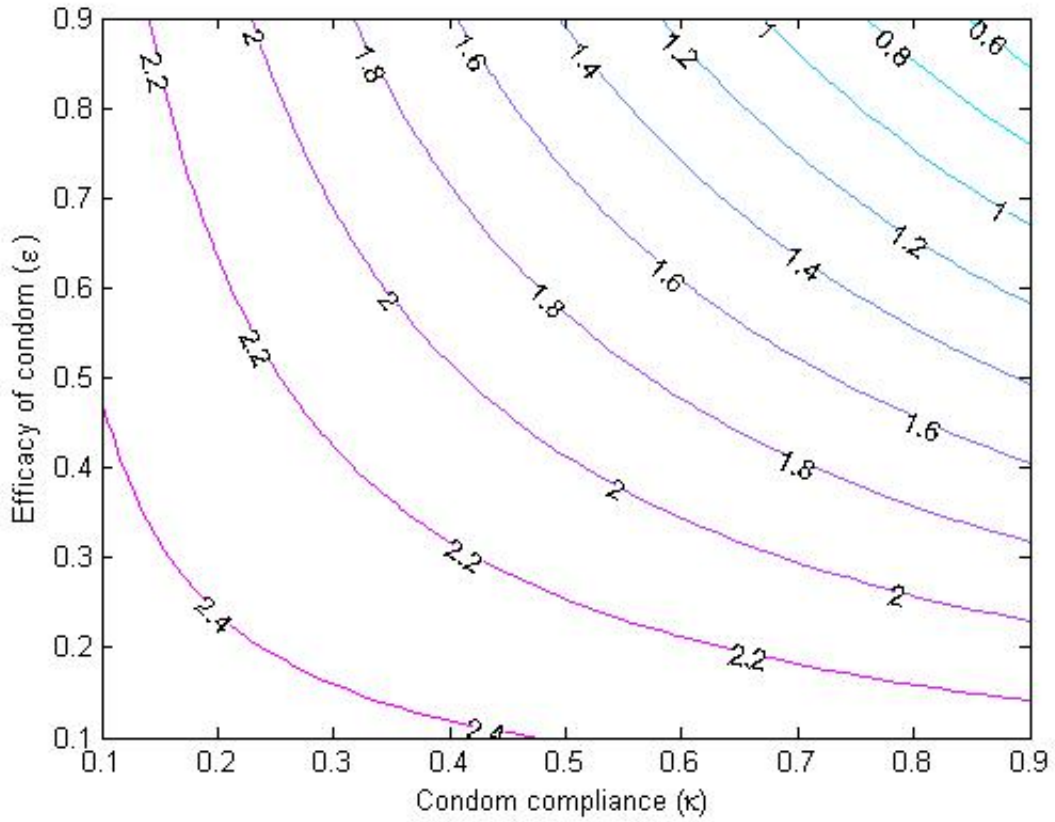

Figure 7: *Simulatins of the Model* (1) *showing a contour plot of* $\mathcal{R}_{c1}$. *Parameter values used are as given in Table 8 with* $\sigma = 0$.