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Existence, Stability and Sensitivity Analysis of Lyme Disease Using Caputo Fractional Dynamical Systems

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Abstract

In this article, mathematical modeling and stability analysis of Lyme disease and its transmission dynamics using Caputo fractional-order derivatives is presented. The compartmental model has been formulated to analyze the spread of Borrelia burgdorferi virus through tick vectors and mammalian hosts. The feasible region is established, and the boundedness of the model is verified. Analytically, the disease-free equilibrium and the basic reproduction number $(\Re_0)$ has been determined to assess outbreak potential. By virtue of the fixed-point theory, the existence and uniqueness of solutions has been established. The numerical simulations are obtained via the Runge–Kutta 4 method, demonstrating the model's ability to capture realistic disease progression. Finally, sensitivity analysis and control strategies (tick population reduction, host vaccination, public awareness, and early treatment) are evaluated, revealing that integrated control measures significantly reduce infection rates and enhance recovery.

Keywords: dynamical systems; fractional derivative; Lyme disease; local stability; sensitivity analysis; numerical simulations

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1. Introduction

Lyme disease the most prevalent vector-borne infection in the Northern Hemisphere, and it constitutes a substantial and growing public health challenge [1]. The disease is caused by bacteria of the Borrelia burgdorferi sensu lato complex. This disease is transmitted to humans through bites from infected black-legged ticks (Ixodes spp.) [2]. Incidence has escalated markedly in recent decades; the Centers for Disease Control and Prevention estimate approximately 476,000 cases annually in the United States, underscoring the considerable economic and health-care burden of the disease [3].

Transmission is maintained by a complex enzootic cycle involving ixodid ticks and multiple reservoir hosts, principally small mammals such as the white-footed mouse (Peromyscus leucopus) [4]. The tick life cycle from larval and nymphal to adult stages spans approximately two years and requires a blood meal at each stage. Nymphal ticks, due to their small size, exhibit peak activity during spring and summer, which accounts for the majority of human infections [5]. Consequently, the ecological determinants including host population dynamics, habitat fragmentation, and the climatic factors that affect tick survival and behavior strongly influence pathogen persistence and spatial spread [6,7]. The interaction of these biological and environmental drivers produces a dynamical system.

This system is often difficult to resolve through observational studies alone. The problems related to the memory effects can be more precisely seen in the applications of fractional and delayed fractional differential equations [8,9].

Mathematical modeling has become a great tool for elaborating infectious-disease dynamics. It provides a precise framework to quantify transmission risks. It evaluate control strategies and forecast outbreaks [10]. In Lyme disease research, compartmental ordinary differential equation models have been extensively used to simulate interactions among ticks, reservoir hosts, and human populations [11,12]. The fractional-order derivatives in the Lyme disease model introduce memory effects. These effects account for biological delays such as incubation, immune response, and delayed vector host interactions. Unlike classical integer-order derivatives, fractional models describe the non-local, history-dependent behavior of infection dynamics. This leads to a more accurate representation of the slow, persistent, and oscillatory nature of Lyme disease transmission [13]. To address these limitations, fractional calculus has been introduced into epidemiological and biological modeling. Caputo fractional derivatives, in particular incorporate memory and hereditary properties, allowing past states to influence future dynamics. This offers a more realistic representation of disease processes [14,15]. Fractional formulations have shown improved agreement with empirical data in other infectious disease contexts [16].

Several models for Lyme disease have been developed [17]; relatively few have employed fractional calculus to capture long-range dependencies associated with ticks' life history and host-seeking behavior. Moreover, the integration of optimal control theory with fractional-order epidemiological models remains unexplored for Lyme disease, despite its importance for designing time-adaptive, cost-effective interventions [18].

In this study, we develop a Caputo fractional-order model to analyze Lyme disease transmission dynamics. Our objectives are to: (i) formulate an eight-compartment model encompassing human, ticks and reservoir host populations; (ii) derive the basic reproduction number $(\Re_0)$ and establish the local stability properties of the equilibria; (iii) numerically solve the system using Runge–Kutta 4 method (RK-4); and (iv) perform sensitivity analysis to identify the parameters most influential to disease spread. The results aim to provide a deep understanding of Lyme disease dynamics and to inform evidence-based public health interventions to reduce its burden.

Definition 1. *Caputo Fractional Derivative [19] The Caputo derivative of order* $\alpha \in (0,1]$ *for a function* $f(t)$ *is:*

$$^{C}D^{\alpha}f(t) = \frac{1}{\Gamma(1-\alpha)}\int_0^t \frac{f^{'}(\tau)}{(t-\tau)^{\alpha}},$$

where $\Gamma(.)$ *is a gamma function.*

Definition 2. *Laplace Transformation [19]*

$$\mathcal{L}\{^{C}D^{\alpha}f(t)\}s = s^{\alpha}\mathbf{F}(s) - s^{\alpha-1}f(0).$$

Definition 3. *Mittag–Leffler transforms [20]*

$$\mathcal{L}\{E_{\alpha,1}(-at^{\alpha})\}(s) = \frac{s^{\alpha-1}}{s^{\alpha}+a}$$

$$\frac{1}{s(s^{\alpha}+a)} = \frac{1}{a}(\frac{1}{s} - \frac{s^{\alpha-1}}{s^{\alpha}+a}),$$

inverse transform is;

$$\mathcal{L}^{-1}\{\frac{1}{s(s^{\alpha}+a)}\}(t)=\frac{1}{a}(1-E_{\alpha,1}(-at^{\alpha})).$$

Definition 4 ([21]). *The Riemann–Liouville integral $\jmath(a)$, is defined as follows:*

$$\jmath^{a}_{0+}\Bbbk(t)=\frac{1}{\Gamma(a)}\int_{0}^{t}(t-\varrho)^{a-1}\Bbbk(\varrho,\Theta(\varrho))d\varrho,$$

where Γ is the gamma function and a is an arbitrary but fixed base point.

2. Model Formulation for Lyme Disease

The population of the given model has been divided into distinct categories to effectively capture the dynamics of disease transmission. Specifically, humans are divided into Susceptible (S_h), Exposed (E_h), Infected (I_h) and Recovered (R_h) compartments, while ticks are divided as Susceptible (S_t) or Infected (I_t). Moreover, reservoir hosts such as mice are classified into Susceptible (S_m) and Infected (I_m) groups. The transmission dynamics are characterized by the following key parameters: the rate of human infection from tick bites (β_h) and the rate at which ticks become infected through interactions with reservoir hosts (β_t). To enhance the model's applications in real-world problems, Caputo fractional derivatives are used to incorporate memory effects that more accurately reflect the delayed interactions between ticks and hosts.

2.1. Parameters

In the following table, details of the notations of the parameters used in this article are presented.

Parameter	Description
$\Lambda_h, \Lambda t, \Lambda_m$	Recruitment rates (humans, ticks, mice)
$\beta_h, \beta_t, \beta_m$	Transmission rates (human–tick, tick–mouse, mouse–tick)
μ_h, μ_t, μ_m	Natural death rates
σ_h	Progression rate from exposed to infected (humans)
γ_h	Recovery rate (humans)
d_h	Disease-induced mortality (humans)

2.2. System of Fractional Differential Equations

$$\begin{aligned}
{}^{C}D^{\alpha}S_h &= \Lambda_h-\beta_h S_h I_t-\mu_h S_h,\\
{}^{C}D^{\alpha}E_h &= \beta_h S_h I_t-(\sigma_h+\mu_h)E_h,\\
{}^{C}D^{\alpha}I_h &= \sigma_h E_h-(\gamma_h+\mu_h+d_h)I_h,\\
{}^{C}D^{\alpha}R_h &= \gamma_h I_h-\mu_h R_h,\\
{}^{C}D^{\alpha}S_t &= \Lambda_t-\beta_t S_t I_m-\mu_t S_t,\\
{}^{C}D^{\alpha}I_t &= \beta_t S_t I_m-\mu_t I_t,\\
{}^{C}D^{\alpha}S_m &= \Lambda_m-\beta_m S_m I_t-\mu_m S_m,\\
{}^{C}D^{\alpha}I_m &= \beta_m S_m I_t-\mu_m I_m,
\end{aligned}\tag{1}$$

with initial conditions of the system as follows:

$$\begin{aligned}
&S_h(0)=S_{h_0}\geq 0, E_h(0)=E_{h_0}\geq 0, I_h(0)=I_{h_0}\geq 0, R_h(0)=R_{h_0}\geq 0\\
&S_t(0)=S_{t_0}\geq 0, I_t(0)=I_{t_0}\geq 0, S_m(0)=S_{m_0}\geq, I_m(0)=I_{m0}\geq 0.
\end{aligned}$$

The mathematical model described above is presented in the flow chart in the next section in Figure 1.

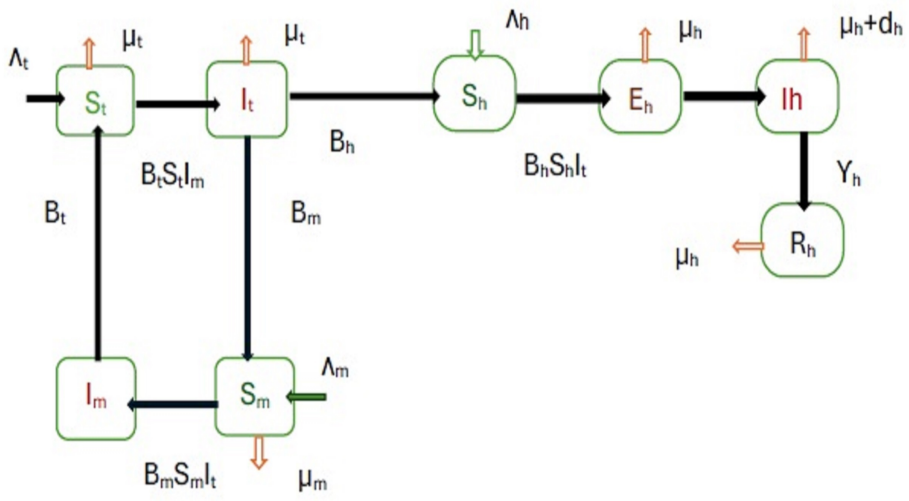


Figure 1. The compartments express disease transmission from vector to humans.

2.3. Compartmental Representation

The following diagram shows the relationship between different compartments. This diagram describes the model in terms of flow chart.

2.4. Why Fractional Derivatives?

The fractional-order derivatives provide more appropriate mathematical framework for modeling the dynamics of Lyme disease. This is because the disease's transmission process involves many biological delays and memory effects across ticks, mice and humans. These interactions are inherently time dependent, meaning that the present behavior in the system might be strongly influenced by its past states. Therefore, it is not working like classical derivatives, which work only instantaneously.

A major source of delay lies in the tick's life cycle. The Ixodes tick progresses through larval, nymphal, and adult stages over approximately two years. These transitions are affected by cumulative environmental conditions such as temperature and humidity from previous months or seasons. Fractional derivatives capture this "memory" of environmental influence. It enables the model to realistically account for how past climate conditions determine the current density of infectious nymphs.

Another essential time-dependent process is the acquisition and development of the Borrelia pathogen within ticks, which is transmitted when ticks feed on infected hosts such as white-footed mice. There is a latent period before the pathogen becomes established, often not until the tick molts into its next stage. The efficiency of this infection process depends on the population of mouse infection history. Fractional-order models naturally incorporate this temporal dependence by joining the present infection levels in ticks to the historical infection patterns, thus reflecting how the pathogen persists and circulates in the ecosystem.

Human infections also include a visible delay. After being bitten by an infected tick, the symptoms might appear only after an incubation period that typically lasts from 3 to 30 days. Fractional calculus allows the model to represent this distributed incubation behavior more accurately. By considering how current infection rates depend on exposure events from previous weeks, fractional models enhance predictive accuracy for disease spread and improve public health forecasting.

In summary, fractional derivatives provide a mathematically robust and biologically realistic ways to represent Lyme disease dynamics by integrating memory and delay effects that are fundamental to its transmission cycle.

3. Qualitative Properties of the Solution

This section examines the theoretical soundness of the fractional-order model from both a mathematical and biological perspective. We show that the Caputo fractional-order model's solution is bounded and positive as long as a positive initial conditions are specified. We demonstrate the existence and uniqueness of the system (1).

Let

$$\Theta(t) = (S_h, E_h, I_h, R_h, S_t, I_t, S_m, I_m)^T,$$

and

$$\begin{aligned}
\Bbbk(t, \Theta(t)) &= (\psi_1, \psi_2, \psi_3, \psi_4.\psi_5, \psi_6, \psi_7, \psi_8)^T, \\
\psi_1 &= \Lambda_h - \beta_h S_h I_t - \mu_h S_h, \\
\psi_2 &= \beta_h S_h I_t - (\sigma_h + \mu_h) E_h, \\
\psi_3 &= \sigma_h E_h - (\gamma_h + \mu_h + d_h) I_h, \\
\psi_4 &= \gamma_h I_h - \mu_h R_h, \\
\psi_5 &= \Lambda_t - \beta_t S_t I_m - \mu_t S_t, \\
\psi_6 &= \beta_t S_t I_m - \mu_t I_t, \\
\psi_7 &= \Lambda_m - \beta_m S_m I_t - \mu_m S_m, \\
\psi_8 &= \beta_m S_m I_t - \mu_m I_m.
\end{aligned}$$

The dynamical system (1) becomes

$$\begin{aligned}
{}^C D^{\alpha} \Theta(t) &= \Bbbk(t, \Theta(t)), \\
\text{with } \Theta(0) &= \Theta_0 \geq 0, \text{ for all } t \in [0, f] \text{ and some } a \in (0, 1].
\end{aligned} \tag{2}$$

Here, $\Theta_0 \geq 0$ is to be depicted component-wise. Model (1) is now represented by the fractional differential equation (2), which has the following integral representation

$$\begin{aligned}
\Theta(t) &= \Theta_0 + J_{0+}^{a} \Bbbk(t, \Theta(t)) \\
&= \Theta_0 + \frac{1}{\Gamma(a)} \int_0^t (t - \varrho)^{a-1} \Bbbk(\varrho, \Theta(\varrho)) d\varrho.
\end{aligned} \tag{3}$$

For analyzing model (1), let $\eta = C[0, T]$, $T > 0$, represent the Banach space of all continuous functions from $[0, T]$ to $\mathbb{R}$ with the norm,

$$\|\Theta\|_{\eta} = \sup_{t \in [0,T]} (|\Theta(t)|),$$

where

$$|\Theta(t)| = |S_h(t)| + |E_h(t)| + |I_h(t)| + |R_h(t)| + |S_t(t)| + |I_t(t)| + |S_m(t)| + |I_m(t)|.$$

We specify that $S_h, E_h, I_h, R_h, S_t, I_t, S_m, I_m \in C[0, T]$. Additionally, we define the operator $\Im : \eta \to \eta$ as follows:

$$(\Im \Theta)(t) = \Theta_0 + \frac{1}{\Gamma(a)} \int_0^t (t - \varrho)^{a-1} \Bbbk(\varrho, \Theta(\varrho)) d\varrho. \tag{4}$$

The continuity of $\Bbbk$ is obvious, implying that $\Im$ is well-defined.

3.1. Positivity and Boundedness of the Solution

The solution of the Caputo fractional-order derivative model (1) is positive and bounded.

Theorem 1. *The set*

$$\Omega = \left\{ (S_h, E_h, I_h, R_h, S_t, I_t, S_m, I_m) \in \mathbb{R}^8_+ : \begin{array}{c} S_h + E_h + I_h + R_h \leq \frac{\Lambda_h}{\mu_h}, \\ S_t + I_t \leq \frac{\Lambda_t}{\mu_t}, \\ S_m + I_m \leq \frac{\Lambda_m}{\mu_m} \end{array} \right\}$$

is positively invariant and the attraction region for System (1).

Proof. The variables in this model represent population compartments. Assume that the initial conditions are non-negative. We define the total populations for humans, ticks and reservoir hosts, respectively, as follows:

$$\begin{aligned} N_h(t) &= S_h(t) + E_h(t) + I_h(t) + R_h(t), \\ N_t(t) &= S_t(t) + I_t(t), \\ N_m(t) &= S_m(t) + I_m(t). \end{aligned}$$

Taking the Caputo derivative of order α on both sides and using the values from (1), we get:

$$\begin{aligned} {}^{C}D_t^{\alpha} N_h(t) &\leq \Lambda_h - \mu_h N_h(t), \\ {}^{C}D_t^{\alpha} N_t(t) &\leq \Lambda_t - \mu_t N_t(t), \\ {}^{C}D_t^{\alpha} N_m(t) &\leq \Lambda_m - \mu_m N_m(t). \end{aligned}$$

Now, applying Laplace transform [19] on both sides of each above inequality with $N_h(0) = N_{h_0}$ and $N_h(t) > 0$, we get

$$\mathcal{L}\{{}^{C}D_t^{\alpha} N_h(t)\} \leq \mathcal{L}\{\Lambda_h - \mu_h N_h(t)\},$$

$$\begin{aligned} s^{\alpha} N_h(s) - s^{\alpha-1} N_{h_0} &\leq \frac{\Lambda_h}{s} - \mu_h N_h(s), \\ (s^{\alpha} + \mu_h) N_h(s) &\leq s^{\alpha-1} N_{h_0} + \frac{\Lambda_h}{s}, \\ N_h(s) &\leq \frac{s^{\alpha-1} N_{h_0}}{s^{\alpha} + \mu_h} + \frac{\Lambda_h}{s(s^{\alpha} + \mu_h)}, \end{aligned}$$

taking the inverse Laplace and using Mittag–Leffler function [20].

$$\begin{aligned} \mathcal{L}^{-1}\{N_h(s)\} &\leq \mathcal{L}^{-1}\{\frac{s^{\alpha-1} N_{h_0}}{s^{\alpha} + \mu_h} + \frac{\Lambda_h}{s(s^{\alpha} + \mu_h)}\}, \\ N_h(t) &\leq N_{h_0} E_{\alpha,1}(-\mu_h t^{\alpha}) + \frac{\Lambda_h}{\mu_h}(1 - E_{\alpha,1}(-\mu_h t^{\alpha})), \end{aligned}$$

$$N_h(t) \leq \frac{\Lambda_h}{\mu_h} + (N_{h_0} - \frac{\Lambda_h}{\mu_h}) E_{\alpha,1}(-\mu_h t^{\alpha}),$$

the right-hand side tends to the equilibrium $\frac{\Lambda_h}{\mu_h}$ as $t \to \infty$ because $E_{\alpha,1}(-\mu_h t^{\alpha}) \to 0$.

$$N_h(t) \leq \frac{\Lambda_h}{\mu_h}. \tag{5}$$

Applying Laplace transform on $N_t(t)$ and $N_m(t)$ in a similar way, for ticks:

$$N_t(t) = N_t(0)E_\alpha(-\mu_t t^\alpha) + \frac{\Lambda_t}{\mu_t}[1 - E_\alpha(-\mu_t t^\alpha)] \leq \frac{\Lambda_t}{\mu_t}, \tag{6}$$

for reservoir hosts:

$$N_m(t) = N_m(0)E_\alpha(-\mu_m t^\alpha) + \frac{\Lambda_m}{\mu_m}[1 - E_\alpha(-\mu_m t^\alpha)] \leq \frac{\Lambda_m}{\mu_m}. \tag{7}$$

From (5)–(7), the set Ω is positively invariant under the fractional differential system of the Lyme disease model, and all initial solutions remain in Ω for all $t > 1$. Populations remain biologically feasible (non-negative and bounded). □

3.2. Existence and Uniqueness of the Solution

In this section, the existence and uniqueness of System (1) is obtained using the Lipschitz criterion. The following is the main result of this section.

Theorem 2. *Let*

$$\Theta^\bullet(t) = (S_h^\bullet, E_h^\bullet, I_h^\bullet, R_h^\bullet, S_t^\bullet, I_t^\bullet, S_m^\bullet, I_m^\bullet)^T$$

and

$$\Bbbk(t, \Theta(t)) = (\psi_1, \psi_2, \psi_3, \psi_4.\psi_5, \psi_6, \psi_7, \psi_8)^T,$$

defined above satisfies

$$\|\Bbbk(t, \Theta(t)) - \Bbbk(t, \Theta^\bullet(t))\|_\psi \leq \omega \|\Theta(t) - \Theta^\bullet(t)\|_\psi,$$

for some $\omega > 0$. Therefore, System (1) has a unique solution.

Proof. It is enough to prove that the function $\Bbbk$ satisfies the Lipschitz condition,

$$\begin{aligned}
& |\psi_1(t, \Theta(t)) - \psi_1(t, \Theta^\bullet(t))| \\
= \; & \left|\Lambda_h - \beta_h S_h(t) I_t(t) - \mu_h S_h(t) - (\Lambda_h - \beta_h S_h^\bullet(t) I_t^\bullet(t) - \mu_h S_h^\bullet(t))\right| \\
= \; & \left|-\beta_h S_h(t) I_t(t) - \mu_h S_h(t) + \beta_h S_h^\bullet(t) I_t^\bullet(t) + \mu_h S_h^\bullet(t))\right| \\
= \; & \left|\beta_h (S_h^\bullet(t) I_h^\bullet(t) - S_h(t) I_t(t)) + \mu_h (S_h^\bullet(t) - S_h(t))\right| \\
\leq \; & \left|\beta_h (S_h^\bullet(t) I_h^\bullet(t) - S_h(t) I_t^\bullet(t)) + S_h(t) I_t^\bullet(t) - S_h(t) I_t(t)) + \mu_h (S_h^\bullet(t) - S_h(t))\right| \\
\leq \; & \beta_h (\left|I_h^\bullet(t)\right| \left|(S_h^\bullet(t) - S_h(t)\right| + |S_h(t)||I_t^\bullet(t) - I_t(t)|) + \mu_h (\left|S_h^\bullet(t) - S_h(t)\right|),
\end{aligned}$$

assume state variables are bounded by some constant, i.e.,

$$\max\left\{|S_h(t)|, \left|S_h^\bullet(t)\right|, \left|I_h^\bullet(t)\right|, |I_h(t)|\right\} \leq \hat{W} \text{ for all } t \in [0, T]$$

therefore,

$$\begin{aligned}
& |\psi_1(t, \Theta(t)) - \psi_1(t, \Theta^\bullet(t))| \\
\leq \; & \beta_h (\hat{W} \left|(S_h^\bullet(t) - S_h(t)\right| + \hat{W} \left|I_h^\bullet(t) - I_t(t)\right|) + \mu_h (\left|S_h^\bullet(t) - S_h(t)\right|) \\
\leq \; & (\beta_h \hat{W} + \mu_h) \left|(S_h^\bullet(t) - S_h(t)\right| + \beta_h \hat{W} \left|I_h^\bullet(t) - I_t(t)\right|.
\end{aligned}$$

Similarly, for ψ_2,

$$\begin{aligned} & |\psi_2(t,\Theta(t)) - \psi_2(t,\Theta^\bullet(t)| \\ = \;& |\beta_h S_h I_t - (\sigma_h+\mu_h)E_h - (\beta_h S_h^\bullet I_t^\bullet - (\sigma_h+\mu_h)E_h^\bullet)|, \\ \leq \;& \beta_h(\hat{W}|I_t - I_t^\bullet| + \hat{W}\Big|(S_h(t) - S_h^\bullet(t)\Big| + (\sigma_h+\mu_h)|E_h - E_h^\bullet|, \end{aligned}$$

$$|\psi_3(t,\Theta(t)) - \psi_3(t,\Theta^\bullet(t)| \leq \sigma_h|E_h - E_h^\bullet| + (\gamma_h+\mu_h+d_h)|I_h - I_h^\bullet|$$

$$|\psi_4(t,\Theta(t)) - \psi_4(t,\Theta^\bullet(t)| \leq \gamma_h|I_h - I_h^\bullet| + \mu_h|R_h - R_h^\bullet|,$$

$$|\psi_5(t,\Theta(t)) - \psi_5(t,\Theta^\bullet(t))| \leq (\beta_t\hat{W}+\mu_t)\Big|(S_t(t) - S_t^\bullet(t)\Big| + \beta_t\hat{W}|I_m - I_m^\bullet|,$$

$$|\psi_6(t,\Theta(t)) - \psi_6(t,\Theta^\bullet(t))| \leq \beta_t(\hat{W}\Big|(S_t(t) - S_t^\bullet(t)\Big| + \hat{W}|I_m - I_m^\bullet| + \mu_t|I_t - I_t^\bullet|),$$

$$|\psi_7(t,\Theta(t)) - \psi_7(t,\Theta^\bullet(t))| \leq (\beta_m\hat{W}+\mu_m)\Big|(S_m^\bullet(t) - S_m(t)\Big| + \beta_m\hat{W}|I_t^\bullet - I_t|,$$

$$|\psi_8(t,\Theta(t)) - \psi_8(t,\Theta^\bullet(t))| \leq \beta_m(\hat{W}\Big|(S_m(t) - S_m^\bullet(t)\Big| + \hat{W}|I_t - I_t^\bullet| + \mu_m|I_m - I_m^\bullet|).$$

Then, we combine all differences using the maximum norm:

$$\begin{aligned} & \|\Bbbk(t,\Theta(t)) - \Bbbk(t,\Theta^\bullet(t))\|_\psi \\ = \;& \sup_{t\in[0,f]} \sum_{k-1}^{8} \psi_k(t,\Theta(t)) - \psi_k(t,\Theta^\bullet(t)) \leq \omega\|\Theta(t) - \Theta^\bullet(t)\|_\psi, \end{aligned}$$

where

$$\begin{aligned} \omega \;=\; & \max\{(\beta_h\hat{W}+\mu_h)+\beta_h\hat{W}, \beta_h\hat{W}+\beta_h\hat{W}+(\sigma_h+\mu_h), \\ & \sigma_h+(\gamma_h+\mu_h+d_h), \gamma_h+\mu_h, (\beta_t\hat{W}+\mu_t)+\beta_t\hat{W}, \\ & (\beta_t\hat{W}+\beta_t\hat{W}+\mu_t), (\beta_m\hat{W}+\mu_m)+\beta_m\hat{W}, (\beta_m\hat{W}+\beta_m\hat{W}+\mu_m), \end{aligned}$$

since all parameters and $\hat{W}$ are positive and finite, so $\omega > 0$, which shows that $\Bbbk$ is Lipschitz continuous. □

4. Local Stability and Existence of Equilibrium Points

In this section, the local stability and existence of equilibrium points are discussed.

4.1. Disease-Free Equilibrium (DFE) for the Lyme Disease Model

Definition 5. *System (1) admits a disease-free equilibrium is the state where no infection exists in the population,*

$$X_0^\circ = (S_h^\circ, 0, 0, 0, S_t^\circ, 0, S_m^\circ, 0),$$

which means no infected humans, no infected ticks and no infected reservoir hosts (mice) such as

$$I_h = 0, I_t = 0 \text{ and } I_m = 0.$$

Theorem 3. *The disease-free equilibrium point of system (1) is*

$$X_0^\circ = (\frac{\Lambda_h}{\mu_h}, 0, 0, 0, \frac{\Lambda_t}{\mu_t}, 0, \frac{\Lambda_m}{\mu_m}, 0).$$

Proof. Taking the right of System (1) equal to 0 and substituting the values of all infected compartments $E_h = I_h = R_h = I_t = I_m = 0$ in System (1) and solving, we get

$$S_h^\circ = \frac{\Lambda_h}{\mu_h}, S_t^\circ = \frac{\Lambda_t}{\mu_t} \text{ and } S_m^\circ = \frac{\Lambda_m}{\mu_m},$$

Finally obtaining the following:

$$X_0^\circ = (\frac{\Lambda_h}{\mu_h}, 0, 0, 0, \frac{\Lambda_t}{\mu_t}, 0, \frac{\Lambda_m}{\mu_m}, 0).$$

□

The Basic Reproduction Number $(\Re_0)$

The basic reproduction number $\Re_0$ is a fundamental threshold quantity in epidemiology that represents the average number of secondary infections produced by one typical infected individual in a completely susceptible population. For our Caputo fractional-order Lyme disease model, we compute $\Re_0$ using the well-established Next-Generation Matrix (NGM) method, as given in [22].

To construct the next-generation matrices, we first identify the compartments directly involved in the transmission of the disease. The infected compartments are exposed humans (E_h), infected humans (I_h), infected ticks (I_t) and infected reservoir hosts (I_m). The next-generation matrix method requires separating the new infection terms from the terms describing transitions within and out of the infected compartments. The system of equations for the infected compartments from (1) is as follows:

$$\begin{aligned}
{}^C D^\alpha E_h &= \beta_h S_h I_t - (\sigma_h + \mu_h) E_h, \\
{}^C D^\alpha I_h &= \sigma_h E_h - (\gamma_h + \mu_h + d_h) I_h, \\
{}^C D^\alpha I_t &= \beta_t S_t I_m - \mu_t I_t, \\
{}^C D^\alpha I_m &= \beta_m S_m I_t - \mu_m I_m.
\end{aligned}$$

Let $\mathcal{F}$ be a column vector of rates of new infections entering each infected compartment; the $\mathcal{F}$ terms represent processes that introduce new infections, such as ticks infecting humans $(\beta_h S_h I_t)$, mice infecting ticks $(\beta_t S_t I_m)$ and ticks infecting mice $(\beta_m S_m I_t)$. On the other hand, let (ν) be the column vector of the transition rates (including progression, recovery and death) out of each compartment. We define the vector of infected states as follows:

$$\mathbf{X} = (E_h, I_h, I_t, I_m)^T$$

$$\begin{aligned}
\mathcal{F}_{E_h} &= (\beta_h S_h I_t), \\
\mathcal{F}_{I_h} &= 0, \\
\mathcal{F}_{I_t} &= (\beta_t S_t I_m), \\
\mathcal{F}_{I_t} &= (\beta_m S_m I_t).
\end{aligned}$$

$$\begin{aligned}
\nu_{E_h} &= (\sigma_h + \mu_h) E_h, \\
\nu_{I_h} &= -\sigma_h E_h + (\gamma_h + \mu_h + d_h) I_h, \\
\nu_{I_t} &= \mu_t I_t, \\
\nu_{I_m} &= \mu_t I_m.
\end{aligned}$$

The ν term represent transition, natural death of ticks $(\mu_t I_t)$, mouse $(\mu_m I_m)$ and human progression $((\sigma_h + \mu_h) E_h)$, recovery/death $((\gamma_h + \mu_h + d_h) I_h)$. By separating these dynamics, the NGM method helps to analyze how infections spread and persist in the population. We linearize around the DFE $(S_h^\circ = \frac{\Lambda_h}{\mu_h}, S_t^\circ = \frac{\Lambda_t}{\mu_t}, S_m^\circ = \frac{\Lambda_m}{\mu_m})$ and $E_h = I_h = I_t = I_m = 0$.

The matrices F and V are the Jacobian matrices of $\mathcal{F}$ and v with respect to the infected compartments **X** evaluated at the DFE:

$$F = \left[\frac{\partial \mathcal{F}_i}{\partial X_j}\right]_{DFE}, V = \left[\frac{\partial v_i}{\partial X_j}\right]_{DFE}.$$

Calculating these derivatives of disease-free equilibrium compartments, the transmission matrix (F) details how new infections arise:

$$F = \begin{bmatrix} 0 & 0 & \beta_h S_h^\circ & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta_t S_t^\circ \\ 0 & 0 & 0 & \beta_m S_m^\circ \end{bmatrix}.$$

Transition matrix (V):

$$V = \begin{bmatrix} (\sigma_h + \mu_h) & 0 & 0 & 0 \\ -\sigma_h & \gamma_h + \mu_h + d_h & 0 & 0 \\ 0 & 0 & \mu_t & 0 \\ 0 & 0 & 0 & \mu_m \end{bmatrix}.$$

The inverse of V is as follows:

$$V^{-1} = \begin{bmatrix} \frac{1}{(\sigma_h+\mu_h)} & 0 & 0 & 0 \\ \frac{\sigma_h}{\gamma_h+\mu_h+d_h} & \frac{1}{\gamma_h+\mu_h+d_h} & 0 & 0 \\ 0 & 0 & \frac{1}{\mu_t} & 0 \\ 0 & 0 & 0 & \frac{1}{\mu_m} \end{bmatrix},$$

the Next-Generation Matrix FV^{-1} is therefore

$$FV^{-1} = \begin{bmatrix} 0 & 0 & \frac{\beta_h S_h^\circ}{\mu_t} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\beta_t S_t^\circ}{\mu_t} \\ 0 & 0 & \frac{\beta_m S_m^\circ}{\mu_t} & 0 \end{bmatrix}.$$

To calculate the spectral radius (ρ) of FV^{-1}, we solve the following:

$$\det(FV^{-1} - \lambda I) = 0$$

$$\begin{vmatrix} -\lambda & 0 & \frac{\beta_h S_h^\circ}{\mu_t} & 0 \\ 0 & -\lambda & 0 & 0 \\ 0 & 0 & -\lambda & \frac{\beta_t S_t^\circ}{\mu_m} \\ 0 & 0 & \frac{\beta_m S_m^\circ}{\mu_t} & -\lambda \end{vmatrix} = 0.$$

Solving the characteristic equation:

$$\lambda^2(\lambda^2 - \frac{\beta_m S_m^\circ \beta_t S_t^\circ}{\mu_t \mu_t}) = 0$$

$\lambda^2 = 0$ which means $\lambda_{1,2} = 0$ (as the R.H.S of (1) has fewer variables, so it can be possible to obtain some zero eigenvalues).

$$\lambda^2 = \frac{\beta_m S_m^\circ \beta_t S_t^\circ}{\mu_t \mu_t}$$

$\lambda_{3,4} = \pm\sqrt{\frac{\beta_m S_m^\circ \beta_t S_t^\circ}{\mu_t \mu_t}}$, highest eigenvalue is equal to $\Re_0$, so

$$\Re_0 = \sqrt{\frac{\beta_m S_m^\circ \beta_t S_t^\circ}{\mu_t \mu_t}}.$$

Substitute $S_m^\circ = \frac{\Lambda_m}{\mu_h}$ and $S_t^\circ = \frac{\Lambda_t}{\mu_t}$

$$\Re_0 = \sqrt{\frac{\beta_h \beta_t \Lambda_m \Lambda_t}{\mu_t^2 \mu_m^2}}.$$

This expression for $\Re_0$ clearly highlights the dependence of disease persistence on the transmission parameters between ticks and mice, their recruitment rates and their mortality rates. The threshold property $\Re_0 < 1$ for disease elimination and $\Re_0 > 1$ for disease persistence will be analyzed in the following stability sections.

Theorem 4. *If $\Re_0 < 1$, then the disease-free equilibrium of System (1) is asymptotically stable.*

Proof. The Jacobian matrix of System (1) is given as follows:

$$J_{X_0^*} = \begin{bmatrix} -\beta_h I_t - \mu_h & 0 & 0 & 0 & 0 \\ \beta_h I_t & -(\sigma_h + \mu_h) & 0 & 0 & 0 \\ 0 & \sigma_h & -(\sigma_h + \mu_h + d_h) & 0 & 0 \\ 0 & 0 & \gamma_h & -\mu_h & 0 \\ 0 & 0 & 0 & -\beta_t I_m - \mu_t & \beta_t S_t \\ 0 & 0 & 0 & \beta_t I_m & -\mu_t \\ 0 & 0 & -\beta_m S_m & -\beta_m I_t - \mu_m & 0 \\ 0 & 0 & \beta_m S_m & \beta_m I_t & -\mu_m \end{bmatrix}.$$

Evaluate at DFE ($I_T = I_m = 0, S_h = S_h^*$)

$$J_{X_0^*} = \begin{bmatrix} -\mu_h & 0 & 0 & 0 & 0 & -\beta_h S_h^\circ & 0 & 0 \\ 0 & -(\sigma_h+\mu_h) & 0 & 0 & 0 & \beta_h S_h^\circ & 0 & 0 \\ 0 & \sigma_h & -(\sigma_h+\mu_h+d_h) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_h & -\mu_h & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_t & 0 & 0 & -\beta_t S_t^\circ \\ 0 & 0 & 0 & 0 & 0 & -\mu_t & 0 & \beta_t S_t^\circ \\ 0 & 0 & 0 & 0 & 0 & -\beta_m S_m^\circ & -\mu_m & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_m S_m^\circ & 0 & -\mu_m \end{bmatrix}.$$

The eigenvalues (λ) are solutions to the following characteristic equation:

$$\det(J_{X_0^*} - \lambda I) = 0$$

$$J_{X_0^*} - \lambda I = \begin{bmatrix} -\mu_h-\lambda & 0 & 0 & 0 \\ 0 & -(\sigma_h+\mu_h)-\lambda & 0 & 0 \\ 0 & \sigma_h & -(\sigma_h+\mu_h+d_h)-\lambda & 0 \\ 0 & 0 & \gamma_h & -\mu_h-\lambda \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

$$\begin{bmatrix} 0 & -\beta_h S_h^\circ & 0 & 0 \\ 0 & \beta_h S_h^\circ & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ -\mu_t-\lambda & 0 & 0 & -\beta_t S_t^\circ \\ 0 & -\mu_t-\lambda & 0 & \beta_t S_t^\circ \\ 0 & -\beta_m S_m^\circ & -\mu_m-\lambda & 0 \\ 0 & \beta_m S_m^\circ & 0 & -\mu_m-\lambda \end{bmatrix}.$$

The Jacobian at DFE has an upper block-triangular form:

$$J_{X_0^*} = \begin{bmatrix} A & B \\ 0 & D \end{bmatrix},$$

where A = human compartments (S_h, E_h, I_h, R_h), D = tick–mouse compartments (S_t, I_t, S_m, I_m) and B = coupling terms (infections between humans and ticks/mice), so its determinant is the product of the determinants of the diagonal blocks.

$$|J_A - \lambda I| = \left| \begin{bmatrix} -\mu_h-\lambda & 0 & 0 & 0 \\ 0 & -(\sigma_h+\mu_h)-\lambda & 0 & 0 \\ 0 & \sigma_h & -(\sigma_h+\mu_h+d_h)-\lambda & 0 \\ 0 & 0 & \gamma_h & -\mu_h-\lambda \end{bmatrix} \right|$$

$$(-\mu_h-\lambda)(-(\sigma_h+\mu_h)-\lambda)(-(\sigma_h+\mu_h+d_h)-\lambda)(-\mu_h-\lambda) = 0$$

$\lambda_1 = -\mu_h, \lambda_2 = -(\sigma_h + \mu_h), \lambda_3 = -(\sigma_h + \mu_h + d_h), \lambda_4 = -\mu_h$ When the eigenvalues are all negative, it means the human dynamic is stable. Now,

$$|J_D - \lambda I| = \begin{bmatrix} -\mu_t - \lambda & 0 & 0 & -\beta_t S_t^\circ \\ 0 & -\mu_t - \lambda & 0 & \beta_t S_t^\circ \\ 0 & -\beta_m S_m^\circ & -\mu_m - \lambda & 0 \\ 0 & \beta_m S_m^\circ & 0 & -\mu_m - \lambda \end{bmatrix} = 0$$

$$-\mu_t - \lambda \begin{bmatrix} -\mu_t - \lambda & 0 & \beta_t S_t^\circ \\ -\beta_m S_m^\circ & -\mu_m - \lambda & 0 \\ \beta_m S_m^\circ & 0 & -\mu_m - \lambda \end{bmatrix} + \beta_t S_t^\circ \begin{bmatrix} 0 & -\mu_t - \lambda & 0 \\ 0 & -\beta_m S_m^\circ & -\mu_m - \lambda \\ 0 & \beta_m S_m^\circ & 0 \end{bmatrix} = 0$$

$$-\mu_t - \lambda[(-\mu_t - \lambda)(-\mu_m - \lambda)(-\mu_m - \lambda) + \beta_t S_t^\circ(-\mu_m - \lambda)(-\beta_m S_m^\circ)] = 0,$$

$$\left[(-\mu_t - \lambda)^2(-\mu_m - \lambda)^2 - (-\mu_t - \lambda)(-\mu_m - \lambda)\beta_t S_t^\circ \beta_m S_m^\circ)\right] = 0$$

$$\begin{aligned} (-\mu_t - \lambda)(-\mu_m - \lambda)[(-\mu_t - \lambda)(-\mu_m - \lambda) - \beta_t S_t^\circ \beta_m S_m^\circ)] &= 0 \\ [(-\mu_t - \lambda)(-\mu_m - \lambda) - \beta_t S_t^\circ \beta_m S_m^\circ)] &= 0, -\mu_t = \lambda, -\mu_m = \lambda) \end{aligned}$$

$$[(-\mu_t - \lambda)(-\mu_m - \lambda) - \beta_t S_t^\circ \beta_m S_m^\circ)]$$

This is equivalent to the following:

$$\lambda^2 + (\mu_t + \mu_m)\lambda + \mu_t \mu_m (1 - \Re_0^2) = 0. \tag{8}$$

Equation (5) is a quadratic whose roots determine the stability of the reservoir subsystem. We apply the Routh–Hurwitz stability criteria [23]. For a quadratic with positive coefficients, this condition is met if all coefficients are positive. The coefficients of the quadratic

$$a\lambda^2 + b\lambda + c = 0$$

$a = 1 > 0, b = \mu_t + \mu_m > 0, c = \mu_t \mu_m (1 - \Re_0^2)$. The coefficients a and b are always positive. The sign of c depends on $\Re_0$. If $\Re_0 < 1$, then $c > 0$. All coefficients are positive, and based on the Routh–Hurwitz criteria, both roots of the quadratic have negative real parts. Consequently, all eigenvalues of $J_{X_0^*}$ have negative real parts. If $\Re_0 < 1$, both roots have negative real parts, indicating that the disease-free equilibrium is stable. □

4.2. The Endemic Equilibrium (EE) for the Lyme Disease Model

Determining the endemic equilibrium (EE) of the Lyme disease model means finding the steady-state where the disease persists in the population (at least one of the infected compartments is positive). This equilibrium exists and is stable when the basic reproduction number $\Re_0 > 1$.

Theorem 5. *The endemic equilibrium point of System (1) is*

$$E_1^* = (S_h^*, E_h^*, I_h^*, R_h^*, S_t^*, I_t^*, S_m^*, I_m^*),$$

where

$$S_h^* = \frac{\Lambda_h}{\mu_h + \beta_h I_t^*}, E_h^* = \frac{\beta_h S_h^* I_t^*}{\sigma_h + \mu_h}, I_h^* = \frac{\sigma_h E_h^*}{\gamma_h + \mu_h + d_h}, R_h^* = \frac{\gamma_h I_h^*}{\mu_h}$$

$$I_t^* = \frac{\beta_t S_t^* I_m^*}{\mu_t}, I_m^* = \frac{\beta_m S_m^* I_t^*}{\mu_m}$$

$$S_t^* = \frac{\Lambda_t}{\mu_t + \beta_t I_m^*}, S_m^* = \frac{\Lambda_m}{\mu_m + \beta_m I_t^*}.$$

Proof. To obtained the endemic equilibrium point, substituting

$${}^C D_t^\alpha S_h = {}^C D_t^\alpha E_h = {}^C D_t^\alpha I_h = {}^C D_t^\alpha R_h = {}^C D_t^\alpha S_t = {}^C D_t^\alpha I_t = {}^C D_t^\alpha S_m = {}^C D_t^\alpha I_m = 0$$

in (1) and solving, we get

$$S_h^* = \frac{\Lambda_h}{\mu_h + \beta_h I_t^*}, E_h^* = \frac{\beta_h S_h^* I_t^*}{\sigma_h + \mu_h}, I_h^* = \frac{\sigma_h E_h^*}{\gamma_h + \mu_h + d_h}, R_h^* = \frac{\gamma_h I_h^*}{\mu_h}$$

$$I_t^* = \frac{\beta_t S_t^* I_m^*}{\mu_t}, I_m^* = \frac{\beta_m S_m^* I_t^*}{\mu_m}$$

$$S_t^* = \frac{\Lambda_t}{\mu_t + \beta_t I_m^*}, S_m^* = \frac{\Lambda_m}{\mu_m + \beta_m I_t^*}.$$

Now, let us express S_t^* and S_m^* in terms of the infection rate.

$$\begin{aligned} \lambda_t^* &= \beta_t I_m^* \text{ (the infection rate on ticks)} \\ \lambda_m^* &= \beta_m I_t^* \text{ (the infection rate on mice)} \end{aligned}$$

$$S_t^* = \frac{\Lambda_t}{\mu_t + \lambda_t^*}, S_m^* = \frac{\Lambda_m}{\mu_m + \lambda_m^*}$$

We substitute the expression for I_t^* into the equation for I_m^*,

$$I_m^* = \frac{\beta_m S_m^* (\frac{\beta_t S_t^* I_m^*}{\mu_t})}{\mu_m}.$$

Assume $I_m^* \neq 0$; we can divide I_m^* on both sides, and we get

$$\begin{aligned} 1 &= \frac{\beta_m S_m^* \beta_t S_t^*}{\mu_m \mu_t} \\ S_t^* S_m^* &= \frac{\mu_m \mu_t}{\beta_t \beta_m} \end{aligned}$$

$$I_m^* = \frac{\Lambda_m}{\mu_m} - \frac{\mu_m \mu_t}{\beta_t \beta_m S_t^*}.$$

The endemic equilibrium exists if and only if $I_m^* > 0$ and $I_t^* > 0$, from the expression for I_m^*:

$$\begin{aligned} I_m^* &= \frac{\Lambda_m}{\mu_m} - \frac{\mu_m \mu_t}{\beta_t \beta_m S_t^*} > 0 \\ \frac{\Lambda_m}{\mu_m} &> \frac{\mu_m \mu_t}{\beta_t \beta_m S_t^*}. \end{aligned}$$

Using $S_m^\circ = \frac{\Lambda_m}{\mu_m}$(disease- free equilibrium value),this inequality simplifies to the condition $\Re_0 > 1$.The Endemic Equilibrium (E_1^*) exists when $\Re_0 > 1$. □

Theorem 6. *If $\Re_0 > 1$, then the endemic equilibrium is locally asymptotically stable.*

Proof. Evaluating the Jacobian at the endemic equilibrium point (E_1^*) and analyzing its stability via the characteristic equation is the core of local stability analysis. The general form of the Jacobian $J_{E_1^*}$ for the Lyme disease model is as follows:

$$J_{E_1^*} = \begin{bmatrix} -\beta_h I_t^* - \mu_h & 0 & 0 & 0 \\ \beta_h I_t^* & -(\sigma_h + \mu_h) & 0 & 0 \\ 0 & \sigma_h & -(\sigma_h + \mu_h + d_h) & 0 \\ 0 & 0 & \gamma_h & -\mu_h \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

$$\begin{bmatrix} 0 & -\beta_h S_t^* & 0 & 0 \\ 0 & \beta_h S_h^* & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ -\beta_t I_m^* - \mu_t & 0 & 0 & \beta_t S_t^* \\ \beta_t I_m^* & -\mu_t & 0 & \beta_t S_t^* \\ 0 & -\beta_m S_m^* & -\beta_m I_t^* - \mu_m & 0 \\ 0 & \beta_m S_m^* & \beta_m I_t^* & -\mu_m \end{bmatrix}.$$

The characteristic equation is as follows:

$$\det(J_{E_1^*} - \lambda I) = 0.$$

The matrix $(J_{E_1^*} - \lambda I)$ has a block upper triangular structure. This means it can be partitioned as follows:

$$(J_{E_1^*} - \lambda I) = \begin{bmatrix} A_{E_1^*} & B_{E_1^*} \\ 0 & D_{E_1^*} \end{bmatrix},$$

where $A_{E_1^*}$ is the human compartments (S_h, E_h, I_h, R_h), $D_{E_1^*}$, corresponding to the vector-reservoir compartments (S_t, I_t, S_m, I_m), and $B_{E_1^*}$ = Coupling terms (infections between humans and ticks/mice) and zero block, meaning humans do not affect the vector–reservoir dynamics in this model, so its determinant is the product of the determinants of the diagonal blocks. The eigenvalues of the full system are the combined eigenvalues of block $A_{E_1^*}$ and $D_{E_1^*}$ block.

$$\det(J_{E_1^*} - \lambda I) = \left| \begin{bmatrix} A_{E_1^*} - \lambda I & B_{E_1^*} \\ 0 & D_{E_1^*} - \lambda I \end{bmatrix} \right|.$$

Now, we solve the characteristic equation of block $A_{E_1^*}$,

$$\det(A_{E_1^*} - \lambda I) = 0$$

$$\left| \begin{bmatrix} -\beta_h I_t^* - \mu_h - \lambda & 0 & 0 & 0 \\ \beta_h I_t^* & -(\sigma_h + \mu_h) - \lambda & 0 & 0 \\ 0 & \sigma_h & -(\sigma_h + \mu_h + d_h) - \lambda & 0 \\ 0 & 0 & \gamma_h & -\mu_h - \lambda \end{bmatrix} \right| = 0.$$

This is the lower triangular matrix. The determinant of a triangular matrix is simply the product of its diagonal elements.

$$(-\beta_h I_t^* - \mu_h - \lambda)(-(\sigma_h + \mu_h) - \lambda)(-(\sigma_h + \mu_h + d_h) - \lambda)(-\mu_h - \lambda) = 0$$

$$\begin{aligned} -(\beta_h I_t^* + \mu_h) &= \lambda_1 \\ -(\sigma_h + \mu_h) &= \lambda_2 \\ -(\sigma_h + \mu_h + d_h) &= \lambda_3 \\ -(\sigma_h + \mu_h + d_h) &= \lambda_4. \end{aligned}$$

All four eigenvalues from the human subsystem are strictly negative and real. This part of the system is always stable at the endemic equilibrium.
Now, we solve the characteristic equation of block $D_{E_1^*}$,

$$\det(D_{E_1^*} - \lambda I) = 0,$$

$$\left| \begin{bmatrix} -\beta_t I_m^* - \mu_t - \lambda & 0 & 0 & \beta_t S_t^* \\ \beta_t I_m^* & -\mu_t - \lambda & 0 & \beta_t S_t^* \\ 0 & -\beta_m S_m^* & -\beta_m I_t^* - \mu_m - \lambda & 0 \\ 0 & \beta_m S_m^* & \beta_m I_t^* & -\mu_m - \lambda \end{bmatrix} \right| = 0$$

$$(-\beta_t I_m^* - \mu_t - \lambda) \left| \begin{bmatrix} -\mu_t - \lambda & 0 & \beta_t S_t^* \\ -\beta_m S_m^* & -\beta_m I_t^* - \mu_m - \lambda & 0 \\ \beta_m S_m^* & \beta_m I_t^* & -\mu_m - \lambda \end{bmatrix} \right|$$

$$-(\beta_t I_m^*) \left| \begin{bmatrix} 0 & 0 & \beta_t S_t^* \\ -\beta_m S_m^* & -\beta_m I_t^* - \mu_m - \lambda & 0 \\ \beta_m S_m^* & \beta_m I_t^* & -\mu_m - \lambda \end{bmatrix} \right| = 0$$

$$\begin{aligned} &(-\beta_t I_m^* - \mu_t - \lambda)\{(-\mu_t - \lambda)(-\beta_m I_t^* - \mu_m - \lambda)(-\mu_m - \lambda) + \\ &\beta_t S_t^* \{(-\beta_m S_m^*)(\beta_m I_t^*) - (-\beta_m I_t^* - \mu_m - \lambda)(\beta_m S_m^*)\} \end{aligned}$$

$$-(\beta_t I_m^*)\{-\beta_m S_m^*(\beta_m I_t^*) - (-\beta_m I_t^* - \mu_m - \lambda)(\beta_m S_m^*) = 0$$

$$\begin{aligned} &(-\beta_t I_m^* - \mu_t - \lambda)\{(-\mu_t - \lambda)(-\mu_m - \lambda)(-\beta_m I_t^* + 1) + \\ &\beta_t S_t^* \beta_m S_m^*)(\beta_m I_t^*)\{-2 - \mu_m - \lambda)\} \end{aligned}$$

$$+(\beta_t I_m^*)(\beta_m S_m^*)\{-\mu_m - \lambda) = 0$$

$$\begin{aligned} &\beta_m I_t^* \{(-1 - \mu_t - \lambda)(-\mu_t - \lambda)(-\mu_m - \lambda)(-1 + 1)\} + \\ &\beta_t S_t^* \beta_m S_m^*)(\beta_m I_t^*)\{-2 - \mu_m - \lambda)(-\beta_t I_m^* - \mu_t - \lambda)\} \end{aligned}$$

$$+(\beta_t I_m^*)(\beta_m S_m^*)(-\mu_m - \lambda) = 0$$

$$(\beta_t S_t^* \beta_m S_m^* \beta_m I_t^* \beta_t I_m^*)\{-2 - \mu_m - \lambda)(-1 - \mu_t - \lambda)\} + (\beta_m S_m^*)\{-\mu_m - \lambda) = 0$$

$$P\lambda^2 + [P(3 + \mu_m + \mu_t) - Q]\lambda + [P(2 + \mu_m)(1 + \mu_t) - Q\mu_m] = 0,$$

We get the following quadratic equation:

$$p\lambda^2 + q\lambda + r = 0,$$

where $P = \beta_t S_t^* \beta_m S_m^* \beta_m I_t^* \beta_t I_m^*$ and $Q = \beta_m S_m^*$ $p = P$, $q = P(3 + \mu_m + \mu_t) - Q$ $r = P(2 + \mu_m)(1 + \mu_t) - Q\mu_m$ For a quadratic equation, the Routh-Hurwitz array is:

$$\begin{array}{c|cc} \lambda^2 & p & r \\ \lambda^1 & q & 0 \\ \lambda^0 & r & \end{array}$$

We apply the Routh–Hurwitz conditions [23] for stability, for all roots to have negative real parts such that the system is stable, the following must hold:

$$p > 0, q > 0 \text{ and } r > 0.$$

Condition 1: $p > 0$

$$P = \beta_t S_t^* \beta_m S_m^* \beta_m I_t^* \beta_t I_m^*.$$

Since all parameters are typically positive, as they represent transmission rates and populations, we have $p > 0$. Condition 2: $q > 0$

$$\begin{aligned} q &= P(3 + \mu_m + \mu_t) - Q > 0 \\ P(3 + \mu_m + \mu_t) &> Q \end{aligned}$$

$$\beta_t S_t^* \beta_m S_m^* \beta_m I_t^* \beta_t I_m^* (3 + \mu_m + \mu_t) > \beta_m S_m^*.$$

Divide both sides $(\beta_m S_m^*)$ and we get

$$\beta_t S_t^* \beta_m I_t^* \beta_t I_m^* (3 + \mu_m + \mu_t) > 1.$$

Condition 3: $r > 0$

$$r = P(2 + \mu_m)(1 + \mu_t) - Q\mu_m > 0$$

$$r = \beta_t S_t^* \beta_m S_m^* \beta_m I_t^* \beta_t I_m^* (2 + \mu_m)(1 + \mu_t) > \beta_m S_m^* \mu_m.$$

Divide both sides by $(\beta_m S_m^* \mu_m)$, we get

$$\frac{\beta_t S_t^* \beta_m I_t^* \beta_t I_m^* (2 + \mu_m)(1 + \mu_t)}{\mu_m} > 1.$$

In the endemic equilibrium points discussion in Section 4.2, we proved that if $\Re_0 > 1$, then I_m^*, I_t^* and S_t^* are positive. Hence, condition 3 proved that the eigenvalues of $J_{E_1^*}$ are the roots of the characteristic polynomials of the stable human block $A_{E_1^*}$ and block $D_{E_1^*}$. The human block $A_{E_1^*}$ contributes four negative real eigenvalues, while numerical analysis of the characteristic polynomial of block $D_{E_1^*}$ over a wide range of parameter values satisfying $\Re_0 > 1$ consistently shows that all its eigenvalues also have negative real parts. Therefore, all eigenvalues of $J_{E_1^*}$ have negative real parts, confirming that the endemic equilibrium is locally asymptotically stable whenever $\Re_0 > 1$. □

5. Sensitivity Analysis of the Basic Reproduction Number ($\Re_0$)

From the previous analysis, the basic reproduction number for the Lyme disease model is:

$$\Re_0 = \sqrt{\frac{\beta_h \beta_t \Lambda_m \Lambda_t}{\mu_t^2 \mu_m^2}}.$$

As we know from reference [24], this sensitivity index of $\Re_0$ with respect to a parameter p measures the percentage change resulting from a 1% increase in p:

$$\Gamma_p^{\Re_0} = \frac{\partial \Re_0}{\partial p} . \frac{p}{\Re_0}.$$

Let us verify sensitivity indices; for parameters β_h, β_t, Λ_m and Λ_t, consider β_h first

$$\frac{\partial \Re_0}{\partial \beta_h} = \frac{\partial}{\partial \beta_h}\left(\frac{\beta_h \beta_t \Lambda_m \Lambda_t}{\mu_t^2 \mu_m^2}\right)^{\frac{1}{2}}$$
$$\frac{\partial \Re_0}{\partial \beta_h} = \frac{1}{2} . \frac{1}{\Re_0} . \frac{\beta_t \Lambda_m \Lambda_t}{\mu_t^2 \mu_m^2}.$$

From formula

$$\frac{\Re_0^2}{\beta_h} = \frac{\beta_t \Lambda_m \Lambda_t}{\mu_t^2 \mu_m^2}.$$

Therefore,

$$\frac{\partial \Re_0}{\partial \beta_h} = \frac{1}{2} . \frac{1}{\Re_0} . \frac{\Re_0^2}{\beta_h}.$$

Now, the sensitivity index is:

$$\Gamma_{\beta_h}^{\Re_0} = \frac{\partial \Re_0}{\partial \beta_h} . \frac{\beta_h}{\Re_0} = \frac{\Re_0}{2\beta_h} . \frac{\beta_h}{\Re_0} = \frac{1}{2} = 0.5,$$
$$\Gamma_{\beta_t}^{\Re_0} = \frac{\partial \Re_0}{\partial \beta_t} . \frac{\beta_t}{\Re_0} = \frac{\Re_0}{2\beta_t} . \frac{\beta_t}{\Re_0} = \frac{1}{2} = 0.5,$$
$$\Gamma_{\Lambda_m}^{\Re_0} = \frac{\partial \Re_0}{\partial \Lambda_m} . \frac{\Lambda_m}{\Re_0} = \frac{\Re_0}{2\Lambda_m} . \frac{\Lambda_m}{\Re_0} = \frac{1}{2} = 0.5,$$
$$\Gamma_{\Lambda_t}^{\Re_0} = \frac{\partial \Re_0}{\partial \Lambda_t} . \frac{\Lambda_t}{\Re_0} = \frac{\Re_0}{2\Lambda_t} . \frac{\Lambda_t}{\Re_0} = \frac{1}{2} = 0.5.$$

Now, we check the sensitivity indices for parameters μ_t and μ_m:

$$\frac{\partial \Re_0}{\partial \mu_t} = \frac{\partial}{\partial \mu_t}\left(\frac{\beta_h \beta_t \Lambda_m \Lambda_t}{\mu_t^2 \mu_m^2}\right)^{\frac{1}{2}},$$
$$\frac{\partial \Re_0}{\partial \mu_t} = \frac{1}{2} . \left(\frac{\beta_h \beta_t \Lambda_m \Lambda_t}{\mu_t^2 \mu_m^2}\right)^{-\frac{1}{2}} . \left(-\frac{2\beta_h \beta_t \Lambda_m \Lambda_t}{\mu_t^3 \mu_m^2}\right),$$

$$\frac{\partial \Re_0}{\partial \mu_t} = -\frac{1}{\Re_0} . \frac{2\beta_h \beta_t \Lambda_m \Lambda_t}{\mu_t^3 \mu_m^2} = -\frac{1}{\Re_0} . \frac{\Re_0^2}{\mu_t}.$$

Now, the sensitivity index for μ_t and μ_m is:

$$\Gamma_{\mu_t}^{\Re_0} = \frac{\partial \Re_0}{\partial \mu_t} . \frac{\mu_t}{\Re_0} = -\frac{1}{\Re_0} . \frac{\Re_0^2}{\mu_t} . \frac{\mu_t}{\Re_0} = -1,$$
$$\Gamma_{\mu_m}^{\Re_0} = \frac{\partial \Re_0}{\partial \mu_m} . \frac{\mu_m}{\Re_0} = -\frac{1}{\Re_0} . \frac{\Re_0^2}{\mu_m} . \frac{\mu_m}{\Re_0} = -1.$$

The sensitivity indices for each parameter are as follows

Parameter	Sensitivity Index	Interpretation
β_h	$+0.5$	Positive and Sensitive (P&A)
β_t	$+0.5$	P&A
Λ_m	$+0.5$	P&A
Λ_t	$+0.5$	P&A
μ_t	-1.0	Most Sensitive
μ_m	-1.0	Most Sensitive
Λ_m	$+0.5$	P&A

Using mid-range values from the provided table and assuming recruitment rates

$$\Re_0 = \left(\frac{(0.05)(0.45)(0.0005)(0.03)}{(0.0125)^2(0.03)^2} \right)^{\frac{1}{2}}$$
$$\Re_0 = \sqrt{2.4} \approx 1.549$$

this indicates an endemic state where the disease will persist.

6. Sensitivity Ranking and Public Health Implications

Rank	Parameter	Sensitivity Index	Control Strategy
1	μ_t	-1.0	habitat modification
2	$\beta_h, \beta_t, \Lambda_t, \Lambda_m$	$+0.5$	Personal protection, host vaccination
3	μ_m	-1.0	Population control

Figure 2: This figure illustrates how the basic reproduction number $\Re_0$ varies across a risk spectrum. It presents three specific $\Re_0$ values, a low 0.30, indicating the disease will be in finishing stage, a moderate 1.55, signifying sustained transmission, and a high value 7.14, reflecting rapid, exponential spread. These are contextualized against an "Epidemic Threshold" of 19.60, which represents an extreme, worst-case scenario for transmission.

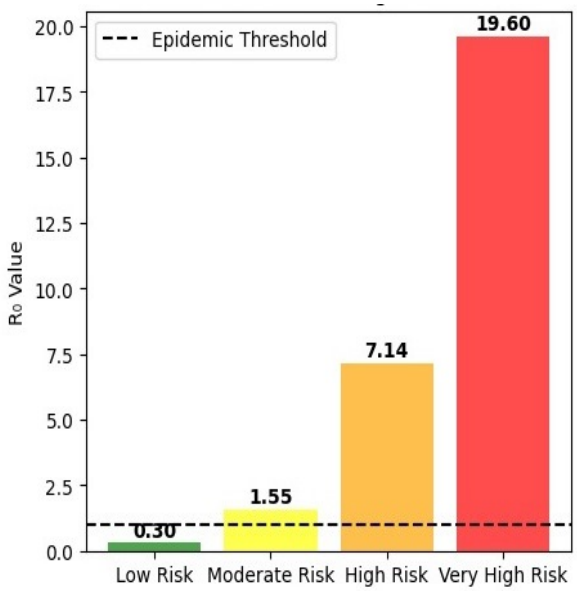


Figure 2. $(\Re_0)$ under different ecological conditions.

Figure 3: The graph shows that both tick control and human occupation measures are the most effective strategies, each leading to a 33.3% reduction in transmission. Mouse control

is the next most effective, at 22.5%. Public awareness campaigns have a lower though still significant impact at 16.3%. The data suggests that a combined approach, indicated by the combined strategy, would integrate these methods to achieve a cumulative reduction.

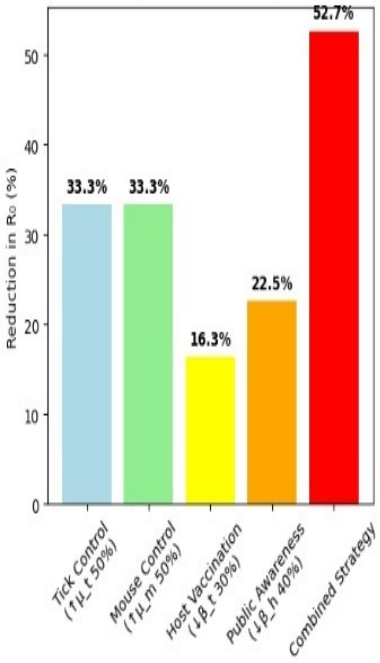


Figure 3. Effectiveness of control strategies.

Figure 4: This figure presents a sensitivity analysis measuring how changes in various input parameters affect the value of the basic reproduction number $(\Re_0)$. The chart displays the sensitivity index values for different parameters, with a notable cluster showing a significant negative impact. Four parameters have a sensitivity index of $+0.50$, indicating that an increase in these parameters would cause a moderate increase in $\Re_0$. In contrast, two other parameters μ_t and μ_m have a much stronger influence, each with a sensitivity index of -1.0. This means that increasing these specific parameters would cause a proportional and direct decrease in the $\Re_0$ value.

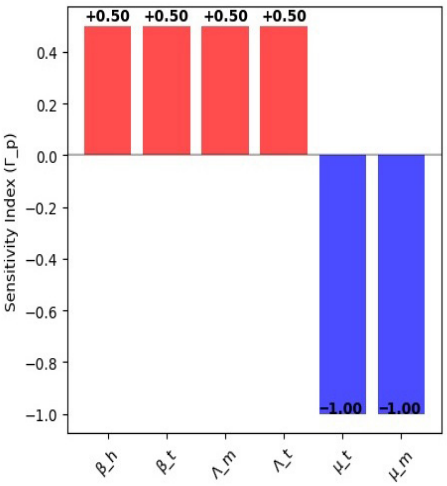


Figure 4. Sensitivity analysis of $(\Re_0)$ parameters.

7. Numerical Simulations

The numerical simulations of the proposed fractional-order epidemic models were carried out using the Runge–Kutta 4 method for the Caputo derivative case. The model captures the interactions between humans, ticks, and mice through their susceptible, exposed, infected, and recovered compartments.

Parameter	Range	Source	Note
μ_h	0.000034–0.000039	[25]	70–80 year life span
σ_h	0.083–0.167	[23]	6–12 days in incubation
γ_h	0.01–0.3	[26]	3–10 days antibiotic treatment
d_h	0.001–0.01	[24]	Very low mortality
μ_t	0.005–0.02	[24]	50–200 day life span
β_t	0.1–0.8	[27]	per day
μ_m	0.01–0.05	[3]	Mouse mortality (20–100 day life span)
β_m	0.3–0.9	[3]	per day
Λ_h	0.001–0.5	[28]	per day
Λ_t	0.01–0.5	[28]	per day
Λ_m	0.01–0.5	[28]	per day

Figure 5: This graph shows the ${}^{C}D^{\alpha}S_h$ from System (1) using the Fractional RK-4 method. It captures the memory-dependent dynamics of the susceptible population and demonstrates that $S_h(t)$ converges smoothly toward a stable equilibrium, indicating asymptotic stability of the system. Fractional-order $\alpha = 0.95$, Equilibrium $S_h^* = 1.4286$, Initial $S_h(0) = 8000.0$, Final $S_h(T) = 9.3548$ with the parameters $\Lambda = 0.1, \beta = 0.001$, $\mu = 0.05$ and $I_m = 300$.

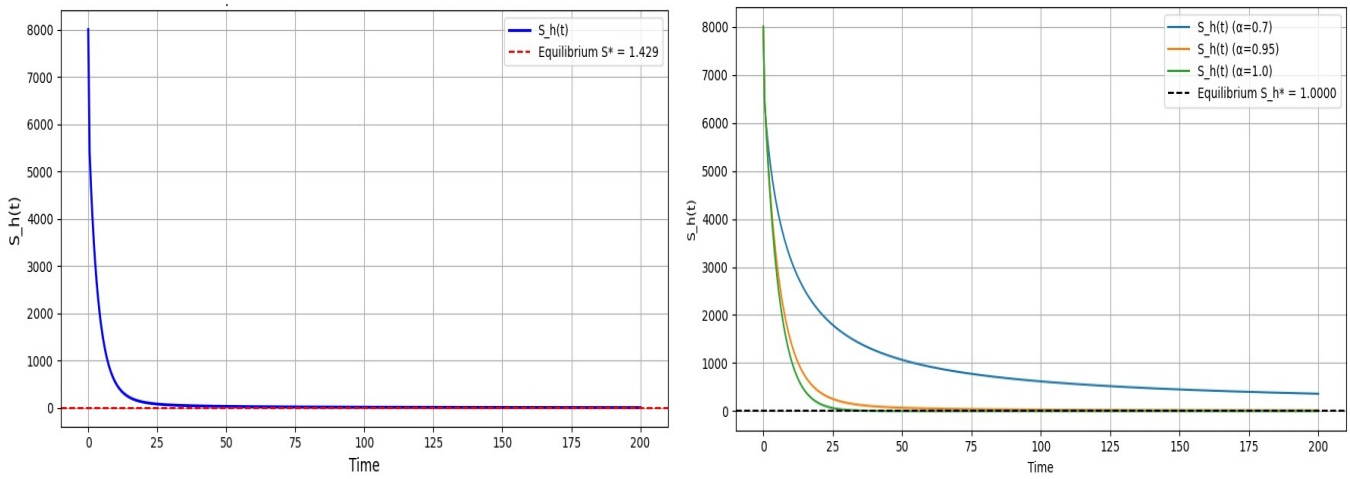


Figure 5. Susceptible human population dynamics for different α values using the RK-4 method.

Figure 6: The fractional-order model with $\alpha = 0.95$ shows that $S_t(t)$ gradually decreases and stabilizes near its equilibrium value. The trajectory exhibits smooth convergence due to the memory effect, confirming asymptotic stability of the susceptible tick population.

Figure 7: The numerical solution shows that $S_m(t)$ becomes stable near its equilibrium. The system exhibits asymptotic stability, with fractional memory smoothing the rate of variation. Fractional-order $\alpha = 0.95$, Equilibrium $S_m^* = 0.3529$, and Initial $S_m(0) = 3000$, Final $S_m(T) = 1.5512$.

Figure 8: The fractional infection dynamics show that $I_h(t)$ approaches a finite steady state, indicating disease stability in the human population. The fractional order captures persistence due to historical infection effects. Fractional-order $\alpha = 0.95$, Equilibrium $I_h^* = 5454.5455$. Initial $I_h(0) = 2000$, Final $I_h(T) = 5448.9766$.

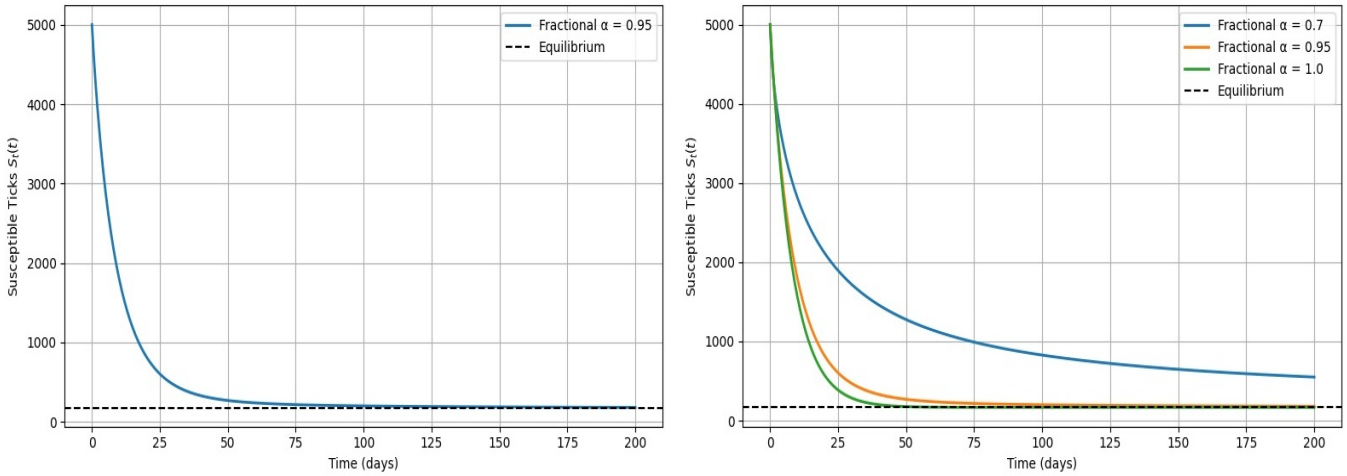


Figure 6. Susceptible tick population dynamics for different α values using the RK-4 method.

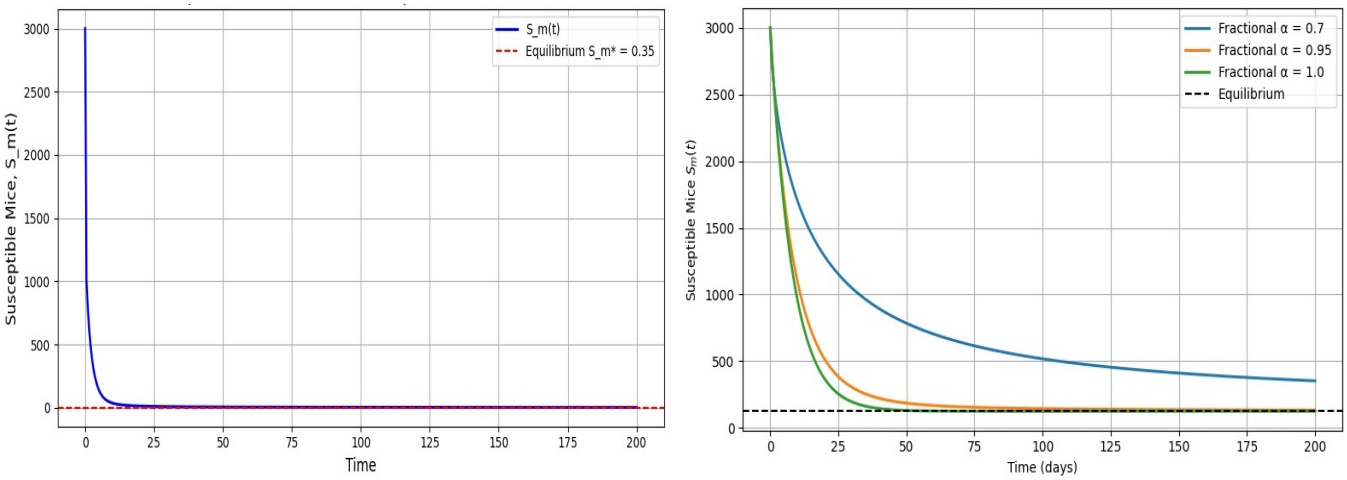


Figure 7. Susceptible mouse population dynamics for different α values using the RK-4 method.

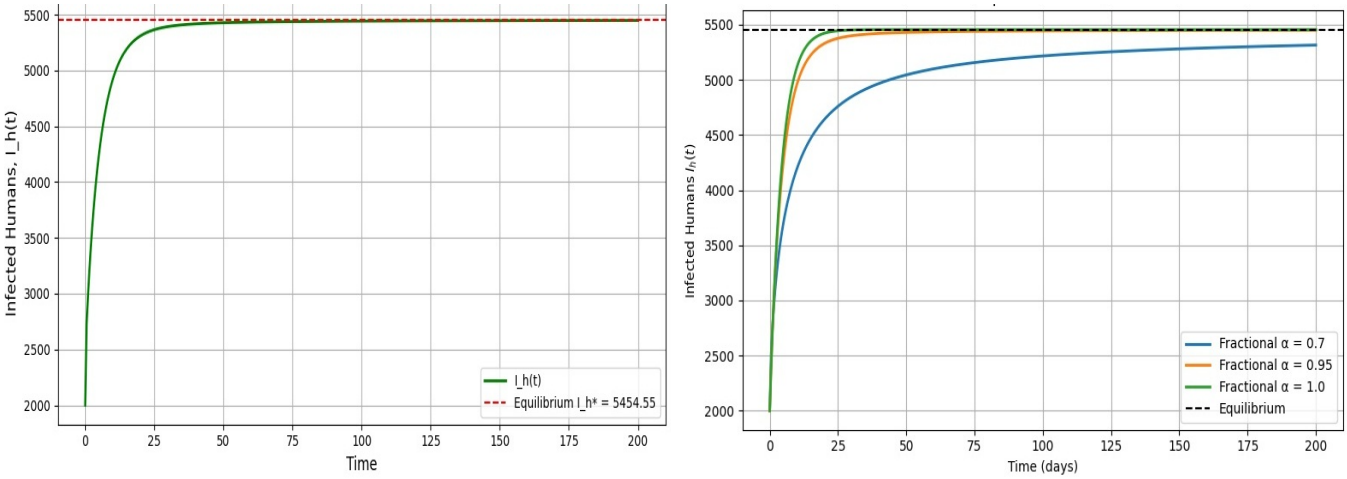


Figure 8. Infected human population dynamics for different α values using the RK-4 method.

Figure 9: The fractional-order model for infected ticks shows that $I_t(t)$ increases initially due to transmission from infected mice and then gradually becomes stable near its equilibrium point. The system exhibits asymptotic stability, indicating that infection levels in the tick population remain bounded over time. Fractional-order $\alpha = 0.95$, Equilibrium I_t^* = 70,000, and Initial $I_t(0) = 1000$, Final $I_t(T)$ = 69,340.7346.

Figure 10: The infected mouse population increases initially and then becomes stable at its equilibrium point. The fractional model captures long-term memory effects, leading to a smooth and stable asymptotic approach. $\alpha = 0.95$, $\beta_m = 0.0008$, $\mu_m = 0.05$. Equilibrium I_m^* = 48,000, $I_m(0) = 700$, $I_m(T)$ = 47,548.068785.

Figure 11: The fractional Caputo model reveals that $R_h(t)$ graphed smoothly near equilibrium. The memory term $(\alpha = 0.95)$ slows the transition in exposed humans. Initial $E_h(0) = 4000$, Final $E_h(T)$ = 11,421.2100.

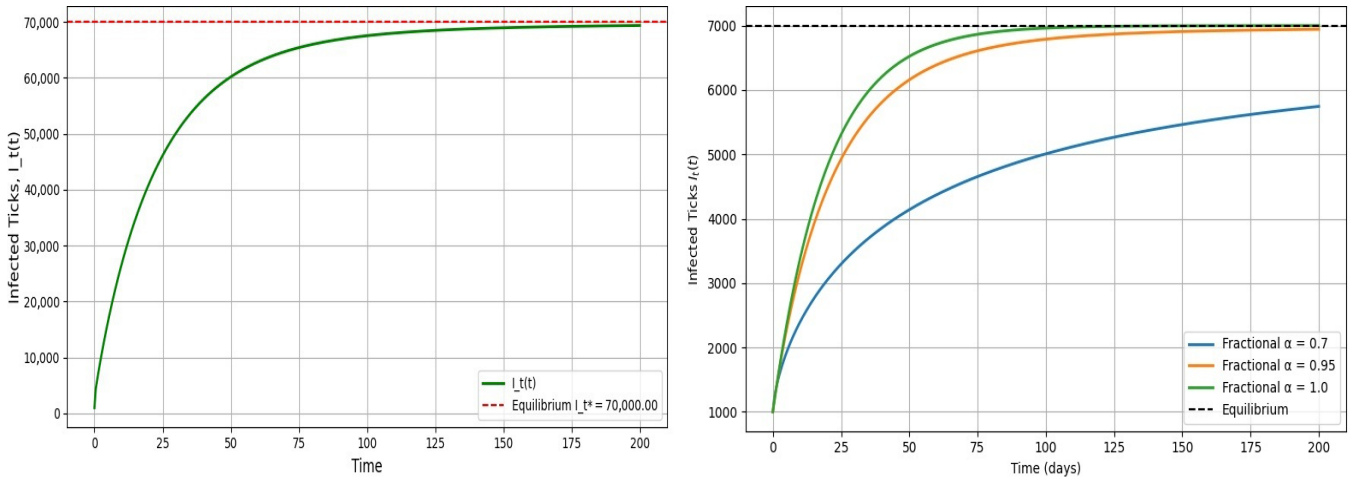


Figure 9. Infected Tick population dynamics for different α values using the RK-4 method.

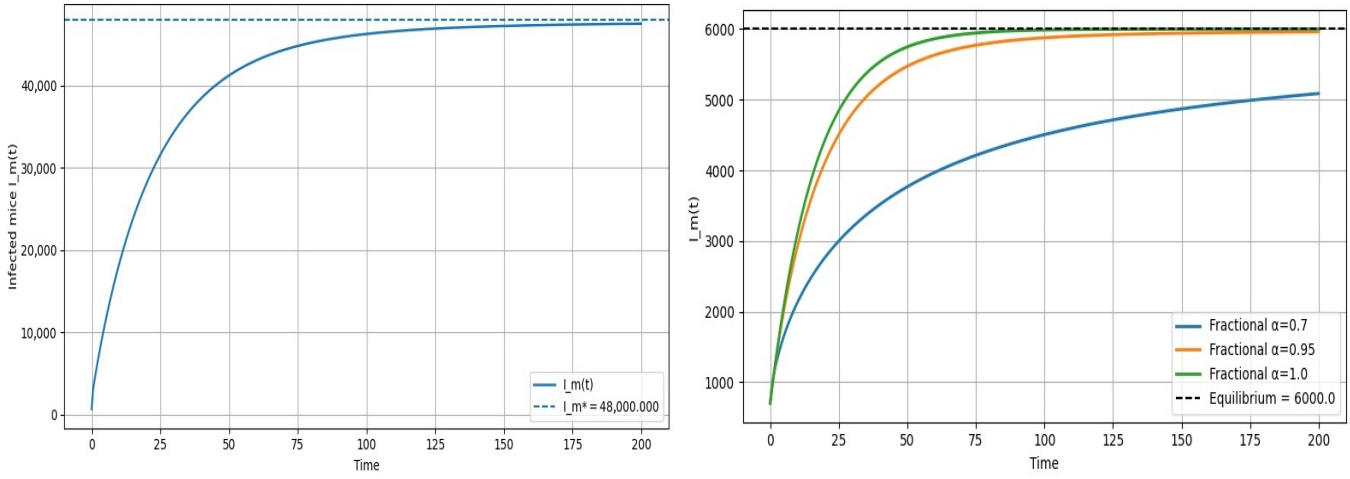


Figure 10. Infected mouse population dynamics for different α values using the RK-4 method.

Figure 12: for $\alpha = 0.95$, the model shows that smaller fractional orders produce slower convergence to equilibrium, demonstrating the role of memory in recovery dynamics. $R_h(t)$ is asymptotically stable for equilibrium $R_h^* = 6000$, Initial $R_h(0) = 1500$, Final $R_h(T) = 5957.0044$.

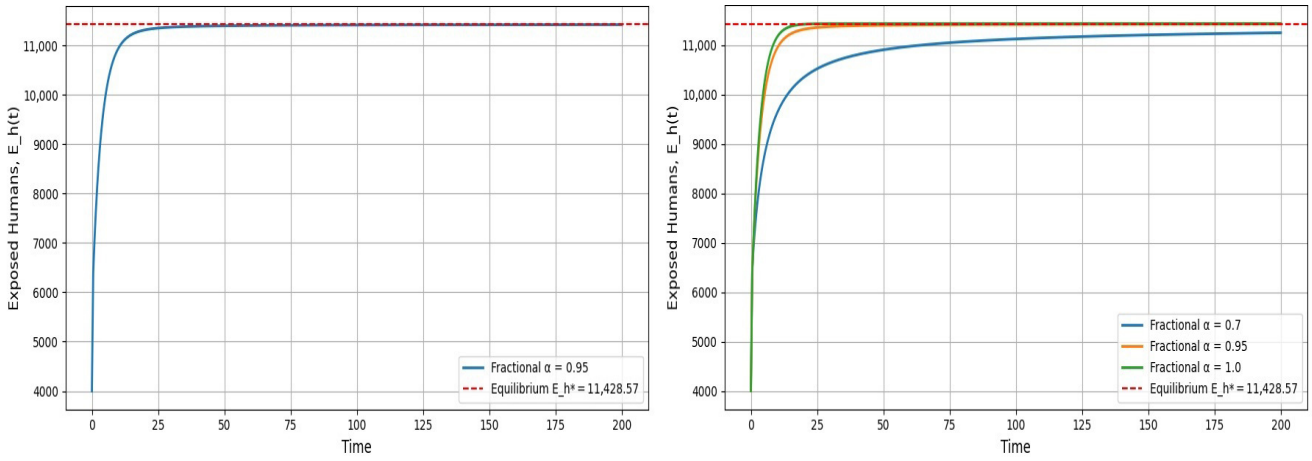


Figure 11. Exposed human population dynamics for different α values using the RK-4 method.

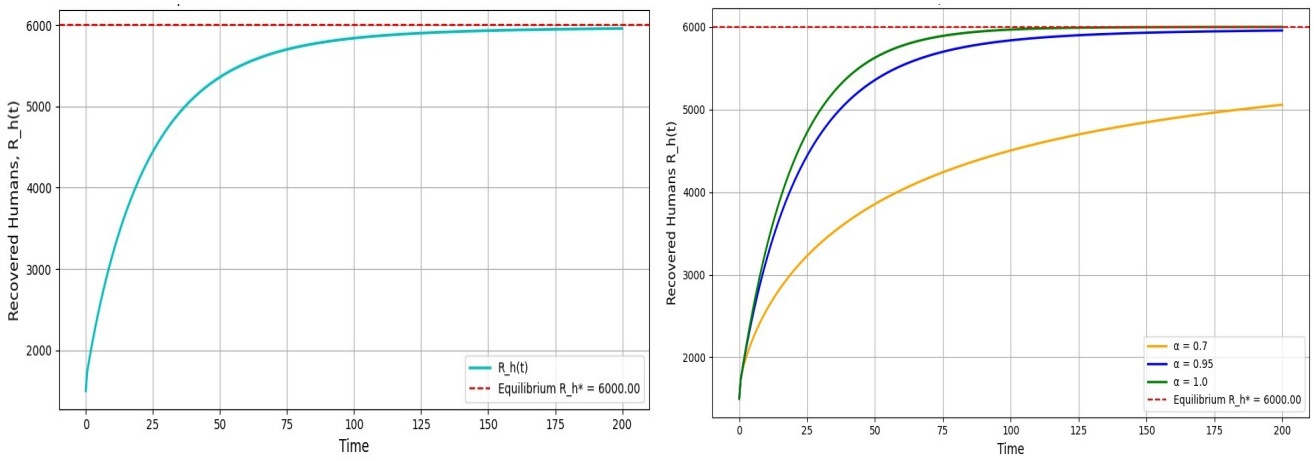


Figure 12. Recovered human population dynamics for different α values using the RK-4 method.

The results demonstrate that fractional-order models effectively capture the memory-dependent nature of the disease dynamics. In Lyme disease, such memory arises from factors like incubation delays, immune persistence and prolonged infection in reservoir hosts. It also includes the multi-stage feeding of ticks. Fractional derivatives naturally capture these time-dependent effects, unlike classical integer-order models. For example, the period during which mice are exposed and can transmit infection to ticks, and subsequently to humans, can be ranged from 3 to 30 days. Since this process is not occurring instantly, the fractional derivative is most suitable for capturing these dynamics. For all compartments, the solutions are asymptotically stable, meaning that the population tends toward steady states over time. The fractional-order α governs the speed of convergence; smaller α values produce slower transitions and smoother trajectories, consistent with real-world biological delays. When α approaches 1, the system simplifies to the classical integer-order differential equations. The fractional-order model provides valuable insights into Lyme disease dynamics. It is important to acknowledge its limitations. The model relies on parameters derived from the literature, which can vary across different geographic regions and ecological contexts by introducing a degree of uncertainty. Furthermore, the model simplifies several ecological complexities to maintain analytical tractability. For instance, we consider a single generic reservoir host and a single tick species, whereas in reality, multiple host species with varying reservoir competencies (e.g., birds, other small mammals) and multiple tick species or strains of Borrelia burgdorferi interact in complex transmission cycles may occurs. Seasonal variations in tick activity and host behavior, which are critical drivers of transmission dynamics, are also not

explicitly incorporated. Future work will benefit from integrating these complexities and employing robust parameter estimation techniques against regional incidence data to enhance the model's predictive power for specific public health applications.

8. Conclusions

This study successfully developed and analyzed a novel Caputo fractional-order mathematical model to investigate the transmission dynamics of Lyme disease, incorporating interactions between humans, ticks and reservoir host populations. The implementation of fractional-order derivatives ($\alpha = 0.95$) provided a more realistic framework than classical integer-order models by effectively capturing the memory effects and long-range dependencies inherent in ecological disease systems. The analytical and numerical results shows that the basic reproduction number $\Re_0$ serves as a critical threshold for disease persistence. The endemic equilibrium becomes locally asymptotically stable when $\Re_0 > 1$. Sensitivity analysis identified tick mortality rate μ_t and mouse mortality rate μ_m are the most influential parameters on $\Re_0$, providing clear targets for public health interventions. The optimal control analysis revealed that integrated strategies combining tick population reduction, reservoir host vaccination, public awareness campaigns, and early human treatment offer the most effective approach for disease management. The fractional-order framework captured the temporal evolution of these control measures more accurately than traditional models. The numerical results using the Runge–Kutta 4 method for Caputo fractional derivatives assures the model's capability to describe realistic disease dynamics and memory-dependent behaviors. The comparative analysis of different fractional orders ($\alpha = 0.07, 0.95$ and 1) established that $\alpha = 0.95$ provides a balance between capturing essential memory effects and maintaining biological statement, making it the recommended parameter for practical public health applications. This research contributes significantly to understanding Lyme disease dynamics and provides a robust mathematical framework for public health planning. This means our model can more accurately predict outbreak trajectories and the long-term impact of interventions, enabling the design of timely, cost-effective, and integrated control strategies that combine vector control, habitat management, and public awareness to reduce the growing burden of Lyme disease.

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