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AN EPIDEMIC MODEL FOR THE TRANSMISSION DYNAMICS OF HIV AND ANOTHER PATHOGEN

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Abstract

A five-dimensional deterministic model is proposed for the dynamics between HIV and another pathogen within a given population. The model exhibits four equilibria: a disease-free equilibrium, an HIV-free equilibrium, a pathogen-free equilibrium and a co-existence equilibrium. The existence and stability of these equilibria are investigated. A competitive finite-difference method is constructed for the solution of the non-linear model. The model predicts the optimal therapy level needed to eradicate both diseases.

1. Introduction

Following the discovery of the human immunodeficiency virus (HIV) three decades ago, much attention has been focussed on the development and analysis of realistic mathematical models for the transmission dynamics, pathogenesis and control mechanisms of HIV (see, for instance, [1, 2, 4, 5, 7]).

Since HIV is known to replicate only in activated CD4+ T cells (see [4, 5, 7]), the study of the effect of the interactions between HIV and other pathogens (such as the flu virus, mycobacterium tuberculosis, the hepatitis virus *etc*.) is important. McLean and Nowak [5] have proposed within host (*in vivo*) models for the dynamics between HIV and activated CD4+ T cells specific to other pathogens.

Our study is focussed on the design and analysis of a new population model for the transmission dynamics of HIV and another competing pathogen (assumed to be curable) within a given population. Such an epidemiological model can be used to assess anti-HIV programmes in nations where access to highly-active anti-retroviral therapy (HAART) [7] for controlling HIV is not readily available, but cures for the competing pathogens (causing tuberculosis, flu, hepatitis *etc*.) are generally

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available and affordable. The central question to ask is whether an effective treatment mechanism that solely focuses on eradicating competing pathogens can reduce the spread of HIV within the population.

In this paper, we propose a new deterministic model for the transmission dynamics of HIV and a single competing pathogen that is curable. Qualitative and numerical analyses of the resulting five-dimensional nonlinear model are carried out to determine the optimal therapeutic coverage levels needed for eradicating both diseases. The model is formulated in Section 2 and analysed in Section 3. The effect of treatment on the stability of the associated equilibria is discussed in Section 4. In Section 5, we construct a robust numerical method, which is free of scheme-dependent numerical instabilities (such as oscillations, chaos, bifurcations and convergence to spurious zeros). Numerical experiments are reported in Section 6.

2. Mathematical model

The model monitors five populations: susceptibles (X), individuals infected with a curable pathogen (Y_1), HIV-infected individuals (Y_2), HIV-infected individuals that have progressed to clinical AIDS (A) and the population of individuals infected with the pathogen who become non-infectious following effective treatment (Z). The total population size is $N = N(t) = X(t) + Y_1(t) + Y_2(t) + A(t) + Z(t)$.

2.1. Susceptible individuals, $X(t)$. All individuals recruited into the population at a rate of Π per year are considered to be susceptible to both infectious diseases (HIV and the other pathogen). Susceptibles die of natural causes at a rate of μ per year. Susceptibles are lost following contact with members of the Y_i populations ($i = 1, 2$) at rate β_i, respectively. The average number of contacts per unit time is denoted by c. Thus

$$\frac{dX}{dt} = \Pi - \mu X - \left(\frac{c\beta_1 Y_1 + c\beta_2 Y_2}{N}\right) X.$$

2.2. Pathogen-infected individuals, $Y_1(t)$. This population is generated following the infection of susceptibles with the pathogen at rate β_1. Since members of this (Y_1) population can be cured, we model the effect of treatment in terms of inhibition of transmission probability β_1 given by $(1-\tau)\beta_1$. This population is diminished by HIV infection (at rate β_2) and by natural death (at rate μ). This gives

$$\frac{dY_1}{dt} = \frac{(1-\tau)c\beta_1 Y_1 X}{N} - \frac{c\beta_2 Y_1 Y_2}{N} - \mu Y_1.$$

2.3. HIV-infected individuals, $Y_2(t)$. This population is generated by the HIV infection of both the susceptibles and pathogen-infected individuals (at rate β_2). It

is diminished by natural death (at rate μ) and by the development of clinical AIDS (at rate v). For computational convenience, it is assumed that Y_2 individuals are not susceptible to infection by the pathogen. It is further assumed that anti-HIV treatment is not available within the community; hence members of the Y_2 and A populations are not treated. This suggests

$$\frac{dY_2}{dt} = \frac{c\beta_2 Y_2 X}{N} + \frac{c\beta_2 Y_1 Y_2}{N} - (\mu + v)Y_2.$$

2.4. Individuals with clinical AIDS, $A(t)$. The population of individuals with clinical AIDS, $A(t)$, increases when members of the Y_2 population progress to clinical AIDS (at rate v). This population is reduced by natural death (at rate μ) and by AIDS-induced death (at rate d). Thus

$$\frac{dA}{dt} = vY_2 - (\mu + d)A.$$

2.5. Non-infectious individuals, $Z(t)$. This population is generated following the effective treatment of members of the Y_1 population (at a rate τ) and is diminished by natural death (at a rate μ), so that

$$\frac{dZ}{dt} = \frac{\tau c\beta_1 Y_1 X}{N} - \mu Z.$$

In summary, the model consists of the equations

$$\begin{aligned}
\frac{dX}{dt} &= \Pi - \mu X - \left(\frac{c\beta_1 Y_1 + c\beta_2 Y_2}{N}\right) X,\\
\frac{dY_1}{dt} &= \frac{(1-\tau)c\beta_1 Y_1 X}{N} - \frac{c\beta_2 Y_1 Y_2}{N} - \mu Y_1,\\
\frac{dY_2}{dt} &= \frac{c\beta_2 Y_2 X}{N} + \frac{c\beta_2 Y_1 Y_2}{N} - (\mu + v)Y_2,\\
\frac{dA}{dt} &= vY_2 - (\mu + d)A,\\
\frac{dZ}{dt} &= \frac{\tau c\beta_1 Y_1 X}{N} - \mu Z.
\end{aligned} \tag{2.1}$$

It should be mentioned that since the physical situation being modelled is that of population dynamics, it is necessary to impose the condition that all the dependent variables and parameters of the model are non-negative.

3. Existence and stability of equilibria

Defining, first of all, the force of infection given by (see [1, 8])

$$G(t) = \frac{c\beta_1 Y_1(t) + c\beta_2 Y_2(t)}{N(t)}, \quad H(t) = \frac{c\beta_2 Y_2(t)}{N(t)}, \tag{3.1}$$

it follows from (2.1) that the associated expressions for the population densities at equilibrium are (see [1, 8])

$$X^* = \frac{\Pi}{\mu + G^*}, \quad Y_1^* = \frac{(1-\tau)(G^* - H^*)X^*}{\mu + H^*}, \quad Y_2^* = \frac{H^*(X^* + Y_1^*)}{\mu + v},$$
$$A^* = \frac{vY_2^*}{\mu + d}, \quad Z^* = \frac{\tau(G^* - H^*)X^*}{\mu} \quad \text{and} \quad N^* = \frac{1}{\mu}(\Pi - dA^*).$$

Substituting the above into the expressions for G and H in (3.1) gives

$$G^* = \frac{1}{N^*}\left(\frac{c\beta_1(1-\tau)\Pi(G^*-H^*)}{(\mu+H^*)(\mu+G^*)} + \frac{c\beta_2 H^*}{\mu+v}\left[\frac{\Pi}{\mu+G^*} + \frac{(1-\tau)\Pi(G^*-H^*)}{(\mu+H^*)(\mu+G^*)}\right]\right) \tag{3.2}$$

and

$$H^* = \frac{1}{N^*}\left(\frac{c\beta_2 H^*}{\mu+v}\left[\frac{\Pi}{\mu+G^*} + \frac{(1-\tau)\Pi(G^*-H^*)}{(\mu+H^*)(\mu+G^*)}\right]\right). \tag{3.3}$$

To find the equilibria of the model, we need to determine the fixed points of the equation

$$x = \Phi(x) = \begin{pmatrix}\phi_1(G,H)\\ \phi_2(G,H)\end{pmatrix},$$

where ϕ_1 and ϕ_2 are the right-hand sides of (3.2) and (3.3), respectively.

3.1. Disease-free equilibrium. Clearly $(0,0)$ is a fixed point of Φ (which corresponds to the disease-free equilibrium of the model) since $\phi_1(0,0) = 0$ and $\phi_2(0,0) = 0$. In the context of (2.1), this fixed point corresponds to

$$E_0 = (\Pi/\mu, 0, 0, 0, 0).$$

The Jacobian of Φ is given by

$$J = \begin{pmatrix}\partial\phi_1(G,H)/\partial G & \partial\phi_1(G,H)/\partial H\\ \partial\phi_2(G,H)/\partial G & \partial\phi_2(G,H)/\partial H\end{pmatrix}.$$

Evaluating J at $(0,0)$ gives

$$J_0 = \begin{pmatrix}R_1 & \left.\dfrac{\partial\phi_1(G,H)}{\partial H}\right|_{(0,0)}\\ 0 & R_2\end{pmatrix},$$

where

$$R_1 = c\beta_1(1-\tau)/\mu \quad \text{and} \quad R_2 = c\beta_2/(\mu+v). \tag{3.4}$$

The eigenvalues of J_0 are R_1 and R_2. The dominant eigenvalue of J_0, given by $\mathscr{R} = \max\{R_1, R_2\}$, is the basic reproductive number [1, 3, 8]. It follows then that the disease-free equilibrium is locally asymptotically stable if $\mathscr{R} < 1$ and unstable if $\mathscr{R} > 1$.

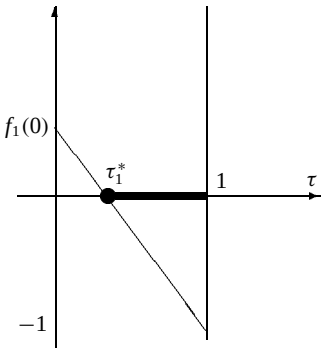

FIGURE 1. $f_1(0) = c\beta_1/\mu - 1 > 0$.

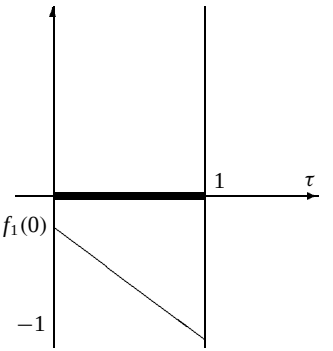

FIGURE 2. $f_1(0) \leq 0$.

3.2. Non-trivial equilibria. Now we discuss the existence and stability of the non-trivial equilibria. Note that if $Y_2^* = 0$, then $H^* = 0$, so that $G^* = c\beta_1 Y_1^*/N^*$. Therefore $(G^*, 0)$ is a fixed point of Φ where

$$G^* = \mu\left[\frac{c\beta_1(1-\tau)}{\mu} - 1\right] = \mu(R_1 - 1),$$

with R_1 defined in (3.4). Since μ is assumed positive, G^* is positive whenever $R_1 > 1$. By substituting G^* (above) and $H^* = 0$ in the population densities, it is easy to see that the fixed point $(G^*, 0)$ of Φ corresponds to a non-trivial equilibrium (free of HIV and AIDS) given by

$$E_1 = \left[\frac{\Pi}{\mu R_1}, \frac{(1-\tau)\Pi(R_1-1)}{\mu R_1}, 0, 0, \frac{\tau\Pi(R_1-1)}{\mu R_1}\right].$$

It should be noted that we require $\mathscr{R} > 1$ ($\mathscr{R}$ is the basic reproductive number of infection) to ensure the existence of a non-trivial equilibrium. It can be shown, after some manipulation, that the Jacobian of Φ at $(G^*, 0)$ is

$$J_1 = \begin{pmatrix} D_1 & \left.\dfrac{\partial\phi_1(G,H)}{\partial H}\right|_{(G^*,0)} \\ 0 & D_2 \end{pmatrix},$$

where $D_1 = 1/R_1$, $D_2 = (R_2/R_1)[1 + (1-\tau)(R_1 - 1)]$ and R_2 is as defined in (3.4).

The eigenvalues of J_1 are D_1 and D_2. Since $R_1 > 1$, it follows that $D_1 < 1$. Thus E_1 is locally asymptotically stable if $D_2 < 1$ and unstable if $D_2 > 1$.

Suppose now that $G^* = H^*$ (that is, $Y_1^* = 0$). In this case, the fixed point of Φ is

$$\left[\frac{(\mu+d)(\mu+v)(R_2-1)}{\mu+v+d}, \frac{(\mu+d)(\mu+v)(R_2-1)}{\mu+v+d}\right].$$

Thus G^* and H^* are positive if $R_2 > 1$. This fixed point corresponds to another non-trivial equilibrium (pathogen-free)

$$E_2 = \left[\frac{(\mu + v + d)\Pi}{R_2(\mu + v)(\mu + d) - vd}, 0, \frac{(\mu + d)\Pi(R_2 - 1)}{R_2(\mu + v)(\mu + d) - vd}, \right.$$
$$\left. \frac{v\Pi(R_2 - 1)}{R_2(\mu + v)(\mu + d) - vd}, 0 \right].$$

It is worth mentioning that if $R_2 > 1$, then $R_2(\mu+v)(\mu+d)-vd > \mu(\mu+v+d) > 0$. Thus none of the components of E_2 is negative for $R_2 > 1$.

The Jacobian of Φ at (G^*, H^*) is given by

$$J_2 = \begin{pmatrix} \left.\dfrac{\partial(\phi_2 + \phi_3)}{\partial G}\right|_{(G^*,H^*)} & \left.\dfrac{\partial(\phi_2 + \phi_3)}{\partial H}\right|_{(G^*,H^*)} \\ \left.\dfrac{\partial \phi_2}{\partial G}\right|_{(G^*,H^*)} & \left.\dfrac{\partial \phi_2}{\partial H}\right|_{(G^*,H^*)} \end{pmatrix},$$

where $\phi_1 = \phi_2 + \phi_3$ and ϕ_1 and ϕ_2 are defined as before. The characteristic polynomial of J_2 is

$$\lambda^2 - \lambda \left(\frac{\partial \phi_2}{\partial H} + \frac{\partial \phi_2}{\partial G} + \frac{\partial \phi_3}{\partial G} \right)\Bigg|_{(G^*,H^*)} + \left(\frac{\partial \phi_2}{\partial H}\frac{\partial \phi_3}{\partial G} - \frac{\partial \phi_2}{\partial G}\frac{\partial \phi_3}{\partial H} \right)\Bigg|_{(G^*,H^*)}.$$

It is easy to see that

$$\left.\frac{\partial \phi_3}{\partial H}\right|_{(G^*,H^*)} = -\left.\frac{\partial \phi_3}{\partial G}\right|_{(G^*,H^*)}.$$

Thus the roots of the characteristic polynomial of J_2 are

$$\lambda_1 = \left.\frac{\partial \phi_3}{\partial G}\right|_{(G^*,H^*)} = \frac{c\beta_1(1 - \tau)}{((\mu + d)(\mu + v)(R_2 - 1)/(\mu + v + d) + \mu)\, R_2}$$

and

$$\lambda_2 = \left(\frac{\partial \phi_2}{\partial G} + \frac{\partial \phi_2}{\partial H} \right)\Bigg|_{(G^*,H^*)} = \frac{1}{R_2}.$$

Therefore E_2 is locally asymptotically stable if $\max\{\lambda_1, \lambda_2\} < 1$.

It should be mentioned that another positive fixed point of Φ, which corresponds to the coexistence of the two diseases (that is, $Y_1 \neq 0, Y_2 \neq 0$), exists. However, its closed-form expression could not be found, thereby making it impossible to analyse it qualitatively at this point.

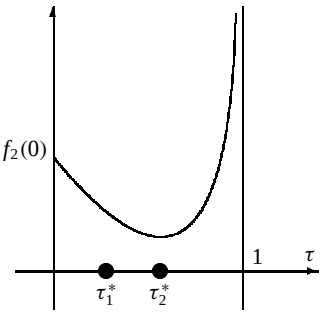

FIGURE 3. $f_2(0) = R_2 - 1 > 0$ and $\tau_1^* < \tau_2^*$.

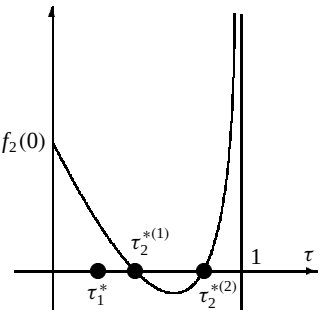

FIGURE 4. $f_2(\tau) < 0$ for some $\tau \in (0, 1)$ and $\tau_1^* < \tau_2^{*(1)} < \tau_2^{*(2)}$.

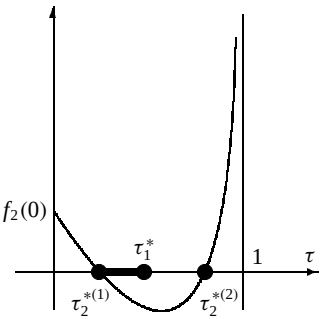

FIGURE 5. $f_2(\tau) < 0$ for some $\tau \in (0, 1)$ and $\tau_2^{*(1)} < \tau_1^* < \tau_2^{*(2)}$.

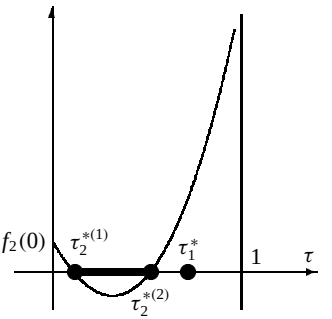

FIGURE 6. $f_2(\tau) < 0$ for some $\tau \in (0, 1)$ and $\tau_2^{*(1)} < \tau_2^{*(2)} < \tau_1^*$.

4. Effect of the treatment rate (τ)

In this section, we shall discuss the effect of the treatment rate on the stability of the three equilibria.

4.1. Effect of τ on the stability of E_0. Without loss of generality, we assume that $R_2 < 1$ (otherwise $\mathscr{R} \geq 1$ and the disease-free equilibrium is unstable). Furthermore, let $f_1(\tau) = c\beta_1(1-\tau)/\mu - 1$, for $0 \leq \tau \leq 1$. We consider the cases $f_1(0) > 0$ and $f_1(0) \leq 0$.

Case 1. Suppose $f_1(0) = c\beta_1/\mu - 1 > 0$. In this case, it is easy to see that there is a unique τ_1^* such that $f_1(\tau_1^*) = 0$ and the region of the local asymptotic stability of E_0 is where $f_1(\tau) < 0$. Thus E_0 is stable provided $\tau_1^* < \tau \leq 1$ (see Figure 1).

Case 2. Suppose $f_1(0) \leq 0$. Here $f_1(\tau) < 0$ for every $\tau \in [0, 1]$ (see Figure 2). Thus $R_1 < 1$. Hence E_0 is locally asymptotically stable. These results can be summarised in the following theorem.

THEOREM 4.1. (a) *If $c\beta_1 > \mu$, then E_0 is locally asymptotically stable whenever $\tau_1^* < \tau < 1$.*
(b) *If $c\beta_1 \leq \mu$, then E_0 is locally asymptotically stable for every $\tau \in [0, 1]$.*

4.2. Effect of τ on the stability of E_1. Define now a function $f_2(\tau)$ given by

$$f_2(\tau) = \frac{\mu R_2}{c\beta_1(1-\tau)}\left[1 + (1-\tau)\left(\frac{c\beta_1(1-\tau)}{\mu} - 1\right)\right] - 1, \quad \text{for} \quad 0 \leq \tau < 1.$$

We consider two cases as follows.
Case 1. Suppose $f_2(0) = R_2 - 1 > 0$. It is easy to see that $\lim_{\tau\to 1} f_2(\tau) = +\infty$. Further, there is a unique τ_2^* such that $f_2'(\tau_2^*) = 0$. If $f_2(\tau) > 0$ for $\tau \in (0, 1)$, then $D_2 \geq 1$ for every $\tau \in [0, 1]$ and hence E_1 is unstable (see Figure 3). Suppose that $f_2(\tau) < 0$ for some $\tau \in (0, 1)$. Then there exist $\tau_2^{*(1)}$ and $\tau_2^{*(2)}$ with $\tau_2^{*(1)} < \tau_2^{*(2)}$ such that $f_2(\tau_2^{*(1)}) = f_2(\tau_2^{*(2)}) = 0$. If $\tau_2^{*(1)} > \tau_1^*$, then $D_2 < 1$ whenever $\tau \in (\tau_2^{*(1)}, \tau_2^{*(2)})$. In this case, $D_1 > 1$ and thus E_1 is unstable (see Figure 4). If $\tau_2^{*(1)} < \tau_1^*$ and $\tau_2^{*(2)} > \tau_1^*$, then we need $\tau \in (\tau_2^{*(1)}, \tau_1^*)$ to establish the local asymptotic stability of E_1 (see Figure 5). In this case, if $\tau \in (\tau_1^*, \tau_2^{*(2)})$ ($D_2 < 1$), then $D_1 > 1$ (because here $R_1 < 1$); thus E_1 is also unstable. Finally, if $\tau_2^{*(2)} < \tau_1^*$, then E_1 is locally asymptotically stable provided $\tau \in (\tau_2^{*(1)}, \tau_2^{*(2)})$ (see Figure 6).

THEOREM 4.2. *Suppose that $R_2 > 1$.*
(a) *If $f_2(\tau) \geq 0$ for $0 < \tau < 1$ or $\tau_2^{*(1)} \geq \tau_1^*$, then E_1 is unstable for every $\tau \in (0, 1)$.*
(b) *If $\tau_2^{*(1)} < \tau_1^* \leq \tau_2^{*(2)}$, then E_1 is locally asymptotically stable whenever $\tau \in (\tau_2^{*(1)}, \tau_1^*)$.*
(c) *If $\tau_2^{*(2)} \leq \tau_1^*$, then E_1 is locally asymptotically stable whenever $\tau \in (\tau_2^{*(1)}, \tau_2^{*(2)})$.*

Case 2. Suppose $f_2(0) = R_2 - 1 \leq 0$. In this case, there is a unique $\tau_2^{*(3)}$ such that $f_2(\tau_2^{*(3)}) = 0$. If $\tau_2^{*(3)} \geq \tau_1^*$, then $D_2 < 1$ whenever $\tau \in (0, \tau_2^{*(3)})$. Thus E_1 is locally asymptotically stable if $\tau \in (0, \tau_1^*)$ (see Figure 7). If $\tau_2^{*(3)} < \tau_1^*$, then E_1 is locally asymptotically stable whenever $\tau \in (0, \tau_2^{*(3)})$ (see Figure 8).

THEOREM 4.3. *Suppose that $R_2 < 1$.*
(a) *If $\tau_2^{*(3)} \geq \tau_1^*$, then E_1 is locally asymptotically stable whenever $\tau \in (0, \tau_1^*)$.*
(b) *If $\tau_2^{*(3)} < \tau_1^*$, then E_1 is locally asymptotically stable whenever $\tau \in (0, \tau_2^{*(3)})$.*

4.3. Effect of τ on the stability of E_2. Define

$$f_3(\tau) = \frac{c\beta_1(1-\tau)}{((\mu+d)(\mu+v)(R_2-1)/(\mu+v+d)+\mu)\,R_2} - 1, \quad \text{for} \quad 0 < \tau < 1.$$

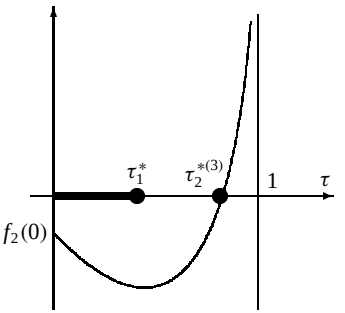

FIGURE 7. $f_2(0) = R_2 - 1 \le 0$ and $\tau_1^* \le \tau_2^{*(3)}$.

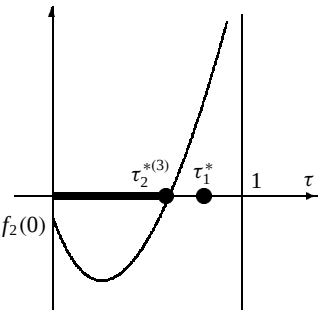

FIGURE 8. $f_2(0) = R_2 - 1 \le 0$ and $\tau_2^{*(3)} \le \tau_1^*$.

Notice that $f_3(\tau) < R_1/R_2 - 1$. It follows that if $R_1 < 1$, then E_2 is locally asymptotically stable, since $R_2 > 1$ (a necessary condition for the existence of E_2). Now we consider two cases as follows. If $f_3(0) > 0$, then there exists a unique τ_3^* such that $f_3(\tau_3^*) = 0$. Therefore E_2 is locally asymptotically stable if $\tau_3^* < \tau < 1$ (that is, $f_3(\tau) < 0$ or $\lambda_1 < 1$). Now suppose that $f_3(0) \le 0$. Then $f_3(\tau) < 0$ for every τ and thus E_2 is locally asymptotically stable for every $\tau \in [0, 1]$.

THEOREM 4.4. (a) *If $R_1<1$, then E_2 is locally asymptotically stable (for $R_2>1$).*
(b) *If $f_3(0) > 0$, then E_2 is locally asymptotically stable whenever $\tau \in (\tau_3^*, 1)$.*
(c) *If $f_3(0) \le 0$, then E_2 is locally asymptotically stable for every τ.*

5. Construction of a robust numerical method

It should be noted that the five dependent variables of the model are populations and must therefore be non-negative. Thus any discrete model (numerical method) for approximating the model given by (2.1) must satisfy the "positivity" property of the model (see [6]). To construct a scheme with such a property, we use forward-difference approximation for the derivatives in (2.1) and approximate the right-hand side functions appropriately as follows:

$$\frac{X^{n+1} - X^n}{\ell} = \Pi - \mu X^{n+1} - \left(\frac{c\beta_1 Y_1^n + c\beta_2 Y_2^n}{X^n + Y_1^n + Y_2^n + A^n + Z^n} \right) X^{n+1}, \tag{5.1a}$$

$$\frac{Y_1^{n+1} - Y_1^n}{\ell} = \frac{c\beta_1(1-\tau)\left(2Y_1^n - Y_1^{n+1}\right) X^{n+1}}{X^{n+1} + Y_1^n + Y_2^n + A^n + Z^n} - \frac{c\beta_2 Y_1^{n+1} Y_2^n}{X^{n+1} + Y_1^n + Y_2^n + A^n + Z^n} - \mu Y_1^{n+1}, \tag{5.1b}$$

$$\frac{Y_2^{n+1}-Y_2^n}{\ell}=\frac{c\beta_2 X^{n+1}\left(2Y_2^n-Y_2^{n+1}\right)}{X^{n+1}+Y_1^{n+1}+Y_2^n+A^n+Z^n}$$
$$+\frac{c\beta_2 Y_1^{n+1}\left(2Y_2^n-Y_2^{n+1}\right)}{X^{n+1}+Y_1^{n+1}+Y_2^n+A^n+Z^n}-(\mu+v)Y_2^{n+1}, \tag{5.1c}$$

$$\frac{A^{n+1}-A^n}{\ell}=vY_2^{n+1}-(\mu+d)A^{n+1}, \tag{5.1d}$$

$$\frac{Z^{n+1}-Z^n}{\ell}=\frac{c\beta_1\tau Y_1^{n+1}X^{n+1}}{X^{n+1}+Y_1^{n+1}+Y_2^{n+1}+A^{n+1}+Z^n}-\mu Z^{n+1}, \tag{5.1e}$$

where $X(t_n)=X^n$, $Y_1(t_n)=Y_1^n$, $Y_2(t_n)=Y_2^n$, $A(t_n)=A^n$, $Z(t_n)=Z^n$; $n=0,1,2,\ldots$ and $\ell>0$ is the time-step.

In (5.1b) and (5.1c), non-local implicit approximations for Y_1 and Y_2, given respectively by $Y_1^n\to 2Y_1^n-Y_1^{n+1}$ and $Y_2^n\to 2Y_2^n-Y_2^{n+1}$, have been used to ensure "positivity". Furthermore, although the equations in (5.1) are implicit in nature, they can be solved in sequence to give the following (Gauss-Seidel type) explicit formulation

$$X^{n+1}=\frac{X^n+\ell\Pi}{1+\ell\left[\mu+\dfrac{c(\beta_1Y_1^n+\beta_2Y_2^n)}{X^n+Y_1^n+Y_2^n+A^n+Z^n}\right]}, \tag{5.2}$$

$$Y_1^{n+1}=\frac{\left[1+\dfrac{2\ell c\beta_1(1-\tau)X^{n+1}}{X^{n+1}+Y_1^n+Y_2^n+A^n+Z^n}\right]Y_1^n}{1+\ell\left[\dfrac{c\beta_1(1-\tau)X^{n+1}+c\beta_2Y_2^n}{X^{n+1}+Y_1^n+Y_2^n+A^n+Z^n}+\mu\right]}, \tag{5.3}$$

$$Y_2^{n+1}=\frac{Y_2^n+\dfrac{2\ell c\beta_2Y_2^n\left(X^{n+1}+Y_1^{n+1}\right)}{X^{n+1}+Y_1^{n+1}+Y_2^n+A^n+Z^n}}{1+\dfrac{\ell c\beta_2\left(X^{n+1}+Y_1^{n+1}\right)}{X^{n+1}+Y_1^{n+1}+Y_2^n+A^n+Z^n}+\ell(\mu+v)}, \tag{5.4}$$

$$A^{n+1}=\frac{A^n+\ell vY_2^{n+1}}{1+\ell(\mu+d)}, \tag{5.5}$$

$$Z^{n+1}=\frac{Z^n+\dfrac{\ell\tau c\beta_1Y_1^{n+1}X^{n+1}}{X^{n+1}+Y_1^{n+1}+Y_2^{n+1}+A^{n+1}+Z^n}}{1+\ell\mu}. \tag{5.6}$$

It can be seen from the right-hand sides of (5.2)–(5.6) that none of the methods admits negative terms for $0<\tau<1$. Thus the method {(5.2)–(5.6)} satisfies the "positivity" requirement of the model (2.1).

TABLE 1. Effect of τ.

τ	R_1	R_2	D_1	D_2	λ_1	λ_2	Y_1^*	Y_2^*	S.E.
0	6.451	0.392	0.155	0.392	91.306	2.550	54516	0	E_1
0.2	5.161	0.392	0.194	0.329	73.045	2.250	41613	0	E_1
0.4	3.871	0.392	0.258	0.276	54.783	2.250	28710	0	E_1
0.6	2.581	0.392	0.388	0.248	36.552	2.550	15807	0	E_1
0.8	1.290	0.392	0.775	0.322	18.261	2.250	2903	0	E_1
0.85	0.968	0.392	1.033	0.403	13.696	2.550	0	0	E_0

TABLE 2. Effect of β_2 (S.E. represents stable equilibrium).

β_2	R_1	R_2	D_1	D_2	λ_1	λ_2	Y_1^*	Y_2^*	S.E.
1	0.645	78.431	1.550	100	$7.802E-5$	$1.275E-5$	0	38844	E_2
0.8	0.645	62.745	1.550	80	$1.220E-4$	$1.593E-2$	0	38750	E_2
0.6	0.645	47.059	1.550	60	$2.172E-4$	$2.125E-2$	0	38594	E_2
0.4	0.645	31.373	1.550	40	$4.900E-4$	$3.188E-2$	0	38281	E_2
0.2	0.645	15.686	1.550	20	$1.976E-3$	$6.375E-2$	0	37331	E_2
0.1	0.645	7.843	1.550	10	$8.041E-3$	0.128	0	35382	E_2
0.01	0.645	0.784	1.550	1	1.160	1.275	0	0	E_0

6. Numerical experiments

In order to test the behaviour of the numerical scheme constructed above, numerous numerical simulations were carried out. The effect of the treatment parameter τ was monitored by simulating the method using the following parameter and initial values: $\Pi = 2000$, $\mu = 0.031$, $\beta_1 = 0.05$, $\beta_2 = 0.005$, $v_2 = 0.02$, $d = 0.06$, $c = 4$, $X^0 = 120,000$, $Y_1^0 = 10$, $Y_2^0 = 70,100$, $A^0 = 80,000$, $Z^0 = 30$. The results obtained, given in Table 1, are consistent with the theoretical predictions of Sections 3 and 4. As expected, higher values of τ lead to decreasing values of Y_1^*. In line with Theorem 4.1 (a), at $\tau = \tau_1^* = 0.85$, we found that $Y_1^* = 0$, implying that the pathogen can be eradicated provided at least 85% of the pathogen-infected population is effectively treated. This eradication condition coincides with the case where both R_1 and R_2 are less than unity (so that the disease-free equilibrium is stable).

Further numerical simulations were carried out to study the effect of β_2 on the dynamics of the HIV-infected population Y_2. The parameter and initial values used in these experiments were: $\Pi = 2000$, $\mu = 0.031$, $\beta_1 = 0.01$, $\tau = 0.5$, $v = 0.02$, $d = 0.06$, $c = 4$, $X^0 = 120,000$, $Y_1^0 = 10$, $Y_2^0 = 70,100$, $A^0 = 80,000$, $Z^0 = 30$. The results are depicted in Table 2, where it is evident that as $\beta_2 \to 0$, $Y_2^* \to 0$. Thus, if an effective treatment mechanism is used to eliminate the competing pathogen from the population (making $Y_1 = 0$), then HIV eradication can be achieved in about 3 years (see Table 3 for the number of HIV infected individuals as a function of time) by (additionally) administering an active anti-retroviral therapy to the HIV-infected

TABLE 3. Dynamics of an HIV-infected population for $\beta_2 = 0.01$.

Time (days)	100	200	400	600	800	1000
HIV-infected population (Y_2)	5300	1375	140	16	2	0

population that can reduce the probability of HIV transmission below 1% (that is, make $\beta_2 \leq 0.01$).

It should be mentioned that numerical simulations reveal that although E_1 and E_2 are both locally asymptotically stable (under the conditions of Theorems 4.2–4.4), neither of the two equilibria is globally asymptotically stable. Furthermore, in all the numerical experiments carried out, the numerical method gave profiles that converged only to the correct steady-state solutions and did not suffer any scheme-dependent instability for any set of parameter values used.

7. Discussion and conclusion

A new epidemic model has been developed and analysed for the interactions between HIV and a curable pathogen. Although pathogen-infected individuals are generally assumed to be more susceptible to HIV infection (in comparison to individuals who are not infected by the curable pathogen), our model assigns the same probability of HIV infection (β_2) for both subpopulations (X and Y_1). The associated steady-state solutions of the model were found and analysed qualitatively. A novel finite-difference method, which is free of the scheme-dependent instabilities associated with the use of standard methods (such as the Euler and Runge-Kutta methods), was constructed and used to compute the solution of the model. Based on the parameter values used in our numerical simulations, the model predicts that the pathogen can be eradicated from the population if 85% of the pathogen-infected populace is effectively treated. Furthermore, with the pathogen eliminated, the model suggests that HIV eradication is feasible if an active anti-HIV therapy can reduce the HIV transmission parameter below a certain threshold. This threshold is 1% for the parameter values used in our simulations.

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