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Stability analysis of a fractional order model for Host-Parasite interactions

Chaima Moutalibi, Ahmed Aghriche* and Youssef El Hadfi
IPIM Laboratory, National School of Applied Sciences, Sultan Moulay Slimane University, Morocco.

Abstract

This study analyzes a fractional-order SI parasite-host model. We focus on examining key properties of the solutions, such as existence, uniqueness, positivity, and boundedness. Additionally, we examine the local and global stability of the equilibrium points, with particular attention to the basic reproduction number R_0. Finally, numerical simulations are carried out to illustrate the theoretical part.

Keywords. Parasite-host model, Fractional-order, Caputo derivative, Basic reproduction number, Stability.
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1. Introduction and mathematical model

Mathematical modeling has increasingly become a recognized and vital research tool for public health, particularly in epidemiology, which examines the distribution and determinants of diseases in populations, how diseases spread, their causes, and methods of control [8, 14]. These models enhance our understanding of disease transmission dynamics and help address pertinent challenges [19]. Dynamical system models in this context are commonly formulated using ordinary or partial differential equations. This approach provides a rigorous framework for analyzing the temporal and spatial dynamics of disease transmission, enabling the resolution of complex public health challenges.

The parasite-host model is a vital framework for studying the transmission dynamics of infectious diseases and the complex interactions between hosts and parasites. Deterministic models, as discussed in [4, 13], serve as a foundation for studying the dynamics of these interactions. These mathematical frameworks provide valuable insights into the evolution and consequences of parasite-host relationships, with applications in public health and evolutionary biology [21, 28]. By offering a theoretical perspective, they help uncover the biological mechanisms driving these interactions and their long-term evolutionary implications, which are often difficult to observe experimentally. Additionally, factors such as direct contact and specific behaviors influence disease transmission. Several studies have explored parasite-host and epidemiological models using ordinary differential equations to capture the complex dynamics of these interactions. One study reveals that an outbreak can originate from a small population of infected individuals [7]. Another explores the influence of host mobility and environmental heterogeneity on disease dynamics [9]. A separate investigation revises an existing model to demonstrate that parasite-induced host extinction may occur under specific conditions [16]. Finally, another analysis corrects earlier models, emphasizing the role of initial conditions in determining host extinction [17].

Fractional models generalize classical calculus to non-integer orders, offering a robust framework for analyzing systems with memory, long-term dependencies, and complex natural phenomena. This approach is extensively applied in disciplines such as physics, finance, biology, and engineering [5, 6, 18, 22], where such characteristics or anomalous behaviors are prevalent. In epidemiology, fractional calculus proves invaluable for capturing the multifaceted dynamics of disease spread, including memory effects, anomalous diffusion, and persistent infections [11, 33]. A central element in this framework is the fractional kernel function, which accounts for the influence of past events on the present

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* Corresponding author. Email: a.aghriche@usms.ma.

system [26]. Building on these theoretical foundations, recent studies have employed fractional modeling techniques to understand and simulate the dynamics of infectious diseases. For instance, the study [25] introduces a stochastic framework that incorporates random perturbations and behavioral shifts, providing a more realistic representation of epidemic dynamics. Another proposes a fractional-order model for toxoplasmosis, capturing complex host interactions and memory effects inherent in disease transmission [1]. Moreover, the study in [31] presents advanced numerical techniques based on Bessel polynomials to solve a fractional HIV-1 model that accounts for the impact of antiviral treatment. Also, COVID-19 transmission models based on fractional derivatives, incorporating both the Caputo and fractal-fractional approaches, have been proposed [23, 24]. These studies rigorously examine the models' fundamental properties, integrate the effects of public health interventions, and substantiate their results through numerical simulations, highlighting their potential applicability on a global scale. In parasite-host models, the kernel captures the effects of prior infections, interactions, or environmental factors on disease transmission, effectively addressing non-local and historical influences. By incorporating fractional-order terms and kernel functions, these models provide a more precise representation of disease progression, improving predictions and supporting the development of effective control strategies. We focus on the following SI model, adapted from [32], which classifies parameters as either susceptible or infected:

$$\begin{cases} \dfrac{dS}{dt} = \eta(S+\zeta I)[1-\gamma(S+I)] - \dfrac{\beta SI}{S+I}, \\ \dfrac{dI}{dt} = \dfrac{\beta SI}{S+I} - \mu I. \end{cases} \tag{1.1}$$

We consider $N(t) = S(t) + I(t)$ with $S(0) = S_0 \geq 0$ and $I(0) = I_0 \geq 0$, where the functions $S := S(t)$ and $I := I(t)$ denote the density of susceptible and infected hosts at time t respectively. All parameters are non-negative, such as $1/\gamma$ is the carrying capacity of $\gamma \neq 0$ and $\dfrac{\beta SI}{S+I}$ is the frequency-dependent transmission. See [32] for more details.

TABLE 1. Parameter descriptions.

Parameter	Description
η	Maximum birth rate of the hosts
γ	Reduction in the per capita birth rate
μ	Natural death rate of the host population
ζ	Reduction reproduction ability of infected hosts
β	Disease transmission rate

In the case of susceptible-free, i.e., $S = 0$, if infected hosts remain at low prevalence, i.e., $\gamma I < 1$, the net reproduction rate of newborns of susceptible hosts will be $\eta\zeta I(1-\gamma I) > 0$, and susceptible hosts will grow; if infected hosts remain at high prevalence, i.e., $\gamma I \geq 1$, the net reproduction rate of newborns of susceptible hosts will be zero [9].

This study introduces a fractional-order SI parasite-host model, providing a more comprehensive understanding of the dynamics of parasite-host infections, particularly in terms of disease transmission and system stability. The model employs fractional order derivatives, specifically the Liouville-Caputo fractional derivative, and does not account for diffusion. The fractional-order formulation of (1.1) is outlined as follows:

$$\begin{cases} {}^{LC}D_t^{\alpha} S(t) = \eta(S+\zeta I)[1-\gamma(S+I)] - \dfrac{\beta SI}{S+I}, \\ {}^{LC}D_t^{\alpha} I(t) = \dfrac{\beta SI}{S+I} - \mu I. \end{cases} \tag{1.2}$$

The following sections detail the paper's contents: Section 2 includes preliminaries and definitions. Section 3 introduces the fundamental properties of the epidemic model. Section 4 outlines the equilibrium points. Section

5 provides the stability analysis of the equilibrium points and a bifurcation analysis. Section 6 presents numerical simulations, and section 7 concludes the study.

2. Preliminaries

This section provides brief definitions of essential concepts in the theory of fractional-order ordinary differential equations. For more details, one can refer to [11].

Definition 2.1. ([11]) Riemann–Liouville fractional integral of order $\beta > 0$ of a function $g(t) \in C[a, b]$ is expressed as

$$I_t^{\beta} g(t) = \frac{1}{\Gamma(\beta)} \int_a^t (t-x)^{\beta-1} g(x) dx.$$

Definition 2.2. ([27]) The Liouville-Caputo fractional derivative of order $\beta > 0$ of a function $g \in C^n[a, b]$ such that $n \in \mathbb{N}$ and $n > 0$, is defined as

$${}^{\mathrm{LC}}D_a^{\beta} g(t) := \begin{cases} g^{(n)}(t), & \beta = n, \\ \dfrac{1}{\Gamma(n-\beta)} \displaystyle\int_a^t (t-x)^{n-\beta-1} g^{(n)}(x)\, dx, & n-1 < \beta < n. \end{cases}$$

Proposition 2.3. *([11]) The Liouville-Caputo fractional derivation operator* $\left({}^{LC}D_t^{\beta}\right)$ *has the following properties:*

(1) $\left({}^{LC}D_t^{\beta}\right)$ *is a linear operator.*
(2) $\left({}^{LC}D_t^{\beta}\right)\left(I_t^{\beta} g\right)(t) = g(t)$.
(3) $I_t^{\beta}\left({}^{LC}D_t^{\beta} g(t)\right) = g(t) + \sum_{j=0}^{n-1} c_j (t-a)^j$, $(c)_{j=0,\ldots,n-1} \in \mathbb{R}$.

Definition 2.4. ([26]) For $\beta, \alpha \in \mathbb{C}$ with $\Re(\beta) > 0$ and $\Re(\alpha) > 0$, the two-parameter Mittag-Leffler function is defined as follows:

$$E_{\beta,\alpha}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\beta k + \alpha)}, \quad z \in \mathbb{C}.$$

Theorem 2.5. *([26]) Let $g(t)$ be a function that has a Liouville–Caputo fractional derivative of order β, where $\beta \in (n-1, n)$, $\forall n \in \mathbb{N}$ and $n > 0$. The Laplace transform of this fractional derivative is given by*

$$\mathcal{L}\left({}^{LC}D_t^{\beta} g(t)\right)(s) = s^{\beta} G(s) - \sum_{k=0}^{n-1} s^{\beta-k-1} g^{(k)}(0),$$

where $G(s) = \mathcal{L}(g(t))(s)$ represents the Laplace transform of $g(t)$.

Theorem 2.6. *([26]) The Laplace transform of the function defined by the two-parameter Mittag-Leffler function can be written as*

$$\mathcal{L}\left(t^{\alpha-1} E_{\beta,\alpha}\left(\pm\gamma t^{\beta}\right)\right)(s) = \frac{s^{\beta-\alpha}}{s^{\beta} \mp \gamma},$$

where $\alpha, \beta > 0$, $\gamma \in \mathbb{R}$, and s is the Laplace variable.

The global stability of the equilibrium points is proven by introducing a fundamental lemma that establishes sufficient conditions for their stability.

Lemma 2.7. *([15]) Let A be a bounded closed set. For every solution of ${}^{LC}D^{\beta} z(t) = g(z(t))$ with $z \in \mathbb{R}$, suppose there exists a function $V(z) : A \to \mathbb{R}$ with continuous first partial derivatives that satisfies the condition*

$${}^{LC}D^{\beta} V(z) \leq 0.$$

Let $F = \left\{z, {}^{LC}D^{\beta} V(z) = 0\right\}$ and B denote the largest invariant set of F. Then, every solution $z(t)$ originating in A converges to B as $t \to \infty$. In particular, if $B = \{0\}$, then $z \to 0$ as $t \to \infty$.

3. Basic proprieties of the system

In this section, we explore the various mathematical properties of the model. These include examining the existence, uniqueness, positivity, and boundedness of the solution.

3.1. **Existence and uniqueness of solution.** Let g be a C^∞ function and $z(t) \in \mathbb{R}^2_+$ be the solution to the fractional system given by

$$\begin{cases} {}^{LC}D_t^\alpha z(t) = g(t, z(t)), \\ z(t_0) = z_0, \qquad\qquad \alpha \in (0, 1], \end{cases} \tag{3.1}$$

where

$$\mathbb{R}^2_+ = \left\{ z \in \mathbb{R}^2 \mid z \geq 0 \right\} \quad \text{and} \quad z(t) = (S(t), I(t))^\top.$$

To analyze the existence and uniqueness of the system solution (3.1), we consider $D \subset \mathbb{R}^2_+$ to be a compact positively invariant region for the system defined by

$$D = \left\{ z = (S, I)^\top \in \mathbb{R}^2_+ \mid 0 \leq S, I \leq M \right\}.$$

Proposition 3.1. *If $g(t, z)$ is continuous and satisfies the Lipschitz condition in $z \in D$, then the fractional system in (3.1) has a unique solution for any initial condition $z_0 = (S(0), I(0))^\top$.*

Proof. Let g be defined as

$$g(t, z(t)) = \begin{pmatrix} g_1(t, z(t)) \\ g_2(t, z(t)) \end{pmatrix} = \begin{pmatrix} \eta(S + \zeta I)[1 - \gamma(S + I)] - \dfrac{\beta SI}{S + I} \\ \dfrac{\beta SI}{S + I} - \mu I \end{pmatrix}.$$

Let $z, \bar{z} \in D$ such as $z = (S, I)^\top$ and $\bar{z} = (\bar{S}, \bar{I})^\top$. Then, we have

$$\begin{aligned} \|g(t, z(t)) - g(t, \bar{z}(t))\| &= \|g_1(t, z(t)) - g_1(t, \bar{z}(t))\| + \|g_2(t, z(t)) - g_2(t, \bar{z}(t))\|, \\ &= \left| \eta(S + \zeta I)[1 - \gamma(S + I)] - \frac{\beta SI}{S + I} - \eta(\bar{S} + \zeta \bar{I})[1 - \gamma(\bar{S} + \bar{I})] + \frac{\beta \bar{S}\bar{I}}{\bar{S} + \bar{I}} \right| \\ &+ \left| \frac{\beta SI}{S + I} - \mu I - \frac{\beta S\bar{I}}{\bar{S} + \bar{I}} + \mu \bar{I} \right| \\ &= \left| \eta\big[((S - \bar{S}) + \zeta(I - \bar{I}))\big(1 - \gamma(S + I)\big) - \gamma(\bar{S} + \zeta \bar{I})((S - \bar{S}) + (I - \bar{I}))\big] - \beta\Big(\frac{SI}{S + I} - \frac{\bar{S}\bar{I}}{\bar{S} + \bar{I}}\Big) \right| \\ &+ \left| \beta\Big(\frac{SI}{S + I} - \frac{\bar{S}\bar{I}}{\bar{S} + \bar{I}}\Big) - \mu(I - \bar{I}) \right|, \end{aligned}$$

where

$$\frac{SI}{S + I} - \frac{\bar{S}\bar{I}}{\bar{S} + \bar{I}} = \frac{S\bar{S}(I - \bar{I}) + \bar{I}I(S - \bar{S})}{(S + I)(\bar{S} + \bar{I})}.$$

We obtain

$$\begin{aligned} \|g(t, z(t)) - g(t, \bar{z}(t))\| \quad &\leq \eta \left| ((S - \bar{S}) + \zeta(I - \bar{I}))\big(1 - \gamma(S + I)\big) - \gamma(\bar{S} + \zeta \bar{I})((S - \bar{S}) + (I - \bar{I})) \right| \\ &+ 2\beta \left| \frac{S\bar{S}(I - \bar{I}) + \bar{I}I(S - \bar{S})}{(S + I)(\bar{S} + \bar{I})} \right| + \mu \left| (I - \bar{I}) \right|. \end{aligned}$$

Since $z, \bar{z} \in D$ and all parameters are strictly positive, there exist constants $C_1, C_2 > 0$ such that the nonlinear terms are uniformly bounded on D. Then

$$\begin{aligned} |1 - \gamma(S + I)| &\leq 1 + 2\gamma M = C_1, \\ |\bar{S} + \zeta \bar{I}| &\leq (1 + \zeta) M = C_2. \end{aligned}$$

As all variables are bounded on D, there exists $\delta > 0$ such that $(S+I)(\bar{S}+\bar{I}) \geq \delta$, we have

$$\left| \frac{S\bar{S}(I-\bar{I}) + \bar{I}I(S-\bar{S})}{(S+I)(\bar{S}+\bar{I})} \right| \leq \frac{M^2}{\delta} \left| (I-\bar{I}) + (S-\bar{S}) \right|.$$

Then

$$\begin{aligned} \|g(t,z(t)) - g(t,\bar{z}(t))\| &\leq \eta \left(|S-\bar{S}| + \zeta |I-\bar{I}| \right) C_1 + \eta C_2 \gamma \left(|S-\bar{S}| + |I-\bar{I}| \right) + \mu \left| (I-\bar{I}) \right| + 2\beta \frac{M^2}{\delta} \left| (I-\bar{I}) + (S-\bar{S}) \right| \\ &\leq L_1 |S-\bar{S}| + L_2 |I-\bar{I}|, \end{aligned}$$

with

$$L_1 = \eta C_1 + \eta C_2 \gamma + 2\beta \frac{M^2}{\delta}, \quad \text{and} \quad L_2 = \eta \zeta C_1 + \eta C_2 \gamma + 2\beta \frac{M^2}{\delta} + \mu.$$

Then, we have

$$\|g(t,z(t)) - g(t,\bar{z}(t))\| \leq L \, \|z - \bar{z}\|,$$

where $L = \max\{L_1, L_2\}$. Therefore, $g(t,z(t))$ is a Lipschitz function, ensuring the existence and uniqueness of the solution to the model in (3.1).

□

3.2. **Positivity and boundedness.** Positivity is crucial for ensuring biologically meaningful model solutions, while boundedness guarantees that the solutions remain finite.

Proposition 3.2. *The solutions of (1.2) remain non-negative and bounded for all positive values of t, given any non-negative initial conditions.*

Proof. - Positivity:
From model (1.2), we have

$${}^{LC}D_t^{\alpha} S(t) \geq -\beta S(t).$$

We apply the Laplace transform method, assuming the initial condition $S(0) \geq 0$. We have

$$S(t) \geq E_{\alpha}(-\beta t^{\alpha}) S(0),$$

where $E_{\alpha,\eta}$ is the Mittag-Leffler function. Since $S(0) \geq 0$, one obtains $S(t) \geq 0$. Thus, $S(t)$ stays non-negative for all $t > 0$.
Likewise, we get

$$I(t) \geq E_{\alpha}(-\mu t^{\alpha}) I(0) \geq 0.$$

Then $I(t)$ is non-negative for all $t > 0$.
-Boundedness:
To demonstrate that the system in (1.2) is bounded, the growth of the population is given by the following expression:

$${}^{LC}D_t^{\alpha} N(t) = {}^{LC}D_t^{\alpha} S(t) + {}^{LC}D_t^{\alpha} I(t).$$

We have

$${}^{LC}D_t^{\alpha} N(t) \leq -\eta\gamma N(t).$$

Then

$$N(t) \leq E_{\alpha}(-\eta\gamma t^{\alpha}) N(0).$$

Due to $0 \leq E_{\alpha}(-\eta\gamma t^{\alpha}) \leq 1$ then one has $N(t) \leq N(0)$. Consequently, all solutions of (1.2) with non-negative initial conditions remain bounded, with the realizable domain for this system given by

$$\Omega = \{(S,I) \in \mathbb{R}^2_+ \;\mid\; S + I \leq N(0)\}.$$

□

4. Equilibrium Points

4.1. **Disease-free equilibrium.** We set the expressions ${}^{LC}D_t^{\alpha}S(t) = 0$ and ${}^{LC}D_t^{\alpha}I(t) = 0$. By assuming $I = 0$, we can determine the equilibrium E_0 of the system (1.2) as

$$E_0 = (S_0, I_0) = \left(\frac{1}{\gamma}, 0\right).$$

4.2. **The basic reproductive number R_0.** The basic reproduction number R_0 is a measure of the average number of secondary infections generated by a single infected individual. If R_0 is less than one, it indicates a decline in disease prevalence, while a value of one indicates stability. Conversely, when R_0 exceeds 1, it signals the potential for disease spread that could lead to an outbreak. A value below one indicates the presence of the disease within the community, with the potential for effective management [29]. If we consider $z(t) = (I(t), S(t))$, then we represent the system (1.2) as

$$\frac{dz}{dt} = \mathcal{F}(z) - \mathcal{V}(z),$$

with

$$F = \left[\frac{\partial \mathcal{F}}{\partial z}(z_0)\right] \quad \text{and} \quad V = \left[\frac{\partial \mathcal{V}}{\partial z}(z_0)\right].$$

Then R_0 is described as the spectral radius of the next-generation matrix $K = -FV^{-1}$, such as $R_0 = \rho(-FV^{-1})$. In this case, we have $E_0 = (S_0, I_0)$ with $S_0 = \frac{1}{\gamma}$. We can easily obtain

$$F = \beta \quad \text{and} \quad V = -\mu, \quad \text{then} \quad R_0 = \frac{\beta}{\mu}.$$

4.3. **Endemic equilibrium.** We can resolve the equations ${}^{LC}D_t^{\alpha}S(t) = 0$ and ${}^{LC}D_t^{\alpha}I(t) = 0$ simultaneously, to determine the equilibrium E^* of Equation (1.2). This yields the following results:

$$S^* = \frac{\mu\theta}{\gamma\beta\eta(\zeta(R_0 - 1) + 1)} \quad \text{and} \quad I^* = (R_0 - 1)S^*.$$

We can define

$$\eta_e := \frac{\mu(R_0 - 1)}{\zeta(R_0 - 1) + 1}.$$

If $R_0 > 1$ and $\eta > \eta_e$, then we have $\theta = \eta[\zeta(R_0 - 1) + 1] - \mu(R_0 - 1) > 0$. Thus, $E^* = (S^*, I^*)$ represents the unique endemic equilibrium of system (1.2).

In addition, the proposal ensures the uniqueness of the positive equilibrium solution by considering the value of R_0.

Proposition 4.1.

- *If $R_0 \leq 1$, (1.2) has only one equilibrium E_0.*
- *Besides the disease-free equilibrium E_0, system (1.2) admits a unique equilibrium E^* when $R_0 > 1$ and $\eta > \eta_e$.*

5. Equilibrium stability analysis

This section will analyze the local and global stability of the fractional model at the disease-free and endemic equilibrium points.

5.1. **Disease-Free equilibrium stability.** Here, we present the stability analyses for the disease-free equilibrium point E_0.

Lemma 5.1. *([20]) Let z^* be an equilibrium of (3.1). Then, for all eigenvalues λ of the Jacobian matrix $J(z^*)$, z^* is locally asymptotically stable if*

$$|\arg(\lambda)| > \frac{\alpha\pi}{2}.$$

Proposition 5.2. *The equilibrium E_0 is locally asymptotically stable when $R_0 < 1$.*

Proof. Consider the Jacobian matrix obtained from (1.2) at E_0, we have

$$J(E_0) = \begin{pmatrix} -\eta & -(\eta+\beta) \\ 0 & \mu(R_0-1) \end{pmatrix}.$$

The eigenvalues of $J(E_0)$ are $\lambda_1 = -\eta$ and $\lambda_2 = \mu(R_0-1)$. Thus, all eigenvalues of $J(E_0)$ are negative if and only if $R_0 < 1$, thus $|\arg(\lambda_i)| = \pi$, $i = 1, 2$. Therefore, E_0 is locally asymptotically stable.

□

Lemma 5.3. *([2]) Let $z : [0, +\infty) \to \mathbb{R}_+$ be a continuous and differentiable function. Then*

$$\frac{1}{2}{}_{t_0}^{LC}D_t^{\beta} z^2(t) \leq z(t)\,{}_{t_0}^{LC}D_t^{\beta} z(t), \quad \forall t \geq t_0, \quad \beta \in (0,1).$$

Proposition 5.4. *The equilibrium E_0 is globally asymptotically stable when $R_0 \leq 1$.*

Proof. We present

$$V : \{(S, I) \in \mathbb{R}_+^2 : S > 0, I \geq 0\} \to \mathbb{R},$$

by

$$V(S, I) = \frac{1}{2} I^2.$$

The time derivative of V is expressed as

$$\begin{aligned} {}^{LC}D_t^{\alpha} V(S, I) = \frac{1}{2}\,{}^{LC}D_t^{\alpha} I^2 &\leq \frac{I^2}{S+I}(\beta S - \mu(S+I)) \\ &\leq -\mu \frac{I^2}{S+I}((1-R_0)+I). \end{aligned}$$

We have ${}^{LC}D_t^{\alpha} V(S, I) \leq 0$ for $R_0 \leq 1$ with ${}^{LC}D_t^{\alpha} V(S, I) = 0$ if and only if $I = I_0 = 0$, then $\{(S, I) \in \mathbb{R}_+^2 : {}^{LC}D_t^{\alpha} V(S, I) = 0\} = \{E_0\}$. Then, by Lemma 2.7, E_0 is globally asymptotically stable, whenever $R_0 \leq 1$.

□

5.2. **Endemic equilibrium stability.** Now let's demonstrate the stability of the endemic equilibrium point E^*.

Proposition 5.5. *The equilibrium E^* is locally asymptotically stable when $R_0 > 1$.*

Proof. Consider the Jacobian matrix obtained from (1.2) at E^*, we have

$$J(E^*) = \begin{pmatrix} a_{11} & a_{12} \\ a_{21} & a_{22} \end{pmatrix},$$

where

$$a_{11} = \frac{1}{\beta(\zeta(\beta-\mu)+\mu)}\left[-(\zeta(\beta-\mu)+\mu)\theta - (\beta-\mu)\left(\zeta(\mu-\beta)^2 - \mu^2\right)\right],$$

$$a_{12} = \frac{1}{\beta(\zeta(\beta-\mu)+\mu)}\left[-(\zeta(\beta-\mu)+\mu)\theta + \mu\left(\zeta(\mu-\beta)^2 - \mu^2\right)\right],$$

$$a_{21} = \frac{(\beta-\mu)^2}{\beta}, \quad \text{and} \quad a_{22} = -\frac{\mu(\beta-\mu)}{\beta}.$$

The characteristic equation of $J(E^*)$ is

$$X^2 - \mathrm{Tr(J(E^*))}X + \det(J(E^*)) = 0,$$

with

$$\mathrm{Tr}(J(E^*)) = \frac{1}{\beta(\zeta\mu(R_0-1)+\mu)}\left[-(\zeta\mu(R_0-1)+\mu)\theta - \beta\zeta(\beta-\mu)^2\right],$$

$$\det(J(E^*)) = \frac{(R_0-1)\mu\theta}{\beta}.$$

Thus, if $R_0 > 1$ then $\det(J(E^*)) > 0$ and $Tr(J(E^*)) < 0$. By the Routh-Hurwitz criterion [3] for a second-order polynomial $X^2 + b_1X + b_0 = 0$, all roots have negative real parts if and only if $b_1 > 0$ and $b_0 > 0$. In this case,

$$b_1 = -\mathrm{Tr}(J(E^*)) > 0, \quad \text{and} \quad b_0 = \det(J(E^*)) > 0.$$

Therefore, both eigenvalues of $J(E^*)$ have negative real parts, implying that the endemic equilibrium E^* is locally asymptotically stable whenever $R_0 > 1$. □

Lemma 5.6. *([30]) Let $z : [0, +\infty) \to \mathbb{R}_+$ be a continuous and differentiable function. Then*

$$ {}^{LC}_{t_0}D^{\beta}_t\left[z(t) - z^* - z^*\ln\frac{z(t)}{z^*}\right] \le \left(1 - \frac{z^*}{z(t)}\right) {}^{LC}_{t_0}D^{\beta}_t z(t),\ z^* \in \mathbb{R}^+,\ \beta \in (0,1),\ \forall t \ge t_0.$$

Proposition 5.7. *If $R_0 > 1$, then E^* is globally asymptotically stable.*

Proof. We assume Λ the per-capita birth rate define by $\Lambda = \eta(S + \zeta I)[1 - \gamma(S + I)]$.
We define

$$V : \{(S, I) \in \mathbb{R}^2_+ : S > 0, I > 0\} \to \mathbb{R},$$

by

$$V(S,I) = \left[(S+I) - (S^*+I^*) - (S^*+I^*)\ln\frac{(S+I)}{(S^*+I^*)}\right] + \frac{\mu(S^*+I^*)}{\beta I^*}\left(I - I^* - I^*\ln\frac{I}{I^*}\right).$$

We take

$$\Lambda = \mu I^* \quad \text{and} \quad \mu = \frac{\beta S^*}{S^*+I^*}.$$

Using the result of the Lemma 5.6, we obtain

$$\begin{aligned}
{}^{LC}D^{\alpha}_t V(S,I) &\le \left(1 - \frac{S^*+I^*}{S+I}\right) {}^{LC}D^{\alpha}_t(S+I) + \frac{\mu(S^*+I^*)}{\beta I^*}\left(1 - \frac{I^*}{I}\right) {}^{LC}D^{\alpha}_t(I) \\
&\le \left(1 - \frac{S^*+I^*}{S+I}\right)(\Lambda - \mu I) + \frac{\mu(S^*+I^*)}{\beta I^*}\left(1 - \frac{I^*}{I}\right)\left(\frac{\beta SI}{S+I} - \mu I\right) \\
&\le \left(\frac{(S-S^*)+(I-I^*)}{S+I}\right)(-\mu(I-I^*)) + \frac{\mu(S^*+I^*)}{I^*}(I-I^*)\left(\frac{S}{S+I} - \frac{S^*}{S^*+I^*}\right).
\end{aligned}$$

Notice that

$$\frac{S}{S+I} - \frac{S^*}{S^*+I^*} = \frac{I^*(S-S^*) - S^*(I-I^*)}{(S+I)(S^*+I^*)}.$$

Thus,

$$\begin{aligned}
{}^{LC}D^{\alpha}_t V(S,I) &\le \left(\frac{(S-S^*)+(I-I^*)}{S+I}\right)(-\mu(I-I^*)) + \frac{\mu(I-I^*)}{I^*}\left(\frac{I^*(S-S^*) - S^*(I-I^*)}{(S+I)}\right) \\
&\le -\mu\frac{(I-I^*)^2}{S+I}\left(1 + \frac{S^*}{I^*}\right).
\end{aligned}$$

Then ${}^{LC}D^{\alpha}_t V(S,I)$ is negative definite. In addition ${}^{LC}D^{\alpha}_t V(S,I) = 0$ if and only if $S = S^*$ and $I = I^*$ then $\left\{(S,I) \in \mathbb{R}^2_+ : {}^{LC}D^{\alpha}_t V(S,I) = 0\right\} = \{E^*\}$. Then, by Lemma 2.7, if $R_0 > 1$ then E^* is globally asymptotically stable. □

5.3. **Bifurcation analysis.** We assume that $z = (z_1, z_2)^\top$ and $G = (G_1, G_2)^\top$, then (1.2) can be written in the form ${}^{LC}D_t^\alpha z = G(z)$ as

$$\begin{cases} {}^{LC}D_t^\alpha z_1 = \eta(z_1 + \zeta z_2)\left[1 - \gamma(z_1 + z_2)\right] - \dfrac{\beta z_1 z_2}{z_1 + z_2} := G_1, \\ {}^{LC}D_t^\alpha z_2 = \dfrac{\beta z_1 z_2}{z_1 + z_2} - \mu z_2 := G_2. \end{cases} \tag{5.1}$$

We apply the result of Castillo-Chavez and Song [10] to analyze the nature of the bifurcation occurring at the critical threshold $R_0 = 1$, where the bifurcation parameter β^* satisfies $\beta^* = \beta = \mu$.

The Jacobian matrix of system (5.1) at the equilibrium point E_0 and bifurcation parameter β^* can be expressed as

$$J(E_0, \beta^*) = \begin{pmatrix} -\eta & -(\eta + \mu) \\ 0 & 0 \end{pmatrix},$$

with the eigenvalues are $\lambda_1 = -\eta$ and $\lambda_2 = 0$. Therefore, the Jacobian matrix $J(E_0, \beta^*)$ has a simple zero eigenvalue, and the remaining eigenvalue has a negative real part. The corresponding left and right eigenvectors associated with this zero eigenvalue at the critical equilibrium are respectively given by $v = (v_1, v_2) = (0, 1)$ and $w = (w_1, w_2)^\top = \left(-\frac{\eta+\mu}{\eta}, 1\right)^\top$.

We now compute the bifurcation coefficients a and b, as rigorously detailed in Theorem 4.1 of [10], which are defined by

$$a = \sum_{k,i,j=1}^{2} v_k w_i w_j \frac{\partial^2 G_k}{\partial x_i \partial x_j}(E_0, \beta^*), \quad \text{and} \quad b = \sum_{k,i=1}^{2} v_k w_i \frac{\partial^2 G_k}{\partial x_i \partial \beta}(E_0, \beta^*).$$

Since the left eigenvector is $v = (0, 1)$, only the second component G_2 of the vector field contributes to the sums. Therefore, the coefficients reduce to

$$a = \sum_{i,j=1}^{2} w_i w_j \frac{\partial^2 G_2}{\partial x_i \partial x_j}(E_0,\, \beta^*) = w_1^2 \frac{\partial^2 G_2}{\partial S^2} + 2 w_1 w_2 \frac{\partial^2 G_2}{\partial S \partial I} + w_2^2 \frac{\partial^2 G_2}{\partial I^2},$$

and

$$b = \sum_{i=1}^{2} w_i \frac{\partial^2 G_2}{\partial x_i \partial \beta}(E_0,\, \beta^*) = w_1 \frac{\partial^2 G_2}{\partial S \partial \beta} + w_2 \frac{\partial^2 G_2}{\partial I \partial \beta}.$$

The only non-zero second-order partial derivatives of the functions G are

$$\frac{\partial^2 G_2}{\partial I^2}(E_0,\, \beta^*) = -2\mu\gamma, \quad \text{and} \quad \frac{\partial^2 G_2}{\partial I \partial \beta}(E_0,\, \beta^*) = 1.$$

The bifurcation coefficients are given by

$$a = -2\mu\gamma < 0, \quad \text{and} \quad b = 1 > 0.$$

The opposite signs of the bifurcation coefficients $a < 0$ and $b > 0$, as described by the Castillo-Chavez and Song theory, indicate that the system experiences a forward (transcritical) bifurcation at the critical threshold $R_0 = 1$. This means that when $R_0 < 1$, the disease-free equilibrium E_0 is unique and stable. However, when $R_0 > 1$, this equilibrium loses stability, and a unique, stable endemic equilibrium E^* emerges. The following figure illustrates the bifurcation behavior of the system as described.

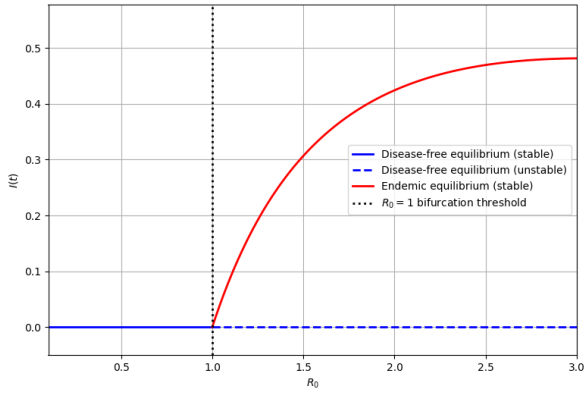

FIGURE 1. Forward bifurcation at $R_0 = 1$.

6. Numerical simulations

In this part, we present a numerical simulation to support the theoretical results derived in the previous sections. We used Python software with the parameters provided in Table 2.

We examine the Liouville-Caputo fractional differential equation of the form

$$\begin{cases} {}^{LC}D_t^{\alpha} z(t) = g(t, z(t)), \\ z(t_0) = z_0, \qquad \alpha \in (0, 1], \end{cases}$$

where g is a Lipschitz-continuous function, such as

$$g(t, z(t)) = \begin{pmatrix} \eta(S + \zeta I)[1 - \gamma(S + I)] - \dfrac{\beta SI}{S + I} \\ \dfrac{\beta SI}{S + I} - \mu I \end{pmatrix}, \quad z(t) = (S(t), I(t))^{\top}.$$

Taking the Riemann-Liouville integral on each side, we have

$$z(t) = z_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \lambda)^{\alpha} g(\lambda, z(\lambda)) d\lambda.$$

To construct an iterative procedure, a uniform grid $[0, T]$ with step size $h = \frac{T}{l}$ is considered, where $t_0 = 0 < t_1 < \cdots < t_l = T$, $l \in \mathbb{N}$, $l > 0$, and $t_j = t_0 + jh$. Let z_m be the approximate solution of $z(t_m)$ at $t = t_m$. Employing the fractional Euler method [12], well-regarded for its simplicity and compatibility with the Liouville–Caputo fractional derivative, we use the following numerical scheme:

$$z_{m+1} = z_0 + \frac{h^{\alpha}}{\Gamma(\alpha + 1)} \sum_{i=0}^{m} \left[(m + 1 - i)^{\alpha} - (m - i)^{\alpha} \right] g\left(t_i, z\left(t_i\right)\right). \tag{6.1}$$

To ensure numerical stability and accuracy, the interval $[0, T]$ is discretized into $l = 400$ time steps with a final time $T = 400$, resulting in a time step size $h = 1$. This choice effectively balances computational cost while accurately capturing the system's dynamic behavior. Moreover, it is well established that the fractional Euler method converges under standard regularity assumptions on the function $g(t, z(t))$, particularly when it satisfies a Lipschitz condition [12]. As the assumptions in our model are satisfied, the method is deemed stable and convergent.

Thus, by using the scheme derived in (6.1), the iterative formulae for the proposed fractional case system (1.2) are obtained as

$$S_{m+1} = S_0 + \frac{h^\alpha}{\Gamma(\alpha+1)} \sum_{i=0}^{m} [(m+1-i)^\alpha - (m-i)^\alpha] \left(\eta(S_i + \zeta I_i)[1-\gamma(S_i+I_i)] - \frac{\beta S_i I_i}{S_i + I_i}\right),$$

$$I_{m+1} = I_0 + \frac{h^\alpha}{\Gamma(\alpha+1)} \sum_{i=0}^{m} [(m+1-i)^\alpha - (m-i)^\alpha] \left(\frac{\beta S_i I_i}{S_i + I_i} - \mu I_i\right).$$

In the following, we utilize the parameters listed in Table 1 along with various values of α to illustrate the accuracy of the theoretical findings.

TABLE 2. Parameters estimation.

Parameters	Values	References
η	0.6	[32]
γ	1	[32]
μ	0.1-1	Assumed
ζ	0.1	[32]
β	0.1-1	Assumed

The analysis indicates that the disease will be effectively eliminated from the population. This conclusion is derived using the parameter values outlined in Table 2. Specifically, with $\beta = 0.2$ and $\mu = 1$, we have $R_0 = 0.2 \leq 1$. For values of $\alpha = 0.6, 0.7, 0.8, 0.9, 1$, numerical simulations show that the susceptible population asymptotically approaches $S_0 = 1 = 1/\gamma$ (Figure 2(a)), while the infected population tends to $I_0 = 0$ (Figure 2(b)), confirming the stability of the equilibrium E_0. Biologically, this result suggests that the disease cannot persist in the population under the given conditions, emphasizing the crucial impact of model parameters on the long-term dynamics of disease transmission.

However, for the choice of parameter values $\beta = 1$ and $\mu = 0.9$, we obtain $R_0 = 1.1112 > 1$, indicating the possibility of an endemic equilibrium. This equilibrium is represented by $E = (S, I) = (0.7888, 0.0877)$. Numerical simulations for $\alpha = 0.6, 0.7, 0.8, 0.9, 1$ show that the susceptible population asymptotically approaches $S = 0.7888$ (Figure 3(a)), while the infected population converges to $I^* = 0.0877$ (Figure 3(b)), confirming the stability of the equilibrium E^*. Biologically, this suggests that under these specific conditions, the disease will sustain a stable presence in the population, with the susceptible and infected groups reaching equilibrium. The transmission and recovery rates are crucial in determining the persistence and steady-state levels of the disease.

In this study, the classical integer-order model corresponds to $\alpha = 1$. Comparing the dynamics at $\alpha = 1$ with those at fractional orders $\alpha < 1$ reveals that smaller values (e.g., $\alpha = 0.6$) result in slower convergence to equilibrium due to the memory effects inherent to fractional derivatives. In contrast, the classical model stabilizes more rapidly, closely matching the behavior of integer-order formulations, as illustrated in Figures 2 and 3. These observations underscore the enhanced flexibility of the fractional model in capturing the complex, history-dependent temporal dynamics of disease spread, offering a more accurate description compared to classical approaches. That highlights the importance of α not only in determining the system's steady state but also in modulating the speed of disease progression.

7. Conclusions

This study aimed to investigate the stability of a fractional-order SI parasite-host model incorporating the Liouville-Caputo fractional derivative. By considering memory effects and long-term dependencies, factors often overlooked in traditional integer-order models, this approach offers valuable insights into disease persistence and transmission dynamics. The study successfully demonstrated the existence and uniqueness of the solution, guaranteeing its positivity and boundedness. Furthermore, the asymptotic analysis identified two equilibrium points characterized by the basic reproduction number R_0: the disease-free equilibrium and the endemic equilibrium. When $R_0 \leq 1$, the equilibrium E_0 is globally stable, signifying the eventual elimination of the disease. Conversely, when $R_0 > 1$, the equilibrium E^*

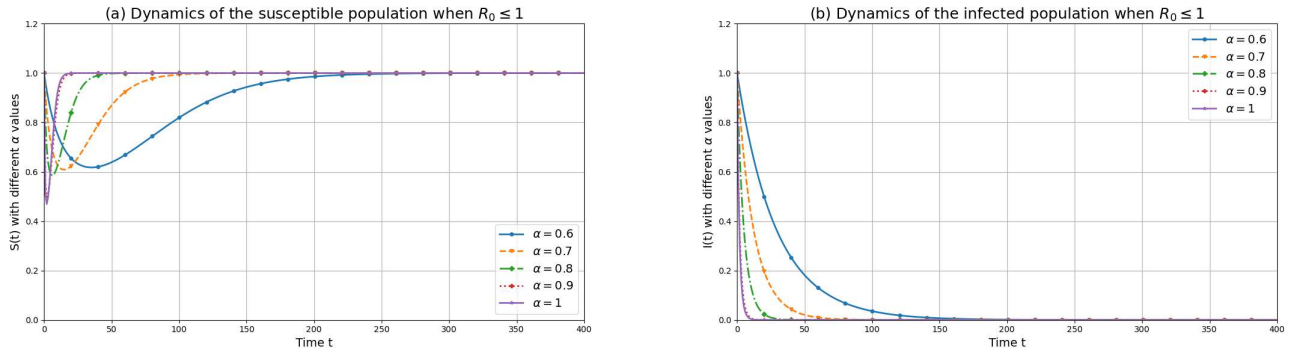

FIGURE 2. Stability of E_0 for various values of α.

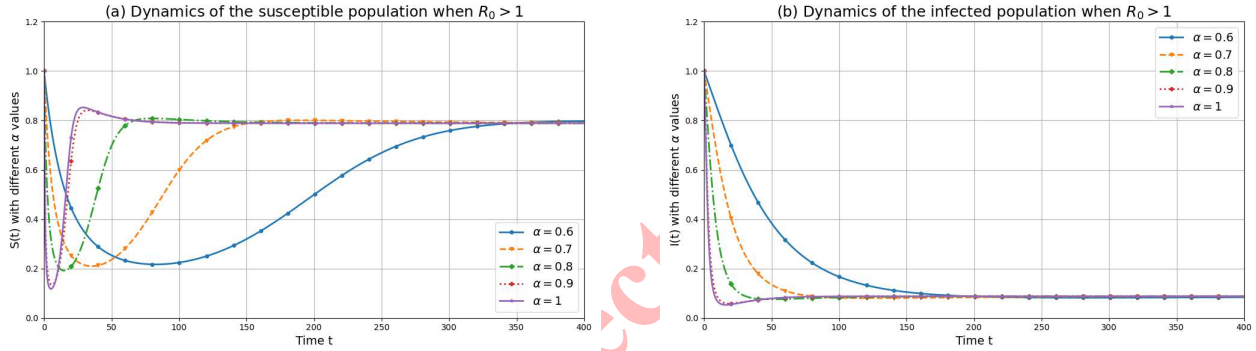

FIGURE 3. Stability of E^* for various values of α.

becomes stable, indicating the potential for the disease to persist within the population. Additionally, the bifurcation analysis confirmed that the model exhibits a forward transition at the critical threshold $R_0 = 1$, ensuring a smooth change in disease dynamics as parameters vary. Numerical simulations verified the stability of these equilibria, emphasizing the effect of changes in α on the system's dynamics. From a biological standpoint, this study highlights the long-term dynamics of parasite-host interactions. It underscores the significance of critical parameters, including transmission and recovery rates, in shaping disease dynamics and determining the persistence of infections. Our future research could first focus on exploring alternative numerical methods to enhance the accuracy and efficiency of simulations, thus providing a deeper qualitative understanding of the model. Subsequently, extending the fractional-order framework to incorporate spatial diffusion would allow for a more realistic modeling of disease spread in heterogeneous environments. Finally, applying our study to real existing diseases would allow for model validation and more accurate estimation of key epidemiological parameters.

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