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A POSITIVITY- AND THRESHOLD-PRESERVING NONSTANDARD FINITE DIFFERENCE SCHEME FOR A RESERVOIR-DRIVEN VISCERAL LEISHMANIASIS MODEL

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Abstract. A dynamically consistent numerical method for an epidemic model should reproduce the main qualitative features of the underlying continuous system. This requirement is especially important in vector-borne infections with reservoir transmission, where standard explicit methods may create negative populations, spurious oscillations, or step-size-dependent threshold behavior. This paper develops and analyzes a nonstandard finite difference (NSFD) scheme for a structured model of visceral leishmaniasis involving humans, sandfly vectors, and animal reservoir hosts. The continuous model includes human latency, treatment, temporary immunity, vector incubation, asymptomatic and symptomatic reservoir infection, host-attractiveness-based bite allocation, and density-regulated vector demography. Positivity, boundedness, a positively invariant feasible region, the disease-free equilibrium, the basic reproduction number, and local stability of the disease-free equilibrium are established for the continuous model. The basic reproduction number decomposes into human-vector and reservoir-vector transmission contributions, giving a direct interpretation of the two infection loops that can sustain invasion.

A structure-preserving NSFD discretization is then constructed using a denominator function and nonlocal approximations of incidence, transfer, and density-dependent vector-regulation terms. The discrete scheme is shown to preserve nonnegativity and the biological bounds for all admissible time steps, to share the same disease-free equilibrium as the continuous model, and to preserve the threshold condition determined by the next-generation number. In particular, the disease-free equilibrium of the discrete system is locally asymptotically stable when

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$R_0 < 1$ and unstable when $R_0 > 1$. Numerical experiments compare the NSFD method with forward Euler discretization under subcritical and supercritical transmission regimes. The results illustrate that the proposed scheme preserves biologically meaningful trajectories and threshold behavior for larger time steps, whereas Euler discretization can lose positivity or become unstable. The framework provides a reliable computational basis for reservoir-driven leishmaniasis models and related vector-borne disease systems.

Keywords: nonstandard finite difference scheme; visceral leishmaniasis; vector-host-reservoir model; positivity preservation; threshold preservation; dynamically consistent discretization.

2020 AMS Subject Classification: 34D05, 65L20.

1. INTRODUCTION

Leishmaniasis is a neglected tropical disease caused by protozoan parasites of the genus *Leishmania* and transmitted through the bites of infected female phlebotomine sandflies. The disease has several clinical manifestations, including cutaneous, mucocutaneous, and visceral leishmaniasis. Visceral leishmaniasis is the most severe form and may be fatal when untreated. The World Health Organization reports that more than 20 *Leishmania* species can infect humans and that more than 90 sandfly species are known to transmit *Leishmania* parasites [15]. This biological diversity partly explains why leishmaniasis control remains difficult: transmission depends on host infection, vector abundance, reservoir availability, feeding preference, environmental suitability, and intervention coverage.

A central feature of many leishmaniasis systems is the presence of non-human reservoirs. In zoonotic visceral leishmaniasis, domestic dogs are often important reservoir hosts, while other mammals may contribute to local transmission in particular ecological settings. Consequently, infection can persist even when human diagnosis and treatment are improved, because the parasite may continue circulating through a reservoir-vector cycle. A model that ignores reservoir hosts may therefore underestimate persistence mechanisms and overestimate the effectiveness of human-only interventions.

Mathematical models have contributed substantially to the analysis of leishmaniasis transmission and control. Existing studies have considered human-vector dynamics, dog-vector transmission, asymptomatic infection, vertical transmission in dogs, treatment, vaccination, culling, vector control, and environmental drivers [6, 3, 4, 5, 8, 12, 16, 11]. These works show that the

basic reproduction number and the expected impact of control strategies can change substantially when reservoir infection is included. They also indicate that zoonotic visceral leishmaniasis is not adequately represented by a simple two-population host-vector model.

Continuous-time ordinary differential equations provide a natural framework for model construction and qualitative analysis. However, numerical simulation is needed for scenario exploration, parameter sensitivity, bifurcation studies, and intervention assessment. The difficulty is that qualitative properties of the continuous system are not automatically inherited by standard numerical methods. A forward Euler method, for example, may generate negative state variables, artificial oscillations, incorrect long-term behavior, or stability properties that depend on the time step. These failures are not merely computational; in epidemiological models they can lead to biologically impossible interpretations.

Nonstandard finite difference schemes, introduced and developed by Mickens, provide a systematic way to construct discrete models that preserve essential properties of differential equations [9, 10]. The central principles are to replace the classical denominator h by a denominator function $\phi(h)$ satisfying $\phi(h) = h + O(h^2)$ and to approximate nonlinear terms in a nonlocal manner. In epidemiological models, these ideas have been used to preserve positivity, boundedness, equilibria, and threshold conditions associated with the basic reproduction number [1, 14, 7].

The present paper develops a dynamically consistent NSFD scheme for a biologically structured vector-host-reservoir model of visceral leishmaniasis. The model distinguishes susceptible, exposed, clinically infected, treated, and recovered humans; susceptible, exposed, and infectious sandflies; and susceptible, exposed, asymptomatic infectious, and symptomatic infectious reservoir hosts. The biting process is described by an attractiveness-weighted allocation mechanism so that sandfly bites are distributed between humans and reservoir hosts according to host abundance and relative attractiveness. Vector demography is represented through density-dependent mortality, which produces logistic regulation at the total vector-population level while retaining a compartmentwise gain-loss structure suitable for positivity preservation.

The main contribution is twofold. First, the continuous model is formulated and analyzed in a way that separates the human-vector and reservoir-vector contributions to R_0 . This decomposition clarifies how a reservoir cycle can sustain transmission even when human transmission is weak. Second, a discrete NSFD model is constructed so that nonnegativity, boundedness, equilibrium consistency, and the local threshold behavior of the disease-free equilibrium are preserved for all admissible step sizes. The threshold-preservation proof is formulated through a regular splitting and the nonsingular M -matrix structure of the next-generation linearization.

The paper is organized as follows. Section 2 formulates the continuous model. Section 3 establishes the continuous-model properties. Section 4 constructs the NSFD discretization. Section 5 proves the main qualitative properties of the discrete model. Section 6 presents numerical comparisons with the Euler method. Section 7 concludes the paper.

2. MODEL FORMULATION

Let S_h, E_h, I_h, T_h, R_h denote susceptible, exposed, clinically infected, treated, and recovered humans, respectively. The total human population is

$$N_h = S_h + E_h + I_h + T_h + R_h.$$

Let S_v, E_v, I_v denote susceptible, exposed, and infectious sandfly vectors, with

$$N_v = S_v + E_v + I_v.$$

Let S_r, E_r, A_r, I_r denote susceptible, exposed, asymptomatic infectious, and symptomatic infectious reservoir hosts, with

$$N_r = S_r + E_r + A_r + I_r.$$

Sandfly bites are allocated between humans and reservoir hosts according to host abundance and attractiveness. Let $q_h > 0$ and $q_r > 0$ be the attractiveness weights of humans and reservoir hosts. Define

$$\mathcal{H} = q_h N_h + q_r N_r.$$

The forces of infection are

$$(1) \quad \lambda_h = ab_{vh} \frac{q_h I_v}{\mathcal{H}},$$

$$(2) \quad \lambda_r = ab_{vr} \frac{q_r I_v}{\mathcal{H}},$$

$$(3) \quad \lambda_v = a \frac{q_h c_h I_h + q_r (c_A A_r + c_I I_r)}{\mathcal{H}}.$$

Here a is the average biting rate per sandfly; b_{vh} and b_{vr} are transmission probabilities from vector to human and vector to reservoir, respectively; c_h, c_A, c_I are transmission probabilities from infectious humans, asymptomatic infectious reservoirs, and symptomatic infectious reservoirs to susceptible sandflies. We assume

$$0 \leq c_A \leq c_I \leq 1,$$

which allows symptomatic reservoir hosts to be more infectious to vectors than asymptomatic reservoirs.

To retain logistic regulation at the total vector-population level while preserving compartment-wise positivity, vector demography is written with linear recruitment into the vector population and density-dependent mortality acting on all vector classes. Thus

$$\frac{dN_v}{dt} = (r_v - \mu_v)N_v - \delta_v N_v^2,$$

and the vector carrying capacity is

$$K_v = \frac{r_v - \mu_v}{\delta_v}, \quad r_v > \mu_v, \quad \delta_v > 0.$$

The full model is

$$(4) \quad \begin{aligned} \frac{dS_h}{dt} &= \Lambda_h - \lambda_h S_h - \mu_h S_h + \omega_h R_h, \\ \frac{dE_h}{dt} &= \lambda_h S_h - (\sigma_h + \mu_h) E_h, \\ \frac{dI_h}{dt} &= \sigma_h E_h - (\tau_h + \gamma_h + \mu_h + \alpha_h) I_h, \\ \frac{dT_h}{dt} &= \tau_h I_h - (\eta_h + \mu_h) T_h, \\ \frac{dR_h}{dt} &= \gamma_h I_h + \eta_h T_h - (\omega_h + \mu_h) R_h, \\ \frac{dS_v}{dt} &= r_v N_v - \lambda_v S_v - \mu_v S_v - \delta_v N_v S_v, \\ \frac{dE_v}{dt} &= \lambda_v S_v - (\sigma_v + \mu_v) E_v - \delta_v N_v E_v, \end{aligned}$$

$$\begin{aligned}
\frac{dI_v}{dt} &= \sigma_v E_v - \mu_v I_v - \delta_v N_v I_v, \\
\frac{dS_r}{dt} &= \Lambda_r - \lambda_r S_r - \mu_r S_r, \\
\frac{dE_r}{dt} &= \lambda_r S_r - (\sigma_r + \mu_r) E_r, \\
\frac{dA_r}{dt} &= p_r \sigma_r E_r - (\kappa_r + \mu_r) A_r, \\
\frac{dI_r}{dt} &= (1 - p_r) \sigma_r E_r + \kappa_r A_r - (\mu_r + \alpha_r) I_r.
\end{aligned}$$

For compactness, define

$$\begin{aligned}
D_{E_h} &= \sigma_h + \mu_h, & D_{I_h} &= \tau_h + \gamma_h + \mu_h + \alpha_h, & D_{T_h} &= \eta_h + \mu_h, \\
D_{E_r} &= \sigma_r + \mu_r, & D_{A_r} &= \kappa_r + \mu_r, & D_{I_r} &= \mu_r + \alpha_r.
\end{aligned}$$

At the disease-free vector equilibrium, the vector removal rates are

$$D_{E_v}^0 = \sigma_v + \mu_v + \delta_v K_v, \quad D_{I_v}^0 = \mu_v + \delta_v K_v.$$

TABLE 1. State variables of the leishmaniasis transmission model.

Variable	Description
S_h, E_h, I_h, T_h, R_h	Susceptible, exposed, clinically infected, treated, and recovered humans
N_h	Total human population
S_v, E_v, I_v	Susceptible, exposed, and infectious sandfly vectors
N_v	Total vector population
S_r, E_r, A_r, I_r	Susceptible, exposed, asymptomatic infectious, and symptomatic infectious reservoirs
N_r	Total reservoir population

3. PROPERTIES OF THE CONTINUOUS MODEL

Let

$$X = (S_h, E_h, I_h, T_h, R_h, S_v, E_v, I_v, S_r, E_r, A_r, I_r)^T.$$

TABLE 2. Parameters of the model.

Parameter	Description	Unit
Λ_h, Λ_r	Recruitment rates of humans and reservoirs	population time ⁻¹
μ_h, μ_v, μ_r	Natural death rates of humans, vectors, and reservoirs	time ⁻¹
α_h, α_r	Disease-induced death rates of humans and reservoirs	time ⁻¹
$\sigma_h, \sigma_v, \sigma_r$	Progression rates from exposed classes	time ⁻¹
τ_h	Treatment initiation rate for clinically infected humans	time ⁻¹
γ_h, η_h	Recovery rates of infected and treated humans	time ⁻¹
ω_h	Loss of human immunity	time ⁻¹
r_v	Intrinsic vector recruitment parameter	time ⁻¹
δ_v	Density-dependent vector mortality coefficient	vector ⁻¹ time ⁻¹
K_v	Vector carrying capacity, $(r_v - \mu_v)/\delta_v$	vectors
a	Average biting rate per sandfly	bites vector ⁻¹ time ⁻¹
q_h, q_r	Human and reservoir attractiveness weights	dimensionless
b_{vh}, b_{vr}	Transmission probabilities from vector to human/reservoir	dimensionless
c_h, c_A, c_I	Transmission probabilities from infectious hosts to vectors	dimensionless
p_r	Fraction of exposed reservoirs becoming asymptomatic infectious	dimensionless
κ_r	Progression rate from asymptomatic to symptomatic reservoir infection	time ⁻¹

The biologically feasible state space is

$$\mathbb{R}_+^{12} = \{X \in \mathbb{R}^{12} : X_i \geq 0, i = 1, \dots, 12\}.$$

Throughout the analysis all parameters are nonnegative, with

$$q_h > 0, \quad q_r > 0, \quad a > 0, \quad r_v > \mu_v, \quad \delta_v > 0,$$

and the initial data are nonnegative with $N_h(0) + N_r(0) > 0$.

3.1. Existence, uniqueness, and positivity.

Theorem 3.1. *For every initial condition $X(0) \in \mathbb{R}_+^{12}$ satisfying $N_h(0) + N_r(0) > 0$, model (4) has a unique local solution. Moreover, this solution remains nonnegative and can be extended for all $t \geq 0$.*

Proof. The right-hand side of (4) consists of polynomial terms and rational incidence terms with denominator $\mathcal{H} = q_h N_h + q_r N_r$. In the region where $\mathcal{H} > 0$, the vector field is locally Lipschitz. Thus a unique local solution exists.

It remains to verify positive invariance. On each boundary face of $\mathbb{R}_+^{12}$, the corresponding derivative is nonnegative:

$$\left. \frac{dS_h}{dt} \right|_{S_h=0} = \Lambda_h + \omega_h R_h \geq 0, \quad \left. \frac{dE_h}{dt} \right|_{E_h=0} = \lambda_h S_h \geq 0, \quad \left. \frac{dI_h}{dt} \right|_{I_h=0} = \sigma_h E_h \geq 0,$$

$$\left. \frac{dT_h}{dt} \right|_{T_h=0} = \tau_h I_h \geq 0, \quad \left. \frac{dR_h}{dt} \right|_{R_h=0} = \gamma_h I_h + \eta_h T_h \geq 0,$$

$$\left. \frac{dS_v}{dt} \right|_{S_v=0} = r_v N_v \geq 0, \quad \left. \frac{dE_v}{dt} \right|_{E_v=0} = \lambda_v S_v \geq 0, \quad \left. \frac{dI_v}{dt} \right|_{I_v=0} = \sigma_v E_v \geq 0,$$

and

$$\left. \frac{dS_r}{dt} \right|_{S_r=0} = \Lambda_r \geq 0, \quad \left. \frac{dE_r}{dt} \right|_{E_r=0} = \lambda_r S_r \geq 0, \quad \left. \frac{dA_r}{dt} \right|_{A_r=0} = p_r \sigma_r E_r \geq 0,$$

$$\left. \frac{dI_r}{dt} \right|_{I_r=0} = (1 - p_r) \sigma_r E_r + \kappa_r A_r \geq 0.$$

The vector field is therefore quasi-positive, and $\mathbb{R}_+^{12}$ is positively invariant. The boundedness estimates below rule out finite-time blow-up, so the solution extends globally. $\square$

3.2. Boundedness and feasible region. Adding the human equations gives

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \alpha_h I_h \leq \Lambda_h - \mu_h N_h.$$

Therefore

$$N_h(t) \leq N_h(0)e^{-\mu_h t} + \frac{\Lambda_h}{\mu_h} (1 - e^{-\mu_h t}), \quad \limsup_{t \rightarrow \infty} N_h(t) \leq \frac{\Lambda_h}{\mu_h}.$$

Similarly,

$$\frac{dN_r}{dt} = \Lambda_r - \mu_r N_r - \alpha_r I_r \leq \Lambda_r - \mu_r N_r,$$

and hence

$$\limsup_{t \rightarrow \infty} N_r(t) \leq \frac{\Lambda_r}{\mu_r}.$$

For vectors,

$$\frac{dN_v}{dt} = (r_v - \mu_v)N_v - \delta_v N_v^2 = (r_v - \mu_v)N_v \left(1 - \frac{N_v}{K_v}\right),$$

so

$$\limsup_{t \rightarrow \infty} N_v(t) \leq K_v.$$

Define

$$(5) \quad \Omega = \left\{ X \in \mathbb{R}_+^{12} : N_h \leq \frac{\Lambda_h}{\mu_h}, \quad N_v \leq K_v, \quad N_r \leq \frac{\Lambda_r}{\mu_r} \right\}.$$

Theorem 3.2. *The region Ω is positively invariant for model (4). Every nonnegative solution is ultimately bounded and satisfies the three limsup estimates above.*

Proof. On the boundary $N_h = \Lambda_h/\mu_h$,

$$\frac{dN_h}{dt} = -\alpha_h I_h \leq 0.$$

On the boundary $N_r = \Lambda_r/\mu_r$,

$$\frac{dN_r}{dt} = -\alpha_r I_r \leq 0.$$

On the boundary $N_v = K_v$,

$$\frac{dN_v}{dt} = 0,$$

and if $N_v > K_v$, then $dN_v/dt < 0$. Thus no trajectory starting in Ω crosses outward. The attracting estimates follow from the comparison inequalities for N_h, N_r and the logistic equation for N_v . $\square$

3.3. Disease-free equilibrium and reproduction number. At the disease-free equilibrium, all infected, exposed, treated, and recovered classes vanish. The biologically relevant vector-present disease-free equilibrium is

$$(6) \quad E_0 = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, 0, K_v, 0, 0, \frac{\Lambda_r}{\mu_r}, 0, 0, 0 \right).$$

Let

$$N_h^0 = \frac{\Lambda_h}{\mu_h}, \quad N_v^0 = K_v, \quad N_r^0 = \frac{\Lambda_r}{\mu_r}, \quad \mathcal{H}_0 = q_h N_h^0 + q_r N_r^0.$$

Define

$$B_{hv} = ab_{vh} \frac{q_h S_h^0}{\mathcal{H}_0}, \quad B_{rv} = ab_{vr} \frac{q_r S_r^0}{\mathcal{H}_0},$$

$$B_{vh} = ac_h \frac{q_h S_v^0}{\mathcal{H}_0}, \quad B_{vA} = ac_A \frac{q_r S_v^0}{\mathcal{H}_0}, \quad B_{vI} = ac_I \frac{q_r S_v^0}{\mathcal{H}_0}.$$

The infected subsystem is ordered as

$$x = (E_h, I_h, E_v, I_v, E_r, A_r, I_r)^T.$$

The new-infection matrix and transition matrix at E_0 are

$$F = \begin{pmatrix} 0 & 0 & 0 & B_{hv} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & B_{vh} & 0 & 0 & 0 & B_{vA} & B_{vI} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & B_{rv} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

and

$$V = \begin{pmatrix} D_{E_h} & 0 & 0 & 0 & 0 & 0 & 0 \\ -\sigma_h & D_{I_h} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & D_{E_v}^0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\sigma_v & D_{I_v}^0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & D_{E_r} & 0 & 0 \\ 0 & 0 & 0 & 0 & -p_r \sigma_r & D_{A_r} & 0 \\ 0 & 0 & 0 & 0 & -(1-p_r) \sigma_r & -\kappa_r & D_{I_r} \end{pmatrix}.$$

Since V is a nonsingular M -matrix and $F \geq 0$, the basic reproduction number is

$$R_0 = \rho(FV^{-1}).$$

Direct calculation gives

$$(7) \quad R_0 = \sqrt{\mathcal{R}_{hv}^2 + \mathcal{R}_{rv}^2},$$

where

$$(8) \quad \mathcal{R}_{hv}^2 = \frac{B_{hv} B_{vh} \sigma_h \sigma_v}{D_{E_h} D_{I_h} D_{E_v}^0 D_{I_v}^0},$$

and

$$(9) \quad \mathcal{R}_{rv}^2 = \frac{B_{rv}\sigma_v}{D_{I_v}^0 D_{E_v}^0} \frac{\sigma_r}{D_{E_r}} \left[p_r \left(\frac{B_{vA}}{D_{A_r}} + \frac{\kappa_r B_{vI}}{D_{A_r} D_{I_r}} \right) + (1 - p_r) \frac{B_{vI}}{D_{I_r}} \right].$$

Remark 3.3. The decomposition $R_0^2 = \mathcal{R}_{hv}^2 + \mathcal{R}_{rv}^2$ separates the human-vector transmission loop from the reservoir-vector transmission loop. Thus disease invasion may be driven by the reservoir cycle even when the human-vector contribution is relatively small.

3.4. Local stability of the disease-free equilibrium.

Theorem 3.4. The disease-free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof. The Jacobian at E_0 separates into uninfected and infected blocks. The uninfected demographic block has eigenvalues with negative real parts, including $-\mu_h$, $-\mu_r$, and $-(r_v - \mu_v)$ for the total vector direction. The infected block is $F - V$. By the next-generation theorem [13], $F - V$ is Hurwitz if and only if $\rho(FV^{-1}) < 1$. Hence E_0 is locally asymptotically stable for $R_0 < 1$. If $R_0 > 1$, then $F - V$ has an eigenvalue with positive real part, and E_0 is unstable. $\square$

4. NSFD DISCRETIZATION

Let $t_n = nh$, $h > 0$, and write X^n for the approximation of $X(t_n)$. The discrete total populations are

$$N_h^n = S_h^n + E_h^n + I_h^n + T_h^n + R_h^n, \quad N_v^n = S_v^n + E_v^n + I_v^n, \quad N_r^n = S_r^n + E_r^n + A_r^n + I_r^n,$$

with

$$\mathcal{H}^n = q_h N_h^n + q_r N_r^n.$$

The discrete forces of infection are

$$\lambda_h^n = ab_{vh} \frac{q_h I_v^n}{\mathcal{H}^n}, \quad \lambda_r^n = ab_{vr} \frac{q_r I_v^n}{\mathcal{H}^n},$$

and

$$\lambda_v^n = a \frac{q_h c_h I_h^n + q_r (c_A A_r^n + c_I I_r^n)}{\mathcal{H}^n}.$$

The denominator function is chosen as

$$(10) \quad \phi(h) = \frac{1 - e^{-Qh}}{Q},$$

where

$$Q \geq \max \{ \omega_h, \mu_h, D_{E_h}, D_{I_h}, D_{T_h}, \mu_v, D_{E_v}^0, D_{I_v}^0, \mu_r, D_{E_r}, D_{A_r}, D_{I_r}, r_v \}.$$

Then $\phi(h) > 0$, $\phi(h) = h + O(h^2)$ as $h \rightarrow 0$, and $0 < \phi(h) \leq 1/Q$.

The NSFD scheme is

$$(11) \quad S_h^{n+1} = \frac{S_h^n + \phi(\Lambda_h + \omega_h R_h^n)}{1 + \phi(\lambda_h^n + \mu_h)},$$

$$(12) \quad E_h^{n+1} = \frac{E_h^n + \phi \lambda_h^n S_h^{n+1}}{1 + \phi D_{E_h}},$$

$$(13) \quad I_h^{n+1} = \frac{I_h^n + \phi \sigma_h E_h^{n+1}}{1 + \phi D_{I_h}},$$

$$(14) \quad T_h^{n+1} = \frac{T_h^n + \phi \tau_h I_h^{n+1}}{1 + \phi D_{T_h}},$$

$$(15) \quad R_h^{n+1} = \frac{(1 - \phi \omega_h) R_h^n + \phi(\gamma_h I_h^{n+1} + \eta_h T_h^{n+1})}{1 + \phi \mu_h},$$

$$(16) \quad S_v^{n+1} = \frac{S_v^n + \phi r_v N_v^n}{1 + \phi(\lambda_v^n + \mu_v + \delta_v N_v^n)},$$

$$(17) \quad E_v^{n+1} = \frac{E_v^n + \phi \lambda_v^n S_v^{n+1}}{1 + \phi(\sigma_v + \mu_v + \delta_v N_v^n)},$$

$$(18) \quad I_v^{n+1} = \frac{I_v^n + \phi \sigma_v E_v^{n+1}}{1 + \phi(\mu_v + \delta_v N_v^n)},$$

$$(19) \quad S_r^{n+1} = \frac{S_r^n + \phi \Lambda_r}{1 + \phi(\lambda_r^n + \mu_r)},$$

$$(20) \quad E_r^{n+1} = \frac{E_r^n + \phi \lambda_r^n S_r^{n+1}}{1 + \phi D_{E_r}},$$

$$(21) \quad A_r^{n+1} = \frac{A_r^n + \phi p_r \sigma_r E_r^{n+1}}{1 + \phi D_{A_r}},$$

$$(22) \quad I_r^{n+1} = \frac{I_r^n + \phi [(1 - p_r) \sigma_r E_r^{n+1} + \kappa_r A_r^{n+1}]}{1 + \phi D_{I_r}}.$$

Here and below $\phi = \phi(h)$.

4.1. Unique solvability of the NSFD scheme.

Theorem 4.1. *Assume that all parameters are nonnegative and that*

$$q_h > 0, \quad q_r > 0, \quad a > 0.$$

Suppose that

$$X^0 \in \mathbb{R}_+^{12}, \quad N_h^0 + N_r^0 > 0.$$

Then, for every $n \geq 0$, the NSFD scheme (11)–(22) admits a unique solution

$$X^{n+1} \in \mathbb{R}_+^{12}.$$

Proof. Assume that $X^n \in \mathbb{R}_+^{12}$ is known. Since

$$H^n = q_h N_h^n + q_r N_r^n$$

and $q_h, q_r > 0$, the condition $N_h^n + N_r^n > 0$ implies $H^n > 0$. Hence the discrete forces of infection

$$\lambda_h^n, \quad \lambda_v^n, \quad \lambda_r^n$$

are uniquely determined and nonnegative.

The update formulae (11)–(22) are sequential rational formulae. Each denominator is strictly positive because $\phi > 0$ and all parameters and state variables appearing in the denominator are nonnegative. For example,

$$1 + \phi(\lambda_h^n + \mu_h) > 0, \quad 1 + \phi D_{E_h} > 0, \quad 1 + \phi(\mu_v + \delta_v N_v^n) > 0.$$

Therefore S_h^{n+1} is uniquely determined first, then E_h^{n+1} , I_h^{n+1} , T_h^{n+1} , and R_h^{n+1} . The same sequential argument applies to the vector variables S_v^{n+1} , E_v^{n+1} , I_v^{n+1} and to the reservoir variables S_r^{n+1} , E_r^{n+1} , A_r^{n+1} , I_r^{n+1} .

Moreover, all numerators are nonnegative. The only potentially delicate term is

$$(1 - \phi \omega_h) R_h^n$$

in the update for R_h^{n+1} . By the choice of Q , we have $0 < \phi \leq 1/Q$ and $Q \geq \omega_h$, so

$$1 - \phi \omega_h \geq 0.$$

Thus $X^{n+1} \in \mathbb{R}_+^{12}$. By induction, the scheme is uniquely solvable and nonnegative for all $n \geq 0$. □

4.2. Consistency and convergence.

Theorem 4.2. *Assume that the exact solution $X(t)$ of model (4) is twice continuously differentiable on a finite interval $[0, T]$, and suppose that*

$$\phi(h) = h + O(h^2) \quad \text{as } h \rightarrow 0.$$

Then the local truncation error of the NSFD scheme (11)–(22) is $O(h^2)$.

Proof. Let $X(t)$ be the exact solution of the continuous model. Taylor expansion gives

$$X(t_{n+1}) = X(t_n) + hF(X(t_n)) + O(h^2).$$

Since

$$\phi(h) = h + O(h^2),$$

we also have

$$X(t_{n+1}) = X(t_n) + \phi(h)F(X(t_n)) + O(h^2).$$

The NSFD scheme differs from the continuous vector field only through nonlocal approximations of incidence, transfer, and loss terms. Since $X(t)$ is continuously differentiable on $[0, T]$, every state variable satisfies

$$X(t_{n+1}) = X(t_n) + O(h).$$

Thus replacing a local factor by a nonlocal factor changes the vector field by $O(h)$. After multiplication by $\phi(h) = O(h)$, this contributes $O(h^2)$ to the one-step error. Therefore the local truncation error is $O(h^2)$. $\square$

Theorem 4.3. *Let $X(t)$ be the exact solution of model (4) on $[0, T]$, and let X^n be the NSFD approximation generated by (11)–(22). Assume that $X(0) = X^0$, that $\phi(h) = h + O(h^2)$, and that the exact and numerical solutions remain in a compact subset of the feasible region where*

$$H = q_h N_h + q_r N_r$$

is bounded away from zero. Then there exist constants $C > 0$ and $h_0 > 0$, independent of h , such that for $0 < h < h_0$,

$$\max_{0 \leq n \leq T/h} \|X(t_n) - X^n\| \leq Ch.$$

Hence the NSFD scheme is first-order convergent on finite time intervals.

Proof. Let

$$e^n = X(t_n) - X^n$$

be the global error. On a compact subset of the feasible region where $H > 0$, the continuous vector field is locally Lipschitz. The NSFD one-step map, denoted by Ψ_h , is also Lipschitz because it consists of rational functions whose denominators are bounded away from zero.

By the consistency result, the exact solution satisfies

$$X(t_{n+1}) = \Psi_h(X(t_n)) + \varepsilon^{n+1},$$

where

$$\|\varepsilon^{n+1}\| \leq C_1 h^2.$$

The numerical solution satisfies

$$X^{n+1} = \Psi_h(X^n).$$

Subtracting gives

$$\|e^{n+1}\| \leq \|\Psi_h(X(t_n)) - \Psi_h(X^n)\| + C_1 h^2.$$

Since $\phi(h) = O(h)$, there is a constant $C_2 > 0$ such that

$$\|\Psi_h(X) - \Psi_h(Y)\| \leq (1 + C_2 h)\|X - Y\|.$$

Hence

$$\|e^{n+1}\| \leq (1 + C_2 h)\|e^n\| + C_1 h^2.$$

Using $e^0 = 0$ and the discrete Gronwall inequality, we obtain

$$\|e^n\| \leq Ch$$

for all $0 \leq t_n \leq T$. This proves first-order convergence. □

5. PROPERTIES OF THE DISCRETE MODEL

5.1. Positivity.

Theorem 5.1. *Assume $X^0 \in \mathbb{R}_+^{12}$ and $N_h^0 + N_r^0 > 0$. Then the NSFD scheme (11)–(22) satisfies $X^n \in \mathbb{R}_+^{12}$ for all $n \geq 0$.*

Proof. All denominators in (11)–(22) are strictly positive. All numerators are nonnegative except possibly the first term in (15). However, by the choice of Q in (10), $\phi\omega_h \leq 1$, so $(1 - \phi\omega_h)R_h^n \geq 0$. Therefore each update is nonnegative whenever the previous state is nonnegative. The result follows by induction. $\square$

5.2. Boundedness.

Theorem 5.2. *The NSFD scheme preserves the population bounds*

$$N_h^n \leq \frac{\Lambda_h}{\mu_h}, \quad N_v^n \leq K_v, \quad N_r^n \leq \frac{\Lambda_r}{\mu_r}$$

whenever they hold at $n = 0$. Moreover, all nonnegative solutions are ultimately bounded.

Proof. Adding the human updates in balance form gives

$$\frac{N_h^{n+1} - N_h^n}{\phi} = \Lambda_h - \mu_h N_h^{n+1} - \alpha_h I_h^{n+1}.$$

Hence

$$N_h^{n+1} \leq \frac{N_h^n + \phi\Lambda_h}{1 + \phi\mu_h}.$$

If $N_h^n \leq \Lambda_h/\mu_h$, then $N_h^{n+1} \leq \Lambda_h/\mu_h$.

Adding the reservoir updates gives

$$\frac{N_r^{n+1} - N_r^n}{\phi} = \Lambda_r - \mu_r N_r^{n+1} - \alpha_r I_r^{n+1},$$

and the same argument yields $N_r^{n+1} \leq \Lambda_r/\mu_r$ whenever $N_r^n \leq \Lambda_r/\mu_r$.

Adding the vector updates gives

$$\frac{N_v^{n+1} - N_v^n}{\phi} = r_v N_v^n - \mu_v N_v^{n+1} - \delta_v N_v^n N_v^{n+1}.$$

Thus

$$N_v^{n+1} = \frac{N_v^n(1 + \phi r_v)}{1 + \phi(\mu_v + \delta_v N_v^n)}.$$

If $N_v^n = K_v$, then $N_v^{n+1} = K_v$ because $\delta_v K_v = r_v - \mu_v$. If $0 \leq N_v^n \leq K_v$, then $N_v^{n+1} \leq K_v$. The ultimate boundedness follows from the scalar comparison recurrences above. $\square$

5.3. Equilibrium consistency.

Theorem 5.3. *The continuous disease-free equilibrium E_0 in (6) is also a fixed point of the NSFD scheme.*

Proof. At E_0 , all forces of infection vanish and all infected, treated, and recovered classes are zero. Substitution into the update formulae gives

$$S_h^{n+1} = \frac{S_h^n + \phi \Lambda_h}{1 + \phi \mu_h}, \quad S_r^{n+1} = \frac{S_r^n + \phi \Lambda_r}{1 + \phi \mu_r},$$

whose fixed points are $S_h^0 = \Lambda_h / \mu_h$ and $S_r^0 = \Lambda_r / \mu_r$. For vectors,

$$N_v^{n+1} = \frac{N_v^n (1 + \phi r_v)}{1 + \phi (\mu_v + \delta_v N_v^n)},$$

whose positive fixed point is $K_v = (r_v - \mu_v) / \delta_v$. Therefore E_0 is a fixed point of the discrete system. $\square$

5.4. Threshold preservation. Linearizing the infected part of the NSFD scheme at E_0 gives

$$\left(\frac{1}{\phi} I + V \right) x^{n+1} = \left(\frac{1}{\phi} I + F \right) x^n.$$

Equivalently,

$$x^{n+1} = G_\phi x^n, \quad G_\phi = \left(\frac{1}{\phi} I + V \right)^{-1} \left(\frac{1}{\phi} I + F \right).$$

Lemma 5.4. *For every $\phi > 0$,*

$$\rho(G_\phi) < 1 \quad \Longleftrightarrow \quad \rho(FV^{-1}) < 1.$$

Proof. Let

$$M_\phi = \frac{1}{\phi} I + V, \quad N_\phi = \frac{1}{\phi} I + F.$$

Then

$$M_\phi - N_\phi = V - F.$$

Since V is a nonsingular M -matrix, M_ϕ is also a nonsingular M -matrix and $M_\phi^{-1} \geq 0$. Moreover $N_\phi \geq 0$. Thus $V - F = M_\phi - N_\phi$ is a regular splitting. A standard result for regular splittings

states that $\rho(M_\phi^{-1}N_\phi) < 1$ if and only if $V - F$ is a nonsingular M -matrix. The next-generation theorem gives that $V - F$ is a nonsingular M -matrix if and only if $\rho(FV^{-1}) < 1$. Since $G_\phi = M_\phi^{-1}N_\phi$, the result follows. $\square$

Theorem 5.5. *The disease-free equilibrium of the NSFD scheme is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof. The local stability of the discrete infected subsystem is determined by $\rho(G_\phi)$. By the lemma,

$$\rho(G_\phi) < 1 \iff \rho(FV^{-1}) < 1 \iff R_0 < 1.$$

Therefore the disease-free fixed point is locally asymptotically stable when $R_0 < 1$. If $R_0 > 1$, then $\rho(G_\phi) > 1$, so the disease-free fixed point is unstable. $\square$

6. NUMERICAL SIMULATIONS

This section illustrates the theoretical results and compares the NSFD scheme with the forward Euler method. The parameter values in Table 3 are scaled illustrative values used to test positivity, boundedness, and threshold preservation. They are not intended as a fitted parameter set for a particular outbreak.

Two transmission regimes are generated by changing the biting rate a . For $a = 0.20$, the reproduction number is

$$R_0 = 0.7024, \quad \mathcal{R}_{hv} = 0.2561, \quad \mathcal{R}_{rv} = 0.6541.$$

For $a = 0.40$, the reproduction number is

$$R_0 = 1.4048, \quad \mathcal{R}_{hv} = 0.5122, \quad \mathcal{R}_{rv} = 1.3081.$$

These two cases represent subcritical and supercritical transmission, respectively. In both cases the reservoir-vector contribution is larger than the human-vector contribution, illustrating the potential role of reservoir hosts in sustaining infection.

TABLE 3. Illustrative parameter values used in the numerical simulations.

Parameter	Value	Parameter	Value
Λ_h	10	Λ_r	2
μ_h	0.001	μ_r	0.001
r_v	0.08	μ_v	0.04
K_v	5000	δ_v	8.0×10^{-6}
q_h	1.0	q_r	1.2
b_{vh}	0.18	b_{vr}	0.16
c_h	0.12	c_A	0.08
c_I	0.16	p_r	0.60
σ_h	1/30	σ_v	1/10
σ_r	1/25	κ_r	1/90
τ_h	1/45	γ_h	1/180
η_h	1/60	ω_h	1/365
α_h	0.0001	α_r	0.0005

6.1. Subcritical case: $R_0 < 1$. Figure 1 compares the NSFD and Euler solutions for the subcritical case. The NSFD trajectories remain nonnegative and bounded, and the infected classes decay toward the disease-free state. The Euler method follows the same qualitative behavior for sufficiently small time steps, but it becomes sensitive to step-size enlargement, as shown in Table 4.

6.2. Supercritical case: $R_0 > 1$. Figure 2 shows the corresponding comparison for $R_0 > 1$. The disease invades and approaches a positive persistence regime. The NSFD method preserves positivity and boundedness across all compartments. The reservoir classes are correctly labelled as S_r, E_r, A_r, I_r ; no recovered-reservoir compartment is present in the model.

6.3. Step-size robustness. Table 4 summarizes the step-size robustness test. The NSFD scheme remains nonnegative in all tested cases. By contrast, the Euler scheme loses positivity for larger time steps.

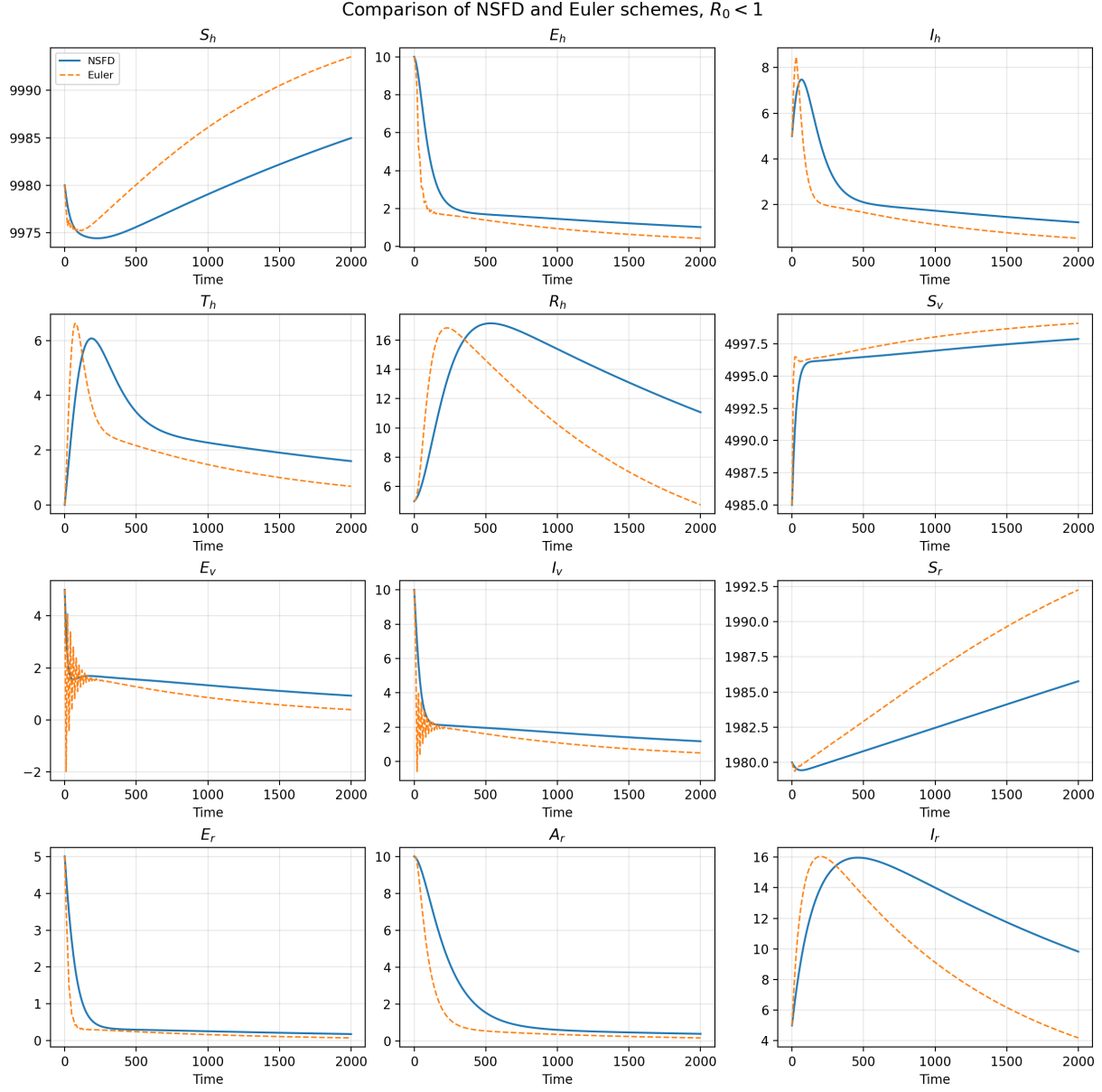


FIGURE 1. Comparison of the NSFD and Euler schemes for the subcritical regime $R_0 < 1$. The infectious classes decay toward the disease-free state.

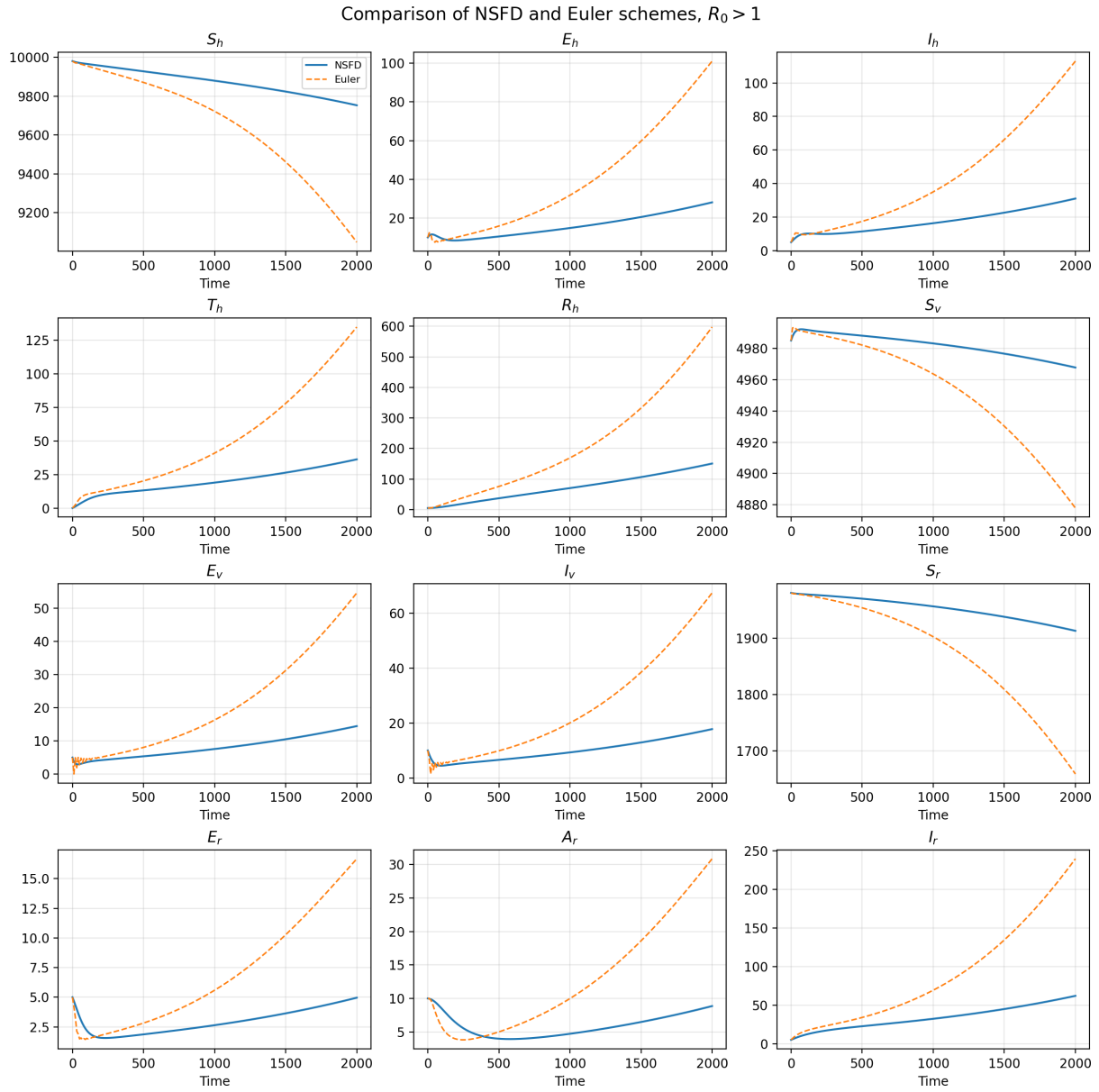


FIGURE 2. Comparison of the NSFD and Euler schemes for the supercritical regime $R_0 > 1$. The infection persists and approaches a positive endemic regime.

TABLE 4. Step-size robustness summary. “Positive” means all state variables remained nonnegative on the simulated interval $0 \leq t \leq 500$.

Regime	Step size h	NSFD minimum	Euler minimum	Euler status
$R_0 < 1$	1	0.0000	0.0000	Positive
$R_0 < 1$	5	0.0000	0.0000	Positive
$R_0 < 1$	10	0.0000	-1.9738	Negative values
$R_0 < 1$	20	0.0000	-8.75×10^{25}	Unstable
$R_0 > 1$	1	0.0000	0.0000	Positive
$R_0 > 1$	5	0.0000	0.0000	Positive
$R_0 > 1$	10	0.0000	0.0000	Positive
$R_0 > 1$	20	0.0000	-1.87×10^{18}	Unstable

The simulations support the theoretical analysis. When $R_0 < 1$, small introductions of infection decay. When $R_0 > 1$, infection persists. The NSFD scheme preserves non-negativity and boundedness in both regimes and avoids the step-size artifacts observed with the Euler method.

7. CONCLUSION

A structured visceral leishmaniasis model involving humans, sandfly vectors, and animal reservoir hosts was formulated and analyzed. The model incorporates human latency, treatment, temporary immunity, vector incubation, asymptomatic and symptomatic reservoir infection, host-attractiveness-based bite allocation, and density-regulated vector demography. The continuous system was shown to be epidemiologically well-posed: solutions remain nonnegative, are ultimately bounded, and evolve within a biologically meaningful feasible region. The disease-free equilibrium and the basic reproduction number were derived using the next-generation method, and R_0 was shown to decompose into human-vector and reservoir-vector contributions.

A dynamically consistent NSFD discretization was then constructed. The scheme uses a denominator function and nonlocal approximations of incidence, transfer, and density-dependent demographic terms. The resulting discrete model preserves positivity, population bounds, the

disease-free equilibrium, and the local stability threshold determined by R_0 . The threshold-preservation argument was established using a regular splitting and the nonsingular M -matrix structure of the infected subsystem.

Numerical experiments confirmed the theoretical findings and showed that the proposed NSFD scheme remains biologically meaningful for larger time steps. In contrast, the forward Euler method can generate negative values or unstable trajectories. The results emphasize the value of structure-preserving discretization for reservoir-driven vector-borne disease models.

Future work may extend the model to include seasonal forcing, spatial heterogeneity, vector mobility, stochastic perturbations, optimal control, or parameter estimation using outbreak data. Higher-order dynamically consistent NSFD methods would also be valuable for large-scale simulation and inference.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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