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Mathematical Age-Structured Model for HIV/AIDS Incorporating Two Stages of Infection and Treatment Strategy

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Abstract: In this paper, we introduce a novel age-structured mathematical model for HIV/AIDS, incorporating two distinct stages of infection: individuals who are aware of their infection and those who are unaware. Additionally, we include the dynamics of treated individuals to analyze the impact of treatment in reducing the transmission of HIV/AIDS. To establish the mathematical well-posedness of our age-structured model, we employ semigroup theory to demonstrate the existence and uniqueness of solutions. Furthermore, we derive the equilibrium states and analyze their local stability, as well as compute the basic reproduction number $\mathscr{R}_0$ and its role in determining the stability of these steady states. Finally, in the numerical simulations section, we validate our theoritical findings regarding the stability of the steady states. We also explore the dynamics of the virus in scenarios where one of the infected compartments (aware or unaware individuals) is absent. Additionally, we investigate the impact of treatment on the overall dynamics of HIV/AIDS.

Keywords: Age-strecture epidemic model- Steady state- HIV/AIDS disease-Stability analysis

1 Introduction

The human immunodeficiency virus (HIV) attacks and weakens the immune system, potentially progressing to acquired immunodeficiency syndrome (AIDS), the most severe stage of the infection. HIV spreads through bodily fluids such as blood, breast milk, semen, and vaginal secretions but cannot be transmitted through casual contact, including hugging, kissing, or sharing food. Additionally, the virus can be passed from mother to child during pregnancy, childbirth, or breastfeeding [4,6,7].

Antiretroviral therapy (ART) is the primary treatment and prevention strategy for HIV. If left untreated, HIV can develop into AIDS, often years after the initial infection. Symptoms vary depending on the stage of the disease. In the first few months, when HIV is most contagious, many individuals are unaware of their infection. Some may experience no symptoms, while others develop flu-like signs such as fever, headache, rash, and sore throat.

Certain behaviors and conditions increase the risk of HIV transmission, including unprotected anal or vaginal sex, coexisting sexually transmitted infections (STIs), and drug or alcohol use during sexual activity. Additionally, sharing contaminated needles or undergoing unsafe medical procedures heightens the risk.

Although there is no cure for HIV, ART effectively controls the virus by suppressing its replication within the body. While it does not eliminate HIV, ART strengthens the immune system, reducing susceptibility to opportunistic infections. To remain effective, treatment

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must be taken daily for life. ART significantly lowers the viral load, preventing disease progression and enabling individuals to lead healthy lives. Importantly, people undergoing ART who maintain undetectable viral levels cannot transmit HIV to their sexual partners. Furthermore, HIV-positive pregnant women should initiate ART as early as possible to protect their health and prevent transmission of the virus to their unborn child during pregnancy, childbirth, or breastfeeding. [5,7].

Globally, there were a displayed 39.9 million HIV-positive individuals in 2023, with 1.3 million new infections and 630,000 deaths from AIDS-related causes. Despite these obstacles, 30.7 million patients were receiving antiretroviral therapy, demonstrating the advancements in treatment. The fact that 42.3 million people have died from AIDS-related illnesses and that 88.4 million people have contracted HIV since the epidemic's beginning highlights the disease's continued effect [9].

For HIV/AIDS and other infectious diseases, mathematical models are created using four main modeling approaches: deterministic models, stochastic models, statistical models (such as the state back-calculation method and direct extrapolation), and Space-Kalman filter models [8,6].

The focus of this research is on HIV/AIDS deterministic models. These models make use of HIV transmission dynamics, including the development of AIDS. And usually divide the population into groups such as susceptible individuals, individuals in different stages of infection, and those in the AIDS phase. Transitions between these compartments—through infection, AIDS progression to the next stage, migration, or death—are characterized by differential equations or systems of difference in deterministic transmission models.

In the deterministic models, there are many models provided for studying HIV/AIDS transmission by using ordinary differential equations. In 2013, Hai-Feng and Li-Xiang [10] proposed a $HIV/AIDS$ model with different latent stages and treatments. They analyzed the model and established it using the reproduction number; If the basic reproduction number (R_0) is less than 1, then the disease-free equilibrium point will be globally and asymptotically stable, while if R_0 is greater than 1, then the endemic equilibrium point will be globally asymptotically stable. 2016 Hai-Feng Rui and Xun-Yang [11] studied a novel model treated with susceptible-infected AIDS and recovered $SIATR$ that included a new compartment T of treated individuals. They also determined the stability of the endemic equilibrium using the reproduction number; if the basic reproduction number (R_0) is greater than 1.

In 2023, Nadiah Wan-Arfah, Ling Shing Wong stated that [11] Multivariable analysis is extremely important to statistically adjust the estimated effect of each variable in the model and for more comprehensive statistical modeling. However, the equilibrium point will be globally and asymptotically stable.

A structured population model is a framework for studying population dynamics in which the distribution of persons throughout a range of values is defined as the state variable that reflects the population at any given time. Each individual is associated with one specific value at each moment. In age-structured models, for example, people are classified according to their age, and people who are in the same age group at a certain point in time are referred to as a cohort or age structure.

In age-structured models, compartmental modeling is employed to partition the population into distinct subgroups, each characterized by specific attributes. These models are typically represented using integropartial differential equations. Notable examples include the age-structured measles model [12], where the authors are studied an age-structured epidemic model with vaccination and standard incidence rate for measles disease. The HIV model in [13] is about an age-structured model where they take into consideration three groups, $T(t)$, $V(t)$, and $i(a,t)$, where $T(t)$ and $V(t)$ denote the densities of uninfected target T cells and infectious, also $i(a,t)$ denotes the density of infected T cells of infection age a at time t free virion at time t. The tuberculosis (TB) model [14] was studied by Juan Pablo Aparicio et al. in 2009. Further, the Buruli ulcer model [15] is studied by using age-strecture models.

In this research, we study an age-structured model of HIV population dynamics. First, we divide the total population into five subgroups: the susceptible, the infected individuals who are aware of their infection, the infected individuals who are unaware of their infection, those with AIDS, and those receiving treatment. Then, we formulate a mathematical model that incorporates the age distribution a at time t. Next, we analyze the well-posedness of our integropartial differential system. Furthermore, we derive the explicit form of the steady states and examine their stability in relation to $\mathscr{R}_0$. Finally, we present numerical findings to validate our theoretical results, illustrate the dynamics of HIV/AIDS over time and age, and assess the impact of treated individuals in reducing disease spread and controlling the infection.

The reminder of this paper is structured as follows: In Section 2 we have proposed our age structure model of HIV/AIDS. In Section 3 the mathematical well-posedness is established by using the semigroup theory. The existence of steady states and their stability are given in Section 4. In Section 5 we have illustrated our numerical finding with a discussion to clarify each scenario. Finally we finish by the conclusion in Section 6.

2 Mathematical model

In this part, we disscussing and exploring each step in our model. Then, the first step in modeling the dynamics of HIV-AIDS is to grouping the total population into five important sub-populations. The first sub-population is the

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susceptible individuals, then the infected who know their infection, and the compartment of those who don't know their infection, also, those who develop the infection to be infected with AIDS, finally the individuals who take treatment see figure 1. Before giving the equations

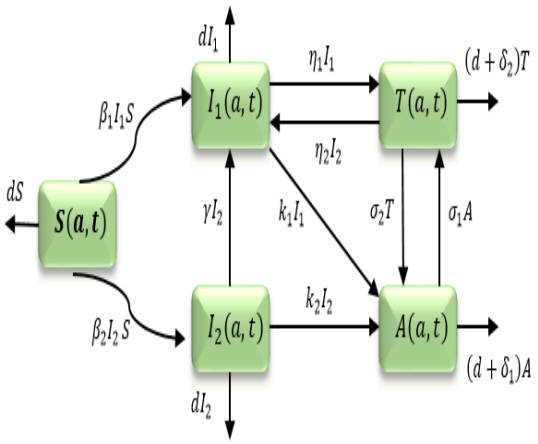

Fig. 1

associated with the model let us set some notations, definitions and assumptions

The distribution of susceptible individuals $S(a,t)$ is the distribution given at time t and age a, are those persons who have the risk of contacting the HIV virus, a contact with an infected individual provides one of the two types of infections, the individuals who aware their infection and those who unware infection.

The distribution of individuals known their infection $I_1(a,t)$ is the distribution given at time t and at age a, are the humans known to their infection. The individuals in this compartment may go to treatment or develop the virus and become infected with AIDS; additionally, those individuals may increase due to stopping using medications against the virus for treated individuals, or the inefficacy of treatment, or due to the infection with other diseases.

The infected who don't know infection $I_2(a,t)$ The distribution at time t and age a represents individuals who are unaware of their infection. These individuals belong to a compartment where their numbers decrease over time as they become aware of their infection status. Additionally, individuals in this compartment may progress to develop the disease and transition to an AIDS-infected state.

The treated individuals $T(a,t)$ is the distribution given at time t and at age a, are the humans who go to treatment.

The infected individuals with AIDS $A(a,t)$ is the distribution given at time t and at age a, are the humans who have infected and develop the infection to later stage with HIV (AIDS).

The total population $N(a,t)$ is the distribution by age a and at time t of the total population. Also, note that the total size of the population at time t given by

$$N(t) = \int_0^\infty N(a,t)da. \tag{1}$$

The parameters that we used it in this model are defined as

$\beta_1(a,b)$: is the average number of new contact per unit of time of one infective that know his infection of age b with a susceptible individual of age a.

$\beta_2(a,b)$: is the average number of new contact per-unit of time of one infective that do not know his infection of age b with a susceptible individual of age a.

$\gamma(a)$: is the rate at which the individuals of age a in I_2 become in the sub-population I_1.

$k_1(a)$: is the rate of infected individual with age a, I_1, become infected with AIDS.

$k_2(a)$: is the rate of infected individual with age a, I_2, become infected with AIDS.

$\sigma_1(a)$: is the rate at which an infected with AIDS with age a go to treatment.

$\sigma_2(a)$: is the inficacity or stopping using treatment for individuals with AIDS of age a.

$\eta_1(a)$: is the rate at which an infected with age a (I_1) go to treatment.

$\eta_2(a)$: The inficacity or stopping using treatment.

$d(a)$: is the natural death rate for individuals with age a.

$\delta_1(a)$: is the additional rate of mortality due to AIDS infection.

$\delta_2(a)$: is the additional rate of mortality due to complications of AIDS or the incompability of treatment with the immune system.

We note that the number of contact at time t with age a of susceptivble individuals with another one of infected of age b is given by $\beta_1(a,b)I_1(b,t)$. The same for infected I_2, the number of comtact with one individual with age b with susceptible individual with age a is given by $\beta_2(a,b)I_2(b,t)$. Therefore, the number of contacts at time t due to one infected I_1 are given by

$$\int_0^\infty \beta_1(a,b)I_1(b,t)db. \tag{2}$$

Also, the number of contacts due to infected one from I_2, are

$$\int_0^\infty \beta_2(a,b)I_2(b,t)db. \tag{3}$$

However, not all individuals sussceptible contact individuals in I_1 or I_2 are infected, thus, we multiply with the susceptible function, then, the loss from age-a susceptible class given by

$$-\int_0^\infty \beta_1(a,b)I_1(b,t)db \times \frac{S(a,t)}{N(a,t)} - \int_0^\infty \beta_2(a,b)I_2(b,t)db \times \frac{S(a,t)}{N(a,t)}. \tag{4}$$

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The loss from age-a susceptible due to natural death is given by $-d(a)S(a,t)$. Also the loss from infected I_1, infected I_2, infected with AIDS A, and the treated T, is given by "$-d(a)I_1(a,t)$", "$-d(a)I_2(a,t)$", "$-d(a)A(a,t)$", and "$-d(a)T(a,t)$" respectively. The additional loss from the class A, and T due to additional death rate is given by "$-\delta_1(a)A(a,t)$", and "$-\delta_2(a)T(a,t)$" respectively.

Assumptions 1 *We assume that,*

($\mathscr{H}_1$) :*$d(.)$, $\gamma(.)$, $k_1(.)$, $k_2(.)$, $\sigma_1(.)$, $\sigma_2(.)$, $\eta_1(.)$, $\eta_2(.)$, $\delta_1(.)$, and $\delta_2(.)$ are in $L^\infty_+(0,\sigma)$, and $\beta_{1,2}(.,.) \in L^\infty_+((0,\sigma)\times(0,\sigma))$, also, all of those functions are extended by zero outside of intervall $(0,\sigma)$, also they are locally integrable*

($\mathscr{H}_2$) :*$(P_1\varphi_2)(.) \in L^\infty_+(0,\sigma)$, and $(P_2\varphi_3)(.) \in L^\infty_+(0,\sigma)$.*

($\mathscr{H}_3$) :*$\beta_1(a,b) = \theta_1(a)\lambda_1(b)$, with $\theta_1(a)$ is the probability of individuals susceptible becoming infected aware infection. Additionally, $\beta_2(a,b) = \theta_2(a)\lambda_2(b)$, with $\theta_2(a)$ is the probability of individuals susceptible becoming infected not aware infection.*

According to above hypothesis, definitions, notations, and the descriptive scheme 1 we have the following system of equations

This system is equipied with boundary and initial conditions as follows

$$S(0,t)=\Lambda,\ I_1(0,t)=0,\ I_2(0,t)=0,\ A(0,t)=0,\ T(0,t)=0,$$

Where, Λ is the newborn. Also the initial conditions are

$$S(a,0)=S_0(a),\ I_1(a,0)=I_{1,0}(a),\ I_2(a,0)=I_{2,0}(a),\ A(a,0)=A_0(a),\ T(a,0)=T_0(a).$$

Let $N(a)$ with $0 \le a \le \sigma$ the density with respect to the age of the total number of individuals. Under assumption $N(a)$ satisfie

$$N(a) = m^* N e^{(-\int_0^a d(\sigma)d\sigma)}, \tag{6}$$

where the constant N is the total size of the population and m^* indicates the crude death rate, is determined such that

$$m^* \int_0^\sigma l(a)da = 1, \tag{7}$$

where,

$$l(a) = e^{\left(-\int_0^\sigma d(a)da\right)},$$

is the survival function, is proportional to the individuals who survive to age a, then

$$N(a) = m^* N l, \tag{8}$$

Let $\Lambda = m^*N$, and consider the functions

$$s(a,t)=\frac{S(a,t)}{N(a,t)},\quad i_1(a,t)=\frac{I_1(a,t)}{N(a,t)},\quad i_2(a,t)=\frac{I_2(a,t)}{N(a,t)},$$
$$x(a,t)=\frac{A(a,t)}{N(a,t)},\quad y(a,t)=\frac{T(a,t)}{N(a,t)}. \tag{9}$$

We noted by

$$\beta_1(a,t) = \theta_1(a)\int_0^\sigma \lambda_1(b)i_1(b,t)db,$$
$$\beta_2(a,t) = \theta_2(a)\int_0^\sigma \lambda_2(b)i_2(b,t)db, \tag{10}$$

Therefore, the system (5) be modified to

Where, $\partial_a = \frac{\partial}{\partial a}$, and $\partial_t = \frac{\partial}{\partial t}$ are the partial derivatives according age a and time t respectively. With the boundary and initial conditions as follows

$$s(0,t)=1,\ i_1(0,t)=0,\ i_2(0,t)=0,\ x(0,t)=0,\ y(0,t)=0,$$

and

$$s(a,0)=\frac{S(a,0)}{N(a,0)}=s_0(a),\quad i_1(a,0)=\frac{I_1(a,0)}{N(a,0)}=i_{1,0}(a)$$
$$,\quad i_2(a,0)=\frac{I_2(a,0)}{N(a,0)}=i_{2,0}(a),$$
$$x(a,0)=\frac{A(a,0)}{N(a,0)}=x_0(a),\quad y(a,0)=\frac{T(a,0)}{N(a,0)}=y_0(a),$$

respectively.

Now, we are going to descussing the mathematical propreities of the proposed model

3 Well-posedness of the model

This section interessed in providing the existence of the solution for the system (11). First, we introduce a new variable $\tilde{s}(a,t) = s(a,t) - 1$, the system (11) can be written as

Under the boundary and initial conditions

$$\tilde{s}(0,t)=0,\ i_1(0,t)=0,\ i_2(0,t)=0,\ x(0,t)=0,\ y(0,t)=0,$$

and

$$\tilde{s}(a,0)=\tilde{s}_0(a),\ i_1(a,0)=i_{1,0}(a)$$
$$,\ i_2(a,0)=i_{2,0}(a),\ x(a,0)=x_0(a),\ y(a,0)=y_0(a),$$

respectively.

We define the Banach space, $\mathscr{X} = L^1(0,\sigma)\times L^1(0,\sigma)\times L^1(0,\sigma)\times L^1(0,\sigma)\times L^1(0,\sigma)$, with L^1- norm which is defined for $\varphi = (\varphi_1,\varphi_2,\varphi_3,\varphi_4,\varphi_5) \in \mathscr{X}$

$$\|\varphi\|_{\mathscr{X}} = \sum_{i=1}^{5} \|\varphi_i\|_{L^1}, \tag{13}$$

where,

$$\|\varphi_i\|_{L^1} = \int_0^\sigma |\varphi_i(a)|da.$$

Additionally, we define the linear operator $\mathscr{A} : \mathscr{D}(\mathscr{A}) \subset \mathscr{X} \to \mathscr{X}$ as

$$A(\varphi)(a) = \left(-\frac{d\varphi_1}{da}, -\frac{d\varphi_2}{da}, -\frac{d\varphi_3}{da}, -\frac{d\varphi_4}{da}, -\frac{d\varphi_5}{da}\right)^t. \tag{14}$$

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$$
\begin{aligned}
\frac{\partial S}{\partial t}(a,t)+\frac{\partial S}{\partial a}(a,t) &= -\int_0^\infty \beta_1(a,b)I_1(b,t)db\times\frac{S(a,t)}{N(a,t)}-\int_0^\infty \beta_2(a,b)I_2(b,t)db\times\frac{S(a,t)}{N(a,t)}\\
&\quad -d(a)S(a,t),\\
\frac{\partial I_1}{\partial t}(a,t)+\frac{\partial I_1}{\partial a}(a,t) &= \int_0^\infty \beta_1(a,b)I_1(b,t)db\times\frac{S(a,t)}{N(a,t)}+\gamma(a)I_2(a,t)+\eta_2(a)T(a,t)\\
&\quad -(k_1(a)+\eta_1(a)+d(a))I_1(a,t),\\
\frac{\partial I_2}{\partial t}(a,t)+\frac{\partial I_2}{\partial a}(a,t) &= \int_0^\infty \beta_2(a,b)I_2(b,t)db\times\frac{S(a,t)}{N(a,t)}-(k_2(a)+\gamma(a)+d(a))I_2(a,t),\\
\frac{\partial A}{\partial t}(a,t)+\frac{\partial A}{\partial a}(a,t) &= k_1(a)I_1(a,t)+k_2(a)I_2(a,t)+\sigma_2(a)T(a,t)\\
&\quad -(\sigma_1(a)+d(a)+\delta_1(a))A(a,t),\\
\frac{\partial T}{\partial t}(a,t)+\frac{\partial T}{\partial a}(a,t) &= \sigma_1(a)A(a,t)+\eta_1(a)I_1(a,t)-(\sigma_2(a)+\eta_2(a)+\delta_2(a)+d(a))T(a,t).
\end{aligned}
\tag{5}
$$

$$
\begin{aligned}
(\partial_a+\partial_t)s(a,t) &= -\beta_1(a,t)s(a,t)-\beta_2(a,t)s(a,t)-d(a)s(a,t),\\
(\partial_a+\partial_t)i_1(a,t) &= \beta_1(a,t)s(a,t)+\gamma(a)i_2(a,t)+\eta_2(a)y(a,t)-(k_1(a)+\eta_1(a)+d(a))i_1(a,t),\\
(\partial_a+\partial_t)i_2(a,t) &= \beta_2(a,t)s(a,t)-(k_2(a)+\gamma(a)+d(a))i_2(a,t),\\
(\partial_a+\partial_t)x(a,t) &= k_1(a)i_1(a,t)+k_2(a)i_2(a,t)+\sigma_2(a)y(a,t)-(\sigma_1(a)+d(a)+\delta_1(a))x(a,t),\\
(\partial_a+\partial_t)y(a,t) &= \sigma_1(a)x(a,t)+\eta_1(a)i_1(a,t)-(\sigma_2(a)+\eta_2(a)+\delta_2(a)+d(a))y(a,t).
\end{aligned}
\tag{11}
$$

$$
\begin{aligned}
(\partial_a+\partial_t)\tilde{s}(a,t) &= -\left(\beta_1(a,t)+\beta_2(a,t)+d(a)\right)\left(\tilde{s}(a,t)+1\right),\\
(\partial_a+\partial_t)i_1(a,t) &= \beta_1(a,t)\left(\tilde{s}(a,t)+1\right)+\gamma(a)i_2(a,t)+\eta_2(a)y(a,t)-(k_1(a)+\eta_1(a)+d(a))i_1(a,t),\\
(\partial_a+\partial_t)i_2(a,t) &= \beta_2(a,t)\left(\tilde{s}(a,t)+1\right)-(k_2(a)+\gamma(a)+d(a))i_2(a,t),\\
(\partial_a+\partial_t)x(a,t) &= k_1(a)i_1(a,t)+k_2(a)i_2(a,t)+\sigma_2(a)y(a,t)-(\sigma_1(a)+d(a)+\delta_1(a))x(a,t),\\
(\partial_a+\partial_t)y(a,t) &= \sigma_1(a)x(a,t)+\eta_1(a)i_1(a,t)-(\sigma_2(a)+\eta_2(a)+\delta_2(a)+d(a))y(a,t).
\end{aligned}
\tag{12}
$$

$$
(\mathscr{F}\varphi)(a)=\begin{bmatrix}
-\left[(P_1\varphi_2)(a)+(P_2\varphi_3)(a)+d(a)\right](\varphi_1(a)+1)\\
(P_1\varphi_2)(a)(\varphi_1(a)+1)+\gamma(a)\varphi_3(a)+\eta_2(a)\varphi_5(a)-(k_1(a)+\eta_1(a)+d(a))\varphi_2\\
(P_2\varphi_3)(a)\left(\varphi_1(a)+1\right)-(k_2(a)+\gamma(a)+d(a))\varphi_3(a)\\
k_1(a)\varphi_2(a)+k_2(a)\varphi_3(a)+\sigma_2(a)\varphi_5(a)-(\sigma(a)+d(a)+\delta_1(a))\varphi_4(a)\\
\sigma_1(a)\varphi_4(a)+\eta_1(a)\varphi_2(a)-(\sigma_2(a)+\eta_2(a)+\delta_2(a)+d(a))\varphi_5(a)
\end{bmatrix},
\tag{15}
$$

where, $\mathscr{D}(\mathscr{A})$ is the domain of $\mathscr{A}$

$$
\mathscr{D}(\mathscr{A})=\left\{\varphi\in\mathscr{X},\ \ \varphi_i\in W^{1,1}(0,\sigma),\ \ \varphi(0)=(0,0,0,0,0)^t\right\}.
$$

Furthermore, we define the non-linear operator $\mathscr{F}:\mathscr{X}\to\mathscr{X}$ as where,

Let $u(t)=(\tilde{s}(.,t),i_1(.,t),i_2(.,t),x(.,t),y(.,t))$, then we can rewrite the system (12) as an abstract semilinear problem

$$
\frac{du}{dt}=\mathscr{A}(u(t))+\mathscr{F}(u(t)),\ \ u\in\mathscr{D}(\mathscr{A}).
\tag{16}
$$

with the initial conditions $u(0)=(\tilde{s}_0,i_{1,0},i_{2,0},x_0,y_0)\in\mathscr{X}$.

To demonstrate the existence and uniqueness of the system, it's necessary the following achievements

Lemma 1

(i)The linear operator $\mathscr{A}$ is the infinitesimal generator of c_0-semigroup $\mathscr{T}=\{e^{t\mathscr{A}}\}$.
(ii)The non-linear operator $\mathscr{F}$ is locally Lipshitz.

Proof.

(i)By applying (Hille-Yosida) [1], $\mathscr{A}$ is a linear operator, $\mathscr{A}$ is the infinitesimal generator of c_0-semigroup $\mathscr{T}(t)$ if and only if,
(a)$\overline{\mathscr{D}(\mathscr{A})}=\mathscr{X}$,
(b)$(\lambda I-\mathscr{A})^{-1}$ is bounded from $\mathscr{X}$ into itself
(c)The resolvent set $\rho(\mathscr{A})=\{\lambda\in\mathbf{C}:\ \ \lambda I-\mathscr{A}:\mathscr{D}(\mathscr{A})\to\mathscr{X}$ is bijective$\}$ [2] contain $\mathbf{R}^+$, and for

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$$\beta_1(.) := \int_0^\sigma \beta_1(.,b)\varphi_2(b)db = \theta_1(.)\int_0^\sigma \lambda_1(b)\varphi_2(b)db := (P_1\varphi_2)(.) \in L^\infty(0,\sigma),$$
$$\beta_2(.) := \int_0^\sigma \beta_2(.,b)\varphi_3(b)db = \theta_2(.)\int_0^\sigma \lambda_2(b)\varphi_3(b)db := (P_2\varphi_3)(.) \in L^\infty(0,\sigma).$$

every $\lambda > 0$

$$\|(\lambda I - \mathscr{A})^{-n}\| \leq \frac{1}{\lambda^n},\ n \geq 1.$$

Then it is clear that $\overline{\mathscr{D}(\mathscr{A})} = \mathscr{X}$.
For the second and third properties, we consider the following abstract Cauchy problem,

$$\begin{cases} \frac{du}{dt} = \mathscr{A}u(t),\ t \geq 0, \\ u(0) = u_0 \in \mathscr{X}, \end{cases} \tag{17}$$

and the linear operator $\mathscr{A}$ is given in the above equation (14).
To demonstrate that $\lambda I - \mathscr{A}$ is bijective function, it suffices that $(\lambda I - \mathscr{A})\varphi(a) = \Psi(a) \Leftrightarrow \varphi(a) = (\lambda I - \mathscr{A})^{-1}\Psi(a)$, with the determination of $(\lambda I - \mathscr{A})^{-1}(.)$. Therefore, for each $\lambda > 0$ and all $a \in (0,\sigma)$, we have:

$$\begin{aligned} &(\lambda I - \mathscr{A})\varphi(a) = \Psi(a) \Leftrightarrow \lambda\varphi(a) + \varphi'(a) = \Psi(a), \\ &\Leftrightarrow \varphi(a) = \int_0^\sigma e^{-\lambda(s-a)}\Psi(s)ds, \\ &\Leftrightarrow (\lambda I - \mathscr{A})^{-1}\Psi(a) = \int_0^\sigma e^{-\lambda(a-s)}\Psi(s)ds, \end{aligned} \tag{18}$$

which means that $(\lambda I - \mathscr{A})$ is a bijection from $\mathscr{D}(\mathscr{A})$ to $\mathscr{X}$, and

$$\begin{aligned} &\|(\lambda I - \mathscr{A})^{-1}\Psi\|_{\mathscr{X}} \leq \sum_{i=1}^{5}\int_0^\sigma \left|\int_0^a e^{-\lambda(a-s)}\Psi_i(s)ds\right| da, \\ &\Leftrightarrow \|(\lambda I - \mathscr{A})^{-1}\| \leq \int_0^\sigma e^{-\lambda(a-s)}da \leq \frac{1}{\lambda}, \\ &\Leftrightarrow \|(\lambda I - \mathscr{A})^{-n}\| \leq \frac{1}{\lambda^n}, \end{aligned} \tag{19}$$

By Hille-Yosida Theorem, $\mathscr{A}$ is the infinitesimal generator of a c_0-semigroup $\mathscr{T}(t) = \{e^{t\mathscr{A}}\}_{t\geq 0}$.

(*ii*) The nonlinear operator $\mathscr{F}$ of system (16) is Lipshitz continuous in $\mathscr{X}$, *i.e.*, for φ, and $\Psi \in \mathscr{X}$ then there exists a constant $L \in \mathbf{R}$ such that

$$\|(\mathscr{F}\varphi) - (\mathscr{F}\Psi)\| \leq L\|\varphi - \Psi\|. \tag{20}$$

Therefore, for every $u_0 \in \mathscr{X}$ there exists a maximal interval of existence (0,T) and a unique mild solution $t \mapsto u(t,u_0)$ such that,

$$u(t,u_0) = \mathscr{T}(t)u_0 + \int_0^t \mathscr{T}(t-s)\mathscr{F}(u(s,u_0))ds,\ t \in (0,T), \tag{21}$$

also for $t = \infty$ or $lim_{t\to T}\|u(t,u_0)\| = \infty$. Furthermore, if $u_0 \in \mathscr{D}(\mathscr{A})$, then for $t \in (0,T)$, $u(t,u_0) \in \mathscr{D}(\mathscr{A})$ and the function $t \mapsto u(t,u_0)$ is continuously differentiable and satisfies (16) on (0,T) [3].

Currently, allow us to note by

$$\Omega := \{(\tilde{s},i_1,i_2,x,y) \in \mathscr{X},\ \tilde{s} \geq -1, i_1 \geq 0, i_2 \geq 0, x \geq 0, y \geq 0\},$$

and

$$\Omega_0 := \{(\tilde{s},i_1,i_2,x,y) \in \mathscr{X},\ -1 \leq \tilde{s} + i_1 + i_2 + x + y \leq 1\},$$

state space or admissible region , and the feasible subregion respectively.

Lemma 2 *The mild solution $u(t,u_0)$, $u_0 \in \Omega$ of (12) enter into Ω_0 after finite time and the set Ω_0 is positively invariant.*

Proof. According to the system (12), we have following presentation

$$s(a,t) = \begin{cases} e^{-\int_0^a (\beta_1(\gamma,t-a+\gamma)+\beta_2(\gamma,t-a+\gamma)+d(\gamma))d\gamma}, & (t-a) > 0, \\ s_0(a-t)e^{-\int_0^t (\beta_1(a-t+\gamma,\gamma)+\beta_2(a-t+\gamma,\gamma))d\gamma}, & a-t > 0, \end{cases} \tag{22}$$

Since we know that the exponential function is always positive, we also know that $s_0(a) \geq 0$, which means that $s(a,t) \geq 0$. Consequently, $\tilde{s}(a,t) \geq -1$.
For the second equation in (12) is now rewritten as an abstract Cauchy problem

$$\frac{d}{dt}i_1(t) = (P_1 i_1(t))(\tilde{s}(t)+1) + \gamma i_2(t) + \eta_2 y(t) + \mathscr{B}_1 i_1(t), \tag{23}$$

where, the operator $\mathscr{B}_1$ is defined by $\mathscr{B}_1 = -\frac{d}{da} - (k_1 + \eta_1 + d)$, and the domaine of this operatos is defined as $\mathscr{D}(\mathscr{B}_1) = \{\psi \in L^1(0,\sigma) \mid \psi(0) = 0\}$. Therefore, the solutio of this last equation is given by

$$i_1(t) = \mathscr{T}_1(t)i_1(0) + \int_0^T \mathscr{T}_1(t-s)[P_1 i_1(s)(\tilde{s}(s)+1) + \gamma i_2(s) + \eta_2 y(s)]ds \tag{24}$$

with $\mathscr{T}_1(t) := e^{t\mathscr{B}_1}$, is a c_0-semigroup generated by the operator $\mathscr{B}_1$. Therefore, we have $\tilde{s} \geq -1$, and $i_{1,0} \geq 0$, also the operator $\mathscr{T}_1$, is a positive semigroup, then we show that $i_1(t)$ is a positive function.
As the same for the third, fourth, and fifth equations we can express them as

$$\begin{aligned} \frac{d}{dt}i_2(t) &= (P_2 i_2(t))(\tilde{s}(t)+1) - \mathscr{B}_2 i_2(t), \\ \frac{d}{dt}x(t) &= k_1 i_1(t) + \sigma_2 y(t) + \mathscr{B}_3 x(t), \\ \frac{d}{dt}y(t) &= \sigma_1 x(t) + \eta_1 i_1(t) + \mathscr{B}_4 y(t). \end{aligned} \tag{25}$$

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Where the operators $\mathscr{B}_2, \mathscr{B}_3$, and $\mathscr{B}_4$ and their domaines are defined as

$$\mathscr{B}_2 = -\frac{d}{da} - (k_1+\gamma+d),\ \ \mathscr{D}(\mathscr{B}_2) = \{\psi \in L^1(0,\sigma), \psi(0) = 0\},$$
$$\mathscr{B}_3 = -\frac{d}{da} - (\sigma_1+\delta_1+d),\ \ \mathscr{D}(\mathscr{B}_3) = \{\psi \in L^1(0,\sigma), \psi(0) = 0\},$$
$$\mathscr{B}_4 = -\frac{d}{da} - (\sigma_2+\eta_2+\delta_2+d),\ \ \mathscr{D}(\mathscr{B}_4) = \{\psi \in L^1(0,\sigma), \psi(0) = 0\}.$$

The solutions given by equation (25) are

$$\begin{aligned} i_2(t) &= \mathscr{T}_2(t)i_2(0) + \int_0^T \mathscr{T}_2(t-s)[P_2 i_2(s)(\tilde{s}+1)]ds, \\ x(t) &= \mathscr{T}_3(t)x(0) + \int_0^T \mathscr{T}_3(t-s)[k_1 i_1(s) + k_2 y(s)]ds, \\ y(t) &= \mathscr{T}_4(t)y(0) + \int_0^T \mathscr{T}_4(t-s)[\sigma_1 x(s) + \eta_1 i_1(s)]ds, \end{aligned} \tag{26}$$

with, $\mathscr{T}_2 := e^{t\mathscr{B}_2}$, $\mathscr{T}_3 := e^{t\mathscr{B}_3}$, and $\mathscr{T}_4 := e^{t\mathscr{B}_4}$ are the c_0-semigroups generated by the operators $\mathscr{B}_2$, $\mathscr{B}_3$, and $\mathscr{B}_4$ respectively. As we observe, $i_2(0)$, $x(0)$, and $y(0)$ are positives also from (26), (23), and(22) $i_1(t) \geq 0$ and $\tilde{s}(t)+1 \geq 0$, for $t \geq 0$, then $i_2(t)$, $x(t)$, and $y(t)$ are positives. Hence, we know that $u(t,u_0) \in \Omega$ for all $u_0 \in \Omega$.
Next we note $\omega = \tilde{s}+i_1+i_2+x+y$, then we have

$$(\partial_t + \partial_a)\omega(a,t) = -d(a)\omega(a,t) - \delta_1(a)x(a,t) - \delta_2(a)y(a,t),$$

this last equation can be written as an abstract semilinear Cauchy problem

$$\frac{d}{dt}\omega(t) = -\delta_1 x(t) - \delta_2 y(t) + \mathscr{B}_5\omega,$$

where $\mathscr{B}_5 = -\frac{d}{da} - d$, and $\omega(0) = \tilde{s}(0) + i_1(0) + i_2(0) + x(0) + y(0) = 0$ also, the domain of this linear operator is defined as $\mathscr{D}(\mathscr{B}_5) = \{\psi \in L^1(0,\sigma), \psi(0) = 0\}$. Therefore, we deduce the solution as follows,

where $\mathscr{T}_5 := e^{(t\mathscr{B}_5)}$ is the positive c_0-semigroup generated by the operator $\mathscr{B}_5$.
Thus, we have two results,
Case 1; if $u_0 \in \Omega_0$ it is clear that the mild solution $u(t,u_0) \in \Omega_0$ for all $t \geq 0$.
Case 2; if $u_0 \in \Omega$, Then the mild solution enters into Ω_0 for all $a > t$.

By the Lemma 2, we have the following results:

Theorem 1.*The abstract Cauchy problem (16) has a unique global classical solution on $\mathscr{X}$ for the initial conditions in $u_0 \in \Omega \cap \mathscr{D}(\mathscr{A})$.*

4 Existence of steady states and their stability

4.1 Existence of disease-free steady states

Let us represent the disease-free steady states for the set of equations (11) by $\mathscr{E}^0 = \left(s^0(a), i_1^0(a), i_2^0(a), x^0(a), y^0(a)\right)$.

Since we assume that there is no sickness in this steady state scenario, $i_1^0(a), i_2^0(a)$, and $x^0(a)$ are all equal to zero. Therefore, we obtain the following

$$\begin{cases} \frac{d}{da}s^0(a) = -d(a)s^0(a), \\ y^0(a) = 0,\ i_1^0(a) = 0,\ i_2^0(a) = 0, \text{ and } x^0(a) = 0, \\ s^0(0) = 1. \end{cases} \tag{28}$$

The solution of this system is given by

$$s^0(a) = e^{(-\int_0^a d(\tau)d\tau)}.$$

Then, the disease-free steady state existe and given by $\mathscr{E}^0 = \left(s^0(a), 0, 0, 0, 0\right)$.

4.2 Local stability of the disease-free steady state

To demonstrate the local stability of the disease-free steady state, we need to calculate the linearized system of our model at this steady state.
Let us first make the following translations $\hat{s} = s(a,t) - s^0(a)$, $\hat{i}_1 = i_1(a,t) - i_1^0(a)$, $\hat{i}_2 = i_2(a,t) - i_2^0(a)$, $\hat{x} = x(a,t) - x^0(a)$, and $\hat{y} = y(a,t) - y^0(a)$. Therefore, the system is transformed to

The linearized part of the above system is written as follows.

where,

$$\begin{aligned} \hat{\beta}_1(a,t) &= \theta_1(a)\int_0^\sigma \lambda_1(b)\hat{i}_1(b,t)db, \\ \hat{\beta}_2(a,t) &= \theta_2(a)\int_0^\sigma \lambda_2(b)\hat{i}_2(b,t)db, \end{aligned} \tag{31}$$

Currently, let's consider the non-zero exponential solution of the system (30), $\hat{s}(a,t) = \hat{s}(a)e^{\lambda t}$, $\hat{i}_1(a,t) = \hat{i}_1(a)e^{\lambda t}$, $\hat{i}_2(a,t) = \hat{i}_2(a)e^{\lambda t}$, $\hat{x}(a,t) = \hat{x}(a)e^{\lambda t}$, and $\hat{y}(a,t) = \hat{y}(a)e^{\lambda t}$, then the last system become

$$\begin{aligned} \hat{\beta}_1(a) &= \theta_1(a)\int_0^\sigma \lambda_1(b)\hat{i}_1(b)db, \\ \hat{\beta}_2(a) &= \theta_2(a)\int_0^\sigma \lambda_2(b)\hat{i}_2(b)db. \end{aligned} \tag{33}$$

Define, Λ_1, and Λ_2 as

$$\begin{aligned} \Lambda_1 &= \int_0^\sigma \lambda_1(b)\hat{i}_1(b)db, \\ \Lambda_2 &= \int_0^\sigma \lambda_2(b)\hat{i}_2(b)db. \end{aligned} \tag{34}$$

Then, $\hat{\beta}_1 = \Lambda_1\theta_1(a)$, and $\hat{\beta}_2 = \Lambda_2\theta_2(a)$, and the solution of the system (32) is given by

Now, we shall to calculate the basic reproduction number, which gives a direct relation between the equilibrium and its stability. This rate is the number of

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$$\omega(t) = \mathscr{T}_5(t)\omega(0) - \int_0^T \mathscr{T}_5(t-s)[\delta_1 x(s) + \delta_2 y(s)]ds \leq \mathscr{T}_5(t)\omega(0) \leq e^{\left(-t\left(\frac{d}{da}\omega(0)+d\omega(0)\right)\right)} \leq 1, \tag{27}$$

$$\begin{aligned}
(\partial_a+\partial_t)\hat{s}(a,t) &= -\hat{\beta}_1(a,t)\hat{s}(a,t) - \hat{\beta}_2(a,t)\hat{s}(a,t) - \hat{\beta}_1(a,t)s^0(a) - \hat{\beta}_2(a,t)s^0(a) - d(a)\hat{s}(a,t),\\
(\partial_a+\partial_t)\hat{i}_1(a,t) &= \hat{\beta}_1(a,t)\hat{s}(a,t) + \hat{\beta}_1(a,t)s^0(a) + \gamma(a)\hat{i}_2(a,t) + \eta_2(a)\hat{y}(a,t) - (k_1(a)+\eta_1(a)+d(a))\hat{i}_1(a,t),\\
(\partial_a+\partial_t)\hat{i}_2(a,t) &= \hat{\beta}_2(a,t)\hat{s}(a,t) + \hat{\beta}_2(a,t)s^0(a) - (k_2(a)+\gamma(a)+d(a))\hat{i}_2(a,t),\\
(\partial_a+\partial_t)\hat{x}(a,t) &= k_1(a)\hat{i}_1(a,t) + k_2(a)\hat{i}_2(a,t) + \sigma_2(a)\hat{y}(a,t) - (\sigma_1(a)+d(a)+\delta_1(a))\hat{x}(a,t),\\
(\partial_a+\partial_t)\hat{y}(a,t) &= \sigma_1(a)\hat{x}(a,t) + \eta_1(a)\hat{i}_1(a,t) - (\sigma_2(a)+\eta_2(a)+\delta_2(a)+d(a))\hat{y}(a,t).
\end{aligned} \tag{29}$$

$$\begin{aligned}
(\partial_a+\partial_t)\hat{s}(a,t) &= -\hat{\beta}_1(a,t)s^0(a) - \hat{\beta}_2(a,t)s^0(a) - d(a)\hat{s}(a,t),\\
(\partial_a+\partial_t)\hat{i}_1(a,t) &= \hat{\beta}_1(a,t)s^0(a) + \gamma(a)\hat{i}_2(a,t) + \eta_2(a)\hat{y}(a,t) - (k_1(a)+\eta_1(a)+d(a))\hat{i}_1(a,t),\\
(\partial_a+\partial_t)\hat{i}_2(a,t) &= \hat{\beta}_2(a,t)s^0(a) - (k_2(a)+\gamma(a)+d(a))\hat{i}_2(a,t),\\
(\partial_a+\partial_t)\hat{x}(a,t) &= k_1(a)\hat{i}_1(a,t) + k_2(a)\hat{i}_2(a,t) + \sigma_2(a)\hat{y}(a,t) - (\sigma_1(a)+d(a)+\delta_1(a))\hat{x}(a,t),\\
(\partial_a+\partial_t)y(a,t) &= \sigma_1(a)\hat{x}(a,t) + \eta_1(a)\hat{i}_1(a,t) - (\sigma_2(a)+\eta_2(a)+\delta_2(a)+d(a))\hat{y}(a,t).
\end{aligned} \tag{30}$$

$$\begin{aligned}
\frac{d}{da}\hat{s}(a) + \lambda\hat{s}(a) &= -\hat{\beta}_1(a)s^0(a) - \hat{\beta}_2(a)s^0(a) - d(a)\hat{s}(a),\\
\frac{d}{da}\hat{i}_1(a) + \lambda\hat{i}_1(a) &= \hat{\beta}_1(a)s^0(a) + \gamma(a)\hat{i}_2(a) + \eta_2(a)\hat{y}(a) - (k_1(a)+\eta_1(a)+d(a))\hat{i}_1(a),\\
\frac{d}{da}\hat{i}_2(a) + \lambda\hat{i}_2(a) &= \hat{\beta}_2(a)s^0(a) - (k_2(a)+\gamma(a)+d(a))\hat{i}_2(a),\\
\frac{d}{da}\hat{x}(a) + \lambda\hat{x}(a) &= k_1(a)\hat{i}_1(a) + k_2(a)\hat{i}_2(a) + \sigma_2(a)\hat{y}(a) - (\sigma_1(a)+d(a)+\delta_1(a))\hat{x}(a),\\
\frac{d}{da}\hat{y}(a) + \lambda\hat{y}(a) &= \sigma_1(a)\hat{x}(a) + \eta_1(a)\hat{i}_1(a) - (\sigma_2(a)+\eta_2(a)+\delta_2(a)+d(a))\hat{y}(a).
\end{aligned} \tag{32}$$

$$\begin{aligned}
\hat{s}(a) &= -\Lambda_1\int_0^a \theta_1(\sigma)s^0(\sigma)e^{\{-\int_\sigma^a d(\xi)+\lambda d\xi\}}d\sigma - \Lambda_2\int_0^a \theta_2(\sigma)s^0(\sigma)e^{\{-\int_\sigma^a d(\xi)+\lambda d\xi\}}d\sigma,\\
\hat{i}_1(a) &= \int_0^a \left[\Lambda_1\theta_1(\sigma)s^0(\sigma) + \gamma(\sigma)\hat{i}_2(\sigma) + \eta_2(\sigma)\hat{y}(\sigma)\right]e^{\{-\int_\sigma^a \lambda+k_1(\xi)+\eta_1(\xi)+d(\xi)d\xi\}}d\sigma\\
\hat{i}_2(a) &= \Lambda_2\int_0^a \theta_2(\sigma)s^0(\sigma)e^{\{-\int_\sigma^a \lambda+k_2(\xi)+\gamma(\xi)+d(\xi)d\xi\}}d\sigma,\\
\hat{x}(a) &= \int_0^a \left[k_1(\sigma)\hat{i}_1(\sigma) + k_2(\sigma)\hat{i}_2(\sigma)\right]e^{\{-\int_\sigma^a \lambda+\sigma_1(\xi)+d(\xi)+\delta_1(\xi)d\xi\}}d\sigma,\\
\hat{y}(a) &= \int_0^a \left[\sigma_1(\tau)\hat{x}(\tau) + \eta_1(\tau)\hat{i}_1(\tau)\right]e^{\{-\int_\sigma^a \lambda+\sigma_2(\xi)+\eta_2(\xi)+\delta_2(\xi)+d(\xi)d\xi\}}d\tau.
\end{aligned} \tag{35}$$

infections caused by one infected individual in a population totally susceptible. Then this last is expressed without taking treatment strategy into consideration, then for this purpose we consider, $\eta_1(a) = \eta_2(a) = \sigma_1(a) = \sigma_2(a) = 0$. Then, the solutions $\hat{s}(a), \hat{i}_1(a), \hat{i}_2(a)$, and $\hat{x}(a)$ are expressed in this case as follows

From the third equation of system (36) and the second equation in (34), we have,

$$\Lambda_2 = \Lambda_2\int_0^\sigma \lambda_2(b)\Phi_2(a,\lambda)db. \tag{37}$$

Further, from the second equation in (36) and the first in (34) we have

$$\Lambda_1 = \Lambda_1\int_0^\sigma \lambda_1(b)\Phi_1(b,\lambda)db + \Lambda_2\int_0^\sigma \lambda_1(b)\Psi(b,\lambda)db, \tag{38}$$

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$$\begin{aligned}
\hat{s}(a) &= -\Lambda_1 \int_0^a \theta_1(\sigma)s^0(\sigma)e^{\{-\int_\sigma^a d(\xi)+\lambda d\xi\}}d\sigma - \Lambda_2 \int_0^a \theta_2(\sigma)s^0(\sigma)e^{\{-\int_\sigma^a d(\xi)+\lambda d\xi\}}d\sigma,\\
\hat{i}_1(a) &= \int_0^a \left[\Lambda_1\theta_1(\sigma)s^0(\sigma)+\gamma(\sigma)\hat{i}_2(\sigma)\right]e^{\{-\int_\sigma^a \lambda+k_1(\xi)+d(\xi)d\xi\}}d\sigma\\
\hat{i}_2(a) &= \Lambda_2 \int_0^a \theta_2(\sigma)s^0(\sigma)e^{\{-\int_\sigma^a \lambda+k_2(\xi)+\gamma(\xi)+d(\xi)d\xi\}}d\sigma,\\
\hat{x}(a) &= \int_0^a \left[k_1(\sigma)\hat{i}_1(\sigma)+k_2(\sigma)\hat{i}_2(\sigma)\right]e^{\{-\int_\sigma^a \lambda+d(\xi)+\delta_1(\xi)d\xi\}}d\sigma.
\end{aligned} \tag{36}$$

where,

$$\begin{aligned}
\Phi_1(b,\lambda) &= \int_0^b \theta_1(\sigma)s^0(\sigma)e^{\{-\int_\sigma^b \lambda+k_1(\xi)+d(\xi)d\xi\}}d\sigma,\\
\Phi_2(b,\lambda) &= \int_0^b \theta_2(\sigma)s^0(\sigma)e^{\{-\int_\sigma^b \lambda+k_2(\xi)+\gamma(\xi)+d(\xi)d\xi\}}d\sigma,\\
\Psi(b,\lambda) &= \int_0^b \gamma(\sigma)\Phi_2(\sigma,\lambda)e^{\{-\int_\sigma^b \lambda+k_1(\xi)+d(\xi)d\xi\}}d\sigma.
\end{aligned} \tag{39}$$

From (38) we have

$$\Lambda_2 = \Lambda_1 \frac{\left(1-\int_0^\sigma \lambda_1(b)\Phi_1(b,\lambda)db\right)}{\int_0^\sigma \lambda_1(b)\Psi(b,\lambda)db}, \tag{40}$$

Therefore, the basic reproduction number, $\mathscr{R}_0$, is defined as $\mathscr{R}_0 = F(0)$. This represents the total number of infections caused by both infected individuals who are aware of their infection and those who are unaware of it. The variables and expressions describe various probabilities and parameters in the model: $\theta_1(\sigma)$ represents the probability of infection, while $\gamma(\sigma)$ denotes the probability of transmission to infected individuals who are aware of their infection. The rate $s^0(\sigma)$ indicates the initial number of susceptible individuals. The term $e^{\{-\int_\sigma^a k_1(\xi)d\xi\}}$ expresses the probability of becoming infected with AIDS. Similarly, $e^{\{-\int_\sigma^a d(\xi)d\xi\}}$ represents the probability of death. Furthermore, $\theta_2(\sigma)$ is the probability of infection from individuals in the i_2 compartment, $e^{\{-\int_\sigma^a k_2(\xi)d\xi\}}$ signifies the probability of being in the AIDS compartment, and $e^{\{-\int_\sigma^a \gamma(\xi)d\xi\}}$ represents the transition probability to the i_1 compartment.

Theorem 2.*The disease-free steady state, $\mathscr{E}^0$, is locally asymptotically stable if $\mathscr{R}_0 < 1$, indicating that the disease will eventually die out. Conversely, it becomes unstable if $\mathscr{R}_0 > 1$, suggesting that the disease will persist and potentially spread within the population.*

replacing this last into equation (37), we obtain

$$1 = \int_0^\sigma \lambda_1(b)\Phi_1(b,\lambda)db + \left(1-\int_0^\sigma \lambda_1(b)\Phi_1(b,\lambda)db\right)\times\int_0^\sigma \lambda_2(b)\Phi_2(b,\lambda)db. \tag{41}$$

Let us noted by F the right hand side of equation (41),

$$F(\lambda) = \int_0^\sigma \lambda_1(b)\Phi_1(b,\lambda)db + \left(1-\int_0^\sigma \lambda_1(b)\Phi_1(b,\lambda)db\right)\times\int_0^\sigma \lambda_2(b)\Phi_2(b,\lambda)db. \tag{42}$$

*Proof.*By the definition of $F(\lambda)$, we have,

$$\lim_{\lambda\to+\infty} F(\lambda) = 0, \tag{43}$$

and the limite at $-\infty$ is given as

$$\lim_{\lambda\to-\infty} F(\lambda) = +\infty, \tag{44}$$

furthermore,

If $\int_0^\sigma \lambda_1(b)\Phi_1(b,\lambda)db \leq 1$, and $\int_0^\sigma \lambda_2(b)\Phi_2(b,\lambda)d \leq 1$, then $F'(\lambda) < 0$.

Therefore, this function F is a decreasing function with the limite at $+\infty$ is zero and at $-\infty$ is the $+\infty$ then the courb of this function is cross the axe of ordonnee at a positif point see the figure 2

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$$F'(\lambda)=\frac{\partial}{\partial\lambda}\left(\int_0^{\sigma}\lambda_1(b)\Phi_1(b,\lambda)db+\left(1-\int_0^{\sigma}\lambda_1(b)\Phi_1(b,\lambda)db\right)\times\int_0^{\sigma}\lambda_2(b)\Phi_2(b,\lambda)db\right). \tag{45}$$

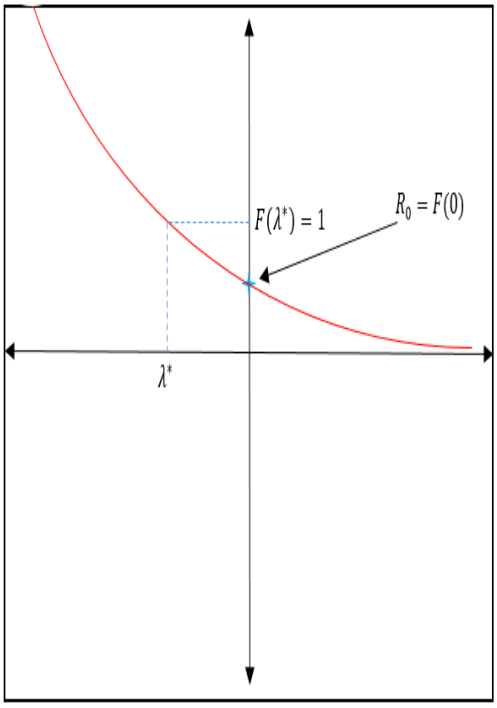

Fig. 2: Courbe representatif of the function F.

Then, there exists a unique root λ^* solution of $F(\lambda^*)=1$. And we have the two following cases,

- **–case 1:** It is clear that if we have $R_0<1$ the solution $\lambda^*\leq 0$, see figure 2 .
- **–case 2:** if $F(\lambda)=1$ has a complex roots, and let $\lambda=a+ib$ a root satisfying $F(\lambda)=1$, Then $Re(F(\lambda))=1$ and $Im(F(\lambda))=0$. Also,

$$Re(e^{(\lambda)})=Re(e^{(ai+b)})=e^{(a)}cos(b)\leq exp(a)=e^{Re((\lambda))}$$

Then,

$$1=Re(F(\lambda))\leq F(Re(\lambda))\Leftrightarrow F(\lambda^*)\leq F(Re(\lambda))\Leftrightarrow Re(\lambda)\leq\lambda^*<0\ (\mathscr{R}_0<F(\lambda^*)),$$

.

Therefore, the disease-free steady state is stable if and only if $\mathscr{R}_0<1$, Then we complete the proof of the theorem.

4.3 Existence of endemic steady state

The endemic steaday state satisfy the following system with the initial conditions

$$s^*(0)=1,\ i_1^*(0)=0,\ i_2^*(0)=0,\ x^*(0)=0,\ y^*(0)=0.$$

Where,

$$\begin{aligned}\beta_1^*(a)&=\theta_1(a)\int_0^{\sigma}\lambda_1(b)i_1^*(b)db\\ \beta_2^*(a)&=\theta_2(a)\int_0^{\sigma}\lambda_2(b)i_2^*(b)db\end{aligned} \tag{47}$$

Let, as denote by

$$\begin{aligned}\Lambda_1^*&=\int_0^{\sigma}\lambda_1(b)i_1^*(b)db\Rightarrow\beta_1^*(a)=\theta_1(a)\Lambda_1^*,\\ \Lambda_2^*&=\int_0^{\sigma}\lambda_2(b)i_2^*(b)db\Rightarrow\beta_2^*(a)=\theta_2(a)\Lambda_2^*,\end{aligned} \tag{48}$$

the solution of each differential equation in system (46) are given by

Therefore, simillarly to equations (37), and (38) we have

$$\begin{aligned}\Lambda_1^*&=\Lambda_1^*\int_0^{\sigma}\lambda_1(b)\psi_1(b)db+\Lambda_2\int_0^{\sigma}\lambda_1(b)\psi_3(b)db,\\ \Lambda_2&=\Lambda_2\int_0^{\sigma}\lambda_2(b)\psi_2(a)db,\end{aligned} \tag{50}$$

where,

$$\begin{aligned}\psi_1(b)&=\int_0^{b}\theta_1(\sigma)s^*(\sigma)e^{\{-\int_{\sigma}^{b}+k_1(\xi)+d(\xi)d\xi\}}d\sigma,\\ \psi_2(b)&=\int_0^{b}\theta_2(\sigma)s^*(\sigma)e^{\{-\int_{\sigma}^{b}k_2(\xi)+\gamma(\xi)+d(\xi)d\xi\}}d\sigma,\\ \psi_3(b)&=\int_0^{b}\gamma(\sigma)\psi_2(\sigma)e^{\{-\int_{\sigma}^{b}k_1(\xi)+d(\xi)d\xi\}}d\sigma.\end{aligned} \tag{51}$$

From (51)

$$\Lambda_2^*=\Lambda_1^*\frac{\left(1-\int_0^{\sigma}\lambda_1(b)\psi_1(b)db\right)}{\int_0^{\sigma}\lambda_1(b)\psi_3(b)db}, \tag{52}$$

replacing this last into equation (37), we obtain

$$1=\int_0^{\sigma}\lambda_1(b)\psi_1(b)db+\left(1-\int_0^{\sigma}\lambda_1(b)\psi_1(b)db\right)\times\int_0^{\sigma}\lambda_2(b)\psi_2(b)db \tag{53}$$

Let us define the functional G as follows

$$G(\Lambda_2^*)=\int_0^{\sigma}\lambda_1(b)\psi_1(b)db+\left(1-\int_0^{\sigma}\lambda_1(b)\psi_1(b)db\right)\times\int_0^{\sigma}\lambda_2(b)\psi_2(b)db. \tag{54}$$

It is clear that when $\Lambda_1^*=\Lambda_2^*=0$, the functional $G(0)=F(0)=\mathscr{R}_0$. Also, we can show that there exists a unique endemic steady state if and only if there exists a unique Λ_2^* such that $G(\Lambda_2^*)=1$, and $\Lambda_2^*>0$, furtheremore, we have $G'(\Lambda_2^*)<0$, $\lim_{\Lambda_2^*\to-\infty}G(\Lambda_2^*)=+\infty$, $G(0)=\mathscr{R}_0$. Then, if $\mathscr{R}_0>1$ the equation $G(\Lambda_2^*)=1$ has a unique positive real root noted

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$$
\begin{aligned}
&\frac{d}{da}s^*(a) = -\beta_1^*(a)s^*(a) - \beta_2^*(a)s^*(a) - d(a)s^*(a),\\
&\frac{d}{da}i_1^*(a) = \beta_1^*(a)s^*(a) + \gamma(a)i_2^*(a) + \eta_2(a)y^*(a) - (k_1(a) + \eta_1(a) + d(a))i_1^*(a),\\
&\frac{d}{da}i_2^*(a) = \beta_2^*(a)s^*(a) - (k_2(a) + \gamma(a) + d(a))i_2^*(a),\\
&\frac{d}{da}x^*(a) = k_1(a)i_1^*(a) + k_2(a)i_2^*(a) + \sigma_2(a)y^*(a) - (\sigma_1(a) + d(a) + \delta_1(a))x^*(a),\\
&\frac{d}{da}y^*(a) = \sigma_1(a)x^*(a) + \eta_1(a)i_1^*(a) - (\sigma_2(a) + \eta_2(a) + \delta_2(a) + d(a))y^*(a).
\end{aligned}
\tag{46}
$$

$$
\begin{aligned}
s^*(a) &= e^{\{-\int_0^a \Lambda_1^*\theta_1(\sigma) + \Lambda_2^*\theta_2(\sigma) + d(\sigma)d\sigma\}},\\
i_1^*(a) &= \int_0^a [\theta_1\Lambda_1^*s^*(\sigma) + \gamma(\sigma)i_2^*(\sigma) + \eta_2(\sigma)y^*(\sigma)]\, e^{\{-\int_\sigma^a k_2(\xi) + \eta_2(\xi) + d(\xi)d\xi\}}d\sigma,\\
i_2^*(a) &= \int_0^a \theta_2(\sigma)\Lambda_2^*s^*(\sigma)e^{\{-\int_\sigma^a k_2(\xi) + \gamma(\xi) + d(\xi)d\xi\}}d\sigma,\\
x^*(a) &= \int_0^a [k_1(\sigma)i_1^*(a) + k_2(\sigma)i_2^*(\sigma) + \sigma_2(\sigma)y^*(\sigma)]\, e^{\{-\int_\sigma^a \sigma_1(\xi) + d(\xi) + \delta_1(\xi)d\xi\}}d\sigma,\\
y^*(a) &= \int_0^a [\sigma_1(\sigma)x^*(\sigma) + \eta_1(\sigma)i_1^*(\sigma)i_1^*(\sigma)]\, e^{\{-\int_\sigma^a \sigma_2(\xi) + \eta_2(\xi) + \delta_2(\xi)d(\xi)d\xi\}}d\sigma.
\end{aligned}
\tag{49}
$$

as $\Lambda_{2\sim}^* > 0$, see figure 3, from (52) we get also a unique $\Lambda_{1\sim}^* > 0$. Therefore, the system has a unique positive endemic steady state. $\mathscr{E}^* = (s^*(a), i_1^*(a), i_2^*(a), x^*(a), y^*(a))$ with $s^*(a), e^*(a), v^*(a)$, and $i^*(a)$ defined above.

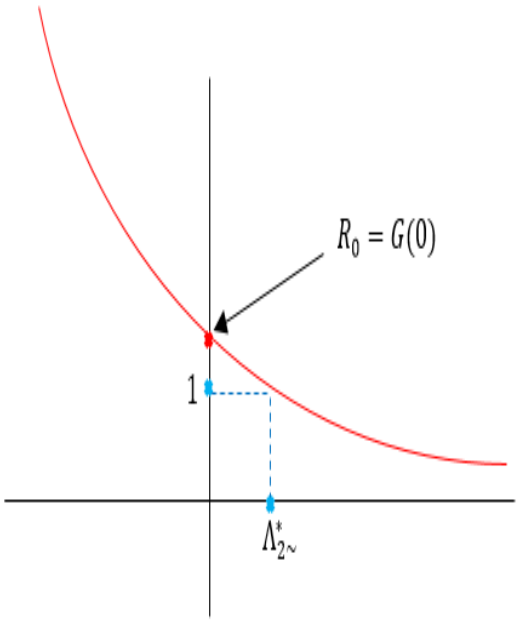

Fig. 3: Courbe representative of the function G.

4.4 Local stability of the endemic steady state

In this part, we looking to demonstrate the local stability of the endemic steady state. First, we have to introduce the linearized system associated to the system (11), let denote by $\check{s}(a,t) = s(a,t) - s^*(a)$, $\check{i}_1(a,t) = i_1(a,t) - i_1^*(a)$, $\check{i}_2(a,t) = i_2(a,t) - i_2^*(a)$, $\check{x}(a,t) = x(a,t) - x^*(a)$, and $\check{y}(a,t) = y(a,t) - y^*(a)$. Then the system (11) take the form

with,

$$
\begin{aligned}
&\check{\beta}_1(a,t) = \theta_1(a)\int_0^\sigma \lambda_1(b)\check{i}_1(b,t)db, \text{ and, } \check{\beta}_2(a,t) = \theta_2(a)\int_0^\sigma \lambda_2(b)\check{i}_2(b,t)db,\\
&\beta_1(a,t) = \check{\beta}_1(a,t) + \beta_1^*(a), \text{ and, } \beta_2(a,t) = \check{\beta}_2(a,t) + \beta_2^*(a).
\end{aligned}
\tag{56}
$$

The linearized part in system (55) is expressed as

This system is equipied with boundary conditions : $\check{s}(0,t) = 0$, $\check{i}_1(0,t) = 0$, $\check{i}_2(0,t) = 0$, $\check{x}(0,t) = 0$, and $\check{y}(0,t) = 0$, also the initial conditions are $\check{s}(a,0) = \check{s}_0(a)$, $\check{i}_1(a,0) = \check{i}_{1,0}(a)$, $\check{i}_2(a,0) = \check{i}_{2,0}(a)$, $\check{x}(a,0) = \check{x}_0(a)$, and $\check{y}(a,0) = \check{y}_0(a)$.

We consider the following exponential solution of system (57)

therefore, the variables $\check{s}(a), \check{i}_1(a), \check{i}_2(a), \check{x}(a)$, and $\check{y}(a)$ are satisfies the folowwing equations

where,

$$
\check{\Lambda}_1 = \int_0^\sigma \lambda_1(b)\check{i}_1(b)db, \text{ and } \check{\Lambda}_2 = \int_0^\sigma \lambda_2(b)\check{i}_2(b)db.
\tag{59}
$$

The states solution for this last system take the following form

Theorem 3.*The endemic steady state $\mathscr{E}^*$ exists if and only if $\mathscr{R}_0 > 1$, furthermore, this equilibrium is stable if the*

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$$
\begin{aligned}
(\partial_a+\partial_t)\check{s}(a,t) &= -\check{\beta}_1(a,t)\check{s}(a,t)-\check{\beta}_1(a,t)s^*(a)-\beta_1^*(a)\check{s}(a,t)\\
&\quad -\check{\beta}_2(a,t)\check{s}(a,t)-\check{\beta}_2(a,t)s^*(a)-\beta_2^*(a)\check{s}(a,t)-d(a)\check{s}(a,t),\\
(\partial_a+\partial_t)\check{i}_1(a,t) &= \check{\beta}_1(a,t)\check{s}(a,t)+\check{\beta}_1(a,t)s^*(a)+\beta_1^*(a)\check{s}(a,t)+\gamma(a)\check{i}_2(a,t)\\
&\quad +\eta_2(a)\check{y}(a,t)-(k_1(a)+\eta_1(a)+d(a))\check{i}_1(a,t),\\
(\partial_a+\partial_t)\check{i}_2(a,t) &= \check{\beta}_2(a,t)\check{s}(a,t)+\check{\beta}_2(a,t)s^*(a)+\beta_2^*(a)\check{s}(a,t)-(k_2(a)+\gamma(a)+d(a))\check{i}_2(a,t),\\
(\partial_a+\partial_t)\check{x}(a,t) &= k_1(a)\check{i}_1(a,t)+k_2(a)\check{i}_2(a,t)+\sigma_2(a)\check{y}(a,t)-(\sigma_1(a)+d(a)+\delta_1(a))\check{x}(a,t),\\
(\partial_a+\partial_t)\check{y}(a,t) &= \sigma_1(a)\check{x}(a,t)+\eta_1(a)\check{i}_1(a,t)-(\sigma_2(a)+\eta_2(a)+\delta_2(a)+d(a))\check{y}(a,t),
\end{aligned}
\tag{55}
$$

$$
\begin{aligned}
(\partial_a+\partial_t)\check{s}(a,t) &= -\check{\beta}_1(a,t)s^*(a)-\beta_1^*(a)\check{s}(a,t)-\check{\beta}_2(a,t)s^*(a)-\beta_2^*(a)\check{s}(a,t)-d(a)\check{s}(a,t),\\
(\partial_a+\partial_t)\check{i}_1(a,t) &= \check{\beta}_1(a,t)s^*(a)+\beta_1^*(a)\check{s}(a,t)+\gamma(a)\check{i}_2(a,t)+\eta_2(a)\check{y}(a,t)-(k_1(a)+\eta_1(a)+d(a))\check{i}_1(a,t),\\
(\partial_a+\partial_t)\check{i}_2(a,t) &= \check{\beta}_2(a,t)s^*(a)+\beta_2^*(a)\check{s}(a,t)-(k_2(a)+\gamma(a)+d(a))\check{i}_2(a,t),\\
(\partial_a+\partial_t)\check{x}(a,t) &= k_1(a)\check{i}_1(a,t)+k_2(a)\check{i}_2(a,t)+\sigma_2(a)\check{y}(a,t)-(\sigma_1(a)+d(a)+\delta_1(a))\check{x}(a,t),\\
(\partial_a+\partial_t)\check{y}(a,t) &= \sigma_1(a)\check{x}(a,t)+\eta_1(a)\check{i}_1(a,t)-(\sigma_2(a)+\eta_2(a)+\delta_2(a)+d(a))\check{y}(a,t).
\end{aligned}
\tag{57}
$$

$$
\check{s}(a,t)=\check{s}(a)e^{\omega t},\ \check{i}_1(a,t)=\check{i}_1(a)e^{\omega t},\ \check{i}_2(a,t)=\check{i}_2(a)e^{\omega t},\ \check{x}(a,t)=\check{x}(a)e^{\omega t},\ \check{y}(a,t)=\check{y}(a)e^{\omega t},
$$

$$
\begin{aligned}
\frac{d}{da}\check{s}(a) &= -\omega\check{s}(a)-\theta_1(a)\check{\Lambda}_1 s^*(a)-\theta_1(a)\Lambda_{1\sim}^*\check{s}(a)-\theta_2(a)\check{\Lambda}_2 s^*(a)-\theta_2(a)\Lambda_{2\sim}^*\check{s}(a)-d(a)\check{s}(a),\\
\frac{d}{da}\check{i}_1(a) &= -\omega\check{i}_1(a)+\theta_1(a)\check{\Lambda}_1 s^*(a)+\theta_1(a)\Lambda_{1\sim}^*\check{s}(a)+\gamma(a)\check{i}_2(a)+\eta_2(a)\check{y}(a)-(k_1(a)+\eta_1(a)+d(a))\check{i}_1(a),\\
\frac{d}{da}\check{i}_2(a) &= -\omega\check{i}_2(a)+\theta_2(a)\check{\Lambda}_2 s^*(a)+\theta_2(a)\Lambda_{2\sim}^*\check{s}(a)-(k_2(a)+\gamma(a)+d(a))\check{i}_2(a),\\
\frac{d}{da}\check{x}(a,t) &= -\omega\check{x}(a)+k_1(a)\check{i}_1(a)+k_2(a)\check{i}_2(a)+\sigma_2(a)\check{y}(a)-(\sigma_1(a)+d(a)+\delta_1(a))\check{x}(a),\\
\frac{d}{da}\check{y}(a) &= -\omega\check{y}(a)+\sigma_1(a)\check{x}(a)+\eta_1(a)\check{i}_1(a)-(\sigma_2(a)+\eta_2(a)+\delta_2(a)+d(a))\check{y}(a).
\end{aligned}
\tag{58}
$$

$$
\begin{aligned}
\check{s}(a) &= -\check{\Lambda}_1\int_0^a \theta_1(\sigma)s^*(\sigma)e^{\{-\int_\sigma^a \omega+\Lambda_{1\sim}^*\theta_1(\xi)+\Lambda_{2\sim}^*\theta_2(\xi)+d(\xi)d\xi\}}d\sigma\\
&\quad -\check{\Lambda}_2\int_0^a \theta_2(\sigma)s^*(\sigma)e^{\{-\int_\sigma^a \omega+\Lambda_{1\sim}^*\theta_1(\xi)+\Lambda_{2\sim}^*\theta_2(\xi)+d(\xi)d\xi\}}d\sigma,\\
\check{i}_1(a) &= \int_0^a \left[\check{\Lambda}_1\theta_1(\sigma)s^*(\sigma)+\Lambda_{1\sim}^*\theta_1(\sigma)\check{s}(\sigma)+\gamma(\sigma)\check{i}_2(\sigma)+\eta_2(\sigma)\check{y}(\sigma)\right]\times e^{\{-\int_\sigma^a \omega+k_1(\xi)+\eta_1(\xi)+d(\xi)d\xi\}}d\sigma,\\
\check{i}_2(a) &= \int_0^a \left[\check{\Lambda}_2\theta_2(\sigma)s^*(\sigma)+\Lambda_{2\dim}^*\theta_2(\sigma)\check{s}(\sigma)\right]\times e^{\{-\int_\sigma^a \omega+k_2(\xi)+\delta_1(\xi)+d(\xi)d\xi\}}d\sigma,\\
\check{x}(a) &= \int_0^a \left[k_1(\sigma)\check{i}_1(\sigma)+k_2(\sigma)\check{i}_2(\sigma)+\sigma_2(\sigma)\check{y}(\sigma)\right]\times e^{\{-\int_\sigma^a \omega+\sigma_1(\xi)+\delta_1(\xi)+d(\xi)d\xi\}}d\sigma\\
\check{y}(a) &= \int_0^a \left[\sigma_1(\sigma)\check{x}(\sigma)+\eta_1(\sigma)\check{i}_1(\sigma)\right]\times e^{\{-\int_\sigma^a \omega+\sigma_2(\xi)+\eta_2(\xi)+\delta_2(\xi)+d(\xi)d\xi\}}d\sigma.
\end{aligned}
\tag{60}
$$

eigenvalues of the linearized system (57) have a negative real part.

5 Numerical simulations

In this section, we will show the behavior of each state (susceptible, infected aware of the infection, infected unaware of infection, infected with AIDS, and treated individuals) with the consideration of age a and the time

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Table 1: Values of model parameters for simulations

Parameter	value	Parameter	value
k_1	0.25	k_2	0.03
σ_1	0.45	σ_2	0.02
η_1	$\in (0,2)$	η_2	0.45
γ	0.05	δ_1	0.0909
δ_2	0.0679	λ_1	$\in (0,0.03)$
λ_2	$\in (0,0.02)$	Λ	100
θ_1	1	θ_2	1

t. For this purpose we consider the age-function $d(a) = \frac{(a-30)^2}{10^4}$, is depend of age variable a. Also, the rates of transmission $\lambda_1 \in (0,0.03)$, $\lambda_2 \in (0,0.02)$. Further, the other parameters values are given in table 1.

Senario 1 : This paragraph discusses the stability and convergence to the equilibrium steady states. We begin by selecting the parameters $\lambda_1(a) = 0.01$ and $\lambda_2(a) = 0.001$, which clearly demonstrate the stability of the disease-free steady state $\mathscr{E}^0$, as the corresponding $\mathscr{R}_0$ value is less than one. As illustrated in Figures 4, 5, 6, 7, and 8, which depict the behavior of state variables over time, it is evident that the system converges to the equilibrium $\mathscr{E}^0 = (2826,0,0,0,0)$. On the other hand we fixed the parameters λ_1, and λ_2 at the values 0.03, and 0.02 respectively. Figures 9, 10,11,12, and 13, show the behavior of those state in the presense of the disease, and the convergence to the disease steady state $\mathscr{E}^* = (1037,14.39,260.6,36.39,7.25)$, where $\mathscr{R}_0 > 1$.

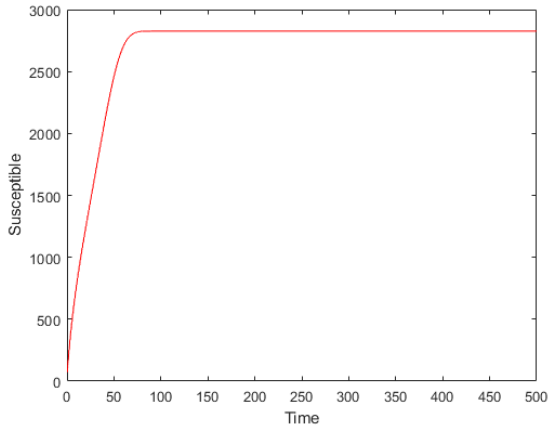

Fig. 4: Behaviour of the susceptible individuals $\int_0^\sigma S(a,t)da$ over the time when $\mathscr{R}_0 < 1$

The distribution of the system's states with respect to age and time is illustrated in the figures 14-18. We consider two distinct cases: the first case corresponds to $\lambda_1(a) = 0.01$ and $\lambda_2(a) = 0.001$ where the basic

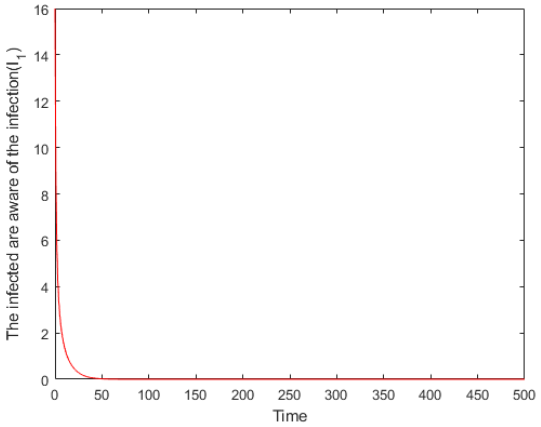

Fig. 5: Behaviour of the infected aware of infection $\int_0^\sigma I_1(a,t)da$ over the time when $\mathscr{R}_0 < 1$

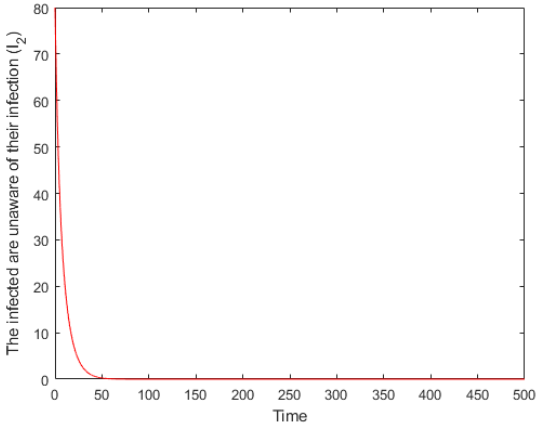

Fig. 6: Behaviour of the infected unware of infection $\int_0^\sigma I_2(a,t)da$ over the time when $\mathscr{R}_0 < 1$

reproduction number $\mathscr{R}_0$ is less than one ($\mathscr{R}_0 < 1$). The second case involves higher values of $\lambda_1 = 0.03$, and $\lambda_2 = 0.02$ resulting in $\mathscr{R}_0 > 1$. Figure 14 depicts the age distribution of susceptible individuals for the case $\mathscr{R}_0 < 1$. Here, we observe that the dynamics occur primarily when $t < a$, meaning that the behavior of the system is influenced by individuals whose age a exceeds the elapsed time t. On the other hand, when examining the distribution over time, the number of susceptible individuals remains positive and constant, while the other states (such as infected or treated individuals) gradually diminish and eventually vanish. This indicates a clear convergence to the disease-free steady state when $\mathscr{R}_0 < 1$. These findings are further supported by Figures 15, 16,

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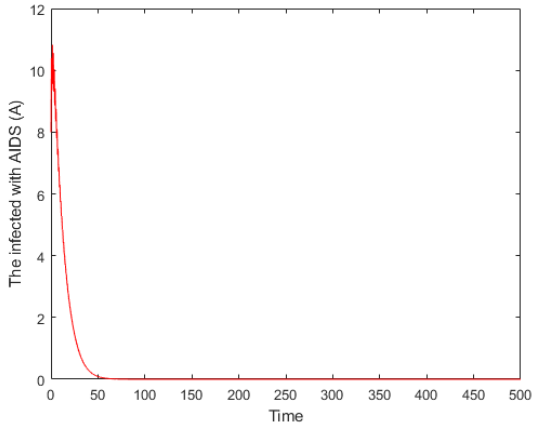

Fig. 7: Behaviour of the infected with AIDS $\int_0^\sigma A(a,t)da$ over the time when $\mathscr{R}_0 < 1$

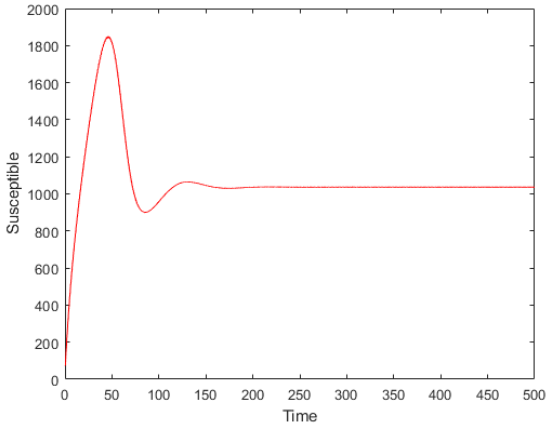

Fig. 9: Behaviour of the susceptible individuals $\int_0^\sigma S(a,t)da$ over the time when $\mathscr{R}_0 > 1$

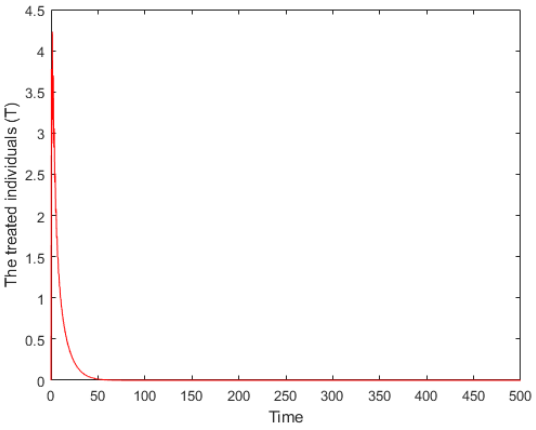

Fig. 8: Behaviour of thetreaated individuals $\int_0^\sigma T(a,t)da$ over the time when $\mathscr{R}_0 < 1$

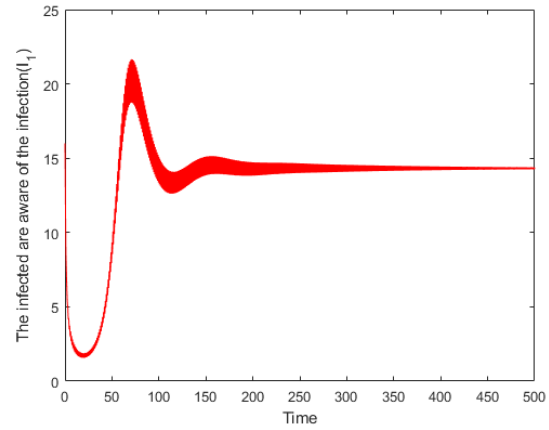

Fig. 10: Behaviour of the infected aware of infection $\int_0^\sigma I_1(a,t)da$ over the time when $\mathscr{R}_0 > 1$

17, and 18, which illustrate the temporal evolution of the system's states.

In the case where, $\mathscr{R}_0 > 1$, Figures 19, 20,21, 22, and 23 show the distribution of susceptible S, Infected I_1, Infected I_2, Infected with AIDS A, and Treated T individuals over time t and age a. These dynamics are showd when $t < a$, this condition The condition arises naturally because, at time $t = 0$, the population consists of individuals of all ages $a \geq 0$, as time progresses ($t > 0$), the age of individuals increases, and the dynamics of the system are influenced by the interaction between time and age. The condition $t < a$ ensures that only individuals who were already present at the start of the simulation (i.e., those with age $a \geq t$) are considered in the dynamics. In the distribution of infected individuals, the majority are situated between the ages of 10 and 30. This age range represents the most vulnerable group for the development of HIV/AIDS, as individuals within this demographic are more likely to contract the infection or transmit it to others.

Senario 2 : In this scenario, we demonstrate the impact of infected individuals who are aware of their infection to transmit the disease. To illustrate this, we set the transmission rate λ_2 to zero ($\lambda_2 = 0$), effectively eliminating the contribution of unaware infected individuals (I_2) to the spread of the disease. The results are depicted in Figures 24, 25, 26, and 27. From these figures, we observe that the dynamics of I_2 over time become null, confirming that aware infected individuals

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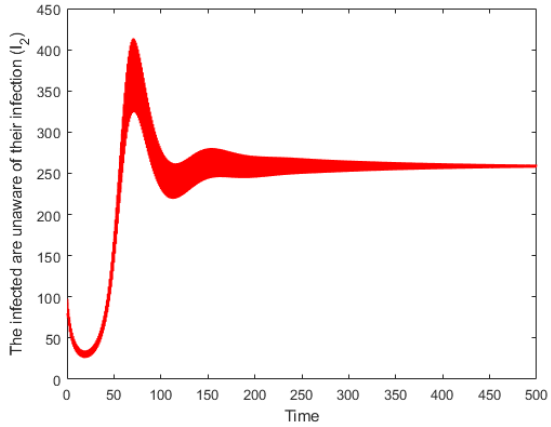

Fig. 11: Behaviour of the infected unware of infection $\int_0^\sigma I_2(a,t)da$ over the time when $\mathscr{R}_0 > 1$

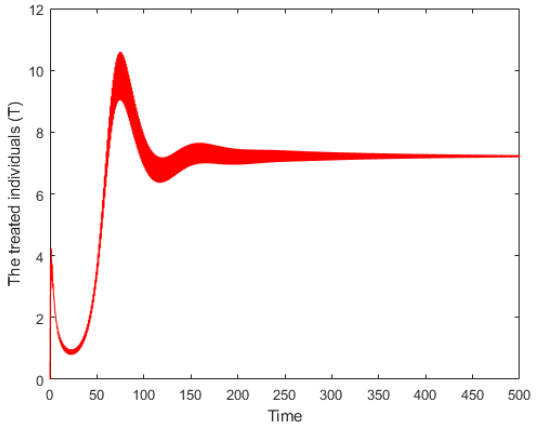

Fig. 13: Behaviour of thetreaated individuals $\int_0^\sigma T(a,t)da$ over the time when $\mathscr{R}_0 > 1$

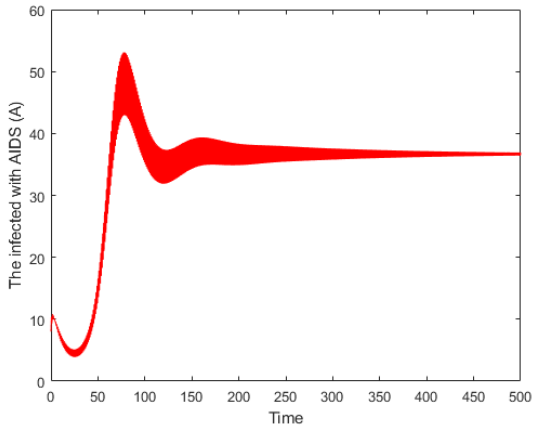

Fig. 12: Behaviour of the infected with AIDS $\int_0^\sigma A(a,t)da$ over the time when $\mathscr{R}_0 > 1$

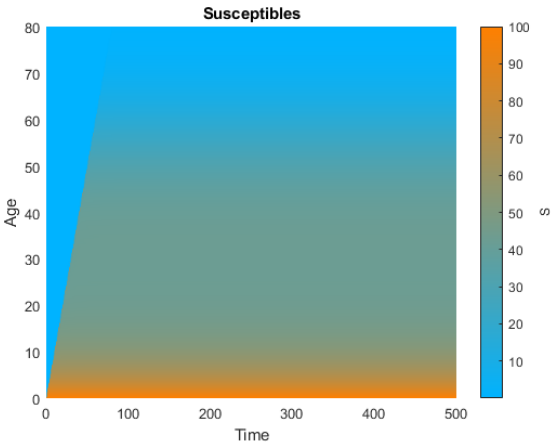

Fig. 14: The behaviors of susceptible individuals according to time and age, when $\mathscr{R}_0 < 1$

do not contribute to new unware infections when $\lambda_2 = 0$. However, we note the continued existence of AIDS cases over time, which is attributed to the presence of infected individuals who are aware of their status (I_1).
Additionally, the model shows the presence of treated individuals over time, highlighting the positive impact of awareness on disease management. When infected individuals (I_1) that aware of their infection, they are more likely to demand for treatment, thereby reducing their infectiousness and contributing to the overall control of the disease.

Senario 3 : Now, we evaluate the effectiveness of infected individuals who are unaware of their infection (I_2) in transmitting the disease. To achieve this, we set the transmission rate λ_1 to zero ($\lambda_1 = 0$), effectively removing the contribution of aware infected individuals (I_1) to the spread of the disease. The dynamics of HIV/AIDS under this scenario are illustrated in Figures 29, 30, 31, 32, and 33. These figures reveal that the presence of unaware infected individuals (I_2) can lead to the emergence of aware infected individuals (I_1) due to the transition rate γ, which represents the rate at which unaware individuals become aware of their infection [16]-[21].

Over time and across different age groups, we observe the distribution of both types of infected individuals: those who are aware (I_1) and those who are unaware (I_2) of their infection. Additionally, the model shows the presence of individuals with AIDS, as well as treated individuals. The latter group arises because aware infected individuals (I_1) are more likely to demand

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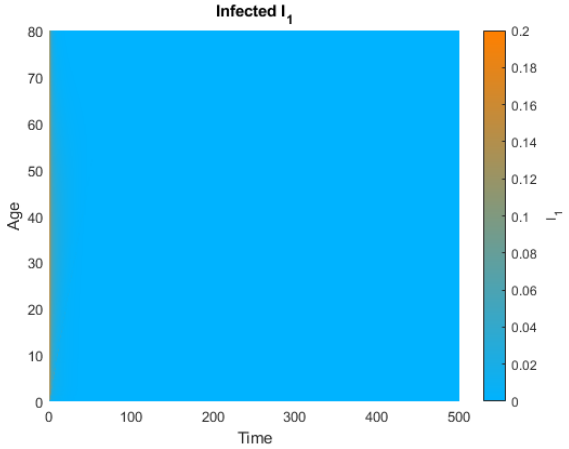

Fig. 15: The behaviors of infected individuals I_1 according to time and age, when $\mathscr{R}_0 < 1$

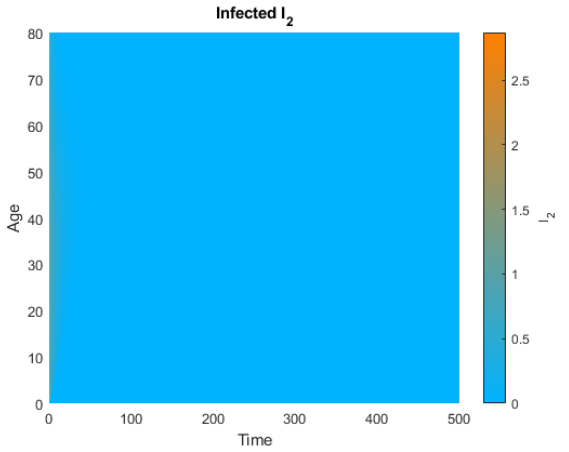

Fig. 16: The behaviors of infected individuals I_2 according to time and age, when $\mathscr{R}_0 < 1$

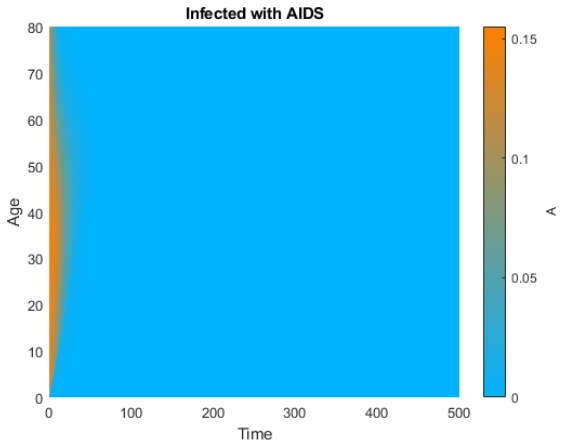

Fig. 17: The behaviors of infected with AIDS individuals according to time and age, when $\mathscr{R}_0 < 1$

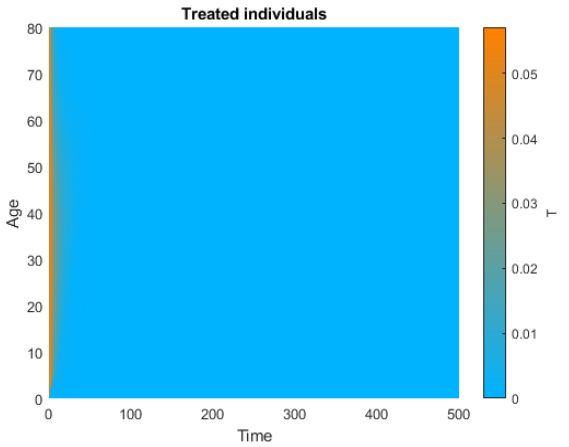

Fig. 18: The behaviors of treated individuals according to time and age, when $\mathscr{R}_0 < 1$

treatment, thereby reducing their infectiousness and contributing to the control of the disease.

Senario 4 : In this scenario, we increase the treatment rate to a higher value ($\eta_1 = 2$) to evaluate its impact on the dynamics of the HIV/AIDS disease. The results are illustrated in Figures 34, 35, 36, 37, and 38, which depict the behavior of the susceptible individuals (S), unaware infected individuals (I_1), aware infected individuals (I_2), individuals with AIDS (A), and treated individuals (T), respectively. Compared to Figures 19, 20, 21, 22, and 23, it is evident that the number of treated individuals increases significantly when $\eta_1 = 2$. Furthermore, the populations of both unaware (I_1) and aware (I_2) infected individuals decrease due to the higher transition rate to treatment.

These findings underscore the critical role of treatment in controlling the spread of HIV/AIDS. By increasing the treatment rate, more infected individuals are moved into the treated category, reducing their infectiousness and preventing further transmission [22]-[28].

This highlights the importance of awareness campaigns and early detection programs, as they encourage infected individuals to demand for treatment promptly. Such interventions not only reduce the number of new infections but also improve overall public health outcomes by curbing the spread of the disease.

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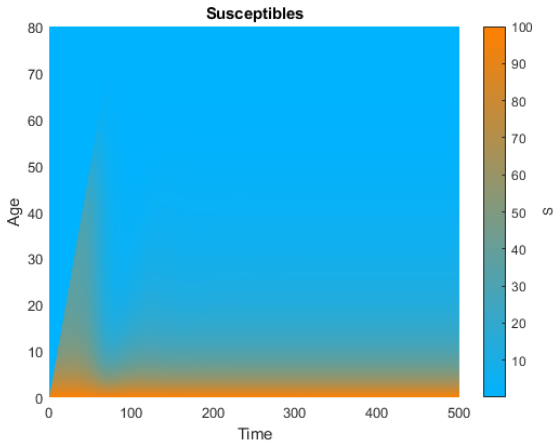

Fig. 19: The behaviors of susceptible individuals according to time and age, when $\mathscr{R}_0 > 1$.

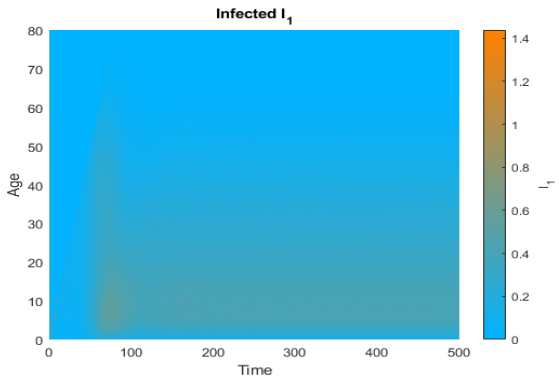

Fig. 20: The behaviors of infected individuals I_1 according to time and age, when $\mathscr{R}_0 > 1$.

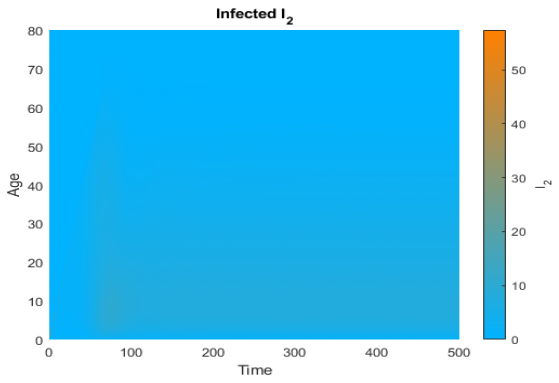

Fig. 21: The behaviors of infected individuals I_2 according to time and age, when $\mathscr{R}_0 > 1$.

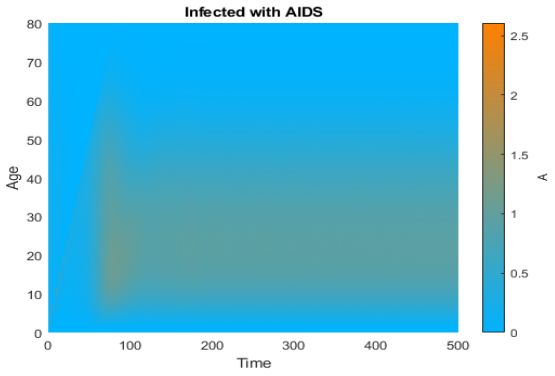

Fig. 22: The behaviors of infected with AIDS individuals according to time and age, when $\mathscr{R}_0 > 1$.

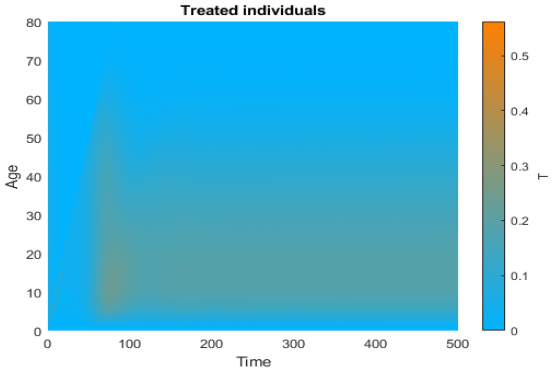

Fig. 23: The behaviors of treated individuals according to time and age, when $\mathscr{R}_0 > 1$.

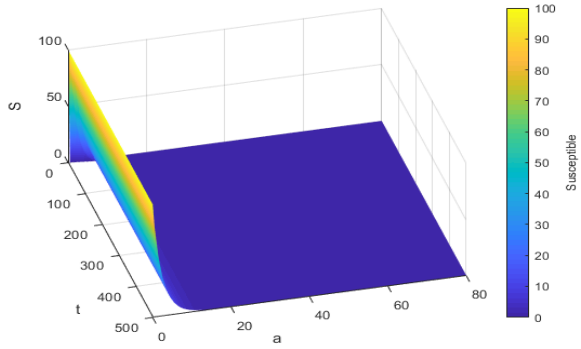

Fig. 24: The behaviors of susceptible individuals in the absence of infected I_2.

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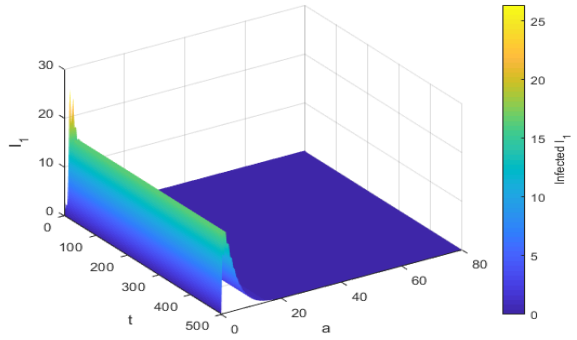

Fig. 25: The behaviors of infected individuals I_1in the absence of infected I_2.

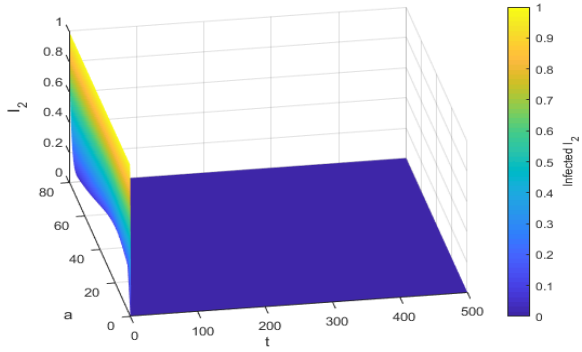

Fig. 26: The behaviors of infected individuals I_2 in the absence of infected I_2.

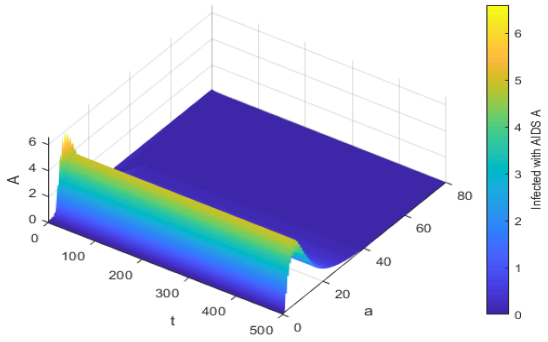

Fig. 27: The behaviors of infected with AIDS individuals in the absence of infected I_2.

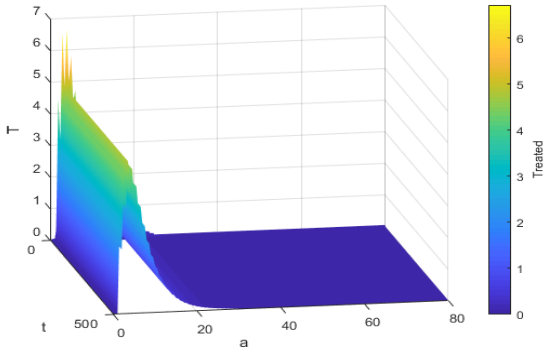

Fig. 28: The behaviors of treated individuals in the absence of infected I_2.

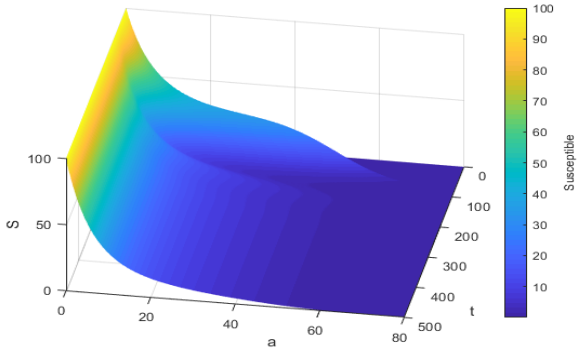

Fig. 29: The behaviors of susceptible individuals in the absence of infected I_1

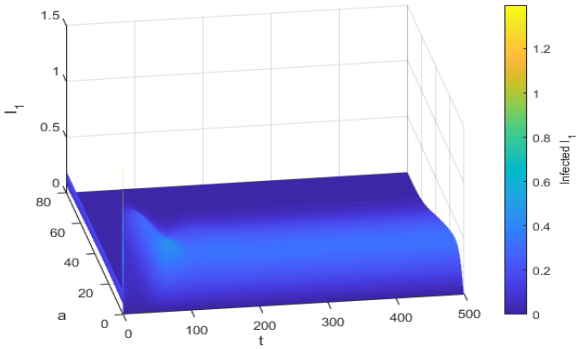

Fig. 30: The behaviors of infected individuals I_1 in the absence of infected I_1.

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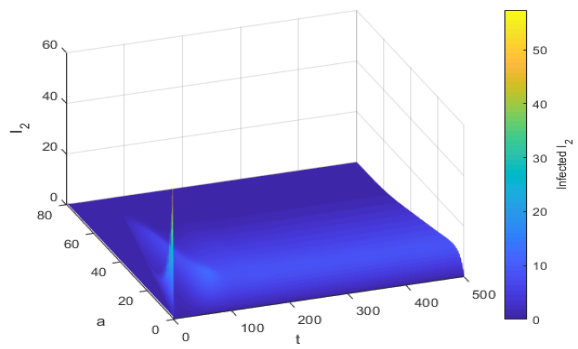

Fig. 31: The behaviors of infected individuals I_2 in the absence of infected I_1.

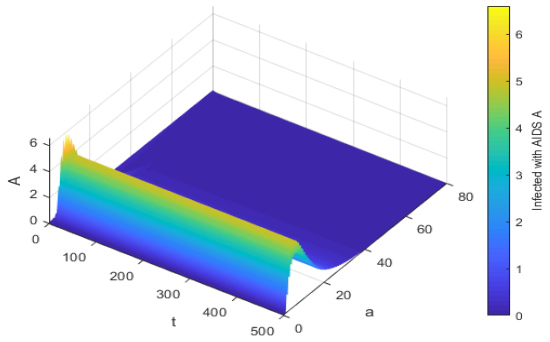

Fig. 32: The behaviors of infected with AIDS individuals in the absence of infected I_1.

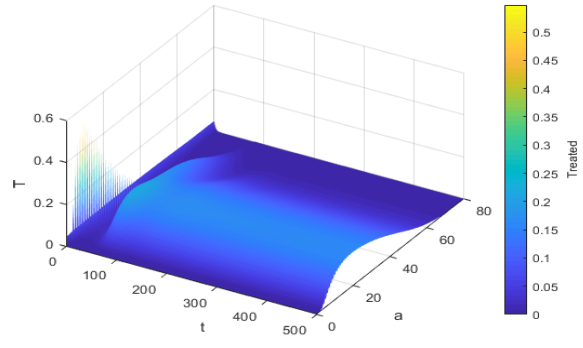

Fig. 33: The behaviors of treated individuals in the absence of infected I_1.

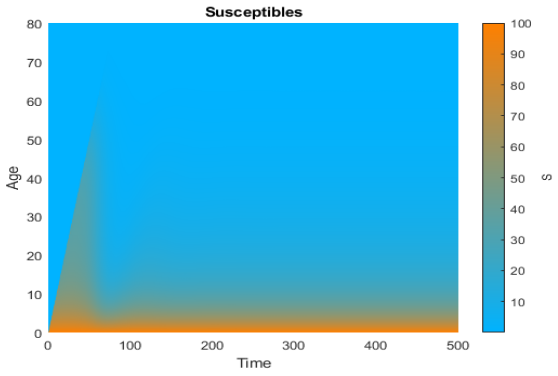

Fig. 34: The behaviors of susceptible individuals with $\eta_1 = 2$.

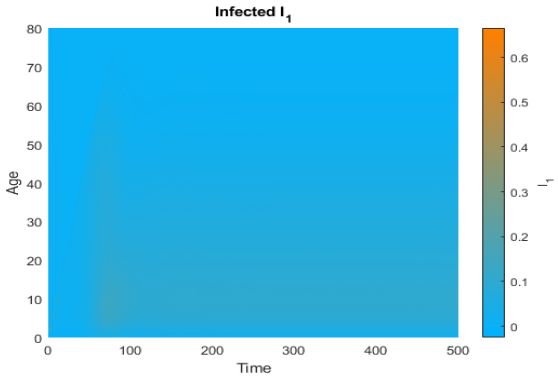

Fig. 35: The behaviors of infected individuals I_1 with $\eta_1 = 2$.

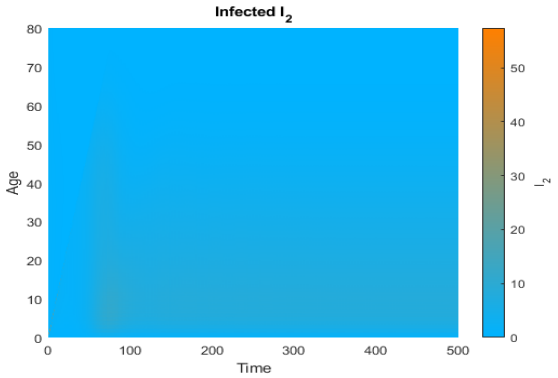

Fig. 36: The behaviors of infected individuals I_2 with $\eta_1 = 2$.

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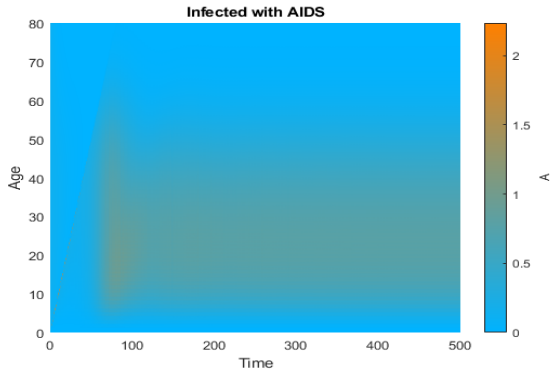

Fig. 37: The behaviors of infected with AIDS individuals with $\eta_1 = 2$.

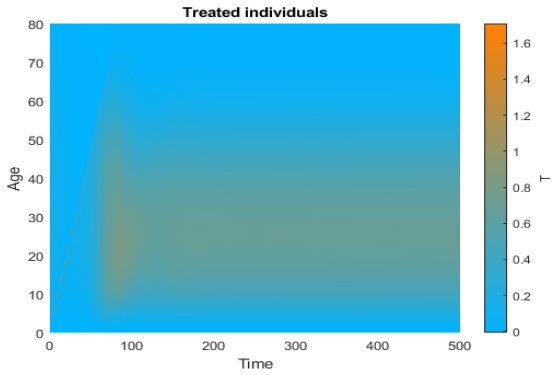

Fig. 38: The behaviors of treated individuals with $\eta_1 = 2$.

6 Conclusion

In this paper, we proposed a novel age-structured mathematical model for HIV/AIDS, incorporating two distinct stages of infection: individuals who are aware of their infection and those who are unaware. Additionally, we included a compartment for treated individuals to account for those undergoing treatment. To demonstrate the mathematical well-posedness of the model, we reformulated it as an abstract semi-linear Cauchy problem on a Banach space and proved the existence and uniqueness of solutions. Furthermore, we focused on analyzing the existence and stability of disease steady states, which are crucial for understanding the dynamics of diseases with long latent periods. In the numerical simulations section, we demonstrated the convergence of state variables to the steady states in two scenarios: when $\mathscr{R}_0 < 1$ (stability of the disease-free steady state) and when $\mathscr{R}_0 > 1$ (stability of the endemic steady state). Additionally, we investigated the role of individuals unaware of their infection in spreading HIV and developing AIDS, with a particular focus on the age group most affected by the disease (10 to 30 years). We also examined the impact of treatment in reducing infection rates and controlling the spread of HIV/AIDS [16]. Our analysis demonstrates that enhancing treatment accessibility and promoting awareness are essential strategies for managing HIV/AIDS. By prioritizing early detection and timely treatment, the burden of the disease can be significantly reduced, leading to healthier communities and better long-term outcomes [17].

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