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Article

New Trends in the Modeling of Diseases Through Computational Techniques

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Abstract: The computational techniques are a set of novel problem-solving methodologies that have attracted wider attention for their excellent performance. The handling strategies of real-world problems are artificial neural networks (ANN), evolutionary computing (EC), and many more. An estimated fifty thousand to ninety thousand new leishmaniasis cases occur annually, with only 25% to 45% reported to the World Health Organization (WHO). It remains one of the top parasitic diseases with outbreak and mortality potential. In 2020, more than ninety percent of new cases reported to World Health Organization (WHO) occurred in ten countries: Brazil, China, Ethiopia, Eritrea, India, Kenya, Somalia, South Sudan, Sudan, and Yemen. The transmission of visceral leishmaniasis is studied dynamically and numerically. The study included positivity, boundedness, equilibria, reproduction number, and local stability of the model in the dynamical analysis. Some detailed methods like Runge Kutta and Euler depend on time steps and violate the physical relevance of the disease. They produce negative and unbounded results, so in disease dynamics, such developments have no biological significance; in other words, these results are meaningless. But the implicit non-standard finite difference method does not depend on time step, positive, bounded, dynamic and consistent. All the computational techniques and their results were compared using computer simulations.

Keywords: Real-world problem; differential equations; computational techniques; analysis

1 Introduction

In 2017, Boukhalfa et al. presented a mathematical model which describes the dynamics of visceral leishmaniasis in the dog population. They observed the effect of primary reproduction numbers and showed global stability using the Lyapunov function [1]. In 2020, Coffeng et al. predicted the impact of reduced detection delays and increased population coverage on observed visceral leishmaniasis cases using a mathematical model [2]. In 2020, Gandhi et al. discussed the delayed visceral leishmaniasis model's dynamical characteristics and presented the steady states' global stability [3]. Zamir et al. formulated well known mathematical model with the help of deterministic techniques [4]. In 2019, Nadeem et al. constructed a mathematical model of zoonotic cutaneous leishmaniasis, including vector populations, reservoirs, and humans. Based on sensitivity analysis of threshold number, they proposed some strategies for eliminating the disease [5]. Rutte et al. studied the infectiousness of kala-azar dermal leishmaniasis [6]. In 2019, Adamu et al. presented a mathematical model with time delay for zoonotic visceral leishmaniasis transmission dynamics. They concluded that the incubation period significantly affects the stability of the equilibrium points [7]. Zamir et al. proposed a mathematical model of visceral leishmaniasis disease with a saturated infection rate. They recommended different control strategies based on sensitivity analysis to manage the spread of this disease in the community [8]. In 2017, Shimozako et al. proposed a new model for zoonotic visceral leishmaniasis using a modified set of differential equations on Brazilian human data. They recommended that sandfly population control be prioritized to eliminate the disease in Brazil [9]. Ghosh et al. proposed a compartmental model of visceral leishmaniasis cases from South Sudan in 2013. They also performed a cost-effectiveness study and cost sensitivity analysis on different interventions [10]. In 2017, Lmojtaba et al. proposed the visceral leishmaniasis dynamics with seasonality's effect. They applied two control, treatment, and vaccination, to the model that forces the system to be non-periodic [11]. Biswas studied disease dynamics to analyze the seasonal visceral leishmaniasis incidence data from South Sudan. He also discussed the optimal control strategy using vaccination and possible treatment of infective humans [12]. In 2017, Debroy et al. studied vector-borne diseases and collected challenges and successes related to modeling transmission dynamics of visceral leishmaniasis [13]. In 2017, Rutte et al. presented three transmission models of visceral leishmaniasis in the Indian subcontinent with structural differences regarding the disease stage [14]. In 2017, Zou et al. developed a mathematical model to study the transmission dynamics of visceral leishmaniasis considering three populations: dogs, sandflies, and humans [15]. In 2016, Siewe et al. developed a mathematical model to explain the evolution of the disease and used the model to simulate treatment by existing or potential new drugs [16]. Rock et al. presented the next-generation matrix methods of visceral leishmaniasis with clinical infection [17]. In 2015, Roy et al. considered a mathematical model of cutaneous leishmaniasis with a time delay effect in the disease transmission [18]. In 2015, Subramanian et al. developed a compartmental-based mathematical model of zoonotic visceral leishmaniasis transmission. They analyzed the model for positivity, boundedness, and stability around steady states indifferent to diseased and disease-free scenarios and derived the primary reproduction number [19]. In 2015, Medley et al. used empirical data on health-seeking behavior and health-system performance from the Indian state of Bihar, Bangladesh, and Nepal to parametrize a mathematical model [20]. Some well-known mathematical models are studied through different aspects [21–29]. The paper's strategy is as follows: Section 2 goes to the formulation, and Section 3 states the fundamental properties of the model. Section 4 goes to the model's numerical results, and the last quarter delivers the results, discussion, and concluding remarks.

2 Variables and Parameters

The parameters and variables of the leishmaniasis model are described as follows: $S_1(t)$: Susceptible (uninfected) dogs at any time t. $L_1(t)$: Latent (infected but not infectious) dogs at any time t. $I_1(t)$: Infectious dogs at any time t. $R_1(t)$: Uninfected dogs at any time t.$Q_1(t)$: Infected dogs at any time t. $D(t)$: Total dog population. δ: Natural death rate in dogs. α: represented the birth rate, β: the natural birth rate of dogs, σ: designated the rate of latent dogs to infectious dogs, and C: defined vectorial capacity of the sandfly population. The 1st order, nonlinear, and coupled ordinary differential equations of the visceral leishmaniasis epidemic model are as follows:

$$\frac{dS_1}{dt} = \alpha\beta D - \frac{CI_1S_1}{D} - \delta S_1. \tag{1}$$

$$\frac{dL_1}{dt} = \frac{CI_1S_1}{D} - (\sigma + \delta)L_1. \tag{2}$$

$$\frac{dI_1}{dt} = \sigma L_1 - \delta I_1. \tag{3}$$

$$\frac{dR_1}{dt} = (1 - \alpha)\beta D - \frac{CI_1R_1}{D} - \delta R_1. \tag{4}$$

$$\frac{dQ_1}{dt} = \frac{CI_1R_1}{D} - \delta Q_1. \tag{5}$$

Following non-negative initial conditions, $S_1(0) \geq 0,\ L_1(0) \geq 0,\ I_1(0) \geq 0,\ R_1(0) \geq 0,\ Q_1(0) \geq 0$ and $\mathrm{S_1(t) + L_1(t) + I_1(t) + R_1(t) + Q_1(t) = D(t)}$.

2.1 Normalization

The system (1–5) can be normalized by subsidizing variables to avoid complications $\mathrm{S} = \frac{\mathrm{S_1}}{\mathrm{D}},\ \mathrm{L} = \frac{L_1}{\mathrm{D}}$, $I = \frac{I_1}{\mathrm{D}},\ R = \frac{R_1}{D},\ Q = \frac{Q_1}{D}$ as follows:

$$\frac{dS}{dt} = \alpha\beta - CIS - \delta S. \tag{6}$$

$$\frac{dL_1}{dt} = CIS - (\sigma + \delta)L. \tag{7}$$

$$\frac{dI_1}{dt} = \sigma L - \delta I. \tag{8}$$

$$\frac{dR_1}{dt} = (1 - \alpha)\beta - CIR - \delta R. \tag{9}$$

$$\frac{dQ_1}{dt} = CIR - \delta Q. \tag{10}$$

with nonnegative initial conditions $S(0) \geq 0,\ L(0) \geq 0,\ \mathrm{I(0)} \geq 0,\ R(0) \geq 0,\ Q(0) \geq 0$ and $S(t)+ L(t) + I(t) + R(t) + Q(t) = 1$.

The feasible region of the model as follows:

$$\mathcal{H} = \Big\{ (S,\ L,\ I,\ R,\ Q)\epsilon\mathbb{R}_{+}^{5} : S(t) + L(t) + I(t) + R(t) + Q(t) = D(t) \le \frac{\beta}{\delta},$$
$$S \ge 0,\ \mathrm{L} \ge 0,\ \mathrm{I} \ge 0,\ \mathrm{R} \ge 0,\ \mathrm{Q} \ge 0 \Big\}.$$

2.2 Positivity of Model

Theorem 1: For any time, t, the system (6–10) admits a positive solution.

Proof: By letting the system,

$$\left.\frac{\mathrm{dS}}{\mathrm{dt}}\right|_{\mathrm{S}=0} = \alpha\beta \ge 0,\ \left.\frac{\mathrm{dL}}{\mathrm{dt}}\right|_{\mathrm{L}=0} = CIS \ge 0,\ \left.\frac{\mathrm{dI}}{\mathrm{dt}}\right|_{\mathrm{I}=0} = \sigma L \ge 0,\ \left.\frac{\mathrm{dR}}{\mathrm{dt}}\right|_{\mathrm{R}=0} = (1-\alpha)\beta \ge 0$$
$$\left.\frac{\mathrm{dQ}}{\mathrm{dt}}\right|_{\mathrm{Q}=0} = CIR \ge 0, \text{ as desired.}$$

2.3 Boundedness of Model

Theorem 2: For any time, t, the system (6–10) admits a bounded solution and lies in the feasible region. For considerable time t, the following inequality satisfies $\lim_{t\to\infty} Sup\ D(t) \le \frac{\beta}{\delta}$.

Proof: Consider the population function as follows:

$$D(t) = S(t) + L(t) + I(t) + R(t) + Q(t).$$
$$\frac{dD}{dt} = \beta - \delta(S + L + I + R + Q)$$
$$\frac{dD}{dt} \le \beta - \delta D$$
$$D(t) \le D(0)e^{-\delta t} + \frac{\beta}{\delta},\quad t \ge 0$$
$$\lim_{t\to\infty} Sup\ D(t) \le \frac{\beta}{\delta}, \text{ as desired.}$$

2.4 Equilibria of Model

The model admits two types of equilibria as follows:

Leishmaniasis free equilibrium point $(\mathcal{L}_nFE - \mathrm{D}_1) = (S^1,\ L^1,\ I^1,\ R^1,\ Q^1) = (\alpha,\ 0,\ 0,\ 1-\alpha,\ 0)$ and Leishmaniasis existing equilibrium point $(\mathcal{L}_nEE - \mathrm{D}_2) = (S^*,\ L^*,\ I^*,\ R^*,\ Q^*)$.

$$S^* = \frac{\delta(\sigma+\delta)}{C\sigma},\ L^* = \frac{\alpha\beta C\sigma - \delta^2(\sigma+\delta)}{C\sigma(\sigma+\delta)},\ I^* = \frac{\alpha\beta C\sigma - \delta^2(\sigma+\delta)}{C\delta(\sigma+\delta)},\ R^* = \frac{(1-\alpha)(\sigma+\delta)\beta\delta}{\alpha\beta C\sigma - \delta^2(\sigma+\delta) + (\sigma+\delta)\delta^2},$$
$$Q^* = \frac{(1-\alpha)\beta\left[\alpha\beta C\sigma - \delta^2(\sigma+\delta)\right]}{\delta\left[\alpha\beta C\sigma - \delta^2(\sigma+\delta) + \delta^2(\sigma+\delta)\right]}.$$

2.5 Reproduction Number

In this section, the calculation of the infection force ratio by using the next-generation matrix method and the leishmaniasis-free equilibrium of the model as follows:

$$\begin{bmatrix} L' \\ I' \end{bmatrix} = \begin{bmatrix} 0 & CS \\ 0 & 0 \end{bmatrix} \begin{bmatrix} L \\ I \end{bmatrix} - \begin{bmatrix} \sigma+\delta & 0 \\ -\sigma & \delta \end{bmatrix} \begin{bmatrix} L \\ I \end{bmatrix}$$

where $F = \begin{bmatrix} 0 & CS \\ 0 & 0 \end{bmatrix}$, $V = \begin{bmatrix} \sigma+\delta & 0 \\ -\sigma & \delta \end{bmatrix}$. F and V are the transmission and transition matrices, respectively.

$$FV^{-1} = \frac{1}{\delta(\sigma+\delta)} \begin{bmatrix} C\sigma S & (\sigma+\delta)CS \\ 0 & 0 \end{bmatrix}.$$

The largest eigenvalue of FV^{-1} is called reproduction number and is defined as $R_o = \dfrac{C\sigma\alpha}{\delta(\sigma+\delta)}$

3 Local Stability

***Theorem 3*:** The leishmaniasis free equilibrium $(\mathcal{L}_{\mathrm{n}}\mathrm{FE} - \mathrm{D}_1)\ D_1 = (S^1,\ L^1,\ I^1,\ R^1,\ Q^1) = (\alpha,\ 0,\ 0,\ 1-\alpha,\ 0)$ for the system (2.6–2.10) is locally asymptotically stable (LAS) if $R_o < 1$.

Proof: The Jacobian matrix at D_1 as follows:

$$J_{\mathcal{L}_n}|_{\mathrm{D}_1} = \begin{bmatrix} -\delta & 0 & 0 & 0 & 0 \\ 0 & -(\sigma+\delta) & C\alpha & 0 & 0 \\ 0 & \sigma & -\delta & 0 & 0 \\ 0 & 0 & -C(1-\alpha) & -\delta & 0 \\ 0 & 0 & C(1-\alpha) & 0 & -\delta \end{bmatrix}$$

Consider, $\left| J_{\mathcal{L}_n}|_{\mathrm{D}_1} - \lambda I \right| = 0$

$$\begin{vmatrix} -\delta-\lambda & 0 & 0 & 0 & 0 \\ 0 & -(\sigma+\delta)-\lambda & C\alpha & 0 & 0 \\ 0 & \sigma & -\delta-\lambda & 0 & 0 \\ 0 & 0 & -C(1-\alpha) & -\delta-\lambda & 0 \\ 0 & 0 & C(1-\alpha) & 0 & -\delta-\lambda \end{vmatrix} = 0$$

$\lambda_1 = -\delta < 0,\ \ \lambda_2 = -\delta < 0,\ \ \lambda_3 = -\delta < 0$

$\lambda^2 + a_1\lambda + a_0 = 0.$

where $a_1 = \sigma + 2\delta,\ \ a_0 = \delta\sigma + \delta^2 - \sigma C\alpha.$

Since $a_1,\ \ a_0 > 0$ if $\mathcal{R}_0 < 1$, it is stable with the reference Routh-Hurwitz properties.

Theorem 4: The leishmaniasis existing equilibrium $(\mathcal{L}_n\mathrm{EE} - \mathrm{D}_2),\ \ D_2 = (S^*,\ \ L^*,\ \ I^*,\ \ R^*,\ \ Q^*)$ for the system (6–10) is locally asymptotical stable (LAS) if $\mathcal{R}_0 > 1$.

Proof: The Jacobian matrix at D_2 as follows:

$$\left| J_{\mathcal{L}_n}|_{\mathrm{D}_2} - \lambda I \right| = 0$$

$$\begin{bmatrix} \frac{-\alpha\beta C\sigma}{\delta(\sigma+\delta)}-\lambda & 0 & \frac{\delta^2(\sigma+\delta)-\alpha\beta C\sigma}{\delta(\sigma+\delta)} & 0 & 0 \\ \frac{\alpha\beta C\sigma-\delta^2(\sigma+\delta)}{\delta(\sigma+\delta)} & -(\sigma+\delta)-\lambda & \frac{\delta(\sigma+\delta)}{\sigma} & 0 & 0 \\ 0 & \sigma & -\delta-\lambda & 0 & 0 \\ 0 & 0 & \frac{-C(1-\alpha)(\sigma+\delta)\beta\delta}{\alpha\beta C\sigma-\delta^2(\sigma+\delta)+(\sigma+\delta)\delta^2} & \frac{\delta^2(\sigma+\delta)-\alpha\beta C\sigma-\delta^2(\sigma+\delta)}{\delta(\sigma+\delta)}-\lambda & 0 \\ 0 & 0 & \frac{C(1-\alpha)(\sigma+\delta)\beta\delta}{\alpha\beta C\sigma-\delta^2(\sigma+\delta)+(\sigma+\delta)\delta^2} & \frac{\alpha\beta C\sigma-\delta^2(\sigma+\delta)}{\delta(\sigma+\delta)} & -\delta-\lambda \end{bmatrix} = 0$$

$$\lambda_1 = -\delta < 0, \quad \lambda_2 = \frac{\delta^2(\sigma+\delta)-\alpha\beta C\sigma-\delta^2(\sigma+\delta)}{\delta(\sigma+\delta)} < 0, \text{ if } \mathcal{R}_0 > 1$$

$$\lambda^3 + b_2\lambda^2 + b_1\lambda + b_0 = 0,$$

where $b_2 = \frac{\alpha\beta C\sigma + \delta(\sigma+\delta)(\sigma+2\delta)}{\delta(\sigma+\delta)}, \quad b_1 = \alpha\beta C\sigma\left[\frac{\sigma+2\delta}{\delta(\sigma+\delta)}\right], \quad b_0 = \frac{\sigma\left[\alpha\beta C\sigma - \delta^2(\sigma+\delta)\right]^2}{\delta^2(\sigma+\delta)^2}$

Applying Routh-Hurwitz Criterion for 3rd order, $b_2 > 0$, $b_0 > 0$, and $b_1 b_2 > b_0$, if $R_0 > 1$. Therefore, the $(\mathcal{L}_n \text{EE} - \text{D}_2)$ of the given system (6–10) is locally asymptotically stable.

4 Numerical Results

Using the command-built software such as Matlab and simulating the system (1–6) at both equilibria of the model using scientific literature presented in Tab. 1 as follows:

Table 1: Values of parameters

Parameters	$\%\mathcal{L}_n FE - \text{D}_1$	$\%\mathcal{L}_n EE - \text{D}_2$
α	0.50	0.50
β	0.50	0.50
δ	0.50	0.50
σ	0.40	0.40
C	0.15	3.15

4.1 Euler's Scheme

The Euler method could be applied to the system (1–5) as follows:

$$S^{n+1} = S^n + h(\alpha\beta - CI^nS^n - \delta S^n) \tag{11}$$

$$L^{n+1} = L^n + h(CI^nS^n - (\sigma+\delta)L^n) \tag{12}$$

$$I^{n+1} = I^n + h(\sigma L^n - \delta I^n) \tag{13}$$

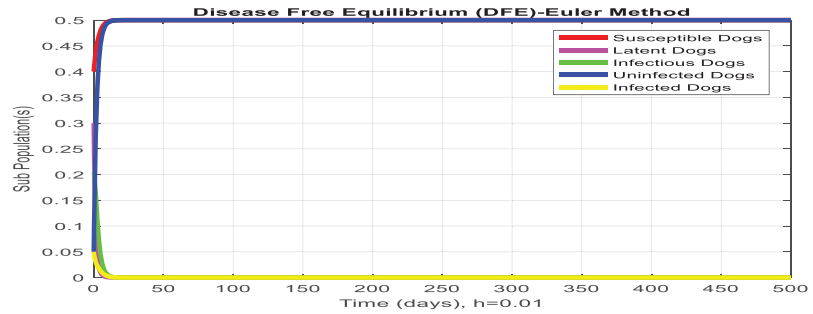
$$R^{n+1} = R^n + h((1-\alpha)\beta - CI^nR^n - \delta R^n) \tag{14}$$

$$Q^{n+1} = Q^n + h(CI^nR^n - \delta Q^n) \tag{15}$$

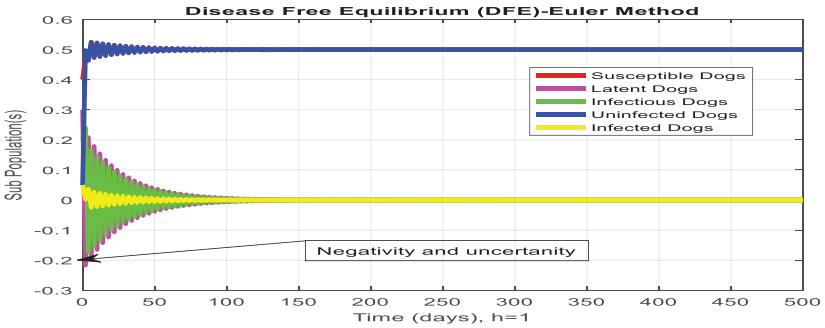
where h is any time step size?

4.2 Diagrams

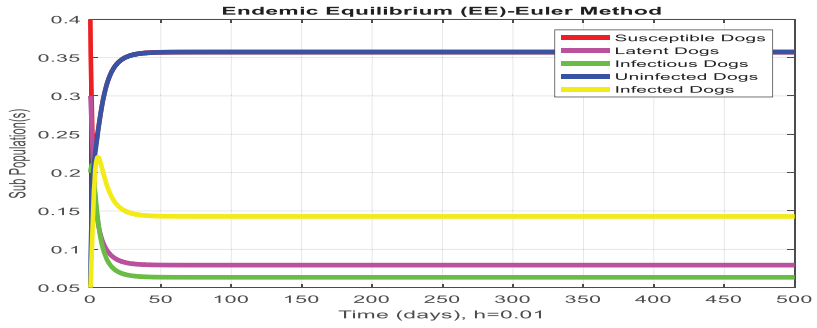
The Euler's method graphs are plotted for both equilibria of the model as follows:



(a)



(b)



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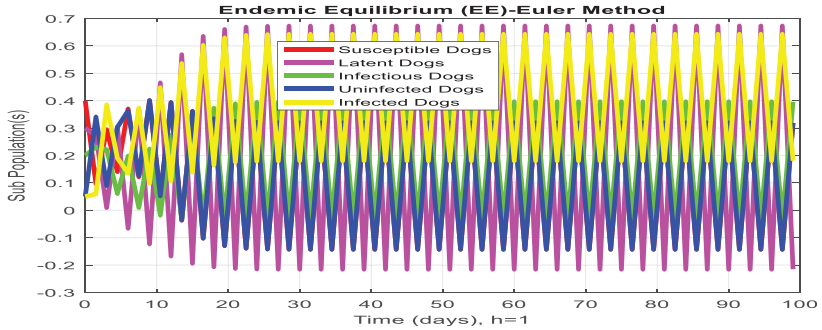


(d)

Figure 1: (Euler simulations) subpopulation at $\mathcal{L}_nFE - \mathrm{D}_1$ for $h = 0.01$ (b) subpopulation at $\mathcal{L}_nFE - \mathrm{D}_1$ for $h = 1$ (c) subpopulation at $\mathcal{L}_nEE - \mathrm{D}_2$ for $h = 0.01$ (d) subpopulation at $\mathcal{L}_nEE - \mathrm{D}_2$ for $h = 1$

4.3 Runge-Kutta Scheme

The Runge Kutta method could be applied to the system (1–5) as follows:

Stage I

$$J_1 = h(\alpha\beta - CI^nS^n - \delta S^n)$$

$$K_1 = h[CI^nS^n - (\sigma + \delta)L^n]$$

$$L_1 = h(\sigma L^n - \delta I^n)$$

$$M_1 = h[(1-\alpha)\beta - CI^nR^n - \delta R^n]$$

$$N_1 = h(CI^nR^n - \delta Q^n)$$

Stage II

$$J_2 = h\left[\alpha\beta - C\left(I^n + \frac{L_1}{2}\right)\left(S^n + \frac{J_1}{2}\right) - \delta\left(S^n + \frac{J_1}{2}\right)\right]$$

$$K_2 = h\left[C\left(I^n + \frac{L_1}{2}\right)\left(S^n + \frac{J_1}{2}\right) - (\sigma + \delta)\left(L^n + \frac{K_1}{2}\right)\right]$$

$$L_2 = h\left[\sigma\left(L^n + \frac{K_1}{2}\right) - \delta\left(I^n + \frac{L_1}{2}\right)\right]$$

$$M_2 = h\left[(1-\alpha)\beta - C\left(I^n + \frac{L_1}{2}\right)\left(R^n + \frac{M_1}{2}\right) - \delta\left(R^n + \frac{M_1}{2}\right)\right]$$

$$N_2 = h\left[C\left(I^n + \frac{L_1}{2}\right)\left(R^n + \frac{M_1}{2}\right) - \delta\left(Q^n + \frac{N_1}{2}\right)\right]$$

Stage III

$$J_3 = h\left[\alpha\beta - C\left(I^n + \frac{L_2}{2}\right)\left(S^n + \frac{J_2}{2}\right) - \delta\left(S^n + \frac{J_2}{2}\right)\right]$$

$$K_3 = h\left[C\left(I^n + \frac{L_2}{2}\right)\left(S^n + \frac{J_2}{2}\right) - (\sigma + \delta)\left(L^n + \frac{K_2}{2}\right)\right]$$

$$L_3 = h\left[\sigma\left(L^n + \frac{K_2}{2}\right) - \delta\left(I^n + \frac{L_2}{2}\right)\right]$$

$$M_3 = h\left[(1-\alpha)\beta - C\left(I^n + \frac{L_2}{2}\right)\left(R^n + \frac{M_2}{2}\right) - \delta\left(R^n + \frac{M_2}{2}\right)\right]$$

$$N_3 = h\left[C\left(I^n + \frac{L_2}{2}\right)\left(R^n + \frac{M_2}{2}\right) - \delta\left(Q^n + \frac{N_2}{2}\right)\right]$$

Stage IV

$$J_2 = h[\alpha\beta - C(I^n + L_3)(S^n + J_3) - \delta(S^n + J_3)]$$

$$K_2 = h[C(I^n + L_3)(S^n + J_3) - (\sigma + \delta)(L^n + K_3)]$$

$$L_2 = h[\sigma(L^n + K_3) - \delta(I^n + L_3)]$$

$$M_2 = h\left[(1-\alpha)\beta - C(I^n + L_3)\left(R^n + \frac{M_1}{2}\right) - \delta(R^n + M_3)\right]$$

$$N_2 = h[C(I^n + L_3)(R^n + M_3) - \delta(Q^n + N_3)]$$

Final Stage

$$S^{n+1} = S^n + \frac{1}{6}(J_1 + 2J_2 + 2J_3 + J_4) \tag{16}$$

$$L^{n+1} = L^n + \frac{1}{6}(K_1 + 2K_2 + 2K_3 + K_4) \tag{17}$$

$$I^{n+1} = I^n + \frac{1}{6}(L_1 + 2L_2 + 2L_3 + L_4) \tag{18}$$

$$R^{n+1} = R^n + \frac{1}{6}(M_1 + 2M_2 + 2M_3 + M_4) \tag{19}$$

$$Q^{n+1} = Q^n + \frac{1}{6}(N_1 + 2N_2 + 2N_3 + N_4) \tag{20}$$

where h is any time step size.

4.4 Diagrams

The Runge Kutta method graphs are plotted for both equilibria of the model as follows:

4.5 NSFD Method

The NSFD method that could be applied to the system (1–5) as follows:

Eq. (1)

$$S^{n+1} = S^n + h\left(\alpha\beta - CI^n S^{n+1} - \delta S^{n+1}\right)$$

$$S^{n+1} = \frac{S^n + h\alpha\beta}{1 + hCI^n + h\delta} \tag{21}$$

Like, Eq. (21),

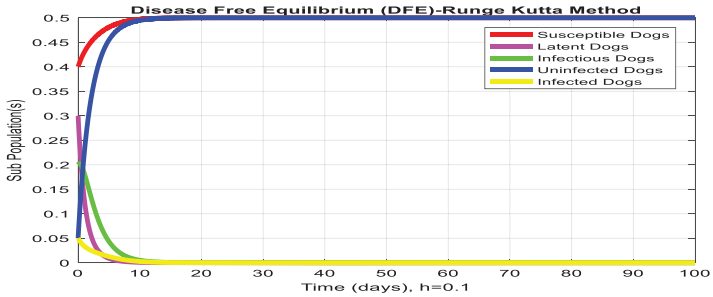
$$L^{n+1} = \frac{L^n + hCI^n S^n}{1 + h(\sigma + \delta)} \tag{22}$$

$$I^{n+1} = \frac{I^n + h\sigma L^n}{1 + h\delta} \tag{23}$$

$$R^{n+1} = \frac{R^n + h(1-\alpha)\beta}{1 + hCI^n + h\delta} \tag{24}$$

$$Q^{n+1} = \frac{Q^n + hCI^n R^n}{1 + h\delta} \tag{25}$$

where h is any time step size.



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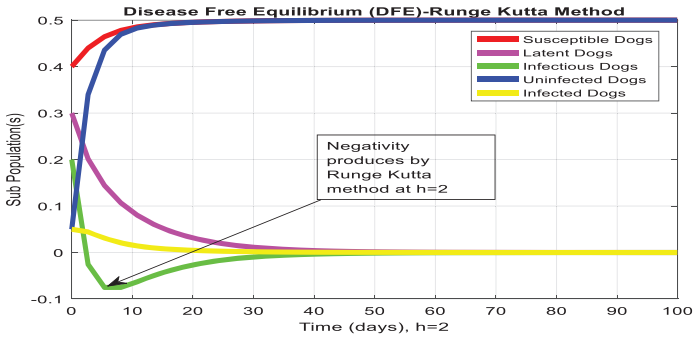

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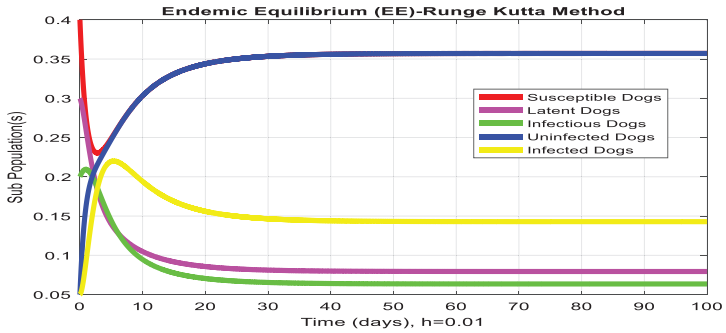

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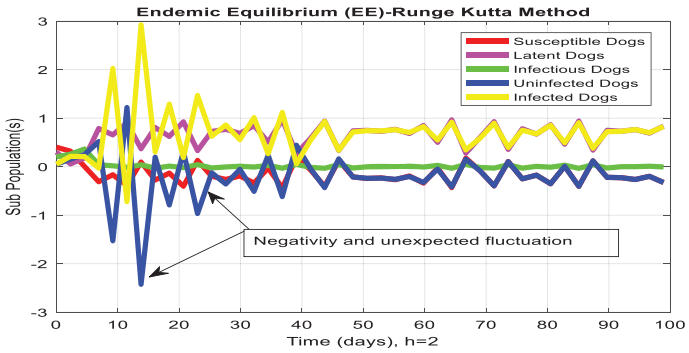


(d)

Figure 2: (Runge-Kutta simulations) **(a)** subpopulation at $\mathcal{L}_nFE - \mathrm{D}_1$ for $h = 0.1$ (b) subpopulation at $\mathcal{L}_nFE - \mathrm{D}_1$ for $h = 2$ (c) subpopulation at $\mathcal{L}_nEE - \mathrm{D}_2$ for $h = 0.01$ (d) subpopulation at $\mathcal{L}_nEE - \mathrm{D}_2$ for $h = 2$

4.6 Stability Results of NSFD Method

Considering the function for the system (3.11–3.15),

$$M=\frac{S+h\alpha\beta}{1+hCI+h\delta},\quad P=\frac{L+hCIS}{1+h(\sigma+\delta)},\quad U=\frac{I+h\sigma L}{1+h\delta},\quad V=\frac{R+h(1-\alpha)\beta}{1+hCI+h\delta}\text{ and }W=\frac{Q+hCIR}{1+h\delta}$$

The partial derivatives of the Jacobean matrix,

$$J(S,\,L,\,I,\,R,\,Q)=\begin{bmatrix}\frac{1}{1+hCI+h\delta} & 0 & \frac{-hC(S+h\alpha\beta)}{(1+hCI+h\delta)^2} & 0 & 0\\ \frac{hCI}{1+h(\sigma+\delta)} & \frac{1}{1+h(\sigma+\delta)} & \frac{hCS}{1+h(\sigma+\delta)} & 0 & 0\\ 0 & \frac{h\sigma}{1+h\delta} & \frac{1}{1+h\delta} & 0 & 0\\ 0 & 0 & \frac{-hC[R+h(1-\alpha)\beta]}{(1+hCI+h\delta)^2} & \frac{1}{1+hCI+h\delta} & 0\\ 0 & 0 & \frac{hCR}{1+h\delta} & \frac{hCI}{1+h\delta} & \frac{1}{1+h\delta}\end{bmatrix}\tag{26}$$

At disease free equilibrium point $(S,\,L,\,I,\,R,\,Q)=(\alpha,\,0,\,0,\,1-\alpha,\,0)$, Eq. (26) becomes

$$J(\alpha,\,0,\,0,\,1-\alpha,\,0)=\begin{bmatrix}\frac{1}{1+h\delta} & 0 & \frac{-hC\alpha(1+h\beta)}{(1+h\delta)^2} & 0 & 0\\ 0 & \frac{1}{1+h(\sigma+\delta)} & \frac{hC\alpha}{1+h(\sigma+\delta)} & 0 & 0\\ 0 & \frac{h\sigma}{1+h\delta} & \frac{1}{1+h\delta} & 0 & 0\\ 0 & 0 & \frac{-hC(1-\alpha)(1+h\beta)}{(1+h\delta)^2} & \frac{1}{1+h\delta} & 0\\ 0 & 0 & \frac{hC(1-\alpha)}{1+h\delta} & 0 & \frac{1}{1+h\delta}\end{bmatrix}$$

$$|J-\lambda I|=0$$

$$\begin{bmatrix}\frac{1}{1+h\delta}-\lambda & 0 & \frac{-hC\alpha(1+h\beta)}{(1+h\delta)^2} & 0 & 0\\ 0 & \frac{1}{1+h(\sigma+\delta)}-\lambda & \frac{hC\alpha}{1+h(\sigma+\delta)} & 0 & 0\\ 0 & \frac{h\sigma}{1+h\delta} & \frac{1}{1+h\delta}-\lambda & 0 & 0\\ 0 & 0 & \frac{-hC(1-\alpha)(1+h\beta)}{(1+h\delta)^2} & \frac{1}{1+h\delta}-\lambda & 0\\ 0 & 0 & \frac{hC(1-\alpha)}{1+h\delta} & 0 & \frac{1}{1+h\delta}-\lambda\end{bmatrix}=0$$

$$\lambda_1=\left|\frac{1}{1+h\delta}\right|<1,\quad \lambda_2=\left|\frac{1}{1+h\delta}\right|<1,\quad \lambda_3=\left|\frac{1}{1+h\delta}\right|<1$$

$$J(\alpha,\ 0,\ 0,\ 1-\alpha,\ 0)=\begin{bmatrix}\dfrac{1}{1+h(\sigma+\delta)} & \dfrac{hC\alpha}{1+h(\sigma+\delta)}\\ \dfrac{h\sigma}{1+h\delta} & \dfrac{1}{1+h\delta}\end{bmatrix}$$

$A = Trace\ of\ J,\ \ B = Determinant\ of\ J$

$$A=\frac{2+2h\delta+h\sigma}{(1+h\delta)[1+h(\sigma+\delta)]},\ \ B=\frac{1-h^2C\alpha\sigma}{(1+h\delta)[1+h(\sigma+\delta)]}$$

For stability, to prove the following conditions as follows:

i) $1-A+B>0$
ii) $1+A+B>0$
iii) $B<1$
iv) $1-A+B>0$

$$1-\frac{2+2h\delta+h\sigma}{(1+h\delta)[1+h(\sigma+\delta)]}+\frac{1-h^2C\alpha\sigma}{(1+h\delta)[1+h(\sigma+\delta)]}>0$$

$$(1+h\delta)[1+h(\sigma+\delta)]-2-2h\delta-h\sigma+1-h^2C\alpha\sigma>0$$

$$1+h\sigma+h\delta+h\delta+h^2\sigma\delta+h^2\delta^2-2-2h\delta-h\sigma+1-h^2C\alpha\sigma>0$$

$$h^2\left(\sigma\delta+\delta^2-C\alpha\sigma\right)>0$$

$$h^2>0.$$

$$h>0$$

Since the step size is never zero, so the condition is satisfied.

(ii) $1+A+B>0$

$$1+\frac{2+2h\delta+h\sigma}{(1+h\delta)[1+h(\sigma+\delta)]}+\frac{1-h^2C\alpha\sigma}{(1+h\delta)[1+h(\sigma+\delta)]}>0$$

$$(1+h\delta)[1+h(\sigma+\delta)]+2+2h\delta+h\sigma+1-h^2C\alpha\sigma>0$$

$$1+h\sigma+h\delta+h\delta+h^2\sigma\delta+h^2\delta^2+2+2h\delta+h\sigma+1-h^2C\alpha\sigma>0$$

$$h^2\left(\sigma\delta+\delta^2-C\alpha\sigma\right)+2h(\sigma+2\delta)+4>0$$

$$h^2a+2hb+4>0$$

where $a=\sigma\delta+\delta^2-C\alpha\sigma,\ \ b=\sigma+2\delta$

$$h^2+2h\frac{b}{a}+\frac{4}{a}>0$$

$$h^2+2h\frac{b}{a}+\left(\frac{b}{a}\right)^2+\frac{4}{a}>\left(\frac{b}{a}\right)^2$$

$$\left(h+\frac{b}{a}\right)^2+\frac{4}{a}>\left(\frac{b}{a}\right)^2$$

Hence, this condition is also satisfied.

(iii) $B<1$

$$\frac{1-h^2C\alpha\sigma}{(1+h\delta)[1+h(\sigma+\delta)]}<1$$

$$1-h^2C\alpha\sigma<(1+h\delta)[1+h(\sigma+\delta)]$$

$$1-h^2C\alpha\sigma<1+h\sigma+h\delta+h\delta+h^2\sigma\delta+h^2\delta^2$$

$$h\sigma+2h\delta++h^2\sigma\delta+h^2\delta^2+h^2C\alpha\sigma>0$$

$$h^2\left(\sigma\delta+\delta^2+C\alpha\sigma\right)+h(\sigma+2\delta)>0$$

$$h^2c+hb>0$$

where $c=\sigma\delta+\delta^2+C\alpha\sigma$

$$h^2+h\frac{b}{c}>0$$

$$h^2+2h\frac{b}{2c}+\left(\frac{b}{2c}\right)^2>\left(\frac{b}{2c}\right)^2$$

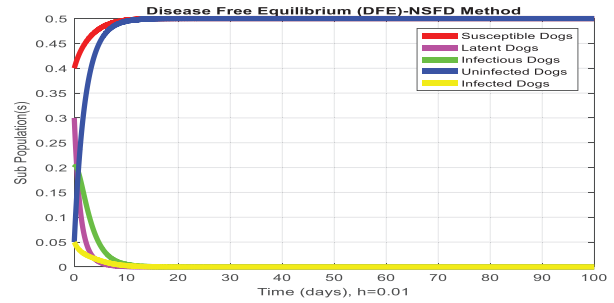
$$\left(h+\frac{b}{2c}\right)^2>\left(\frac{b}{2c}\right)^2$$

So, condition (iii) is also satisfied, as desired.

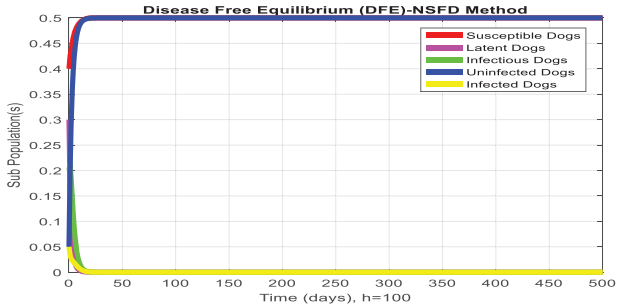
4.7 Diagrams

The NSFD method graphs are plotted for both equilibria of the model as follows:

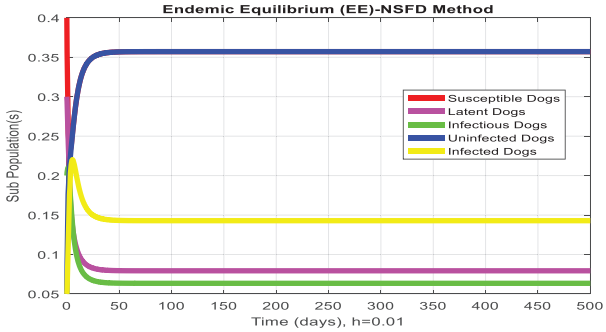
4.8 Comparison Section



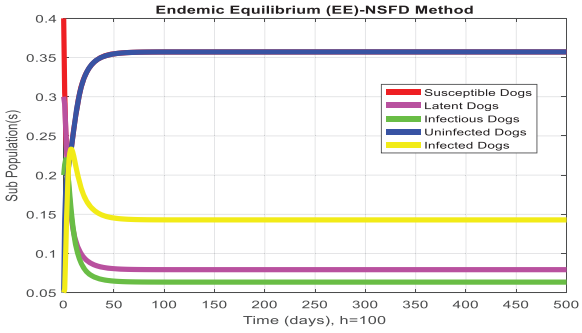
(a)



(b)



(c)



(d)

Figure 3: (NSFD simulations) subpopulation at $\mathcal{L}_nFE - \mathrm{D}_1$ for $h = 0.01$ (b) subpopulation at $\mathcal{L}_nFE - \mathrm{D}_1$ for $h = 100$ (c) subpopulation at $\mathcal{L}_nEE - \mathrm{D}_2$ for $h = 0.01$ (d) subpopulation at $\mathcal{L}_nEE - \mathrm{D}_2$ for $h = 100$

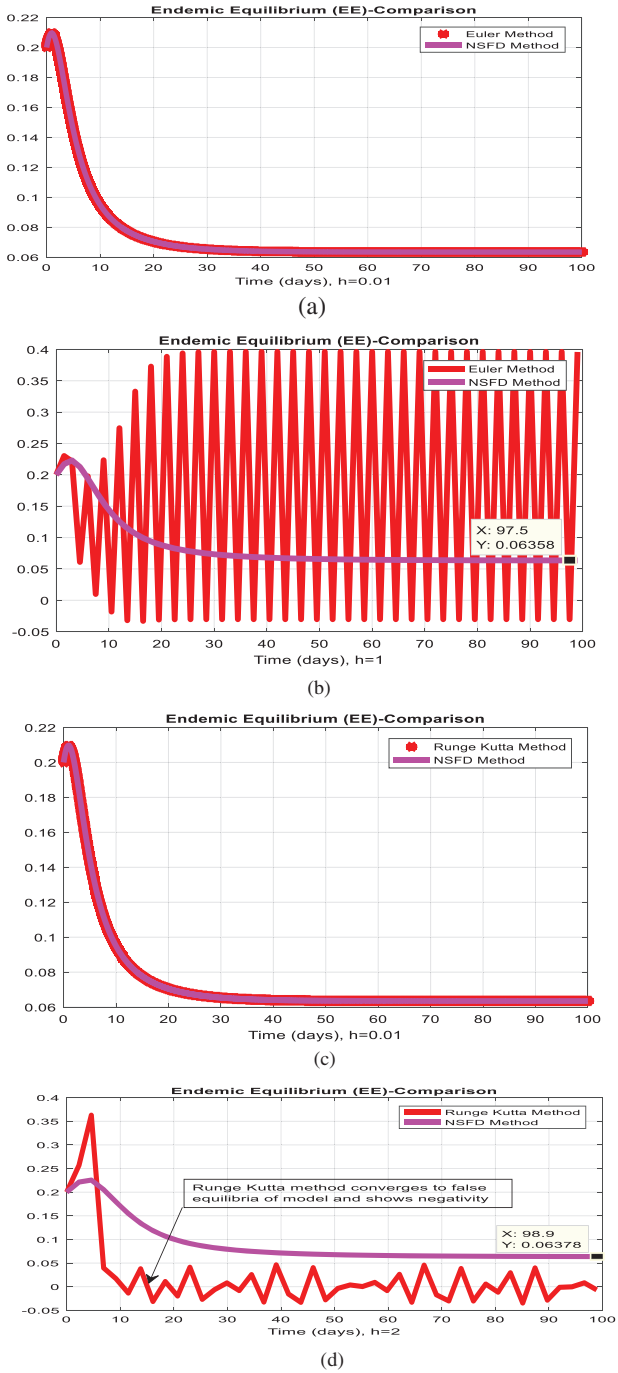


Figure 4: Comparison of methods (a) infected humans at $\mathcal{L}_nEE - \mathrm{D}_2$ for $h = 0.01$ (b) infected humans at $\mathcal{L}_nEE - \mathrm{D}_2$ for $h = 1$ (c) infected humans at $\mathcal{L}_nEE - \mathrm{D}_2$ for $h = 0.01$ (d) infected humans at $\mathcal{L}_nEE - \mathrm{D}_2$ for $h = 2$

5 Results and Concluding Remarks

The simulation of the Euler method is presented in Figs. 1a–1d. Figs. 1b and Fig. 1d show that the system violates the properties of the real-world problem like positivity, boundedness, and dynamical

consistency by an increase in the time step size. The Runge Kutta method of order fourth depicts the graphical representation in Figs. 2a–2d, but the technique is time-dependent and converges to the false steady state of the model. The NSFD process depicts the graphical solution of the model in Figs. 3a–3d; the NSFD method converges to proper equilibria of the model at any time step size and fulfils all the model properties. The support comparison of the numerical methods is presented in Figs. 4a–4d. The Euler and RK-4 schemes are dependent on time step size. These numerical schemes are convergent for small step sizes while divergent for large step sizes. But the nonstandard finite difference method used for communication dynamics of leishmaniasis disease is independent of the time step size. It is convergent even for any time step size like hundreds and thousands. NSFD scheme shows positivity, boundedness, and stability and behaves similarly to the behavior of the continuous model. Hence, the NSFD scheme is more efficient, fast convergent, and reliable than the remaining method. In the future, the work will extend to the field as presented in [30–33].

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