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Mathematical Model for Coinfection of HIV/AIDS and Kaposi's Sarcoma with Treatment

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Abstract: HIV destroys T-cells in order to target the body's defence mechanism. Without treatment HIV infection advances in stages causing destruction and reduction in T-cells thus, rendering the body incapable of fighting other infections such as respiratory infections, sexually transmitted diseases and some cancers. Kaposi's sarcoma is the cancer that allows a tumour to grow in an HIV patient and its presence in a patient is an indication that HIV has fully developed into AIDS in the patient. Research has indicated that AIDS-associated Kaposi Sarcoma was on the rise in sub-Saharan Africa until the introduction of Antiretroviral Therapy (ART). The Kenyan community has struggled in the past decade to combat the spread of HIV/AIDS and successes have been recorded in many areas. However, Kaposi Sarcoma, an opportunistic infection, has continued to rise steadily through the years. In this study, a simple model for the coinfection of HIV/AIDS and KS is developed and studied. The model solution is explored for positivity and boundedness while the DFE point is determined for stability where it was verified that the infection-free equilibrium E_0 is locally asymptotically stable when $\mathcal{R} < 1$. The NGM is used to derive the basic reproduction number of the model. By providing treatment to the HIV and the co-infected population immune system is strengthened and thus progression rate to AIDS is reduced.

Keywords: HIV/AIDS, Coinfection, Kaposi's Sarcoma, Treatment, Reproduction Number

1. Introduction

The CD4 Lymphocyte cells also called the T-cells, are white cells that protect humans against any external infection and are in abundance among the white blood cells. They are produced in the bone marrow and are transported to the area of the body under attack to fight the incoming disease, illness or infection. HIV is a virus that aims at destroying T-cells. HIV infection, as stated by Wang and Li [1], gradually eradicate the T-cells and eventually, exposes the host to subsequent infections. These opportunistic infections include respiratory infections (such as tuberculosis and pneumonia) and STDs (such as syphilis and gonorrhoea among others). HIV infection advances in three stages. The first stage of HIV infection is Acute HIV infection, which is experienced within less than four weeks of infection. During this time, the patient has symptoms like rash, fever and/or headache. HIV replicates speedily and transmits throughout the body destroying the soldier T cells. Introducing ART at this early stage usually proves very beneficial to the patient's health. The next stage is the chronic (or asymptomatic or clinical latency) HIV infection, where the virus rate of replication is very low. At this stage, the symptoms are subdued and the patient may seem very healthy while the virus continues to destroy the remaining T cells. Without treatment, the third stage is reached. This is the fully developed AIDS, a situation where the body's immune system has been utterly destroyed beyond repair. At this stage, the body's immune system cannot fight incoming diseases or infections. It is at this stage that cancer in the form of KS prevails. People with AIDS easily transmit HIV to other humans through the exchange of body fluids. HIV is commonly transmitted from infected individuals to uninfected ones through unprotected sex, and an exchange of body fluids, mother-to-child transmission at birth or during breastfeeding, and sharing tattooing or piercing needles and needles used for injecting drugs. HIV

prevention and treatment with an aim of ending AIDS is a key global health strategic objective**.** This is achieved by improving the medicine, promoting diagnosis and enhancing a patient-centred mode of service delivery. The management of people living with advanced HIV disease has been strengthened. Despite the measures in place, 1.5 million people are estimated to live with HIV in Kenya, out of which 1,136,000 are on ART, according to National AIDS & STI Control Program 2018. AIDS-related Kaposi sarcoma (AIDS-KS) caused by HIV co-infection with HHV-8 remains endemic in Kenya and sub-Saharan Africa [2, 3].

A common illness among HIV patients who have a good CD4 count is KS. It is cancer in form of a tumour in HIV patients. The human herpes virus 8 (HHV-8) is the causative agent with spreads commonly among African gay men [4]. According to Warpe [5], KS builds up when cancer cells, septic blood cells and infected blood vessels begin to grow, without stoppage, under the skin, in lymph nodes, or the mouth lining. They show up as red or purple patches or lesions starting from one or many body parts simultaneously. The presence of the HHV-8 in the mouth makes its transmission possible via kissing with an infected individual, lubricating the genitals with saliva during sex, or during oral sex (common among gays). The immune system of a healthy individual is able to keep the HHV-8 under control and to also ensure it does not develop into KS. Meanwhile, the presence of HHV-8 in an HIV patient, not using ART, is prone to quickly metamorphose into KS. To understand the mechanisms and the dynamics of HIV and KS coinfection as well as establish the disease transmission, prevention and control strategies, a mathematical model has been used. Through quantitative and qualitative analysis and numerical simulations, definite treatment regimens to boost anti-viral immunity and enhance long-term control of the virus have been designed. Mathematical models for HIV dynamics, disease progression, and therapy as well as HIV and KS coinfection have been proposed on different scales.

Wang and Li [1] proposed a model with a simplified logistic growth of the susceptible CD4+T cells. Their main interest was to thoroughly investigate the global dynamics of the model and the qualitative changes resulting from changes in logistic terms. The results indicated that if $\mathcal{R} < 1$, the T cells will get rid of the HIV infection but otherwise, the infection remains. The global stability region for chronic infection was also obtained. Srivastava *et a*l. [6] developed and analysed a stochastic model for the effect of reverse transcriptase inhibitors on the viremia level. Cai *e*t al. [7] included treatment in the model. The authors established the model has two infective stages; the asymptomatic and symptomatic phases and proved that $\mathcal{R}$ is enough to unravel the dynamics of HIV. Ogunlaran and Noutchie [8] used minimum drug therapy to minimize the viral load. They assumed that the logistic growth function incorporated constant recruitment and death of new uninfected cells. The T-cells infection rate by free virions was assumed to be saturated by overcrowding of free virions and the choice of the patient on protection measures. Numerical simulation results were used to confirm the effectiveness of the treatment strategy. In 2016, Hikal and Zahra [9] investigated a fractional order time delay model with treatment. The population in their model was divided into a susceptible class, asymptomatic infection phase, symptomatic infection phase and the group of AIDS patients. The obtained results concurred with those of Cai *e*t al. [7]. Vaidya and Rong [10] integrated several drug-related parameters into the model. The results show that once a patient reaches the latent stage, it is impossible to remove the virus completely by any treatment; rather the treatment suppresses the viral load. Tarfulea [11] unravelled the influence of how mitosis influences HIV transmission using a mathematical model. Four dynamic variables were considered in the model. It was shown that the DFE is locally asymptotically stable if $\mathcal{R} \ll 1$. The author further recommended that the stability and other properties of the infected equilibrium be determined in future. In their work, Zhang and Wang [12] proposed a model for the variability of HIV infection of T cells, with the response of the immune and rate of cure. They developed a mathematical model that catered for the concentration of cells that are not infected, the concentration of cells that are infected and can produce a virus and the concentration of *CTLs*. They analysed the local stability of the DFE, immune absence and immune present equilibriums using the characteristic equation and Hurwitz criterion and obtained $\mathcal{R}$. Their model suggested that the T-cells concentration is a good criterion to measure the progression of HIV infection. Thus, the cure rate increases as the concentration of T cells increase. Modelling within-host viral infections with drug therapy give rise to dynamical systems whose local and global analysis is very essential. Prevention of HIV and providing tests and ART treatment as a fight against HIV infection was quantified by Omondi *et* al. [13] who constructed a model for epidemiological HIV trends. Through sensitivity analysis, the authors concluded that effective contact rates intensify HIV transmission while the effectiveness of ART inhibits the incidence rate.

It is recorded that 84% of the world case of KS is found in sub-Saharan Africa, making KS a common cancer in Sub-Saharan Africa occurring in the context of immunodeficiency. According to the available statistics, the same region is more heavily impacted by HIV/AIDS than any other [14-16]. Currently, 38 million people have HIV and 690,000 died in 2019 from HIV and/or its complications according to the World Health Organization. Without treatment HIV and KS coinfection can reduce lifespan [17]. Therefore, massively expanded prevention and treatment are fundamental in disease monitoring. Modelling the coinfection of HIV and KS is uncommon in literature yet they redound to the understanding of HIV and KS synergy. The model proposed in the current study incorporates prevention and treatment for both HIV/AIDS and KS. A mathematical model for the coinfection of HIV/AIDS and KS is studied. The solution is explored for positivity and boundedness. The stability of the DFE point is obtained. The basic reproduction number is derived using the Next Generation Matrix. The specific

objectives of this study are to:

1) Model coinfection of HIV and Kaposi's sarcoma with treatment.
2) Verify the model solutions are non-negative and bounded.
3) Determine the DFE point of the model and the local stability.
4) Establish the basic reproduction number.

2. Methodology

2.1. Model Formulation

The force of infection in the above model is taken as the probability of exposure to HIV, KS or both HIV and KS co-infected individuals. Susceptible individuals increases due to (i) newly recruited individuals, (ii) those who recover naturally from KS infection and (iii) full recovery after treatment. Infected individuals join the HIV, KS or co-infected classes $H(t), K(t)$ and $I(t)$ respectively and progress for treatment at the rates β_1, β_2 and β_3 respectively. We assume that the recovered individuals can still be infected by KS or HIV. Thus a fraction of the HIV and co-infected individuals join the treatment class $T(t)$ while the rest progress to AIDS class $A(t)$. The KS-infected individuals join the treatment compartment and the fraction that fully recovers after treatment moves back to the susceptible class while the remaining join the AID class where they die a natural death or death due to the suppressed immunity.

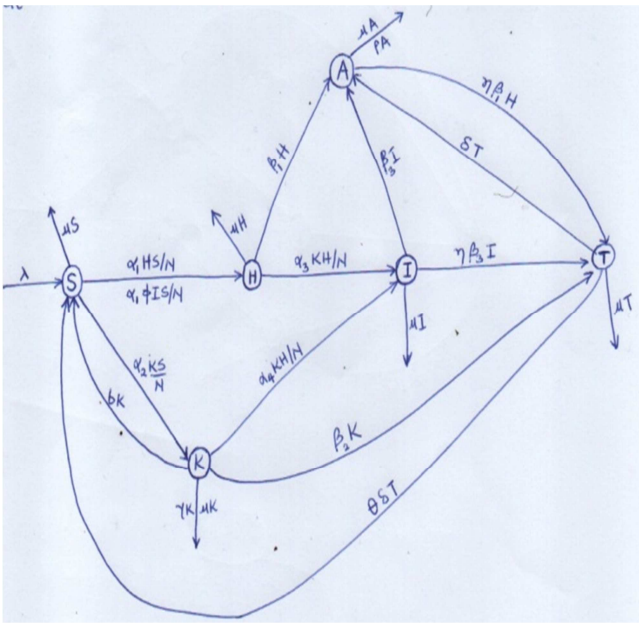

Figure 1. *HIV/Kaposi Sarcoma Coinfection flowchart.*

The following assumptions have been taken into account in formulating the model:

1) The susceptible population is a general population that is at risk of getting an HIV and KS infection at a rate proportional to the density of HIV- and KS-infected people respectively.
2) The transmission of HIV from an infective to susceptive is through horizontal transmission.
3) All parameters are positive.
4) The treatment is for both HIV and KS patients
5) The coinfection of HIV and Kaposi Sarcoma is modelled by the system;

$$\frac{dS}{dt} = \lambda - \frac{\alpha_1(H+\phi I)S}{N} - \frac{\alpha_2 KS}{N} - \mu S + bK + \theta\delta T \quad (1)$$

$$\frac{dH}{dt} = \frac{\alpha_1(H+\phi I)S}{N} - \frac{\alpha_3 KH}{N} - (\beta_1 + \mu)H \quad (2)$$

$$\frac{dK}{dt} = \frac{\alpha_2 KS}{N} - \frac{\alpha_4 KH}{N} - (\beta_2 + b + \gamma + \mu)K \quad (3)$$

$$\frac{dI}{dt} = \frac{\alpha_3 KH}{N} + \frac{\alpha_4 KH}{N} - \big((1+\eta)\beta_3 + \mu\big)I \quad (4)$$

$$\frac{dT}{dt} = \eta\beta_1 H + \eta\beta_3 I + \beta_2 K - (\delta + \mu)T \quad (5)$$

$$\frac{dA}{dt} = (1-\eta)\beta_1 H + (1-\eta)\beta_3 I + (1-\theta)\delta T - (\rho + \mu)A \quad (6)$$

The flow chart for the transmission of HIV/AIDS and KS, with their coinfection in a single host, is shown in Figure 1.

2.2. Equilibrium Points

The equilibrium points are obtained by setting each of the equations to 0.

$$\lambda - \frac{\alpha_1(H+\phi I)S}{N} - \frac{\alpha_2 \mathrm{KS}}{N} - \mu S + bK + \theta\delta T = 0 \quad (7)$$

$$\frac{\alpha_1(H+\phi I)S}{N} - \frac{\alpha_3 KH}{N} - (\beta_1 + \mu)H = 0 \quad (8)$$

$$\frac{\alpha_2 KS}{N} - \frac{\alpha_4 KH}{N} - (\beta_2 + b + \gamma + \mu)K = 0 \quad (9)$$

$$\frac{\alpha_3 KH}{N} + \frac{\alpha_4 KH}{N} - \big((1+\eta)\beta_3 + \mu\big)I = 0 \quad (10)$$

$$\eta\beta_1 H + \eta\beta_3 I + \beta_2 K - (\delta + \mu)T = 0 \quad (11)$$

$$(1-\eta)\beta_1 H + (1-\eta)\beta_3 I + (1-\theta)\delta T - (\rho + \mu)A = 0 \quad (12)$$

From equation (10)

$$I = \frac{(\alpha_3+\alpha_4)KH}{((1+\eta)\beta_3+\mu)N} \quad (13)$$

From equation (11)

$$T = \left(\frac{\eta\beta_1}{(\delta+\mu)K} + \frac{\beta_2}{(\delta+\mu)H} + \frac{\eta\beta_3(\alpha_3+\alpha_4)}{(\delta+\mu)((1+\eta)\beta_3+\mu)N}\right)KH \quad (14)$$

From equation (12)

$$A = \left[\frac{(1-\eta)\beta_1}{K} + \frac{(1-\eta)\beta_3(\alpha_3+\alpha_4)}{((1+\eta)\beta_3+\mu)N} + (1-\theta)\delta\left(\frac{\eta\beta_1}{(\delta+\mu)K} + \frac{\beta_2}{(\delta+\mu)H} + \frac{\eta\beta_3(\alpha_3+\alpha_4)}{(\delta+\mu)((1+\eta)\beta_3+\mu)N}\right)\right]\frac{KH}{\rho+\mu} \quad (15)$$

By adding (7), (8), and (9)

$$S = \frac{\lambda}{\mu} + \frac{1}{\mu}\left(\frac{\theta\delta T}{KH} - \frac{(\alpha_3+\alpha_4)}{N} - \frac{(\beta_1+\mu)}{K} - \frac{\beta_2+\gamma+\mu}{H}\right)KH \tag{16}$$

For the disease-free equilibrium DFE, we set $K = H = 0$, and that means,

$$I = 0, T = 0, A = 0, S = \frac{\lambda}{\mu},$$

So that the disease-free equilibrium point E_0 is

$$E_0 = (S_0, H_0, K_0, I_0, T_0, A_0) = \left(\frac{\lambda}{\mu}, 0, 0, 0, 0, 0\right).$$

and for the endemic equilibrium point E_1 is obtained by setting $H = H_1$ and $K = K_1$ so that

$$I_1 = \frac{(\alpha_3+\alpha_4)K_1H_1}{((1+\eta)\beta_3+\mu)N}, T_1 = \left(\frac{\eta\beta_1}{(\delta+\mu)K_1} + \frac{\beta_2}{(\delta+\mu)H_1} + \frac{\eta\beta_3(\alpha_3+\alpha_4)}{(\delta+\mu)((1+\eta)\beta_3+\mu)N}\right)K_1H_1$$

$$A_1 = \left[\frac{(1-\eta)\beta_1}{K_1} + \frac{(1-\eta)\beta_3(\alpha_3+\alpha_4)}{((1+\eta)\beta_3+\mu)N} + (1-\theta)\delta\left(\frac{\eta\beta_1}{(\delta+\mu)K_1} + \frac{\beta_2}{(\delta+\mu)H_1} + \frac{\eta\beta_3(\alpha_3+\alpha_4)}{(\delta+\mu)((1+\eta)\beta_3+\mu)N}\right)\right]\frac{K_1H_1}{\rho+\mu}$$

$$S_1 = \frac{\lambda}{\mu} + \frac{1}{\mu}\left(\frac{\theta\delta T}{K_1H_1} - \frac{(\alpha_3+\alpha_4)}{N} - \frac{(\beta_1+\mu)}{K_1} - \frac{\beta_2+\gamma+\mu}{H_1}\right)K_1H_1.$$

and thus

$$E_1 = (S_1, H_1, K_1, I_1, T_1, A_1).$$

2.3. Reproduction Number

The reproduction number is obtained using the next-generation matrix [**18-23**]. Start by setting

$$F = \frac{1}{N}\begin{pmatrix} \alpha_1(H+\phi I)S - \alpha_3 KH \\ \alpha_2 KS - \alpha_4 KH \\ (\alpha_3+\alpha_4)KH \end{pmatrix},$$

$$V = \begin{pmatrix} (\beta_1+\mu)H \\ (\beta_2+b+\gamma+\mu)K \\ \left((1+\eta)\beta_3+\mu\right)I \end{pmatrix}$$

and we find the Jacobian of F and V as follows

$$f = \nabla F = \frac{1}{N}\begin{pmatrix} \alpha_1 S - \alpha_3 K & -\alpha_3 K & \alpha_1\phi S \\ -\alpha_4 K & \alpha_2 S - \alpha_4 H & 0 \\ (\alpha_3+\alpha_4)K & (\alpha_3+\alpha_4)H & 0 \end{pmatrix},$$

$$v = \nabla V = \begin{pmatrix} \beta_1+\mu & 0 & 0 \\ 0 & \beta_2+b+\gamma+\mu & 0 \\ 0 & 0 & (1+\eta)\beta_3+\mu \end{pmatrix},$$

The characteristics equation of fv^{-1} at the DFE is

$$\begin{vmatrix} \frac{\alpha_1\lambda}{(\beta_1+\mu)\mu N} - m & 0 & \frac{\alpha_1\phi\lambda}{((1+\eta)\beta_3+\mu)\mu N} \\ 0 & \frac{\alpha_2\lambda}{(\beta_2+b+\gamma+\mu)\mu N} - m & 0 \\ 0 & 0 & -m \end{vmatrix} = 0$$

From where

$$m = 0, \frac{\alpha_1\lambda}{(\beta_1+\mu)\mu N}, \frac{\alpha_2\lambda}{(\beta_2+b+\gamma+\mu)\mu N}.$$

The reproduction numbers are;

$$R_0^H = \frac{\alpha_1\lambda}{(\beta_1+\mu)\mu N}, R_0^K = \frac{\alpha_2\lambda}{(\beta_2+b+\gamma+\mu)\mu N}.$$

2.4. Stability of the Disease-Free Equilibrium Point

Theorem 1: The DFE is locally asymptomatic stable if $R_0^H < 1$ and $R_0^K < 1$.

Proof:

Evaluating the Jacobian J_0 of the system (1 – 6) at the disease-free equilibrium point E_0, we have

$$J_0 = \begin{pmatrix} -\mu & \frac{\alpha_1\lambda}{\mu N} & b & -\frac{\alpha_1\phi\lambda}{\mu N} & \theta\delta & 0 \\ 0 & \frac{\alpha_1\lambda}{\mu N} - (\beta_1+\mu) & 0 & \frac{\alpha_1\phi\lambda}{\mu N} & 0 & 0 \\ 0 & 0 & \frac{\alpha_2\lambda}{\mu N} - (\beta_2+b+\gamma+\mu) & 0 & 0 & 0 \\ 0 & 0 & 0 & -\big((1+\eta)\beta_3+\mu\big) & 0 & 0 \\ 0 & \eta\beta_1 & \beta_2 & 0 & -(\delta+\mu) & 0 \\ 0 & (1-\eta)\beta_1 & 0 & (1-\eta)\beta_3 & (1-\theta)\delta & -(\rho+\mu) \end{pmatrix}$$

The characteristic equation is

$$\begin{vmatrix} -\mu-m & \frac{\alpha_1\lambda}{\mu N} & b & -\frac{\alpha_1\phi\lambda}{\mu N} & \theta\delta & 0 \\ 0 & \frac{\alpha_1\lambda}{\mu N} - (\beta_1+\mu) - m & 0 & \frac{\alpha_1\phi\lambda}{\mu N} & 0 & 0 \\ 0 & 0 & \frac{\alpha_2\lambda}{\mu N} - (\beta_2+b+\gamma+\mu) - m & 0 & 0 & 0 \\ 0 & 0 & 0 & -\big((1+\eta)\beta_3+\mu\big) - m & 0 & 0 \\ 0 & \eta\beta_1 & \beta_2 & 0 & -(\delta+\mu) - m & 0 \\ 0 & (1-\eta)\beta_1 & 0 & (1-\eta)\beta_3 & (1-\theta)\delta & -(\rho+\mu) - m \end{vmatrix} = 0$$

where m is the eigenvalue and by evaluating the determinant, we have;

$$(-\mu-m)(-(\rho+\mu)-m)(-(\delta+\mu)-m)\left(\frac{\alpha_1\lambda}{\mu N} - (\beta_1+\mu) - m\right)$$

$$\left(\frac{\alpha_2\lambda}{\mu N} - (\beta_2+b+\gamma+\mu) - m\right)\left(-\big((1+\eta)\beta_3+\mu\big) - m\right) = 0,$$

from which,

$$m_1 = -\mu, m_2 = -(\rho+\mu), m_3 = -(\delta+\mu), m_4 = \frac{\alpha_1\lambda}{\mu N} - (\beta_1+\mu),$$

$$m_5 = \frac{\alpha_2\lambda}{\mu N} - (\beta_2+b+\gamma+\mu), m_6 = -\big((1+\eta)\beta_3+\mu\big).$$

m_1, m_2, m_3 and m_6 are all negative for all values of the parameters, but

$$m_4 < 0 \text{ only if } \frac{\alpha_1\lambda}{\mu N} - (\beta_1+\mu) < 0 \Rightarrow \frac{\alpha_1\lambda}{\mu N(\beta_1+\mu)} < 1 \Rightarrow R_0^H < 1$$

and

$$m_5 < 0 \text{ only if } \frac{\alpha_2\lambda}{\mu N} - (\beta_2+b+\gamma+\mu) < 0 \Rightarrow \frac{\alpha_2\lambda}{\mu N(\beta_2+b+\gamma+\mu)} < 1 \Rightarrow R_0^K < 1$$

Hence, the DFE is locally asymptomatically stable if and only if $R_0^H < 1$ and $R_0^K < 1$. ■

2.5. Positivity and Boundedness of Solution

Theorem 1: The solution space is bounded in the region

$$\mathcal{R} = \left\{(S,H,K,I,T,A) \mid S+H+K+I+T+A \leq \frac{\lambda}{\mu}\right\}.$$

Proof:

By summing up equations (1 – 6)

$$\frac{d}{dt}(S+H+K+I+T+A)=\lambda-\ \mu(S\ +H+K+I+T+A)-\gamma K-\eta\beta_3 I-\rho A$$

and setting $N=S+H+K+I+T+A$, we have

$$\frac{dN}{dt}=\lambda-\ \mu N-\gamma K-\eta\beta_3 I-\rho A\leq\lambda-\ \mu N$$

And thus,

$$N(t)\leq\frac{\lambda}{\mu}-\left(\frac{\lambda}{\mu}-\ N(t_0)\right)\exp\left(-\mu(t-t_0)\right)\ \Rightarrow\ \lim_{t\to\infty}N(t)=\frac{\lambda}{\mu}$$

Thus, we can conclude that $N(t)\leq\frac{\lambda}{\mu}$, for all time t. Therefore, the solution is bounded in the region

$$\mathcal{R}=\left\{(S,H,K,I,T,A)\mid S+H+K+I+T+A\leq\frac{\lambda}{\mu}\right\}.\ \blacksquare$$

Theorem 2: The solution space (S,H,K,I,T,A) remains positive whenever the initial conditions are such that

$$S(t_0)>0,H(t_0)>0,K(t_0)>0,I(t_0)>0,T(t_0)>0,A(t_0)>0,$$

Proof: Start with equation (1)

$$\frac{dS}{dt}=\lambda-\frac{\alpha_1(H+\phi I)S}{N}-\frac{\alpha_2 KS}{N}-\ \mu S\ +bK+\ \theta\delta T\geq-\mu S\Rightarrow S(t)\geq S(t_0)\exp\left(-\mu(t-t_0)\right).$$

This clearly indicates that $S(t)\geq 0$ provided $S(t_0)>0$. Next, from equation (2)

$$\frac{dH}{dt}=\frac{\alpha_1(\mathrm{H}+\phi I)S}{N}-\frac{\alpha_3 KH}{N}-(\beta_1+\mu)H\geq-(\beta_1+\mu)H\Rightarrow H(t)\geq H(t_0)\exp\left(-(\beta_1+\mu)(t-t_0)\right).$$

Thus, $H(t)\geq 0$ provided $H(t_0)>0$.
From equation (3)

$$\frac{dK}{dt}=\frac{\alpha_2 KS}{N}-\frac{\alpha_4 KH}{N}-(\beta_2+b+\gamma+\mu)K\geq-(\beta_2+b+\gamma+\mu)K$$

$$\Rightarrow K(t)\geq K(t_0)\exp\left(-(\beta_2+b+\gamma+\mu)(t-t_0)\right)>0\text{ provided }K(t_0)>0.$$

Considering equation (4)

$$\frac{dI}{dt}=\frac{\alpha_3 KH}{N}+\frac{\alpha_4 KH}{N}-\left((1+\eta)\beta_3+\mu\right)I\geq-\left((1+\eta)\beta_3+\mu\right)I$$

$$\Rightarrow I(t)\geq I(t_0)\exp\left(-\left((1+\eta)\beta_3+\mu\right)(t-t_0)\right)>0\text{ provided }I(t_0)>0.$$

Considering the Treatment class in equation (5)

$$\frac{dT}{dt}=\eta\beta_1 H+\eta\beta_3 I+\beta_2 K-(\delta+\mu)T\geq-(\delta+\mu)T$$

$$\Rightarrow T(t)\geq T(t_0)\exp\left(-(\delta+\mu)(t-t_0)\right)>0\text{ provided }T(t_0)>0.$$

Finally, from equation (6)

$$\frac{dA}{dt}=(1-\eta)\beta_1 H+(1-\eta)\beta_3 I+(1-\theta)\delta T-(\rho+\mu)A\geq-(\rho+\mu)A\Rightarrow A(t)\geq A(t_0)\exp\left(-(\rho+\mu)(t-t_0)\right)>0\text{ provided }A(t_0)>0.$$

Hence, any solution (S,H,K,I,T,A) of the system (1 – 6) remains positive provided the initial conditions are positive.

3. Numerical Procedure

The Runge-Kutta RK (4,5) (also called the Dorman-Prince method) is an adaptive numerical technique that computes the accurate solutions of the initial value problem.

$$X'=F(X),X(t_0)=X_0$$

where

$$X=(x_1\quad x_2\quad\cdots\quad x_n)^T,F=(f_1,f_2,\cdots,f_n)^T,X_0=(x_1(t_0)\quad x_2(t_0)\quad\cdots\quad x_n(t_0))^T,$$

using the Runge-Kutta of orders 4 and 5. The difference between the two solutions is considered as the error and the

stepsize is adjusted based on the tolerance chosen. The modified Butcher tableau of the Dorman-Prince method is

c	A
	b_{RK4}
	b_{RK5}

where

$$A = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 1 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{1}{4} & \frac{3}{4} & 0 & 0 & 0 & 0 & 0 \\ \frac{11}{9} & -\frac{14}{3} & \frac{40}{9} & 0 & 0 & 0 & 0 \\ \frac{4843}{1458} & -\frac{3170}{243} & \frac{8056}{729} & -\frac{53}{162} & 0 & 0 & 0 \\ \frac{9017}{3168} & -\frac{355}{33} & \frac{46732}{5247} & \frac{49}{176} & -\frac{5103}{18656} & 0 & 0 \\ \frac{35}{384} & 0 & \frac{500}{1113} & \frac{125}{192} & -\frac{2187}{6784} & \frac{11}{84} & 0 \end{bmatrix}$$

$$b_{RK4} = \left[\frac{5179}{57600} \quad 0 \quad \frac{7571}{16695} \quad \frac{393}{640} \quad -\frac{92097}{339200} \quad \frac{187}{2100} \quad \frac{1}{40}\right]^T;$$

$$b_{RK4} = \left[\frac{35}{384} \quad 0 \quad \frac{500}{1113} \quad \frac{125}{192} \quad -\frac{2187}{6784} \quad \frac{11}{84} \quad 0\right]^T;$$

$$c = \left[0 \quad \frac{1}{5} \quad \frac{3}{10} \quad \frac{4}{5} \quad \frac{8}{9} \quad 1 \quad 1\right]^T.$$

The Dorman-Prince method is coded into the ode45 MATLAB solver. The system of equations (1 – 6) is solved with the tolerance set to 10^{-8} (see [24-25] for other methods of solutions). The parameter values are chosen based on the study of Wang et al. [1] and Onyango and Njiru [2] as follows;

$$\lambda = 800; \eta = 0.1; \delta = 0.01; \gamma = 0.1; \theta = 0.3; \mu = 0.02;$$

$$\alpha_1 = 0.4801; \alpha_2 = 0.002; \alpha_3 = \alpha_4 = 0.001; b = 0.2;$$

$$\rho = 0.333; \phi = 0.05;\ \beta_1 = 0.2, \beta_2 = 0.1; \beta_3 = 0.05.$$

4. Analysis and Discussion of Results

The effects of varying the parameters of the model on the different classes of the population are analysed and discussed in this section.

Figures 1 – 6 illustrate the effects of varying the proportion of HIV-infected individuals who have access to treatment. Figure 1 shows that increasing the access of HIV-infected individuals to treatment increases the population of the treated individuals, which means the susceptible class also increases as shown in figure 3. The class of individuals who are coinfected with HIV and KS will decrease as the treatment rate for HIV individuals increases (figure 4) and the HIV class decreases (figure 5). Clearly, a decrease in the HIV class will also lead to a decrease in the AIDS class as revealed in figure 6. Figure 7 shows that the population of the coinfected individuals increases as the rate of coinfection increases. By increasing the rate at which HIV-infected individuals get access to treatment, the population of the susceptible class increases (figure 8), the population of the coinfected individuals decreases (figure 9) and the population of individuals who are infected with HIV only decreases (figure 10).

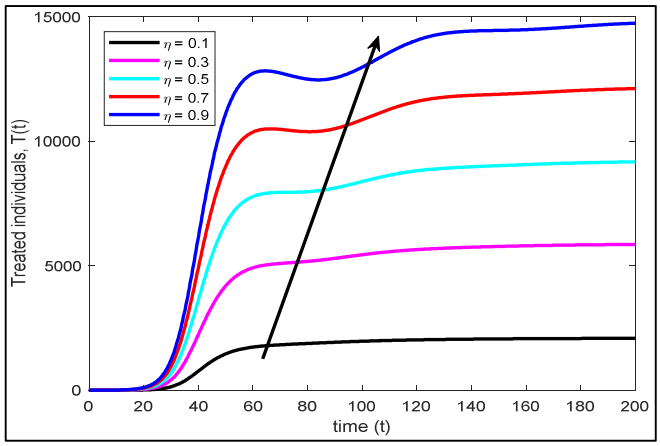

Figure 2. *Effects of the proportion of HIV-infected individuals who get treated on the Treatment class.*

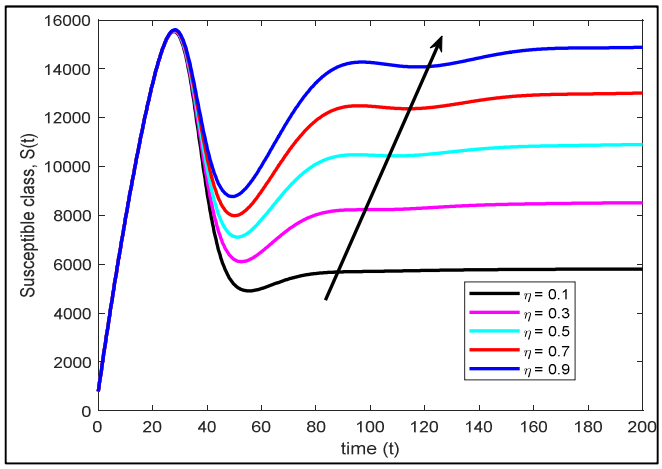

Figure 3. *Effects of the proportion of HIV-infected individuals who get treated on the Susceptible class.*

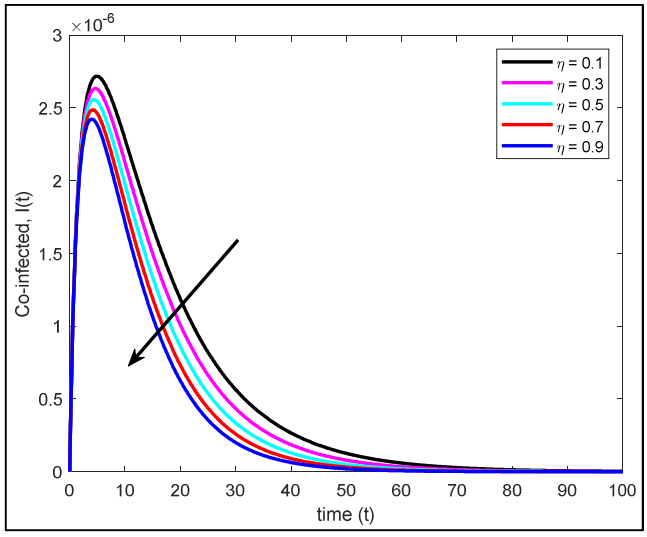

Figure 4. *Effects of the proportion of HIV-infected individuals who get treated on the individuals coinfected with HIV and Kaposi Sarcoma.*

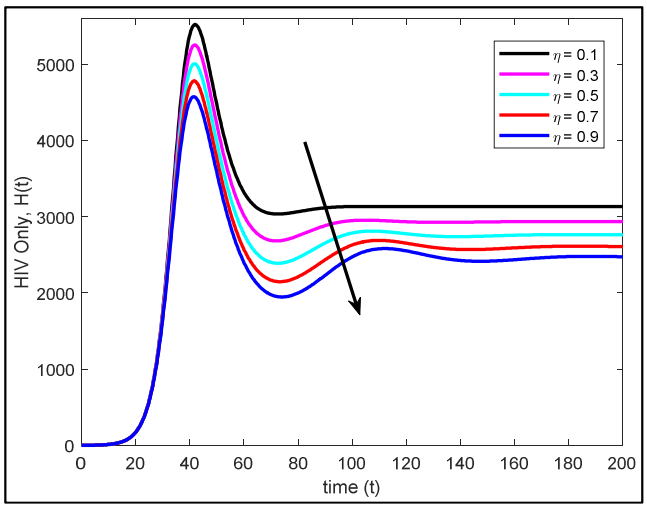

Figure 5. *Effects of the proportion of HIV-infected individuals who get treated on HIV-infected individuals.*

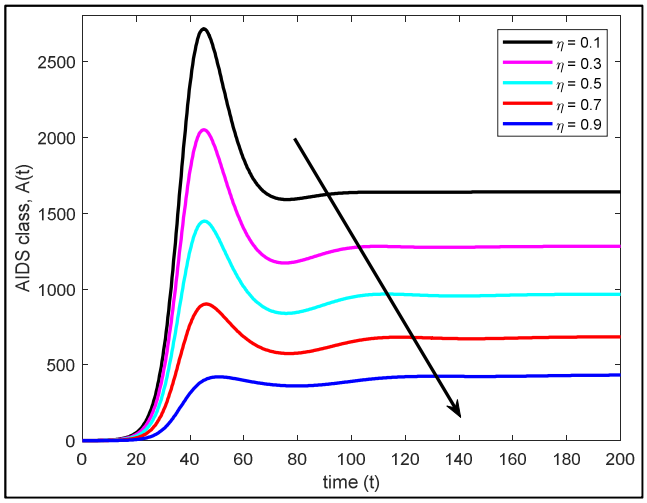

Figure 6. *Effects of the proportion of HIV-infected individuals who get treated on the AIDS class.*

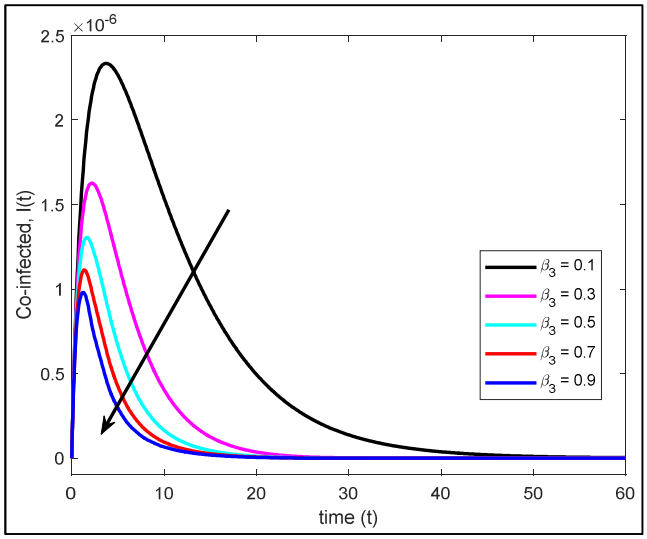

Figure 7. *Effects of the rate of coinfection on the coinfected class.*

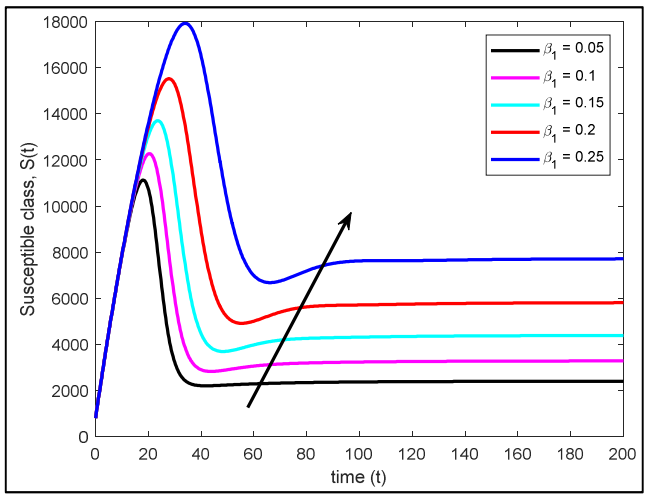

Figure 8. *Effects of the rate at which HIV-infected individuals get treated on the Susceptible class.*

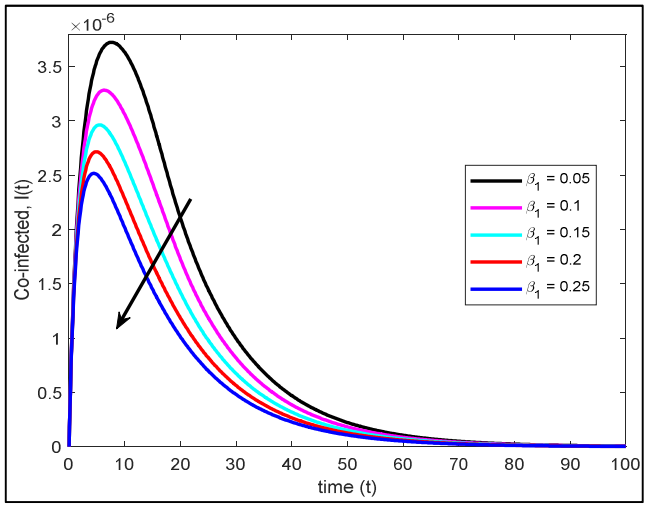

Figure 9. *Effects of the rate at which HIV-infected individuals get treated on coinfected individuals.*

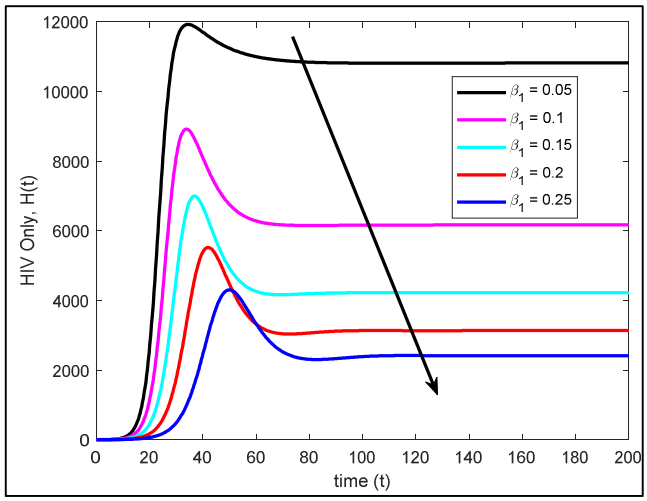

Figure 10. *Effects of the rate at which HIV-infected individuals get treated on HIV class.*

5. Conclusion and Recommendation

5.1. Conclusion

HIV and KS affect millions of people across overlapping geographic locations. The risk of transmission of both diseases can be increased because of co-infection if there is no treatment and preventive measures in place. The model predicts that there is a potential increase in HIV transmission if treatment of HIV and KS is not administered. Mathematical analysis of the model showed that it had a (DFE) with $E_0 = \left(\frac{\lambda}{\mu}, 0, 0, 0, 0, 0\right)$. Depending on the value of the infection rate and the death rate, the results proposed the existence of a unique endemic equilibrium $E_1 = (S_1, H_1, K_1, I_1\, T_1, A_1)$. The infection-free equilibrium E_0 is locally asymptotically stable whenever $R_0^K < 1$ and $R_0^K < 1$. This means that KS and HIV diseases will persist in the population if the reproductive number is > 1 and it go to extinction if it is < 1.

5.2. Recommendation

HIV/AIDS and KS eradication are of concern in developing countries. Thus, there exists a need to strengthen the control interventions and develop new ones that are more effective in combating the scourge. In this regard, we recommend that:

1) KS should be treated as soon as possible to assist strengthen the immune response, which lowers the rate at which AIDS class progression occurs.
2) Co-infection of KS and HIV/AIDS programs should strengthen the awareness and education campaigns with a lot of emphasis on the importance of prompt recognition of symptoms, correct disease diagnosis, HIV screening and early commencement of ARV treatment.

5.3. Possible Future Work

There is a need to have comprehensive research aimed at discovering effective methodological control strategies to curb the scourge of the two diseases thus analysis of the global properties and optimal control of the model should be incorporated. In the future fractional differential equations instead of ordinary differential equations should be adopted.

Abbreviations Nomenclature

HIV	Human immunodeficiency virus
AIDS	Acquired immune deficiency syndrome
KSHV	Kaposi's sarcoma-associated herpes virus
RT	Reverse transcriptase inhibitor
HAART	Highly active antiretroviral therapies
CD4+/T cell	White blood cells in charge of body immunity
PrEP	Post-exposure prophylaxis
NRTIs	Nucleoside or nucleoside reverse transcriptase inhibitors
NNRTIs	Non-nucleoside reverse transcriptase inhibitors
KS	Kaposi’s sarcoma
HHV-8	Human herpes virus 8
AIDS-KS	AIDS-related Kaposi’s sarcoma
ART	Antiretroviral therapy
PIs	Protease inhibitors
TB	Tuberculosis
DFE	Disease free equilibrium
NGM	Next generation matrix
α_2	rate of infection of susceptible individuals with Kaposi’s sarcoma
α_3	contact infection rate of HIV-infected individuals with KS
ϕ	probability of coinfection of susceptible individuals with HIV and KS
A_4	contact infection rate of KS-infected individuals with HIV
θ	proportion of KS infective who recovered after treatment
β_1	rate at which $H(t)$ compartment go for treatment
$\mathcal{R}$	Basic reproduction number
λ	recruitment rate into the susceptible population
$K(t)$	KS individuals infected at time, t
$N(t)$	total human population at time, t
δ	treatment rate
γ	Kaposi sarcoma induced mortality rate
μ	per capita natural death rate
b	natural recovery rate for KS infectives
ρ	AIDS induced mortality rate

β_3	progression rate of coinfected HIV and KS for treatment
η	proportion of HIV infected individuals receiving treatment
β_2	rate at which $K(t)$ compartment go for treatment

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