Mathematical Model on the Effect of Hospital Admission for HIV Infected Individuals on the Rate of Transmission and Mortality

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Abstract

Large scale programmes to provide Anti-retrovirals (ARVs) in Human Immunodeficiency Virus (HIV) patients have expanded and become established in Kenya so access is no longer a problem. However, high transmission and mortality rates still remain a concern. In our study we formulated a mathematical model using Ordinary Differential Equations (ODEs) to determine the effect of increasing admission period for the first (initial) hospital visit of HIV infected individuals on the rate of HIV transmission and mortality. The stability of the equilibria points was analyzed. The existence of locally asymptotic stability of the Disease Free Equilibrium (DFE) was investigated based on the Reproduction number $R_0$. The DFE is Locally Asymptotically Stable (LAS) if $R_0 < 1$ and unstable if $R_0 > 1$. The results show that the DFE is globally asymptotically stable (GAS) when $R_0 < 1$. Sensitivity analysis shows that the most sensitive parameter is the rate of no admission followed by the rate of no hospitalization. The endemic equilibrium is LAS and from the simulations it is observed that increasing admission reduces transmission of HIV.

Keywords: Mortality, Stability

1Corresponding author
1 Introduction

HIV is a lentivirus (a subgroup of retrovirus) that causes HIV infection\[20, 8\]. There are three stages of HIV; acute(early) stage, clinical latency stage (chronic HIV infection) and progression to AIDS (late stage)\[17, ?\]. People in acute stage cause more HIV transmission. Once one is infected the virus duplicates very quickly and the amount of virus and body fluids rises rapidly. The acute period takes place 4-6 weeks after one has been infected by the virus. Following initial infection, a person may experience a bad period of influenza-like illness such as fever, chills, rash, night sweats, muscle aches, sore throat, fatigue, swollen lymph nodes and mouth ulcers\[21, ?\]. Most infected people thus visit hospital at this stage. This is typically followed by a prolonged period without symptoms which may take up to 10 years for some people. As the infection progresses, it interferes more and more with the immune system making the person much more susceptible to common infections like tuberculosis and other opportunistic infections such as kaposi sarcoma and tumors that do not affect people that have working immune system\[14\]. HIV attack the body’s immune system, specifically the CD4 cells (T-cells), which help the immune system fight of infections. When the number of CD4 cells fall below 200 cells per cubic millimeter of blood, one is considered to have progressed to AIDS. Normally CD4 counts are between 500 to 1600 cells per cubic millimeter of blood.

1.1 Antiretrovirals (ARVs)

The common methods of HIV prevention includes; encouraging and practicing safe sex, avoiding sharing of sharp objects such as needles, razor blades and treating with ARVs for those who are already infected\[2\]. There is no cure or vaccine however antiretroviral treatment can slow the cause of the disease and may lead to a near normal life expectancy, antiretroviral treatment reduces the risk of death and complication from the disease\[10\]. These medication are free and easily available. Treatment is recommended as soon as the diagnosis is made. Without treatment the average survival time after infection with HIV is estimated to be 9 to 11 years depending on the HIV subtype.

1.2 Cultural and religious beliefs on HIV

When people become aware of their HIV infection and are counselled well on treatment procedure and provided with care services, most take measures to reduce their risk of transmitting HIV and also are able to take good care of their health. However, if a patient is not closely monitored, counselled and
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guided early enough, strong religious beliefs about faith and healing defer HIV infected persons from continuing with ARVs treatment. Preachers who define healing, culture that points fingers and witches have always turned desperate patients against their life prolonging ARV medication, thus risking their health and making them health risk to HIV negative individuals[6]. An alarming proportion of HIV-positive people have stopped taking ARVs once they have been to diviners and healers who convince them that they are bewitched by particular person(s) or they are cursed. Many more believe that they are being punished by God. It is important that the infected people are closely kept in contact with health care providers so as to dissociate these fears, especially at the early stages of ARVs dispensing. Hence a longer admission period for initial hospital visit is recommended.

1.3 Introduction of the model

Antiretroviral (ARVs) is a life-long commitment that requires patients to adhere diligently to daily medication, dosing schedules and make frequent clinic visits for care. Otherwise levels of virus continue rising and the probability of risk of infection by such patients is high. Despite the availability of free and effective ARVs treatment, there is continued rise in transmission and mortality rates. It therefore means that there are high rates of non compliance of those with HIV in acute (early) stage[9]. Several mathematical models have been formulated on HIV but none on the first (initial) HIV admission period in the hospital has been formulated. We have formulated a mathematical model on the effect of increased admission period for first (initial) hospital visit of HIV infected individuals on the rate of transmission and mortality. With better understanding of the reasons for defaulting (non-compliance), interventions can be designed to improve treatment retention i.e. adopting extended stay in the hospital particularly during the acute stage of HIV infection.

2 Model Description Formulation and Analysis

The compartments of the model are given by the figure below:

The model subdivides the total human population $N_H$ into various components depending on their disease status. $\Lambda_H$ is the constant per capita recruitment rate into susceptible individuals. At time $t$ there are $S_H$ susceptible individuals who are likely to be infected by HIV. $I$ is the number of individuals infected by HIV virus. $I_H$ and $I_{NH}$ represents the HIV infected individuals who visit the hospital and who do not visit the hospital respectively. Those who are admitted at the hospital after visiting are represented by $I_A$. 
whereas those who are not admitted are represented by \( I_{NA} \). The number of the HIV infected individuals who are compliant to ARVs treatment is given by \( I_C \). Those who are not admitted in the hospital and also non compliant to ARVs treatment are denoted by \( I_{NC} \). \( I_{HT} \) denotes HIV infected individuals who have progressed to AIDS, and \( \lambda_s \) is the rate at which the susceptible individuals contact HIV.

\[
\lambda_s = \omega \left( \frac{\phi I_{NA} + \rho I_{NH} + \psi I_{NC}}{N_H} \right)
\]  

(1)

where \( \omega \) is the effective contact rate.

The rate at which individuals infected by HIV in the acute stage visit the hospital is given by \( \alpha \) and \( \beta \) is the rate at which individuals affected by HIV do not visit hospital. \( \epsilon \) is the rate at which individual infected by HIV visit the hospital and are admitted. We assume that every individual admitted in the hospital is compliant to ARV’s treatment and does not progressed to HIV/AIDS. The rate at which the HIV infected individuals who visit the hospital for treatment but are not admitted is given by \( \delta \). \( \pi \) is the rate at which the individuals who are admitted in the hospital are compliant to ARVs treatment. We assume that those people who are compliant to ARVs treatment
are forever in medication. \( \gamma \) and \( \phi \) are the rates at which the individuals who are not admitted in the hospital are compliant to ARVs treatment and not compliant to ARVs treatment respectively usually due to cultural and religious beliefs. The rate at which the individuals who are non compliant to ARVs progress to AIDS is given by \( \psi \).

All individuals in human population experience per capita natural death rate of \( \theta \). Death rate of the individuals infected with HIV in the acute stage is given by \( \mu_A \). \( \mu_B \) and \( g \mu_E \) are the death rates of the individuals infected by HIV after visiting the hospital and those individuals who do not visit the hospital due to the influence of cultural and religious beliefs respectively. \( g \) accounts to increased mortality due to non treatment. Death rates of individuals infected by HIV who visit the hospital and are admitted and those who visit the hospital but are not admitted are given by \( \mu_C \) and \( \mu_D \) respectively.

The death rates of individuals infected by HIV who are admitted in the hospital and are compliant to ARVs treatment is given by \( \mu_F \). \( j \mu_G \) is the death rate of individuals infected by HIV who are not admitted in the hospital and not compliant to ARVs treatment, while \( j \) accounts for increased rate of death due to mistreatment. Finally the death rate of individuals who are infected by HIV and progresses to AIDS is \( \mu \).

From the above definitions and explanations we have formulated the following model of the effect of increased admission period for the first(initial) hospital visit of HIV infected individuals on the rate of transmission and mortality due to influence of cultural and religious beliefs.

\[
\begin{align*}
\frac{dS_H}{dt} &= \Lambda_H - \lambda_S S_H - \theta S_H \\
\frac{dI}{dt} &= \lambda_S S_H - \alpha I - \beta I - \theta I - \mu_A I \\
\frac{dI_H}{dt} &= \alpha I - \epsilon I_H - \delta I_H - \theta I_H - \mu_B I_H \\
\frac{dI_{NH}}{dt} &= \beta I - \mu_F I_{NH} - \theta I_{NH} - \rho I_{NH} \\
\frac{dI_A}{dt} &= \epsilon I_H - \pi I_A - \theta I_A - \mu_C I_A \\
\frac{dI_{NA}}{dt} &= \delta I_H - \phi I_{NA} - \theta I_{NA} - \mu_D I_{NA} - \gamma I_{NA} \\
\frac{dI_C}{dt} &= \pi I_A + \gamma I_{NA} - \mu_F I_C - \theta I_C \\
\frac{dI_{NC}}{dt} &= \phi I_{NA} - \psi I_{NC} - \theta I_{NC} - \mu_G I_{NC} \\
\frac{dI_{HT}}{dt} &= \psi I_{NC} + \rho I_{NH} - \theta I_{HT} - \mu_{HT}
\end{align*}
\]
2.1 Assumption of the model

The following assumptions have been made

(i) Admission in this case is for the individuals who are visiting the hospital for the first time after HIV infection.

(ii) Those who are admitted start seeing positive effects of the ARVs before leaving hospital and remain compliant for the rest of their lives.

(iii) There is no denial of the infection.

(iv) Those who are not admitted maybe influenced by their cultural and religious beliefs and abandon the ARVs.

2.2 Local stability analysis of the disease free equilibrium

The disease free equilibrium (DFE) of the system is given by;

\[ E_0 = (S_H(t), I(t), I_H(t), I_{NH}(t), I_A(t), I_{NA}(t), I_C(t), I_{NC}(t), I_{HT}(t)) \]

\[ = \left( \frac{\Lambda_H}{\mu_H}, 0, 0, 0, 0, 0, 0, 0, 0 \right). \]

To study the stability of disease free equilibrium, we find the basic reproduction number \( R_H \). It is defined as the number of secondary infections that occur when an infected individual is introduced into a completely susceptible population\cite{22}. To calculate \( R_H \) we use the next generation matrix approach. In this method we define curl \( f \) as the rates of the appearance of the new infections in each compartment and curl \( v \) is the rate of transfer of individuals in and out of the particular compartment by any other means. Hence from our model we have;

\[
\begin{bmatrix}
\lambda_S S_H \\
0 \\
0 \\
0 \\
0 \\
0 \\
0 \\
0
\end{bmatrix}
\]

where \( \lambda_S = \omega \left( \frac{\phi I_{NA} + \rho I_{NH} + \psi I_{NC}}{N_H} \right) \).

and
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\[ v = \begin{bmatrix}
    \alpha I + \beta I + \theta I + \mu_A I \\
    -\alpha I + \epsilon I_H + \delta I_H + \theta I_H + \mu_B I_H \\
    -\beta I + \mu_F I_{NH} + \theta I_{NH} + \rho I_{NH} \\
    -\epsilon I_H + \pi I_A + \theta I_A + \mu_C I_A \\
    -\delta I_H + \phi I_{NA} + \theta I_{NA} + \mu_D I_{NA} + \gamma I_{NA} \\
    -\pi I_A - \gamma I_{NA} + \mu_F I_C + \theta I_C \\
    -\phi I_{NA} + \psi I_{NC} + \theta I_{NC} + \mu_G I_{NC} \\
    -\psi I_{NC} - \rho I_{NH} + \theta I_{HT} + \mu I_{HT}
\end{bmatrix} \]

The matrices \( F \) and \( V \) from the partial derivatives of \( f \) and \( v \) with respect to the infected classes computed at disease free equilibrium are given by:

\[
F = \begin{bmatrix}
0 & 0 & \omega \rho & 0 & \omega \phi & 0 & \omega \psi & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0
\end{bmatrix}
\]

and

\[
V = \begin{bmatrix}
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
-\alpha & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
-\beta & 0 & r_3 & 0 & 0 & 0 & 0 & 0 \\
0 & -\epsilon & 0 & r_4 & 0 & 0 & 0 & 0 \\
0 & -\delta & 0 & 0 & r_5 & 0 & 0 & 0 \\
0 & 0 & 0 & -\pi & 0 & r_6 & -\gamma & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & r_7 & 0 \\
0 & 0 & -\rho & 0 & -\phi & 0 & -\psi & r_8
\end{bmatrix}
\]

where

\[
\begin{align*}
    r_1 &= \alpha + \theta + \beta + \mu_A, \\
    r_2 &= \delta + \epsilon + \theta + \mu_B, \\
    r_3 &= \rho + \theta + \mu_E, \\
    r_4 &= \pi + \theta + \mu_C, \\
    r_5 &= \phi + \gamma + \theta + \mu_D, \\
    r_6 &= \theta + \mu_F, \\
    r_7 &= \psi + \theta + \mu_G
\end{align*}
\]
and
\[ r_8 = \theta + \mu - \rho. \]
The eigenvalues of $FV^{-1}$ are 0,0,0,0,0,0,0 and
\[
\frac{\omega \rho \alpha \epsilon}{(\alpha + \theta + \beta + \mu_A)(\delta + \epsilon + \theta + \mu_B)(\pi + \theta + \mu_C)}
\]
The basic reproduction number which is the spectral radius of the matrix $FV^{-1}$ is given by
\[
\rho(FV^{-1}) = \frac{\omega \rho \alpha \epsilon}{(\alpha + \theta + \beta + \mu_A)(\delta + \epsilon + \theta + \mu_B)(\pi + \theta + \mu_C)}.
\]
Therefore,
\[
R_H = \rho(FV^{-1}) = \frac{\omega \rho \alpha \epsilon}{(\alpha + \theta + \beta + \mu_A)(\delta + \epsilon + \theta + \mu_B)(\pi + \theta + \mu_C)}.
\]

**Lemma 1.** The disease free equilibrium is locally asymptotically stable if $R_H < 1$ and unstable if $R_H > 1$. When $R_H < 1$ implies that compliance to ARVs by the HIV infected individuals suppresses the HIV virus to very low level that does not cause transmission.

The above lemma can also be proved using the jacobian matrix at Disease Free Equilibrium $J[E_0]$. We compute the trace and the determinant and apply the set conditions. The jacobian of the full model (2) at $E_0$ is given as;

\[
J = \begin{bmatrix}
-\theta & 0 & 0 & -\omega \rho & 0 & -\omega \phi & 0 & -\omega \psi & 0 \\
0 & s_1 & 0 & -\omega \rho & 0 & -\omega \phi & 0 & -\omega \psi & 0 \\
0 & \alpha & s_2 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & \beta & 0 & s_3 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \epsilon & 0 & s_4 & 0 & 0 & 0 & 0 \\
0 & 0 & \delta & 0 & 0 & s_5 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \pi & \gamma & s_6 & 0 & 0 \\
0 & 0 & 0 & 0 & \phi & 0 & s_7 & 0 & 0 \\
0 & 0 & 0 & 0 & \rho & 0 & 0 & \psi & s_8 \\
\end{bmatrix}
\]

where
\[
s_1 = -\alpha - \beta - \theta - \mu_A, \\
s_2 = -\epsilon - \delta - \theta - \mu_B, \\
s_3 = -\rho - \theta - \mu_F, \\
s_4 = -\pi - \theta - \mu_C, \\
s_5 = -\phi - \theta - \mu_D,
\]
\( s_6 = -\theta - \mu_F, \)
\( s_7 = -\psi - \theta - \mu_G, \)
\( s_8 = -\theta - \mu \)

The trace at DFE is given by;
\[
Tr(E_0) = -[\theta + \alpha + \beta + \mu_A + \epsilon + \delta + \mu_B + \rho + \mu_F + \pi + \mu_C + \phi + \mu_D + \mu_F + \psi + \mu_G + \mu]
\]
which is negative and the determinant at DFE is given by;
\[
Det(E_0) = [\alpha(\theta + \mu_F)(\pi + \theta + \mu_C)(-\omega \rho \epsilon)\{(\alpha + \beta + \theta + \mu_A)(\epsilon + \delta + \theta + \mu_B)(\rho + \theta + \mu_F)(\phi + \theta + \mu_D) \\
(\psi + \theta + \mu_G) + \beta \omega \phi (\epsilon + \delta + \theta + \mu_B)(\psi + \theta + \mu_G)(\phi + \theta + \mu_D) + \alpha \delta (\rho + \theta + \mu_F)(\omega \phi (\psi + \theta + \mu_G) + \phi \psi))\}]
\]
and on substitution with \( R_H \) we obtain
\[
Det(E_0) = [\alpha(\theta + \mu_F)(-\omega \rho \epsilon)\{(\rho + \theta + \mu_F)(\phi + \theta + \mu_D)(R_H - 1) \\
(\psi + \theta + \mu_G) + (\epsilon + \delta + \theta + \mu_B)(\psi + \theta + \mu_G)(\phi + \theta + \mu_D) + (\rho + \theta + \mu_F)(\phi (\psi + \theta + \mu_G) + \phi \psi))\}]
\]
which is positive when \( R_H < 1 \), thus the DFE is locally asymptotically stable.

### 2.3 Global Asymptotic Stability (GAS) of the disease free equilibrium

In this section we analyze the global asymptotic stability of the disease free equilibrium. Using the theorem by Castillo Chavez et. al. The system (2) is rewritten in the form
\[
\frac{dX(t)}{dt} = F(X, Z) \\
\frac{dZ(t)}{dt} = G(X, Z), G(X, 0) = 0 \tag{3}
\]
where \( X = (S_H) \) and \( Z = (I, I_H, I_{NH}, I_A, I_{NA}, I_C, I_{NC}, I_{HT}) \) in which the components of \( X \in \mathbb{R}^{+1} \) denotes the number of uninfected individuals and the components of \( Z \in \mathbb{R}^{+7} \) denote the number of infected individuals with \( E^u(X, 0) \) denoting the DFE of this system.

The two conditions that if met will guarantee the global asymptotic stability of the DFE are
\[
M_1 : \frac{dX(t)}{dt} = F(X, 0), X^* \text{is globally asymptotically stability} \\
M_1 : G(X, Z) = AZ - \hat{G}(X, Z), \hat{G}(X, Z) \geq 0 \text{for } (X, Z) \in \Omega
\]
where \( A = D_Z G(X^*, 0) \) is an M-matrix (off diagonal elements are non-negative) and \( \Omega \) is the region where the model makes biological sense.

If the system (2) satisfies the conditions \( M_1 \) and \( M_2 \) then the theorem (4.1) holds.

**Theorem 1.** The fixed points \( E_0 = (X^*, 0) \) is asymptotically stable equilibrium of the system (2) provided \( R_0 < 1 \) and the assumptions \( M_1 \) and \( M_2 \) are satisfied

**Proof.** Consider

\[
F(X, 0) = (\Lambda_H - \mu_H S_H) \\
G(X, Z) = AZ - \hat{G}(X, Z)
\]

where

\[
A = \begin{bmatrix}
    a & 0 & \omega \rho & 0 & \omega \phi & 0 & \omega \psi & 0 \\
    \alpha & b & 0 & 0 & 0 & 0 & 0 & 0 \\
    \beta & 0 & c & 0 & 0 & 0 & 0 & 0 \\
    0 & \epsilon & 0 & d & 0 & 0 & 0 & 0 \\
    0 & \delta & 0 & e & 0 & 0 & 0 & 0 \\
    0 & 0 & 0 & 0 & \pi + \gamma & f & 0 & 0 \\
    0 & 0 & 0 & 0 & \phi & 0 & g & 0 \\
    0 & 0 & \rho & 0 & 0 & 0 & \psi & -(\theta + \mu)
\end{bmatrix}
\]

where

\[
a = - (\alpha + \beta + \theta + \mu_H),
\]

\[
b = (\epsilon + \delta + \theta + \mu_H),
\]

\[
c = -(\theta + \rho),
\]

\[
d = -(\pi + \theta + \mu_C),
\]

\[
e = -(\phi + \theta + \mu_D + \gamma),
\]

\[
f = -(\theta + \mu_F),
\]

and

\[
g = -(\psi + \theta + \mu_g)
\]

and

\[
\hat{G}(X, Z) = \begin{bmatrix}
    \omega(\rho + \phi + \psi)(1 - \frac{S_H}{N_H}) \\
    0 \\
    0 \\
    0 \\
    0 \\
    0 \\
    0 \\
    0
\end{bmatrix}
\]
Since \( \hat{G}(X, Z) \geq 0 \) for all \((X, Z) \in \mathbb{R}^{+8} \) and matrix \( A \) is an M-Matrix, and since all off diagonal elements of \( A \) are non-negative, the system (2) is Globally Asymptotically Stable.

3 Local stability of the endemic equilibrium (EE) of the model

A disease is endemic if it is persistent in the population. The endemic equilibrium point is denoted by \( E^* \) where 
\[
\]

\[
S^* = \frac{\Lambda_H}{\mu_H \lambda_S^*}
\]

\[
I^* = \frac{\Lambda_H \lambda_S^*}{(\alpha + \beta + \theta + \mu_A)(\mu_H + \lambda_S^*)}
\]

\[
I_H = \frac{\alpha(\Lambda_H \lambda_S^*)}{(\alpha + \beta + \theta + \mu_A)(\mu_H + \lambda_S^*)(\epsilon + \delta + \theta + \mu_B)}
\]

\[
I^*_NH = \frac{\Lambda_H \lambda_S^*}{(\alpha + \beta + \theta + \mu_A)(\mu_H + \lambda_S^*)(\mu_H + \lambda_S^*)}(\mu_E + \theta + \rho)
\]

\[
I_A^* = \frac{\epsilon \alpha \Lambda_H \lambda_S^*}{(\alpha + \beta + \theta + \mu_A)(\mu_H + \lambda_S^*)}(\epsilon + \delta + \theta + \mu_B)(\pi + \theta + \mu_C)
\]

\[
I^*_NA = \frac{\delta \alpha \Lambda_H \lambda_S^*}{(\alpha + \beta + \theta + \mu_A)(\mu_H + \lambda_S^*)(\mu_H + \lambda_S^*)}(\phi + \theta + \mu_B + \gamma)
\]

\[
I_C^* = \frac{\pi I^*_A + \gamma I^*_NA}{(\lambda + \mu_F)}
\]

\[
I^*_NC = \frac{\phi \delta \alpha \Lambda_H \lambda_S^*}{(\alpha + \beta + \theta + \mu_A)(\mu_H + \lambda_S^*)(\epsilon + \delta + \theta + \mu_B)(\phi + \theta + \mu_D + \gamma)(\psi + \theta + j \mu_G)}
\]

\[
I^*_HT = \frac{\psi I^*_NC + \rho I^*_NH}{\theta + \mu}
\]

The stability of the EE of the model is investigated by calculating the trace and determinant of the Jacobian matrix below.

\[
A = \begin{bmatrix}
-\lambda_S^* & 0 & 0 & \omega \lambda^*_H & 0 & 0 & 0 & 0 & 0 \\
\lambda_S^* & u & 0 & \omega \lambda^*_H & 0 & 0 & 0 & 0 & 0 \\
0 & \alpha & v & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & \beta & x & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \epsilon & 0 & y & 0 & 0 & 0 & 0 \\
0 & 0 & \delta & 0 & z & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \pi & \gamma & -(\theta + \mu_F) & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \phi & -(\psi + \theta + j \mu_G) & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \rho & 0 & 0 & \psi & -(\theta + \mu)
\end{bmatrix}
\]
where
\[ u = -(\alpha + \beta + \theta + \mu_A), \]
\[ v = -(\alpha + \epsilon + \delta + \theta + \mu_B), \]
\[ x = -(\rho + \theta + g\mu_E), \]
\[ y = -(-\pi + \theta + \mu_C), \]
\[ z = -(\phi + \theta + \mu_D + \gamma). \]

The trace at Endemic equilibrium is given by;
\[ Tr(E^*) = -[\lambda^*_S + 9\theta + 2\alpha + \beta + \mu_A + \epsilon + \delta + \mu_B + \rho + \mu_F + \pi + \mu_C + \phi + \mu_D + g\mu_E + \psi + j\mu_G + \gamma + \mu] \]

and the determinant at Endemic Equilibrium is given by;
\[ Det(E^*) = -(\pi + \theta + \mu_C)(\theta + \mu_F)(\theta + \mu){((\lambda^*_S + \theta)(\alpha + \beta + \theta + \mu_A)(\alpha + \epsilon + \delta + \theta + \mu_B)(\rho + \theta + j\mu_G)(\phi + \gamma + \theta + \mu_D)) + \alpha((\alpha + \epsilon + \delta + \theta + \mu_B)(\phi + \theta + \gamma + \mu_D)(\psi + \theta + j\mu_G) \omega \rho e + \alpha \delta(\rho + \theta + g\mu_E)(\phi \omega \psi + \omega \phi(\psi + \theta + j\mu_G)))} \]

and on substitution with \( R_H \) we obtain
\[ Det(E^*) = (\omega \rho \epsilon)(\theta + \mu_F)(\theta + \mu)(R_H - 1){((\lambda^*_S + \theta)(\rho + \theta + j\mu_G)(\phi + \gamma + \theta + \mu_D)) + (\phi + \theta + j\mu_G) + \alpha \delta(\rho + \theta + g\mu_E)(\phi \omega \psi + \omega \phi(\psi + \theta + j\mu_G)))}. \]

The trace is negative and the determinant is positive whenever \( R_H > 1 \). Hence, the endemic equilibrium (EE) is asymptotically stable whenever \( R_H > 1 \).

### 4 Sensitivity Analysis

Sensitivity analysis was carried out in order to determine the parameters which have high impact on \( R_H \) that should be targeted for intervention strategies. In order to determine how best to reduce transmission and mortality rates due to HIV infection, it is necessary to know the relative importance of the different factors responsible for its spread and prevalence. The sensitivity indices of the reproduction number are computed using Chitni’s approach [5] as follows: \( \frac{\partial R_H}{\partial B} \frac{B}{R_H} \) and the results are as shown below. The parameter values in Table 2 have been used to calculate the sensitivity indices. The most sensitive parameter is \( \delta \) the rate of no admission followed by the rate of no hospitalization \( \beta \). This means that hospitalization has positive impact on control of HIV transmission and mortality rates.
Table 1: Parameter values

<table>
<thead>
<tr>
<th>parameter</th>
<th>Value</th>
<th>reference</th>
<th>Sensitivity index</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\beta$</td>
<td>0.00998</td>
<td>Assumed</td>
<td>-0.00001502</td>
</tr>
<tr>
<td>$\alpha$</td>
<td>0.00197</td>
<td>Assumed</td>
<td>-0.00020085</td>
</tr>
<tr>
<td>$\delta$</td>
<td>0.00271</td>
<td>Assumed</td>
<td>-0.000013731</td>
</tr>
<tr>
<td>$\phi$</td>
<td>0.00743</td>
<td>Assumed</td>
<td>-0.018355</td>
</tr>
</tbody>
</table>

5 Numerical Simulations

In this section we use numerical simulations in order to give graphical projection of the results of the model. Some of the parameters were obtained from, some were assumed or made varying from realistic simulations results. The simulations are done with varying initial conditions. The parameter values are in Table 2.
Table 2: Numerical Simulations

<table>
<thead>
<tr>
<th>Parameter Description</th>
<th>Symbol</th>
<th>Value</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>human recruitment rate</td>
<td>( \Lambda_H )</td>
<td>( 8.748 \times 10^{-3} \text{day}^{-1} )</td>
<td>CIA(2013)</td>
</tr>
<tr>
<td>human natural mortality rate</td>
<td>( \theta )</td>
<td>( 2.740 \times 10^{-3} \text{day}^{-1} )</td>
<td>CIA(2013)</td>
</tr>
<tr>
<td>Rate of infecting HIV virus</td>
<td>( \lambda_S )</td>
<td>variable</td>
<td>variable</td>
</tr>
<tr>
<td>Rate of visiting the hospital</td>
<td>( \alpha )</td>
<td>0.00197\text{day}^{-1}</td>
<td>Assumed</td>
</tr>
<tr>
<td>Rate of not visiting the hospital</td>
<td>( \beta )</td>
<td>0.00998\text{day}^{-1}</td>
<td>Assumed</td>
</tr>
<tr>
<td>Rate of admission in the hospital</td>
<td>( \epsilon )</td>
<td>variable\text{day}^{-1}</td>
<td>variable</td>
</tr>
<tr>
<td>Rate of not being admitted in the hospital after visiting it</td>
<td>( \delta )</td>
<td>0.00271\text{day}^{-1}</td>
<td>Assumed</td>
</tr>
<tr>
<td>Rate of compliance to ARVs after admission in the hospital</td>
<td>( \pi )</td>
<td>variable\text{day}^{-1}</td>
<td>variable</td>
</tr>
<tr>
<td>Rate of compliance to ARVs after one refuses to be admitted in the hospital</td>
<td>( \gamma )</td>
<td>variable\text{day}^{-1}</td>
<td>variable</td>
</tr>
<tr>
<td>Rate of being non compliance after one refuses to be admitted in the hospital</td>
<td>( \phi )</td>
<td>0.00743\text{day}^{-1}</td>
<td>Assumed</td>
</tr>
<tr>
<td>Rate of progression to AIDS after one is non compliance to ARVs treatment</td>
<td>( \psi )</td>
<td>variable\text{day}^{-1}</td>
<td>variable</td>
</tr>
<tr>
<td>Rate of progression to AIDS after one refuses to visit the hospital</td>
<td>( \rho )</td>
<td>variable\text{day}^{-1}</td>
<td>variable</td>
</tr>
<tr>
<td>Death rate caused by HIV in the acute stage</td>
<td>( \mu_A )</td>
<td>0.0043\text{day}^{-1}</td>
<td>Estimate</td>
</tr>
<tr>
<td>Death rate caused by HIV after visiting the hospital</td>
<td>( \mu_B )</td>
<td>0.0051\text{day}^{-1}</td>
<td>Estimate</td>
</tr>
<tr>
<td>Death rate caused by HIV after not visiting the hospital</td>
<td>( \mu_E )</td>
<td>0.096\text{day}^{-1}</td>
<td>assumed</td>
</tr>
<tr>
<td>Death rate caused by HIV after visiting the hospital and being admitted</td>
<td>( \mu_C )</td>
<td>0.0047\text{day}^{-1}</td>
<td>Assumed</td>
</tr>
<tr>
<td>Death rate caused by HIV after visiting the hospital and one refuses to be admitted</td>
<td>( \mu_D )</td>
<td>0.0062\text{day}^{-1}</td>
<td>Assumed</td>
</tr>
<tr>
<td>Death rate caused by HIV after being compliance to ARVs treatment</td>
<td>( \mu_F )</td>
<td>0.0046\text{day}^{-1}</td>
<td>Estimate</td>
</tr>
<tr>
<td>Death rate caused by HIV after being non compliance to ARVs treatment</td>
<td>( \mu_G )</td>
<td>0.0077\text{day}^{-1}</td>
<td>Assumed</td>
</tr>
<tr>
<td>Death rate caused by HIV after the virus progresses to HIV/AIDS</td>
<td>( \mu )</td>
<td>0.0099\text{day}^{-1}</td>
<td>Estimate</td>
</tr>
<tr>
<td>Modification parameter</td>
<td>( j,g )</td>
<td>0.0008423,0.000756</td>
<td>Assumed</td>
</tr>
</tbody>
</table>

Figure 1 shows the solution dynamics of Equation (2) in the absence of infectives. In this case, the only stable steady state is the HIV/AIDS free equilibrium \( E_0 \).
Transmission and mortality

From Figure 1, we observe that the solutions converge to HIV/AIDS free steady state $E_0$, where the disease is wiped out.

In the presence of interventions as shown in Figure 2, the increase in the number of admissions results in the decrease in the transmission of HIV, i.e. the reduction of the number of infectives in the population.

From Figure 3, the increase in the number of individuals hospitalized results in a decrease in the transmission of HIV, i.e. the reduction of the number of
infectives in the population.

![Figure 3: Numerical solution on the impact of hospitalization in the transmission of HIV](image1)

In the absence of intervention strategies; admissions and hospitalization, the number of infectives persists in the population. This is as described in Figure 4 below;

![Figure 4: Numerical solution on the absence of intervention strategies](image2)
6 Conclusion

The study has addressed the treatment of HIV in its very early stages using deterministic mathematical model which incorporates the admission of patients for some period. From our results there is evidence that LAS and the GAS are asymptotically stable when $R_H < 1$. The sensitivity analysis has indicated that hospitalization is an important prenominal in reducing the transmission of HIV. Those admitted are further counselled and become more compliant to ARV treatment thus reducing transmission and mortality rates even further, as is observed in the simulation graphs. The endemic equilibrium which is LAS is an indication that hospitalization and admission reduces the possibility of epidemic.

References


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