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Modelling the spread of HIV and AIDS epidemic trends in male and female populations*

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Abstract. We develop and use mathematical models that describe a theoretical analysis of how to model the spread of human immunodeficiency virus (HIV) and acquired immune deficiency syndrome (AIDS) epidemics with constant inflow of migration into the male and female populations. We did not consider all of the stages of HIV transmission; however, we present a comparative model that is sufficiently comprehensive to investigate the spread of HIV/AIDS in aheterogeneous population. Migration is included in the model to consider its effect on the spread of HIV/AIDS to the general population.We calculate the reproduction number for each population separately to determine whether the disease-free equilibrium is globally stable. The Routh-Hurwitz condition and the Lyapunov function are used to validate the variety of model. Finally, we establish the global stability of the various endemic equilibrium states.

Keywords: epidemiological model, threshold parameter, stability, Lyapunov function, Routh-Hurwitz condition

1 Introduction

Globally human immunodeficiency virus (HIV) as the aetiological agent of the acquired immune deficiency syndrome (AIDS)is the most devastating disease humankind has ever faced. AIDS, which is the most advanced stage of HIV, has caused the deaths of millions of individuals in both developed and developing countries. Currently, there is no cure for HIV or AIDS. However, treatment is available that reduces the spread. Since the beginning of this chronic disease epidemic, 78 million people have been infected,and out of this number 39 million people have died of HIV[35]. The World Health Organ is at ion in 2014 reported that one out of every 20 adults lives with HIV in Sub-Saharan Africa, which has the fastest growing rate of disease spread,representing 71% of individuals living with HIV globally,followed by South and Southeast Asia, while Oceania has the slowest rate[35]. Mathematical models have dealt widely with how to model the epidemiology of HIV and AIDS to improve awareness of the factors that affect the spread of the epidemic. For instance, during the initial stages of HIV and AIDS emergence in the 1980s, May and Anderson produced various models to outline the factors that have impacted the spread of the disease[1–3, 29]. Vincenzi[16] identified how to show the risk factors for heterosexual transmission of HIV by comparing the efficiency of male-to-female and female-to-male transmission.In 2008, Kandala et al.[22] presented an epidemiological study ofHIV infection in Zambia for the male and female populations. The average ages that were used for males and females diagnosed with HIV were 30 and 28,respectively.Dushoffet al.[12] formulated a simple model to present the gendered perspective of male circumcision to reduce the spread of the HIV epidemic in Sub-Saharan Africa. They determined that the

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effect of circumcision is good for men but bad for women since this contributes to the spread of HIV during sex.The effects of outbound migrants and outbound migration on the spread of the HIV/AIDS epidemic in India were model led by Deering et al.[9]. Mathematical studies of HIV and AIDS have been an area of vehement theoretical analysis. Recently, further studies have strengthened ideas of how to model the spread of HIV and AIDS using prevention and control systems to provide public health education and counselling [4, 10, 31, 34, 42]. Nyabadza et al.[15] developed a simple mathematical model to study trends in the HIV/AIDS epidemic in South Africa. They used their model to predict the HIV prevalence trend from 1990-2007. Reproduction numbers play a pivotal role in determining the spread of an infectious disease.To give a good description of epidemic, the reproduction number gives an idea about the disease and whether the epidemic will either grow or decline[36]. Reproduction numbers give a vivid description as to the number of secondary infections caused by one infected individual on average throughout his/herinfected period and give better insight into the probable course of the infection[18, 19, 36, 40]. The reproduction number is the expected number of secondary cases produced by a typical infection in a completely susceptible population[40]. If $R_0 > 1$, an epidemic occurs; if$R_0 < 1$, an epidemic does not occur.And if $R_0 = 1$(the threshold),a change in stability occurs[19]. There is very little understanding of how to model infection for the two different genders.

In this paper, we address the question of how to model the transmission of HIV and AIDS in two different populations while taking other demographic and epidemiologic parameters into account to study the relationship between various reproduction numbers. It is important to mention here that our work is different from some of the other related works cited in this paper because the model takes into account the recruitment of external migrants into the susceptibility of males and females. We also assume that the infected individuals are capable of having children that are either infected with HIV or will not have HIV.In addition, most of the papers cited did not take into consideration the possible contact rates between the male and female populations but we are incorporating the contact rates between the AIDS class and the susceptible class, which is more realistic. Moreover, none of the models presented in those papers address how closely linked the two genders are in heterosexual transmission as well as homosexual transmission. The combination of heterosexual and homosexual transmission[14, 17, 39] is different from most of the papers cited that concentrate on either of the two routes.

2 Model formulation

There are several mathematical models for describing HIV/AIDS epidemics with different properties with respect to mortality, immunity, and time horizon[8, 13, 21, 32, 41]. In this paper, one of these mathematical models is examined. Precisely,the population is divided into two categories (that is, the male and female populations). For HIV, once an individual becomes infected, that individual remains either infected or infectious for life until she or he dies. Due to this reality we use Susceptible-Infected-Removal (SIR) models[23–26]. We replaced the removal class by the AIDS class, which then becomes aSusceptible-Infected-AIDS (SLA) model. A susceptible individual acquires HIV when an infected person's bodily fluids (blood, semen, fluids from the vagina or breast milk) enter his or her bloodstream. When a susceptible individual becomes infected, she/he moves to the infected HIV compartment and finally moves to the AIDS category.

Let $S_1(t)$, $S_2(t)$, $I_1(t)$, $I_2(t)$, $A_1(t)$ and$A_2(t)$ denote the number of susceptible males and females, HIV-infected males and females, and males and females with AIDS, respectively, at time with a constant inflow of migrations at a rate . The subscripts represent gender (1 for male and 2 for female), and all individuals are sexually active and have a natural mortality rate in all the classes. In 2011, Zhang et al.[43] presented an epidemic model of HIV/AIDS in Yunnan, China using local prevalence rates. They took into account injection drug users and individuals who engage in commercial sex work. Unlike the model from [43], and Naresh[33], who also assumed in his paper that the AIDS class and those in the pre-AIDS class were sexually inactive and therefore were not capable of giving birth, in our model, we include the novel feature of newborn cases of infected individuals $b(I_2 + A_2)$, for which we assume that $I_1(t)$, $I_2(t)$, $A_1(t)$ and$A_2(t)$ are sexually active[5, 7, 30], and $\varepsilon\omega\, b\,(I_2 + A_2)$ are infected with HIV and join the population of females with HIV. The remaining $\varepsilon(1 - \omega)b(I_2 + A_2)$ are susceptible females, where ω is the fraction of newborns not living with HIV. In a similar way, there will be $\rho(1 - \varepsilon)b(I_2 + A_2)$ individuals who are infected with HIV and join the population of males with HIV and the proportion of newborn males is ρ. The remaining $(1 - \rho)(1 - \varepsilon)b(I_2 + A_2)$ individuals

are susceptible male individuals, where ε is the fraction of newborn males born to females with HIV. Let $d_i(i = 1, 2)$ denote the disease-induced death rates due to infected HIV individuals for males and females, respectively, while $\delta_i(i = 1, 2)$ denotes the disease-induced death rates due to individuals with AIDS for males and females, respectively. The rates of removal of HIV-infected individuals who move to become individuals with AIDS are α_1 and α_2 for males and females, respectively. β_1, β_2, σ_1, σ_2, γ_1, γ_{12}, θ_1 and θ_2 are the contact rates for susceptible individuals with HIV and AIDS, respectively, as indicated by the dotted lines shown in Fig. 1.

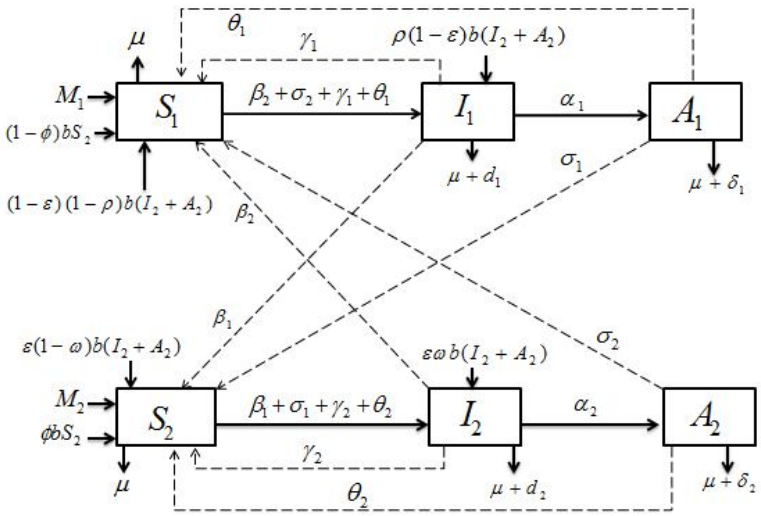

Fig. 1: Schematic flow diagram of male and female of HIV/AIDS populations

With these assumptions the dynamics of the male and female populations are assumed to bed escribed by the following set of nonlinear ordinary differential equations:

$$\begin{aligned}
S_1' &= M_1 + (1-\varphi)bS_2 + (1-\rho)(1-\varepsilon)b(I_2+A_2) - \beta_2 I_2 S_1 - \sigma_2 A_2 S_1 - \gamma_1 I_1 S_1 \\
&\quad - \theta_1 A_1 S_1 - \mu S_1 \\
I_1' &= \rho(1-\varepsilon)b(I_2+A_2) + \beta_2 I_2 S_1 + \sigma_2 A_2 S_1 + \gamma_1 I_1 S_1 + \theta_1 A_1 S_1 - (d_1+\alpha_1+\mu)I_1 \\
A_1' &= \alpha_1 I_1 - (\delta_1+\mu)A_1 \\
S_2' &= M_2 + \varphi bS_2 + \varepsilon(1-\omega)b(I_2+A_2) - \beta_1 I_1 S_2 - \sigma_1 A_1 S_2 - \gamma_2 I_2 S_2 - \theta_2 A_2 S_2 - \mu S_2 \\
I_2' &= \varepsilon\omega b(I_2+A_2) + \beta_1 I_1 S_2 + \sigma_1 A_1 S_2 + \gamma_2 I_2 S_2 + \theta_2 A_2 S_2 - (d_2+\alpha_2+\mu)I_2 \\
A_2' &= \alpha_2 I_2 - (\delta_2+\mu)A_2
\end{aligned} \tag{1}$$

The variables must satisfy $N_1(t) = S_1(t)+I_1(t)+A_1(t)$ and $N_2(t) = S_2(t)+I_2(t)+A_2(t)$, respectively, where N_1 and N_2 are the total number of males and females in the population at time t.

Let $N_1(t) = S_1(t) + I_1(t) + A_1(t)$ and $N_2(t) = S_2(t) + I_2(t) + A_2(t)$, then Eq. (1) can be reduced to

$$\begin{aligned}
I_1' &= \rho(1-\varepsilon)b(I_2+A_2) + (\beta_2 I_2 + \sigma_2 A_2 + \gamma_1 I_1 + \theta_1 A_1)(N_1 - I_1 - A_1) - (d_1+\alpha_1+\mu)I_1 \\
A_1' &= \alpha_1 I_1 - (\delta_1+\mu)A_1 \\
I_2' &= \varepsilon\omega b(I_2+A_2) + (\beta_1 I_1 + \sigma_1 A_1 + \gamma_2 I_2 + \theta_2 A_2)(N_2 - I_2 - A_2) - (d_2+\alpha_2+\mu)I_2 \\
A_2' &= \alpha_2 I_2 - (\delta_2+\mu)A_2.
\end{aligned} \tag{2}$$

The feasible region Δ of Eq. (2) is defined as $\Delta = \{x = (I_i, A_i) \in \Re_+^4 \,|I_i \geq 0, A_i \geq 0, \text{for } i = 1,\ 2\}$.

Lemma 1. *System (2) has no periodic solution in Δ, and this closed set is forward invariant.*

Proof. The proof follows from the Dulac's criterion and the Poincare-Bendixson Theorem. Let us rewrite system (2) as

$$
\begin{aligned}
I_1' &= E(I_1A_1I_2A_2) = \rho(1-\varepsilon)b(I_2+A_2) + (\beta_2 I_2 + \sigma_2 A_2 + \gamma_1 I_1 + \theta_1 A_1)(N_1 - I_1 - A_1) \\
&\quad - (d_1 + \alpha_1 + \mu)I_1 \\
A_1' &= F(I_1A_1I_2A_2) = \alpha_1 I_1 - (\delta_1 + \mu)A_1 \\
I_2' &= G(I_1A_1I_2A_2) = \varepsilon\omega b(I_2 + A_2) + (\beta_1 I_1 + \sigma_1 A_1 + \gamma_2 I_2 + \theta_2 A_2)(N_2 - I_2 - A_2) \\
&\quad - (d_2 + \alpha_2 + \mu)I_2 \\
A_2' &= H(I_1A_1I_2A_2) = \alpha_2 I_2 \\
&\quad - (\delta_2 + \mu)A_2.
\end{aligned} \tag{3}
$$

Choose $B(I_iA_i) = \frac{1}{I_i}$ as a Dulac function. Then,

$$
\begin{aligned}
&\tfrac{\partial}{\partial I_1}(BE) + \tfrac{\partial}{\partial A_1}(BF) + \tfrac{\partial}{\partial I_2}(BG) + \tfrac{\partial}{\partial A_2}(BH) = \\
&+ \tfrac{\partial}{\partial I_1}\left(\rho(1-\varepsilon)b(I_2+A_2) + (\beta_2 I_2 + \sigma_2 A_2 + \gamma_1 + \theta_1 \tfrac{A_1}{I_1})(N_1 - I_1 - A_1) - (d_1 + \alpha_1 + \mu)\right) \\
&+ \tfrac{\partial}{\partial A_1}\left(\alpha_1 - (\delta_1 + \mu)\tfrac{A_1}{I_1}\right) \\
&+ \tfrac{\partial}{\partial I_2}\left(\varepsilon\omega b(1 + \tfrac{A_2}{I_2}) + (\beta_1 I_1 + \sigma_1 A_1 + \gamma_2 + \theta_2 \tfrac{A_2}{I_2})(N_2 - I_2 - A_2) - (d_2 + \alpha_2 + \mu)\right) \\
&+ \tfrac{\partial}{\partial A_2}\left(\alpha_2 - (\delta_2 + \mu)\tfrac{A_2}{I_2}\right) \\
&= -\gamma_i - \tfrac{(\delta_i + \mu)}{I_i} < 0, \text{for all } I_i > 0.
\end{aligned}
$$

By the Dulac's criterion, system (2) has no period solutions contained in Δ. With the derivatives in the normal directions to the boundaries of Δ, we have shown that this region is positively invariant under model (2), hence the system is both epidemiologically and mathematically well posed. Therefore, it is sufficient to study the dynamics of the model in Δ.

3 Stability analysis

In this section, we consider the stability analysis of the disease at its equilibrium points.

3.1 Equilibria of the model

From our model (1), there are two non-negative equilibrium points named below.

(i) The disease-free equilibrium

We determine the disease-free equilibrium $E_0 = (S_1^0, 0, 0, S_2^0, 0, 0)$ by simple calculation, and we obtain: $S_1^0 = M_1 + \frac{M_2(1-\varphi)b}{\mu - \varphi b}$, and $S_2^0 = \frac{M_2}{\mu - \varphi b}$. .

(ii) The endemic equilibria

From Eq. (1) we obtain results by using these two boundary equilibria $E_1 = (S_{11}^0, I_{11}^0, A_{11}^0, S_{21}^0, 0, 0)$ with these conditions because the parameters are non-zeros where $k_1 S_{11} \neq 0$, $k(\theta_1 M_1 - k_3) + \alpha_1^2 k_2 \theta_1 \sigma_1 \neq 0$, and $E_2 = (S_{12}^0, 0, 0, S_{22}^0, I_{22}^0, A_{22}^0)$ are given by

$$
\begin{aligned}
S_{21} &= \tfrac{(\delta_1+\mu)[M_1 - S_{11}(\gamma_1 I_{11} + \mu)] - \theta_1 \alpha_1 S_{11} I_{11}}{(\delta_1 + \mu)}, \\
S_{11} &= \tfrac{(\delta_1+\mu)(d_1+\alpha_1+\mu)}{(\delta_1+\mu)\gamma_1 + \theta_1\alpha_1}, \\
I_{11} &= \tfrac{k_1(1-\varphi)bM_2 - k_1k_4}{k_1(\theta_1 M_1 - k_3) + \alpha_1^2 k_2 \theta_1 \sigma_1}, \\
A_{11} &= \tfrac{\alpha_1}{\delta_1 + \mu} I_{11}, \\
S_{12} &= \tfrac{M_1 + (1-\varphi)bS_{22} + (1-\rho)(1-\omega)(I_{22} + A_{22})}{\beta_2 I_{22} + \sigma_2 A_{22} + \mu}, \\
S_{22} &= \tfrac{d_2 + \alpha_2 + \mu}{(\delta_2+\mu)(\gamma_2 + \varepsilon\omega b) + \alpha_2(\theta_2 + \varepsilon\omega b)}, \\
I_{22} &= \tfrac{q_1(\mu - \varphi b) - M_2 q_2}{\varepsilon(1-\omega)bq_3 - q_4}, \\
A_{22} &= \tfrac{\alpha_2}{\delta d_2 + \mu} I_{22}.
\end{aligned}
$$

where

$$
\begin{aligned}
k_1 &= (\mu+\delta_1)\beta_1\gamma_1 + \beta_1\theta_1\alpha_1 + \gamma_1\theta_1\alpha_1,\\
k_2 &= \tfrac{(\varphi\theta b-\sigma_1\mu-\theta_1\mu)(\mu+\delta_1)(\mu+d_1+\alpha_1)+\beta_1 M_1[(\mu+\delta_1)\gamma_1+\theta_1\alpha_1]}{(\mu+\delta_1)\gamma_1+\theta_1\alpha_1},\\
k_3 &= \tfrac{(\mu+\delta_1)(\mu+d_1+\alpha_1)\alpha_1(\varphi\theta b-\sigma_1\mu-\theta_1\mu)}{(\mu+\delta_1)\gamma_1+\theta_1\alpha_1},\\
k_4 &= \tfrac{(\varphi b-\mu)(\mu+\delta_1)}{(\mu+\delta_1)\gamma_1+\theta_1\alpha_1}\left\{(\mu+d_1+\alpha_1) - M_1((\mu+\delta_1)\gamma_1+\theta_1\alpha_1)\right\},\\
q_1 &= (\delta_2+\mu)(d_2+\alpha_2+\mu),\\
q_2 &= (\delta_2+\mu)[(\delta_2+\mu)(\gamma_2+\varepsilon\omega b)+\alpha_2(\theta_2+\varepsilon\omega b)],\\
q_3 &= q_2(d_2+\alpha_2+\mu),\\
q_4 &= (d_2+\alpha_2+\mu)[(\delta_2+\mu)\gamma_2+\theta_2\alpha_2)].
\end{aligned}
$$

The system (1) also has a unique endemic equilibrium $E^0 = (S_1^0, I_1^0, A_1^0, S_2^0, I_2^0, A_2^0)$ with

$$
\begin{aligned}
S_1^0 &= \tfrac{(\delta d_1+\mu)[(1-\delta)d_1+\alpha_1+\mu]}{(\delta d_1+\mu)\gamma_1+\theta_1\alpha_1},\\
I_1^0 &= \tfrac{k_1(1-\varphi)bM_2-k_1k_4}{k_1(\theta_1M_1-k_3)+\alpha_1^2k_2\theta_1\sigma_1},\\
A_1^0 &= \tfrac{\alpha_1}{\delta_1+\mu}I_1^0,\\
S_2^0 &= \tfrac{(\delta d_2+\mu)[(1-\delta)d_2+\alpha_1+\mu]-\omega b(\delta d_2+\alpha_1+\mu)}{(\delta d_2+\mu)\gamma_2+\theta_2\alpha_2},\\
I_2^0 &= \tfrac{(\mu-\varphi b)q_1-M_2(\delta_2+\mu)q_2}{\varepsilon(1-\omega)b(\delta_2+\alpha_2+\mu)q_2-q_3},\\
A_2^0 &= \tfrac{\alpha_2}{d_2+\mu}I_2^0.
\end{aligned}
$$

3.2 Local stability of the equilibrium

The local stability of E^0 will be investigated using the next generation approach [11, 19, 20, 37, 38, 40] with the same boundary conditions[6]. Similar notations were used in[11], and the non-negative matrix, is the new infection term of an matrix, while is the transition term associated with model (1) for the male population with

$$
F_1 = \begin{bmatrix} \gamma_1 S_1^0 & \theta_1 S_1^0 \\ 0 & 0 \end{bmatrix}, \tag{4}
$$

$$
V_1 = \begin{bmatrix} d_1+\alpha_1+\mu & 0 \\ -\alpha_1 & \delta_1+\mu \end{bmatrix}. \tag{5}
$$

The reproduction number is a threshold value or number that determines the stability of the disease-free equilibrium. From Eq. (4) and Eq. (5), the reproduction number denoted by $R_0 = \rho(F_1V_1^{-1})$, where represents the spectral radius, is given by

$$
R_0^M = \frac{\gamma_1 S_1^0}{d_1+\alpha_1+\mu}.
$$

On the other hand, for the female population the same approach is applied to find the reproduction number. For a non-negative matrix, is the new infection term of a 2×2 matrix, and V_2 is the transition term associated with model (1) for the female population by

$$
F_2 = \begin{bmatrix} \gamma_2 S_2^0 & \theta_2 S_2^0 \\ 0 & 0 \end{bmatrix} \tag{6}
$$

$$
V_2 = \begin{bmatrix} d_2+\alpha_2+\mu-\varepsilon\omega b & -\varepsilon\omega b \\ -\alpha_2 & \delta_2+\mu \end{bmatrix}. \tag{7}
$$

From Eq. (6) and Eq. (7), the reproduction number denoted by $R_0 = \rho(F_2V_2^{-1})$, where ρ represents the spectral radius, is given by

$$
R_0^F = \frac{\gamma_2 S_2^0(\delta_2+\mu)}{(\alpha_2+\mu+d_2)(\delta_2+\mu-\varepsilon\omega b)}.
$$

Theorem 1. *The disease-free equilibrium E_0 is globally asymptotically stable if $R_0 \leq 1$ but is unstable if $R_0 > 1$.*

Proof. The stability of the endemic equilibrium is determined using the eigenvalues of the characteristic equation of the corresponding Jacobian matrix, $J(S_1, I_1, A_1) = J(E_{01})$, which is given by:

$$J(E_{01}) = \begin{bmatrix} -\mu & -\gamma_1 S_1^0 & -\theta_1 S_1^0 \\ 0 & \gamma_1 S_1^0 - (\mu + d_1 + \alpha_1) & \theta_1 S_1^0 \\ 0 & \alpha_1 & -(\mu + \delta_1) \end{bmatrix}. \tag{8}$$

The characteristic equation corresponding to $J(E_{01})$ is given by

$$f(\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0. \tag{9}$$

where

$$\begin{aligned} a_1 &= \alpha_1 + d_1 + \delta_1 + 3\mu - \mu S_1^0, \\ a_2 &= (\alpha_1\theta_1 - \delta_1\gamma_1)S_1^0 + 2\mu(\alpha_1 - \gamma_1 S_1^0) + (\alpha_1 + 2\mu)(d_1 + \delta_1) + 3\mu^2, \\ a_3 &= \mu^2(\alpha_1 + \delta_1 + d_1) + \alpha_1\mu(\delta_1 - \theta_1 S_1^0) - \mu\gamma_1 S_1^0(\mu + \delta_1) + \mu^3. \end{aligned}$$

Therefore, by the Routh-Hurwitz criteria, E_{01} is locally asymptotically stable as it can be seen that $a_1 > 0$, $a_3 > 0$ and $a_1a_2 > a_3$.

$$J(E_{02}) = \begin{bmatrix} \varphi b - \mu & \varepsilon(1-\omega)b - \gamma_2 S_2^0 & \varepsilon(1-\omega)b - \theta_2 S_2^0 \\ 0 & \varepsilon\omega\, b + \gamma_2 S_2^0 - (\mu + d_2 + \alpha_2) & \varepsilon\omega\, b + \theta_2 S_2^0 \\ 0 & \alpha_2 & -(\mu + \delta_2) \end{bmatrix} \tag{10}$$

The characteristic equation corresponding to $J(E_{02})$ is given by

$$f(\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0 \tag{11}$$

where

$$\begin{aligned} a_1 &= \alpha_2 - \varphi b - \varepsilon\omega b + d_2 + \delta_2 + \gamma_2 S_2^0 + 3\mu, \\ a_2 &= \alpha_2\delta_2 - \alpha_2\varphi b - \alpha_2\varepsilon\omega b + \alpha_2\theta_2 S_2^0 + 2\alpha_2\mu + d_2\delta_2 + 2\mu d_2 - 2\mu\gamma_2 S_2^0 + \varepsilon\omega\varphi b^2 \\ &\quad - \varphi b d + \varphi\gamma_2 b S_2^0 - \varphi b\delta - 2\varphi b\mu - 2\mu\varepsilon\omega b - \varepsilon\omega b + 2\mu d + 3\mu^2 + 2\mu\delta_2, \\ a_3 &= \alpha_2\varphi\varepsilon\omega b^2 - \alpha_2\varphi\delta_2 b + \alpha_2\varphi\theta_2 b S_2^0 - \alpha_2\varphi\mu b - \alpha_2\varepsilon\omega\mu b - \alpha_2\theta_2 S_2^0 + \alpha_2\mu^2 \\ &\quad + \alpha_2\mu\delta + \mu\varphi\varepsilon\omega b^2 + \delta_2\varphi\varepsilon\omega b^2 - \delta_2\varphi b d_2 - \mu\varphi b d_2 + \varphi\mu\gamma_2 b S_2^0 - \varphi\mu^2 b \\ &\quad - \varphi\mu\delta b - \mu^2\varepsilon\omega b - \mu\varepsilon\omega\delta b + \mu^2 d + \mu\delta d + \mu^2\gamma_2 S_2^0 - \mu\gamma_2\delta\, S_2^0 + \mu^3 + \delta\mu^2. \end{aligned}$$

Therefore, by the Routh-Hurwitz criteria, E_{02} is locally asymptotically stable as it can be seen that $a_1 > 0$, $a_3 > 0$ and $a_1a_2 > a_3$. The proof is complete.

3.3 Global stability of the disease-free equilibrium E_0

First and foremost, we find the maximum invariant set Ω of model (1)-(6), and we obtain $\Omega = \left\{(S_1, I_1, A_1, S_2, I_2, A_2) \in \Re_+^6 : S \le S_i^0, i = 1, 2, N_1 \le \frac{M_1 + (1-\varphi)bS_2^0}{\mu}, N_2 \le \frac{M_2}{\mu - \varphi b}\right\}$. where $S_1'(t) \le M_1 + (1-\varphi)bS_2 - \mu S_1$, and $S_2'(t) \le M_2 + \varphi bS_2 - \mu S_2$, because $S_1(0) \le S_1^0$ and $S_2(0) \le S_2^0$, which implies that $S_1(t) \le S_1^0$ and $S_2(t) \le S_2^0$ for all $t \ge 0$.

Furthermore, $\begin{aligned} N_1'(t) &\le M_1 + (1-\varphi)bS_2 - \mu N_1, \\ N_2'(t) &\le M_2 + \varphi bS_2 - \mu N_2. \end{aligned}$

$N_1(0) \le \frac{M_1 + (1-\varphi)bS_2^0}{\mu}$ and $N_2(0) \le \frac{M_2}{\mu - \varphi b}$ gives $N_1(t) \le \frac{M_1 + (1-\varphi)bS_2^0}{\mu}$ and $N_2(t) \le \frac{M_2}{\mu - \varphi b}$ for all $t > 0$. Thus, it is sufficient to consider the model (1)-(6) with respect to Ω.

3.3.1 Male population

When $R_0 \le 0$, we consider the following linear Lyapunov function to address the disease-free equilibrium:

$$
\begin{aligned}
V_1(t) &= \phi I_1 + A_1 \qquad (12)\\
V_1'(t) &= \phi I_1' + A_1' \le \phi(\gamma_1 I_1 S_1 + \theta_1 A_1 S_1 - (d_1+\alpha_1+\mu)I_1) + \alpha_1 I_1 - (\mu+\delta_1)A_1\\
&= \phi(d_1+\alpha_1+\mu)\left\{\left(\frac{\gamma_1 S_1^0}{d_1+\alpha_1+\mu} - 1\right) I_1 + \alpha_1 I_1 + A_1\left(\frac{\theta_1 S_1^0}{d_1+\alpha_1+\mu} - (\mu+\delta_1)\right)\right\} \qquad (13)\\
&= \phi(d_1+\alpha_1+\mu)(R_0^M - 1)I_1 + (d_1+\alpha_1+\mu)\alpha_1 I_1 + \theta_1 A_1 S_1^0 - (d_1+\alpha_1+\mu)(\mu+\delta_1)A_1\\
&\le 0
\end{aligned}
$$

where $\phi = \frac{1}{(d_1+\alpha_1+\mu)}$, $S_1 = S_1^0$, only $V_1'(t) = 0$, $I_1 = 0$ and $A_1 = 0$.

3.3.2 Female population

When $R_0 < 0$, we consider the following linear Lyapunov function to address the disease-free equilibrium:

$$
\begin{aligned}
V_2(t) &= \eta I_2 + A_2 \qquad (14)\\
V_2'(t) &= \eta I_2' + A_2'\\
&= \eta\left(\varepsilon\omega b(I_2+A_2) + \gamma_2 I_2 S_2 + \theta_2 A_2 S_2 - (d_2+\alpha_2+\mu)I_2\right) + \alpha_2 I_2 - (\mu+\delta_2)A_2\\
&= \eta\left\{\varepsilon\omega b\left(I_2 + \tfrac{\alpha_2 I_2}{\mu+\delta_2}\right) + \gamma_2 I_2 S_2 + \tfrac{\alpha_2\theta_2 I_2 S_2}{\mu+\delta_2} - (d_2+\alpha_2+\mu)I_2\right\} + \alpha_2 I_2 - (\mu+\delta_2)A_2\\
&= \eta\left\{\varepsilon\omega b(\mu+\delta_2+\alpha_2)I_2 + (\mu+\delta_2)\gamma_2 I_2 S_2 + \alpha_2\theta_2 I_2 S_2 - (\mu+\delta_2)(d_2+\alpha_2+\mu)I_2\right\}\\
&\quad + (\mu+\delta_2)\left\{\alpha_2 I_2 - (\mu+\delta_2)A_2\right\}\\
&= \eta\left\{(\mu+\delta_2)\gamma_2 I_2 S_2 - (\mu+\delta_2+\alpha_2)(\mu+\delta_2-\varepsilon\omega b)I_2 + \alpha_2\theta_2 I_2 S_2\right\} \qquad (15)\\
&\quad + (\mu+\delta_2)\left\{\alpha_2 I_2 - (\mu+\delta_2)A_2\right\}\\
&= \eta\left\{\tfrac{(\mu+\delta_2)\gamma_2 I_2 S_2}{(\mu+\delta_2+\alpha_2)(\mu+\delta_2-\varepsilon\omega b)} - 1\right\} I_2 + \eta\alpha_2\theta_2 I_2 S_2 + (\mu+\delta_2)\left\{\alpha_2 I_2 - (\mu+\delta_2)A_2\right\}\\
&= \eta\left(R_0^F - 1\right)I_2 + \eta\alpha_2\theta_2 I_2 S_2 + (\mu+\delta_2)\left\{\alpha_2 I_2 - (\mu+\delta_2)A_2\right\}\\
&= \tfrac{(\mu+\delta_2+\alpha_2)(\mu+\delta_2-\varepsilon\omega b)}{(\mu+\delta_2)}\left[\eta\left(R_0^F - 1\right)I_2 + \eta\alpha_2\theta_2 I_2 S_2 + (\mu+\delta_2)\left\{\alpha_2 I_2 - (\mu+\delta_2)A_2\right\}\right]\\
&\le 0
\end{aligned}
$$

where $\eta = \frac{(\mu+\delta_2)}{(d_2+\alpha_2+\mu)(\mu+\delta_2-\varepsilon\omega b)}$, $S_2 = S_2^0$, only $V_2'(t) = 0$, $I_2 = 0$ and $A_2 = 0$.

From the above scenario, using the Lyapunov-LaSalle theorem, E_0 is global asymptotically stable provided that $R_0 \le 1$. The proof is complete.

3.4 Global stability of the endemic equilibrium E^*

In this section, we find the global stability of the endemic equilibrium using the Lyapunov function for each of the populations.

Theorem 2. *If the endemic equilibrium E^* exists, then it is globally asymptotically stable provided the conditions are satisfied using the following:*

$$
\begin{aligned}
&M_1 = \mu(S_1^* + I_1^* + A_1^*) + d_1 I_1^* + \delta_1 A_1^*,\\
&S_1^*\left(\gamma_1 + \tfrac{\theta_1\alpha_1}{\delta_1+\mu}\right) = (d_1+\alpha_1+\mu)\, I_2^*,\\
&\alpha_1 I_1^* = (\delta_1+\mu)\, A_1^*.
\end{aligned}
$$

Proof. Let us consider the following positive definite function for E^*:

$$
\begin{aligned}
L_1 =& \frac{1}{2}[(S_1 - S_1^*) + (I_1 - I_1^*) + (A_1 - A_1^*)]^2 + x_1\left(I_1 - I_1^* - I_1^*\ln\frac{I_1}{I_1^*}\right)\\
&+ \frac{1}{2}x_2\,(\delta_1+\mu)\,(A_1 - A_1^*)^2
\end{aligned} \qquad (16)
$$

where the constants x_1 and x_2 are chosen.

$$\begin{aligned} L_1' = \; & [(S_1 - S_1^*) + (I_1 - I_1^*) + (A_1 - A_1^*)] \, \tfrac{d}{dt} (S_1 + I_1 + A_1) + x_1 \left(\tfrac{I_1 - I_1^*}{I_1} \right) \tfrac{dI_1}{dt} \\ & + x_2 (\delta_1 + \mu) (A_1 - A_1^*) \, \tfrac{dA_1}{dt}. \end{aligned} \tag{17}$$

After substitution, algebraic calculations and summing the quadratics we obtain

$$\begin{aligned} L_1' &= [(S_1 - S_1^*) + (I_1 - I_1^*) + (A_1 - A_1^*)] \{M_1 - \mu (S_1 + I_1 + A_1) - d_1 I_1 - \delta_1 A_1\} \\ &\quad + x_2 \left(\tfrac{I_1 - I_1^*}{I_1} \right) \left\{ S_1 \left(\gamma_1 + \tfrac{\theta_1 \alpha_1}{\delta_1 + \mu} \right) I_1 - (d_1 + \alpha_1 + \mu) I_1 \right\} \\ &\quad + (\delta_1 + \mu) (A_1 - A_1^*) \{\alpha_1 I_1 - (\delta_1 + \mu) A_1\} \\ &= [(S_1 - S_1^*) + (I_1 - I_1^*) + (A_1 - A_1^*)] \{\mu (S_1^* + I_1^* + A_1^*) + d_1 I_1^* + \delta_1 A_1^* - \mu (S_1 + I_1 + A_1) - d_1 I_1 - \delta_1 A_1\} \\ &\quad + x_1 \left(\gamma_1 + \tfrac{\theta_1 \alpha_1}{\delta_1 + \mu} \right) \left(\tfrac{I_1 - I_1^*}{I_1} \right) (S_1 - S_1^*) I_1 - x_2 \, (A_1 - A_1^*) \{(\delta_1 + \mu) A_1^* - (\delta_1 + \mu) A_1\} \\ &= -\mu (S_1 - S_1^*)^2 - (d_1 + \mu) (I_1 - I_1^*)^2 + (\delta_1 + \mu) (A_1 - A_1^*)^2 \\ &\quad + x_1 \left(\gamma_1 + \tfrac{\theta_1 \alpha_1}{\delta_1 + \mu} \right) (S_1 - S_1^*)(I_1 - I_1^*) \qquad + x_2 (\delta_1 + \mu) (A_1 - A_1^*)^2. \end{aligned} \tag{18}$$

There are sufficient conditions for L_1' to be negative. Thus, $L_1' = 0$ only if $S_1 = S_1^*$, $I_1 = I_1^*$ and $A_1 = A_1^*$. Hence, we may conclude that L_1' is a Lyapunov function for (1), and by the Lyapunov asymptotic stability theorem[28] and the LaSalle invariance principle [27], we can conclude that the endemic equilibrium is globally asymptotically stable.

Theorem 3. *If the endemic equilibrium E^* exists, then it is globally asymptotically stable provided the conditions are satisfied using the following:*

$$\begin{aligned} & M_2 = \mu (S_2^* + I_2^* + A_2^*) + d_2 I_2^* + \delta_2 A_2^* - \varphi b S_2^* - \varepsilon b (I_2^* + A_2^*), \\ & S_2^* \left(\gamma_2 + \tfrac{\theta_2 \alpha_2}{\delta_2 + \mu} \right) = (d_2 + \alpha_2 + \mu) I_2^*, \\ & \alpha_2 I_2^* = (\delta_2 + \mu) A_2^*. \end{aligned}$$

Proof. : Let us consider the following positive definite function for E^*:

$$\begin{aligned} L_2 = & \frac{1}{2} [(S_2 - S_2^*) + (I_2 - I_2^*) + (A_2 - A_2^*)]^2 + y_1 \left(I_2 - I_2^* - I_2^* \ln \frac{I_2}{I_2^*} \right) \\ & + \frac{1}{2} y_2 (\delta_1 + \mu) (A_2 - A_2^*)^2 \end{aligned} \tag{19}$$

where the constants y_1 and y_2 are chosen.

$$\begin{aligned} L_2' = & [(S_2 - S_2^*) + (I_2 - I_2^*) + (A_2 - A_2^*)] \frac{d}{dt} (S_2 + I_2 + A_2) + y_1 \left(\frac{I_2 - I_2^*}{I_2} \right) \frac{dI_2}{dt} \\ & + y_2 (A_2 - A_2^*) \frac{dA_2}{dt} \end{aligned} \tag{20}$$

After substitution, algebraic calculations and summing the quadratics we obtain

$$\begin{aligned} L_2' &= [(S_2 - S_2^*) + (I_2 - I_2^*) + (A_2 - A_2^*)] \{M_2 + \varphi b S_2 + \varepsilon b (I_2 + A_2) - \mu (S_2 + I_2 + A_2) - d_1 I_1 - \delta_1 A_1\} \\ &\quad + y_1 \left(\tfrac{I_2 - I_2^*}{I_2} \right) \left\{ \gamma_2 S_2 I_2 + \tfrac{\theta_2 \alpha_2 S_2 I_2}{\delta_2 + \mu} - (d_2 + \alpha_2 + \mu) I_2 \right\} + y_2 (A_2 - A_2^*) \{\alpha_2 I_2 - (\delta_2 + \mu) A_2\} \\ &= [(S_2 - S_2^*) + (I_2 - I_2^*) + (A_2 - A_2^*)] \left\{ \begin{array}{r} \mu (S_2^* + I_2^* + A_2^*) + d_2 I_2^* + \delta_2 A_2^* - \varphi b S_2 - \varepsilon b (I_2 + A_2) \\ + \varphi b S_2 + \varepsilon b (I_2 + A_2) - \mu (S_2 + I_2 + A_2) - d_2 I_2 - \delta_2 A_2 \end{array} \right\} \\ &\quad + \left(\tfrac{I_2 - I_2^*}{I_2} \right) \left\{ \left(\gamma_1 + \tfrac{\theta_2 \alpha_2}{\delta_2 + \mu} \right) (S_2 - S_2^*) I_2 \right\} + \; (A_2 - A_2^*) \{(\delta_2 + \mu) A_2^* - (\delta_2 + \mu) A_2\} \\ &= - (\mu + \varphi b) (S_2 - S_2^*)^2 - (d_2 + \mu + \varepsilon b) (I_2 - I_2^*)^2 + (\delta_1 + \mu + \varepsilon b) (A_1 - A_1^*)^2 \\ &\quad + y_1 \left(\gamma_2 + \tfrac{\theta_2 \alpha_2}{\delta_2 + \mu} \right) (S_2 - S_2^*)(I_2 - I_2^*) + y_2 (\delta_1 + \mu) (A_2 - A_2^*)^2. \end{aligned} \tag{21}$$

There are sufficient conditions for to be negative. Thus, $L_2' = 0$ if and only if $S_2 = S_2^*$, $I_2 = I_2^*$ and $A_2 = A_2^*$. Hence, we may conclude that is a Lyapunov function for (1) for the female population, and by the Lyapunov asymptotic stability theorem[28] and the LaSalle invariance principle[27], we can conclude that the endemic equilibrium is globally asymptotically stable. These entries complete the proof of these theorems.

4 Conclusion and limitation

In this paper, we have demonstrated how to compartment model six differential equations within the male and female populations depicting a good dynamics trend of the spread of HIV and AIDS epidemics with a constant inflow of migration into the susceptible populations. The model is analysed using the threshold parameter to determine all of the various reproduction numbers. The various equilibrium states of are locally asymptotically stable if $R_0 < 1$ and are unstable if $R_0 > 1$. These equilibria are shown to be globally asymptotically stable under certain conditions.

We acknowledge that there are limitations to this study and to the analysis of our results. For example, it is difficult to knowhow many babies are born to females with HIV. Additionally, there are certain assumptions that are based on the mathematical models.Wealso assume that external migration is constant for the male and female susceptible populations,which is another limitation of the model.In future research, we will take into account preventive measures such as effective control strategy programmes. Finally, the proposed model will be tested against real-world data to test its fitness and to evaluate its accuracy. It is our hope that this mathematical model will address these challenges in part by fitting the model to epidemiological data to yield better estimates for the unknown parameters that will inform public health decision-makers.

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