



Stability Thresholds and Bounded Hybrid Neural Modifiers in a Fractional HIV-CD4+ T-Cell Model

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Abstract

A commensurate Caputo model of HIV infection of CD4+ T cells is analysed by separating biologically admissible equilibria from algebraic roots. Building on the fractional model of Okeke et al. [1] and the hybrid activation construction of Essang et al. [2], this study introduces bounded neural modifiers of infection and viral production rates. Explicit equilibrium expressions and an infection threshold are derived. The disease-free equilibrium is locally asymptotically stable below the threshold, while a unique positive endemic equilibrium exists and is locally asymptotically stable above it for every common order in $(0,1]$. A recalculation using the parameter values reported in [1] produces a stable endemic equilibrium, contrary to the published numerical classification. A conservative bound on neural modifiers provides a sufficient condition for local disease-free stability across fixed covariate settings. A completed numerical study reports dimensionless fractional trajectories, threshold surfaces, Jacobian spectra and time-step refinement. These results concern mathematical equilibria rather than clinical treatment efficacy.

Keywords: Caputo derivative; HIV dynamics; CD4+ T cells; local stability; hybrid activation; bounded neural modifier.

1. Introduction

Okeke et al. [1] considered a fractional HIV model with healthy cells, infected cells and free virions. Its numerical conclusions motivate a fresh examination of equilibrium algebra and fractional stability. Essang et al. [2] combined sigmoid, ReLU, tanh, leaky ReLU and ELU through nonnegative coefficients summing to one. That construction provides a flexible scalar map but does not itself ensure that a biological rate remains nonnegative or bounded.

This study connects these ideas through a bounded output transformation, while treating the core equilibrium results as mathematical consequences of the model. The contribution is a corrected numerical audit together with an explicit link between modifier bounds and the infection threshold. Neither hybrid activations nor fractional stability criteria are claimed as new inventions. Adaptive activations have already been studied in physics-informed networks [4].

2. Related work and contribution boundaries

The experimental observations reported by Ho et al. [12] and Perelson et al. [11] helped establish the relevance of rapid viral turnover and infected-cell loss in within-host HIV models. These studies concern biological observations, whereas the present calculations concern a deliberately simplified mathematical system. The survey by Perelson and Nelson [13] explains why model structure and interpretation must be considered together. In particular, replacing an ordinary derivative by a fractional operator does not, by itself, demonstrate improved agreement with patient measurements.

Fractional HIV dynamics have already been investigated by Arafa et al. [23]. Their model and numerical setting differ from the logistic system examined here, but their work establishes that fractional HIV modelling predates this study. The supplied study [1] motivates the particular equilibrium audit. For the mathematical background, I use the Caputo initial-value formulation described by Diethelm [6] and the nonlinear linearisation result of Cong et al. [3]. The threshold terminology is consistent with the next-generation approach developed by van den Driessche and Watmough [10], although the threshold in this manuscript is obtained directly from the two-dimensional infection block.

Several numerical methods are available for fractional systems. Diethelm et al. [7] developed an Adams predictor-corrector approach, and Garrappa [9] discusses implementation and verification issues across fractional solvers. I use an implicit L1 discretisation and report refinement differences rather than asserting an unverified formal convergence rate. The nonsmooth-data analysis of Jin et al. [8] provides a reason to treat the initial-time behaviour carefully; its subdiffusion results are not presented as a convergence theorem for this nonlinear HIV system.

The neural construction also has a substantial prior history. Glorot and Bengio [14] related training difficulty to activation saturation and Jacobian conditioning; rectifier networks were studied by Glorot et al. [15], and exponential linear units by Clevert et al. [16]. Trainable activations are reviewed by Apicella et al. [22]. Jagtap et al. [4] investigate adaptive activations for physics-informed problems. The mixture used here is explicitly attributed to Essang et al. [2]. Accordingly, I do not claim that combining these elementary nonlinearities is a new activation principle.

Physics-informed neural networks [21] and their fractional extension [5] provide possible estimation tools. Optimisers such as Adam [17] can support such a future implementation, but no optimiser or network is trained in the numerical results reported here. The bounded neural architecture is analysed symbolically; the plotted modifier surface is the exact threshold formula evaluated on a grid. Structural identifiability [19], profile likelihood [18] and identifiable combinations [20] are relevant to subsequent calibration because a mathematically stable model can still possess parameters that data cannot uniquely determine.

The contribution is therefore specific: I provide a transparent correction of the supplied model's endemic equilibrium and Jacobian, a complete cubic stability calculation, and a bounded-modifier threshold certificate. These results should be assessed as an analytical audit and extension. The literature considered here does not establish that every formula is unprecedented, and no claim of clinical novelty is made.

3. Model and assumptions

Time and states are nondimensionalised before introducing fractional differentiation. Consequently, the numerical values in Section 5 are an algebraic reproduction of the published vector field, not a dimensional calibration of fractional biological rates.

Let $0 < q \leq 1$ and define, for $0 < q < 1$,

$${}^C D_t^q x(t) = \frac{1}{\Gamma(1-q)} \int_0^t (t-u)^{-q} x'(u) du.$$

At $q=1$ the ordinary derivative is used. Consider

$${}^C D_t^q T = s + (\alpha - d)T - \frac{\alpha}{K} T^2 - bTV, \quad (1)$$

$${}^C D_t^q I = bTV - \delta I, \quad {}^C D_t^q V = pI - cV. \quad (2)$$

All eight coefficients $s, \alpha, d, K, b, \delta, p$ and c are strictly positive. T, I and V represent uninfected CD4+ cells, infected cells and virions. Initial conditions are nonnegative. The smooth vector field is locally Lipschitz. Its values on the coordinate boundaries are respectively s, bTV and pI , all nonnegative; the usual positivity result for quasipositive Caputo systems applies. Only local equilibrium stability is asserted here; no global convergence theorem is claimed.

For a fixed covariate vector z , define

$$H_w(u) = w_1 \sigma(u) + w_2 \text{ReLU}(u) + w_3 \tanh(u) + w_4 \text{LReLU}_a(u) + w_5 \text{ELU}_\kappa(u), \quad (3)$$

where $w_j \geq 0, \sum w_j = 1$ and $a, \kappa > 0$. Following the mixture idea in [2], a network N_θ built from H_w may specify

$$U_\theta(z) = u_- + (u_+ - u_-) \sigma(N_\theta(z)), \quad 0 \leq u_- < u_+ < 1. \quad (4)$$

Similarly define W_θ with bounds v_- and v_+ . Set $b = \beta(1 - U_\theta(z))$ and $p = \rho(1 - W_\theta(z))$, with $\beta, \rho > 0$. For each fixed z these are constant positive rates. This is a bounded mathematical modifier architecture. Its output is not a drug recommendation. The analysis does not apply unchanged to time-varying policies or state feedback.

4. Admissible equilibria and infection threshold

Write $f(T) = s + (\alpha - d)T - \alpha T^2/K$. Its positive root is

$$T_0 = \frac{K}{2\alpha} \left[(\alpha - d) + \sqrt{(\alpha - d)^2 + 4\alpha s/K} \right]. \quad (5)$$

The other root is negative because $s > 0$. It is excluded from the biological state space. Define

$$\mathcal{R} = \frac{bpT_0}{c\delta}. \quad (6)$$

Proposition 1. The disease-free equilibrium is $E_0 = (T_0, 0, 0)$. A positive endemic equilibrium exists precisely when $\mathcal{R} > 1$ and is unique. It is

$$T_* = \frac{c\delta}{bp}, \quad V_* = \frac{f(T_*)}{bT_*}, \quad I_* = \frac{cV_*}{p}. \quad (7)$$

Proof. If $V=0$, equations (2) imply $I=0$, and the only nonnegative root of $f(T)=0$ is T_0 . If $V>0$, equations (2) yield $I=bTV/\delta$ and $pI=cV$, hence $T=T_0$. Equation (1) then gives V . The concave polynomial f has a negative root and the positive root T_0 , so $f(T)>0$ for $0<T<T_0$. Therefore $V>0$ exactly when $T<T_0$, equivalently $R>1$. Each coordinate is then uniquely determined. $\square$

5. Local stability

For a smooth commensurate Caputo system the strict spectral condition $\text{arg } \lambda > q\pi/2$ implies local asymptotic stability [3]. In particular, a Hurwitz Jacobian satisfies this condition for every $q \in (0, 1]$.

Theorem 1. E_0 is locally asymptotically stable when $R<1$. When $R>1$ its linearisation has a positive real eigenvalue. The case $R=1$ is nonhyperbolic and is not settled by the strict spectral criterion.

Proof. The first eigenvalue is

$$f'(T_0) = -\sqrt{(\alpha - d)^2 + 4\alpha s/K} < 0.$$

The remaining two satisfy

$$\lambda^2 + (c + \delta)\lambda + c\delta(1 - R) = 0. \quad (8)$$

When $R<1$ the coefficients give roots with negative real parts. When $R>1$ the constant coefficient is negative and the roots are real with opposite signs. $\square$

Theorem 2. Whenever $R>1$, E^* is locally asymptotically stable for every common fractional order $0 < q \leq 1$.

Proof. Put $A=s/T+\alpha T/K>0$, $B=bV>0$ and $D=c+\delta$. Using $f(T)=bTV$, the Jacobian is

$$J_* = \begin{pmatrix} -A & 0 & -bT_* \\ B & -\delta & bT_* \\ 0 & p & -c \end{pmatrix}.$$

Since $bpT^*=c\delta$, its characteristic polynomial is

$$\lambda^3 + (A + D)\lambda^2 + (AD + bT_*B)\lambda + bcT_*B. \quad (9)$$

All coefficients are positive, and the remaining cubic Hurwitz inequality follows from

$$(A + D)(AD + bT_*B) - bcT_*B = AD(A + D) + bT_*B(A + \delta) > 0.$$

Thus all roots have negative real parts. The fractional spectral condition proves the claim. $\square$

Corollary 1. Let $R_{\text{base}} = \beta\rho T_0/(c\delta)$. If

$$\mathcal{R}_{\text{base}}(1 - u_-)(1 - v_-) < 1, \quad (10)$$

then the disease-free equilibrium is locally asymptotically stable at every fixed covariate setting allowed by (4).

Proof. Both modifier factors are at most their values at the lower bounds, so $R \leq R_{\text{base}}(1 - u_-)(1 - v_-)$. Apply Theorem 1 pointwise. $\square$

This is a pointwise equilibrium guarantee, not a theorem of uniform stability under arbitrary switching or fractional state feedback.

6. Reproducible numerical audit

Use the values in [1]: $s=5$, $d=0.01$, $\alpha=6.8$, $K=1300$, $b=0.0002$, $\delta=0.5$, $p=1000$ and $c=10$. Direct calculation gives

Quantity	Recomputed value
T_0	1298.824195
R	51.952968
T^*	25.000000
I^*	342.961538
V^*	34296.153846

The corrected endemic Jacobian is

$$\begin{pmatrix} -0.330769231 & 0 & -0.005 \\ 6.859230769 & -0.5 & 0.005 \\ 0 & 1000 & -10 \end{pmatrix},$$

with eigenvalues approximately -10.80314473 and $-0.01381225 \pm 1.78169993i$. Its stability agrees with Theorem 2. The positive disease-free root remains unstable at these parameters. The negative disease-free root has no biological interpretation. Thus the numerical statement that all three algebraic equilibria are unstable in [1] does not describe the corrected positive endemic equilibrium of equations (1)–(2).

With $W=0$, the local disease-free condition is $U > 1 - 1/R_{\text{base}} \approx 0.9807518$. This number is a property of the illustrative parameter set; it is not a clinical efficacy estimate.

7. Dimensional consistency and fractional interpretation

A fractional derivative of a dimensional state has units of state divided by time to the power q . Consequently, importing all ordinary differential-equation rates unchanged into a fractional equation creates a dimensional inconsistency unless a reference time or a rate rescaling is specified. Let physical time be $t_{\text{phys}} = t_{\text{ref}}$ times t , where t is dimensionless. Let each compartment be divided by a positive reference concentration. The coefficients in the resulting dimensionless vector field can be studied consistently for any q .

I follow this convention for the illustrative simulations. The parameter values copied from [1] are used only to audit the algebraic vector field printed there. The corrected numbers do not constitute a re-estimation of biological rates. In particular, the proliferation value and viral production coefficient in that table must not be interpreted as validated fractional rates in a patient model.

For q below one, the Caputo derivative includes the full time history. Its kernel gives greater weight to recent increments while retaining a slowly decaying memory of earlier increments. The resulting trajectories are not obtained by a simple rescaling of the ordinary time axis. The equilibrium equations remain the same because the derivative of a constant is zero. This distinction explains why the stability classification can remain unchanged while transient trajectories differ.

8. A robust certificate under coefficient uncertainty

Suppose the base coefficients β , ρ , c and δ are known only within positive intervals. Let $\beta_{\max}$ and $\rho_{\max}$ be their upper bounds, and $c_{\min}$ and $\delta_{\min}$ their lower bounds. Suppose further that the positive healthy-cell equilibrium is bounded above by $T_{\max 0}$ over the admissible demographic coefficients. The latter bound can be computed from formula (5) or obtained by maximising that expression over a specified parameter box.

Proposition 2. If

$$\frac{\beta_{\max} \rho_{\max} T_{\max 0}}{c_{\min} \delta_{\min}} (1 - u_-)(1 - v_-) < 1, \quad (11)$$

then every fixed admissible parameter vector and every fixed admissible covariate setting has a locally asymptotically stable disease-free equilibrium.

Proof. Positivity of the interval bounds gives

$$\frac{\beta \rho T_0}{c \delta} (1 - U)(1 - W) \leq \frac{\beta_{\max} \rho_{\max} T_{\max 0}}{c_{\min} \delta_{\min}} (1 - u_-)(1 - v_-).$$

The strict inequality and Theorem 1 establish the conclusion for each admissible frozen system. The argument does not invoke a common Lyapunov function and therefore does not imply stability under arbitrary time variation. This distinction is essential for systems with memory. $\square$

If the demographic parameters also vary, a conservative $T_{\max 0}$ can be obtained directly. For fixed K and α , T_0 increases with s and decreases with d . If α and K vary simultaneously, using an independently validated upper bound avoids an unsupported monotonicity assumption. The certificate is sufficient rather than necessary: a failed upper-bound test may reflect conservatism instead of actual instability.

9. Numerical method and reproducibility

I discretise the common Caputo order on a uniform grid $t_n = nh$. For q below one, the L1 formula is

$$\mathcal{D}_h^q x_n = \frac{1}{\Gamma(2 - q)h^q} \sum_{j=0}^{n-1} a_j (x_{n-j} - x_{n-j-1}), \quad a_j = (j + 1)^{1-q} - j^{1-q}. \quad (12)$$

At each time level I solve the three-dimensional implicit nonlinear equation $D_h^q x_n = F(x_n)$. The previous state supplies the initial iterate, and the analytical Jacobian supplies the derivative in the nonlinear solver. The accepted equation residual is required to be below 10^{-7} in the infinity norm. At $q=1$, the procedure reduces to backward Euler. I do not clip negative states; instead, the program rejects any computed value below a small numerical tolerance.

The history sum is evaluated directly. This gives quadratic total history work in the number of time steps and linear storage of the trajectory. The small three-dimensional solve is sufficient for the present experiments, but a faster history algorithm would be appropriate for substantially longer records. The implemented method is independently checked at $q=1$ against a high-accuracy DOP853 ordinary differential-equation solution. For fractional orders, I report comparisons with a finer L1 grid; these comparisons establish numerical consistency of the plotted calculations, not a rigorous error bound against an exact nonlinear solution.

The illustrative coefficient set is $s=0.1$, $\alpha=0.8$, $d=0.1$, $K=2$, $\beta=0.5$, $\delta=0.4$, $\rho=1$ and $c=0.8$. The initial state is $(1.5, 0.05, 0.1)$. All values are dimensionless. I use $q=0.6, 0.8, 1$ and $h=0.025$ on the interval $[0, 40]$ for the trajectory figure. Refinement comparisons use

$h=0.1, 0.05, 0.025, 0.0125$ against $h=0.00625$ on $[0, 10]$. CSV files, the calculation script and numerical results accompany the manuscript.

10. Numerical results and interpretation

For the illustrative parameter set, the disease-free healthy-cell value is 1.882782219 and the threshold is 2.941847216. The endemic state is $(0.64, 0.9604, 1.2005)$. Its eigenvalues are approximately -1.351355484 and -0.130447258 plus or minus $0.353726276i$. Their real parts are strictly negative, agreeing with the parameter-independent Hurwitz proof.

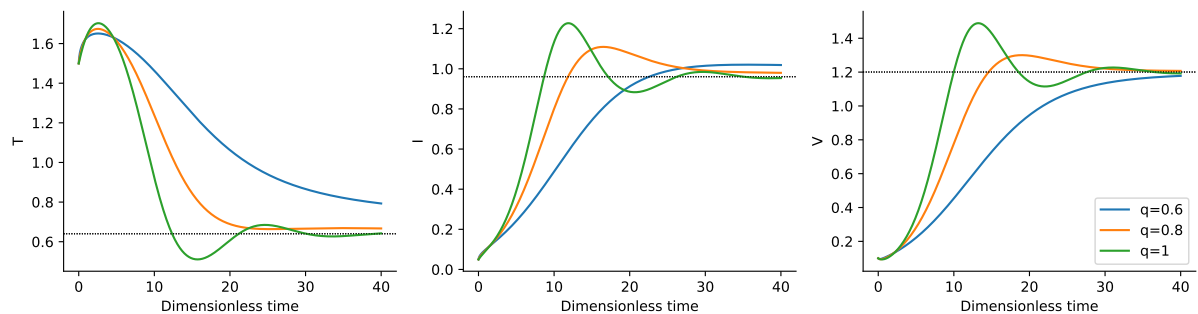


Figure 1: Computed dimensionless trajectories for $q=0.6, 0.8$ and 1 with $h=0.025$. Dotted horizontal lines indicate the common endemic equilibrium. These are synthetic model trajectories, not observations of patients.

The finite-horizon curves illustrate different transient responses despite the same equilibrium. The ordinary-order trajectory exhibits a visibly oscillatory approach for this parameter set. Lower-order curves retain longer transients. A plotted approach over a finite interval is not a proof of global convergence; the stability conclusion remains the local result in Theorem 2.

The boundary in the modifier plane is hyperbolic because the two factors enter multiplicatively. If the production modifier is fixed at zero, the infection modifier must exceed approximately 0.66008 for the illustrative coefficients. For the audited values from [1], the corresponding threshold is approximately 0.98075. The difference demonstrates dependence on the selected coefficients; it cannot be interpreted as a universal treatment target.

At $h=0.0125$, the largest state differences against the finer grid are 0.00120949 for $q=0.6$, 0.00041343 for $q=0.8$ and 0.00110639 for $q=1$. The values decrease across every reported refinement sequence. For $q=1$, the largest discrepancy against the independent DOP853 solution is 0.00221351. The latter check is especially useful because it tests the implementation against a different integration algorithm rather than against itself.

11. Limitations, interpretation and reporting scope

The proofs assume strictly positive constant coefficients and a common order. Unequal fractional orders require a different stability calculation. The system omits latent reservoirs, immune effector dynamics, spatial structure, adherence variation and viral mutation. It also retains a simplified mass-action infection term and logistic proliferation of only the uninfected compartment. These choices are appropriate for an audit of the supplied equations, but they restrict biological interpretation.

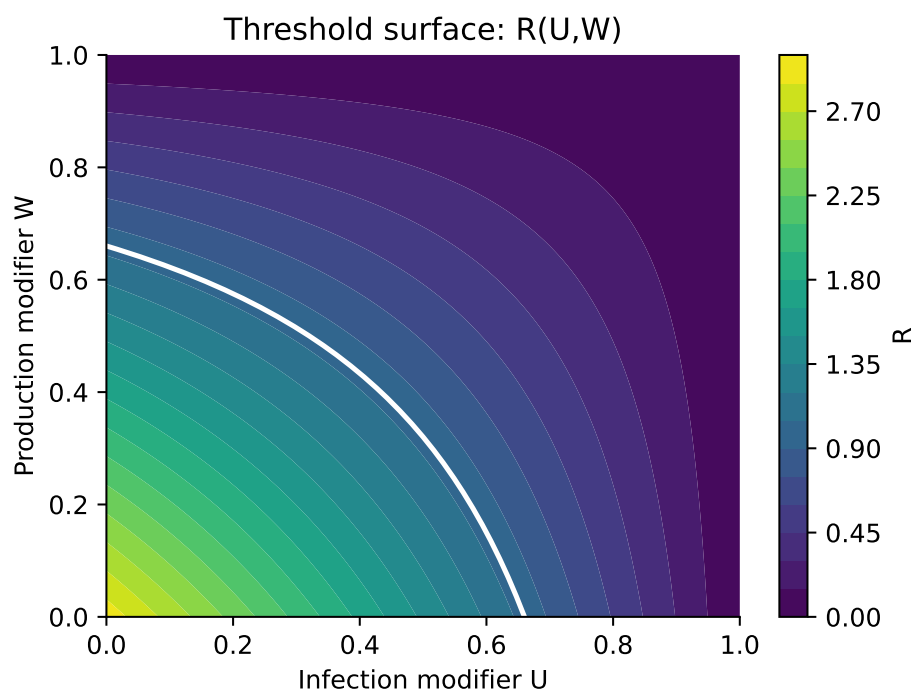


Figure 2: Exact modifier threshold surface for the illustrative coefficients. The white contour is $R=1$; only frozen systems in the region $R<1$ meet the local disease-free condition.

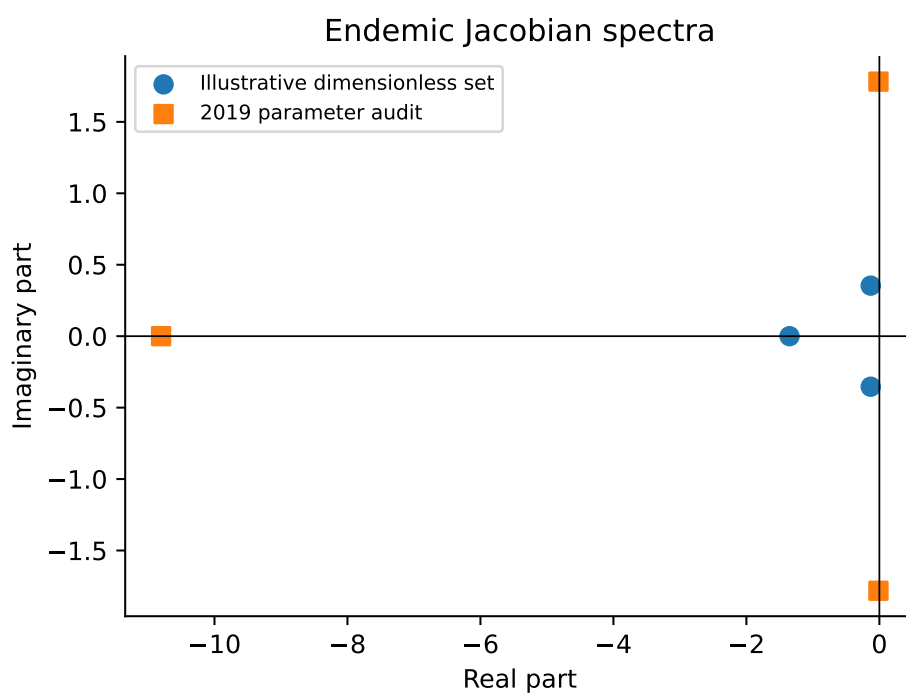


Figure 3: Endemic Jacobian spectra for the illustrative coefficients and the audited values from [1]. All plotted eigenvalues are strictly in the left half-plane.

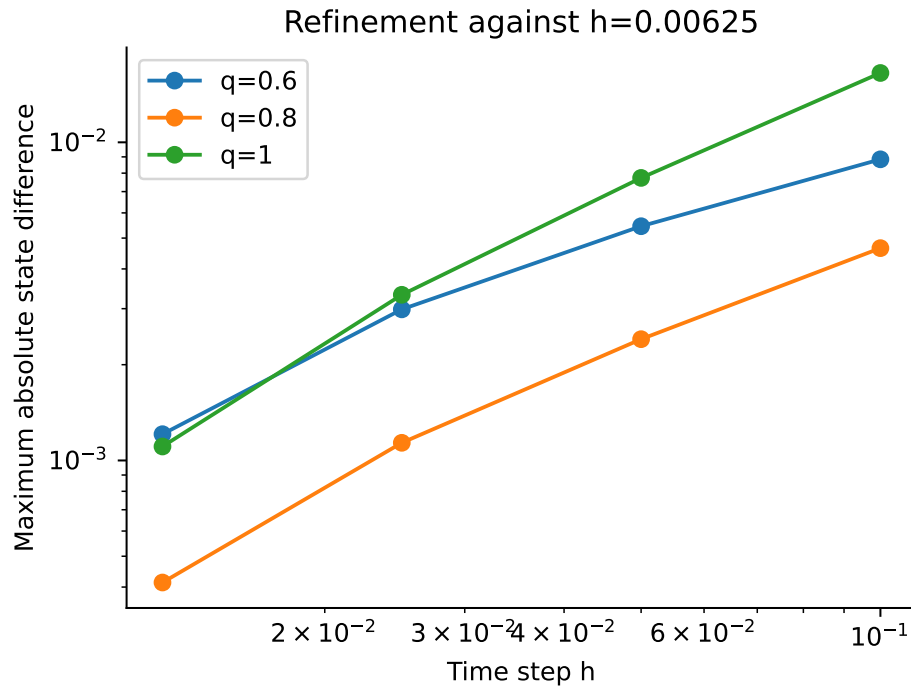


Figure 4: Maximum state discrepancies relative to the $h=0.00625$ L1 trajectory on $[0,10]$. Each curve decreases under refinement. The reference is itself numerical and does not supply an exact fractional solution.

The neural modifier is bounded by construction, but neither its covariates nor its weights are estimated here. The plots therefore do not validate a learned intervention policy. If the architecture is fitted later, an identifiability study and out-of-sample assessment must precede claims about personalised prediction. Different parameter combinations can produce indistinguishable trajectories, as established generally in identifiable-combination work [20]. The use of a flexible network can compound rather than remove that difficulty.

The numerical evidence supports the stated analytical and implementation claims. It does not establish superiority over other fractional solvers, superiority of hybrid activations over tanh in supervised learning, or better clinical prediction than an ordinary differential model. I present the corrected equilibrium analysis as a reproducible mathematical result that can serve as a sound basis for those later comparisons.

12. Conclusion

The model has one biologically meaningful disease-free equilibrium and, above the infection threshold, one positive endemic equilibrium. The endemic equilibrium is Hurwitz for every positive parameter choice for which it exists. A bounded hybrid neural output connects the activation construction in [2] to an explicit sufficient condition for local disease-free stability. The corrected parameter audit identifies a concrete error to resolve in subsequent work based on [1].

13. Declarations

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Ethics and consent: This study used synthetic computational data and involved no human participants or identifiable personal data. Ethical approval and informed consent were therefore not required.

Author contributions: Okeke Ikenna Stephen conducted the research, developed the methodology, performed the mathematical analysis and numerical simulations, and prepared the manuscript. The author reviewed and approved the final version for publication.

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