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Global Dynamics of a Social Hierarchy-Stratified Malaria Model: Insight from Fractional Calculus

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Abstract: In this study, a mathematical model for the transmission dynamics of malaria among different socioeconomic groups in the human population interacting with a susceptible-infectious vector population is presented and analysed using a fractional-order derivative of the Caputo type. The total human population is stratified into two distinguished classes of lower and higher income individuals, with each class further subdivided into susceptible, infectious, and recovered populations. The socio hierachy-structured fractional-order malaria model is analyzed through the application of different dynamical system tools. The theory of positivity and boundedness based on the generalized mean value theorem is employed to investigate the basic properties of solutions of the model, while the Banach fixed point theory approach is used to prove the existence and uniqueness of the solution. Furthermore, unlike the existing related studies, comprehensive global asymptotic dynamics of the fractional-order malaria model around both disease-free and endemic equilibria are explored by generalizing the usual classical methods for establishing global asymptotic stability of the steady states. The asymptotic behavior of the trajectories of the system are graphically illustrated at different values of the fractional (noninteger) order.

Keywords: fractional-order system; social hierarchy model; malaria dynamics; Banach fixed point theory; global stability

MSC: 37N25; 34D23; 34A08; 92D25

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1. Introduction

The evolution of infectious diseases has been a regular threat to humanity and a bone of contention for policymakers [1]. Several dangerous infectious diseases such as Ebola, malaria, measles, Zika, Acquired Immune Deficiency Syndrome (AIDS), tuberculosis, chickenpox, chikungunya virus (CHIKV) and COVID-19 have posed an intensifying threat to humanity due to their emergence and re-emergence in the population [2,3]. To date, malaria, which is caused by a single-celled parasite of the genus *Plasmodium* has maintained its stance as one of the vector-borne diseases with an overwhelming adverse effect on the human population [2]. The transmission mode requires the parasitic interaction between a human (host) and a vector (mosquito).

The malaria parasite is typically transmitted to humans through the bite of an infected female *Anopheles* mosquito, which is the main carrier of the parasite. Malaria parasites may also be transmitted to humans through the transfusion of infected blood, organ

transplant, or the sharing of contaminated needles or syringes, as well as from a mother to her unborn infant before or during delivery (congenital malaria) [4]. In 2020, the World Health Organization (WHO) stated in their 2021 malaria global report that there were around 241 million cases of malaria globally and 627,000 deaths due to the whip of the disease [2]. Malaria symptoms may include fever, headache, sweats, muscle aches, chills, tiredness, nausea, vomiting, and diarrhea, among others [2]. If promptly detected, the disease is preventable and curable, but symptoms may go out of hand if not detected early and properly treated [5,6].

Mathematical modeling has become an inestimable tool for finding solutions to the complexities encountered in the transmission of infectious diseases. In particular, several recent mathematical models have been devoted to the study of malaria dynamics in the literature; see for instance [5–18] and some of the cited references in the recent scoping review presented in Anwar et al. [19]. Specifically, Abimbade et al. [5] designed a mathematical model for the evolution of recurrent malaria in the human population. The authors considered all categories of recurrent malaria, including recrudescence, relapse, and reinfection. In a similar development, Tasman et al. [6] developed and analyzed a deterministic model to study the transmission dynamics of recurrent malaria with relapse, reinfection, and recrudescence, thus taking into account the inadequacy of hospital beds. Bakare [8] formulated and analyzed a nonautonomous malaria model that took into account five optimal control measures representing the use of insecticide-treated bed nets, educational campaigns, indoor residual sprayings, the clearance of mosquito breeding sites, and treatment control in mitigating the dynamical spread of malaria in the population. Traore [9] designed a temperature-dependent malaria model where the mosquito population was structured into stages. In [10], the authors presented an optimal framework for the transmission dynamics of malaria that incorporated mosquito seasonal factors, and the impacts of insecticide, prevention, and treatment controls on the malaria model were examined. Furthermore, Keno [14] applied optimal control theory and economic analysis to a deterministic mathematical model with atmospheric variation.

In another development, the authors in [15] presented and rigorously analyzed a malaria model incorporating a direct atmospheric-mediated transmission mode. In [16], the authors formulated and analyzed a malaria model incorporating relapse and unenlightened infected individuals. Furthermore, Olaniyi and coworkers [17] presented an optimal control framework for the transmission dynamics of malaria through the transfusion of infected blood and the indirect horizontal transmission (human vector) route with saturated treatment function. The authors in [20] presented an optimal control framework for recurrent malaria dynamics with a view to providing effective optimal control strategies to be implemented in setting the recurrence of malaria in the human population to extinction. In addition, Olaniyi et al. [21] stressed on the efficiency and economic analyses of a recurrent malaria model. Their focus was centered on identifying the most efficient and most cost-effective strategy that best averts the highest number of recurrent malaria infection in the population.

At this point, it is worth noting that all the aforementioned studies did not consider the stratification of the human population into social classes. However, the authors in [22] developed a mathematical model for the transmission dynamics of malaria by categorizing the human population into two main socioeconomic divisions, namely low-income and high-income individuals. The formulated model was analyzed via optimal control theory and extended to include efficiency and economic analyses to procure the most efficient and effective control strategy for mitigating the spread of malaria among social classes in the population. Modeling infectious disease using a fractional-order derivative operator allows for a more accurate description of the disease transmission than the classical modeling approach. The nonlocal nature of the fractional-order system makes it more suitable to model disease dynamics where prior history of the disease governs its future evolution (see, [23,24]). Thus, modeling malaria spread in a social hierarchy-structured population with fractional-order derivative operator will create a history such that the current behav-

ior of the disease will depend on the previous outbreaks. This explains the capacity of fractional-order models in capturing memory effects in the transmission dynamics of disease. Hence, it is of essence to improve on the existing knowledge of malaria transmission dynamics by studying the effect of memory on the evolution of malaria within social classes in the population. This can be achieved by generalizing the model developed in [22] in the framework of fractional calculus.

It is worthy of note that Atangana–Baleanu and piecewise Caputo–Fabrizio fractional versions of the model have been analyzed in Bonyah [25] and Aldwoah et al. [26], respectively. Both studies in [25,26] focused on establishing the existence and uniqueness of the solution of the model, thereby exploring the crossover effects associated with malaria dynamics. However, the comprehensive global stability dynamics of the social hierarchy-structured malaria model with either a classical or fractional derivative operator remains unexplored thus far. As a result of this, the fractional-order of the Caputo type is employed in this present study, with specific focus on gaining insights into the global asymptotic dynamics of social hierarchy-structured malaria transmission with memory using a more generalized approach for establishing the global asymptotic stability of the steady states of the fractional-order model. In other words, this study examines the influence of memory on the global dynamics of malaria among social classes by extending the usual classical methods for investigating the global asymptotic stabilities of both disease-free and endemic equilibrium points to a more general approach using fractional calculus with a Caputo derivative operator. The remaining aspects of the study are organized as follows: In Section 2, the noninteger-order social hierarchy-stratified model is presented with its qualitative analysis for the existence and uniqueness of solutions. Section 3 presents the global asymptotic dynamics of the fractional-order model with simulations and discussion. Section 4 deals with the concluding remarks of the study.

2. Fractional-Order Social Hierarchy-Stratified Model

The mathematical model presented in this study is a fractionalized version of the classical-order nonlinear malaria model developed in Olaniyi et al. [22]. It has been established by a plethora of researchers that fractional-order derivatives define real-life situations better than the usual classical-order derivatives. This is due to the fact that fractional-order derivatives possess distinctive properties such as memory and heredity, which enable adequate and effective comprehension of real-life phenomena [27–33]. It is on this note that this study is focused on the application of fractional calculus to the epidemiology of malaria with a view to gaining further insights into how the consequences of memory affect the transmission dynamics of the disease in a social heirarchy-structured human population. To start with, it is of essence to state some basic concepts and analytic results in fractional calculus following [3,34,35].

Definition 1. *A Riemann–Liouville fractional integral of order α of relation $h : \mathbb{R}_+ \to \mathbb{R}$, designated by $I_t^\alpha h(t)$, is defined as*

$$I_t^\alpha h(t) = \frac{1}{\Gamma(\alpha)} \int_0^t \frac{h(\xi)}{(t-\xi)^{1-\alpha}} d\xi, \tag{1}$$

where $\alpha \in \mathbb{R}_+$ such that $\alpha \in (0,1)$ and $t > 0$. The gamma function, $\Gamma(\alpha)$, is given by

$$\Gamma(\alpha) = \int_0^\infty x^{\alpha-1} e^{-x} dx. \tag{2}$$

Definition 2. *A fractional derivative of order α of $h : \mathbb{R}_+ \to \mathbb{R}$ of the Caputo type, denoted by ${}^{\mathcal{C}}D_t^{\alpha}h(t)$, is defined as*

$$ {}^{\mathcal{C}}D_t^{\alpha}h(t) = \begin{cases} \dfrac{1}{\Gamma(1-\alpha)} \displaystyle\int_0^t \frac{h'(\varrho)}{(t-\varrho)^{\alpha}} d\varrho, \\ \dfrac{d^{\alpha}}{dt^{\alpha}}h(t), \quad 0 < \alpha \leq 1. \end{cases} \tag{3} $$

Lemma 1 (Generalized Mean Value Theorem)**.** *Let $g(t) \in C[0, t^*]$ and ${}^{\mathcal{C}}D_t^{\alpha}g(t) \in C[0, t^*]$ for $0 < \alpha \leq 1$; then,*

$$ g(t) = g(0) + \frac{{}^{\mathcal{C}}D_t^{\alpha}g(\varphi)t^{\alpha}}{\Gamma(\alpha)}, \quad \varphi \in [0, t], \ \forall\ t \in (0, t^*]. $$

(i) If ${}^{\mathcal{C}}D_t^{\alpha}g(t) \geq 0\ \forall\ t \in [0, t^*]$, then $g(t)$ is nondecreasing for each $t \in (0, t^*)$.
(ii) If ${}^{\mathcal{C}}D_t^{\alpha}g(t) \leq 0\ \forall\ t \in [0, t^*]$, then $g(t)$ is nonincreasing for each $t \in (0, t^*)$.

Lemma 2. *Let $\chi(t) \in C([0, \infty))$ satisfy*

$$ {}^{\mathcal{C}}D_t^{\alpha}\chi(t) + a_1\chi(t) \leq a_2,\ \chi(0) = \chi_0, $$

where $\alpha \in (0, 1]$ and $a_1, a_2 \in \mathbb{R}$, with $a_1 \neq 0$; then,

$$ \chi(t) \leq \left(\chi_0 - \frac{a_2}{a_1}\right) E_{\alpha,1}(-a_1 t^{\alpha}) + \frac{a_2}{a_1}, $$

where $E_{\alpha,1}(\cdot)$ is a Mittag–Leffler function with one parameter α given by

$$ E_{\alpha,1}(x) = \sum_{n=0}^{\infty} \frac{x^n}{\Gamma(\alpha n + 1)}. $$

Lemma 3. *$\chi(t) = \chi(0)E_{\alpha,1}(kt^{\alpha})$ solves the fractional-order differential equation of the form ${}^{\mathcal{C}}D_t^{\alpha}\chi(t) = k\chi(t)$.*

Consequently, the nonlinear fractional-order differential equations of the Caputo type describing the evolution of the social hierarchy-structured malaria model are given by

$$ \begin{aligned} {}^{\mathcal{C}}D_t^{\alpha}S_L(t) &= (1-r)\Lambda_H - \beta_1 S_L(t) I_V(t) + \omega R_L(t) + \sigma_H S_H(t) - (\mu_H + \sigma_L) S_L(t) \\ {}^{\mathcal{C}}D_t^{\alpha}S_H(t) &= r\Lambda_H - b\beta_1 S_H(t) I_V(t) + \epsilon R_H(t) + \sigma_L S_L(t) - (\mu_H + \sigma_H) S_H(t) \\ {}^{\mathcal{C}}D_t^{\alpha}I_L(t) &= \beta_1 S_L(t) I_V(t) - (\mu_H + \gamma + \delta) I_L(t) \\ {}^{\mathcal{C}}D_t^{\alpha}I_H(t) &= b\beta_1 S_H(t) I_V(t) - (\mu_H + \alpha + \phi) I_H(t) \\ {}^{\mathcal{C}}D_t^{\alpha}R_L(t) &= \gamma I_L(t) - (\omega + \mu_H) R_L(t) \\ {}^{\mathcal{C}}D_t^{\alpha}R_H(t) &= \alpha I_H(t) - (\epsilon + \mu_H) R_H(t) \\ {}^{\mathcal{C}}D_t^{\alpha}S_V(t) &= \Lambda_V - \beta_2 (I_L + \theta I_H) S_V(t) - \mu_V S_V(t) \\ {}^{\mathcal{C}}D_t^{\alpha}I_V(t) &= \beta_2 (I_L + \theta I_H) S_V(t) - \mu_V I_V(t). \end{aligned} \tag{4} $$

with initial conditions

$$ \begin{aligned} &S_L(0) = S_{L0},\ S_H(0) = S_{H0},\ I_L(0) = I_{L0},\ I_H(0) = I_{H0}, \\ &R_L(0) = R_{L0},\ R_H(0) = R_{H0},\ S_V(0) = S_{V0},\ I_V(0) = I_{V0}. \end{aligned} \tag{5} $$

Malaria transmission dynamics involve the interaction between human and vector populations. The total human population, $N_H(t)$, is socially structured into six mutually exclusive compartments, namely a low social class susceptible population denoted by $S_L(t)$, a high social class susceptible population denoted by $S_H(t)$, a low social class infectious population denoted by $I_L(t)$, a high social class infectious population denoted by $I_H(t)$, a low social class recovered population denoted by $R_L(t)$, and a high social class recovered population denoted by $R_H(t)$. The vector population is stratified into a susceptible population designated by $S_V(t)$ and an infectious population denoted by $I_V(t)$. Then, the total human population, $N_H(t)$ at time t is given by

$$N_H(t) = S_L(t) + S_H(t) + I_L(t) + I_H(t) + R_L(t) + R_H(t), \tag{6}$$

and the total vector population is given by

$$N_V(t) = S_V(t) + I_V(t). \tag{7}$$

The lower social class human population is a set of lower income individuals, given by $\{S_L(t), I_L(t), R_L(t)\}$, who have little or no accessibility to medical treatment and other resources for sustainance in the population unlike the higher social class population set $\{S_H(t), I_H(t), R_H(t)\}$. The population of lower social class is generated by the fraction of recruitment of individuals into the population assumed susceptible at a rate of $(1-r)\Lambda_H$, while the remaining fraction $r\Lambda_H$ goes to the higher social class population. The susceptible individuals in the lower and higher social groups are infected following effective contact with infectious mosquitoes at incidence rates of $\beta_1 S_L I_V$ and $b\beta_1 S_H I_V$, where β_1 is the transmission probability of infection, and b is the modification parameter responsible for the degree of infection within higher social group individuals.

The susceptible lower and higher social class individuals are further increased by the rate at which recovered humans $R_L(t)$ and $R_H(t)$ loss their immunity at rates ω and ϵ, respectively. The infectious individuals in lower and higher income classes recover from the disease at rates γ and α, respectively. The populations of infectious individuals in both social classes are downsized by the disease-induced death at their respective rates δ and ϕ, while the total human population is dwindled by the natural mortality rate μ_H. Furthermore, the population of susceptible mosquitoes is increased by the recruitment of mosquitoes at a rate Λ_V and become infected due to the contact with both infectious lower and higher social class individuals at incidence rate $\beta_2(I_L + \theta I_H)$, with β_2 being the effective contact rate and θ being the modification parameter responsible for the reduction of infection among the higher social class individuals. The total mosquito population is diminished by the natural mortality rate μ_V. The Caputo fractional derivative operator is chosen to formulate system (4) because of its suitability for initial conditions in a classical sense unlike the Riemann–Liouville derivative operator. In addition, the Caputo derivative of a constant function always yields zero, thus satisfying the fundamental principle of calculus, unlike some other fractional-order derivative operators [27,28]. It should be emphasized that the full description and assumptions governing the model formulation can be found in the classical version presented in [22], but it is pertinent to mention that all the parameters of the model governed by the system (4) are measured per fractional-order time, $t^{-\alpha}$ (see, e.g., [28,32,35]), unlike the classical model in [22], where parameters were measured per unit time t^{-1}.

2.1. Basic Properties of the Fractional Model

Herein, the basic properties of solutions of the fractional-order malaria model (4) are investigated using the theory of positivity and boundedness.

2.1.1. Positivity and Boundedness of Solution

Theorem 1. *The solutions* $\{S_L(t), S_H(t), I_L(t), I_H(t), R_L(t), R_H(t), S_V(t), \text{and } I_V(t)\}$ *of the fractional-order social hierarchy-structured model* (4) *remain non-negative for all* $t > 0$ *if the associated initial conditions* (5) *are non-negative.*

Proof. It is straightforward from system (4) that

$$\begin{aligned}
{}^{\mathcal{C}}D_t^{\alpha}(S_L)(t)|_{S_L=0} &= (1-r)\Lambda_H + \omega R_L(t) + \sigma_H S_H(t) > 0,\\
{}^{\mathcal{C}}D_t^{\alpha}(S_H)(t)|_{S_H=0} &= r\Lambda_H + \epsilon R_H(t) + \sigma_L S_L(t) > 0,\\
{}^{\mathcal{C}}D_t^{\alpha}(I_L)(t)|_{I_L=0} &= \beta_1 S_L(t) I_V(t) \geq 0,\\
{}^{\mathcal{C}}D_t^{\alpha}(I_H)(t)|_{I_H=0} &= b\beta_1 S_H(t) I_V(t) \geq 0,\\
{}^{\mathcal{C}}D_t^{\alpha}(R_L)(t)|_{R_L=0} &= \gamma I_L(t) \geq 0,\\
{}^{\mathcal{C}}D_t^{\alpha}(R_H)(t)|_{R_H=0} &= \alpha I_H(t) \geq 0,\\
{}^{\mathcal{C}}D_t^{\alpha}(S_V)(t)|_{S_V=0} &= \Lambda_V > 0,\\
{}^{\mathcal{C}}D_t^{\alpha}(I_V)(t)|_{I_V=0} &= \beta_2(I_L + \theta I_H) S_V(t) \geq 0.
\end{aligned} \tag{8}$$

Following the fact that the Caputo derivatives in (8) are non-negative on the bounding planes $\mathbb{R}_+^8$ with the non-negative initial conditions, then by using the generalized mean value theorem (see, Lemma 1), it follows that the solutions $S_L(t)$, $S_H(t)$, $I_L(t)$, $I_H(t)$, $R_L(t)$, $R_H(t)$, $S_V(t)$, and $I_V(t)$ are non-decreasing for all time $t > 0$. Hence, we have the proof. □

Theorem 2. *A region* $\mathfrak{D}$ *of the fractional-order social hierarchy-structured malaria model* (4)*, which is defined by* $\mathfrak{D} = \mathfrak{D}_H \times \mathfrak{D}_V \subset \mathbb{R}_+^6 \times \mathbb{R}_+^2$*, where*

$$\mathfrak{D}_H = \left\{ (S_L(t), S_H(t), I_L(t), I_H(t), R_L(t), R_H(t)) \in \mathbb{R}_+^6 : N_H(t) \leq \frac{\Lambda_H}{\mu_H} \right\},$$

$$\mathfrak{D}_V = \left\{ (S_V(t), I_V(t)) \in \mathbb{R}_+^2 : N_V(t) \leq \frac{\Lambda_V}{\mu_V} \right\},$$

is positively invariant.

Proof. Given that $N_H(t) = S_L(t) + S_H(t) + I_L(t) + I_H(t) + R_L(t) + R_H(t)$ and $N_V(t) = S_V(t) + I_V(t)$, it then follows that the Caputo derivatives of $N_H(t)$ and $N_V(t)$ are given by

$$\begin{aligned}
{}^{\mathcal{C}}D_t^{\alpha}N_H(t) &= {}^{\mathcal{C}}D_t^{\alpha}S_L(t) + {}^{\mathcal{C}}D_t^{\alpha}S_H(t) + {}^{\mathcal{C}}D_t^{\alpha}I_L(t) + {}^{\mathcal{C}}D_t^{\alpha}I_H(t) + {}^{\mathcal{C}}D_t^{\alpha}R_L(t) + {}^{\mathcal{C}}D_t^{\alpha}R_H(t)\\
&= \Lambda_H - \mu_H N_H - \delta I_L - \phi I_H,\\
&\leq \Lambda_H - \mu_H N_H.
\end{aligned} \tag{9}$$

Similarly,

$${}^{\mathcal{C}}D_t^{\alpha}N_V(t) + \mu_V N_V \leq \Lambda_V. \tag{10}$$

Now, invoking Lemma 2 on (9) and (10) yields

$$N_H(t) \leq \left(N_H(0) - \frac{\Lambda_H}{\mu_H} \right) E_{\alpha,1}(-\mu_H t^{\alpha}) + \frac{\Lambda_H}{\mu_H}, \tag{11}$$

and

$$N_V(t) \leq \left(N_V(0) - \frac{\Lambda_V}{\mu_V}\right) E_{\alpha,1}(-\mu_V t^{\alpha}) + \frac{\Lambda_V}{\mu_V}. \tag{12}$$

Then, taking the lim sup as $t \to \infty$ implies that $N_H(t) \leq \Lambda_H/\mu_H$ and $N_V(t) \leq \Lambda_V/\mu_V$. Accordingly, the solution path of the system (4) is bounded in $\mathfrak{D}$, thus showing that the region $\mathfrak{D}$ is positively invariant. □

2.1.2. Existence and Uniqueness of Solution

This subsection is dedicated to the investigation of the existence and uniqueness of the solution of the social hierarchy-structured fractional-order malaria model (4) using the Banach's fixed point theory approach [3,33,36,37]. Suppose the noninteger-order malaria model (4) is rewritten in a compact form:

$$\begin{aligned} {}^{C}D_t^{\alpha}(\mathcal{G}(t)) &= \mathcal{H}(t, \mathcal{G}(t)), \quad 0 \leq t \leq \Phi, \\ \mathcal{G}(0) &= \mathcal{G}_0, \end{aligned} \tag{13}$$

where $\mathcal{G}(t) = (S_L(t), S_H(t), I_L(t), I_H(t), R_L(t), R_H(t), S_V(t), I_V(t))^{\intercal}$, and $\mathcal{H}(t, \mathcal{G}(t)) : [0, \Phi] \times \mathbb{R}_+^8 \to \mathbb{R}$ are defined by

$$\mathcal{H}(t, \mathcal{G}(t)) = (\mathcal{H}_i(t, S_L(t), S_H(t), I_L(t), I_H(t), R_L(t), R_H(t), S_V(t), I_V(t))^{\intercal},$$

for $i = 1, 2, ..., 8$ so that

$$\begin{aligned} \mathcal{H}_1(t, \mathcal{G}(t)) &= (1-r)\Lambda_H - \beta_1 S_L(t) I_V(t) + \omega R_L(t) + \sigma_H S_H(t) - (\mu_H + \sigma_L) S_L(t), \\ \mathcal{H}_2(t, \mathcal{G}(t)) &= r\Lambda_H - b\beta_1 S_H(t) I_V(t) + \epsilon R_H(t) + \sigma_L S_L(t) - (\mu_H + \sigma_H) S_H(t), \\ \mathcal{H}_3(t, \mathcal{G}(t)) &= \beta_1 S_L(t) I_V(t) - (\mu_H + \gamma + \delta) I_L(t), \\ \mathcal{H}_4(t, \mathcal{G}(t)) &= b\beta_1 S_H(t) I_V(t) - (\mu_H + \alpha + \phi) I_H(t), \\ \mathcal{H}_5(t, \mathcal{G}(t)) &= \gamma I_L(t) - (\omega + \mu_H) R_L(t), \\ \mathcal{H}_6(t, \mathcal{G}(t)) &= \alpha I_H(t) - (\epsilon + \mu_H) R_H(t), \\ \mathcal{H}_7(t, \mathcal{G}(t)) &= \Lambda_V - \beta_2 (I_L + \theta I_H) S_V(t) - \mu_V S_V(t), \\ \mathcal{H}_8(t, \mathcal{G}(t)) &= \beta_2 (I_L + \theta I_H) S_V(t) - \mu_V I_V(t), \end{aligned} \tag{14}$$

where $\mathcal{G}_0 = (S_{L0}, S_{H0}, I_{L0}, I_{H0}, R_{L0}, R_{H0}, S_{V0}, I_{V0},)^{\intercal}$.

Now, following Definition 1 by integrating (13) fractionally gives

$$\mathcal{G}(t) = \mathcal{G}_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \Psi)^{\alpha - 1} \mathcal{H}(\Psi, \mathcal{G}(\Psi)) d\Psi. \tag{15}$$

Assume that $\mathbf{M} = (C[0, \Phi], \|\cdot\|)$ is a Banach space for all real-valued continuous functions with the supremum norm governed by

$$\|\mathcal{G}(t)\| = \sup\{|\mathcal{G}(t)| : t \in [0, \Phi]\},$$

with

$$\sup|\mathcal{G}(t)| = \sup(|S_L(t)| + |S_H(t)| + |I_L(t)| + |I_H(t)| + |R_L(t)| + |R_H(t)| + |S_V(t)| + |I_V(t)|).$$

At this juncture, it is important to establish that $\mathcal{H}(t, \mathcal{G}(t))$ is Lipschitz continuous, and this is investigated as presented in the next result.

Theorem 3. *The vector function $\mathcal{H}(t, \mathcal{G}(t))$ is Lipschitzian in $\mathcal{G}(t)$ on $C([0, \Phi] \times \mathbb{R}^8_+, \mathbb{R})$ if there exists a constant $\mathcal{P} > 0$ such that*

$$\|\mathcal{H}(t, \mathcal{G}_1(t)) - \mathcal{H}(t, \mathcal{G}_2(t))\| \leq \mathcal{P}\|\mathcal{G}_1(t) - \mathcal{G}_2(t)\|. \tag{16}$$

Proof. Since the states of the fractional-order social hierarchy-structured malaria model (4) are bounded by Λ_H/μ_H for the human population and Λ_V/μ_V for the vector population in a positively invariant region $\mathfrak{D}$, then considering $\mathcal{H}_1(t, S_L(t))$, and for $S_{L1}(t)$ and $S_{L2}(t)$, it follows that

$$\|\mathcal{H}_1(t, S_{L1}(t)) - \mathcal{H}_1(t, S_{L2}(t))\| \leq \|(\beta_1 I_V + \mu_H + \sigma_L)\|\|S_{L1} - S_{L2}\|. \tag{17}$$

Since $I_V \leq \Lambda_V/\mu_V$ in $\mathfrak{D}$, then the inequality (17) becomes

$$\|\mathcal{H}_1(t, S_{L1}(t)) - \mathcal{H}_1(t, S_{L2}(t))\| \leq \mathcal{P}_1\|S_{L1} - S_{L2}\|, \tag{18}$$

where $\mathcal{P}_1 = \left(\frac{\beta_1\Lambda_V}{\mu_V} + (\mu_H + \sigma_L)\right) > 0$.

In a similar manner, for any $S_{H1}(t)$ and $S_{H2}(t)$,

$$\|\mathcal{H}_2(t, S_{H1}) - \mathcal{H}_2(t, S_{H2})\| \leq \mathcal{P}_2\|S_{H1} - S_{H2}\|, \tag{19}$$

where $\mathcal{P}_2 = \left(\frac{b\beta_1\Lambda_V}{\mu_V} + (\mu_H + \sigma_H)\right) > 0$, since $I_V \leq \Lambda_V/\mu_V$. For any $I_{L1}(t)$ and $I_{L2}(t)$,

$$\|\mathcal{H}_3(t, I_{L1}) - \mathcal{H}_3(t, I_{L2})\| \leq \mathcal{P}_3\|I_{L1} - I_{L2}\|, \tag{20}$$

where $\mathcal{P}_3 = (\gamma + \delta + \mu_H) > 0$. For any $I_{H1}(t)$ and $I_{H2}(t)$,

$$\|\mathcal{H}_4(t, I_{H1}) - \mathcal{H}_4(t, I_{H2})\| \leq \mathcal{P}_4\|I_{H1} - I_{H2}\|, \tag{21}$$

where $\mathcal{P}_4 = (\alpha + \phi + \mu_H) > 0$. For any $R_{L1}(t)$ and $R_{L2}(t)$,

$$\|\mathcal{H}_5(t, R_{L1}) - \mathcal{H}_5(t, R_{L2})\| \leq \mathcal{P}_5\|R_{L1} - R_{L2}\|, \tag{22}$$

where $\mathcal{P}_5 = (\omega + \mu_H) > 0$. For any $R_{H1}(t)$ and $R_{H2}(t)$,

$$\|\mathcal{H}_6(t, R_{H1}) - \mathcal{H}_6(t, R_{H2})\| \leq \mathcal{P}_6\|R_{H1} - R_{H2}\|, \tag{23}$$

where $\mathcal{P}_6 = (\epsilon + \mu_H) > 0$. Furthermore, considering $\mathcal{H}_7(t, S_V(t))$, and for any $S_{V1}(t)$ and $S_{V2}(t)$, following a similar procedure yields

$$\|\mathcal{H}_7(t, S_{V1}(t)) - \mathcal{H}_7(t, S_{V2}(t))\| \leq \|\beta_2(I_L + \theta I_H)\|\|S_{V1} - S_{V2}\|. \tag{24}$$

Since I_L and I_H are bounded above by Λ_H/μ_H in the invariant region $\mathcal{D}$, it then follows that the inequality (24) becomes

$$\|\mathcal{H}_7(t, S_{V1}(t)) - \mathcal{H}_7(t, S_{V2}(t))\| \leq \mathcal{P}_7\|S_{V1} - S_{V2}\|, \tag{25}$$

where $\mathcal{P}_7 = \left(\frac{\beta_2\Lambda_H}{\mu_H}(1 + \theta) + \mu_V\right) > 0$. Also, for any $I_{V1}(t)$ and $I_{V2}(t)$,

$$\|\mathcal{H}_8(t, I_{V1}(t)) - \mathcal{H}_8(t, I_{V2}(t))\| \leq \mathcal{P}_8\|I_{V1} - I_{V2}\|, \tag{26}$$

where $\mathcal{P}_8 = \mu_V > 0$.

In view of the foregoing, it is clear that the noninteger-order social hierarchy-structured malaria model (4) is Lipschitz continuous, thus satisfying the condition 16, where the Lipschitz constant $\mathcal{P} = \max\{\mathcal{P}_i\}$, and $i = 1, 2, ..., 8$. □

Now, we define a fixed point of an operator $\mathcal{Q} : \mathbf{M} \to \mathbf{M}$ by $\mathcal{Q}(\mathcal{G}(t)) = \mathcal{G}(t)$ so that

$$\mathcal{Q}(\mathcal{G}(t)) = \mathcal{G}_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \Psi)^{\alpha-1} \mathcal{H}(\Psi, \mathcal{G}(\Psi)) d\Psi. \tag{27}$$

Theorem 4. *The fractional-order system* (4) *has a unique solution* $\mathcal{G}(t) \in \mathbf{M}$ *provided that* $\Phi^{\alpha}\mathcal{P} < \Gamma(\alpha + 1)$.

Proof. The main interest in proving this result is to show that $\mathcal{Q}$ is a contraction. Since $\mathcal{H}(t, \mathcal{G}(t))$ is Lipschitz continuous, as theorized in Equation (16), it follows that for any $\mathcal{G}_1(t), \mathcal{G}_2(t) \in \mathbf{M}$ and since $0 \leq t \leq \Phi$,

$$\begin{aligned}
\|\mathcal{Q}(\mathcal{G}_1(t)) - \mathcal{Q}(\mathcal{G}_2(t))\| &= \left\| \frac{1}{\Gamma(\alpha)} \int_0^t (t - \Psi)^{\alpha-1} [\mathcal{H}(\Psi, \mathcal{G}_1(\Psi)) - \mathcal{H}(\Psi, \mathcal{G}_2(\Psi))] d\Psi \right\| \\
&\leq \frac{1}{\Gamma(\alpha)} \int_0^t (t - \Psi)^{\alpha-1} \|\mathcal{H}(\Psi, \mathcal{G}_1(\Psi)) - \mathcal{H}(\Psi, \mathcal{G}_2(\Psi))\| d\Psi \\
&\leq \frac{\mathcal{P}}{\Gamma(\alpha)} \|\mathcal{G}_1(t) - \mathcal{G}_2(t)\| \int_0^t (t - \Psi)^{\alpha-1} d\Psi \\
&\leq \mathcal{P}^* \|\mathcal{G}_1(t) - \mathcal{G}_2(t)\|,
\end{aligned}$$

where $\mathcal{P}^* = \Phi^{\alpha}\mathcal{P}/(\alpha\Gamma(\alpha))$, thus implying that $\mathcal{Q}$ is a contraction, since $\mathcal{P}^* < 1$. Hence, there exists a unique solution for the fractional-order social hierarchy-structured malaria model (4). □

2.2. Basic Reproduction Number

The malaria-free (disease-free) equilibrium of the fractional-order malaria model (4) is obtained as

$$\varepsilon_0 = (S_L^0, S_H^0, 0, 0, 0, 0, S_V^0, 0), \tag{28}$$

where

$$S_L^0 = \frac{\Lambda_H(\mu_H(1 - r) + \sigma_H)}{\mu_H(\mu_H + \sigma_H + \sigma_L)},$$

$$S_H^0 = \frac{\Lambda_H(\mu_H r + \sigma_L)}{\mu_H(\mu_H + \sigma_H + \sigma_L)},$$

and

$$S_V^0 = \frac{\Lambda_V}{\mu_V}.$$

In what follows, the basic reproduction number of the model is as obtained in [22], and it is given by

$$\mathcal{R}_0 = \sqrt{\frac{\Lambda_H \beta_1 \beta_2 \Lambda_V (\theta b m_1 (r\mu_H + \sigma_L) + m_2(\mu_H(1 - r) + \sigma_H))}{\mu_H \mu_V^2 m_1 m_2 (\mu_H + \sigma_H + \sigma_L)}}, \tag{29}$$

where $m_1 = \mu_H + \gamma + \delta$ and $m_2 = \mu_H + \alpha + \phi$. The basic reproduction number, $\mathcal{R}_0$, is the key epidemiological threshold that determines the average number of secondary cases of malaria infection produced by an infectious individual during its period of infectiousness in a wholly susceptible population [38].

3. Global Asymptotic Dynamics of the Model

This section explores the global asymptotic stability of the fractional-order social hierarchy-structured malaria model (4), since stability analysis has been proven to be an essential performance metric for any dynamical system [29].

3.1. Global Asymptotic Stability of DFE

Since fractional calculus is a generalization of the standard theory of calculus, the global asymptotic stability of the model around the disease-free equilibrium (DFE) of the model (28) is analyzed by extending the classical method that has been x-rayed in [39–42] to a fractional-order derivative operator. To do this, let the fractional-order system (4) be rewritten in a vector form given by

$$\begin{aligned} {}^{\mathcal{C}}D_t^{\alpha}\mathbb{X}(t) &= F(\mathbb{X},\mathbb{Z}),\\ {}^{\mathcal{C}}D_t^{\alpha}\mathbb{Z}(t) &= G(\mathbb{X},\mathbb{Z}),\ G(\mathbb{X},0)=0, \end{aligned} \tag{30}$$

of which $\mathbb{X} \in \mathbb{R}_+^5$ and $\mathbb{Z} \in \mathbb{R}_+^3$, where $\mathbb{X}$ represents the uninfected compartments, and $\mathbb{Z}$ represents the population of infected individuals. In essence, $\mathbb{X} = (S_L, S_H, R_L, R_H, S_V)$, and $\mathbb{Z} = (I_L, I_H, I_V)$. Furthermore, let the disease-free equilibrium of the malaria model (4) be represented by $\varepsilon_0 = (\mathbb{X}^*, 0)$; then, the global asymptotic stability of the social hierarchy-structured malaria model can be established if the following conditions are obeyed:

(N_1): For ${}^{\mathcal{C}}D_t^{\alpha}\mathbb{X}(t) = F(\mathbb{X}, 0)$, $\mathbb{X}^*$ is globally asymptotically stable;
(N_2): $G(\mathbb{X},\mathbb{Z}) = A\mathbb{Z} - \hat{G}(\mathbb{X},\mathbb{Z})$, $\hat{G}(\mathbb{X},\mathbb{Z}) \geq 0$, for $(\mathbb{X},\mathbb{Z}) \in \mathfrak{D}$.

where $A = \partial G/\partial \mathbb{Z}$ is an M matrix evaluated at $(\mathbb{X}^*, 0)$ with non-negative off-diagonal elements.

Theorem 5. *The disease-free equilibrium $\varepsilon_0 = (\mathbb{X}^*, 0)$ of the fractional-order social hierarchy-structured malaria model (4) is globally asymptotically stable if conditions (N_1) and (N_2) are satisfied.*

Proof. $F(\mathbb{X},\mathbb{Z})$ and $G(\mathbb{X},\mathbb{Z})$ are obtained from (4) as follows:

$$F(\mathbb{X},\mathbb{Z}) = \begin{pmatrix} (1-r)\Lambda_H - \beta_1 S_L I_V + \omega R_L + \sigma_H S_H - (\mu_H + \sigma_L) S_L \\ r\Lambda_H - b\beta_1 S_H I_V + \epsilon R_H + \sigma_L S_L - (\mu_H + \sigma_L) S_H \\ \gamma I_L - (\omega + \mu_H) R_L \\ \alpha I_H - (\epsilon + \mu_H) R_H \\ \Lambda_V - \beta_2 (I_L + \theta I_H) S_V - \mu_V S_V \end{pmatrix} \tag{31}$$

and

$$G(\mathbb{X},\mathbb{Z}) = \begin{pmatrix} \beta_1 S_L I_V - (\mu_H + \gamma + \delta) I_L \\ b\beta_1 S_H I_V - (\mu_H + \alpha + \phi) I_H \\ \beta_2 (I_L + \theta I_H) S_V - \mu_V S_V \end{pmatrix}. \tag{32}$$

Since

$$F(\mathbb{X},0)=\begin{pmatrix}(1-r)\Lambda_H+\omega R_L+\sigma_H S_H-(\mu_H+\sigma_L)S_L\\ r\Lambda_H+\epsilon R_H+\sigma_L S_L-(\mu_H+\sigma_L)S_H\\ -(\omega+\mu_H)R_L\\ -(\epsilon+\mu_H)R_H\\ \Lambda_V-\mu_V S_V\end{pmatrix}, \tag{33}$$

then ${}^{\mathcal{C}}D_t^{\alpha}\mathbb{X}(t)=F(\mathbb{X},0)$ implies that

$$\begin{aligned}
{}^{\mathcal{C}}D_t^{\alpha}S_L&=(1-r)\Lambda_H+\omega R_L+\sigma_H S_H-(\mu_H+\sigma_L)S_L,\\
{}^{\mathcal{C}}D_t^{\alpha}S_H&=r\Lambda_H+\epsilon R_H+\sigma_L S_L-(\mu_H+\sigma_L)S_H,\\
{}^{\mathcal{C}}D_t^{\alpha}R_L&=-(\omega+\mu_H)R_L,\\
{}^{\mathcal{C}}D_t^{\alpha}R_H&=-(\epsilon+\mu_H)R_H,\\
{}^{\mathcal{C}}D_t^{\alpha}S_V&=\Lambda_V-\mu_V S_V.
\end{aligned} \tag{34}$$

Using Lemma 3 and solving system (34) simultaneously gives

$$\begin{aligned}
S_L(t) &= \Big(\tfrac{(1-r)\Lambda_H}{(\sigma_H+\sigma_L+\mu_H)}+\tfrac{\sigma_H\Lambda_H}{\mu_H(\sigma_H+\sigma_L+\mu_H)}\Big)(1-E_{\alpha,1}(-(\sigma_H+\sigma_L+\mu_H)t^{\alpha}))\\
&\quad+\Big(\tfrac{\sigma_H}{\sigma_H+\sigma_L}(L(0)+R_L(0)+R_H(0))-\tfrac{\sigma_H\Lambda_H}{\mu_H(\sigma_H+\sigma_L)}\Big)\\
&\quad\times(E_{\alpha,1}(-\mu_H t^{\alpha})-E_{\alpha,1}(-(\sigma_H+\sigma_L+\mu_H)t^{\alpha}))+\tfrac{(\omega-\sigma_H)R_L(0)}{\sigma_H+\sigma_L-\omega}\\
&\quad\times(E_{\alpha,1}(-(\omega+\mu_H)t^{\alpha})-E_{\alpha,1}(-(\sigma_H+\sigma_L+\mu_H)t^{\alpha}))+S_L(0)\\
&\quad\times E_{\alpha,1}(-(\sigma_H+\sigma_L+\mu_H)t^{\alpha})+\tfrac{(\sigma_H)R_H(0)}{\sigma_H+\sigma_L-\omega}\\
&\quad\times(E_{\alpha,1}(-(\sigma_H+\sigma_L+\mu_H)t^{\alpha})-E_{\alpha,1}(-(\epsilon+\mu_H)t^{\alpha})),\\
S_H(t) &= \frac{\Lambda_H}{\mu_H}(1-E_{\alpha,1}(-\mu_H t^{\alpha}))+R_L(0)(E_{\alpha,1}(-\mu_H t^{\alpha})-E_{\alpha,1}(-(\omega+\mu_H)t^{\alpha}))\\
&\quad+R_H(0)(E_{\alpha,1}(-\mu_H t^{\alpha})-E_{\alpha,1}(-(\epsilon+\mu_H)t^{\alpha}))\\
&\quad+L(0)E_{\alpha,1}(-\mu_H t^{\alpha})-S_L(t),\\
R_L(t) &= R_L(0)E_{\alpha,1}(-(\omega+\mu_H)t^{\alpha}),\\
R_H(t) &= R_H(0)E_{\alpha,1}(-(\epsilon+\mu_H)t^{\alpha}),\\
S_V(t) &= \frac{\Lambda_V}{\mu_V}+\Big(S_V(0)-\frac{\Lambda_V}{\mu_V}\Big)E_{\alpha,1}(-\mu_V t^{\alpha}).
\end{aligned} \tag{35, 36}$$

Consequently, as $t\to\infty$ in (35) and (36), regardless of the initial conditions $S_L(0)$, $S_H(0)$, $R_L(0)$, $R_H(0)$, and $S_V(0)$, then $S_L(t)\to S_L^0, S_H(t)\to S_H^0, R_L(t)\to 0, R_H(t)\to 0$ and $S_V(t)\to S_V^0$. As a consequence, the condition (N_1) is satisfied, thus implying that $\mathbb{X}^*$ is globally asymptotically stable.

Further, to establish (N_2), an M matrix with non-negative off-diagonal entries is given by

$$A = \frac{\partial G}{\partial \mathbb{Z}}|_{(\mathbb{X}^*,0)} = \begin{pmatrix} -(\mu_H+\gamma+\delta) & 0 & \beta_1 S_L^0 \\ 0 & -(\mu_H+\alpha+\phi) & b\beta_1 S_H^0 \\ \beta_2 S_V^0 & \theta\beta_2 S_V^0 & -\mu_V \end{pmatrix}. \tag{37}$$

Simplifying $\hat{G}(\mathbb{X},\mathbb{Z}) = A\mathbb{Z} - G(\mathbb{X},\mathbb{Z})$ gives

$$\hat{G}(\mathbb{X},\mathbb{Z}) = \begin{pmatrix} \beta_1 I_V(S_L^0 - S_L) \\ b\beta_1 I_V(S_H^0 - S_H) \\ \beta_2(I_L + \theta I_H)(S_V^0 - S_V) \end{pmatrix}. \tag{38}$$

It is clear that $\hat{G}(X,Z) \geq 0$, since $0 \leq S_L \leq S_L^0$, $0 \leq S_H \leq S_H^0$, and $0 \leq S_V \leq S_V^0$. Hence, property (N_2) is satisfied. Accordingly, the disease-free equilibrium of the fractional-order malaria model (4) is globally asymptotically stable. This ends the proof. □

3.2. Global Asymptotic Stability of EE

Let the endemic equilibrium point of the fractional-order social hierarchy-structured model (4) be represented by ε^{**} so that

$$\varepsilon^{**} = (S_L^{**}, S_H^{**}, I_L^{**}, I_H^{**}, R_L^{**}, R_H^{**}, S_V^{**}, I_V^{**}).$$

It is important to mention that the explicit form of ε^{**} is omitted due to the complexity of the model. However, if it is assumed that the endemic equilibrium point exists, then it is pertinent to establish the asymptotic behavior of the fractional-order social hierarchy-structured model (4) around the endemic equilibrium. To do this, the following result is considered necessary as a consequence of the idea in [29].

Lemma 4. *If $\mathcal{G}^{**}$ is an equilibrium point of the Caputo fractional-order system (13), and $\mathcal{V}(\mathcal{G}(t))$ is a Lyapunov functional defined by*

$$\mathcal{V}(\mathcal{G}(t)) = \sum_{i=1}^{8} \frac{b_i}{2}(\mathcal{G}_i - \mathcal{G}_i^{**})^2, \ \ \forall\, b_i > 0,$$

then

$${}^{\mathcal{C}}D_t^{\alpha}\mathcal{V}(\mathcal{G}(t)) \leq \sum_{i=1}^{8}[b_i(\mathcal{G}_i - \mathcal{G}_i^{**})]\ {}^{\mathcal{C}}D_t^{\alpha}\mathcal{G}_i(t).$$

Theorem 6. *The endemic equilibrium point, ε^{**}, of the social hierarchy-structured malaria model (4) is globally asymptotically stable if the associated threshold quantity, $\mathcal{R}_0$, is greater than unity.*

Proof. Consider a quadratic Lyapunov function $\mathcal{L} : \mathfrak{D} \to \mathbb{R}$ defined by (see, e.g., [43,44])

$$\begin{aligned} \mathcal{L}(\mathcal{G}(t)) = \ & \tfrac{1}{2}[(S_L - S_L^{**}) + (S_H - S_H^{**}) + (I_L - I_L^{**}) + (I_H - I_H^{**}) + (R_L - R_L^{**}) \\ & + (R_H - R_H^{**})]^2 + \tfrac{1}{2}\left[(S_V - S_V^{**}) + (I_V - I_V^{**})\right]^2. \end{aligned} \tag{39}$$

With Lemma 4 in mind, the Caputo fractional time derivative of $\mathcal{L}$, along the solution path of the fractional-order system (4), gives

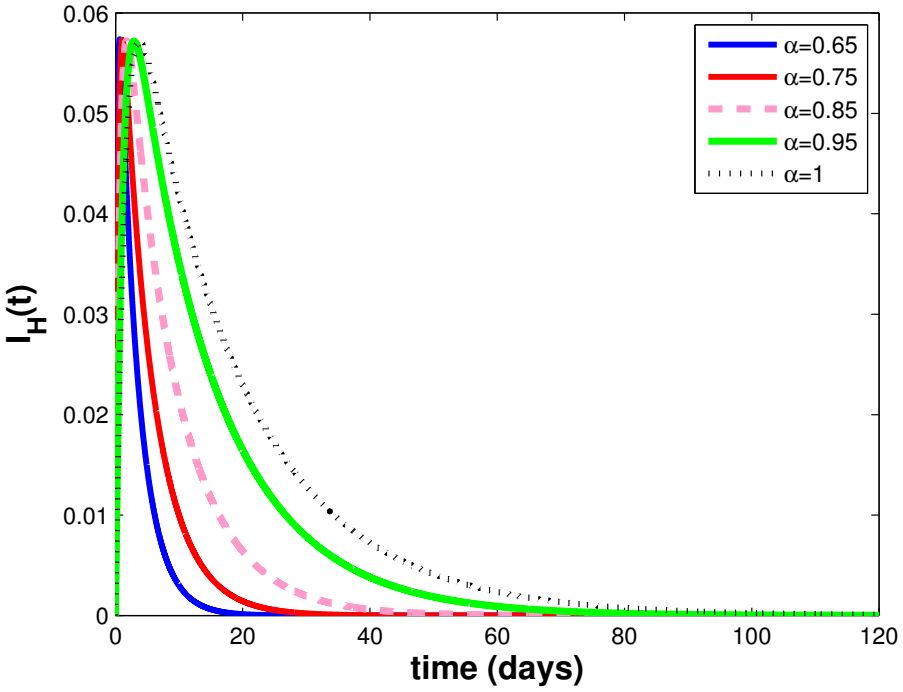
$$
\begin{aligned}
{}^{\mathcal{C}}D_t^{\alpha}\mathcal{L} &\leq [(S_L - S_L^{**}) + (S_H - S_H^{**}) + (I_L - I_L^{**}) + (I_H - I_H^{**}) + (R_L - R_L^{**}) \\
&\quad + (R_H - R_H^{**})]\left[{}^{\mathcal{C}}D_t^{\alpha}(S_L + S_H + I_L + I_H + R_L + R_H)\right] \\
&\quad + \left[(S_V - S_V^{**}) + (I_V - I_V^{**})\right]\left[{}^{\mathcal{C}}D_t^{\alpha}(S_V + I_V)\right] \\
&= [(S_L - S_L^{**}) + (S_H - S_H^{**}) + (I_L - I_L^{**}) + (I_H - I_H^{**}) + (R_L - R_L^{**}) \\
&\quad + (R_H - R_H^{**})][\Lambda_H - \mu_H(S_L + S_H + I_L + I_H + R_L + R_H) - \delta I_L - \phi I_H] \\
&\quad + \left[(S_V - S_V^{**}) + (I_V - I_V^{**})\right](\Lambda_V - \mu_V(S_V + I_V)) \\
&\leq -\mu_H[(S_L - S_L^{**}) + (S_H - S_H^{**}) + (I_L - I_L^{**}) + (I_H - I_H^{**}) + (R_L - R_L^{**}) \\
&\quad + (R_H - R_H^{**})]\left((S_L + S_H + I_L + I_H + R_L + R_H) - \tfrac{\Lambda_H}{\mu_H}\right) \\
&\quad - \mu_V\left[(S_V - S_V^{**}) + (I_V - I_V^{**})\right]\left((S_V + I_V) - \tfrac{\Lambda_V}{\mu_V}\right) \\
&\leq -\mu_H[(S_L - S_L^{**}) + (S_H - S_H^{**}) + (I_L - I_L^{**}) + (I_H - I_H^{**}) + (R_L - R_L^{**}) \\
&\quad + (R_H - R_H^{**})]^2 - \mu_V\left[(S_V - S_V^{**}) + (I_V - I_V^{**})\right]^2.
\end{aligned}
$$

Hence, the Caputo fractional time derivative ${}^{\mathcal{C}}D_t^{\alpha}\mathcal{L}(\mathcal{G}(t))$ is negative semidefinite, that is, ${}^{\mathcal{C}}D_t^{\alpha}\mathcal{L} \leq 0$ with equality if and only if $S_L = S_L^{**}$, $S_H = S_H^{**}$, $I_L = I_L^{**}$, $I_H = I_H^{**}$, $R_L = R_L^{**}$, $R_H = R_H^{**}$, $S_V = S_V^{**}$, and $I_V = I_V^{**}$. This implies that the largest invariant set in $\{\mathcal{G}(t) \in \mathfrak{D} \mid {}^{\mathcal{C}}D_t^{\alpha}\mathcal{L}(\mathcal{G}(t)) = 0\}$ is the singleton $\{\varepsilon^{**}\}$. It follows by LaSalle's invariance principle [45] that the endemic equilibrium point ε^{**} is globally asymptotically stable. □

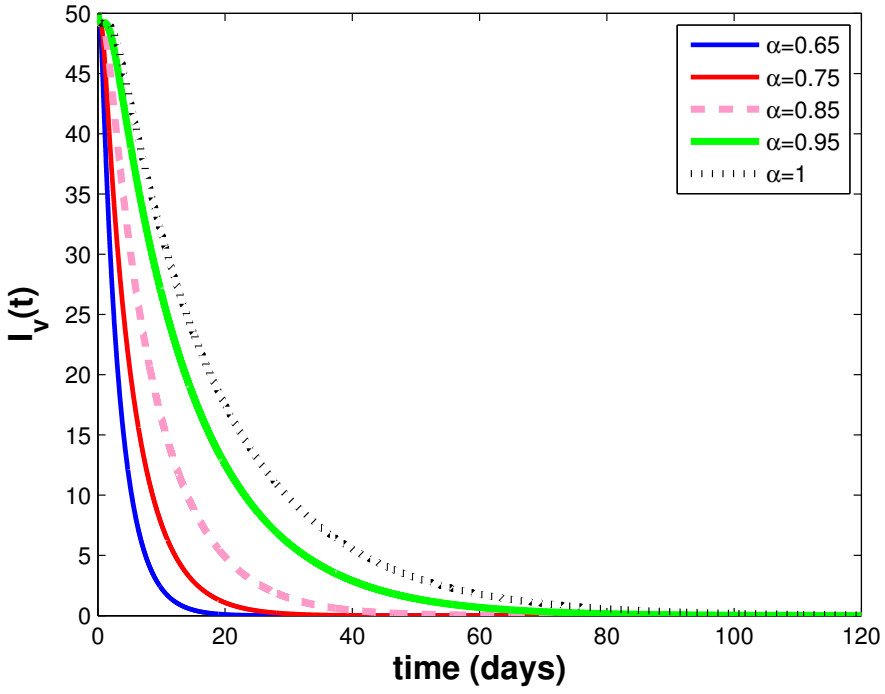
3.3. Simulations and Discussion

To visualize the overall behavior of the fractional-order system (4), the generalized Euler's method discussed in [46,47] was used. Specifically, in Figure 1, the values of the fractional order parameter α were allowed to vary in the interval $0 < \alpha \leq 1$ at the basic reproduction number $\mathcal{R}_0 = 0.7266$. It can be observed that as the memory increased, the size of the high social class infectious human population reduced and converged to the malaria-free equilibrium rapidly. In other words, a lower value of the fractional-order parameter α makes the convergence to the disease-free equilibrium faster when compared with a higer value of the fractional-order α. Similar behavior can be observed for the infectious vector population. In Figure 2, when the basic reproduction number was greater than unity, that is $\mathcal{R}_0 = 2.2978$, it can be observed that the decrease in the value of the fractional order parameter α increased the convergence of the high social class infectious human population to the endemic equilibrium, thus implying that the presence of the memory enabled the fractional-order social hierarchy-structured system to stabilize more quickly when compared to a memoryless system where $\alpha = 1$. Similar behavior can be observed in Figure 2b for the infectious vector population when $\mathcal{R}_0 > 1$.

In another development, Figure 3 shows the global asymptotic behavior of the fractional-order system (4) for $\alpha = 0.85$ at different values of initial data. In particular, when $\mathcal{R}_0 = 0.7266 < 1$, it is shown that every trajectory of the infectious vector population, regardless of the initial conditions, tended to the disease-free equilibrium. This corroborates the theoretical result established in Theorem 5 for the global asymptotic stability of the disease-free equilibrium. Conversely, in the same Figure 3, when $\mathcal{R}_0 = 2.2978 > 1$, it can be seen that every solution originating at different sizes of the infectious vector population converged asymptotically to the endemic equilibrium point. This is in support of Theorem 6.

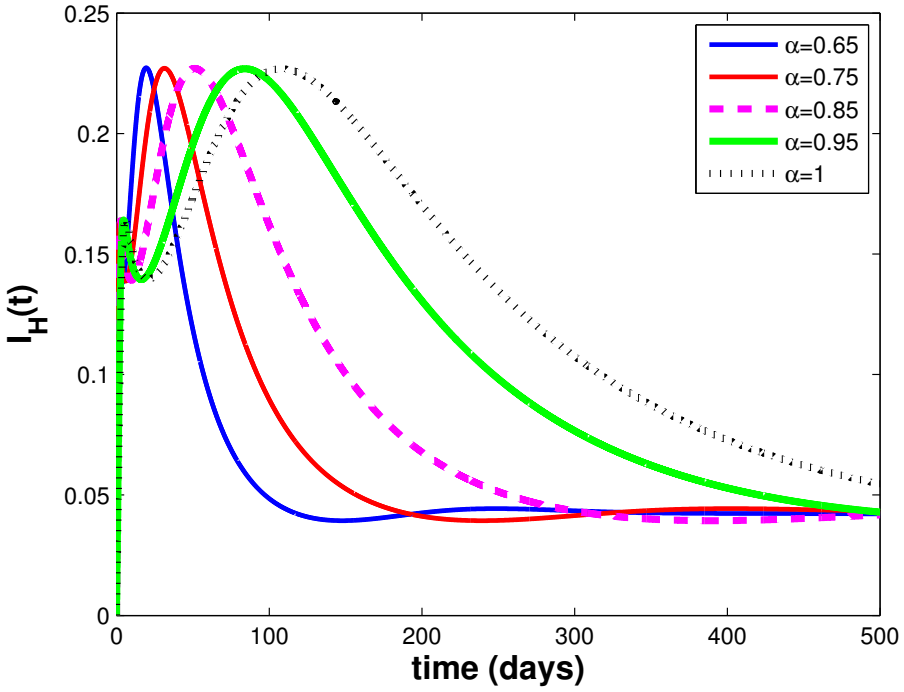


(**a**)

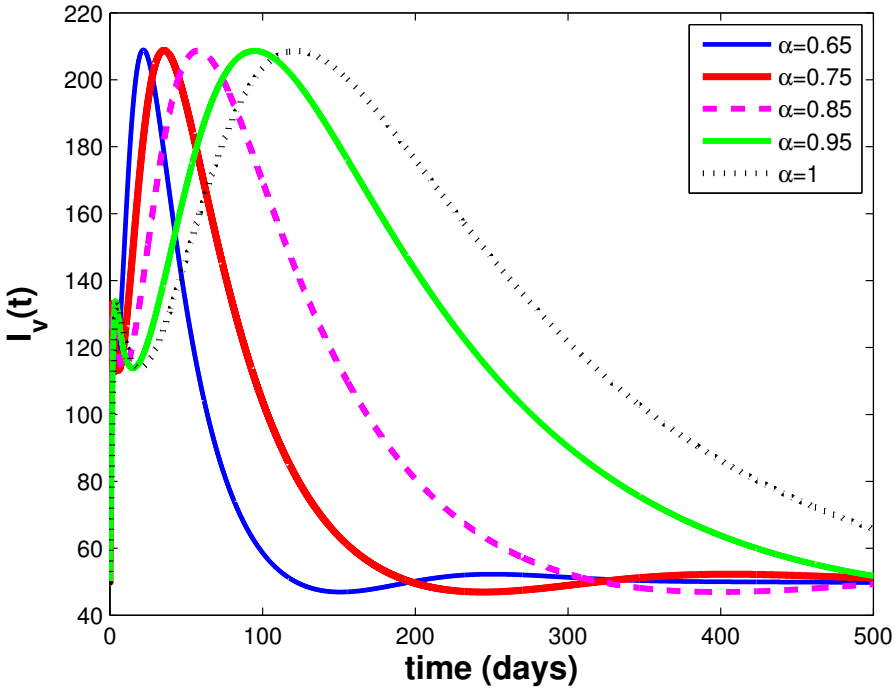


(**b**)

Figure 1. (**a**) Varying effects of the fractional-order parameter α on the high social class infectious human population. (**b**) Varying effects of the fractional-order parameter α on the infectious vector population. In both cases, $r = 0.2$, $\Lambda_H = 0.11$, $\beta_1 = 0.001$, $\beta_2 = 0.002$, $\Lambda_V = 100$, $b = 0.003$, $\theta = 0.65$, $\mu_H = 0.0000548$, $\gamma = 0.82$, $\delta = 0.7$, $\sigma_L = 0.95$, $\alpha = 0.88$, $\phi = 0.5$, $\sigma_H = 0.0065$, and $\mu_V = 0.067$ so that $\mathcal{R}_0 = 0.7266 < 1$.



(**a**)



(**b**)

Figure 2. (**a**) Varying effects of the fractional-order parameter α on the high social class infectious human population. (**b**) Varying effects of the fractional-order parameter α on the infectious vector population. Using the same parameter values as in Figure 1, except for $\beta_2 = 0.02$, so that $\mathcal{R}_0 = 2.2978 > 1$.

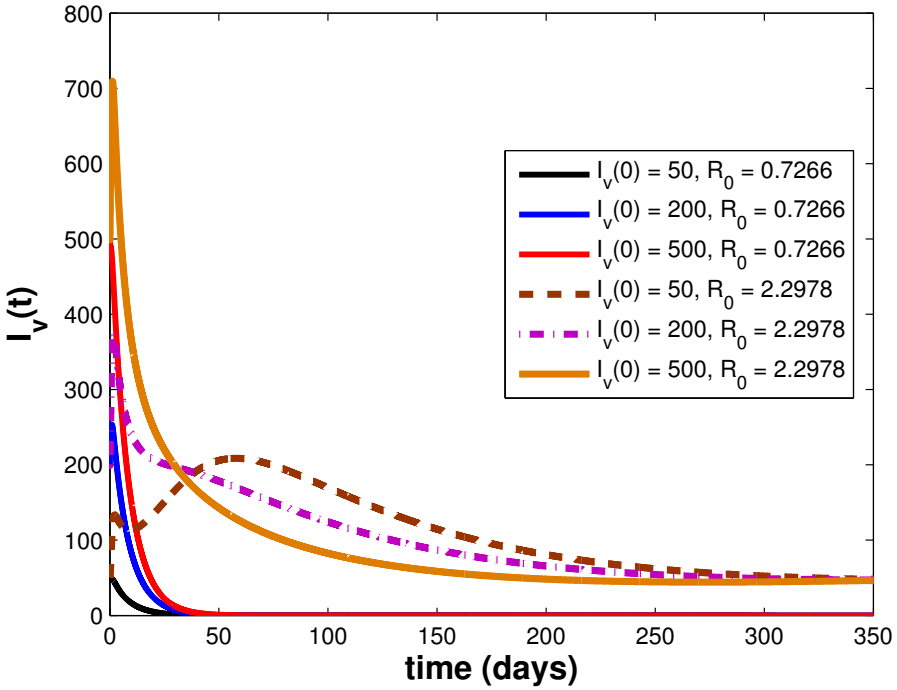


Figure 3. Global asymptotic stability of the fractional-order social hierarchy-structured malaria model (4), at $\alpha = 0.85$, around the disease-free equilirium and endemic equilirium when $\mathcal{R}_0 = 0.7266$ and $\mathcal{R}_0 = 2.2978$, respectively.

4. Conclusions

In this work, fractional calculus has been applied to describe the transmission dynamics of malaria in a social hierarchy-structured population with memory effects. The formulated fractional-order model is a system of differential equations with a Caputo derivative operator. The well-posed nature of the model was established via the generalized mean value theorem for the positivity of bounded solutions, and the Banach fixed point theory was employed for the existence and uniquesness of solutions. The global asymptotic stabilities of both disease-free and endemic equilibria of the fractional-order model were investigated by extending the methods in the classical calculus to the Caputo fractional calculus, and the theoretical results were graphically illustrtated. Consequently, it was proved that at $\mathcal{R}_0 < 1$, the fractional-order social hierarchy-structured malaria model has a globally asymptotically stable disease-free equilibrium where solutions at different initial values converge to. It was also proved that every solution of the model initiating at various values tends to the endemic equilibrium asymptotically when $\mathcal{R}_0 > 1$.

In addition, the effects of various values of the fractional order $0 < \alpha \leq 1$ were tested on the behavior of the fractional-order social hierarchy-structured malaria model. It was revealed that an increase in the fractional order α results in slow convergence of the state solutions of the system to both the disease-free and endemic equilibria. Hence, it was established that solutions of the fractional-order system with values of $\alpha < 1$ stabilize more rapidly than a memoryless system with $\alpha = 1$. Therefore, it can be stated that the presence of memory in a dynamical system operates as a control parameter, which enhances the convergence of the solutions. This underscores the importance of fractional calculus in modeling dynamical systems with memory.

In the presence of real data, fractional-order models can have more degree of freedom for exploring disease dynamics than the classical models. This is so because the fractional order can be employed as a fit parameter to improve the agreement with the real data. Thus, for a more realistic approach and accurate prediction of malaria disease spread in the population, it is worth considering the robust data-driven analysis of the fractional-

order model for malaria dynamics. Also worthy of consideration is modeling malaria dynamics via a reaction–diffusion system in order to describe how the disease spreads through contact between host–vector interactions and spatial movement in a heterogeneous environment. These are the limitations of the present work, which can be explored as future considerations.

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