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Dynamic behavior of a two-dimensional discrete-time HIV infection model: stability and bifurcation analysis

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Abstract

Background: Human immunodeficiency virus (HIV) infection remains a global health challenge, and mathematical modeling has become an essential tool for understanding its dynamics. While continuous-time models have been widely used, discrete-time approaches offer computational advantages and better alignment with clinical data collection. This study aims to analyze the dynamical behavior of a two-dimensional discrete-time HIV infection model.

Methods: We derived a discrete-time HIV model from a continuous system using the forward Euler discretization scheme. We performed stability analysis of both infection-free and endemic fixed points using the Jacobian matrix approach. Bifurcation analysis was conducted using center manifold theory and normal form methods. Numerical simulations, including bifurcation diagrams and Lyapunov exponent computations, were performed to validate the analytical results.

Results: We established necessary and sufficient conditions for the stability of the infection-free fixed point and sufficient conditions for the local stability of the endemic fixed point. The model undergoes flip (period-doubling) and Neimark-Sacker bifurcations as the infection rate parameter varies. Numerical simulations confirmed the emergence of periodic, quasiperiodic, and chaotic oscillations. The bifurcation diagrams and Lyapunov exponent spectra quantitatively demonstrate transitions from order to chaos.

Conclusions: The observed oscillatory and chaotic behaviors in viral dynamics may originate from intrinsic nonlinear interactions between viral replication and immune response rather than external noise. The proposed discrete model provides a robust theoretical framework for understanding complex temporal patterns of HIV infection and offers potential insights into viral persistence and control strategies.

1 Background

HIV (human immunodeficiency virus) is a virus that attacks the body's immune system [11, 16, 19, 20]. If HIV is not treated, it can lead to AIDS (acquired immunodeficiency syndrome) [1, 4, 14, 15, 22, 24, 29–31]. There is currently no effective cure. Once people contract HIV, they have it for life. But with proper medical care, HIV can be controlled. People with HIV who receive effective HIV treatment can live long, healthy lives and protect their partners [2, 12, 13, 27]. HIV infection in humans came from a type of chimpanzee in Central Africa. Studies show that HIV may have jumped from chimpanzees to humans as far back as the late 1800s. The chimpanzee version of the virus is called simian immunodeficiency virus. It was probably passed to humans when humans hunted these chimpanzees for meat and came in contact with their infected blood. Over decades, HIV slowly spread across Africa and later into other parts of the world [34, 35, 37]. Over the past decades, numerous mathematical models have been developed to investigate the transmission and progression dynamics of HIV infection. Nowak and Bangham (1996) and Culshaw and Ruan (2000) introduced continuous-time differential equation models to describe the interactions between viral particles and CD4+ T cells, elucidating the mechanisms of viral replication and immune response [19, 20]. Later, Rong et al. (2007) and Tarfulea (2019) extended these frameworks to include drug therapy and time-delay effects, revealing how treatment interventions influence viral load dynamics [26, 27]. Discrete-time approaches have gained increasing attention in recent years due to their computational advantages and ability to capture complex nonlinear behaviors. Shi and Dong (2014) formulated a discrete HIV-1 virus model with bilinear infection rate and demonstrated the occurrence of rich dynamical patterns such as oscillations and chaos [36]. Similarly, Ackleh et al. (2019) and Hu et al. (2012) studied discrete epidemic and predator-prey systems, establishing rigorous conditions for persistence, stability, and bifurcation [3, 7, 10]. Chavez (2010) and Salman and Elsadany (2023) further analyzed bifurcation structures in discrete systems, showing that local stability loss through flip or Neimark-Sacker bifurcations can give rise to periodic and quasi-periodic oscillations [17, 28]. These studies collectively indicate that discretized epidemic systems can reproduce biologically realistic oscillatory and chaotic dynamics that are not easily captured by continuous-time models. Motivated by these findings, the present work aims to construct and analyze a two-dimensional discrete-time HIV infection model derived from its continuous counterpart using the forward Euler discretization scheme. The model preserves the essential biological properties of the continuous formulation while allowing the exploration of bifurcation phenomena, stability transitions, and complex dynamical behaviors such as periodic, quasiperiodic, and chaotic oscillations.

The virus has existed in the United States since at least the mid-to late 1970s.

AIDS has now surpassed the Black Death on its course to become the worst pandemic in human history. At the end of 2004, 20 million people had been killed by it, and twice that number are currently infected with HIV. Barring a medical breakthrough, it could claim the lives of some 60 million people by 2015. AIDS exerts a terrible toll on societies, crippling their economies, decimating their labor forces and orphaning their children.

Nine out of 10 people living with HIV are in the developing world; 60 to 70% of those are in Sub-Saharan Africa. But the disease is spreading in every region, with fierce epidemics threatening to tear through countries such as India, China, Russia, and the islands of the Caribbean. The statistics are sobering-in some Southern African towns 44% of pregnant women are HIV positive, in Botswana 37% of people carry the virus. Any natural phenomenon can be represented using a mathematical model to make predictions, know what happens, and control the results. Discrete and Continuous Dynamical Systems presented a mathematical model for HIV-1 infection to know whether the virus will spread to all cells or not, and how we can control it. In the late past centuries, researchers tended to represent infectious diseases with mathematical models to know whether the disease will invade the population or not?. The main contributions of this study are:

- Establishing necessary and sufficient conditions for the stability of both infection-free and endemic fixed points;
- Demonstrating the occurrence of flip and Neimark-Sacker bifurcations as the infection rate parameter varies;
- Providing numerical evidence of transitions from order to chaos through period-doubling routes.

2 Methods

2.1 Mathematical model

Our model established in [21] which is given by the following differential equations:

$$\begin{aligned}\dot{x} &= 1 - Dx - (B + \frac{Ay}{y+C})xy, \\ \dot{y} &= (B + \frac{Ay}{y+C})xy - y,\end{aligned}\tag{2.1}$$

where x , y and B are represent, respectively, the population densities of the uninfected $CD4^+T$ cells, infected $CD4^+T$ cells and bifurcation parameter. Taking the parameter values from [21]. Most of the models that were used to describe the HIV

infection were by a set of nonlinear ordinary differential equations. (ODEs), that is, continuous-time models, but the most appropriate to describe HIV infection are the discrete-time models, for the following reasons [8]:

- The data that are obtained from transmitted diseases are usually discrete, which is applied to HIV infection.

- The accurate analytical solutions to continuous-time models are unknown and So a discretization is needed to get discrete-time models that approximate the accurate solutions.

- Generally, simulations of models demand digital computers, which, in turn, demand as a necessity discrete-time models.

Fundamentally, the discretization method [18,32,33,39] should maintain the main properties of their continuous-time correspondence, for example, equilibria and their stability. There are different standard methods for discretizing ODEs, such as Euler and Runge-Kutta methods.

Applying the forward Euler scheme to system (2.1), we obtain the discrete-time HIV system as follows:

$$\begin{aligned}x_{n+1} &= x_n + h[1 - Dx_n - (B + \frac{Ay_n}{y_n + C})x_n y_n], \\y_{n+1} &= y_n + h[(B + \frac{Ay_n}{y_n + C})x_n y_n - y_n],\end{aligned}\tag{2.2}$$

where h is the step size. The system has two fixed points, namely,

HIV-free fixed point, $E^0 = \left(\frac{1}{D}, 0\right)$

Infected fixed point, $E^* = (x^*, y^*)$, where

$x^* = \frac{y^* + C}{y^*(A+B)+BC}$ such that y^* is the solution of the following equation:

$$(A + B)y^2 + (D + BC - B - A)y + C(D - B) = 0,$$

where

$$y_{1,2}^* = \frac{A + B - BC - D \pm \sqrt{(D + BC - B - A)^2 - 4C(A + B)(D - B)}}{2(A + B)}.$$

E^* is biologically possible only if the following conditions hold:

1. $(D + BC - B - A)^2 - 4C(A + B)(D - B) \geq 0$;
2. $A + B - BC - D > 0$;
3. $A + B - BC - D > \sqrt{(D + BC - B - A)^2 - 4C(A + B)(D - B)}$.

Remark on discretization: It is important to note that different discretization schemes may lead to different dynamical behaviors in the discrete model. The forward Euler scheme used here preserves the fixed points of the continuous system, but the bifurcation thresholds and the nature of the resulting oscillations may differ. Moreover, while the continuous system may exhibit limit cycles via Hopf bifurcation, the discrete system can display quasiperiodic orbits via Neimark-Sacker bifurcation. These differences highlight the importance of choosing an appropriate discretization scheme that captures the essential biological dynamics of the HIV infection process.

3 Existence and local stability of fixed points

To discuss the stability of model (2.2). Now, we will study the dynamics of the system (2.2) about the fixed points [3,7,25]. The Jacobian matrix J of the system (2.2) evaluated at any point (x, y) is given by $J(x,y)$ which reads

$$J(x, y) = \begin{pmatrix} 1 - h \left(D + \left(B + \frac{Ay}{y+C} \right) y \right) & -hx \left(B + \frac{Ay(y+2C)}{(y+C)^2} \right) \\ hy \left(B + \frac{A}{y+C} \right) & 1 + h \left(Bx - 1 + \frac{Axy(y+2C)}{(y+C)^2} \right) \end{pmatrix}.$$

Lemma 1. The uninfected fixed point E^0 has the following topological classifications:

1. A **sink** (locally asymptotically stable) if:

$$0 < hD < 2 \quad \text{and} \quad 0 < h \left(1 - \frac{B}{D} \right) < 2$$

2. A **source** (unstable) if:

$$hD > 2 \quad \text{and} \quad B > D, \quad \text{or} \quad h \left(1 - \frac{B}{D} \right) > 2$$

3. A **saddle point** if any of the following conditions hold:

$$(a) \quad hD > 2 \quad \text{and} \quad 0 < h \left(1 - \frac{B}{D} \right) < 2$$

$$(b) \quad 0 < hD < 2 \quad \text{and} \quad B > D, \quad \text{or} \quad h \left(1 - \frac{B}{D} \right) > 2$$

4. **Nonhyperbolic** if any of the following conditions hold:

$$(a) \quad hD = 2, \quad \text{provided that} \quad B \neq D \quad \text{and} \quad h \left(1 - \frac{B}{D} \right) \neq 2$$

(b) $B = D$ or $h(1 - \frac{B}{D}) = 2$, provided that $hD \neq 2$

The characteristic equation of the Jacobian matrix evaluated at the endemic fixed point E^* is:

$$F(\lambda) = \lambda^2 + P\lambda + Q = 0,$$

where

$$P = -2 + h(y^*S + D - Bx^* - M + 1),$$

$$Q = 1 + h(Bx^* + M - 1 - y^*S - D) - h^2((D + y^*S)(Bx^* + M - 1) - x^*y^*S(B + M)),$$

with

$$S = B + \frac{Ay^*}{y^* + C}, \quad M = \frac{Ax^*y^*(y^* + 2C)}{(y^* + C)^2}.$$

We also compute:

$$F(1) = h^2(x^*y^*S(B + M)) - (D + y^*S)(Bx^* + M - 1),$$

$$F(-1) = 4 + 2h(Bx^* + M - 1 - D - y^*S) - h^2((D + y^*S)(Bx^* + M - 1) - x^*y^*S(B + M)).$$

This result discusses the local asymptotic stability of the endemic fixed point $E^* = (x^*, y^*)$.

Lemma 2. Let $F(\lambda) = \lambda^2 + P\lambda + Q$. Suppose that $F(1) > 0$, then E^* is:

1. **a sink** if

$$4 + 2h(Bx^* + M - 1 - D - y^*S) - h^2((D + y^*S)(Bx^* + M - 1) - x^*y^*S(B + M)) > 0$$

and

$$1 + h(Bx^* + M - 1 - y^*S - D) - h^2((D + y^*S)(Bx^* + M - 1) - x^*y^*S(B + M)) < 1$$

2. **a saddle** if

$$4 + 2h(Bx^* + M - 1 - D - y^*S) - h^2((D + y^*S)(Bx^* + M - 1) - x^*y^*S(B + M)) < 0$$

3. **a source** if

$$4 + 2h(Bx^* + M - 1 - D - y^*S) - h^2((D + y^*S)(Bx^* + M - 1) - x^*y^*S(B + M)) > 0$$

and

$$1 + h(Bx^* + M - 1 - y^*S - D) - h^2((D + y^*S)(Bx^* + M - 1) - x^*y^*S(B + M)) > 1$$

4. **nonhyperbolic** if one of the following holds:

$$(a) \quad 4 + 2h(Bx^* + M - 1 - D - y^*S) - h^2((D + y^*S)(Bx^* + M - 1) - x^*y^*S(B + M)) = 0, \text{ and } P \neq 0, 2$$

$$(b) \quad P^2 - 4Q < 0 \text{ and } Q = 1$$

4 Bifurcation analysis

Codimension-One Bifurcations

In this section [5, 9, 17, 23, 28, 39, 40], we consider the parameters A, C , and D as fixed, and vary B as the bifurcation parameter. We select B because it serves as a rescaled measure of the infection rate in the absence of ROS, which plays a critical role in the behavior of model (2.2), particularly in the local stability of the endemic fixed point $E^*(x^*, y^*)$. In essence, B is directly proportional to the infection rate b . When B is used as the bifurcation parameter, the system starts from the uninfected fixed point and transitions to the endemic state as B increases,

two types of local bifurcations may occur at E^* in model (2.2): a flip bifurcation [38] and a Neimark-Sacker bifurcation.

According to part (4)(i) of Lemma 2, a nondegenerate flip bifurcation occurs near E^* if B is perturbed around the set:

$$\text{Flip}(E^*) = \left\{ (A, B, C, D, h) \in \mathbb{R}^5 \mid B_f = \frac{h^2 \left((M-1) \left(D + \frac{Ay^{*2}}{y^* + C} \right) - Mx^* \frac{Ay^{*2}}{y^* + C} \right) + 2h \left(D - M + 1 + \frac{Ay^{*2}}{y^* + C} \right) - 4}{-h^2 \left(Dx^* + (M-1)y^* - Mx^* \left(\frac{Ay^{*2}}{y^* + C} \right) \right) + 2h(x^* - y^*)}, \quad P \neq 0, 2 \right\}$$

A flip bifurcation can arise at the fixed point E^* when the system parameters are perturbed within a small neighborhood around the set $\text{Flip}(E^*)$. Under these conditions, Model 2.2 is expressed in the following form.

$$\begin{cases} x \rightarrow x + h(1 - Dx - (B + B^* + \frac{Ay}{y+C})xy), \\ y \rightarrow y + h((B + B^* + \frac{Ay}{y+C})xy - y), \end{cases} \quad (4.1)$$

where $|B^*| \ll 1$ is a limited perturbation parameter.

Transform E^* to the origin by introducing $u = x - x^*$, $v = y - y^*$, and we obtain

$$\begin{cases} u \rightarrow a_1 u + a_2 v + a_{12} uv + a_{22} v^2 + a_{122} uv^2 + a_{222} v^3 + a_{13} u B^* + a_{23} v B^* + O((|u| + |v| + |B^*|)^4), \\ v \rightarrow b_1 u + b_2 v + b_{12} uv + b_{22} v^2 + b_{122} uv^2 + b_{222} v^3 + b_{13} u B^* + b_{23} v B^* + O((|u| + |v| + |B^*|)^4), \end{cases} \quad (4.2)$$

where

$$\left\{ \begin{array}{l} a_1 = 1 + h(-D - (B + B^* + \frac{Ay^*}{y^*+C})y^*), \quad a_2 = h(-(B + B^*)x^* - \frac{Ax^*y^*(y^*+2C)}{(y^*+C)^2}), \\ a_{12} = h(-(B + B^*) - \frac{Ay^*(y^*+2C)}{(y^*+C)^2}), \quad a_{22} = h\left(\frac{-AC^2x^*}{(y^*+C)^3}\right), \\ a_{122} = h\left(\frac{-AC^2}{(y^*+C)^3}\right), \quad a_{222} = h\left(\frac{AC^2x^*}{(y^*+C)^3}\right), \\ a_{13} = -hy^*, \quad a_{23} = -hx^*, \quad b_{13} = hy^*, \quad b_{23} = hx^*, \\ b_1 = h\left((B + B^*)y^* + \frac{Ay^{*2}}{(y^*+C)}\right), \quad b_2 = 1 + h\left((B + B^*)x^* + \frac{Ax^*y^*(y^*+2C)}{(y^*+C)^2} - 1\right), \\ b_{12} = h\left(B + B^* + \frac{Ay^*(y^*+2C)}{(y^*+C)^2}\right), \quad b_{22} = h\left(\frac{AC^2x^*}{(y^*+C)^3}\right), \\ b_{122} = h\left(\frac{AC^2}{(y^*+C)^3}\right), \quad b_{222} = h\left(\frac{AC^2x^*}{(y^*+C)^3}\right). \end{array} \right. \quad (4.3)$$

Consider the translation

$$\begin{pmatrix} u \\ v \end{pmatrix} = T \begin{pmatrix} \tilde{x} \\ \tilde{y} \end{pmatrix},$$

where T is an invertible matrix given in the form

$$T = \begin{pmatrix} a_2 & a_2 \\ -1 - a_1 & \lambda_2 - a_1 \end{pmatrix},$$

then, model (4.2) has the form

$$\begin{pmatrix} \tilde{x} \\ \tilde{y} \end{pmatrix} \rightarrow \begin{pmatrix} -1 & 0 \\ 0 & \lambda_2 \end{pmatrix} \begin{pmatrix} \tilde{x} \\ \tilde{y} \end{pmatrix} + \begin{pmatrix} f_1(\tilde{x}, \tilde{y}, B^*) \\ f_2(\tilde{x}, \tilde{y}, B^*) \end{pmatrix}, \quad (4.4)$$

where

$$\begin{aligned} f_1(\tilde{x}, \tilde{y}, B^*) &= \frac{1}{a_2(1 + \lambda_2)}((\lambda_2 - a_1)(a_{12}uv + a_{22}v^2 + a_{122}uv^2 + a_{222}v^3 + a_{13}uB^* + a_{23}vB^*) \\ &\quad - a_2(b_{12}uv + b_{22}v^2 + b_{122}uv^2 + b_{222}v^3 + b_{13}uB^* + b_{23}vB^*) \\ &\quad + O((|u| + |v| + |B^*|)^4)), \\ f_2(\tilde{x}, \tilde{y}, B^*) &= \frac{1}{a_2(1 + \lambda_2)}((1 + a_1)(a_{12}uv + a_{22}v^2 + a_{122}uv^2 + a_{222}v^3 + a_{13}uB^* + a_{23}vB^*) \\ &\quad + a_2(b_{12}uv + b_{22}v^2 + b_{122}uv^2 + b_{222}v^3 + b_{13}uB^* + b_{23}vB^*) \\ &\quad + O((|u| + |v| + |B^*|)^4)), \end{aligned}$$

and

$$\begin{aligned} u &= a_2(\tilde{x} + \tilde{y}), \quad v = -(1 + a_1)\tilde{x} + (\lambda_2 - a_1)\tilde{y}, \\ u^2 &= a_2^2(\tilde{x}^2 + 2\tilde{x}\tilde{y} + \tilde{y}^2), \\ uv &= a_2(-(1 + a_1)\tilde{x}^2 + (\lambda_2 - 2a_1 - 1)\tilde{x}\tilde{y} + a_2(\lambda_2 - a_1)\tilde{y}^2), \\ v^2 &= (1 + a_1)^2\tilde{x}^2 + (\lambda_2 - a_1)^2\tilde{y}^2 - 2(1 + a_1)(\lambda_2 - a_1)\tilde{x}\tilde{y}. \end{aligned}$$

According to the center manifold theorem [5], There exists a center manifold $W_c(E^*)(0, 0, 0)$ of (4.2) at E^* in a small neighborhood of B^* in the following form

$$W_c(E^*)(0, 0, 0) = \{(\tilde{x}, \tilde{y}, B^*) \in R^3, \tilde{y} = h(\tilde{x}, B^*), h(0, 0) = 0, Dh(0, 0) = 0\},$$

for $\tilde{x}$ and B^* sufficiently small. Suppose there exists a center manifold in given by

$$h(\tilde{x}, B^*) = c_0 \tilde{x}^2 + c_1 \tilde{x} B^* + c_2 B^{*2} + O((|\tilde{x}| + |B^*|)^3). \quad (4.5)$$

The following relation must be satisfied by the center manifold

$$h(-\tilde{x} + f_1(\tilde{x}, h(\tilde{x}, B^*), B^*), B^*) = \lambda_2 h(\tilde{x}, B^*) + f_2(\tilde{x}, h(\tilde{x}, B^*), B^*). \quad (4.6)$$

Substituting (4.5) into (4.6), then equating like powers' coefficients in (4.6), we obtain

$$\begin{aligned} c_0 &= \frac{(1 + a_1)a_{22} + b_{22}}{a_2(1 - \lambda_2^2)}, \\ c_1 &= \frac{(1 + a_1)a_{23} + b_{13} + (1 + \lambda_2)a_{13} + b_{23}}{a_2(1 - \lambda_2^2)}, \\ c_2 &= 0. \end{aligned}$$

Now system (4.4) restricted to the center manifold $W_c(E^*)(0, 0, 0)$ is given by:

$$F : \tilde{x} \rightarrow -\tilde{x} + A\tilde{x}^2 + C\tilde{x}B^* + D\tilde{x}^2B^* + E\tilde{x}^3 + O((|\tilde{x}| + |B^*|)^4), \quad (4.7)$$

where

$$\begin{aligned} A &= \frac{\lambda_2 - a_1}{a_2(\lambda_2 + 1)}(-a_{22}(1 + a_1)^2 - a_{12}a_2(1 + a_1) + a_{22}(1 + a_1)) \\ &\quad + \frac{1}{a_2(\lambda_2 + 1)}(a_2^2b_{12}(1 + a_1) + a_2b_{22}(1 + a_1)^2), \\ C &= \frac{\lambda_2 - a_1}{a_2(\lambda_2 + 1)}(a_{13}a_2 - a_{23}(1 + a_1)) \\ &\quad - \frac{1}{a_2(\lambda_2 + 1)}(a_2^2b_{13} + a_2b_{23}(1 + a_1)), \\ E &= \frac{\lambda_2 - a_1}{a_2(\lambda_2 + 1)}(a_{122}a_2(1 + a_1)^2 - a_{222}(1 + a_1)^3) \\ &\quad + \frac{1}{a_2(\lambda_2 + 1)}(-a_2^2b_{122}(1 + a_1)^2 + a_2b_{222}(1 + a_1)^3), \\ D &= 0. \end{aligned}$$

The system (4.7) admits a flip bifurcation if the quantities α_1 and α_2 do not equal zero, where

$$\begin{aligned}\alpha_1 &= \left(2\frac{\partial^2 F}{\partial a^* \partial \tilde{x}} + \frac{\partial F}{\partial a^*} \frac{\partial F}{\partial \tilde{x}}\right)_{(0,0)} = 2C \neq 0, \\ \alpha_2 &= \left(\frac{1}{2}\left(\frac{\partial^2 F}{\partial \tilde{x}^2}\right)^2 + \frac{1}{3}\left(\frac{\partial^3 F}{\partial \tilde{x}^3}\right)\right)_{(0,0)} = 2(A^2 + E) \neq 0.\end{aligned}$$

This completes the proof.

Similarly, part (4)(ii) of Lemma 2 indicates that a Neimark-Sacker bifurcation may occur at E^* when B lies in a neighborhood of the set:

$$\text{NS}(E^*) = \left\{ (A, B, C, D, h) \in \mathbb{R}^5 \mid B_{\text{NS}} = \frac{h \left((M-1) \left(D + \frac{Ay^{*2}}{y^* + C} \right) - \frac{Mx^*y^{*2}}{y^* + C} \right) + D + 1 - M + \frac{Ay^{*2}}{y^* + C}}{x^* - y^* - h \left(Dx^* + (M-1)y^* - x^*y^* \left(M + \frac{Ay^*}{y^* + C} \right) \right)}, \quad P < 2\sqrt{Q} \right\}$$

We discuss Neimark-Sacker bifurcation of $E^*(x^*, y^*)$, the eigenvalues associated with $j(E^*)$ are complex conjugate ($\lambda = \bar{\lambda}$) if $P^2 < 4Q$,

$$\lambda, \bar{\lambda} = \frac{-P \pm i\sqrt{4Q - P^2}}{2}$$

We obtain $|\lambda| = \sqrt{Q}$,

$$Q = 1 + h(Bx^* + M - 1 - y^*S - D) - h^2((D + y^*S)(Bx^* + M - 1) - x^*y^*S(B + M)),$$

$$Q(B_{\text{NS}}) = 1,$$

$$\frac{d|\lambda|}{dB} \Big|_{B=B_{\text{NS}}} = \frac{h(x^* - y^*) - h^2(Dx^* + (M-1)y^* - x^*y^*M)}{2\sqrt{Q}} \neq 0.$$

In addition if:

$$P(B_{\text{NS}}) \neq 0, 1 \quad \text{then} \quad h(y^*S + D - Bx^* - M + 1) \neq 2, 3, \quad (4.8)$$

which satisfies $\lambda^m, \bar{\lambda}^m \neq 1$ for $m = 1, 2, 3, 4$. Therefore, the eigenvalues $\lambda, \bar{\lambda}$ of $fi_{x_2} = (x^*, y^*)$ of the system (4.2) do not lie in the intersection of the unit circle with the coordinate axes when $B^* = 0$ and the condition (4.8) holds, now, we discuss the normal form of (4.2) at $B^* = 0$.

Let $B^* = 0$, $\sigma = \frac{1}{2}(2 - h(y^*S + D - Bx^* + M + 1))$,

$$\omega = \frac{1}{2}\sqrt{4h^2((D + y^*S)(Bx^* + M - 1) - x^*y^*S(B + M)) + h^2(y^*S + D - Bx^* - M + 1)^2}.$$

Constructing an invertible matrix

$$T = \begin{pmatrix} a_2 & 0 \\ \sigma - a_1 & -\omega \end{pmatrix},$$

and using the translation

$$\begin{pmatrix} u \\ v \end{pmatrix} = T \begin{pmatrix} \tilde{x} \\ \tilde{y} \end{pmatrix},$$

the system (4.2) becomes

$$\begin{pmatrix} \tilde{x} \\ \tilde{y} \end{pmatrix} \rightarrow \begin{pmatrix} \sigma & -\omega \\ \omega & \sigma \end{pmatrix} \begin{pmatrix} u \\ v \end{pmatrix} + \begin{pmatrix} \bar{f}_1(\tilde{x}, \tilde{y}) \\ \bar{f}_2(\tilde{x}, \tilde{y}) \end{pmatrix}, \quad (4.9)$$

where

$$\begin{aligned}\bar{f}_1(\tilde{x}, \tilde{y}) &= \frac{a_{11}}{a_2}u^2 + \frac{a_{12}}{a_2}uv + \frac{a_{22}}{a_2}v^2 + O((|\tilde{x}| + |\tilde{y}|)^3), \\ \bar{f}_2(\tilde{x}, \tilde{y}) &= \frac{\sigma - a_1}{a_2\omega}(a_{11}u^2 + a_{12}uv + a_{22}v^2) + O((|\tilde{x}| + |\tilde{y}|)^3),\end{aligned}$$

and

$$\begin{aligned}u &= a_2\tilde{x}, v = (\sigma - a_1)\tilde{x} - \omega\tilde{y}, \\ uv &= a_2(\sigma - a_1)\tilde{x}^2 - a_2\omega\tilde{x}\tilde{y}, \\ u^2 &= a_2^2\tilde{x}^2, v^2 = (\sigma - a_1)^2\tilde{x}^2 + \omega^2\tilde{y}^2 - 2(\sigma - a_1)\omega\tilde{x}\tilde{y}.\end{aligned}$$

Therefore,

$$\begin{aligned}\bar{f}_{1\tilde{x}\tilde{x}} &= 2a_{11}a_2 + 2\frac{a_{22}}{a_2}(\sigma - a_1)^2 + 2a_{12}(\sigma - a_1), \\ \bar{f}_{1\tilde{x}\tilde{y}} &= -a_{12}\omega - 2\frac{a_{22}}{a_2}\omega(\sigma - a_1), \\ \bar{f}_{1\tilde{y}\tilde{y}} &= 2\frac{a_{22}}{a_2}\omega^2, \\ \bar{f}_{1\tilde{x}\tilde{y}} &= \bar{f}_{1\tilde{y}\tilde{y}} = \bar{f}_{1\tilde{y}\tilde{y}} = \bar{f}_{1\tilde{x}\tilde{x}} = 0, \\ \bar{f}_{2\tilde{x}\tilde{x}} &= \frac{\sigma - a_1}{a_2\omega}(2a_{11}a_2^2 + 2a_{12}a_2)(\sigma - a_1) + 2a_{22}(\sigma - a_1)^2, \\ \bar{f}_{2\tilde{x}\tilde{y}} &= \frac{\sigma - a_1}{a_2\omega}(-a_{12}a_2\omega - 2a_{22}(\sigma - a_1)\omega), \\ \bar{f}_{2\tilde{y}\tilde{y}} &= 2\frac{\sigma - a_1}{a_2\omega}(a_{22}\omega^2), \\ \bar{f}_{2\tilde{x}\tilde{y}} &= \bar{f}_{2\tilde{y}\tilde{y}} = \bar{f}_{2\tilde{y}\tilde{y}} = \bar{f}_{2\tilde{x}\tilde{x}} = 0\end{aligned}$$

at the origin.

The system (4.9) may undergo a Neimark-Sacker bifurcation if the following quantity does not equal zero:

$$L = \left[-Re\left(\frac{(1 - 2\lambda)\bar{\lambda}^2}{1 - \lambda}L_{11}L_{12}\right) - \frac{1}{2}|L_{11}|^2 - |L_{21}|^2 + Re(\bar{\lambda}L_{22}) \right]_{\bar{a}^*=0},$$

where

$$\begin{aligned}L_{11} &= \frac{1}{4}((\bar{f}_{1\tilde{x}\tilde{x}} + \bar{f}_{1\tilde{y}\tilde{y}}) + i(\bar{f}_{2\tilde{x}\tilde{x}} + \bar{f}_{2\tilde{y}\tilde{y}})), \\ L_{12} &= \frac{1}{8}((\bar{f}_{1\tilde{x}\tilde{x}} - \bar{f}_{1\tilde{y}\tilde{y}} + 2\bar{f}_{2\tilde{x}\tilde{y}}) + i(\bar{f}_{2\tilde{x}\tilde{x}} - \bar{f}_{2\tilde{y}\tilde{y}} - 2\bar{f}_{1\tilde{x}\tilde{y}})), \\ L_{21} &= \frac{1}{8}((\bar{f}_{1\tilde{x}\tilde{x}} - \bar{f}_{1\tilde{y}\tilde{y}} - 2\bar{f}_{2\tilde{x}\tilde{y}}) + i(\bar{f}_{2\tilde{x}\tilde{x}} - \bar{f}_{2\tilde{y}\tilde{y}} + 2\bar{f}_{1\tilde{x}\tilde{y}})), \\ L_{22} &= \frac{1}{16}((\bar{f}_{1\tilde{x}\tilde{x}} + \bar{f}_{1\tilde{y}\tilde{y}} + \bar{f}_{2\tilde{x}\tilde{y}} + \bar{f}_{2\tilde{y}\tilde{y}}) + i(\bar{f}_{2\tilde{x}\tilde{x}} + \bar{f}_{2\tilde{x}\tilde{y}} - \bar{f}_{1\tilde{x}\tilde{y}} - \bar{f}_{1\tilde{y}\tilde{y}})).\end{aligned}$$

According to the preceding analysis we can state the following result based on [5, 40].

Theorem 1. *System (2.2) undergoes Neimark-Sacker bifurcation at the fixed point $fix_2(x^*, y^*)$ under the conditions (4.8) and $L \neq 0$, when B is close to B^* . Moreover, if $L < 0$ (resp., $L > 0$), then an attracting (resp., repelling) invariant closed curve bifurcates from the fixed point for $B > B^*$ (resp., $B < B^*$).*

5 Numerical simulations

In this section, we conduct numerical simulations together with numerical continuation of fixed points to substantiate our analytical findings and to examine the richer dynamical phenomena of model (2.2). Throughout all simulations, the parameters are fixed as ($A = 0.361$, $C = 0.823$, $D = 0.057$), following the assumptions in Ref [21], while the parameters B and h are allowed to vary.

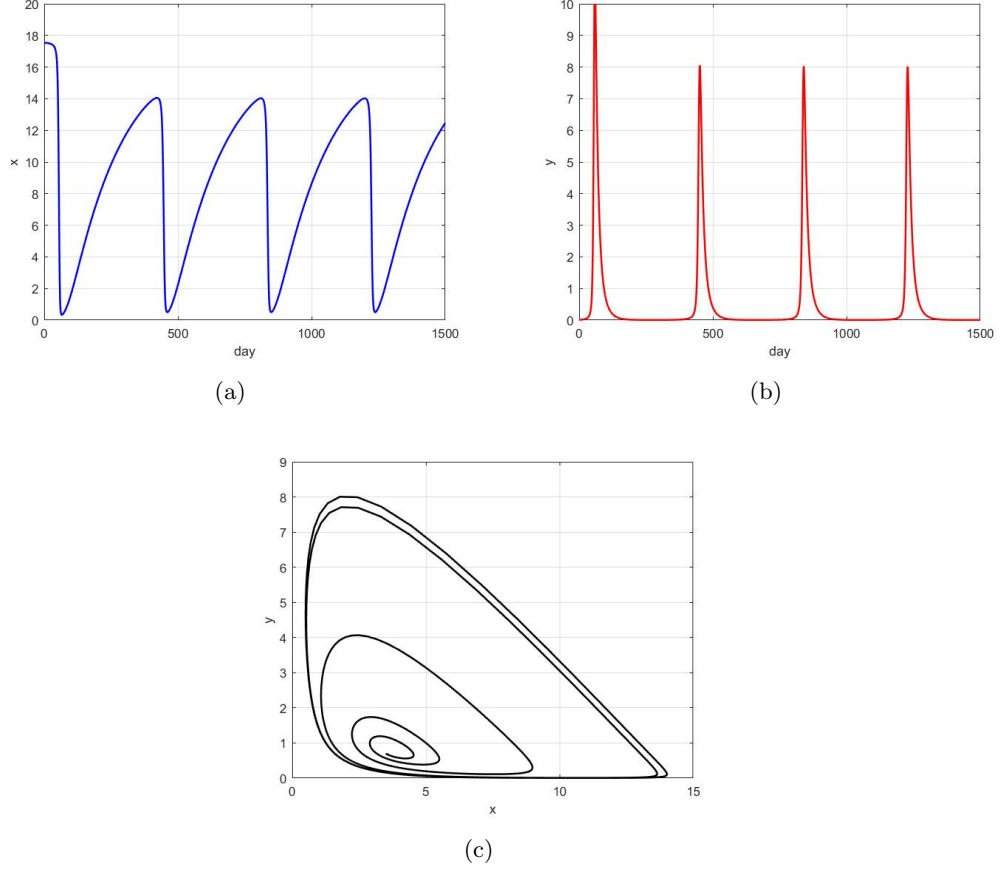


Figure 1: (a) and (b) viral blips trajectories of the model (2.2) and (c) a stable closed orbit. The initial conditions from (a)-(c) are $(x_0, y_0) = (13.543, 0.7)$, and $h = 0.02$.

Figure (1) presents the numerical simulations of the two-dimensional HIV infection model, illustrating the temporal and phase-space dynamics of the system. Panels (a) and (b) depict the time series trajectories of the uninfected cell population $x(t)$ and the viral load $y(t)$, respectively. Both variables exhibit recurrent oscillatory behavior following an initial transient phase, representing the appearance of periodic viral blips over time. This oscillatory pattern highlights the complex nonlinear interactions between viral replication and immune response mechanisms. Panel (c) shows the corresponding phase portrait in the (x, y) -plane, where the discrete points form a closed orbit, confirming the existence of a stable closed orbit. The presence of this closed orbit indicates that the system evolves toward

sustained periodic oscillations rather than a steady-state equilibrium. Overall, the numerical results corroborate the analytical predictions and demonstrate that, for the parameter set ($A = 0.361$, $C = 0.823$, $D = 0.057$), the model exhibits a stable periodic regime. This behavior supports the interpretation that viral blips may arise as deterministic manifestations of intrinsic system dynamics rather than as stochastic fluctuations, providing insight into the persistence and recurrence of low-level viremia observed in HIV-infected individuals.

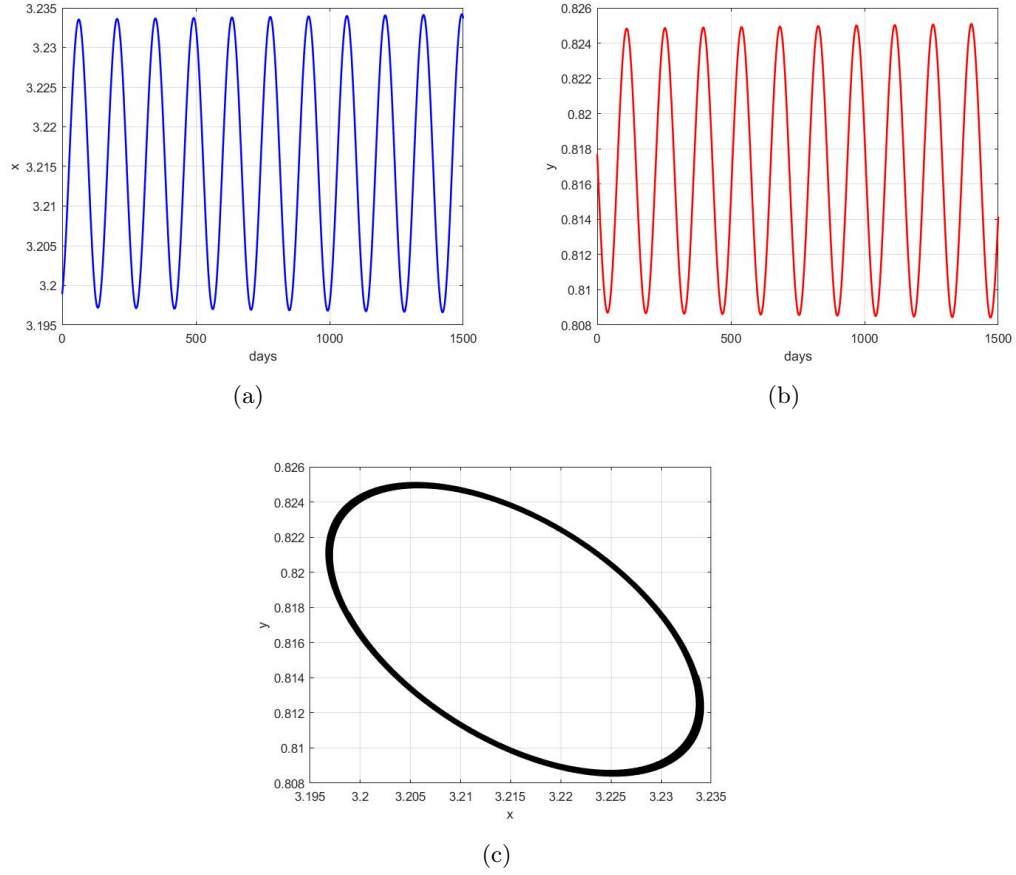


Figure 2: (a) and (b) trajectories of the model (2.2) and (c) Neimark-Sacker bifurcation at $(3.1989, 0.8177)$ with $h = 0.09$ and $B = 0.1312$.

Figure (2) illustrates the numerical evidence of a Neimark-Sacker bifurcation in the two-dimensional HIV infection model. Panels (a) and (b) display the time series trajectories of the uninfected cell population $x(t)$ and the viral load $y(t)$, re-

spectively, near the bifurcation point $(x^*, y^*) = (3.1989, 0.8177)$. The trajectories exhibit sustained small-amplitude oscillations, indicating the onset of quasiperiodic behavior as the system transitions from a stable equilibrium to a closed invariant curve.

Panel (c) presents the corresponding phase portrait in the (x, y) -plane, where the discrete points form a smooth closed orbit surrounding the fixed point. This confirms the emergence of a stable invariant torus characteristic of a Neimark-Sacker bifurcation.

The results obtained for $h = 0.09$ and $B = 0.1312$ demonstrate that as the control parameter B crosses its critical value, the fixed point loses stability and gives rise to persistent oscillations. This behavior highlights the system's transition from steady-state infection dynamics to quasiperiodic oscillations, which may represent complex viral load fluctuations observed in clinical HIV progression.

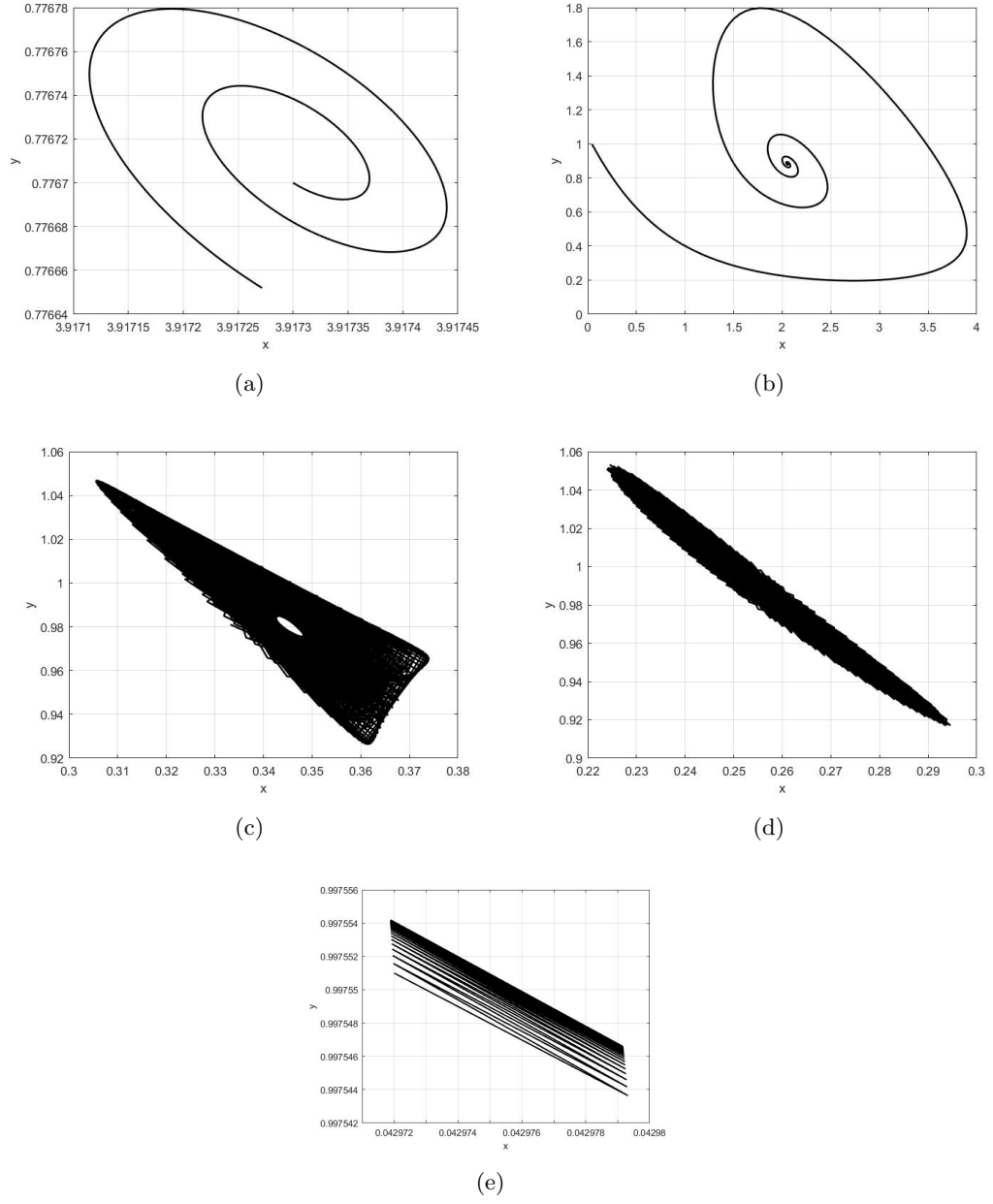


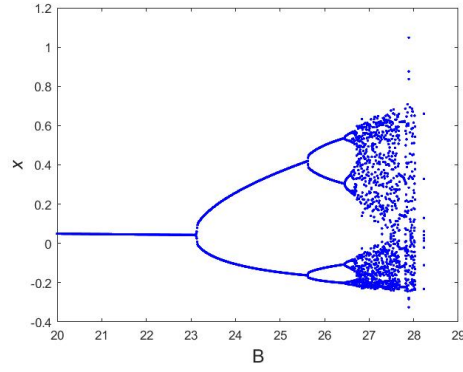
Figure 3: Phase plane of the model (2.2) with different values for x , y , h , and B .

Figure (3) illustrates the phase portraits of the two-dimensional HIV infection

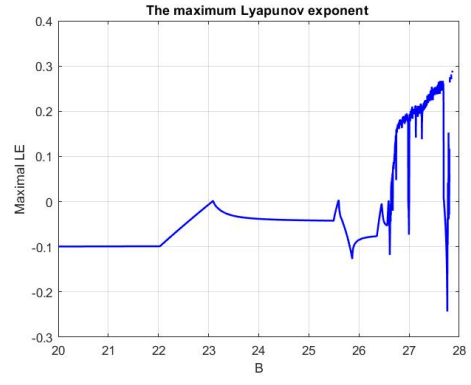
model (2.2) for different parameter values, highlighting the qualitative changes in system dynamics as key parameters vary. Panels (a)-(e) display distinct trajectories in the (x,y)-plane, each representing different dynamical regimes ranging from stable fixed point to oscillatory and quasi-periodic behaviors.

In Figures 3(a) and 3(b), the trajectories converge smoothly toward a fixed point, indicating locally asymptotically stable behavior. As parameters are varied, as seen in Figures 3(c)-(e), the system exhibits closed orbits and more complex attractor structures, suggesting transitions to periodic and quasiperiodic states through bifurcation phenomena. The emergence of such limit cycles and invariant curves reflects the system's sensitivity to parameter changes and the possibility of multiple stable dynamic modes.

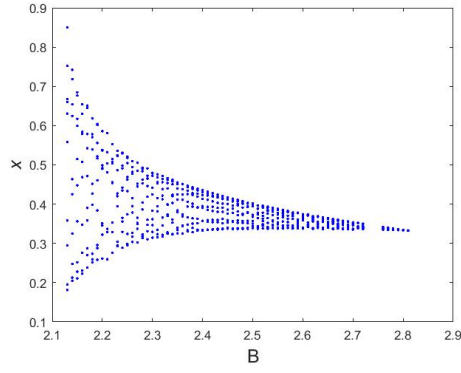
Overall, these phase plane analyses confirm that the HIV model undergoes rich nonlinear dynamical behavior, including stability loss and oscillatory patterns, depending on the control parameters. This underlines the potential of the model to capture diverse biological scenarios such as viral persistence, immune response oscillations, and the emergence of chronic infection states.



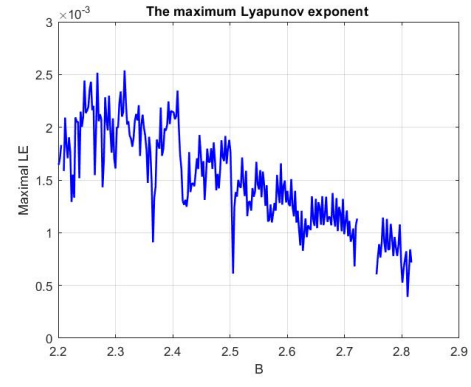
(a)



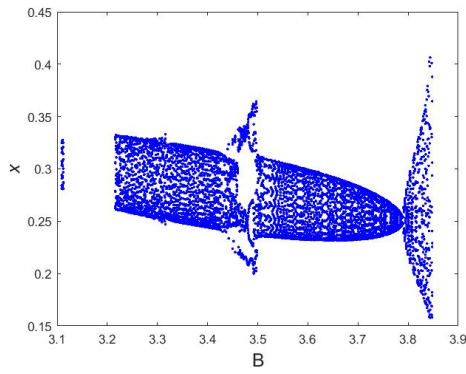
(b)



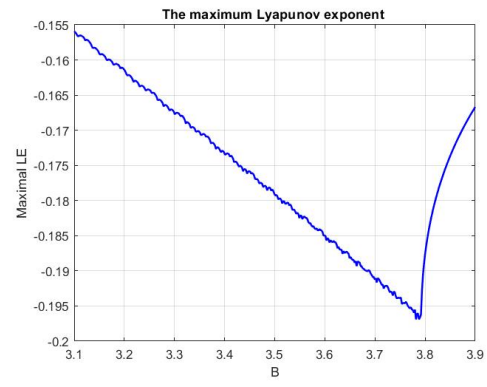
(c)



(d)



(e)



(f)

Figure 4: Bifurcation diagrams and corresponding Lyapunov exponents of map (2.2) with different values for x , y , h , and B .

Figure (4) presents the bifurcation diagrams and the corresponding maximum Lyapunov exponents of the two-dimensional HIV map (2.2) for different parameter values. Each pair of panels (a,b), (c,d) and (e,f) illustrates the qualitative changes in the system dynamics as the bifurcation parameter varies.

In the bifurcation diagrams (Figures 4a, 4c, and 4e), the system exhibits a transition from stable fixed points to periodic oscillations, followed by the emergence of chaotic regimes through a series of period-doubling bifurcations [6]. These complex behaviors reflect the nonlinear nature of the HIV model and demonstrate how small variations in system parameters can lead to significant qualitative changes in the long-term dynamics.

The corresponding plots of the maximum Lyapunov exponent (Figures 4b, 4d, and 4f) provide quantitative confirmation of these transitions. Negative Lyapunov exponents correspond to stable steady states or periodic solutions, while positive values indicate chaotic dynamics. The regions where the Lyapunov exponent crosses zero coincide with the bifurcation points observed in the diagrams, further validating the occurrence of transitions from order to chaos.

Overall, these results highlight the rich dynamical behavior of the HIV model and emphasize the sensitivity of its dynamics to parameter variations. The coexistence of periodic and chaotic attractors suggests potential implications for understanding irregular oscillations in viral load and immune response dynamics in HIV infection.

6 Conclusion

In this study, we investigated the nonlinear dynamical behavior of a two-dimensional discrete-time HIV infection model derived from its continuous counterpart through the forward Euler discretization scheme. The discretization preserved the fundamental qualitative properties of the continuous system while enabling detailed exploration of the discrete dynamics, including local stability, bifurcation phenomena, and the emergence of complex oscillatory behavior.

Analytical investigations established the necessary and sufficient conditions for the stability of both the infection-free and endemic fixed points. The analysis revealed that the system exhibits rich dynamical behavior as key parameters—particularly the infection rate parameter B —vary. Specifically, it was shown that the model undergoes both flip (period-doubling) and Neimark-Sacker (secondary Hopf) bifurcations, leading to the onset of periodic and quasiperiodic oscillations, respectively. These bifurcations mark the transition from steady-state infection dynamics to more intricate oscillatory regimes, reflecting the intrinsic instability and nonlinear feedback inherent in HIV-immune system interactions.

The numerical simulations confirmed the analytical predictions and illustrated the system's transition from equilibrium states to limit cycles and chaotic attractors.

The bifurcation diagrams and corresponding Lyapunov exponent spectra provided quantitative evidence of these transitions, clearly demonstrating routes to chaos via period doubling. The coexistence of multiple attractors and sensitivity to parameter perturbations underscore the model's capacity to reproduce biologically relevant patterns such as viral blips, sustained oscillations, and irregular fluctuations in viral load and immune cell concentrations—phenomena observed in clinical data.

From a biological perspective, these results suggest that the complex oscillations and chaotic behaviors observed in HIV dynamics may not solely arise from random fluctuations or external perturbations but could emerge deterministically from the inherent nonlinearities of viral replication and immune response processes. The presence of bifurcation-induced oscillations offers a theoretical explanation for recurrent episodes of low-level viremia, even under conditions of treatment or immune control.

In conclusion, the discrete HIV infection model provides a robust framework for understanding the diverse dynamical regimes underlying HIV pathogenesis. The combination of analytical bifurcation theory and numerical experimentation not only enhances our comprehension of viral-immune dynamics but also offers potential insights into the timing and nature of therapeutic interventions. Future work may extend this model to higher dimensions or include additional biological mechanisms such as drug therapy effects, immune delay responses, and cellular mutation processes to further elucidate the complex temporal behavior of HIV infection.

List of Abbreviations

HIV Human Immunodeficiency Virus

AIDS Acquired Immunodeficiency Syndrome

CD4+ Cluster of Differentiation 4 positive T cells

ODE Ordinary Differential Equation

Declarations

Ethics approval and consent to participate

Not applicable. This study is purely theoretical and does not involve human participants, human data, or animal experiments.

Consent for publication

Not applicable.

Data Availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

A.E. and S.S. designed the study, developed the mathematical model, and performed the analytical derivations. S.R. conducted the numerical simulations and drafted the manuscript. All authors read and approved the final manuscript.

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