APPLICATIONS OF
MODELLING AND SIMULATION
http://arqiipubl.com/ams eISSN 2600-8084 VOL 4, 2020, 217-236

Mathematical Model for Transmission Dynamics of HIV/ADS and HSV-II Co-infection

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Submitted 7 May 2020, Revised 7 June 2020, Accepted 14 June 2020.

Abstract: In this paper, a mathematical model of HIV/AID and HSV-II co-infection has been formulated and analyzed. The main aim of this study was to give awareness for the community on the transmission dynamics of the disease. The well possedness of the formulated model equations was proved and the equilibrium points of the model have been identified. Qualitative analysis of the formulated model was established using basic reproduction number. The results show that the disease free equilibrium is locally asymptotically stable if the basic reproduction is less than one. The endemic equilibrium of the model equations are considered to exist when the basic reproduction number for each disease is greater than one. Finally, numerical simulations of the model equations are carried out using the software MATLAB R2015b with ODE45 solver. Numerical simulations illustrated that as we increase force of infection, the infections increases.

Keywords: Co-infection; HIV/AIDS; HSV-II; Mathematical model; Stability analysis.

1. INTRODUCTION

Human Immunodeficiency Virus (HIV) is the causative agent of Acquired Immunodeficiency Syndrome (AIDS) [1]. HIV can be transmitted to person through the exchange of a variety of body fluids from infected individuals, such as blood, breast milk, semen, and vaginal secretions. HIV/AIDS was identified since 1983 in the United States, over 60 million people have been infected, and the WHO estimates that a death due to AIDS exceeds 25 million people. In 2015, an estimated 36.7 million people were living with HIV (including 1.8 million children), a global HIV prevalence of 0.8%, with the majority of this number living in low- and middle-income countries [2].

Herpes simplex virus type II (HSV-II) infections are the primary cause of genital herpes. Genital herpes is a chronic, life-long viral infection caused by Herpes Simplex Virus-I (HSV-I) and Herpes Simplex Virus-II (HSV-II). HSV-II can be transmitted during sexual contact with someone who has a genital HSV-II infection [3]. Worldwide, an estimated 19.2 million new HSV-II infections occurred among adults and adolescents aged 15-49 years in 2012 with the highest rates among younger age groups. HSV-II is a lifelong infection and the estimated global HSV-II prevalence of 11.3% translates into an estimated 417 million people with the infection in 2012. The prevalence of HSV-II is highest in the WHO African Region (31.5%), followed by the Region of the Americas (14.4%) [4].

Epidemiological analysis has recognized a link between the prevalence of HSV-II and HIV. In fact, individuals infected with HSV-II are at greater risk of acquiring HIV after exposure, underscoring the fact that herpes infection is an important cofactor for HIV transmission. While the prevalence of HIV is much lower than that of HSV-II, the global burden of HIV is significant [5]. In many countries, the major public health significance of HSV-II relates to its potential role in facilitating HIV transmission. HSV-II is highly widespread in most regions experiencing severe HIV epidemics, with infection rates rising sharply with age to arrive at levels of 70% or more among adult women and men in some African countries [6].

Many mathematical models were developed to control spread of HIV and HSV-II and the interactions between HSV-II and HIV. A Mhlanga *et al.* [7] developed a mathematical model for the spread of HSV-II by incorporating all the relevant biological details and poor treatment adherence. The study illustrates that though time dependent control will be effective on controlling new HSV-II cases it may not be sustainable for certain time intervals. However, Mukandavire *et al.* [8] developed a mathematical model of HIV to compares the impact of increasing condom use or HIV pre-exposure prophylaxis (PrEP) use among sex workers. The authors found that condom promotion interventions should remain the mainstay HIV prevention strategy for female sex workers (FSWs), with PrEP only being implemented once condom interventions have been maximized or to fill prevention gaps where condoms cannot be used. Furthermore, many authors [9-13] developed a mathematical model for sexually transmitted disease to control the transmission dynamics of the disease including HIV, HSV-II and Syphilis. Moreover, Abu-Raddad *et al*. [14] designed a mathematical model to assess whether HSV-II prevalence can be predictive of future HIV spread. Their results illustrated that if HSV-II prevalence is low and stable, then the risk of future HIV epidemics

is low. Also their results shown that, expanding or high HSV-II prevalence (greater than about 20%), implies a risk for a considerable HIV epidemic. Mhlanga [2] also proposed a deterministic mathematical model for the co-interaction of HIV and HSV-II in a community, with all the relevant biological detail and poor HSV-II treatment adherence. In this study threshold parameters of the model are determined and stabilities are analysed. Results from their simulation suggests that more effort should be devoted to monitoring and counseling of individuals dually infected with HIV and HSV-II as compared to those infected with HSV-II only. So far, few mathematical studies have been undertaken to model co-infection of HIV and HSV-II mathematically, but they did not considered HIV-HSV-II compartment in their studies.

This paper is organized as follows: in Section 2, we derive a model consisting of ordinary differential equations that describes the transmission dynamics of HIV-HSV-II co-infection with the fundamental assumptions. In Section 3, qualitative analysis of HIV only model was performed. In Section 4, HSV-II only model was analyzed. Similarly in Section 5 the analysis of HIV and HSV-II co-infection model was performed. In Section 6, numerical simulation of the model equations are performed by conveying various sets of numerical values to the model parameters. The conclusions are discussed in Section 7.

2. MODEL DESCRIPTION AND FORMULATION

HIV-HSV-II co-infection model divided the total population denoted by $N(t)$ into eleven classes at time t depend on their disease status. Those are:

- Susceptible individuals $S(t)$ is the class of individuals who are healthy but can contract the disease.
- Unawared HIV infected individuals $I_{uh}(t)$ consists of individuals which are unaware infected with HIV and are also infectious.
- Unawared HSV-II infected individuals $I_{us}(t)$ consists of individuals which are unaware infected with HSV-II and are also infectious.
- Unawared co-infected individuals $I_{uhs}(t)$ consists of individuals which are unaware infected with HIV-HSV-II and are also infectious.
- Screened HIV infected individuals $I_{sh}(t)$ consists of individuals which are screened infected with virus and provide treatment for those who are found to have HIV infection.
- Screened HSV-II infected individuals $I_{ss}(t)$ consists of cells which are screened infected with virus and provide treatment for those who are found to have HSV-II infection.
- Screened co-infected individuals $I_{shs}(t)$ consists of cells which are screened infected with virus and provide treatment for those who are found to have HIV-HSV-II infection.
- Individuals with AIDS, $A(t)$.
- Individuals with HSV-II, $H(t)$
- Individuals with both AIDS and HSV-II, $AH(t)$.
- Recovered individuals $R(t)$.

It is assumed that susceptible individuals are recruited into the population at a constant rate of Π. Susceptible individuals may acquire HIV infection with force of infection $\lambda_h = \frac{\beta_1(I_{uh}+q_1 I_{sh})}{N_h}$ when they come into effective contact with an infectious individual at the rate β_1 that may lead to infection. Also, susceptible individuals may acquire HSV-II infection with force of infection $\lambda_s = \frac{\beta_2(I_{us}+q_2 I_{ss})}{N_s}$ when they come into effective contact with an infectious individual at the rate β_1 that may lead to infection. The unawared HIV infected individuals are screened and join the screened HIV infected subclass at a rate α. However, some of the unawared HIV infected individual's progress to AIDS at a rate δ and others join the unawared HIV-HSV-II co-infection subclass at a rate ϕ.

Furthermore, screened HIV infected individuals' progress to AIDS at a rate ω and also joined the screened HIV-HSV-II co-infection subclass at a rate φ. Also, the unawared HIV-HSV-II co-infection individuals are screened and join the screened HIV-HSV-II co-infected subclass at a rate θ. But, some of the unawared HIV-HSV-II co-infected individual's progress to AIDS and HSV-II co-infection subclass at rate ρ. The screened HIV-HSV-II co-infection is also progress to AIDS and HSV-II co-infection subclass at rate σ. The unawared HSV-II infected individuals are screened and joined the screened HSV-II infected subclass at a rate γ and others join the unawared HIV-HSV-II co-infection subclass at rate ψ. However, some of them are progress to HSV-II subclass with rate ε and recovered naturally by body immunity at rate κ.

The screened HSV-II infected individuals are treated at rate ϵ and joined the recovered subclass with this rate. Some of them are progress to HSV-II subclass and screened co-infection of HIV-HSV-II subclass with rate η and τ respectively. AIDS individuals and HSV-II individuals are also progress to co-infection of AIDS and HSV-II subclass with rate ν and χ respectively. Finally, recovered individuals revert to susceptible subclass after losing their immunity at a rate ϑ. All individuals suffer natural mortality at a rate μ and sick individuals die of AIDS, HSV-II and AIDS-HSV-II co-infection at rate ξ. The schematic diagram that describes the flow of the model is shown Figure 1.

The above model description and assumptions can be written as linear system of differential equation as:

$$\frac{dS}{dt} = \Pi + \vartheta R - (\lambda_h + \lambda_s + \mu)S$$

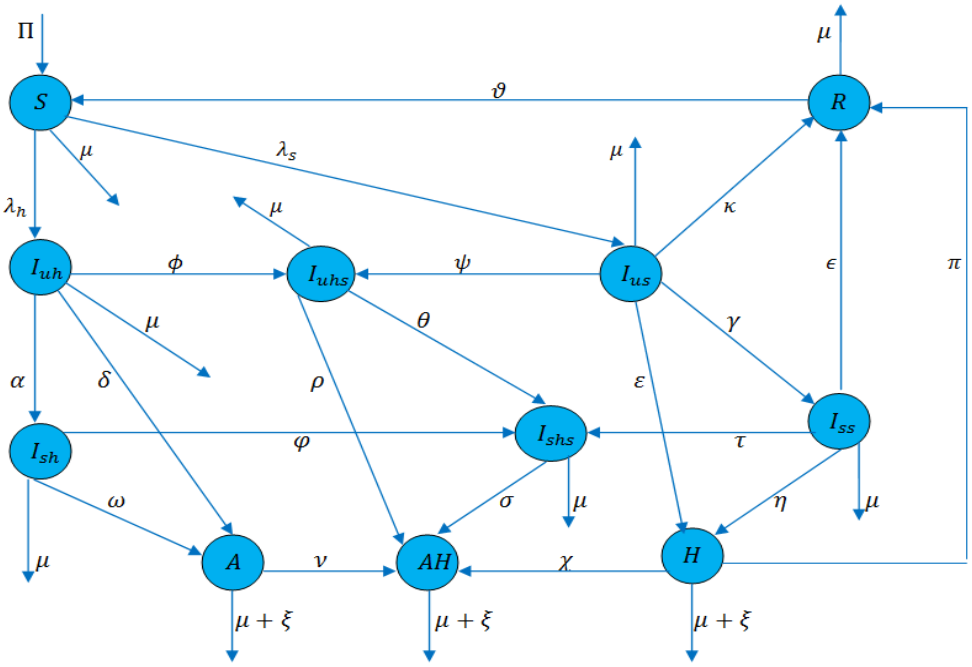

Figure 1. Schematic diagram for HIV-HSV-II co-infection model

$$\begin{aligned}
\frac{dI_{uh}}{dt} &= \lambda_h S - (\phi + \alpha + \delta + \mu) I_{uh} \\
\frac{dI_{us}}{dt} &= \lambda_s S - (\psi + \varepsilon + \gamma + \kappa + \mu) I_{uh} \\
\frac{dI_{uhs}}{dt} &= \phi I_{uh} + \psi I_{us} - (\rho + \theta + \mu) I_{uhs} \\
\frac{dI_{sh}}{dt} &= \alpha I_{uh} - (\varphi + \omega + \mu) I_{sh} \\
\frac{dI_{ss}}{dt} &= \gamma I_{us} - (\tau + \eta + \epsilon + \mu) I_{ss} \\
\frac{dI_{shs}}{dt} &= \theta I_{uhs} + \varphi I_{sh} + \tau I_{ss} - (\sigma + \mu) I_{shs} \\
\frac{dA}{dt} &= \delta I_{uh} + \omega I_{sh} - (\nu + \mu + \xi) A \\
\frac{dH}{dt} &= \varepsilon I_{us} + \eta I_{ss} - (\chi + \pi + \mu + \xi) H \\
\frac{dAH}{dt} &= \rho I_{uhs} + \sigma I_{shs} + \nu A + \chi H - (\mu + \xi) AH
\end{aligned} \tag{1}$$

With initial conditions, $S(0) = S_0, I_{uh}(0) = I_{uh0}$, $I_{us}(0) = I_{us0}$, $I_{uhs}(0) = I_{uhs0}$, $I_{sh}(0) = I_{sh0}$, $I_{ss}(0) = I_{ss0}, I_{shs}(0) = I_{shs0}, A(0) = A_0, H(0) = H_0, AH(0) = AH_0, R(0) = R_0$.

3. ANALYSIS OF HIV ONLY MODEL

In this section, the analysis of the transmission dynamics of HIV only model is considered.

$$\begin{aligned}
\frac{dS}{dt} &= \Pi - (\lambda_h + \mu) S \\
\frac{dI_{uh}}{dt} &= \lambda_h S - (\alpha + \delta + \mu) I_{uh} \\
\frac{dI_{sh}}{dt} &= \alpha I_{uh} - (\omega + \mu) I_{sh} \\
\frac{dA}{dt} &= \delta I_{uh} + \omega I_{sh} - (\mu + \xi) A
\end{aligned} \tag{2}$$

3.1 Invariant Region

Theorem 1: The total population size N_h of the system of model in Equation (2) is bounded in the invariant region Ω_1. That is, size of N_h is bounded for all t.

Proof: In model Equation (2) the total population of N_h is given as

$$N_h = S + I_{uh} + I_{us} + A$$

Differentiating N_h both sides with respect to t leads to

$$\frac{dN_h}{dt} = \frac{dS}{dt} + \frac{dI_{uh}}{dt} + \frac{dI_{us}}{dt} + \frac{dA}{dt} \tag{3}$$

Substituting model Equation (2) into Equation (3), we can get

$$\frac{dN_h}{dt} = \Pi - \mu N_h - \xi A \tag{4}$$

In the absence of mortality due to AIDS($\xi = 0$), then Equation (4) become

$$\frac{dN_h}{dt} \leq \Pi - \mu N_h \tag{5}$$

Rearranging and integrating both sides of Equation (5), we get

$$\int \frac{dN_h}{\Pi - \mu N_h} \leq \int dt$$
$$\Leftrightarrow \frac{-1}{\mu}\ln(\Pi - \mu N_h) \leq t + c_1 \text{ , where } c_1 \text{ is integration constant}$$
$$\Rightarrow \ln(\Pi - \mu N_h) \geq -\mu t + c_2, \text{ where } c_2 = -\mu c_1$$
$$\Rightarrow (\Pi - \mu N_h) \geq ce^{-\mu t}, \text{ where } c = e^{-c_2}$$

Then, applying initial condition, $N_h(0) = N_{h0}$, we obtain

$$\mathrm{c} = \Pi - \mu N_{h0}$$
$$\Rightarrow \Pi - \mu N_h \geq (\Pi - \mu N_{h0})e^{-\mu t}$$
$$\Rightarrow N_h \leq \frac{\Pi}{\mu} - [\frac{\Pi - \mu N_h}{\mu}]e^{-\mu t} \tag{6}$$

As $t \to \infty$ in Equation (6), the population size $N_h(t) \to \frac{\Pi}{\mu}$ which implies that $0 \leq N_h(t) \leq \left(\frac{\Pi}{\mu}\right)$. Thus, the feasible solution set of the model Equation (2) enters and remains in the region:

$$\Omega_1 = \{(S,\ I_{uh},\ I_{sh},\quad A\ ) \in \Re_+^4\ :\ N_h \leq \Pi/\mu\}$$

Therefore, the model Equation (2) is wellposed epidemiologically and mathematically. Hence, it is sufficient to study the dynamics of the model in the region Ω_1.

3.2 Existence of Solution

Lemma 1: Solutions of the model Equation (2) together with the initial conditions$S(0) > 0,\ I_{uh}(0) > 0,\ I_{sh}(0) > 0,\ A(0) > 0$ exist in $\mathbb{R}_+^4$i.e., the solution of the model variables $S(t),\ I_{uh}(t),\ I_{sh}(t)$and $A(t)$ exist for all t and will remain in $\mathbb{R}_+^4$.

Proof: The right hand sides of the system of Equation (2) can be expressed as follows:

$$f_1(S,\quad I_{uh},\quad I_{sh},\quad A) = \Pi - (\lambda_h + \mu)$$
$$f_2(S,\quad I_{uh},\quad I_{sh},\quad A) = \lambda_h S - (\alpha + \delta + \mu)I_{uh}$$
$$f_3(S,\quad I_{uh},\quad I_{sh},\quad A) = \alpha I_{uh} - (\omega + \mu)I_{sh}$$
$$f_4(S,\quad I_{uh},\quad I_{sh},\quad A) = \rho I_{uh} + \omega I_{sh} - (\mu + \xi)A$$

According to Derrick and Groosman theorem, let Ω_1 denote the region $\Omega_1 = \{(S,\quad I_{uh},\quad I_{sh},\quad A) \in \Re_+^4\ :\ N_h \leq \Pi/\mu\}$. Then Equation (2) have a unique solution if $(\partial f_i)/(\partial x_j),\ i,j = 1,2,3,4$ are continuous and bounded inΩ_1. Here, $x_1 = S,\ x_2 = I_{uh},\ x_3 = I_{sh}$ and $x_4 = A$. The continuity and the boundedness are verified as in Table 1. Thus, all the partial derivatives $(\partial f_i)/(\partial x_j), i,j = 1,2,3,4$ exist, continuous and bounded in Ω_1. Hence, by Derrick and Groosman theorem, a solution for the model in Eqaution (2) exists and is unique.

Table 1. Continuity and boundedness of the model solution

$\lvert(\partial f_1)/(\partial S)\rvert = \lvert-(\lambda_h + \mu)\rvert < \infty$ $\lvert(\partial f_1)/(\partial I_{uh})\rvert = \left\lvert\frac{-\beta_1 S}{N_h}\right\rvert < \infty$ $\lvert(\partial f_1)/(\partial I_{us})\rvert = \left\lvert\frac{-\beta_1 q_1 S}{N_h}\right\rvert < \infty$ $\lvert(\partial f_1)/(\partial A)\rvert = 0\ < \infty.$	$\lvert(\partial f_2)/(\partial S)\rvert = \lvert\lambda_h\rvert < \infty$ $\lvert(\partial f_2)/(\partial I_{uh})\rvert = \left\lvert\frac{\beta_1 S}{N_h} - (\alpha + \delta + \mu)\right\rvert < \infty$ $\lvert(\partial f_2)/(\partial I_{us})\rvert = \left\lvert\frac{\beta_1 q_1 S}{N_h}\right\rvert < \infty$ $\lvert(\partial f_2)/(\partial A)\rvert = 0\ < \infty.$
$\lvert(\partial f_3)/(\partial S)\rvert = 0 < \infty$ $\lvert(\partial f_3)/(\partial I_{uh})\rvert = \lvert\alpha\rvert < \infty$ $\lvert(\partial f_3)/(\partial I_{us})\rvert = \lvert-(\omega + \mu)\rvert < \infty$ $\lvert(\partial f_3)/(\partial A)\rvert = 0\ < \infty$	$\lvert(\partial f_4)/(\partial S)\rvert = 0 < \infty$ $\lvert(\partial f_4)/(\partial I_{uh})\rvert = \lvert\delta\rvert < \infty$ $\lvert(\partial f_4)/(\partial I_{us})\rvert = \lvert\omega\rvert < \infty$ $\lvert(\partial f_4)/(\partial A)\rvert = \lvert-(\mu + \xi)\rvert < \infty$

3.3 Positivity of Solution

The solution of the system remains positive at any point in time t, if the initial values of all the variables are positive.

Theorem 2: Let $\Omega_1 = \{(S,\ I_{uh},\ I_{sh},\ A) \in \mathbb{R}_+^4; S_0 > 0,\ I_{uh_0} > 0,\ I_{sh_0} > 0,\ A_0 > 0\}$ then the solutions of $\{S,\ I_{uh},\ I_{sh},\ A\}$ are positive for all $t \geq 0$.

Proof: Positivity is verified separately for each of the model $S(t)$, $I_{uh}(t)$, $I_{sh}(t)$, and $A(t)$.

Positivity of $S(t)$: From model Equation (2) we have:

$$\frac{dS}{dt} = \Pi - (\lambda_h + \mu)S$$

Eliminating the positive terms Π we get,

$$\Leftrightarrow \frac{dS}{dt} \geq -(\lambda_h + \mu)S$$

Using variables separable method we get,

$$\Rightarrow \frac{dP}{S} \geq -(\lambda_h + \mu)dt,$$

Integrating both side we can get,

$$\Rightarrow \int \frac{dS}{S} \geq -\int (\lambda_h + \mu)\, dt$$

$$\Rightarrow \ln S \geq -(\lambda_h + \mu)t + c_4, \text{ where } c_4 \text{ is integration constant}$$

$$\Rightarrow S(t) \geq S_0 e^{-(\lambda_h+\mu)t}, S_0 = e^{c_4} \text{ and } e^{-(\lambda_h+\mu)t} \geq 0, \text{ for all } t \geq 0.$$

Hence, it can be concluded that $S(t) \geq 0$.

Positivity of I_{uh} from model Equation (2) we have:

$$\frac{dI_{uh}}{dt} = \lambda_h S - (\alpha + \delta + \mu)I_{uh}$$

Eliminating the positive terms $(\lambda_h S)$we get,

$$\Leftrightarrow \frac{dI_{uh}}{dt} \geq -(\alpha + \delta + \mu)I_{uh}$$

Using variables separable method we get,

$$\Rightarrow \frac{dI}{I_{uh}} \geq -(\alpha + \delta + \mu)dt,$$

Integrating both side we can get,

$$\Rightarrow \int \frac{dI}{I_{uh}} \geq -\int (\alpha + \delta + \mu)\, dt$$

$$\Rightarrow \ln I_{uh} \geq -(\alpha + \delta + \mu)t + c_5, \text{ where } c_5 \text{ is integration constant}$$

$$\Rightarrow I_{uh}(t) \geq I_{uh0} e^{-(\alpha+\delta+\mu)t}, I_{uh0} = e^{c_5} \text{ and } e^{-(\alpha+\delta+\mu)t} \geq 0, \text{ for all } t \geq 0.$$

Hence, it can be concluded that $I_{uh}(t) \geq 0$.

Positivity of $I_{sh}(t)$: From model Equation (2) we have:

$$\frac{dI_{sh}}{dt} = \alpha I_{uh} - (\omega + \mu)I_{sh}$$

Eliminating the positive terms (αI_{uh}) we get,

$$\Leftrightarrow \frac{dI_{sh}}{dt} \geq -(\omega + \mu)I_{sh}$$

Using variables separable method we get,

$$\Rightarrow \frac{dI_{sh}}{I_{sh}} \geq -(\omega + \mu)dt$$

Integrating both side we can get,

$$\Rightarrow \int \frac{dI_{sh}}{I_{sh}} \geq -\int (\omega + \mu)\, dt$$

$$\Rightarrow \ln I_{sh} \geq -(\omega + \mu)t + c_6, \text{ where } c_6 \text{ is integration constant}$$

$$\Rightarrow I_{sh}(t) \geq I_{sh0} e^{-(\omega+\mu)t}, I_{\mathrm{sh0}} = e^{c_6} \text{ and } e^{-(\omega+\mu)t} \geq 0, \text{ for all } t \geq 0.$$

Hence, it can be concluded that $I_{sh}(t) \geq 0$.

Positivity of $A(t)$: From model Equation (2) we have:

$$\frac{dA}{dt} = \rho I_{uh} + \omega I_{sh} - (\mu + \xi)A$$

Eliminating the positive terms $(\rho I_{uh} + \omega I_{sh})$ we get,

$$\Leftrightarrow \frac{dA}{dt} \geq -(\mu + \xi)A,$$

Using variables separable method we get,

$$\Rightarrow \frac{dA}{A} \geq -(\mu + \xi)dt,$$

Integrating both side we can get,

$$\Rightarrow \int \frac{dA}{A} \geq -\int (\mu + \xi)\, dt$$

$$\Rightarrow \ln A \geq -(\mu + \xi)t + c_7, \text{ where } c_7 \text{ is integration constant}$$

$$\Rightarrow A(t) \geq A_0 e^{-(\mu+\xi)t}, A_0 = e^{c_7} \text{ and } e^{-(\mu+\xi)t} \geq 0, \text{ for all } t \geq 0.$$

Hence, it can be concluded that $A(t) \geq 0$. Therefore, the model variables $S(t), I_{uh}(t), I_{sh}(t)$ and $A(t)$ representing population sizes of various types of cells are positive quantities and will remain in $\mathbb{R}_+^4$ for all t.

3.4 Stability Analysis of the Disease-Free Equilibrium (DFE)

The disease free equilibrium of the HIV only model Equation (2) is determined by equating to zero. Then we get:

$$E_1 = \left\{\left(\frac{\Pi}{\mu}\right),\ 0,\ 0,\ 0,\ 0\right\}$$

The local stability of the DFE, E_1 can be established using the next generation operator method in Van den Driessche and Watmouth [15] on the system in Equation (2). To do this the basic reproduction number can be determined depends on the definition of infected and uninfected compartments. The model Equation (2) are rewritten starting with newly infective classes:

$$\begin{aligned}\frac{dI_{uh}}{dt} &= \lambda_h S - (\alpha+\delta+\mu)I_{uh}\\ \frac{dI_{sh}}{dt} &= \alpha I_{uh} - (\omega+\mu)I_{sh}\\ \frac{dA}{dt} &= \rho I_{uh} + \omega I_{sh} - (\mu+\xi)A\end{aligned}$$

Then by the principle of next-generation matrix, we obtained

$$f_i = \begin{bmatrix}\frac{\beta_1(I_{uh}+q_1 I_{sh})S}{N_h}\\ 0\\ 0\end{bmatrix} \text{ and } v_i = \begin{bmatrix}(\alpha+\delta+\mu)I_{uh}\\ -\alpha I_{uh} + (\omega+\mu)I_{sh}\\ -\rho I_{uh} - \omega I_{sh} + (\mu+\xi)A\end{bmatrix}$$

The Jacobian matrices of f_i and v_i evaluated at DFE are given by F and V, respectively, such that

$$F = \begin{bmatrix}\beta_1 & \beta_1 q_1 & 0\\ 0 & 0 & 0\\ 0 & 0 & 0\end{bmatrix} \text{ and } V = \begin{bmatrix}(\alpha+\delta+\mu) & 0 & 0\\ -\alpha & (\omega+\mu) & 0\\ -\delta & -\omega & (\mu+\xi)\end{bmatrix}$$

It can be verified that the matrix V is non-singular as its determinant $det[V] = (\alpha+\delta+\mu)(\omega+\mu)(\mu+\xi)$ is non-zero and after some algebraic computations its inverse matrix is constructed as

$$V^{-1} = \begin{bmatrix}\frac{1}{(\alpha+\delta+\mu)} & 0 & 0\\ \frac{\alpha}{(\alpha+\delta+\mu)(\omega+\mu)} & \frac{1}{(\omega+\mu)} & 0\\ \frac{\alpha\omega+\delta(\omega+\mu)}{(\alpha+\delta+\mu)(\omega+\mu)(\mu+\xi)} & \frac{\theta}{(\omega+\mu)(\mu+\xi)} & \frac{1}{(\mu+\xi)}\end{bmatrix}.$$

The product of the matrices F and V^{-1} can be computed as:

$$\begin{aligned}FV^{-1} &= \begin{bmatrix}\beta_1 & \beta_1 q_1 & 0\\ 0 & 0 & 0\\ 0 & 0 & 0\end{bmatrix}\begin{bmatrix}\frac{1}{(\alpha+\delta+\mu)} & 0 & 0\\ \frac{\alpha}{(\alpha+\delta+\mu)(\omega+\mu)} & \frac{1}{(\omega+\mu)} & 0\\ \frac{\alpha\omega+\delta(\omega+\mu)}{(\alpha+\delta+\mu)(\omega+\mu)(\mu+\xi)} & \frac{\omega}{(\omega+\mu)(\mu+\xi)} & \frac{1}{(\mu+\xi)}\end{bmatrix}\\ &= \begin{bmatrix}\frac{\beta_1}{(\alpha+\delta+\mu)} + \frac{\beta_1 q_1 \alpha}{(\alpha+\delta+\mu)(\omega+\mu)} & \frac{\beta_1 q_1}{(\omega+\mu)} & 0\\ 0 & 0 & 0\\ 0 & 0 & 0\end{bmatrix}\end{aligned}$$

Now it is possible to calculate the eigenvalue to determine the basic reproduction number $\Re_h$ by taking the spectral radius of the matrix FV^{-1}. Thus, the eigenvalues are computed by evaluating $det[FV^{-1} - \lambda I] = 0$ or equivalently solving

$$\begin{aligned}&\begin{vmatrix}\frac{\beta_1}{(\alpha+\delta+\mu)} + \frac{\beta_1 q_1 \alpha}{(\alpha+\delta+\mu)(\omega+\mu)} - \lambda & \frac{\beta_1 q_1}{(\omega+\mu)} & 0\\ 0 & -\lambda & 0\\ 0 & 0 & -\lambda\end{vmatrix} = 0\\ &\Rightarrow \lambda^2\left[\frac{\beta_1(\omega+\mu)+\beta_1 q_1 \alpha(\alpha+\delta+\mu)}{(\alpha+\delta+\mu)(\omega+\mu)} - \lambda\right] = 0\\ &\Rightarrow \lambda_1 = \left[\frac{\beta_1(\omega+\mu)+\beta_1 q_1 \alpha(\alpha+\delta+\mu)}{(\alpha+\delta+\mu)(\omega+\mu)}\right],\ \lambda_2 = \lambda_3 = 0.\end{aligned}$$

However, the dominant eigenvalue here is $\lambda_1 = \left[\frac{\beta_1(\omega+\mu)+\beta_1 q_1 \alpha(\alpha+\delta+\mu)}{(\alpha+\delta+\mu)(\omega+\mu)}\right]$ and is the spectral radius as the threshold value or the basic reproductive number. Thus, it can be concluded that the reproduction number of the model is $\Re_h =$

$\left[\frac{\beta_1(\omega+\mu)+\beta_1 q_1\alpha(\alpha+\delta+\mu)}{(\alpha+\delta+\mu)(\omega+\mu)}\right]$. Further using Theorem 2 in Van den Driessche and Watmouth [15], the DFE is locally asymptotically stable if $\Re_h < 1$ and unstable is $\Re_h > 1$.

3.5 Existence and Stability Analysis of Endemic Equilibrium

Lemma 2. The HIV only model Equation (2) has a unique endemic equilibrium if and only if the basic reproduction number $\Re_h > 1$.

Proof. Let the endemic equilibrium point of the model Equation (2) be denoted by

$$E_2 = (S^*, \quad I_{uh}^*, \quad I_{sh}^*, \quad A^*)$$

and consider the force of infection

$$\lambda_h^* = \frac{\beta_1[I_{uh}^* + q_1 I_{sh}^*]}{N_h} \tag{7}$$

Solving the model in Equations (2) by setting the right hand sides equal to zero, we get,

$$S^* = \frac{\Pi}{(\lambda_h^*+\mu)}, \quad I_{uh}^* = \frac{\Pi\lambda_h^*}{(\lambda_h^*+\mu)(\alpha+\delta+\mu)},$$
$$I_{sh}^* = \frac{\alpha\Pi\lambda_h^*}{(\lambda_h^*+\mu)(\omega+\mu)} A^* = \frac{\alpha\Pi\lambda_h^*(\omega+\mu)+\omega\alpha\Pi(\lambda_h^*+\mu)}{(\lambda_h^*+\mu)(\alpha+\delta+\mu)(\omega+\mu)(\mu+\xi)} \tag{8}$$

Substituting Eqaution (8) in Equation (7) we get

$$\lambda_h^* = \frac{\beta_1\Pi\lambda_h^*}{(\lambda_h^*+\mu)(\alpha+\delta+\mu)} + \frac{\beta_1 q_1\alpha\Pi\lambda_h^*}{(\lambda_h^*+\mu)(\omega+\mu)}$$
$$(\lambda_h^*)^2 + \lambda_h^*[\mu - \Pi\Re_h] = 0$$

Hence, the HIV force of infection, λ_h^*, satisfies the following polynomial

$$p(\lambda_h^*) = (\lambda_h^*)^2 + D\lambda_h^* = 0, \text{ where } D = \mu - \Pi\Re_h$$

By mathematical induction, $D \geq 0$ whenever the basic reproduction number is less than one ($\Re_h < 1$). This implies that $\lambda_h^* = -1/D \leq 0$. Therefore, the HIV model has no endemic equilibrium whenever $\Re_h < 1$. Hence, the analysis describes the impossibility of backward bifurcation in the HIV only model, implies there is no existence of endemic equilibrium whenever the basic reproduction number is less than one ($\Re_h < 1$).

4. ANALYSIS OF HSV-II ONLY MODEL

In this section, the analysis of the transmission dynamics of HSV-II only model is considered.

$$\begin{aligned}
\frac{dS}{dt} &= \Pi + \vartheta R - (\lambda_s + \mu)S \\
\frac{dI_{us}}{dt} &= \lambda_s S - (\varepsilon + \gamma + \kappa + \mu)I_{us} \\
\frac{dI_{ss}}{dt} &= \gamma I_{us} - (\eta + \epsilon + \mu)I_{ss} \\
\frac{dH}{dt} &= \varepsilon I_{us} + \eta I_{ss} - (\pi + \mu + \xi)H \\
\frac{dR}{dt} &= \kappa I_{us} + \epsilon I_{ss} + \pi H - (\vartheta + \mu)R
\end{aligned} \tag{9}$$

4.1 Invariant Region

Theorem 3: The total population size N_s of the system of model Equation (9) is bounded in the invariant region Ω_2. That is, size of N_s is bounded for all t.

Proof: In model Equation (9), the total population of N_s is given as

$$N_s = S + I_{us} + I_{ss} + H + R$$

Differentiating N_s both sides with respect to t leads to

$$\frac{dN_s}{dt} = \frac{dS}{dt} + \frac{dI_{us}}{dt} + \frac{dI_{ss}}{dt} + \frac{dH}{dt} + \frac{dR}{dt} \tag{10}$$

Substituting model Equation (9) into Equation (10), we can get

$$\frac{dN_s}{dt} = \Pi - \mu N_s - \xi H$$

In the absence of mortality due to HSV-II ($\xi = 0$), then Equation (11) become

$$\frac{dN_s}{dt} \leq \Pi - \mu N_s$$

Rearranging and integrating both sides of Equation (12), we get

$$\int \frac{dN_s}{\Pi - \mu N_s} \leq \int dt$$

$$\Leftrightarrow \frac{-1}{\mu}\ln(\Pi - \mu N_s) \le t + c_7 \text{ , where } c_7 \text{ is integration constant}$$
$$\Rightarrow \ln(\Pi - \mu N_s) \ge -\mu t + c_8, \text{ where } c_8 = -\mu c_7$$
$$\Rightarrow (\Pi - \mu N_s) \ge ce^{-\mu t}, \qquad \text{where } c = e^{-c_8}$$

Then, applying initial condition$N_s(0) = N_{s0}$, we obtain

$$\mathrm{c} = \Pi - \mu N_{s0}$$
$$\Rightarrow \Pi - \mu N_s \ge (\Pi - \mu N_{s0})e^{-\mu t}$$
$$\Rightarrow N_s \le \frac{\Pi}{\mu} - [\frac{\Pi - \mu N_s}{\mu}]e^{-\mu t} \tag{13}$$

As $t \to \infty$ in Equation (13), the population size $N_s(t) \to \frac{\Pi}{\mu}$ which implies that $0 \le N_s(t) \le \left(\frac{\Pi}{\mu}\right)$. Thus, the feasible solution set of the model Equation (9) enters and remains in the region:

$$\Omega_2 = \{(S,\ I_{us},\ I_{ss},\quad H,\ R\ ) \in \Re_+^5\ :\ N_s \le \Pi/\mu\}$$

Therefore, the model in Equation (9) is wellposed epidemiologically and mathematically. Hence, it is sufficient to study the dynamics of the model in the region Ω_2.

4.2 Existence of Solution

Lemma 3: Solutions of the model Equations (9) together with the initial conditions, $S(0) > 0,\ I_{us}(0) > 0,\ I_{ss}(0) > 0,\ H(0) > 0,\ R(0) > 0$ exist in $\mathbb{R}_+^5$ i.e., the solution of the model variables $S(t),\ I_{us}(t),\ I_{ss}(t)$, $H(t)$ and $R(t)$ exist for all t and will remain in$\mathbb{R}_+^5$.

Proof: The right hand sides of the system of Equations (9) can be expressed as follows:

$$f_5(S,\ I_{us},\ I_{ss},\ H,\ R) = \Pi + \vartheta \mathrm{R} - (\lambda_s + \mu)S$$
$$f_6(S,\ I_{us},\ I_{ss},\ H,\ R) = \lambda_s S - (\varepsilon + \gamma + \kappa + \mu)I_{us}$$
$$f_7(S,\ I_{us},\ I_{ss},\ H,\ R) = \gamma I_{us} - (\eta + \epsilon + \mu)I_{ss}$$
$$f_8(S,\ I_{us},\ I_{ss},\ H,\ R) = \varepsilon I_{us} + \eta I_{ss} - (\pi + \mu + \xi)H$$
$$f_9(S,\ I_{us},\ I_{ss},\ H,\ R) = \kappa I_{us} + \epsilon I_{ss} + \pi H - (\vartheta + \mu)R$$

According to Derrick and Groosman theorem, let Ω_2 denote the region $\Omega_2 = \{(S,\ I_{us},\ I_{ss},\ H,\ R) \in \Re_+^5\ :\ N_s \le \Pi/\mu\}$. Then Equations (9) have a unique solution if $(\partial f_i)/(\partial x_j)$, $i,j = 1,2,3,4,5$ are continuous and bounded in Ω_2. Here, $x_1 = S,\ x_2 = I_{us},\ x_3 = I_{ss},\ x_4 = H$ and $x_5 = R$. The continuity and the boundedness are verified in Table 2. Thus, all the partial derivatives $(\partial f_i)/(\partial x_j), i,j = 1,2,3,4,5$ exist, continuous and bounded in Ω_2. Hence, by Derrick and Groosman theorem, a solution for the model (9) exists and is unique.

Table 2. Continuity and boundedness of the model solution

$\lvert(\partial f_5)/(\partial S)\rvert = \lvert-(\lambda_s + \mu)\rvert < \infty$ $\lvert(\partial f_5)/(\partial I_{us})\rvert = \left\lvert\frac{-\beta_2 S}{N_s}\right\rvert < \infty$ $\lvert(\partial f_5)/(\partial I_{ss})\rvert = \left\lvert\frac{-\beta_2 q_2 S}{N_s}\right\rvert < \infty$ $\lvert(\partial f_5)/(\partial H)\rvert = 0\ < \infty$ $\lvert(\partial f_5)/(\partial R)\rvert = \lvert\vartheta\rvert < \infty$	$\lvert(\partial f_6)/(\partial S)\rvert = \lvert\lambda_s\rvert < \infty$ $\lvert(\partial f_6)/(\partial I_{us})\rvert = \left\lvert\frac{\beta_2 S}{N_s} - (\varepsilon + \gamma + \kappa + \mu)\right\rvert < \infty$ $\lvert(\partial f_6)/(\partial I_{ss})\rvert = \left\lvert\frac{\beta_2 q_2 S}{N_s}\right\rvert < \infty$ $\lvert(\partial f_6)/(\partial H)\rvert = 0\ < \infty$ $\lvert(\partial f_6)/(\partial R)\rvert = 0\ < \infty$
$\lvert(\partial f_7)/(\partial S)\rvert = 0 < \infty$ $\lvert(\partial f_7)/(\partial I_{us})\rvert = \lvert\gamma\rvert < \infty$ $\lvert(\partial f_7)/(\partial I_{ss})\rvert = \lvert-(\eta + \epsilon + \mu)\rvert < \infty$ $\lvert(\partial f_7)/(\partial H)\rvert = 0\ < \infty$ $\lvert(\partial f_7)/(\partial R)\rvert = 0\ < \infty$	$\lvert(\partial f_8)/(\partial S)\rvert = 0 < \infty$ $\lvert(\partial f_8)/(\partial I_{us})\rvert = \lvert\varepsilon\rvert < \infty$ $\lvert(\partial f_8)/(\partial I_{ss})\rvert = \lvert\eta\rvert < \infty$ $\lvert(\partial f_8)/(\partial H)\rvert = \lvert-(\pi + \mu + \xi)\rvert < \infty$ $\lvert(\partial f_8)/(\partial R)\rvert = 0\ < \infty$
$\lvert(\partial f_9)/(\partial S)\rvert = 0 < \infty$ $\lvert(\partial f_9)/(\partial I_{us})\rvert = \lvert\kappa\rvert < \infty$ $\lvert(\partial f_9)/(\partial I_{ss})\rvert = \lvert\epsilon\rvert < \infty$ $\lvert(\partial f_9)/(\partial H)\rvert = \lvert\pi\rvert < \infty$ $\lvert(\partial f_9)/(\partial R)\rvert = \lvert-(\vartheta + \mu)\rvert < \infty$	

4.3 Positivity of Solution

The solution of the system remains positive at any point in time t, if the initial values of all the variables are positive.

Theorem 4: Let$\Omega_2 = \{(S,\ I_{us},\ I_{ss},\ H,\ R) \in \mathbb{R}_+^5; S_0 > 0,\ I_{us_0} > 0,\ I_{ss_0} > 0,\ H_0 > 0,\ R_0 > 0\}$ then the solutions of $\{S,\ I_{us},\ I_{ss},\ H,\ R\}$ are positive for all $t \geq 0$.

Proof: Positivity is verified separately for each of the model $S(t)$, $I_{us}(t)$, $I_{ss}(t)$,$H(t)$ and $R(t)$.
Positivity of $S(t)$: From the model in Equation (9) we have:

$$\frac{dS}{dt} = \Pi + \vartheta R - (\lambda_s + \mu)S$$

Eliminating the positive terms $(\Pi + \vartheta R)$ we get,

$$\Leftrightarrow \frac{dS}{dt} \geq -(\lambda_s + \mu)S$$

Using variables separable method we get,

$$\Rightarrow \frac{dP}{S} \geq -(\lambda_s + \mu)dt$$

Integrating both side we can get,

$$\Rightarrow \int \frac{dS}{S} \geq -\int (\lambda_s + \mu)\, dt$$

$$\Rightarrow \ln S \geq -(\lambda_s + \mu)t + c_8, \text{ where } c_8 \text{ is integration constant}$$

$$\Rightarrow S(t) \geq S_0 e^{-(\lambda_s+\mu)t},\ S_0 = e^{c_8} \text{ and } e^{-(\lambda_s+\mu)t} \geq 0, \text{ for all } t \geq 0.$$

Hence, it can be concluded that $S(t) \geq 0$.
Positivity of I_{us}: From model Equation (9) we have:

$$\frac{dI_{us}}{dt} = \lambda_s S - (\varepsilon + \gamma + \kappa + \mu)I_{us}$$

Eliminating the positive terms $(\lambda_s S)$ we get,

$$\Leftrightarrow \frac{dI_{us}}{dt} \geq -(\varepsilon + \gamma + \kappa + \mu)I_{us}$$

Using variables separable method we get,

$$\Rightarrow \frac{dI_{us}}{I_{us}} \geq -(\varepsilon + \gamma + \kappa + \mu)dt$$

Integrating both side we can get,

$$\Rightarrow \int \frac{dI_{us}}{I_{us}} \geq -\int (\varepsilon + \gamma + \kappa + \mu)\, dt$$

$$\Rightarrow \ln I_{us} \geq -(\varepsilon + \gamma + \kappa + \mu)t + c_9, \text{ where } c_9 \text{ is integration constant}$$

$$\Rightarrow I_{us}(t) \geq I_{us0} e^{-(\varepsilon+\gamma+\kappa+\mu)t},\ I_{us0} = e^{c_9} \text{ and } e^{-(\varepsilon+\gamma+\kappa+\mu)t} \geq 0, \text{ for all } t \geq 0.$$

Hence, it can be concluded that $I_{us}(t) \geq 0$.
Positivity of $I_s(t)$: From model Equation (9) we have:

$$\frac{dI_{ss}}{dt} = \gamma I_{us} - (\eta + \epsilon + \mu)I_{ss}$$

Eliminating the positive terms (γI_{us}) we get,

$$\Leftrightarrow \frac{dI_{ss}}{dt} \geq -(\eta + \epsilon + \mu)I_{ss}$$

Using variables separable method we get,

$$\Rightarrow \frac{dI_{ss}}{I_{ss}} \geq -(\eta + \epsilon + \mu)dt$$

Integrating both side we can get,

$$\Rightarrow \int \frac{dI_{ss}}{I_{ss}} \geq -\int (\eta + \epsilon + \mu)\, dt$$

$$\Rightarrow \ln I_{ss} \geq -(\eta + \epsilon + \mu)t + c_{10}, \text{ where } c_{10} \text{ is integration constant}$$

$$\Rightarrow I_{ss}(t) \geq I_{ss0} e^{-(\eta+\epsilon+\mu)t},\ I_{ss0} = e^{10} \text{ and } e^{-(\eta+\epsilon+\mu)t} \geq 0, \text{ for all } t \geq 0.$$

Hence, it can be concluded that$I_{ss}(t) \geq 0$.
Positivity of $H(t)$: From the model in Equation (9) we have:

$$\frac{dH}{dt} = \varepsilon I_{us} + \eta I_{ss} - (\pi + \mu + \xi)H$$

Eliminating the positive terms $(\varepsilon I_{us} + \eta I_{ss})$ we get,

$$\Leftrightarrow \frac{dH}{dt} \geq -(\pi + \mu + \xi)H$$

Using variables separable method we get,

$$\Rightarrow \frac{dH}{H} \geq -(\pi + \mu + \xi)dt$$

Integrating both side we can get,

$$\Rightarrow \int \frac{dH}{H} \geq -\int (\pi + \mu + \xi)\, dt$$

$$\Rightarrow \ln H \geq -(\pi + \mu + \xi)t + c_{11}, \text{ where } c_{11} \text{ is integration constant}$$

$$\Rightarrow H(t) \geq H_0 e^{-(\pi+\mu+\xi)t},\ H_0 = e^{c_{11}} \text{ and } e^{-(\pi+\mu+\xi)t} \geq 0, \text{ for all } t \geq 0.$$

Hence, it can be concluded that $H(t) \geq 0$.
Positivity of $R(t)$: From the model in Equation (9) we have:

$$\frac{dR}{dt} = \kappa I_{us} + \epsilon I_{ss} + \pi H - (\vartheta + \mu)R$$

Eliminating the positive terms $(\kappa I_{us} + \epsilon I_{ss} + \pi H)$ we get,

$$\Leftrightarrow \frac{dR}{dt} \geq -(\vartheta + \mu)R$$

Using variables separable method we get,

$$\Rightarrow \frac{dR}{R} \geq -(\vartheta + \mu)dt$$

Integrating both side we can get,

$$\Rightarrow \int \frac{dR}{R} \geq -\int (\vartheta + \mu)\, dt$$

$$\Rightarrow \ln \mathrm{R} \geq -(\vartheta + \mu)t + c_{12}, \text{ where } c_{12} \text{ is integration constant}$$

$$\Rightarrow R(t) \geq R_0 \mathrm{e}^{-(\vartheta+\mu)t},\ R_0 = \mathrm{e}^{c_{12}} \text{ and } \mathrm{e}^{-(\vartheta+\mu)t} \geq 0, \text{ for all } t \geq 0.$$

Hence, it can be concluded that $R(t) \geq 0$. Therefore, the model variables $S(t)$, $I_{us}(t)$, $I_{ss}(t), H(t)$ and $R(t)$ representing population sizes of various types of cells are positive quantities and will remain in $\mathbb{R}^5_+$ for all t.

4.4 Stability Analysis of the Disease-Free Equilibrium (DFE)

The disease free equilibrium of the HSV-II only is determined by equating Equation (9) to zero. Then we get:

$$E_3 = \left\{\left(\frac{\Pi}{\mu}\right),\ 0,\ 0,\ 0,\ 0\right\}$$

The local stability of the DFE, E_3 can be established using the next generation operator method in Van den Driessche and Watmouth [15] on the system (9). To do this the basic reproduction number can be determined depends on the definition of infected and uninfected compartments. The model in Equation (9) are rewritten starting with newly infective classes:

$$\frac{dI_{us}}{dt} = \lambda_s S - (\varepsilon + \gamma + \kappa + \mu)I_{us}$$
$$\frac{dI_{ss}}{dt} = \gamma I_{us} - (\eta + \epsilon + \mu)I_{ss}$$
$$\frac{dH}{dt} = \varepsilon I_{us} + \eta I_{ss} - (\pi + \mu + \xi)H$$

Then by the principle of next-generation matrix, we obtained

$$f_i = \begin{bmatrix} \frac{\beta_2(I_{us}+q_2 I_{ss})S}{N_s} \\ 0 \\ 0 \end{bmatrix} \text{ and } v_i = \begin{bmatrix} (\varepsilon + \gamma + \kappa + \mu)I_{us} \\ -\gamma I_{us} + (\eta + \epsilon + \mu)I_{ss} \\ -\varepsilon I_{us} - \eta I_{ss} + (\pi + \mu + \xi)H \end{bmatrix}$$

The Jacobian matrices of f_i and v_i evaluated at DFE are given by F and V, respectively, such that

$$F = \begin{bmatrix} \beta_2 & \beta_2 q_2 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} (\varepsilon + \gamma + \kappa + \mu) & 0 & 0 \\ -\gamma & (\eta + \epsilon + \mu) & 0 \\ -\varepsilon & -\eta & (\pi + \mu + \xi) \end{bmatrix}$$

It can be verified that the matrix V is non-singular as its determinant $det[V] = (\varepsilon + \gamma + \kappa + \mu)(\eta + \epsilon + \mu)(\pi + \mu + \xi)$ is non-zero and after some algebraic computations its inverse matrix is constructed as

$$V^{-1} = \begin{bmatrix} \frac{1}{(\varepsilon+\gamma+\kappa+\mu)} & 0 & 0 \\ \frac{\gamma}{(\varepsilon+\gamma+\kappa+\mu)(\eta+\epsilon+\mu)} & \frac{1}{(\eta+\epsilon+\mu)} & 0 \\ \frac{\gamma\eta+\varepsilon(\eta+\epsilon+\mu)}{(\varepsilon+\gamma+\kappa+\mu)(\eta+\epsilon+\mu)(\pi+\mu+\xi)} & \frac{\theta}{\eta} \\ & \frac{\eta}{(\eta+\epsilon+\mu)(\pi+\mu+\xi)} & \frac{1}{(\pi+\mu+\xi)} \end{bmatrix}.$$

The product of the matrices F and V^{-1} can be computed as:

$$FV^{-1} = \begin{bmatrix} \beta_2 & \beta_2 q_2 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{(\varepsilon+\gamma+\kappa+\mu)} & 0 & 0 \\ \frac{\gamma}{(\varepsilon+\gamma+\kappa+\mu)(\eta+\epsilon+\mu)} & \frac{1}{(\eta+\epsilon+\mu)} & 0 \\ \frac{\gamma\eta+\varepsilon(\eta+\epsilon+\mu)}{(\varepsilon+\gamma+\kappa+\mu)(\eta+\epsilon+\mu)(\pi+\mu+\xi)} & \frac{\theta}{(\eta+\epsilon+\mu)(\pi+\mu+\xi)} & \frac{1}{(\pi+\mu+\xi)} \end{bmatrix}$$

$$= \begin{bmatrix} \frac{\beta_2}{(\varepsilon+\gamma+\kappa+\mu)} + \frac{\beta_2 q_2 \gamma}{(\varepsilon+\gamma+\kappa+\mu)(\eta+\epsilon+\mu)} & \frac{\beta_2 q_2}{(\eta+\epsilon+\mu)} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

Now it is possible to calculate the eigenvalue to determine the basic reproduction number $\Re_s$ by taking the spectral radius of the matrix FV^{-1}. Thus, the eigenvalues are computed by evaluating $det[FV^{-1} - \lambda I] = 0$ or equivalently solving

$$\begin{vmatrix} \frac{\beta_2}{(\varepsilon+\gamma+\kappa+\mu)} + \frac{\beta_2 q_2 \gamma}{(\varepsilon+\gamma+\kappa+\mu)(\eta+\epsilon+\mu)} - \lambda & \frac{\beta_2 q_2}{(\eta+\epsilon+\mu)} & 0 \\ 0 & -\lambda & 0 \\ 0 & 0 & -\lambda \end{vmatrix} = 0$$

$$\Rightarrow \lambda^2 \left[\frac{\beta_2(\eta+\epsilon+\mu)+\beta_2 q_2 \gamma(\varepsilon+\gamma+\kappa+\mu)}{(\varepsilon+\gamma+\kappa+\mu)(\eta+\epsilon+\mu)} - \lambda\right] = 0,$$

$$\Rightarrow \lambda_4 = \left[\frac{\beta_2(\eta+\epsilon+\mu)+\beta_2 q_2 \gamma(\varepsilon+\gamma+\kappa+\mu)}{(\varepsilon+\gamma+\kappa+\mu)(\eta+\epsilon+\mu)}\right],\ \lambda_5 = \lambda_6 = 0.$$

However, the dominant eigenvalue here is $\lambda_4 = \left[\frac{\beta_2(\eta+\epsilon+\mu)+\beta_2 q_2 \gamma(\varepsilon+\gamma+\kappa+\mu)}{(\varepsilon+\gamma+\kappa+\mu)(\eta+\epsilon+\mu)}\right]$and is the spectral radius as the threshold value or the basic reproductive number. Thus, it can be concluded that the reproduction number of the model is $\Re_s = \left[\frac{\beta_2(\eta+\epsilon+\mu)+\beta_2 q_2 \gamma(\varepsilon+\gamma+\kappa+\mu)}{(\varepsilon+\gamma+\kappa+\mu)(\eta+\epsilon+\mu)}\right]$. Further using Theorem 2 in Van den Driessche and Watmouth [15], the following result is established. The DFE is locally asymptotically stable if $\Re_s < 1$ and unstable is $\Re_s > 1$.

4.5 Existence and Stability Analysis of Endemic Equilibrium

Lemma 4. The HSV-II only model Equation (9) has a unique endemic equilibrium if and only if the basic reproduction number$\Re_s > 1$.

Proof. Let the endemic equilibrium point of the model in Equation (9) be denoted by,

$$E_4 = (S^*,\ I_{us}^*,\ I_{ss}^*,\ \ H^*,\ R^*)$$

and consider the force of infection

$$\lambda_s^* = \frac{\beta_2[I_{us}^* + q_2 I_{ss}^*]}{N_s} \tag{14}$$

Solving the model by setting the right hand sides of Equation (9) equal to zero, we get,

$$S^* = \frac{\Pi}{(\lambda_s^*+\mu)}, I_{us}^* = \frac{\Pi\lambda_s^*}{(\lambda_s^*+\mu)(\varepsilon+\gamma+\kappa+\mu)}, I_{ss}^* = \frac{\alpha\Pi\lambda_s^*}{(\lambda_s^*+\mu)(\eta+\epsilon+\mu)},$$
$$H^* = \frac{\gamma\Pi\lambda_s^*(\eta+\epsilon+\mu)+\eta\gamma\Pi(\lambda_s^*+\mu)}{(\lambda_s^*+\mu)(\varepsilon+\gamma+\kappa+\mu)(\eta+\epsilon+\mu)(\pi+\mu+\xi)}, R^* = \frac{\kappa I_{us}^* + \epsilon I_{ss}^* + \pi H}{(\vartheta+\mu)} \tag{15}$$

Substituting Equation (15) into Equation (14) we get

$$\lambda_s^* = \frac{\beta_2\Pi\lambda_s^*}{(\lambda_s^*+\mu)(\varepsilon+\gamma+\kappa+\mu)} + \frac{\beta_2 q_2 \gamma\Pi\lambda_s^*}{(\lambda_s^*+\mu)(\eta+\epsilon+\mu)}$$
$$(\lambda_s^*)^2 + \mu\lambda_s^* - \Pi\lambda_s^*\Re_s = 0$$
$$(\lambda_s^*)^2 + \lambda_s^*[\mu - \Pi\Re_s] = 0$$

Hence, the HSV-II force of infection, λ_s^*, satisfies the polynomial:

$$p(\lambda_s^*) = (\lambda_s^*)^2 + M\lambda_s^* = 0, \text{ where } M = \mu - \Pi\Re_s$$

By mathematical induction, $M \geq 0$ whenever the basic reproduction number is less than one ($\Re_s < 1$). This implies that $\lambda_s^* = -1/M \leq 0$. Therefore, the HSV-II model has no endemic equilibrium whenever $\Re_s < 1$. Hence, the analysis describes the impossibility of backward bifurcation in the HSV-II only model, implies there is no existence of endemic equilibrium whenever the basic reproduction number is less than one ($\Re_s < 1$).

5. ANALYSIS OF HIV-HSV-II CO-INFECTION MODEL

In this section, the analysis of HIV-HSV-II co-infection model in Equation (1) was considered.

5.1. Invariant Region

Theorem 5: The total population size N of the system of model Equation (1) is bounded in the invariant region Ω. That is, size of N is bounded for all t.

Proof: In model Equation (1), the total population of N is given as

$$N = S + I_{uh} + I_{us} + I_{uhs} + I_{sh} + I_{ss} + I_{shs} + A + H + AH + R$$

Differentiating N both sides with respect to t leads to

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI_{uh}}{dt} + \frac{dI_{us}}{dt} + \frac{dI_{uhs}}{dt} + \frac{dI_{sh}}{dt} + \frac{dI_{ss}}{dt} + \frac{dI_{shs}}{dt} + \frac{dA}{dt} + \frac{dH}{dt} + \frac{dAH}{dt} + \frac{dR}{dt} \tag{16}$$

Substituting model Equation (1) into Equation (16), we can get

$$\frac{dN}{dt} = \Pi - \mu N - \xi(A + H + AH) \tag{17}$$

In the absence of mortality due to disease ($\xi = 0$), then Equation (17) become

$$\frac{dN}{dt} \leq \Pi - \mu N \tag{18}$$

Rearranging and integrating both sides of Equation (18), we get

$$\int \frac{dN}{\Pi - \mu N} \leq \int dt$$

$$\Leftrightarrow \frac{-1}{\mu}\ln(\Pi - \mu N) \leq t + c_{12} \text{ , where } c_{12} \text{ is integration constant}$$

$$\Rightarrow \ln(\Pi - \mu N) \geq -\mu t + c_{13}, \text{ where } c_{13} = -\mu c_{12}$$

$$\Rightarrow (\Pi - \mu N) \geq ce^{-\mu t}, \text{ where } c = e^{-c_{13}}$$

Then, applying initial condition, $N(0) = N_0$, we obtain $\mathrm{c} = \Pi - \mu N_0$

$$\Rightarrow \Pi - \mu N \geq (\Pi - \mu N_0)e^{-\mu t}$$

$$\Rightarrow N \leq \frac{\Pi}{\mu} - [\frac{\Pi - \mu N}{\mu}]e^{-\mu t} \tag{19}$$

As $t \to \infty$ in Equation (19), the population size $N(t) \to \frac{\Pi}{\mu}$ which implies that $0 \leq N(t) \leq \left(\frac{\Pi}{\mu}\right)$. Thus, the feasible solution set of the model Equation (1) enters and remains in the region: $\Omega = \{(S, I_{uh}, I_{us}, I_{uhs}, I_{sh}, I_{ss}, I_{shs}, A, H, AH, R) \in \mathbb{R}_+^{11} : N_h \leq \Pi/\mu\}$. Therefore, the model in Equation (1) is wellposed epidemiologically and mathematically. Hence, it is sufficient to study the dynamics of the model in the region Ω.

5.2. Existence of Solution

Lemma 5: Solutions of the model Equations (1) together with the initial conditions, $S(0) > 0$, $I_{uh}(0) > 0$, $I_{us}(0) > 0$, $I_{uhs}(0) > 0$, $I_{sh}(0) > 0$, $I_{ss}(0) > 0$, $I_{shs}(0) > 0$, $A(0) > 0$, $H(0) > 0, AH(0) > 0$, $R(0) > 0$ exist in $\mathbb{R}_+^{11}$ i.e., the solution of the model variables $S(t)$, $I_{uh}(t)$, $I_{us}(t)$, $I_{uhs}(t)$, $I_{sh}(t)$, $I_{ss}(t)$, $I_{shs}(t)$, $A(t)$, $H(t)$, $AH(t)$ and $R(t)$ exist for all t and will remain in $\mathbb{R}_+^{11}$.

Proof: Existence of solution for $(S, I_{uh}, I_{us}, I_{sh}, I_{ss}, A, H, R)$ are shown in Sections 3.2 and 4.2, and in Tables 1 and 2. Now, positivity for (I_{uhs}, I_{shs}, AH) are shown in Table 3. Let

$$f_{10}(S, I_{uh}, I_{us}, I_{uhs}, I_{sh}, I_{ss}, I_{shs}, A, H, AH, R) = \phi I_{uh} + \psi I_{us} - (\rho + \theta + \mu)I_{us}$$

$$f_{11}(S, I_{uh}, I_{us}, I_{uhs}, I_{sh}, I_{ss}, I_{shs}, A, H, AH, R) = \theta I_{uhs} + \varphi I_{sh} + \tau I_{ss} - (\sigma + \mu)I_{shs}$$

$$f_{12}(S, I_{uh}, I_{us}, I_{uhs}, I_{sh}, I_{ss}, I_{shs}, A, H, AH, R) = \rho I_{uhs} + \sigma I_{ssh} + \nu A + \chi H - (\mu + \xi)AH$$

According to Derrick and Groosman theorem as in [19], let Ω denote the region, $\Omega = \{(S, I_{uh}, I_{us}, I_{uhs}, I_{sh}, I_{ss}, I_{shs}, A, H, AH, R) \in \mathbb{R}_+^{11} : N \leq \Pi/\mu\}$. Then Equations (1) have a unique solution if $(\partial f_i)/(\partial x_j)$, $i, j = 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11$ are continuous and bounded in Ω. Here, $x_1 = S$, $x_2 = I_{uh}$, $x_3 = I_{us}, x_4 = I_{uhs}$, $x_5 = I_{sh}$, $x_6 = I_{ss}$, $x_7 = I_{shs}$, $x_8 = A, x_9 = H$, $x_{10} = AH$ and $x_{11} = R$. The continuity and the boundedness are verified in Table 3. Thus, all the partial derivatives $(\partial f_i)/(\partial x_j), i, j = 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11$ exist, continuous and bounded in Ω. Hence, by Derrick and Groosman theorem, a solution for the model (1) exists and is unique.

Table 3. Continuity and boundedness of the model solution

$\left\|\frac{(\partial f_1)}{(\partial S)}\right\| = \left\|\frac{(\partial f_1)}{(\partial I_{sh})}\right\| = \left\|\frac{(\partial f_1)}{(\partial I_{ss})}\right\| = \left\|\frac{(\partial f_1)}{(\partial I_{shs})}\right\| = \left\|\frac{(\partial f_1)}{(\partial A)}\right\| = \left\|\frac{(\partial f_1)}{(\partial H)}\right\| = \left\|\frac{(\partial f_1)}{(\partial AH)}\right\| = \left\|\frac{(\partial f_1)}{(\partial R)}\right\| = 0 < \infty$ $\left\|\frac{(\partial f_1)}{(\partial I_{uh})}\right\| = \|\phi\| < \infty, \quad \left\|\frac{(\partial f_1)}{(\partial I_{us})}\right\| = \|\psi\| < \infty, \quad \left\|\frac{(\partial f_1)}{(\partial I_{uhs})}\right\| = \|-(\rho+\theta+\mu)\| < \infty$
$\left\|\frac{(\partial f_2)}{(\partial S)}\right\| = \left\|\frac{(\partial f_2)}{(\partial I_{uh})}\right\| = \left\|\frac{(\partial f_2)}{(\partial I_{us})}\right\| = \left\|\frac{(\partial f_2)}{(\partial A)}\right\| = \left\|\frac{(\partial f_2)}{(\partial H)}\right\| = \left\|\frac{(\partial f_2)}{(\partial AH)}\right\| = \left\|\frac{(\partial f_2)}{(\partial R)}\right\| = 0 < \infty$ $\left\|\frac{(\partial f_2)}{(\partial I_{uhs})}\right\| = \|\theta\| < \infty, \quad \left\|\frac{(\partial f_2)}{(\partial I_{sh})}\right\| = \|\varphi\| < \infty, \quad \left\|\frac{(\partial f_2)}{(\partial I_{ss})}\right\| = \|\tau\| < \infty, \quad \left\|\frac{(\partial f_2)}{(\partial I_{shs})}\right\| = \|-(\sigma+\mu)\| < \infty$
$\left\|\frac{(\partial f_3)}{(\partial S)}\right\| = \left\|\frac{(\partial f_3)}{(\partial I_{uh})}\right\| = \left\|\frac{(\partial f_3)}{(\partial I_{us})}\right\| = \left\|\frac{(\partial f_3)}{(\partial I_{sh})}\right\| = \left\|\frac{(\partial f_3)}{(\partial I_{ss})}\right\| = \left\|\frac{(\partial f_3)}{(\partial R)}\right\| = 0 < \infty$ $\left\|\frac{(\partial f_3)}{(\partial I_{uhs})}\right\| = \|\rho\| < \infty, \left\|\frac{(\partial f_3)}{(\partial I_{shs})}\right\| = \|\sigma\| < \infty, \left\|\frac{(\partial f_3)}{(\partial A)}\right\| = \|\nu\| < \infty, \left\|\frac{(\partial f_3)}{(\partial H)}\right\| = \|\chi\| < \infty, \left\|\frac{(\partial f_3)}{(\partial AH)}\right\| = \|-(\mu+\xi)\| < \infty$

5.3. Positivity of Solution

In this section, we show all the solution of the model Equation (1) remains positive for future time if their respective initial values are positive.

Theorem 6: Let $\Omega = \{(S, I_{uh}, I_{us}, I_{uhs}, I_{sh}, I_{ss}, I_{shs}, A, H, AH, R) \in \mathbb{R}_+^{11}; S_0(0) > 0, I_{uh0}(0) > 0, I_{us0}(0) > 0, I_{uhs0}(0) > 0, I_{sh0}(0) > 0, I_{ss0}(0) > 0, I_{shs0}(0) > 0, A_0(0) > 0, H_0(0) > 0, AH_0(0) > 0, , R_0(0) > 0\}$ then the solutions of $\{S, I_{uh}, I_{us}, I_{uhs}, I_{sh}, I_{ss}, I_{shs}, A, H, AH, R\}$ are positive for all $t \geq 0$.

Proof: Since positivity of $S(t)$, $I_{uh}(t)$, $I_{us}(t)$, $I_{sh}(t)$,$I_{ss}(t)$, $A(t)$, $H(t)$ and $R(t)$are shown in Sections 3.3 and 4.3 separately. Now let us show $I_{uhs}(t)$, $I_{shs}(t)$ and $AH(t)$ are positive for future time.

From model in Equation (1) we have:

$$\frac{dI_{uhs}}{dt} = \phi I_{uh} + \psi I_{us} - (\rho+\theta+\mu)I_{uhs}$$

Eliminating the positive terms $(\phi I_{uh} + \psi I_{us})$ we get,

$$\Leftrightarrow \frac{dI_{uhs}}{dt} \geq -(\rho+\theta+\mu)I_{uhs}$$

Using variables separable method we get,

$$\Rightarrow \frac{dI_{uhs}}{I_{uhs}} \geq -(\rho+\theta+\mu)dt$$

Integrating both side we can get,

$$\Rightarrow \int \frac{dI_{uhs}}{I_{uhs}} \geq -\int(\rho+\theta+\mu)\,dt$$

$$\Rightarrow \ln I_{uhs} \geq -(\rho+\theta+\mu)t + c_{13}, \text{ where } c_{13} \text{ is integration constant}$$

$$\Rightarrow I_{uhs}(t) \geq I_{uhs0}\mathrm{e}^{-(\rho+\theta+\mu)t}, I_{uhs0} = \mathrm{e}^{c_{13}} \text{ and } \mathrm{e}^{-(\rho+\theta+\mu)t} \geq 0, \text{ for all } t \geq 0.$$

Hence, it can be concluded that $I_{uhs}(t) \geq 0$.

From the model in Equation (1) we have:

$$\frac{dI_{shs}}{dt} = \theta I_{uhs} + \varphi I_{sh} + \tau I_{ss} - (\sigma+\mu)I_{shs}$$

Eliminating the positive terms $(\theta I_{uhs} + \varphi I_{sh} + \tau I_{ss})$ we get,

$$\Leftrightarrow \frac{dI_{shs}}{dt} \geq -(\sigma+\mu)I_{shs}$$

Using variables separable method we get,

$$\Rightarrow \frac{dI_{shs}}{I_{shs}} \geq -(\sigma+\mu)dt$$

Integrating both side we can get,

$$\Rightarrow \int \frac{dI_{shs}}{I_{shs}} \geq -\int(\sigma+\mu)\,dt$$

$$\Rightarrow \ln I_{shs} \geq -(\sigma+\mu)t + c_{14}, \text{ where } c_{14} \text{ is integration constant}$$

$$\Rightarrow I_{shs}(t) \geq I_{shs0}\mathrm{e}^{-(\sigma+\mu)t}, I_{\mathrm{shs}0} = \mathrm{e}^{14} \text{and } \mathrm{e}^{-(\sigma+\mu)t} \geq 0, \text{ for all } t \geq 0.$$

Hence, it can be concluded that $I_{shs}(t) \geq 0$.

From the model in Equation (1) we have:

$$\frac{dAH}{dt} = \rho I_{uhs} + \sigma I_{shs} + \nu A + \chi H - (\mu+\xi)AH$$

Eliminating the positive terms $(\rho I_{uhs} + \sigma I_{shs} + \nu A + \chi H)$ we get,

$$\Leftrightarrow \frac{dAH}{dt} \geq -(\mu+\xi)AH$$

Using variables separable method we get,

$$\Rightarrow \frac{dAH}{AH} \geq -(\mu + \xi)dt$$

Integrating both side we can get,

$$\Rightarrow \int \frac{dAH}{AH} \geq -\int (\mu + \xi)\, dt$$
$$\Rightarrow \ln \mathrm{AH} \geq -(\mu + \xi)t + c_{15}, \text{ where } c_{15} \text{ is integration constant}$$
$$\Rightarrow AH(t) \geq AH_0 \mathrm{e}^{-(\mu+\xi)t},\ AH_0 = \mathrm{e}^{c_{15}} \text{ and } \mathrm{e}^{-(\mu+\xi)t} \geq 0, \text{ for all } t \geq 0.$$

Hence, it can be concluded that $AH(t) \geq 0$. Therefore, the model variables $I_{uhs}(t)$, $I_{shs}(t)$ and $AH(t)$ representing population sizes of various types of cells are positive quantities and will remain in $\mathbb{R}_+^{11}$ for all t.

5.4. Stability Analysis of the Disease-Free Equilibrium (DFE)

The disease free equilibrium of the HIV-HSV-II co-infection is obtained by equating the system of model Equation (1) to zero. At disease free equilibrium, there are no infections and recovery. Then we can get;

$$E_5 = \left\{\left(\frac{\Pi}{\mu}\right),\ 0,\ 0,\ 0,\ 0,\quad 0,\quad 0,\quad 0,\quad 0,\ 0,\ 0\right\}$$

The local stability of the DFE, E_5 can be established using the next generation operator method in Van den Driessche and Watmouth [15] on the system (1). It follows that the basic reproduction number of the HIV-HSV-II model Equation (1), denoted by $\Re_{hs}$ is given by $\Re_{hs} = \max\{\Re_h, \Re_s\}$ where,

$$\Re_h = \left[\frac{\beta_1(\omega+\mu)+\beta_1 q_1 \alpha(\alpha+\delta+\mu)}{(\alpha+\delta+\mu)(\omega+\mu)}\right]$$
$$\Re_s = \left[\frac{\beta_2(\eta+\epsilon+\mu)+\beta_2 q_2 \gamma(\varepsilon+\gamma+\kappa+\mu)}{(\varepsilon+\gamma+\kappa+\mu)(\eta+\epsilon+\mu)}\right]$$

Theorem 7: The disease free equilibrium point E_5 of the system (1) is locally asymptotically stable whenever the basic reproduction number is less than one ($\Re_{\mathrm{hs}} < 1$) and unstable if otherwise.

Proof: To proof this theorem first we obtained the Jacobian matrix of the model in Equation (1) at the disease free equilibrium E_5 is given by:

$$J(E_5) = \begin{bmatrix} -\mu & -\beta_1 & -\beta_2 & 0 & -\beta_1 q_1 & -\beta_2 q_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_1 - r_1 & 0 & 0 & \beta_1 q_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \beta_2 - r_2 & 0 & 0 & \beta_2 q_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & \phi & \psi & -r_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha & 0 & 0 & -r_4 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma & 0 & 0 & -r_5 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \theta & \varphi & \tau & -r_6 & 0 & 0 & 0 & 0 \\ 0 & \delta & 0 & 0 & \omega & 0 & 0 & -r_7 & 0 & 0 & 0 \\ 0 & 0 & \varepsilon & 0 & 0 & \eta & 0 & 0 & -r_8 & 0 & 0 \\ 0 & 0 & 0 & \rho & 0 & 0 & \sigma & \nu & \chi & -r_9 & 0 \\ 0 & 0 & \kappa & 0 & 0 & \epsilon & 0 & 0 & \pi & 0 & -r_{10} \end{bmatrix}$$

Now, the eigenvalues of $J(E_5)$ are required to be found. The characteristic equation $det[J(E_5) - \lambda I] = 0$ is expanded and simplified as follows:

$$\begin{vmatrix} -\mu-\lambda & -\beta_1 & -\beta_2 & 0 & -\beta_1 q_1 & -\beta_2 q_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & (\beta_1 - r_1) - \lambda & 0 & 0 & \beta_1 q_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & (\beta_2 - r_2) - \lambda & 0 & 0 & \beta_2 q_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & \phi & \psi & -r_{3-\lambda} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha & 0 & 0 & -r_4 - \lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma & 0 & 0 & -r_5 - \lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \theta & \varphi & \tau & -r_6 - \lambda & 0 & 0 & 0 & 0 \\ 0 & \delta & 0 & 0 & \omega & 0 & 0 & -r_7 - \lambda & 0 & 0 & 0 \\ 0 & 0 & \varepsilon & 0 & 0 & \eta & 0 & 0 & -r_8 - \lambda & 0 & 0 \\ 0 & 0 & 0 & \rho & 0 & 0 & \sigma & \nu & \chi & -r_9 - \lambda & 0 \\ 0 & 0 & \kappa & 0 & 0 & \epsilon & 0 & 0 & \pi & 0 & -r_{10} - \lambda \end{vmatrix} = 0 \qquad (20)$$

From the Jacobian matrix of Eqaution (20), we obtain a characteristic polynomial:

$$[-\mu - \lambda][-r_{10} - \lambda][-r_9 - \lambda][-r_8 - \lambda][-r_7 - \lambda][-r_6 - \lambda][4\lambda^4 + L_1\lambda^3 + L_2\lambda^2 + L_3\lambda + L_4] = 0 \qquad (21)$$

where

$$L_1 = r_4 - \beta_1 + r_5 - \beta_2$$
$$L_2 = 2[\alpha\beta_1 q_1 - \gamma\beta_2 q_2 - r_4(\beta_1 - r_1) - r_5(\beta_2 - r_2)] + (r_5 - \beta_2)(r_4 - \beta_1)$$
$$L_3 = \alpha\beta_1 q_1(r_5 - \beta_2) - \gamma\beta_2 q_2(r_4 - \beta_1) - r_4(r_5 - \beta_2)(\beta_1 - r_1) - r_5(\beta_2 - r_2)(r_4 - \beta_1)$$
$$L_4 = \gamma\beta_2 q_2 r_4(\beta_1 - r_1) + \gamma\beta_2 q_2 \alpha\beta_1 q_1 + r_4 r_5(\beta_2 - r_2)(\beta_1 - r_1) - r_5\alpha\beta_1 q_1(\beta_2 - r_2)$$

Thus, from Equation (21) clearly we see that:

$$\lambda_1 = -\mu,\ \lambda_2 = -r_{10},\ \lambda_3 = -r_9,\ \lambda_4 = -r_8,\ \lambda_5 = -r_7,\ \lambda_6 = -r_6, \lambda_7 = -r_3$$

It can be observed that the eigen values$\lambda_1,\ \lambda_2,\ \lambda_3,\ \lambda_4,\ \lambda_5,\ \lambda_6$and λ_7 are absolutely negative quantities.
For the last expression, that is,

$$4\lambda^4 + L_1\lambda^3 + L_2\lambda^2 + L_3\lambda + L_4 = 0 \tag{22}$$

We applied Routh-Hurwitz criteria. By the principle of Routh-Hurwitz criteria, Equation (22) has strictly negative real root if and only if $L_1 > 0, L_2 > 0, L_3 > 0, L_4 > 0$ and$L_1L_2L_3 > L_3^2 + L_1^2L_4$.Therefore, it is concluded that the DFE E_5 of the system of differential Equations (1) is locally asymptotically stable if $\Re_{hs} < 1$ and unstable if$\Re_{hs} > 1$. Here,$r_1 = (\phi + \alpha + \delta + \mu), r_2 = (\psi + \varepsilon + \gamma + \kappa + \mu), r_3 = (\rho + \theta + \mu), r_4 = (\varphi + \omega + \mu), r_5 = (\tau + \eta + \epsilon + \mu), r_6 = (\sigma + \mu), r_7 = (\nu + \mu + \xi), r_8 = (\chi + \pi + \mu + \xi), r_9 = (\mu + \xi), r_{10} = (\vartheta + \mu)$.

5.5. Global Stability of Disease Free Equilibrium

The global stability of disease free equilibrium is determined using Castillo-Chavez and Song [16] technique. The model Equation (1) can be re-written as

$$dX/dt = F(X,Y)$$
$$dY/dt = G(X,Y),\ \ G(X,0) = 0$$

where X stands for the uninfected population, that is $X = (S,\ R)$ and Y also stands for the infected population, that is $Y = (I_{uh},\ I_{us},\ I_{uhs},\ I_{sh},\ I_{ss},\ I_{shs}, A, H, AH)$. The disease free equilibrium point of the model is denoted by $U = (X^*, 0)$. The point $U = (X^*, 0)$ to be globally asymptotically stable equilibrium for the model provided that $\Re_{hs} < 1$ and the following conditions must be met:

(H_1). For$dX/dt = F(X, 0)$, X^* is globally asymptotically stable.
(H_2). $G(X,Y) = AY - \tilde{G}(X,Y),\ \ \tilde{G}(X,Y) \geq 0$ for $(X,Y) \in \Omega$.

where $A = D_Y G(U, 0)$ is a Metzler matrix (the off diagonal elements of A are non-negative) and G is the region where the model make biologically sense.

If the model Equation(1) met the above two criteria then the following theorem holds.

Theorem 8: The point $U = (X^*, 0)$ is globally asymptotically stable equilibrium provided that $\Re_{\text{hs}} < 1$ and the condition (H_1) and $(H_2)$are satisfied.

Proof: From system (1) we can get $F(X,Y)$ and $G(X,Y)$,

$$dX/dt = F(X,Y) = \begin{bmatrix} \Pi + \vartheta R - (\lambda_h + \lambda_s + \mu)S \\ \kappa I_{us} + \epsilon I_{ss} + \pi H - (\vartheta + \mu)R \end{bmatrix} \text{ and}$$

$$dY/dt = G(X,Y) = \begin{bmatrix} \lambda_h S - r_1 I_{uh} \\ \lambda_s S - r_2 I_{us} \\ \phi I_{uh} + \psi I_{us} - r_3 I_{uhs} \\ \alpha I_{uh} - r_4 I_{sh} \\ \gamma I_{us} - r_5 I_{ss} \\ \theta I_{uhs} + \varphi I_{sh} + \tau I_{ss} - r_6 I_{shs} \\ \delta I_{uh} + \omega I_{sh} - r_7 A \\ \varepsilon I_{us} + \eta I_{ss} - r_8 H \\ \rho I_{uhs} + \sigma I_{shs} + \nu A + \chi H - r_9 AH \end{bmatrix}$$

Consider the reduced system

$$\frac{dX}{dt}_{|Y=0} = \begin{bmatrix} \Pi - \mu S \\ 0 \end{bmatrix} \tag{23}$$

From Equation (23), it is obvious that $X^* = [(\Pi/\mu), 0]$ is the global asymptotic point. This can be verified from the solution, namely $S = [\Pi/\mu] + [S(0) - (\Pi/\mu)]e^{-\mu t}$. As $t \to \infty$, the solution $(S) \to [\Pi/\mu]$, implying that the global convergence of Equation (23) in Ω. From the equation for infected compartments in the model we have:

$$A=\begin{bmatrix} \beta_1-r_1 & 0 & 0 & \beta_1 q_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_2-r_2 & 0 & 0 & \beta_2 q_2 & 0 & 0 & 0 & 0 \\ \phi & \psi & -r_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ \alpha & 0 & 0 & -r_4 & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma & 0 & 0 & -r_5 & 0 & 0 & 0 & 0 \\ 0 & 0 & \theta & \varphi & \tau & -r_6 & 0 & 0 & 0 \\ \delta & 0 & 0 & \omega & 0 & 0 & -r_7 & 0 & 0 \\ 0 & \varepsilon & 0 & 0 & \eta & 0 & 0 & -r_8 & 0 \\ 0 & 0 & \rho & 0 & 0 & \sigma & \nu & \chi & -r_9 \end{bmatrix}$$

Since A is Metzler matrix, i.e. all off diagonal elements are nonnegative. Then, $G(X,Y)$ can be written as, $G(X,Y)=AY-\tilde{G}(X,Y)$, where

$$\tilde{G}(X,Z)=\begin{bmatrix} \beta_1(I_{uh}+q_1 I_{sh})\left[1-\frac{S}{N}\right] \\ \beta_2(I_{us}+q_2 I_{ss})\left[1-\frac{S}{N}\right] \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}=\begin{bmatrix} \tilde{G}_1(X,Z) \\ \tilde{G}_2(X,Z) \\ \tilde{G}_3(X,Z) \\ \tilde{G}_4(X,Z) \\ \tilde{G}_5(X,Z) \\ \tilde{G}_6(X,Z) \\ \tilde{G}_7(X,Z) \\ \tilde{G}_8(X,Z) \\ \tilde{G}_9(X,Z) \end{bmatrix} \quad (24)$$

It follows that, in Equation (24), $\tilde{G}_1(X,Y)\geq 0, \tilde{G}_2(X,Y)\geq 0$, and $\tilde{G}_3(X,Y)=\tilde{G}_4(X,Y)=\tilde{G}_5(X,Y)=\tilde{G}_6(X,Y)=\tilde{G}_7(X,Y)=\tilde{G}_8(X,Y)=\tilde{G}_9(X,Y)=0$. Hence, $\tilde{G}(X,Y)\geq 0$. Therefore, condition (H_1) and (H_2) are satisfied and we conclude that U is globally asymptotically stable for $\Re_{hs}<1$.

5.6. Endemic Equilibrium Point

The endemic equilibrium denoted by(S^*, I_{uh}^*, I_{us}^*, I_{uhs}^*, I_{sh}^*, I_{ss}^*, I_{shs}^*, A^*, H^*, AH^*, R^*) and it occur when the disease persist in the community. To obtain it we equate all the model in Equation (1) to zero. Then we obtained;

$S^*=\frac{\Pi+\vartheta R^*}{(\lambda_h^*+\lambda_s^*+\mu)}$, $I_{uh}^*=\frac{(\Pi+\vartheta R^*)\lambda_h^*}{(\lambda_h^*+\lambda_s^*+\mu)(\phi+\alpha+\delta+\mu)}$, $I_{us}^*=\frac{(\Pi+\vartheta R^*)\lambda_s^*}{(\lambda_h^*+\lambda_s^*+\mu)(\psi+\varepsilon+\gamma+\kappa+\mu)}$, $I_{uhs}^*=\frac{\phi I_{uh}^*+\psi I_{us}^*}{(\rho+\theta+\mu)}$, $I_{sh}^*=\frac{\alpha I_{uh}^*}{(\varphi+\omega+\mu)}$, $I_{ss}^*=\frac{\gamma I_{us}^*}{(\tau+\eta+\epsilon+\mu)}$, $I_{shs}^*=\frac{\theta I_{uhs}^*+\varphi I_{sh}^*+\tau I_{ss}}{(\sigma+\mu)}$, $A^*=\frac{\lambda_h^*(\pi+\vartheta R^*)[\delta(\varphi+\omega+\mu)+\omega\alpha]}{(\lambda_h^*+\lambda_s^*+\mu)(\phi+\alpha+\delta+\mu)(\varphi+\omega+\mu)(\nu+\mu+\xi)}$, $H^*=\frac{\lambda_s^*(\pi+\vartheta R^*)\varepsilon[(\tau+\eta+\epsilon+\mu)+\eta\gamma]}{(\lambda_h^*+\lambda_s^*+\mu)(\psi+\varepsilon+\gamma+\kappa+\mu)(\tau+\eta+\epsilon+\mu)(\chi+\pi+\mu+\xi)}$, $(AH)^*=\frac{\rho I_{uhs}^*+\sigma I_{shs}^*+\nu A^*+\chi H^*}{(\mu+\xi)}$, $R^*=\frac{\kappa I_{us}^*+\epsilon I_{ss}^*+\pi H}{(\vartheta+\mu)}$

After substituting the variables we see that the endemic equilibrium point is very long and complicated. We have therefore decided to use numerical simulation of the co-infection dynamics considering when $\Re_{hs}<1$ and $\Re_{hs}>1$.

6. NUMERICAL SIMULATION

In this section, the numerical simulations of model Equations (1) are carried out using the software MATLAB R2015b with ODE45 solver. To conduct the study, a set of physically meaningful values are assigned to the model parameters. These values are either taken from literature or assumed on the basis of reality. Using the parameter values given in Table 4 and the initial conditions, $S(0)=200, I_{uh}(0)=180, I_{us}(0)=175, I_{uhs}(0)=170, I_{sh}(0)=150, I_{ss}(0)=140, I_{shs}(0)=120, A(0)=60, H(0)=50, AH(0)=40, R(0)=30$ in the model Equations (1), a simulation study is conducted.

Table 4. Parameter values used in simulations

Parameter	Value	Source	Parameter	Value	Source
Π	0.004	[17]	κ	0.02	[18]
λ_s	0.002	[18]	θ	0.003	assumed
λ_h	0.00197	[18]	φ	0.003	assumed
μ	0.02	[17]	ω	0.054	assumed
ϑ	0.0031	assumed	τ	0.003	assumed
ϕ	0.003	assumed	η	0.011	[18]
α	0.003	assumed	ϵ	0.02	assumed
ρ	0.064	assumed	σ	0.017	assumed
δ	0.016	[18]	ν	0.001	assumed
ψ	0.003	assumed	χ	0.001	assumed
ε	0.039	assumed	π	0.0041	[18]
γ	0.003	assumed	ξ	0.0001	assumed

Figure 2 illustrated that all the solutions of model Equation (2) converge towards the disease free equilibrium point while the susceptible individuals decreases and then remains constant. Susceptible individuals remain constant because of AIDS cannot be cured. This was obtained when $\Re_h < 1$ and indicates that the disease free equilibrium point is locally asymptotically stable. Figure 3 indicate that an increase or decrease in the force of infection shows an increase or decrease in the number of infectious.

From Figure 4 we understand that all the solutions converge towards the equilibrium point. At disease free equilibrium point, all infection solutions converge to zero while the susceptible individuals decreases and then remains constant. This indicates that the disease free equilibrium point is locally asymptotically stable when$\Re_s < 1$. Figure 5 shows that a sharp reduction in the number of HSV-II. An increase or decrease in the force of infection shows an increase or decrease in the number of infectious. This indicates that the disease free equilibrium point is locally asymptotically stable when the basic reproduction is less than one.

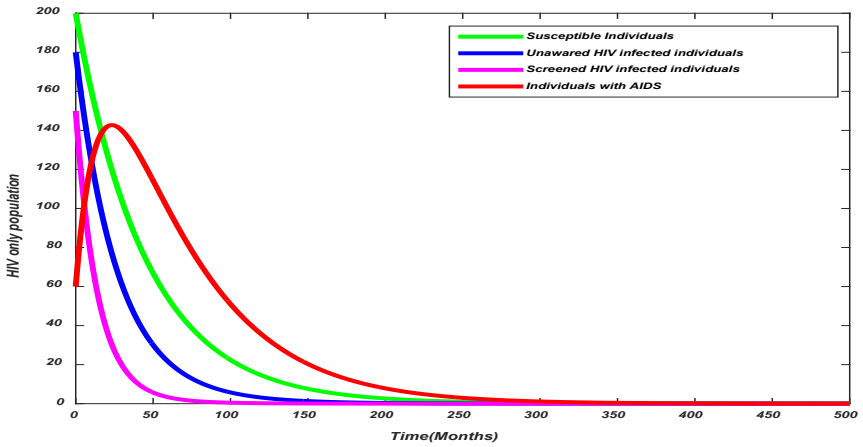

Figure 2. Dynamics of HIV only model

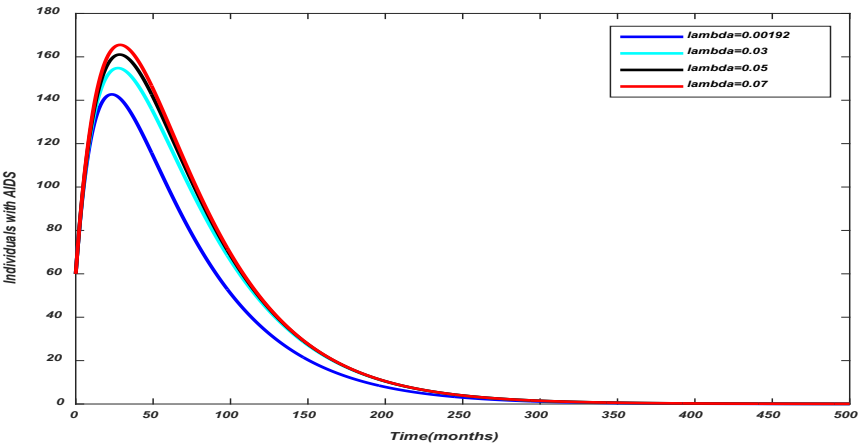

Figure 3. Effect of increasing force infection on individuals with AIDS

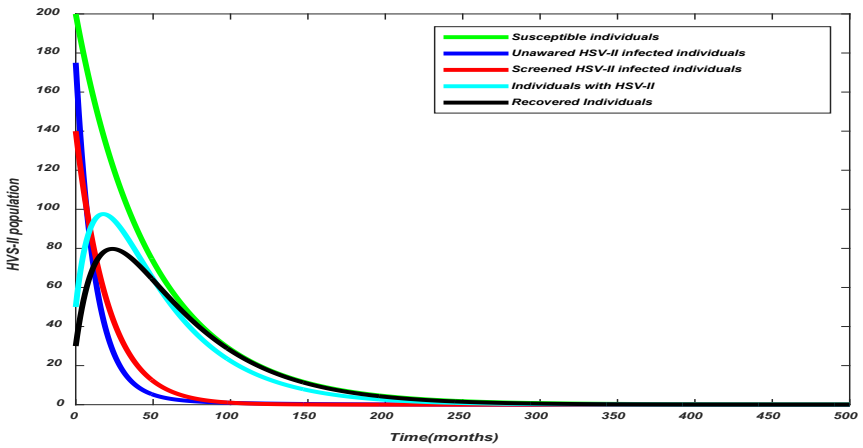

Figure 4. Dynamics of HIV only model

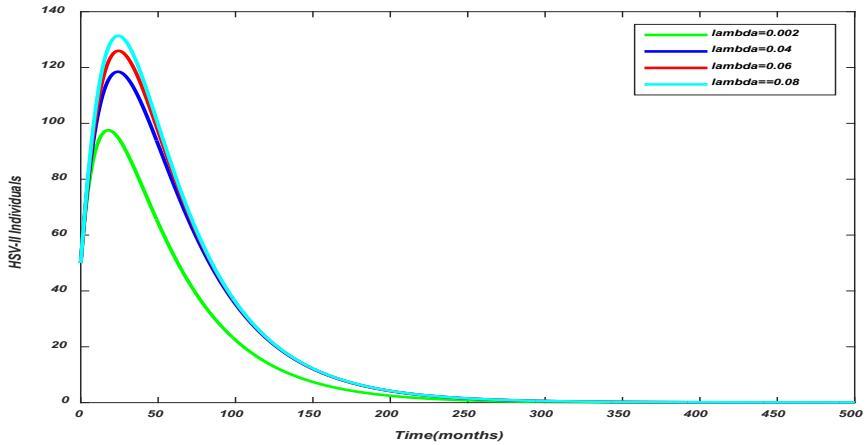

Figure 5. Effect of increasing force of infection on individuals with HSV-II

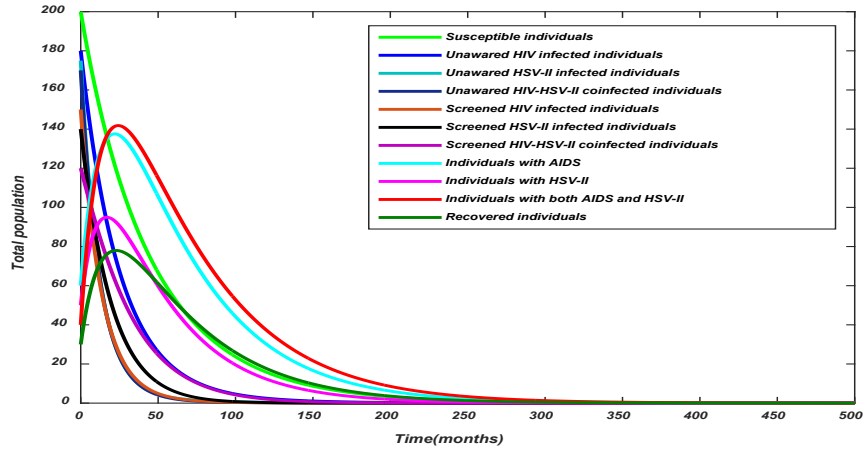

Figure 6. Dynamic of total population

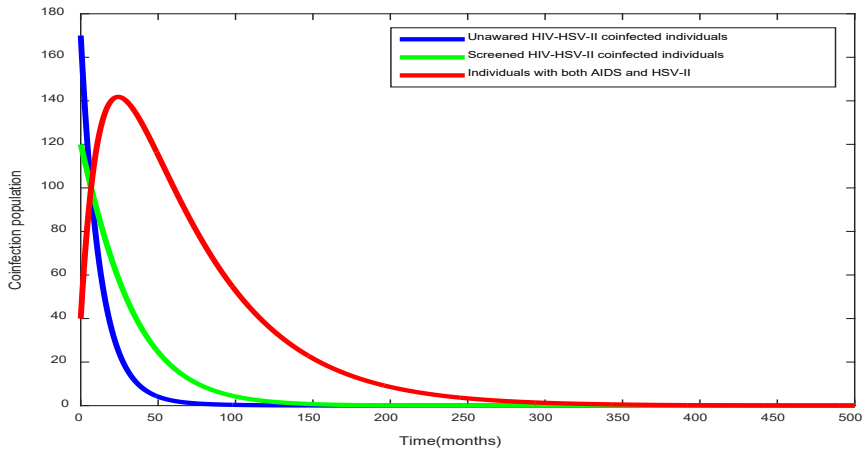

Figure 7. Dynamics of only co-infection individuals

Figure 6 describe that all the solutions of the model Equation (1) converge towards the equilibrium point. At disease free equilibrium point, all infection solutions converge to zero while the susceptible individuals decreases and then remains constant. Susceptible individuals remain constant because of AIDS cannot be cured. This indicates that the disease free equilibrium point is locally asymptotically stable. From Figure 7, we understand that individuals with both unawared HIV-HSV-II co-infection and screened HIV-HSV-II increases the number of individuals with both AIDS and HSV-II and also decreases the number if it is controlled at early stage. Similarly, Figure 8 illustrated that individuals with AIDS and HSV-II increases the number of individuals with both AIDS and HSV-II and also decreases the number if it is controlled at early stage.

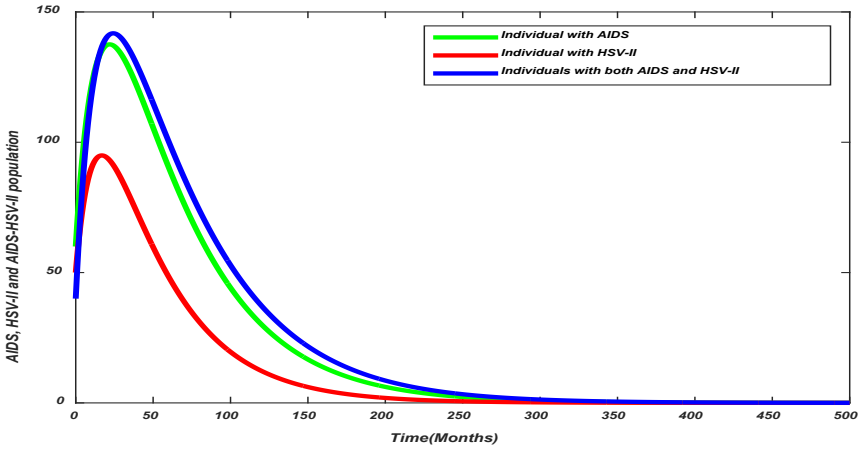

Figure 8. Dynamics of AIDS, HSV-II and co-infection of AIDS-HSV-II

In this study, a non-linear deterministic model of HIV-HSV-II co-infection was formulated and analyzed. The well possedness of model equation have been performed separately for HIV, HSV-II and co-infection of HIV-HSV-II to show it was epidemiologically acceptable and mathematically significant. The equilibria points of the model equations are obtained for each model. The stability analysis of the model was investigated using the basic reproduction number that governs the disease transmission. HIV only model, HSV-II only model and co-infection of HIV-HSV-II model, are locally asymptotically stable whenever the basic reproduction number is less than unity. Also, each model equation has a unique endemic equilibrium whenever the basic reproduction number is greater than unity. We found from the analysis of the impact of HIV on HSV-II that HSV-II infection increases the risk of HIV; similarly, HIV infection increases the risk for HSV-II. Furthermore, results from numerical simulation shows that at disease free equilibrium point, all infection solutions converge to zero when the basic reproduction number is less than unity. In addition to this, an increase or decrease of force of infection has an impact on reducing the disease from community

7. CONCLUSION

In this paper, we formulated a mathematical model on the transmission dynamics HIV-HSV-II co-infection. Moreover, existence, positivity and boundedness for each model equation are verified to illustrate that the model is biologically meaningful and mathematically well posed. In particular, the stability analyses of the model were investigated using the basic reproduction number. And also, the solution of the model equation is numerically supplemented. The result shows that the co-infection of HIV-HSV-II increases the mortality rate in the community other than co-infection of sexually transmitted disease. Although eradication of co-infection remain a challenge especially in developing countries, but from results of this study we recommend that, the government should introduce education programmers on the importance of voluntary and routinely screening HIV-HSV-II co-infection.

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