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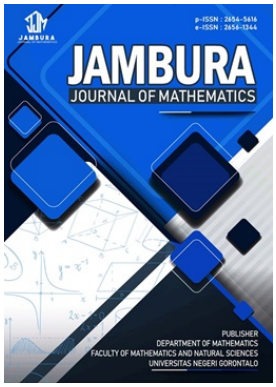

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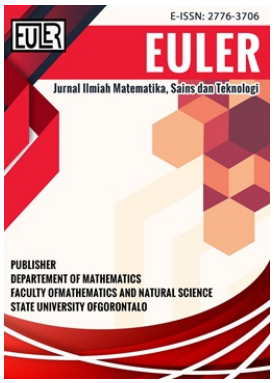

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Research Article

Predator-Prey Dynamics in the Interaction of HIV Virus with CD4+T Cells

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ABSTRACT. *This study analyzes the interaction dynamics between the human immunodeficiency virus (HIV) and CD4+T cells using a predator–prey mathematical model, in which HIV is represented as the predator and CD4+T cells as the prey. The model aims to describe the long-term behavior of the immune system when challenged by the virus. Analytical results show that the system has two equilibrium points: a disease-free equilibrium E_1 and an endemic equilibrium E_2, whose explicit forms are derived in closed form. Stability analyses of both the disease-free and endemic states are conducted through system linearization, Jacobian matrix formulation, and application of the Routh–Hurwitz criteria. The disease-free state is found to be locally asymptotically stable when the viral elimination rate by the immune system exceeds a specific threshold determined by the balance between viral infection and CD4+T cell production, indicating that under certain conditions the immune system can suppress the virus naturally. The endemic state, representing chronic infection, is stable when the combined effects of viral replication and immune response surpass the rate at which healthy CD4+T cells are lost, implying that the virus can persist within the host. Numerical simulations in Python, using parameter values from previous studies, confirm the coexistence of the virus and host cells under specific conditions. The findings emphasize the influence of viral replication and immune response rates on system stability, offering insights into how HIV can maintain chronic infection without completely depleting CD4+T cells.*

1. Introduction

Human Immunodeficiency Virus (HIV) is a virus that attacks the immune system, particularly the CD4+T cells, which play a central role in adaptive immunity. By depleting these helper T cells, HIV weakens the host's defense and increases vulnerability to opportunistic infections. Since the first identification of Acquired Immune Deficiency Syndrome (AIDS) in 1981, HIV has remained a major global health threat, with AIDS representing the terminal stage of infection [1–4]. According to the World Health Organization (WHO), HIV and sexually transmitted infections contribute to approximately 2.7 million deaths annually. In 2022, new HIV infections decreased to 1.3 million from 1.5 million, yet transmission remains concentrated in key populations, including men who have sex with men, injection drug users, sex workers, transgender individuals, and incarcerated populations [1, 3, 5]. Transmission occurs primarily through sexual contact, direct blood exposure, mother to child transmission, and shared needle use.

Beyond medical treatment, mathematical modelling has become an essential tool for understanding HIV–immune system interactions. A widely adopted framework is the predator–prey analogy, in which HIV acts as the predator that consumes CD4+T cells, the prey. This perspective captures the nonlinear interplay between viral replication and immune depletion: as viral populations grow, CD4+T cells decline, followed by cycles of recovery and collapse [6, 7]. Unlike linear models, predator–prey systems can represent feedback mechanisms, allowing for richer insights into immune response and viral persistence [8–11].

Early landmark work by Perelson et al. [12] introduced a compartmental predator–prey formulation including uninfected, latently infected, productively infected cells, and free viruses. While this model clarified viral replication rates and cell lifespans, its complexity hindered straightforward stability analysis. Building on this foundation, subsequent studies refined the framework with additional biological detail. For example, Murray [13] highlighted predator–prey–like stability mechanisms underlying viral rebound after ART interruption, while Toro-Zapata et al. [14] incorporated delayed CD4+T-cell activation, showing that bifurcations could sustain oscillatory viral loads.

Alternative approaches have employed fractional calculus to capture memory effects in infection dynamics. Silva and Torres [15] analyzed a fractional-order HIV/AIDS model with stability conditions derived from Lyapunov functions, and Carvalho et al. [16] extended such models to coinfection scenarios with HCV, showing how HIV accelerates disease progression. Mohyud-Din et al. [17] further applied semi-analytical methods to delay systems for HIV–CD4+ interactions. These studies improve biological realism but often introduce analytical intractability due to high dimensionality and extensive parameterization.

From a computational standpoint, solution techniques

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have also advanced. Attaullah et al. [18] demonstrated that Runge–Kutta, collocation, and Galerkin–Petrov schemes produce accurate results even with larger time steps. Tabassum et al. [19] developed a Padé-approximation method for HIV/AIDS models with vertical transmission, achieving faster convergence than conventional solvers. While these innovations enhance numerical efficiency, they still do not directly isolate the intrinsic dynamics of HIV–CD4+ interactions.

In contrast, the present study adopts a simplified predator–prey framework focusing only on three compartments: uninfected CD4+T cells, infected CD4+T cells, and free viruses, focusing only on the three core compartments essential for capturing the primary infection cycle: uninfected CD4+T cells, infected CD4+T cells, and free viruses. By excluding latent compartments and ART-related effects, the model emphasizes fundamental virus–immune interactions and enables clearer stability analysis. This streamlined approach builds on ecological predator–prey analogies, which have also inspired broader applications in multi-species food chain modeling [20].

The novelty of this study lies in identifying stability conditions as explicit functions of infection rate, immune regeneration, and viral replication efficiency. Rather than focusing solely on temporal viral load fluctuations, this framework highlights threshold dynamics that determine whether HIV persists or the immune system can stabilize. By integrating analytical stability analysis with numerical simulations, the study contributes a tractable yet biologically meaningful model that bridges the gap between complex compartmental systems and oversimplified linear formulations. Ultimately, these findings provide a theoretical basis for exploring intervention strategies, optimizing treatment protocols, and advancing the mathematical understanding of HIV persistence and control.

2. Model

In this section, a model will be developed based on the assumptions regarding the workings of the HIV virus. The assumptions used to create the predator-prey model are as follows:

1. There are CD4+T cells stored in the body before being infected with HIV, denoted as (s).
2. The HIV virus enters the bloodstream and does not immediately attack CD4+T cells; some of it flows within the blood circulation.
3. Some CD4+T cells die at a rate of (d) over time.
4. There are T cells that reproduce at a rate of (r), but these new CD4+T cells are born already infected with the HIV virus.
5. The HIV virus can consume CD4+T cells without any limit, allowing the virus to continue to grow exponentially.
6. The HIV virus is produced when it enters uninfected CD4+T cells, with a birth rate of (b).
7. CD4+T cells that are already infected with HIV will die when the virus attacks those cells again, causing cell destruction.
8. Infected CD4+T cells can also die naturally at a rate of (w).
9. An infected CD4+T cell will generate more HIV viruses, a process called viral duplication, quantified by (k).
10. Once the HIV virus has disseminated its DNA, it does not immediately attack CD4+T cells, which is denoted as (V) for free virus. Nonetheless, this virus will seek CD4+T cells to use as future hosts.
11. After the HIV virus duplicates, it will attack newly infected CD4+T cells, leading to a recursive nature of the virus's spread.

Based on the established assumptions, there are three different compartments: the *prey* population (T), the *predator* (T_{pi}), and *top predator* (V). In this model, the system evolves cyclically until it reaches a state where HIV and CD4+T cells coexist. During this phase, CD4+T cells proliferate at a rate r, after which they are once again 'consumed' by HIV. The CD4+T cells will not be directly eaten by HIV but will go through the infection stage first; after that, the virus will replicate and mutate, leading to the infection of other CD4+T cells. If the interaction between the HIV virus and CD4+T cells is not managed, the spread of HIV will be very rapid, and once infected, there is no possible way to cure it, although antiretroviral drugs can be used to slow down the virus's replication.

The model is a modification of the classic predator-prey model, incorporating an additional compartment to account for the unique dynamics of HIV infection. Unlike traditional predator-prey interactions, where predators directly consume prey, this model considers an intermediate stage where CD4+T cells first become infected before contributing to viral replication and spread. This approach allows for a more accurate representation of the complex interactions between uninfected T cells, infected T cells, and free viruses.

The formation of the model begins with assumptions translated into a diagram. Figure 1 shows the relationships among the three assumed compartments: T, T_{pi} and V.

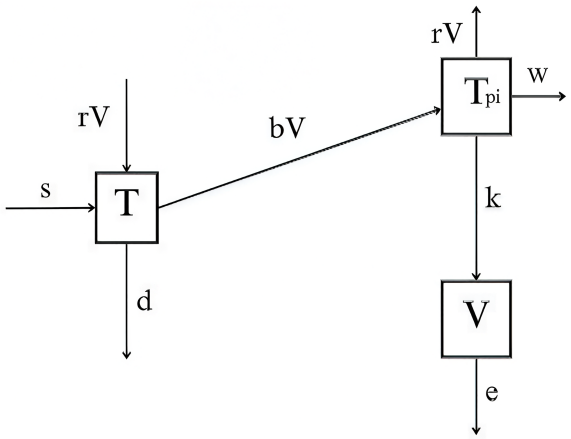

Figure 1. Predator-prey diagram compartment of HIV virus and CD4+T cells

When predation occurs, with prey being consumed at a rate of bV, the prey population will decrease. This is because when the HIV virus attacks uninfected CD4+T cells, it results in the infection of those CD4+T cells. After the HIV virus enters, the CD4+T cells will replicate to prevent the HIV virus at a rate of r. Additionally, there are uninfected CD4+T cells that die naturally at a rate of d. Thus, the rate of the CD4+T cell population over time is given by eq. (1):

$$\frac{dT}{dt} = s - dT + rTV - bTV. \tag{1}$$

The predator population will increase due to the predation

Table 1. List of variables and parameters

No.	Variables	Definition	Condition	Unit
1	$T(t)$	Total population of CD4+T cells not infected by HIV	$T(t) > 0$	cells/mm^3
2	$T_{pi}(t)$	CD4+T cells infected by HIV	$T_{pi} > 0$	cells/mm^3
3	$V(t)$	Virus that has not attacked CD4+T cells	$V(t) > 0$	clone/ml
4	s	CD4+T cells present in the body and not infected by HIV	$s > 200$	cells/mm^3
5	d	Death rate	$d > 0$	cells/mm^3
6	b	Birth rate	$b > 0$	cells/mm^3
7	w	Average death rate of infected CD4+T cells	$w > 0$	cells/mm^3
8	r	Maximum reproduction rate of CD4+T cell population	$r > 0$	cells/mm^3
9	k	Number of HIV replications within infected T cells	$k > 0$	cells/mm^3
10	e	Average number of free HIV viruses present	$e > 0$	cells/mm^3

on the prey, with predation occurring at a rate of bTV, because b denotes the birth of predators that consume the prey. The virus will continue to replicate by infecting the existing CD4+T cells. However, when the HIV virus attacks already infected the T cells, cell destruction occurs. This will reduce the predator population, where cell destruction happens at a rate of $rT_{pi}V$ and a natural decrease of the predator at rate wT_{pi}. In this model, kT_{pi} is not included as a subtraction term since k represents the number of viral copies produced by infected T cells, as viruses typically replicate in this manner. We assume that these replicated copies do not die at the moment they are produced; instead, they are immediately counted as free viruses that have not yet interacted with uninfected CD4+T cells. Hence, the predator population over time is represented by eq. (2):

$$\frac{dT_{pi}}{dt} = bTV - wT_{pi} - rT_{pi}V. \tag{2}$$

After CD4+T cells are infected by the HIV virus, the CD4+T cells will duplicate the viral DNA, then spread the viral duplication to attack uninfected CD4+T cells at a rate of k originating from the infected CD4+T cells, forming kT_{pi}. All duplicated viruses do not immediately attack CD4+T cells but remain in the bloodstream, categorized as not yet having entered the CD4+T cells, denoted by eV. This process will continuously return to the initial state, thus described as a recursive process, and there will be no CD4+T cells that recover and prevent the entry of the HIV virus. Hence, the number of virus populations growing over time is given by eq. (3):

$$\frac{dV}{dt} = kT_{pi} - eV. \tag{3}$$

This model is formed with the addition of a reproductive cycle, assuming that CD4+T cells will proliferate to combat the virus. The CD4+T cells will work optimally, resulting in cell destruction when infected CD4+T cells encounter the HIV virus again. By combining the resulting equations with the existing parameters and variables, we have model (4):

$$\begin{aligned} \frac{dT}{dt} &= s - dT + rTV - bTV, \\ \frac{dT_{pi}}{dt} &= bTV - wT_{pi} - rT_{pi}V, \\ \frac{dV}{dt} &= kT_{pi} - eV. \end{aligned} \tag{4}$$

3. Results and Discussion

3.1. Existence of Equilibrium Points

Model development of the HIV virus with CD4+T cells has the form of linear differential equation models. The equilibrium points of the model (4) are obtained by setting the first derivatives equal to zero, i.e. $\frac{dT}{dt} = \frac{dT_{pi}}{dt} = \frac{dV}{dt} = 0$. Then we have

$$s - dT + rTV - bTV = 0, \tag{5}$$
$$bTV - wT_{pi} - rT_{pi}V = 0, \tag{6}$$
$$kT_{pi} - eV = 0. \tag{7}$$

From eq. (5), we obtain:

$$T = \frac{s}{d - rV + bV}. \tag{8}$$

From eq. (7), we obtain:

$$T_{pi} = \frac{eV}{k}. \tag{9}$$

For the first equilibrium point, we can take the case when there are no viruses attacking, i.e., when $V = 0$. Substituting $V = 0$ into eq. (5) and eq. (6), we obtain:

$$T = \frac{s}{d} \text{ and } T_{pi} = 0.$$

Thus, the equilibrium point, which represents the state with no virus attack, is:

$$E_1 = \left(\frac{s}{d}, 0, 0\right). \tag{10}$$

The first equilibrium represents a state in which there are only uninfected CD4+T cells, while the population of infected CD4+T cells and free HIV virus is entirely absent. From a health perspective, this equilibrium corresponds to an ideal scenario where the immune system is functioning normally without any HIV infection. In this state, the body continuously regenerates CD4+T cells at a rate s and these cells undergo natural cell death at a rate d, maintaining a stable and healthy immune system without external disturbances.

The stability of this equilibrium depends on whether external viral exposure occurs. If an individual remains unexposed to HIV, the immune system remains in equilibrium at E_1, where CD4+T cells exist without any infection. However, once the virus enters the body and starts infecting CD4+T cells, the system shifts away from this equilibrium and moves toward a coexistence state where both infected and uninfected cells, as well as

free viral particles, are present. This transition occurs because HIV targets and integrates itself into CD4+T cells, leading to a chain reaction where more cells become infected, ultimately disrupting the balance maintained at E_1.

Next, we seek the equilibrium point when the virus is present, i.e., when $V > 0$. Substituting eq. (9) into eq. (6), we obtain:

$$bTV - w\frac{eV}{k} - r\frac{eV}{k}V = 0,$$

which can be simplified to:

$$bT - \frac{we}{k} - \frac{reV}{k} = 0. \tag{11}$$

Substituting eq. (8) into eq. (11), we get:

$$\begin{aligned}
\frac{bs}{d - rV + bV} - \frac{we}{k} - \frac{reV}{k} &= 0,\\
\frac{bsk - we\left(d - rV + bV\right) - reV\left(d - rV + bV\right)}{\left(d - rV + bV\right)k} &= 0,\\
bsk - we\left(d - rV + bV\right) - reV\left(d - rV + bV\right) &= 0,\\
bsk - wed + werV - webV - redV + r^2eV^2 - rebV^2 &= 0,\\
\left(bsk - wed\right) + \left(wer - web - red\right)V + \left(r^2e - reb\right)V^2 &= 0,\\
\left(bsk - wed\right) + \left(we\left(r - b\right) - red\right)V + re\left(r - b\right)V^2 &= 0.
\end{aligned} \tag{12}$$

Eq. (12) represents a quadratic equation in the variable V, which can be solved using the quadratic formula, yielding:

$$V_{12} = \frac{-\left(we\left(r - b\right) - red\right) \pm \sqrt{\Delta}}{re\left(r - b\right)}. \tag{13}$$

where:

$$\Delta = \left(we\left(r - b\right) - red\right)^2 - 4re\left(r - b\right)\left(bsk - wed\right).$$

Since V indicates the quantity of viruses, its value must be positive and real. The value of V is a positive real number if: $we\left(r - b\right) - red < 0 \Leftrightarrow r < b$ and $\left(we\left(r - b\right) - red\right)^2 - 4re\left(r - b\right)\left(bsk - wed\right) \geq 0 \Leftrightarrow \left(we\left(r - b\right) - red\right)^2 \geq 4re\left(r - b\right)\left(bsk - wed\right)$.

Thus, the equilibrium point when a virus is attacking is given by:

$$E_2 = \left(T^*, {T_{pi}}^*, V^*\right) = \left(\frac{s}{d - rV^* + bV^*}, \frac{eV^*}{k}, V^*\right), \tag{14}$$

where:
$V^* = \frac{-\left(we\left(r-b\right)-red\right)+\sqrt{\left(we\left(r-b\right)-red\right)^2-4re\left(r-b\right)\left(bsk-wed\right)}}{re\left(r-b\right)}, r < b$, and $\left(we\left(r - b\right) - red\right)^2 \geq 4re\left(r - b\right)\left(bsk - wed\right)$.

The second equilibrium point represents a biological state in which HIV persists within the body while CD4+T cells are continuously replenished. The presence of a stable V^* signifies that the virus remains active, constantly infecting new CD4+T cells, which are represented by T_π^*, while the immune system continues to regenerate uninfected CD4+T cells, represented by T^*. The inequality conditions ensure that the viral load does not become negative or imaginary, meaning that the virus maintains a real, positive presence in the system.

From a health perspective, this equilibrium corresponds to the chronic phase of HIV infection, where the virus and immune system reach a balance. The immune system is still functional, producing new CD4+T cells, but it is unable to clear the infection completely. The stability of this coexistence is determined by factors such as the rate of CD4+T cell production (s), their natural death rate (d), the infection rate (b), and the replication rate of the virus (k).

3.2. *The Stability of Equilibrium Points*

The stability of the equilibrium points is investigated through the results of linearization around the equilibrium points in the system of eq. (3) to obtain the Jacobian matrix:

$$J = \begin{pmatrix} \frac{\partial f}{\partial T} & \frac{\partial f}{\partial T_{pi}} & \frac{\partial f}{\partial V} \\ \frac{\partial g}{\partial T} & \frac{\partial g}{\partial T_{pi}} & \frac{\partial g}{\partial V} \\ \frac{\partial h}{\partial T} & \frac{\partial h}{\partial T_{pi}} & \frac{\partial h}{\partial V} \end{pmatrix},$$

where:

$$\begin{aligned} f &= s - dT + rTV - bTV,\\ g &= bTV - wT_{pi} - rT_{pi}V,\\ h &= kT_{pi} - eV. \end{aligned} \tag{15}$$

The elements of the Jacobian matrix near the equilibrium point E are obtained by differentiating the eq. (15) with respect to T, T_{pi} and V.

$$J(E) = \begin{pmatrix} V\left(r - b\right) - d & 0 & T\left(r - b\right) \\ Vb & -Vr - w & bT - rT_{pi} \\ 0 & k & -e \end{pmatrix}. \tag{16}$$

Theorem 1. *The equilibrium point $E_1 = \left(\frac{s}{d}, 0, 0\right)$ of model (4) is asymptotically stable if $we > \frac{bs}{d}k$.*

Proof. The Jacobian matrix at the first equilibrium point $E_1 = \left(\frac{s}{d}, 0, 0\right)$:

$$J\left(E_1 = \left(\frac{s}{d}, 0, 0\right)\right) = \begin{pmatrix} -d & 0 & \frac{s(r-b)}{d} \\ 0 & -w & \frac{bs}{d} \\ 0 & k & -e \end{pmatrix}.$$

The characteristic equation for $J\left(E_1 = \left(\frac{s}{d}, 0, 0\right)\right)$ is

$$\begin{aligned}
&\begin{vmatrix} -d - \lambda & 0 & \frac{s(r-b)}{d} \\ 0 & -w - \lambda & \frac{bs}{d} \\ 0 & k & -e - \lambda \end{vmatrix} = 0\\
\Leftrightarrow &\left(-d - \lambda\right)\left(-w - \lambda\right)\left(-e - \lambda\right) - \left(-d - \lambda\right)\frac{bs}{d}k = 0\\
\Leftrightarrow &\left(-d - \lambda\right)\left(\left(-w - \lambda\right)\left(-e - \lambda\right) - \frac{bs}{d}k\right) = 0\\
\Leftrightarrow &\left(-d - \lambda\right)\left(\lambda^2 + \left(w + e\right)\lambda + we - \frac{bs}{d}k\right) = 0.
\end{aligned}$$

The equation can be satisfied by two conditions:

1. $\left(-d - \lambda\right) = 0,$

2. $\lambda^2 + (w+e)\,\lambda + we - \frac{bs}{d}k = 0.$

From the first condition we have

$$(-d-\lambda) = 0 \Rightarrow \lambda = -d < 0.$$

From the second condition we have

$$\lambda^2 + (w+e)\,\lambda + we - \frac{bs}{d}k = 0.$$

The quadratic equation has negative real roots or complex conjugates with negative real parts if $\lambda_1 + \lambda_2 < 0$ and $\lambda_1\lambda_2 > 0$:

$$\lambda_1 + \lambda_2 = -(w+e) < 0, \text{ and}$$
$$\lambda_1\lambda_2 = we - \frac{bs}{d}k > 0 \Leftrightarrow we > \frac{bs}{d}k.$$

Hence, the equilibrium point $E_1 = \left(\frac{s}{d}, 0, 0\right)$ is stable if $we > \frac{bs}{d}k$. □

The stability condition for $E_1 = \left(\frac{s}{d}, 0, 0\right)$ indicates that the infection-free equilibrium can persist if the immune system is strong enough to control viral spread. From a health perspective, this means that if the immune response (determined by w and e) is greater than the virus's ability to infect new cells $\left(\frac{bs}{d}k\right)$, then the infection cannot sustain itself in the body and will eventually be eliminated. Conversely, if the virus has a higher infection rate than the immune system's effectiveness, the infection-free equilibrium becomes unstable, leading to a persistent infection. This reflects how the balance between immune response and infection rate determines whether an individual remains healthy or develops a chronic disease.

Theorem 2. *The equilibrium point $E_2 = (T^*, {T_{pi}}^*, V^*)$ of model (4) is asymptotically stable if $r < b$ and $(V^*r + w)\,e > (bT^* - r{T_{pi}}^*)\,k$.*

Proof. The Jacobian matrix at the second equilibrium point $E_2 = (T^*, {T_{pi}}^*, V^*)$:

$$J(E_2) = \begin{pmatrix} V^*(r-b) - d & 0 & T^*(r-b) \\ V^*b & -V^*r - w & bT^* - r{T_{pi}}^* \\ 0 & k & -e \end{pmatrix}.$$

The characteristic equation for $J(E_2 = (T^*, {T_{pi}}^*, V^*))$ is:

$$\begin{vmatrix} V^*(r-b) - d - \lambda & 0 & T^*(r-b) \\ V^*b & -V^*r - w - \lambda & bT^* - r{T_{pi}}^* \\ 0 & k & -e - \lambda \end{vmatrix} = 0,$$
$$\Leftrightarrow (V^*(r-b) - d - \lambda)(-V^*r - w - \lambda)(-e - \lambda) - (V^*(r-b) - d - \lambda)(bT^* - r{T_{pi}}^*)\,k = 0,$$
$$\Leftrightarrow (V^*(r-b) - d - \lambda)((-V^*r - w - \lambda)(-e - \lambda) - (bT^* - r{T_{pi}}^*)\,k) = 0,$$
$$\Leftrightarrow (V^*(r-b) - d - \lambda)\left(\lambda^2 + (V^*r + w + e)\,\lambda + (V^*r + w)\,e - (bT^* - r{T_{pi}}^*)\,k\right) = 0.$$

The equation can be satisfied by these conditions:

1. $(V^*(r-b) - d - \lambda) = 0,$

$$\Rightarrow \lambda_1 = V^*(r-b) - d < 0 \Leftrightarrow r < b.$$

2. $\lambda^2 + (V^*r + w + e)\,\lambda + (V^*r + w)\,e - (bT^* - r{T_{pi}}^*)\,k = 0.$

From the first condition:

$$(V^*(r-b) - d - \lambda) = 0 \Rightarrow \lambda_1 = V^*(r-b) - d < 0 \Leftrightarrow r < b.$$

From the second condition:

$$\lambda^2 + (V^*r + w + e)\,\lambda + (V^*r + w)\,e - (bT^* - r{T_{pi}}^*)\,k = 0.$$

The quadratic has negative real roots or complex conjugates with negative real parts if $\lambda_1 + \lambda_2 < 0$ and $\lambda_1\lambda_2 > 0$.

1. $\lambda_1 + \lambda_2 = -(V^*r + w + e) < 0,$
2. $\lambda_1\lambda_2 = (V^*r + w)\,e - (bT^* - r{T_{pi}}^*)\,k > 0$
   $\Leftrightarrow (V^*r + w)\,e > (bT^* - r{T_{pi}}^*)\,k.$

Thus, the equilibrium point $E_2 = (T^*, {T_{pi}}^*, V^*)$ is stable if $r < b$ and $(V^*r + w)\,e > (bT^* - r{T_{pi}}^*)\,k$. □

The stability condition of the equilibrium $E_2 = (T^*, {T_{pi}}^*, V^*)$ in the HIV-CD4+T cell model provides a mathematical representation of how the immune system and the virus can coexist in a controlled state. From a health perspective, this equilibrium signifies a chronic stage of HIV infection, where the immune system continues to function despite the presence of the virus. It represents a state where the immune system and virus are in balance, preventing both immune collapse and uncontrolled viral growth. This insight is useful for designing therapies aimed at maintaining this equilibrium through controlled viral suppression and immune support.

3.3. Numeric Simulation

Next, numerical simulations will be conducted at each equilibrium point of the System of eq. (3) and simulations for variations of several parameters. The simulations in this model use Python. The parameter values in this simulation are based on parameters from various studies regarding the Predator-Prey model of the HIV virus. For the prey population, it is represented by the number of uninfected CD4+T cells, using research data from Shanghai, China [21], which investigates control measures against the HIV virus infecting individuals there. The parameter values used in this study are as follows:

1. The average prey population in the body is 1000 cells/m³. Due to this population, births and deaths will not lead to exponential growth.
2. The rate of new CD4+T cell birth is $s = 10$ cells/mm³, and the mortality rate is $d = 0.01$ cells/mm³.
3. The birth rate between prey and predator is denoted by $b = 0.00001$ cells/mm³.
4. There is a mortality rate for already infected CD4+T cells set at $w = 0.2$ cells/mm³.
5. As CD4+T cells continue to reproduce, there will be a birth constraint for this population, i.e., $r = 0.000001$ cells/mm³.

When the virus duplicates its genetics, it duplicates by $k = 10$ cells/mm³, resulting in ten HIV duplicates that will be spread. However, not all of these duplicated viruses will enter the uninfected CD4+T cells; some remain in the bloodstream. Thus, there is a level of virus that has not entered uninfected CD4+T cells set at$e = 0.3$ cells/mm³.

Table 2. Parameters values

No.	Parameter	Value
1	s	10
2	d	0.01
3	b	0.00001
4	w	0.2
5	r	0.000001
6	k	10
7	e	0.3

For initial values, predictions at the equilibrium point are taken from Kirscher's [10], which describes the conditions of individuals who have not yet contracted HIV and those who have. Thus:

$$T\left(0\right)=1000,\ T_{pi}\left(0\right)=0, V\left(0\right)=0.$$

After that, numerical simulations are performed at the equilibrium point $E_1 = \left(\frac{s}{d},\ 0,\ 0\right)$ to understand the behavior of the System of eq. (3) around that point.

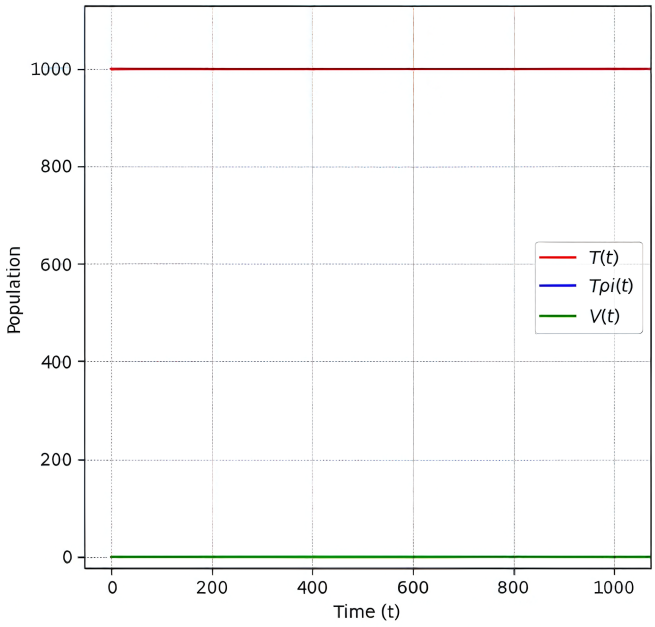

Figure 2. Growth of HIV and CD4+T cell populations when there is no virus

From Figure 2, we can see that at this equilibrium point nothing happens because there is no HIV virus attacking CD4+T cells, which mean without a virus, the population of CD4+T cells will not decrease. If we assume a person has a weak autoimmune system, then its value will not remain constant at 1000, but rather will stabilize below 1000. The changes in predator-prey populations at this equilibrium point are influenced by the birth rate of CD4+T cells and their death rate. This equilibrium point explains the ratio between the production rate and the death rate, which represents the maximum capacity of CD4+T cell population. The higher the ratio, the larger the CD4+T cell population can be sustained.

Now, numerical simulations are conducted at the equilibrium point $E_2 = (T^*, {T_{pi}}^*, V^*) = \left(\frac{s}{d-rV^*+bV^*}, \frac{eV^*}{k}, V^*\right)$, this point explains the relationship between the three variables. Suppose we use the values above; plugging in all values means the stability point will be where CD4+T cells decrease, leading to an increase in the virus population, while the infected CD4+T cells also grow over time. This equilibrium point explains that the predator and prey have a mutual relationship, assuming a predator attacks the prey, then the initial point values will change to:

$$T\left(0\right)=1000,\ T_{pi}\left(0\right)=0, V\left(0\right)=0.3.$$

Assuming the virus enters the body of an individual with $V(0) = 0.3$, and the virus has not yet attacked the CD4+T cells, the simulation graphic is depicted in Figure 3.

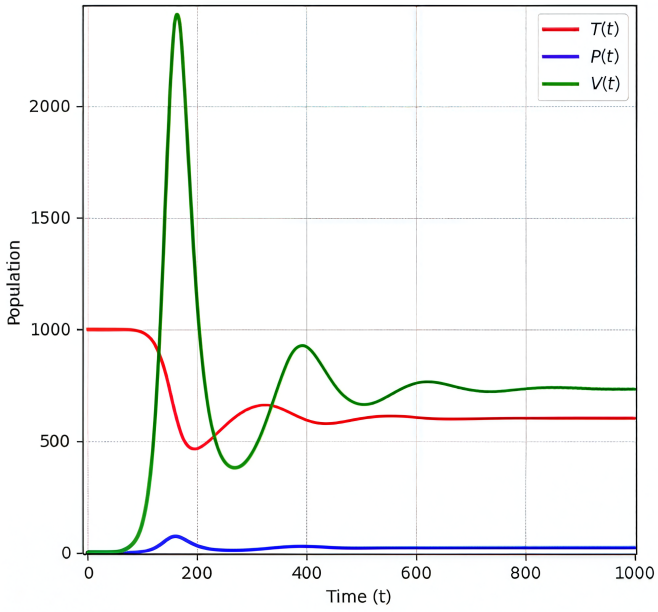

Figure 3. Growth of HIV and CD4+T cell populations when there is a virus

In Figure 3, it shows that when the predator attacks the prey, the population of uninfected CD4+T cells will immediately decrease while the population of infected CD4+T cells will increase. Over time, the HIV virus will continue to attack and grow higher. On day 100, the infected CD4+T cells increase only slightly; however, after one month, the infected CD4+T cells begin to proliferate and start duplicating their DNA. At that point, the population of uninfected CD4+T cells will decrease rapidly, indicating that the autoimmune system will continue to decline. The equilibrium point on the graph is provided in the results below.

$$[T = 602.2019087,\ T_{pi} = 22.01908726,\ V = 733.9695753].$$

After the graph reaches the equilibrium point, it will not return to that point because, according to the eigenvalues obtained, this equilibrium point is unstable. What influences the development of the virus is the number of duplications produced by the virus; the larger the value of k, the greater the amount of virus produced, and the number of infected T cells will also increase.

4. Conclusion

This predator-prey mathematical model provides a framework for understanding the interaction between the HIV virus

and CD4+T cells under various conditions. The model demonstrates that while the introduction of antiretroviral drugs can slow viral replication, it does not lead to the complete eradication of the virus. Through mathematical analysis, two equilibrium points were identified, one representing an uninfected state and another where both the virus and immune cells coexist. Stability conditions for these equilibrium points were examined, and numerical simulations confirmed that under certain parameter values, the system can reach a stable state without complete extinction of either population.

This predator-prey mathematical model will continue without any disturbances. One factor that can affect the prey is the administration of antiretroviral drugs, which can reduce the duplication of the HIV virus, but the virus will still not disappear. In this model, two types of equilibrium points are obtained $E_1 = \left(\frac{s}{d},\ 0,\ 0\right)$ and $E_2 = (T^*, {T_{pi}}^*, V^*) = \left(\frac{s}{d-rV^*+bV^*}, \frac{eV^*}{k}, V^*\right)$, The first equilibrium point is stable. Thus, the equilibrium points $E_1 = \left(\frac{s}{d}, 0, 0\right)$ is stable if $we > \frac{bs}{d}k$. Meanwhile, the second equilibrium point is stable if $r < b$ and $(V^*r + w)\, e > (bT^* - r{T_{pi}}^*)\, k$. Numerical simulations were conducted using Python with parameter values taken from research related to the HIV virus predator-prey model, resulting in the equilibrium point E_2 where the prey population (CD4+T cells) and predator (HIV virus) remain alive. This population does not experience extinction at a certain time.

These findings highlight the persistent nature of HIV infection and the critical balance between viral replication and immune response. Future research could expand this model by incorporating additional biological factors, such as immune activation, drug resistance, or latency mechanisms, to enhance its predictive power and relevance in medical applications.

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