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Dynamics and Simulation of a Within-host HIV Infection Model of CD4⁺ T-Cells with Variable Source Term and Saturated Incidence

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ABSTRACT
A nonlinear within-host HIV model of CD4+ T cells is analysed. The model incorporates a variable CD4⁺ T-cell source term and Holling type II saturated incidence. This accounts for immune exhaustion, finite receptor availability, and physiological limitations in T-cell proliferation, thereby extending classical mass-action formulations. We derive the basic reproduction number R0 and is shown to govern the dynamics of the virus. When R0<1, the infection-free equilibrium is globally asymptotically stable; when R0>1, the system exhibits a locally stable infected equilibrium representing a high level of infection. Local stability of the infection-free equilibrium is investigated via the Routh–Hurwitz criterion, while Lyapunov functional techniques are employed to establish global stability results. Existence and local stability of the infected equilibrium are also demonstrated. Numerical simulations produced clinically observed HIV dynamics. This shows that adding biologically inspired mechanisms improves the within-host HIV models' clinical interpretability and mathematical stability.

KEYWORDS: CD4⁺ T cells; HIV dynamics; Immune exhaustion; Saturated incidence; Stability analysis.

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INTRODUCTION
Human immunodeficiency virus (HIV) continues to be a major global health challenge to date. Approximately 40 million people are living with the virus worldwide [1]. HIV specifically targets the CD4+ T cells (T cells), which are also known as T helper cells. The virus orchestrates adaptive immune responses, leading to progressive immunodeficiency leading to the development of acquired immunodeficiency syndrome (AIDS). To develop effective therapies and to forecast the disease progression, it is essential to understand the complex interplay of virus replication, T cell loss, and viral-immune interactions at the within-host level. Mathematical models have been critical in unravelling HIV pathogenesis, estimating biologically relevant parameters from clinical data, and assessing intervention strategies. The earliest deterministic within-host HIV model was presented in [2] and expresses the dynamics between healthy, infected, and HIV virions in the bloodstream. Various extensions have added other biological features, including time delays [3-5], cell-to-cell transmission [6-9], immune response [10-12], and antiretroviral treatment [14-16], which have greatly extended our knowledge of the HIV pathogenesis.

Even though classical models of mass-action (bilinear) incidence assumption have been successful in predicting underlying HIV resistance dynamics, they suffer from important biological constraints. A saturation of viral entry, immune clearance, and cell-virus binding are believed to have limited the maximal viral load; this is because there are only so many receptors for the virus to bind to (i.e., restricted receptor availability) or immunological resources in the form of cells (e.g., T cells; finite capacity), such that competition during binding results [16]. Most conventional within-host HIV models depend on mass-action infection. However, this assumption is biologically constraining. To address the limitations of these models, various authors have proposed models to capture this characteristic. Numerous studies have discussed SIR models with saturated incidence, as in [5], [17], [18]. This gave rise to improved HIV models with saturation, as studied by [19-25]. The production of new T cells has been observed to diminish with increasing viral load. Experimental evidence demonstrates that thymic output declines significantly with increasing viral burden, indicating immune exhaustion and lymphoid tissue damage [26]. Models that use non-variable source terms fail to capture the declining feedback between viral load and immunological regeneration, necessitating variable source term formulations wherein T-cell recruitment is mostly modelled as a decreasing function of viral load. Models with variable source terms have T cell generation as a decreasing function. Studies that worked on models with variable source terms include [12], [13], [28-32], [35]. Several models have also incorporated multiple features, such as fusion effect, cure effect, and more, to address realism [14], [35-36].

Although saturated incidence and variable CD4⁺ T-cell source terms have been studied separately, few works have analysed models that integrate both mechanisms. This work extends within-host HIV modelling by incorporating additional biological mechanisms. We present the impact of saturated incidence and variable source term on the behaviour of the proposed model.

The source term captures immune exhaustion, the logistic constraint enforces finite lymphoid tissue capacity, and the Holling type II incidence reflects receptor saturation at high viral loads. This model analyses the population dynamics of T cells in the presence and absence of HIV. It studies infection patterns and predicts disease progression trajectories. It provides information regarding threshold conditions for viral clearance versus persistent infection and demonstrates that saturation mechanisms reduce viral overshoot and eliminate spurious oscillations compared to mass-action models, thereby offering more reliable predictions of viral load dynamics.

MODEL EQUATIONS

Mathematical Model

We present three state variables describing the model dynamics: the number of healthy T cells, infected T cells, and HIV virions, denoted as H, I, and V, respectively. We propose the nonlinear system:

$$\dot{H}=\frac{s}{1+V}+rH\left(1-\frac{H+I}{H_{max}}\right)-\delta_H H-\frac{\beta VH}{1+\alpha V}-\frac{\phi VH}{1+\alpha V}+\rho I,$$
$$\dot{I}=\frac{\beta VH}{1+\alpha V}-(\rho+\delta_I)I,$$
$$\dot{V}=A\omega I-\frac{\phi VH}{1+\alpha V}-cV.$$

with $H(0)=H_0>0, I(0)=I_0\geq 0, V(0)=V_0\geq 0.$ (2)

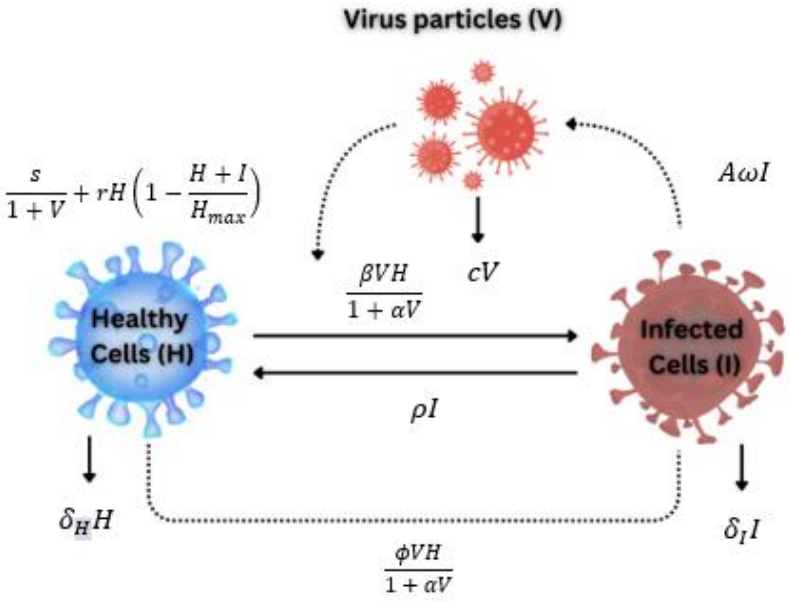

Figure 1: Diagram of the HIV model

The term $\frac{s}{1+V}$ models' immune exhaustion, which reflects the observed suppression of T-cell production at high virus concentration. This captures the negative feedback between viral burden and thymic output observed in clinical studies [26], [32], [34]. The nonlinear terms $\frac{\beta VH}{1+\alpha V}$ and $\frac{\phi VH}{1+\alpha V}$ are of Holling type II form and capture the saturation of viral entry and immune-mediated viral neutralization when the viral burden becomes large [16]. The parameter α measures the half-saturation constant: when $V=\frac{1}{\alpha}$. The infection and neutralization rates reach half of their maximum values. The logistic growth term $rH\left(1-\frac{H+I}{H_{max}}\right)$ enforces a physiological bound on the total T-cell population.

Table 1: Model Parameters and Units

Parameter	Unit	Biological Meaning
s	cells mm^{-3} day^{-1}	$CD4^+$ recruitment rate
δ_h	day^{-1}	Death rate of healthy T cells
δ_i	day^{-1}	Death rate of Infected T cells
r	day^{-1}	T cell proliferation rate
H_{max}	mm^{-3}	T cell carrying capacity

β	virions^{-1} mm^3 day^{-1}	Infection rate
α	constant	Saturation parameter
φ	virions^{-1} mm^3 day^{-1}	Viral neutralization rate
ρ	day^{-1}	Infected cell recovery rate
A	virions/cell	Virions per infected cell
c	day^{-1}	Viral clearance rate
ω	dimensionless	Viral production efficiency

Positivity of Solutions

Theorem For any initial condition satisfying (2.2), the solution $(H(t),I(t),V(t))$ of (2.1) satisfies

$$H(t)>0,I(t)\geq 0,V(t)\geq 0,\forall\, t\geq 0.$$

Proof: For H: If $H(t_0)=0$ while $I,V\geq 0$, then $\left.\frac{dH}{dt}\right|_{\{H=0\}}=s(V)\geq s_0>0$

Thus, H cannot decrease from zero. Similarly, for I and V.

Given that the right-hand side is locally Lipschitz in $\mathbb{R}_+^3$, solutions with positive initial conditions remain positive for all $t\geq 0$.

Boundedness

Let $N(t)=H(t)+I(t)$. By finding the sum of the first and second equations of (2.1), we get

$$\frac{dN}{dt}=\frac{s}{1+V}+rH\left(1-\frac{N}{H_{\max}}\right)-\delta_H H-\delta_I I-\frac{\phi VH}{1+\alpha V} \tag{2.3}$$

Using $\frac{s}{1+V}\leq s$, $H\leq N$, and $\frac{\phi VH}{1+\alpha V}\geq 0$, we obtain

$$\frac{dN}{dt}\leq s+rN\left(1-\frac{N}{H_{\max}}\right)-\delta_{\min}N \tag{2.4}$$

where $\delta_{\min}=\min_{f_0}\{\delta_H,\delta_I\}>0$. The scalar comparison ODE $\dot{y}=g(y)$ with $g(y)=s+ry\left(1-\frac{y}{H_{\max}}\right)-\delta_{\min}y$

has a globally attracting equilibrium $N^*>0$. Thus $N(t)$ is uniformly bounded.

For the virus,

$$\frac{dV}{dt}\leq A\omega N-cV\leq A\omega H_{\max}-cV,$$

So, $V(t)$ is bounded above by the solution $\dot{z}=A\omega H_{\max}-cz$, converging to $\frac{A\omega H_{\max}}{c}$.

Theorem All trajectories of (2.1) enter and remain in the compact positively invariant set

$$\Omega=\{(H,I,V)\in\mathbb{R}_+^3:\, H+I\leq M,\, V\leq\frac{A\omega H_{\max}}{c}\},$$

for some finite $M>0$.

Proof: Define $N(t)=H(t)+I(t)$. Adding the first two equations of system (2.1) and cancelling the $\frac{\beta VH}{1+\alpha V}$terms yields

$$\frac{dN}{dt}=\frac{s}{1+V}+rH\left(1-\frac{N}{H_{\max}}\right)-\delta_H H-\frac{\phi VH}{1+\alpha V}-\delta_I I.$$

Since $\frac{s}{1+V}\leq s$, $\frac{\phi VH}{1+\alpha V}\geq 0$, and $H\leq N$, we obtain

$$\frac{dN}{dt}\leq s+rN\left(1-\frac{N}{H_{\max}}\right)-\delta_{\min}N,$$

where $\delta_{\min}=\min_{f_0}\{\delta_H,\delta_I\}>0$. The scalar comparison ODE $\dot{y}=s+(r-\delta_{\min})y-\frac{r}{H_{\max}}y^2$ has a unique positive equilibrium

$$M_1=\frac{H_{\max}}{2r}\left[(r-\delta_{\min})+\sqrt{\left(r-\delta_{\min}\right)^2+\frac{4rs}{H_{\max}}}\right]<\infty.$$

By standard comparison, $N(t)\leq\max_{f_0}\{N(0),M_1\}$ for all $t\geq 0$.

For the virus, since $\frac{\phi VH}{1+\alpha V}\geq 0$and $I\leq N\leq M_1$,

$$\frac{dV}{dt}=A\omega I-\frac{\phi VH}{1+\alpha V}-cV\leq A\omega M_1-cV.$$

The comparison ODE $\dot{z}=A\omega M_1-cz$ has equilibrium $M_2=\frac{A\omega M_1}{c}$, so $V(t)\leq\max_{f_0}\{V(0),M_2\}$ for all $t\geq 0$.

Therefore, the compact set $\Omega=\{(H,I,V)\in\mathbb{R}_+^3:H+I\leq M_1,\ V\leq M_2\}$ is positively invariant and attracts all solutions of system (2.1). □

Disease- Free Equilibrium

Setting $I=V=0$ in (2.1) yields

$$0=s+rH\left(1-\frac{H}{H_{\max}}\right)-\delta_H H, \tag{2.5}$$

which is quadratic in H:

$$\frac{r}{H_{\max}}H^2-(r-\delta_H)H-s=0$$

The positive root is

$$H^* =\frac{H_{\max}}{2r}((r-\delta_H)+\sqrt{(r-\delta_H)^2+\frac{4rs}{H_{\max}}} \qquad (2.6)$$

The infection-free equilibrium is expressed as $E_0=(H^*, 0, 0)$. (2.7)

STABILITY ANALYSIS

Basic Reproduction Number

The basic reproduction number R_0 is derived via the next-generation matrix method [35]. Following this framework, we partition the state space into infected compartments (I, V) and uninfected compartments (H). The next-generation matrix is constructed with respect to the infected subsystem only, since H represents the susceptible population and does not directly contribute to disease transmission.

Let $x = (I, V, H)^T$ denote the full state vector. For the stability analysis, we focus on the infected subsystem (I, V), with H treated as a dynamic but non-infected variable. System (1) is expressed as follows:

$$\frac{dx}{dt}=\chi(x)-\psi(x),$$

where $\chi(x)$ has the terms describing new infections and $\psi(x)$ collects remaining transitions. For system (1), we identify:

$$\chi(x)=\begin{pmatrix}\frac{\beta VH}{1+\alpha V}\\0\\0\end{pmatrix},\ \psi(x)=\begin{pmatrix}(\rho+\delta_I)I\\\frac{\phi VH}{1+\alpha V}+cV-A\omega I\\\delta_H H-\frac{s}{1+V}-rH\left(1-\frac{H+I}{H_{\max}}\right)-\rho I\end{pmatrix}$$

Linearizing at $E_0=(0,0,H^*)$ gives the Jacobian matrices of χ and ψ with respect to the infected subsystem (I, V):

$$F=\begin{pmatrix}0 & \beta H^*\\0 & 0\end{pmatrix},V=\begin{pmatrix}\rho+\delta_I & 0\\-A\omega & c+\phi H^*\end{pmatrix}$$

The inverse of V is

$$V^{-1}=\frac{1}{(\rho+\delta_I)(c+\phi H^*)}\begin{pmatrix}c+\phi H^* & 0\\A\omega & \rho+\delta_I\end{pmatrix}$$

The next-generation matrix method is

$$FV^{-1}=\frac{1}{(\rho+\delta_I)(c+\phi H^*)}\begin{pmatrix}\beta H^* A\omega & \beta H^*(\rho+\delta_I)\\0 & 0\end{pmatrix}$$

Hence, the basic reproduction number is

$$R_0=\frac{\beta H^* A\omega}{(\rho+\delta_I)(c+\phi H^*)} \qquad (4)$$

Local Stability of the Disease-Free Equilibrium

Theorem The disease-free equilibrium E_0 is locally asymptotically stable if $R_0<1$ and unstable if $R_0>1$.

Proof: We evaluate the Jacobian matrix of system (1) at $E_0=(H^*,0,0)$

$$J(E_0)=\begin{pmatrix}-\delta_H+r\left(1-\frac{2H^*}{H_{\max}}\right) & -\frac{rH^*}{H_{\max}}+\rho & -(\beta+\phi)H^*-s\\0 & -(\rho+\delta_I) & \beta H^*\\0 & A\omega & -(c+\phi H^*)\end{pmatrix}$$

The characteristic polynomial is expressed as

$$\det(J(E_0)-\lambda I)=0$$

One eigenvalue is

$$\lambda_1=-\delta_H+r\left(1-\frac{2H^*}{H_{\max}}\right)$$

Since H^* is the positive root of (2.5), typical parameter regimes yield $2H^*/H_{\max}<1$, ensuring $\lambda_1<0$.

The remaining two eigenvalues are obtained from

$$\begin{vmatrix}-(\rho+\delta_I)-\lambda & \beta H^*\\A\omega & -(c+\phi H^*)-\lambda\end{vmatrix}=0$$

which yields $\lambda^2+a_1\lambda+a_2=0$, where, $a_1=(\rho+\delta_I)+(c+\phi H^*)$, $a_2=(\rho+\delta_I)(c+\phi H^*)-\beta H^* A\omega$

By the Routh–Hurwitz criterion, both roots contain nonpositive real parts if and only if $a_1>0$ and $a_2>0$.

Since $a_1>0$, stability requires $(\rho+\delta_I)(c+\phi H^*)-\beta H^* A\omega>0$, which corresponds to $R_0<1$. If $R_0>1$, then $a_2<0$ and E_0 is unstable.

Lemma If $R_0\le 1$, then $H(t)\le H^*$ for all t sufficiently large.

Proof: As $I,V\to 0$, equation 1a of system (2.1) reduces to

$$\frac{dH}{dt}\to f(H):=s+(r-\delta_H)H-\frac{r}{H_{\max}}H^2.$$

This is a concave-down quadratic satisfying $f(0)=s>0$. Its unique positive root is precisely H^* as given by equation (2.6). Since the parabola opens downward and H^* is its positive root, $f(H)<0$ for all $H>H^*$. Consequently $\frac{dH}{dt}<0$ whenever $H>H^*$, so Hcannot exceed H^* asymptotically as the infection clears. Furthermore, for any $V>0$ the variable source term satisfies $\frac{s}{1+V}<s$, which only reduces T-cell recruitment relative to the limiting equation. By comparison, $H(t)\le H^*$ for all sufficiently large t. □

Global Stability of the Disease-Free Equilibrium

Theorem If $R_0\le 1$, then the disease-free equilibrium E_0 is said to be globally asymptotically stable in the invariant region Ω.
Proof. By Lemma 3.2, when $R_0\le 1$, the healthy T-cell population satisfies $H(t)\le H^*$ for all sufficiently large t. Therefore, we construct a Lyapunov function for the infected subsystem (I,V), with H bounded by H^*.
Define

$$L(I,V)=\frac{A\omega}{\rho+\delta_I}I+V.$$

Differentiating L along solutions of system (2.1) yields

$$\frac{dL}{dt}=\frac{A\omega}{\rho+\delta_I}\left[\frac{\beta VH}{1+\alpha V}-(\rho+\delta_I)I\right]+\left[A\omega I-\frac{\phi VH}{1+\alpha V}-cV\right]$$
$$=\frac{VH}{1+\alpha V}\left(\frac{A\omega\beta}{\rho+\delta_I}-\phi\right)-cV.$$

Using the bound $H\le H^*$ in Ω, we obtain

$$\frac{dL}{dt}\le V\left[\frac{H^*}{1+\alpha V}\left(\frac{A\omega\beta}{\rho+\delta_I}-\phi\right)-c\right]$$

this can be rewritten as

$$\frac{dL}{dt}\le\frac{V(c+\phi H^*)}{1+\alpha V}(R_0-1)-\frac{\alpha cV^2}{1+\alpha V}.$$

Hence, if $R_0\le 1$, then $\frac{dL}{dt}\le 0$ for all $(I,V)\in\Omega$. Moreover, $\frac{dL}{dt}=0$ if and only if $V=0$, which implies $I=0$.
By LaSalle's invariance principle, all trajectories in Ω converge to the infection-free equilibrium E_0. □

ENDEMIC EQUILIBRIUM

When $R_0>1$, the infection-free equilibrium becomes unstable, and the system admits a positive infected equilibrium

$$E^*=(\bar H,\bar I,\bar V),\ \bar I>0,\ \bar V>0,$$

corresponding to chronic infection.

Theorem If $R_0>1$, then system (2.1) admits at least one infected equilibrium $E^*=(\bar H,\bar I,\bar V)$, with all components positive.
Proof: At equilibrium, equations (2.1) give

$$\left.\begin{aligned}0&=\frac{s}{1+\bar V}+r\bar H\left(1-\frac{\bar H+\bar I}{H_{\max}}\right)-\frac{\beta\bar V\bar H}{1+\alpha\bar V}-\frac{\phi\bar V\bar H}{1+\alpha\bar V}+\rho\bar I\\ 0&=\frac{\beta\bar V\bar H}{1+\alpha\bar V}-(\rho+\delta_I)\bar I\\ 0&=A\omega\bar I-\frac{\phi\bar V\bar H}{1+\alpha\bar V}-c\bar V\end{aligned}\right\}\quad(4.1)$$

From the second equation,

$$\bar I=\frac{\beta\bar V\bar H}{(\rho+\delta_I)(1+\alpha\bar V)}\qquad(4.2)$$

Substituting (4.2) into the third equation,

$$A\omega\cdot\frac{\beta\bar V\bar H}{(\rho+\delta_I)(1+\alpha\bar V)}=\frac{\phi\bar V\bar H}{1+\alpha\bar V}+c\bar V$$

Dividing by $\bar V>0$ and multiplying through by $(1+\alpha\bar V)$,

$$\frac{A\omega\beta\bar H}{\rho+\delta_I}=\phi\bar H+c(1+\alpha\bar V)$$

Solving for $\bar H$,

$$\bar H=\frac{c(1+\alpha\bar V)(\rho+\delta_I)}{A\omega\beta-\phi(\rho+\delta_I)}\qquad(4.3)$$

Define a continuous function $g(V)$ by substituting (4.2) and (4.3) into the first equilibrium equation of (4.1). When $V=0$, one finds $g(0)>0$ if $R_0>1$. As $V\to\infty$, the logistic and saturation terms ensure $g(V)<0$. By the intermediate value theorem, there exists $\bar V>0$ such that $g(\bar V)=0$. The corresponding $\bar H,\bar I$ from (4.2)–(4.3) are positive, proving existence. □

Theorem If $R_0>1$, the endemic equilibrium $E^*=(\bar H,\bar I,\bar V)$ is locally asymptotically stable.
Proof: Let $E^*=(\bar H,\bar I,\bar V)$ be the endemic equilibrium. Define

$$\bar{q}=\frac{\beta\bar{V}}{1+\alpha\bar{V}},\ \bar{p}=\frac{\phi\bar{V}}{1+\alpha\bar{V}},$$

which represent the effective infection and neutralization rates at equilibrium. The Jacobian matrix evaluated at E^* is $J(E^*)=(j_{ij})$, where

$$j_{11}=-\delta_H+r(1-(2H+\bar{I})/H_{max})-\bar{q}-\bar{p},\ j_{12}=-(rH)/H_{max}+\rho,$$
$$j_{13}=-((\beta+\varphi)H)/(1+\alpha V)^2-s/(1+V)^2,\ j_{21}=\bar{q},\ j_{22}=-(\rho+\delta_I),$$
$$j_{23}=(\beta H)/(1+\alpha V)^2,\ j_{31}=-\bar{p},\ j_{32}=A\omega,\ j_{33}=-(\varphi H)/(1+\alpha V)^2-c$$

From the equilibrium equation for H, we have

$$\frac{s}{(1+\bar{V})\bar{H}}+r\left(1-\frac{\bar{H}+\bar{I}}{H_{max}}\right)-\delta_H-(\bar{q}+\bar{p})+\frac{\rho\bar{I}}{\bar{H}}=0.$$

Subtracting $\frac{rH}{H_{\max}}$ from both sides gives

$$j_{11}=-\frac{s}{(1+\bar{V})\bar{H}}-\frac{\rho\bar{I}}{\bar{H}}-\frac{r\bar{H}}{H_{max}}<0.$$

Clearly,

$$j_{22}=-(\rho+\delta_I)<0$$
$$j_{33}=-(\varphi H)/(1+\alpha V)^2-c<0$$

The characteristic polynomial of $J(E^*)$ is $\lambda^3+a_1\lambda^2+a_2\lambda+a_3=0$,
Where $a_1=-(j_{11}+j_{22}+j_{33})$.
Thus,

$$a_1=|j_{11}|+(\rho+\delta_I)+\frac{\varphi\bar{H}}{(1+\alpha\bar{V})^2}+c>0.$$

The coefficients $a_2$and $a_3$are given by combinations of the Jacobian entries:

$$a_2=j_{11}j_{22}+j_{11}j_{33}+j_{22}j_{33}-(j_{12}j_{21}+j_{13}j_{31}+j_{23}j_{32}),$$
$$a_3=-\det(J(E^*)).$$

Using the positivity of E^* and the biological feasibility of parameters, it follows that all contributing terms satisfy the required sign conditions, yielding $a_2>0$ and $a_3>0$. Furthermore, direct computation shows that $a_1a_2>a_3$. Hence, all Routh–Hurwitz conditions are satisfied, and the endemic equilibrium E^* is locally asymptotically stable. □

NUMERICAL SIMULATION

To validate analytical results derived in the previous sections, we numerically integrate system (1). Parameter values are chosen based on published HIV modelling studies and clinical data to ensure biological realism. The initial conditions are fixed as $(H_0,I_0,V_0)=(1000, 0, 0.001)$, representing a healthy immune system with baseline CD4⁺ T-cell count close to $H_{\max}$ exposed to a very small viral inoculum. These initial conditions reflect the state of an individual shortly after exposure to HIV. For the HIV-free equilibrium, we employ the parameter values, $s=10$, $\delta_h=0.01$, $\delta_i=0.5$, $r=0.03$, $H_{max}=1500$, $\beta=2.5\times10^{-4}$, $\alpha=1.0\times10^{-4}$, $\varphi=2.0\times10^{-5}$, $\rho=0.2$, $A=10$, $c=3.4$, $\omega=0.2$. Under these conditions, $R_0=0.285<1$, which theoretically predicts that the infection will not establish and the system will converge to the disease-free equilibrium E_0. This threshold condition is confirmed by numerical integration over 500 days.

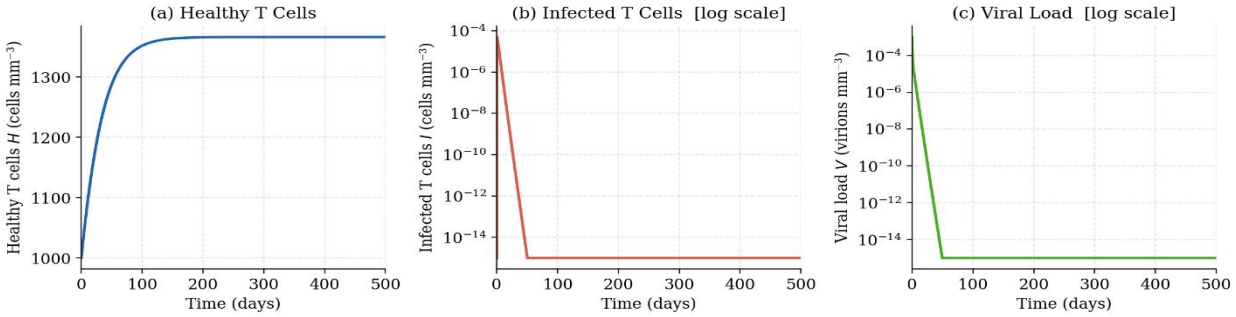

Figure 2: Dynamics of the model at infection-free equilibrium.

The system is capable of clearing viruses successfully, with healthy CD4+ T-cells remaining close to baseline while the infected cells and viral load exhibit a transient early increase before decaying exponentially to negligible levels, eventually converging smoothly and monotonically to the infection-free equilibrium. This clearance emerges from the interplay of three main processes, each driven by: viral saturation at high levels that features poor infection efficiency, sustained immune-mediated neutralization to deplete free virus from circulation, and logistic growth that constrains runaway infection by depleting the susceptible cell pool, and guarantees that the infection cannot be sustained when R_0 falls below unity.

To examine infected dynamics, we use $s=10$, $\delta_h=0.01$, $\delta_i=0.5$, $r=0.03$, $H_{max}=1500$, $\beta=0.0027$, $\alpha=1.0\times10^{-4}$, $\varphi=2.0\times10^{-5}$, $\rho=0.2$, $A=10$, $c=3.4$, $\omega=0.2$. This yields a basic reproduction number of $R_0=3.08>1$, which theoretically predicts that the infection-free equilibrium becomes unstable and the system will evolve toward a persistent infection state corresponding to an infected

equilibrium $E^* = (H^*, I^*, V^*)$ with all components strictly positive.

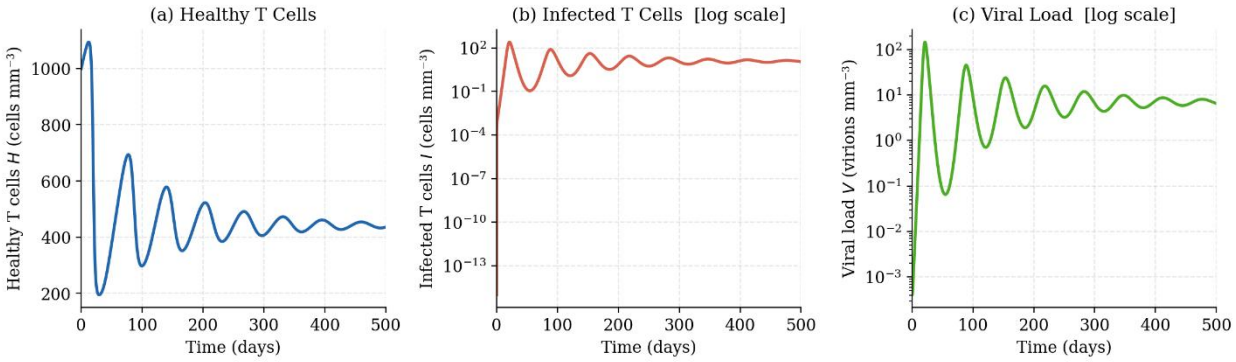

Figure 3: Dynamics of the model at the infected equilibrium

The system then moves to the decaying and converging periods after the acute phase, and finally reaches the endemic equilibrium. With this increased viral load, a healthy population of T cells declines from the baseline through the inhibition of CD4 recruitment and thus imprints immune exhaustion. Saturation feedback processes, harnessing the immune system and limiting infection, constrain viral growth so that infected cells and virus load decline from their maximum levels. The system finally settles down at a moderate level of CD4⁺ depletion and continues to support some amount of viral replication, which is a biologically sound representation for chronic infection without unbounded growth or sustained oscillations.

To demonstrate saturation's stabilizing role, we compare viral dynamics across varying α values while maintaining $R_0>1$. The mass-action model ($\alpha=0$) exhibits threefold higher viral overshoot, pronounced persistent oscillations, and elevated endemic burden compared to saturated formulations. Increasing saturation strength progressively reduces acute viral peaks, accelerates convergence, and lowers chronic viral loads. The baseline saturated model ($\alpha=0.0001$) produces smooth monotonic convergence without spurious oscillations, while stronger saturation ($\alpha=0.001$) further dampens overshoot and expedites equilibration.

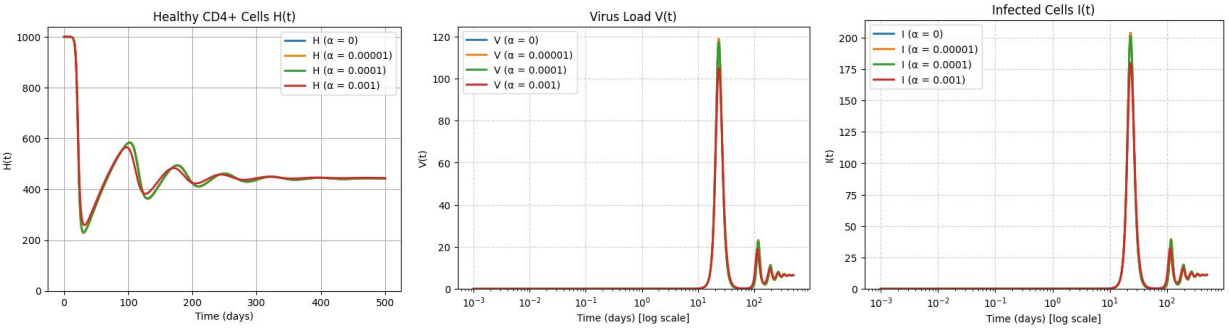

Figure 4: Effect of saturation parameter α on viral dynamics

This systematic trend demonstrates that saturation captures finite receptor availability, yielding biologically realistic dynamics absent in mass-action formulations. The saturated framework thus provides essential improvements, reduced viral overshoot, elimination of nonphysical oscillations, and superior numerical stability, which justify its adoption despite increased mathematical complexity.

CONCLUSION

A nonlinear within-host HIV infection model incorporating immune exhaustion, Holling type II saturated virus–cell interactions, and logistic regulation of T-cells has been developed and rigorously analysed. The proposed model is mathematically well posed, nonnegative, and uniformly bounded. Stability analysis confirmed that the infection-free equilibrium is locally and globally asymptotically stable. Numerical simulations support the theoretical results and reproduce biologically realistic acute-to-chronic infection dynamics. Comparative studies indicate that saturation is an important factor in maintaining stability by lowering viral overshoot, getting rid of nonphysical oscillations that come with mass-action formulations, and improving numerical robustness by limiting infection pressure at high viral loads. In general, the proposed model strikes a balance between analytical tractability and biological realism. It also provides a flexible base for future additions that include spatial effects, immune heterogeneity, therapeutic interventions, and random influences.

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