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ENHANCING FRACTIONAL MODEL FOR DENGUE TRANSMISSION INCORPORATING VERTICAL TRANSMISSION AND VECTOR CONTROL

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Abstract Dengue fever continues to be a major global health concern, particularly in tropical and subtropical regions. This research develops an updated fractional-order dengue transmission model that incorporates vector control methods with vertical transmission in mosquitoes. Memory effects are considered in the model formulation, which are examined using the Caputo fractional derivative. Through vertical transmission, Infected mosquitoes transmit the virus to their offspring, which keeps the infection active when humans are not available as hosts. Spraying insecticides together with biological control measures is part of vector control efforts that help assess how well the disease prevalence decreases. The Banach fixed-point theorem provides a basis to prove existence and uniqueness solutions. The mathematical model uses the basic reproduction number ($\mathscr{R}_0$) to determine disease persistence, while stability conditions depend on equilibrium points. The investigation of local stability uses fractional-order stability theory together with a suitable Lyapunov function to establish global stability. The disease dynamics display changes through numerical results where fractional order variations combine with vector control measures. The simulation data demonstrates that vertical disease transmission functions as a crucial factor for maintaining dengue outbreaks, but strong vector control programs can significantly reduce spreading rates. The presented model functions as a foundation to establish optimal preventive strategies against dengue fever. The subsequent part of this work investigates optimal control models.

Keywords Dengue transmission, fractional-order model, vertical transmission, vector control, basic reproduction number, stability analysis, Banach fixed point theorem, numerical simulations.

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1. Introduction

Due to its complexity, the disease affects approximately 3.9 billion people worldwide, of which 129 countries are already vulnerable to the dengue virus [39]. Due to population growth, climate change, and inadequate vector control initiatives, the number of cases rose by almost 40%

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between 2020 and 2023 [18, 30]. The primary vector of dengue, Aedes aegypti, is found in tropical and subtropical urban centres. It has also adapted to domestic life by spending the day in houses and laying eggs in buckets and other containers [2]. Although vaccine development has progressed, vector control is still required because the primary vaccines now available on the market are ineffective against all four DENV serotypes [14]. In addition, the World Health Organization estimates that dengue is a neglected tropical illness that costs more than $8.9 billion in lost income annually [27].

Vertical transmission (VT), the transfer of DENV from mother mosquitoes to their progeny, has become an important phenomenon explaining the persistence of dengue. Laboratory work has confirmed VT rates of 15-55% in Aedes aegypti species, depending on viral load and temperature [23,38]. Evidence from Brazil shows that VT sustains viral reservoirs in between-epidemic periods, allowing quick resurgence after the post-monsoon period [12]. Desiccation-resistant infected eggs that are capable of dormancy for several months aggravate eradication attempts [11]. Shifts in genomic analysis suggest that vertically transmitted strains have mutations facilitating increased transmissibility, which leads to suspicion of evolutionary benefits [8]. Quite remarkable, however, is that only 12% of the available dengue models consider VT despite the expansion of this phenomenon [7].

While modeling the projection of dengue outbreaks, integer-order differential equations are the dominant tools of choice, but they clearly lack the capability to encompass latent spatial heterogeneities of dengue transmission. Besides non-integer order derivatives, is the framework best suited to system's with time delays as well as long-range interactions [26]. Caputo fractional derivatives, in particular, capture the subdiffusive behavior of mosquitoes and delayed immune responses of the host [6, 32]. More recent uses in forecasting Zika and Chikungunya outbreaks have outperformed classical-based models [9]. For dengue, fractional operators yield an 18–32 % improvement in estimating the effective reproduction number (R_e) due to spatial heterogeneities [19]. Nonetheless, VT is missed in existing fractional models, which oversimplify vector control [24].

Traditional vector control methods depend on the use of pyrethroid insecticides, which is ineffective considering that resistance mutations are above 78% in global Aedes populatios [31]. Adding Wolbachia to mosquitoes lowers transmission rates in trial locations, but issues with scalability and acceptance remain [16]. Gene drives built with CRISPR technology are promising for population control, Such methods are in an ethical uncertainty due to the possibility of ecological changes [35]. New models incorporate the idea of insecticide pulses timed to rainfall in a region, thus making the exploitation of habitats more efficient [1]. Still, these models are not consistent with the fractional order dynamics and VT control, which is an oversight in resource distribution [28].

The purpose of this research study is to address the following gaps: (1) The lack of consideration of VT in dengue fractional models, (2) the oversimplified depiction of control measures, and most importantly, (3) validation deficits against empiric outbreak data. In this paper, we create a Caputo fractional-order model with time-varying control laws, incorporating VT through a transovarial transmission rate ϕ. The model integrates human and vector movement, through a fractional SEIR-SEI model with memory effects represented by a Caputo derivative of order $\alpha \in (0, 1]$. The stability analysis produces threshold conditions suitable for controlling an epidemic, while the optimal control problem determines the most economical mixed strategies of insecticide spraying.

Dengue models need to be improved for three main reasons: (1) The role of VT in maintaining outbreaks in the face of aggressive vector control [23], (2) the attempt towards a target set by

the WHO in 2017 of a 50% reduction in dengue mortality by 2025 [39], and (3) the need for proactive measures regarding climatic spread of Aedes species [26]. This paper brings together fractional calculus, VT dynamics, and time-variant controls to propose a model that allows public health agents to predict outbreaks. The simulations were performed, and the model was validated against Brazilian and Indonesian outbreak data from 2022 to 2024 [13, 17], providing useful information for integrated vector management.

2. Fractional preliminaries

Definition 2.1. [25] Let $\alpha > 0$. The definition of the Caputo fractional derivative applies to a function $f(t)$ under the condition $f(t) \in C^n([a,b],\mathscr{R})$ when $n = \lceil \alpha \rceil$.

$$ {}^{C}D^{\alpha}_{a^+} f(t) = \frac{1}{\Gamma(n-\alpha)} \int_a^t \frac{f^{(n)}(\tau)}{(t-\tau)^{\alpha-n+1}} d\tau. \tag{2.1} $$

Here $\Gamma(\cdot)$ represents the Euler gamma function. The derivative introduced by Caputo differs from Riemann-Liouville derivatives because it needs $f(t)$ to have integer order derivatives to work with initial condition settings.

Definition 2.2. [4] The Mittag-Leffler function presents $E_\alpha, \beta(z)$ as a generalized exponent function through this presentation:

$$ E_{\alpha,\beta}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\alpha k + \beta)}. \tag{2.2} $$

The conditions enable the use of positive values for $\alpha, \beta > 0$. When β equals 1 the expression becomes $E_\alpha(z)$ that holds major importance in fractional differential equation solutions.

Theorem 2.1. [20] *A continuous function $f(t,u)$ exists throughout $[a,b] \times \mathbb{R}$ and fulfills this Lipschitz condition regarding u.*

$$ |f(t,u_1) - f(t,u_2)| \leq \mathscr{L}|u_1 - u_2|. \tag{2.3} $$

For some value $\mathscr{L} > 0$. The initial value problem appears below for the specified fractional value $\mathscr{L} > 0$.

$$ {}^{C}D^{\alpha}_{a^+} u(t) = f(t,u(t)), \quad u^{(k)}(a) = u_k, \quad k = 0,1,\ldots,n-1, \tag{2.4} $$

has a unique solution on $[a,b]$.

Theorem 2.2. [4] *We obtain the Laplace transform of $f(t)$ according to the Caputo definition through the following formula:*

$$ \mathcal{L}\left[{}^{C}D^{\alpha}_{0^+} f(t)\right](s) = s^{\alpha}\mathscr{F}(s) - \sum_{k=0}^{n-1} s^{\alpha-k-1} f^{(k)}(0), \tag{2.5} $$

where $\mathscr{F}(s) = \mathscr{L}\{f(t)\}(s)$. Properies of the fractional transform allow for more efficient solutions of linear fractional differential equations with constant coefficients.

Theorem 2.3. [3] *Suppose $\alpha \in (n-1, n)$. Then the Caputo derivative of $t^\beta, \beta > n-1$, is expressed as follows:*

$$ {}^{C}D^{\alpha}_{0^+} t^{\beta} = \frac{\Gamma(\beta+1)}{\Gamma(\beta-\alpha+1)} t^{\beta-\alpha}. \tag{2.6} $$

This expands upon the integer-order derivative power rule.

3. Model formulation

We built an advanced fractional-order SEIR-SI model that represents vector control and vertical transmission dynamics of mosquitoes within the population model. The human groups comprise four classes: $\mathscr{S}_h$ represents the susceptible while the exposed population uses $\mathscr{E}_h$ and the infected $\mathscr{I}_h$, $\mathscr{R}_h$ stands for recovered hosts. The mosquito population consists of two biological groups where $\mathscr{S}_v$ demonstrates mosquito susceptibility and $\mathscr{I}_v$ indicates infected mosquito. These fractional differential equation systems represent the model according to the following mathematical formulation:

$$\begin{cases} D^{\alpha}\mathscr{S}_h(t) = \Lambda_h - \beta_h \mathscr{S}_h(t)\dfrac{\mathscr{I}_v(t)}{\mathscr{N}_v} - \mu_h \mathscr{S}_h(t), \\ D^{\alpha}\mathscr{E}_h(t) = \beta_h \mathscr{S}_h(t)\dfrac{\mathscr{I}_v(t)}{\mathscr{N}_v} - (\sigma_h + \mu_h)\mathscr{E}_h(t), \\ D^{\alpha}\mathscr{I}_h(t) = \sigma_h \mathscr{E}_h(t) - (\gamma_h + \mu_h)\mathscr{I}_h(t), \\ D^{\alpha}\mathscr{R}_h(t) = \gamma_h \mathscr{I}_h(t) - \mu_h \mathscr{R}_h(t), \\ D^{\alpha}\mathscr{S}_v(t) = \Lambda_v(1-p) - \beta_v \mathscr{S}_v(t)\dfrac{\mathscr{I}_h(t)}{\mathscr{N}_h} - \mu_v \mathscr{S}_v(t) - c\mathscr{S}_v(t), \\ D^{\alpha}\mathscr{I}_v(t) = {}_vp + \beta_v \mathscr{S}_v(t)\dfrac{\mathscr{I}_h(t)}{\mathscr{N}_h} - \mu_v \mathscr{I}_v(t) - c\mathscr{I}_v(t), \end{cases} \tag{3.1}$$

where D^{α} denotes the Caputo fractional derivative of order α $0 < \alpha \leq 1$, and the parameters are defined as follows:

The basic aspects of the dynamics of dengue fever spread among humans and mosquitoes are of just this type, this model represents. Recruitment rate of humans, Λ_h, measures the entry of new individuals to the susceptible class and determines the composition of the human population. In fact, dynamics of mosquitoes, which are controlled by its recruitment rate of mosquitoes Λ_v, can be seen in the population of mosquitoes as well. Each disease transmission rate β_h the probability that a susceptible human becomes infected due to an infected mosquito, or β_v the probability that an infected human passes the disease to a disease susceptible mosquito, is included.

These include an infected human with a rate of recovery γ_h and a human that becomes ineffective when in contact with the virus at a rate σ_h; for the human. Human and mosquito population respectively have natural life spans given by their natural death rates μ_h and μ_v and both, human and mosquito populations have a natural life spans and as such. Since the proportion of infected mosquitoes has a direct effect on the propagation speed of the disease, it is subsequently bounded to the proportion of vertically infected mosquitoes, p, which is synonymous with the number of infected females able to transmit the virulent to the next generation.

The mosquito population control parameter vector (c) includes insecticide spraying, environmental control and biological control of the mosquitoes. In this case, you therefore further stratify the human population ($\mathscr{N}_h$) into four categories: Susceptible ($\mathscr{S}_h$), exposed ($\mathscr{E}_h$), infected ($\mathscr{I}_h$) and recovered ($\mathscr{R}_h$). Similarly, the total number of mosquitoes ($\mathscr{N}_v$) comprise non-infected ($\mathscr{S}_v$) and infected ($\mathscr{I}_v$) mosquitoes. The framework in terms of these key parameters, will inform the mode of dengue transmission and evaluate the control measures on the disease epidemiology.

4. Model analysis

Theorem 4.1. [5,10] *The fractional differential equation system of equations 3.1 with compartments $\mathscr{S}_h(t)$, $\mathscr{E}_h(t)$, $\mathscr{I}_h(t)$, $\mathscr{R}_h(t)$, $\mathscr{S}_v(t)$, and $\mathscr{I}_v(t)$ has initial values $\mathscr{S}_h(0), \mathscr{E}_h(0), \mathscr{I}_h(0), \mathscr{R}_h(0), \mathscr{S}_v(0), \mathscr{I}_v(0)$ which are all non-negative. Consequently, for all $t > 0$, solutions to the system 3.1 remain non-negative, and the total populations $\mathscr{N}_h(t)$ and $\mathscr{N}_v(t)$ are bounded.*

Proof. With the given initial conditions, we shall demonstrate non-negativity and boundedness for each compartment of 3.1.

The first equation of model 3.1 $\mathscr{S}_h(t)$ is:

$$D^{\alpha}\mathscr{S}_h(t) = \Lambda_h - \beta_h \mathscr{S}_h(t)\frac{\mathscr{I}_v(t)}{\mathscr{N}_v} - \mu_h \mathscr{S}_h(t). \tag{4.1}$$

It will be written as:

$$D^{\alpha}\mathscr{S}_h(t) \geq -\left(\beta_h \frac{\mathscr{I}_v(t)}{\mathscr{N}_v} + \mu_h\right)\mathscr{S}_h(t). \tag{4.2}$$

The solution $\mathscr{S}_h(t)$ is:

$$\mathscr{S}_h(t) \geq \mathscr{S}_h(0)\mathcal{E}_{\alpha}\left(-\left(\beta_h \frac{\mathscr{I}_v(t)}{\mathscr{N}_v} + \mu_h\right)t^{\alpha}\right). \tag{4.3}$$

Consequently, given that $\mathscr{S}_h(0) > 0$ and $\mathcal{E}_{\alpha}(\cdot) > 0$, it follows that $\mathscr{S}_h(t) > 0$ for all $t > 0$. This indicates that $\mathscr{S}_h(t)$ is non-negative for every $t > 0$.

Based on this process, all remaining compartments are:

$$\begin{aligned}
&\mathscr{E}_h(t) \geq \mathscr{E}_h(0)\mathcal{E}_{\alpha}\left(-(\sigma_h + \mu_h)t^{\alpha}\right),\\
&\mathscr{I}_h(t) \geq \mathscr{I}_h(0)\mathcal{E}_{\alpha}\left(-(\gamma_h + \mu_h)t^{\alpha}\right),\\
&\mathscr{R}_h(t) \geq \mathscr{R}_h(0)\mathcal{E}_{\alpha}\left(-\mu_h t^{\alpha}\right),\\
&\mathscr{S}_v(t) \geq \mathscr{S}_v(0)\mathcal{E}_{\alpha}\left(-\left(\beta_v \frac{\mathscr{I}_h(t)}{\mathscr{N}_h} + \mu_v + c\right)t^{\alpha}\right),\\
&\mathscr{I}_v(t) \geq \mathscr{I}_v(0)\mathcal{E}_{\alpha}\left(-(\mu_v + c)t^{\alpha}\right),
\end{aligned} \tag{4.4}$$

since $\mathscr{E}_h(0) > 0$, $\mathscr{I}_h(0) > 0$, $\mathscr{R}_h(0) > 0$, $\mathscr{S}_v(0) > 0$, $\mathscr{I}_v(0) > 0$ and $\mathcal{E}_{\alpha}(\cdot) > 0$, we have $\mathscr{S}_h(t) > 0$ for all $t > 0$. Hence, $\mathscr{E}_h(0) > 0$, $\mathscr{I}_h(0) > 0$, $\mathscr{R}_h(0) > 0$, $\mathscr{S}_v(0) > 0$ and $\mathscr{I}_v(0) > 0$ are non-negative for all $t > 0$.

To calculate the boundedness of model (3.1), The total human population is:

$$\mathscr{N}_h(t) = \mathscr{S}_h(t) + \mathscr{E}_h(t) + \mathscr{I}_h(t) + \mathscr{R}_h(t). \tag{4.5}$$

The solution of equation 4.5 is:

$$\mathscr{N}_h(t) \leq \frac{\Lambda_h}{\mu_h} + \left(\mathscr{N}_h(0) - \frac{\Lambda_h}{\mu_h}\right)\mathcal{E}_{\alpha}\left(-\mu_h t^{\alpha}\right). \tag{4.6}$$

For this $t \to \infty$, we obtain:

$$\limsup_{t\to\infty} \mathscr{N}_h(t) \leq \frac{\Lambda_h}{\mu_h}. \tag{4.7}$$

Hence, $\mathscr{N}_h(t)$ is bounded.

Now the total population of vectors:

$$\mathscr{N}_v(t) = \mathscr{S}_v(t) + \mathscr{I}_v(t), \tag{4.8}$$

for this

$$D^\alpha \mathscr{N}_v(t) = \Lambda_v - \mu_v \mathscr{N}_v(t). \tag{4.9}$$

The solution of equation 4.9 is obtained as:

$$\mathscr{N}_v(t) \leq \frac{\Lambda_v}{\mu_v} + \left(\mathscr{N}_v(0) - \frac{\Lambda_v}{\mu_v}\right) \mathcal{E}_\alpha\left(-\mu_v t^\alpha\right), \tag{4.10}$$

as $t \to \infty$, we obtain:

$$\limsup_{t\to\infty} \mathscr{N}_v(t) \leq \frac{\Lambda_v}{\mu_v}. \tag{4.11}$$

Hence, $\mathscr{N}_v(t)$ is bounded [29]. □

Disease-free equilibrium (DFE) points

In order to determine the DFE points for the provided system of fractional differential equations, it is necessary to eliminate the infected compartments and compute the other variable(s) available. The infected compartments for this system are $\mathscr{E}_h(t)$, $\mathscr{I}_h(t)$, and $\mathscr{I}_v(t)$.

Finding the DFE means identifying a point at which the affected population is null, thus, establishing that $\mathscr{E}_h(t) = 0, \mathscr{I}_h(t) = 0, \mathscr{I}_v(t) = 0$.

Let us denote DFE by $(\mathscr{S}_h^0, \mathscr{E}_h^0, \mathscr{I}_h^0, \mathscr{R}_h^0, \mathscr{S}_v^0, \mathscr{I}_v^0)$ in which $\mathscr{E}_h^0 = 0$, $\mathscr{I}_h^0 = 0$, and $\mathscr{I}_v^0 = 0$.

The system of equations 3.1 becomes:

$$\begin{cases} D^\alpha \mathscr{S}_h(t) = \Lambda_h - \mu_h \mathscr{S}_h(t), \\ D^\alpha \mathscr{E}_h(t) = 0, \\ D^\alpha \mathscr{I}_h(t) = 0, \\ D^\alpha \mathscr{R}_h(t) = -\mu_h \mathscr{R}_h(t), \\ D^\alpha \mathscr{S}_v(t) = \Lambda_v(1-p) - \mu_v \mathscr{S}_v(t) - c\mathscr{S}_v(t), \\ D^\alpha \mathscr{I}_v(t) = 0. \end{cases} \tag{4.12}$$

When a system is in equilibrium, the following derivatives are zero: $D^\alpha \mathscr{S}_h(t)$, $D^\alpha \mathscr{E}_h(t)$, $D^\alpha \mathscr{I}_h(t)$, $D^\alpha \mathscr{R}_h(t)$, $D^\alpha \mathscr{S}_v(t)$, and $D^\alpha \mathscr{I}_v(t)$.

$$\begin{cases} 0 = \Lambda_h - \mu_h \mathscr{S}_h^0 \implies \mathscr{S}_h^0 = \dfrac{\Lambda_h}{\mu_h}, \\ 0 = 0 \implies \mathscr{E}_h^0 = 0, \\ 0 = 0 \implies \mathscr{I}_h^0 = 0, \\ 0 = -\mu_h \mathscr{R}_h^0 \implies \mathscr{R}_h^0 = 0, \\ 0 = \Lambda_v(1-p) - \mu_v \mathscr{S}_v^0 - c\mathscr{S}_v^0 \implies \mathscr{S}_v^0 = \dfrac{\Lambda_v(1-p)}{\mu_v + c}, \\ 0 = 0 \implies \mathscr{I}_v^0 = 0. \end{cases}$$

The DFE is given by:

$$(\mathscr{S}_h^0, \mathscr{E}_h^0, \mathscr{I}_h^0, \mathscr{R}_h^0, \mathscr{S}_v^0, \mathscr{I}_v^0) = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, \frac{\Lambda_v(1-p)}{\mu_v + c}, 0\right).$$

Basic reproductive number $\mathscr{R}_0$

Using the Next Generation Matrix method teams first follow these steps to calculate $\mathscr{R}_0$ value. The three infected populations connect to separate parts of the Transmission Matrix $\mathscr{T}$ and Transition Matrix $\mathscr{P}$.

$$\mathscr{T} = \begin{bmatrix} 0 & 0 & \dfrac{\beta_h \mathscr{S}_h^0}{\mathscr{N}_v} \\ 0 & 0 & 0 \\ 0 & \dfrac{\beta_v \mathscr{S}_v^0}{\mathscr{N}_h} & 0 \end{bmatrix}, \quad \mathscr{P} = \begin{bmatrix} \sigma_h + \mu_h & 0 & 0 \\ -\sigma_h & \gamma_h + \mu_h & 0 \\ 0 & 0 & \mu_v + c \end{bmatrix}. \tag{4.13}$$

Next-Generation Matrix ($\mathscr{K} = \mathscr{T}\mathscr{P}^{-1}$):

$$\mathscr{K} = \begin{bmatrix} 0 & 0 & \dfrac{\beta_h \mathscr{S}_h^0}{\mathscr{N}_v(\mu_v + c)} \\ 0 & 0 & 0 \\ \dfrac{\beta_v \mathscr{S}_v^0 \sigma_h}{\mathscr{N}_h(\sigma_h + \mu_h)(\gamma_h + \mu_h)} & \dfrac{\beta_v \mathscr{S}_v^0}{\mathscr{N}_h(\gamma_h + \mu_h)} & 0 \end{bmatrix}. \tag{4.14}$$

Now Spectral Radius of $\mathscr{K}$:

$$\mathscr{R}_0 = \sqrt{\frac{\beta_h \beta_v \mathscr{S}_h^0 \mathscr{S}_v^0 \sigma_h}{\mathscr{N}_h \mathscr{N}_v (\sigma_h + \mu_h)(\gamma_h + \mu_h)(\mu_v + c)}}.$$

By substituting $\mathscr{N}_h = \mathscr{S}_h^0 = \frac{h}{\mu_h}$ and $\mathscr{N}_v = \mathscr{S}_v^0 = \frac{v(1-p)}{\mu_v + c}$.
After simplification we obtained the $\mathscr{R}_0$:

$$\mathscr{R}_0 = \sqrt{\frac{\beta_h \beta_v \sigma_h}{(\sigma_h + \mu_h)(\gamma_h + \mu_h)(\mu_v + c)}}. \tag{4.15}$$

Endemic equilibrium points

We need to calculate the endemic equilibrium solution for equation (3.1). The endemic equilibrium holds if and only if $\mathscr{R}_0 > 1$. Endemic equilibrium values tell us both how long the disease exists and how this period relates to $\mathscr{R}_0^2 - 1$. The system parameters and basic reproductive number $\mathscr{R}_0$ together define how their explicit expressions work.

$$\begin{aligned} \mathscr{S}_h^* &= \frac{\Lambda_h}{\mu_h \mathscr{R}_0^2}, \\ \mathscr{E}_h^* &= \frac{(\gamma_h + \mu_h)}{\sigma_h} \mathscr{I}_h^*, \\ \mathscr{I}_h^* &= \frac{\mu_h(\sigma_h + \mu_h)(\gamma_h + \mu_h)}{\beta_h \beta_v \sigma_h} (\mathscr{R}_0^2 - 1), \\ \mathscr{R}_h^* &= \frac{\gamma_h}{\mu_h} \mathscr{I}_h^*, \\ \mathscr{S}_v^* &= \frac{\Lambda_v(1-p)}{\mu_v + c} \cdot \frac{1}{\mathscr{R}_0^2}, \\ \mathscr{I}_v^* &= \frac{\Lambda_v p}{\mu_v + c} (\mathscr{R}_0^2 - 1). \end{aligned} \tag{4.16}$$

Stability analysis

Theorem 4.2. [20] *The point DFE is considered to be LAS if* $\mathscr{R}_0 < 1$ *and unstable if* $\mathscr{R}_0 > 1$.

Proof. To construct the Jacobian matrix from system equation 3.1 at the DFE point to evaluate local stability. We use this DFE equilibrium to compute system equation partial derivatives before integrating them into the Jacobian matrix.

$$\mathscr{J}_0 = \begin{bmatrix} -\mu_h & 0 & 0 & 0 & 0 & 0 \\ 0 & -(\sigma_h+\mu_h) & 0 & 0 & 0 & 0 \\ 0 & \sigma_h & -(\gamma_h+\mu_h) & 0 & 0 & 0 \\ 0 & 0 & \gamma_h & -\mu_h & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_v - c & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\mu_v+c) \end{bmatrix}. \tag{4.17}$$

The characteristic Jacobian matrix is obtained from the determinant $\mathscr{J}_0 - \lambda\mathscr{I}$:

$$\det(\mathscr{J}_0 - \lambda\mathscr{I}) = \begin{vmatrix} -\mu_h-\lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & -(\sigma_h+\mu_h)-\lambda & 0 & 0 & 0 & 0 \\ 0 & \sigma_h & -(\gamma_h+\mu_h)-\lambda & 0 & 0 & 0 \\ 0 & 0 & \gamma_h & -\mu_h-\lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_v-c-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\mu_v+c)-\lambda \end{vmatrix} = 0. \tag{4.18}$$

Thus, the characteristic equation becomes:

$$(\mu_h+\lambda)(\sigma_h+\mu_h+\lambda)(\gamma_h+\mu_h+\lambda)(\mu_h+\lambda)(\mu_v+c+\lambda)^2 = 0. \tag{4.19}$$

Hence, the eigenvalues are:

$$\lambda_1 = -\mu_h, \quad \lambda_2 = -(\sigma_h+\mu_h), \quad \lambda_3 = -(\gamma_h+\mu_h), \quad \lambda_4 = -\mu_h, \quad \lambda_5 = -(\mu_v+c), \quad \lambda_6 = -(\mu_v+c). \tag{4.20}$$

The Routh-Hurwitz method tests system stability by analyzing the roots of the characteristic polynomial equation. Our evaluation approach meets the requirements of this particular polynomial relationship.

$$\mathscr{P}(\lambda) = (\mu_h+\lambda)(\sigma_h+\mu_h+\lambda)(\gamma_h+\mu_h+\lambda)(\mu_h+\lambda)(\mu_v+c+\lambda)^2. \tag{4.21}$$

In order for a system to remain stable, negative roots are imperative since all eigenvalues must have negative real parts.

- $\lambda_1 = -\mu_h$, which is negative if $\mu_h > 0$.
- $\lambda_2 = -(\sigma_h+\mu_h)$, which is negative if $\sigma_h + \mu_h > 0$.

- $\lambda_3 = -(\gamma_h + \mu_h)$, which is negative if $\gamma_h + \mu_h > 0$.
- $\lambda_4 = -\mu_h$, which is negative if $\mu_h > 0$.
- $\lambda_5 = -(\mu_v + c)$, which is negative if $\mu_v + c > 0$.
- $\lambda_6 = -(\mu_v + c)$, which is negative if $\mu_v + c > 0$.

Therefore, all system eigenvalues have a negative real part which assures that the DFE is locally asymptotically stable. □

Theorem 4.3. [36,37] *The DFE of the fractional order system is said to be locally asymptotically stable if the two conditions below are simultaneously satisfied:*

$tr(\mathscr{J}_0) < 0$ *and* $\det(\mathscr{J}_0) > 0$.

The conditions are met if and only if $\mathscr{R}_0 < 1$.

Proof. From the Jacobian matrix $\mathscr{J}_0$ given in (4.17):

In this instance, we can define the tr of a matrix as the sum of its diagonal elements. Thus, $\text{tr}(\mathscr{J}_0) = \mathscr{J}_{11} + \mathscr{J}_{22} + ... + \mathscr{J}_{66}$.

$$\begin{aligned}\text{tr}(\mathscr{J}) &= -\mu_h - (\sigma_h + \mu_h) - (\gamma_h + \mu_h) - \mu_h - (\mu_v + c) - (\mu_v + c)\\ &= -2\mu_h - (\sigma_h + \mu_h) - (\gamma_h + \mu_h) - 2(\mu_v + c).\end{aligned}$$

All terms are negative so we conclude that it is always true that $\text{tr}(\mathscr{J}_0) < 0$.

Computing the determinant of $\mathscr{J}_0$:

$$\det(\mathscr{J}_0) = \mu_h^2(\sigma_h + \mu_h)(\gamma_h + \mu_h)(\mu_v + c)^2 - \frac{\beta_h\beta_v\sigma_h S_h^0 S_v^0}{N_h N_v}. \tag{4.22}$$

To ensure stability, we need $\det(\mathscr{J}_0) > 0$, which implies that:

$$\mu_h^2(\sigma_h + \mu_h)(\gamma_h + \mu_h)(\mu_v + c)^2 > \frac{\beta_h\beta_v\sigma_h S_h^0 S_v^0}{N_h N_v}. \tag{4.23}$$

Putting DFE values in (4.23), It is simplified as:

$$\mu_h(\sigma_h + \mu_h)(\gamma_h + \mu_h)(\mu_v + c) > \beta_h\beta_v\sigma_h. \tag{4.24}$$

To divide both sides by $\mu_h(\sigma_h + \mu_h)(\gamma_h + \mu_h)(\mu_v + c)$:

$$1 > \frac{\beta_h\beta_v\sigma_h}{\mu_h(\sigma_h + \mu_h)(\gamma_h + \mu_h)(\mu_v + c)}. \tag{4.25}$$

Acknowledge that $\mathscr{R}_0 = \sqrt{\frac{\beta_h\beta_v\sigma_h}{\mu_h(\sigma_h+\mu_h)(\gamma_h+\mu_h)(\mu_v+c)}}$, we assume:

$$1 > \mathscr{R}_0^2 \quad \Longrightarrow \quad \mathscr{R}_0 < 1. \tag{4.26}$$

The DFE is LAS when $\mathscr{R}_0 < 1$. This outcome comes from the trace-determinant method and confirming conditions for stability [40]. □

The provided disease-free equilibrium (DFE) is globally stable concerning a vector-host disease model with a fractional derivative order. Lyapunov functions, LaSalle's Invariance Principle, and comparison theorems are employed, and DFE is achieved. Global stability is related to the basic reproduction number $\mathscr{R}_0$. Stability is reached if $\mathscr{R}_0 < 1$.

Theorem 4.4. [21] *The DFE is GAS in the feasible region* Ω *if* $\mathscr{R}_0 < 1$.

Proof. To construct the Lyapunov function:

$$\mathscr{L}(\mathscr{E}_h, \mathscr{I}_h, \mathscr{I}_v) = \mathscr{E}_h + \frac{\sigma_h + \mu_h}{\sigma_h}\mathscr{I}_h + \frac{\beta_h \mathscr{S}_h^0}{\mathscr{N}_v(\mu_v + c)}\mathscr{I}_v. \tag{4.27}$$

For this $\mathscr{S}_h^0 = \frac{\Lambda_h}{\mu_h}$, $\mathscr{N}_v = \mathscr{S}_v^0 = \frac{\Lambda_v(1-p)}{\mu_v+c}$. Taking the fractional derivative of (4.27):

$$\begin{aligned} D^\alpha \mathscr{L} = &\left[\beta_h \mathscr{S}_h \frac{\mathscr{I}_v}{\mathscr{N}_v} - (\sigma_h + \mu_h)\mathscr{E}_h\right] \\ &+ \frac{\sigma_h + \mu_h}{\sigma_h}\left[\sigma_h \mathscr{E}_h - (\gamma_h + \mu_h)\mathscr{I}_h\right] \\ &+ \frac{\beta_h \mathscr{S}_h^0}{\mathscr{N}_v(\mu_v + c)}\left[\beta_v \mathscr{S}_v \frac{\mathscr{I}_h}{\mathscr{N}_h} - (\mu_v + c)\mathscr{I}_v\right]. \end{aligned} \tag{4.28}$$

After the DFE values and simplify:

$$D^\alpha \mathscr{L} = \frac{(\sigma_h + \mu_h)(\gamma_h + \mu_h)}{\sigma_h}\left(\mathscr{R}_0^2 - 1\right)\mathscr{I}_h. \tag{4.29}$$

Since $\mathscr{R}_0 < 1$, we have $D^\alpha \mathscr{L} \leq 0$ with equality if and only if $\mathscr{I}_h = 0$. The DFE is globally stable. □

Theorem 4.5. [22] *When* $\mathscr{R}_0 < 1$, *all trajectories meet at the* DFE.

Proof. From $D^\alpha \mathscr{L} = 0$, it therefore follows that $\mathscr{I}_h = 0$. Substituting into the system gives:

$$D^\alpha \mathscr{E}_h = -(\sigma_h + \mu_h)\mathscr{E}_h, \quad D^\alpha \mathscr{I}_v = -(\mu_v + c)\mathscr{I}_v. \tag{4.30}$$

For this $\mathscr{E}_h \to 0$ and $\mathscr{I}_v \to 0$ and the other equations:

$$D^\alpha \mathscr{S}_h = \Lambda_h - \mu_h \mathscr{S}_h, \quad D^\alpha \mathscr{S}_v = \Lambda_v(1-p) - (\mu_v + c)\mathscr{S}_v. \tag{4.31}$$

This ensures the convergence towards DFE, $\mathscr{S}_h^0$ and $\mathscr{S}_v^0$. □

Theorem 4.6. [33] *When* R_0 *is less than* 1, *the infected compartments exhibit exponential decay.*

Proof. To construct a Jacobian matrix for infected compartments of model (3.1) at DFE points.

$$\mathscr{J}_1 = \begin{bmatrix} -(\sigma_h + \mu_h) & 0 & \frac{\beta_h \mathscr{S}_h^0}{\mathscr{N}_v} \\ \sigma_h & -(\gamma_h + \mu_h) & 0 \\ 0 & \frac{\beta_v \mathscr{S}_v^0}{\mathscr{N}_h} & -(\mu_v + c) \end{bmatrix}. \tag{4.32}$$

When $\mathscr{R}_0$ is less than one, the dominant eigenvalue λ meets the requirement $\mathrm{Re}(\lambda) < 0$. Through the comparison theorem:

$$\|(E_h, I_h, I_v)\| \leq \|(E_h(0), I_h(0), I_v(0))\| E_\alpha(-\kappa t^\alpha), \tag{4.33}$$

where $\kappa > 0$, this demonstrates that the infected population exhibits at an exponential rate. □

The disease free equilibrium is GAS if $\mathscr{R}_0 < 1$, as proved by the methods of:

- Lyapunov function method.
- LaSalle's invariance principle.
- Comparison theorem.

These results guarantee that effective disease control is achieved with the threshold $\mathscr{R}_0 < 1$ which ensures that the disease dies out.

5. Existence and uniqueness

Theorem 5.1. [32,34] *Analyze the fractional-order system given as:*

$$\begin{cases} D^{\alpha}\mathscr{S}_h(t) = \Lambda_h - \beta_h \mathscr{S}_h(t)\dfrac{\mathscr{I}_v(t)}{\mathscr{N}_v} - \mu_h \mathscr{S}_h(t), \\ D^{\alpha}\mathscr{E}_h(t) = \beta_h \mathscr{S}_h(t)\dfrac{\mathscr{I}_v(t)}{\mathscr{N}_v} - (\sigma_h + \mu_h)\mathscr{E}_h(t), \\ D^{\alpha}\mathscr{I}_h(t) = \sigma_h \mathscr{E}_h(t) - (\gamma_h + \mu_h)\mathscr{I}_h(t), \\ D^{\alpha}\mathscr{R}_h(t) = \gamma_h \mathscr{I}_h(t) - \mu_h \mathscr{R}_h(t), \\ D^{\alpha}\mathscr{S}_v(t) = \Lambda_v(1-p) - \beta_v \mathscr{S}_v(t)\dfrac{\mathscr{I}_h(t)}{\mathscr{N}_h} - \mu_v \mathscr{S}_v(t) - c\mathscr{S}_v(t), \\ D^{\alpha}\mathscr{I}_v(t) = {}_vp + \beta_v \mathscr{S}_v(t)\dfrac{\mathscr{I}_h(t)}{\mathscr{N}_h} - \mu_v \mathscr{I}_v(t) - c\mathscr{I}_v(t). \end{cases} \tag{5.1}$$

Here, $\alpha \in (0,1)$ denotes the fractional order of the system, and all parameters are positive constants. The initial conditions are non-negative and bound. Consequently, the system possesses a unique solution for every $t \geq 0$.

Proof. By using the definition of the Caputo derivative, we write the system of equation (5.1) in integral form as follows:

$$\mathscr{F}(t) = \mathscr{F}(0) + \frac{1}{\Gamma(\alpha)}\int_0^t (t-\tau)^{\alpha-1} D^{\alpha}\mathscr{F}(\tau)\, d\tau. \tag{5.2}$$

To write system of equations (5.1) in integral form as given in (5.2):

$$\begin{cases} \mathscr{S}_h(t) = \mathscr{S}_h(0) + \dfrac{1}{\Gamma(\alpha)}\displaystyle\int_0^t (t-\tau)^{\alpha-1}\Big[\Lambda_h - \beta_h \mathscr{S}_h(\tau)\frac{\mathscr{I}_v(\tau)}{\mathscr{N}_v} - \mu_h \mathscr{S}_h(\tau)\Big]d\tau, \\ \mathscr{E}_h(t) = \mathscr{E}_h(0) + \dfrac{1}{\Gamma(\alpha)}\displaystyle\int_0^t (t-\tau)^{\alpha-1}\Big[\beta_h \mathscr{S}_h(\tau)\frac{\mathscr{I}_v(\tau)}{\mathscr{N}_v} - (\sigma_h + \mu_h)\mathscr{E}_h(\tau)\Big]d\tau, \\ \mathscr{I}_h(t) = \mathscr{I}_h(0) + \dfrac{1}{\Gamma(\alpha)}\displaystyle\int_0^t (t-\tau)^{\alpha-1}\Big[\sigma_h \mathscr{E}_h(\tau) - (\gamma_h + \mu_h)\mathscr{I}_h(\tau)\Big]d\tau, \\ \mathscr{R}_h(t) = \mathscr{R}_h(0) + \dfrac{1}{\Gamma(\alpha)}\displaystyle\int_0^t (t-\tau)^{\alpha-1}\Big[\gamma_h \mathscr{I}_h(\tau) - \mu_h \mathscr{R}_h(\tau)\Big]d\tau, \\ \mathscr{S}_v(t) = \mathscr{S}_v(0) + \dfrac{1}{\Gamma(\alpha)}\displaystyle\int_0^t (t-\tau)^{\alpha-1}\Big[\Lambda_v(1-p) - \beta_v \mathscr{S}_v(\tau)\frac{\mathscr{I}_h(\tau)}{\mathscr{N}_h} - \mu_v \mathscr{S}_v(\tau) - c\mathscr{S}_v(\tau)\Big]d\tau, \\ \mathscr{I}_v(t) = \mathscr{I}_v(0) + \dfrac{1}{\Gamma(\alpha)}\displaystyle\int_0^t (t-\tau)^{\alpha-1}\Big[\Lambda_v p + \beta_v \mathscr{S}_v(\tau)\frac{\mathscr{I}_h(\tau)}{\mathscr{N}_h} - \mu_v \mathscr{I}_v(\tau) - c\mathscr{I}_v(\tau)\Big]d\tau. \end{cases} \tag{5.3}$$

Let the function space:

$$\mathscr{Z} = ([0, \mathscr{T}], \mathbb{R}^6), \tag{5.4}$$

with norm:

$$\|u\| = \sup_{t\in[0,\mathscr{T}]} \sum_{i=1}^{6} |u_i(t)|, \tag{5.5}$$

where $u = (\mathscr{S}_h, \mathscr{E}_h, \mathscr{I}_h, \mathscr{R}_h, \mathscr{S}_v, \mathscr{I}_v)$. This space is a Banach space. Define the operator $\mathscr{T} : \mathscr{Z} \to \mathscr{Z}$:

$$\mathscr{T}(u)(t) = u(0) + \frac{1}{\Gamma(\alpha)} \int_0^t (t-\tau)^{\alpha-1} \mathscr{F}(u(\tau))\, d\tau, \tag{5.6}$$

where $\mathscr{F}(u(\tau))$ represents the system dynamics. We will prove that the right side of the system, represented as $\mathscr{F}(u)$, is Lipschitz continuous in $\mathscr{F}(u)$. To do this we will parametrize as follows: $\mathscr{F}(u) = (\mathscr{S}_h, \mathscr{E}_h, \mathscr{I}_h, \mathscr{R}_h, \mathscr{S}_v, \mathscr{I}_v)$ and $\mathscr{F}(v) = (\mathscr{S}'_h, \mathscr{E}'_h, \mathscr{I}'_h, \mathscr{R}'_h, \mathscr{S}'_v, \mathscr{I}'_v))$. The right hand of the system is:

$$\mathscr{F}(u) = \begin{pmatrix} \Lambda_h - \beta_h \mathscr{S}_h \dfrac{\mathscr{I}_v}{\mathscr{N}_v} - \mu_h \mathscr{S}_h \\ \beta_h \mathscr{S}_h \dfrac{\mathscr{I}_v}{\mathscr{N}_v} - (\sigma_h + \mu_h)\mathscr{E}_h \\ \sigma_h \mathscr{E}_h - (\gamma_h + \mu_h)\mathscr{I}_h \\ \gamma_h \mathscr{I}_h - \mu_h \mathscr{R}_h \\ \Lambda_v(1-p) - \beta_v \mathscr{S}_v \dfrac{\mathscr{I}_h}{\mathscr{N}_h} - \mu_v \mathscr{S}_v - c\mathscr{S}_v \\ \Lambda_v p + \beta_v \mathscr{S}_v \dfrac{\mathscr{I}_h}{\mathscr{N}_h} - \mu_v \mathscr{I}_v - c\mathscr{I}_v \end{pmatrix}. \tag{5.7}$$

In order to establish the Lipschitz continuity condition, we compute the difference $\mathscr{F}(\mathbf{u}) - \mathscr{F}(\mathbf{v})$ and bound it in terms of $\|u - v\|$.

From the inequalities among parts, it follows that there is a constant $\mathscr{L} > 0$ such that:

$$\|\mathscr{F}(u) - \mathscr{F}(v)\| \le \mathscr{L}\|u - v\|, \tag{5.8}$$

$$\begin{aligned}
\mathscr{S}_h &= \left| \left(\Lambda_h - \beta_h \mathscr{S}_h \frac{\mathscr{I}_v}{\mathscr{N}_v} - \mu_h \mathscr{S}_h \right) - \left(\Lambda_h - \beta_h \mathscr{S}'_h \frac{\mathscr{I}'_v}{\mathscr{N}_v} - \mu_h \mathscr{S}'_h \right) \right| \\
&\le \beta_h \left| \mathscr{S}_h \frac{\mathscr{I}_v}{\mathscr{N}_v} - \mathscr{S}'_h \frac{\mathscr{I}'_v}{\mathscr{N}_v} \right| + \mu_h |\mathscr{S}_h - \mathscr{S}'_h| \\
&\le \beta_h \left(\frac{|\mathscr{S}_h - \mathscr{S}'_h|\mathscr{I}_v}{\mathscr{N}_v} + \frac{\mathscr{S}'_h |\mathscr{I}_v - \mathscr{I}'_v|}{\mathscr{N}_v} \right) + \mu_h |\mathscr{S}_h - \mathscr{S}'_h| \\
&\le \left(\frac{\beta_h \mathscr{I}_v}{\mathscr{N}_v} + \mu_h \right) |\mathscr{S}_h - \mathscr{S}'_h| + \frac{\beta_h \mathscr{S}'_h}{\mathscr{N}_v} |\mathscr{I}_v - \mathscr{I}'_v|, \\
\mathscr{E}_h &= \left| \left(\beta_h \mathscr{S}_h \frac{\mathscr{I}_v}{\mathscr{N}_v} - (\sigma_h + \mu_h)\mathscr{E}_h \right) - \left(\beta_h \mathscr{S}'_h \frac{\mathscr{I}'_v}{\mathscr{N}_v} - (\sigma_h + \mu_h)\mathscr{E}'_h \right) \right| \\
&\le \beta_h \left| \mathscr{S}_h \frac{\mathscr{I}_v}{\mathscr{N}_v} - \mathscr{S}'_h \frac{\mathscr{I}'_v}{\mathscr{N}_v} \right| + (\sigma_h + \mu_h) |\mathscr{E}_h - \mathscr{E}'_h| \\
&\le \beta_h \left(\frac{|\mathscr{S}_h - \mathscr{S}'_h|\mathscr{I}_v}{\mathscr{N}_v} + \frac{\mathscr{S}'_h |\mathscr{I}_v - \mathscr{I}'_v|}{\mathscr{N}_v} \right) + (\sigma_h + \mu_h) |\mathscr{E}_h - \mathscr{E}'_h|,
\end{aligned}$$

$$
\begin{aligned}
\mathscr{I}_h &= \left|(\sigma_h\mathscr{E}_h - (\gamma_h+\mu_h)\mathscr{I}_h) - \left(\sigma_h\mathscr{E}'_h - (\gamma_h+\mu_h)\mathscr{I}'_h\right)\right| \\
&\leq \sigma_h|\mathscr{E}_h - \mathscr{E}'_h| + (\gamma_h+\mu_h)|\mathscr{I}_h - \mathscr{I}'_h|, \\
\mathscr{R}_h &= \left|(\gamma_h\mathscr{I}_h - \mu_h\mathscr{R}_h) - \left(\gamma_h\mathscr{I}'_h - \mu_h\mathscr{R}'_h\right)\right| \\
&\leq \gamma_h|\mathscr{I}_h - \mathscr{I}'_h| + \mu_h|\mathscr{R}_h - \mathscr{R}'_h|, \\
\mathscr{S}_v &= \left|\left(\Lambda_v(1-p) - \beta_v\mathscr{S}_v\frac{\mathscr{I}_h}{\mathscr{N}_h} - \mu_v\mathscr{S}_v - c\mathscr{S}_v\right) - \left(\Lambda_v(1-p) - \beta_v\mathscr{S}'_v\frac{\mathscr{I}'_h}{\mathscr{N}_h} - \mu_v\mathscr{S}'_v - c\mathscr{S}'_v\right)\right| \\
&\leq \beta_v\left|\mathscr{S}_v\frac{\mathscr{I}_h}{\mathscr{N}_h} - \mathscr{S}'_v\frac{\mathscr{I}'_h}{\mathscr{N}_h}\right| + (\mu_v+c)|\mathscr{S}_v - \mathscr{S}'_v| \\
&\leq \beta_v\left(\frac{|\mathscr{S}_v - \mathscr{S}'_v|\mathscr{I}_h}{\mathscr{N}_h} + \frac{\mathscr{S}'_v|\mathscr{I}_h - \mathscr{I}'_h|}{\mathscr{N}_h}\right) + (\mu_v+c)|\mathscr{S}_v - \mathscr{S}'_v|, \\
\mathscr{I}_v &= \left|\left({}_vp + \beta_v\mathscr{S}_v\frac{\mathscr{I}_h}{\mathscr{N}_h} - \mu_v\mathscr{I}_v - c\mathscr{I}_v\right) - \left({}_vp + \beta_v\mathscr{S}'_v\frac{\mathscr{I}'_h}{\mathscr{N}_h} - \mu_v\mathscr{I}'_v - c\mathscr{I}'_v\right)\right| \\
&\leq \beta_v\left|\mathscr{S}_v\frac{\mathscr{I}_h}{\mathscr{N}_h} - \mathscr{S}'_v\frac{\mathscr{I}'_h}{\mathscr{N}_h}\right| + (\mu_v+c)|\mathscr{I}_v - \mathscr{I}'_v| \\
&\leq \beta_v\left(\frac{|\mathscr{S}_v - \mathscr{S}'_v|\mathscr{I}_h}{\mathscr{N}_h} + \frac{\mathscr{S}'_v|\mathscr{I}_h - \mathscr{I}'_h|}{\mathscr{N}_h}\right) + (\mu_v+c)|\mathscr{I}_v - \mathscr{I}'_v|. \qquad (5.9)
\end{aligned}
$$

From the preceding inequalities, we note that each element of $\mathscr{F}(u)-\mathscr{F}(v)$ is bounded in terms of a linear combination of $|\mathscr{S}_h - \mathscr{S}'_h|, |\mathscr{E}_h - \mathscr{E}'_h|, |\mathscr{I}_h - \mathscr{I}'_h|, |\mathscr{R}_h - \mathscr{R}'_h|, |\mathscr{S}_v - \mathscr{S}'_v|, |\mathscr{I}_v - \mathscr{I}'_v|$.

This means that $\mathscr{L} > 0$ for which the following holds:

$$\|\mathscr{F}(u) - \mathscr{F}(v)\| \leq \mathscr{L}\|u - v\|.$$

This establishes Lipschitz continuity.

The operator defined in (5.6). For this we demonstrate that $\mathscr{T}$ is a contraction mapping. In particular, we need to show for any $u, v \in \mathscr{Z}$; For this, We utilize the Lipschitz continuity of $\mathscr{F}$ to place a bound on the difference $\|\mathscr{T}(u) - \mathscr{T}(v)\|$:

$$
\begin{aligned}
\|\mathscr{T}(u) - \mathscr{T}(v)\| &= \sup_{t\in[0,\mathscr{T}]}\left|\frac{1}{\Gamma(\alpha)}\int_0^t (t-\tau)^{\alpha-1}\left(\mathscr{F}(u(\tau)) - \mathscr{F}(v(\tau))\right)d\tau\right| \\
&\leq \frac{1}{\Gamma(\alpha)}\int_0^t (t-\tau)^{\alpha-1}\|\mathscr{F}(u(\tau)) - \mathscr{F}(v(\tau))\|d\tau \\
&\leq \frac{\mathscr{L}}{\Gamma(\alpha)}\int_0^t (t-\tau)^{\alpha-1}\|u(\tau) - v(\tau)\|d\tau \\
&\leq \frac{\mathscr{L}}{\Gamma(\alpha)}\|u - v\|\int_0^t (t-\tau)^{\alpha-1}d\tau. \qquad (5.10)
\end{aligned}
$$

To evaluate the integral $\int_0^t (t-\tau)^{\alpha-1}d\tau$ in equation (5.10):

$$\int_0^t (t-\tau)^{\alpha-1}d\tau = \left[\frac{(t-\tau)^\alpha}{\alpha}\right]_0^t = \frac{t^\alpha}{\alpha}. \qquad (5.11)$$

To substitute this into the bound for $\|\mathscr{T}(u) - \mathscr{T}(v)\|$, we obtain:

$$\|\mathcal{T}(\mathbf{u}) - \mathcal{T}(\mathbf{v})\| \leq \frac{L}{\Gamma(\alpha)}\|\mathbf{u} - \mathbf{v}\| \cdot \frac{t^\alpha}{\alpha}. \qquad (5.12)$$

It follows that $t^\alpha \leq \mathscr{T}^\alpha$ since $t \in [0, \mathscr{T}]$. Consequently:

$$\|\mathcal{T}(\mathbf{u}) - \mathcal{T}(\mathbf{v})\| \leq \frac{\mathscr{L}\mathscr{T}^\alpha}{\Gamma(\alpha+1)}\|u - v\|, \tag{5.13}$$

where $\Gamma(\alpha+1) = \alpha\Gamma(\alpha)$.

In case $\mathscr{T}$ is to be a contraction mapping, we need:

$$\frac{\mathscr{L}\mathscr{T}^\alpha}{\Gamma(\alpha+1)} < 1. \tag{5.14}$$

This condition can be satisfied by selecting $\mathscr{T}$ sufficiently small. In particular, we select:

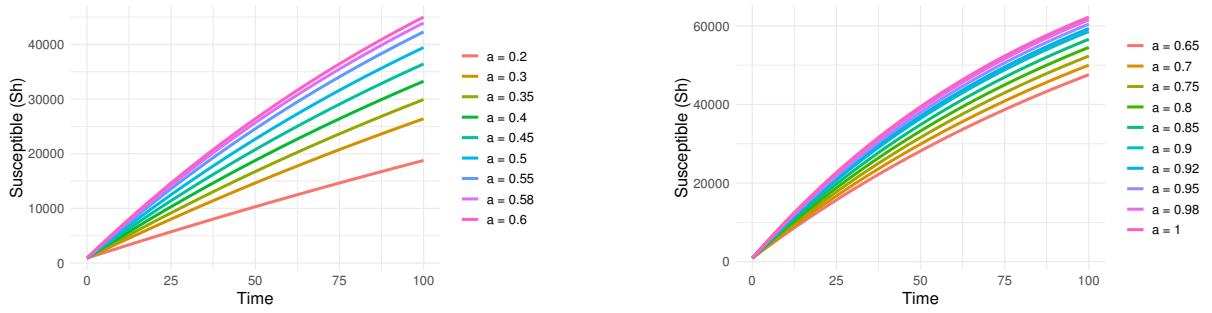
$$\mathscr{T} < \left(\frac{\Gamma(\alpha+1)}{\mathscr{L}}\right)^{1/\alpha}. \tag{5.15}$$

Given that the space $\mathscr{Z}$ is a complete metric space, it implies that $\mathscr{T}$ is a contraction mapping on $\mathscr{Z}$. Thus, by virtue of the Banach fixed-point theorem, it follows that there exists a unique fixed point u such that $\mathscr{T}(u) = u$. This unique fixed point u is the solution of the system(3.1) [15]. □

6. Numerical results and discussion

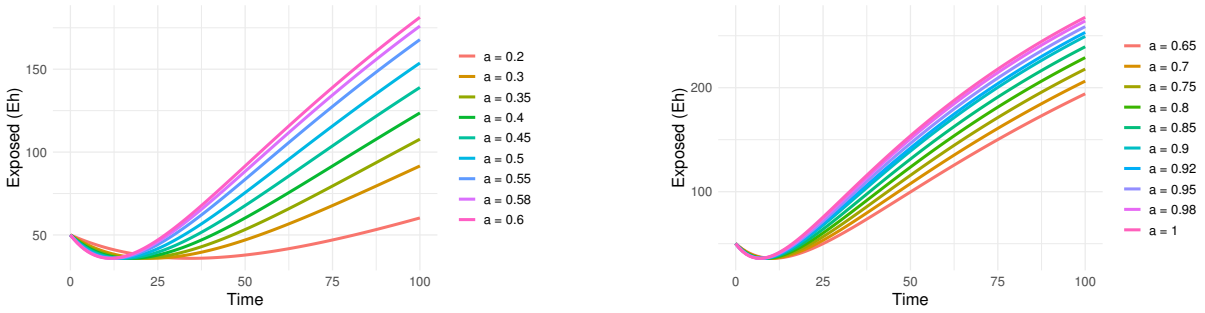
We performed simulations utilizing a Grünwald-Letnikov scheme for fractional-order derivatives to evaluate how the new model performs. The simulations were conducted under different conditions by changing the values of the fractional-order parameter α, the vertical transmission parameter p, and the vector control parameter c.

Results demonstrate that further increasing memory effects leads to a reduction of the infected population peak and also prolonging the duration of the outbreak as a result of decreasing α. Moreover, an increase in vertical transmission permits a higher peaking level of infected individuals, which is certainly important from an outbreak point of view. As adequate vector control measures, including an increase of c, are imposed, there is no corresponding increased infection among humans or mosquitoes; therefore, there is strong evidence for intervention effectiveness in the control of dengue fever.



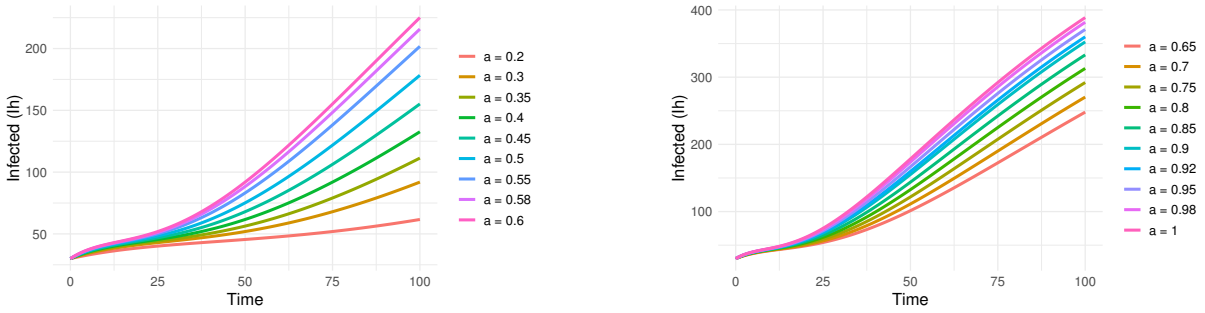
(a) : $\alpha = 0.2 \to 0.6$ (b) : $\alpha = 0.65 \to 1.0$

Figure 1. S_h compartment with fractional order $\alpha = 0.2 \to 1.0$.



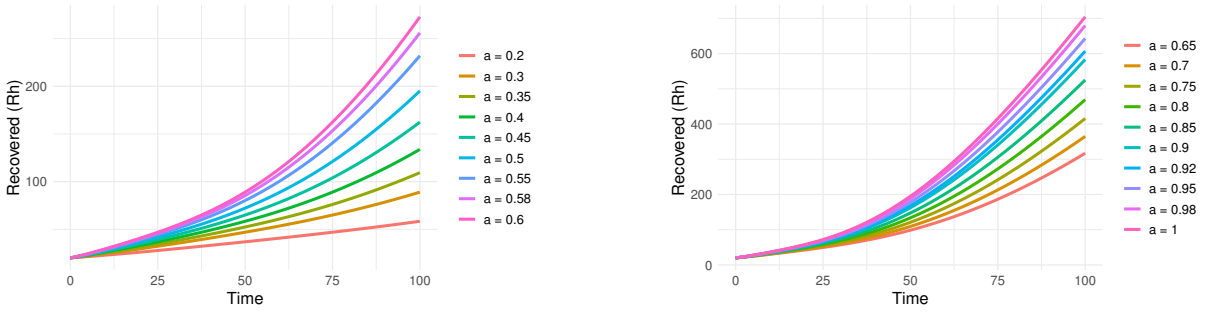
(a) : $\alpha = 0.2 \to 0.6$ (b) : $\alpha = 0.65 \to 1.0$

Figure 2. E_h compartment with fractional order $\alpha = 0.2 \to 1.0$.



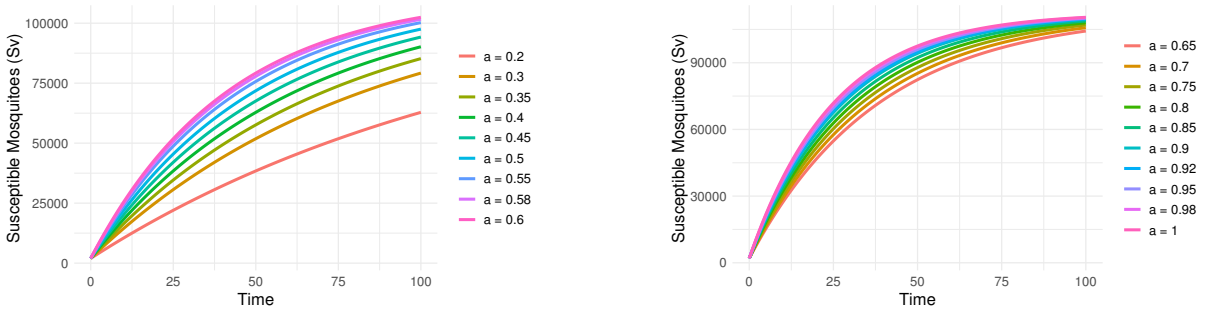
(a) : $\alpha = 0.2 \to 0.6$ (b) : $\alpha = 0.65 \to 1.0$

Figure 3. I_h compartment with fractional order $\alpha = 0.2 \to 1.0$.



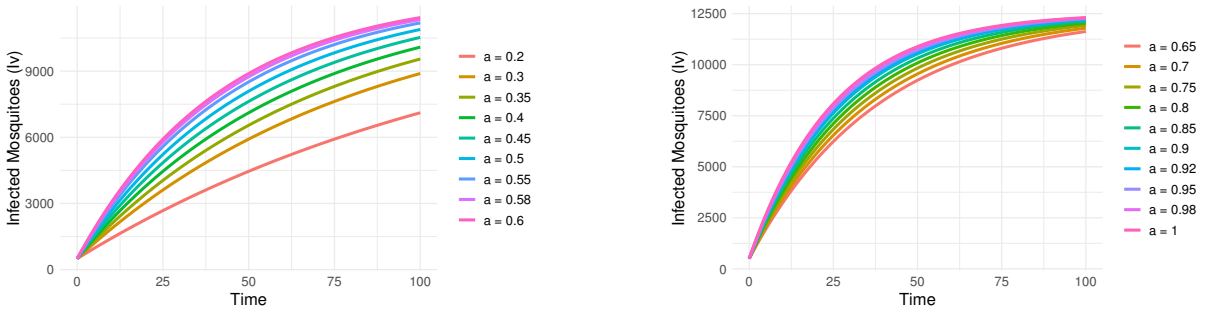
(a) : $\alpha = 0.2 \to 0.6$ (b) : $\alpha = 0.65 \to 1.0$

Figure 4. R_h compartment with fractional order $\alpha = 0.2 \to 1.0$.



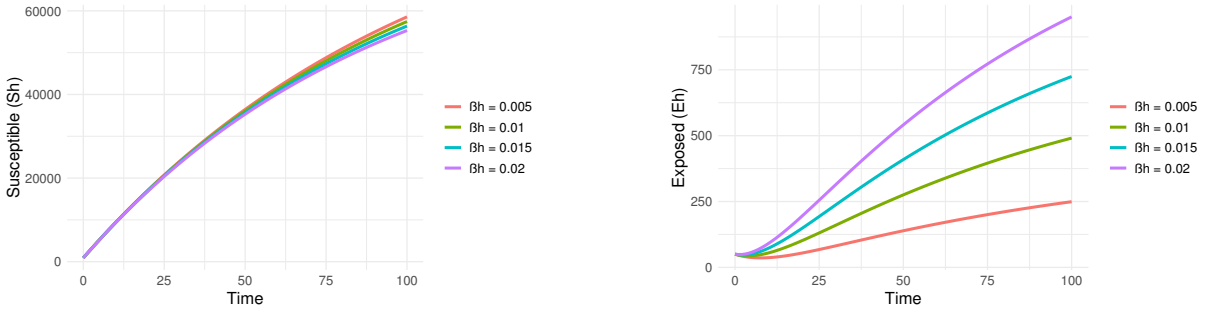
(a) : $\alpha = 0.2 \to 0.6$ (b) : $\alpha = 0.65 \to 1.0$

Figure 5. S_v compartment with fractional order $\alpha = 0.2 \to 1.0$.



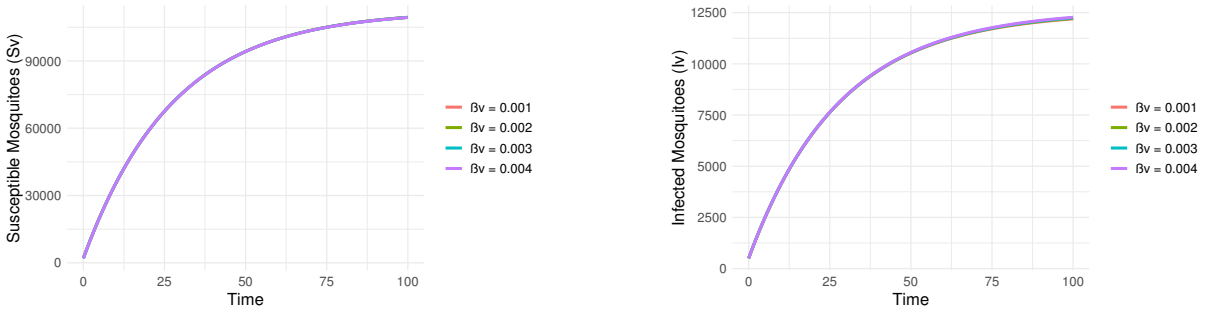
(a) : $\alpha = 0.2 \to 0.6$ (b) : $\alpha = 0.65 \to 1.0$

Figure 6. I_v compartment with fractional order $\alpha = 0.2 \to 1.0$.



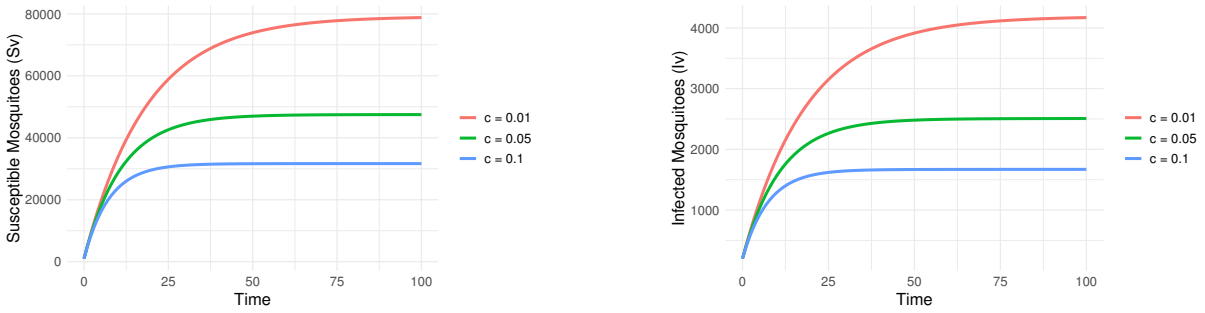
(a) (b)

Figure 7. Impact of transmission rate $\beta_h = 0.005, 0.01, 0.015, 0.02$ on S_h and E_h compartments.



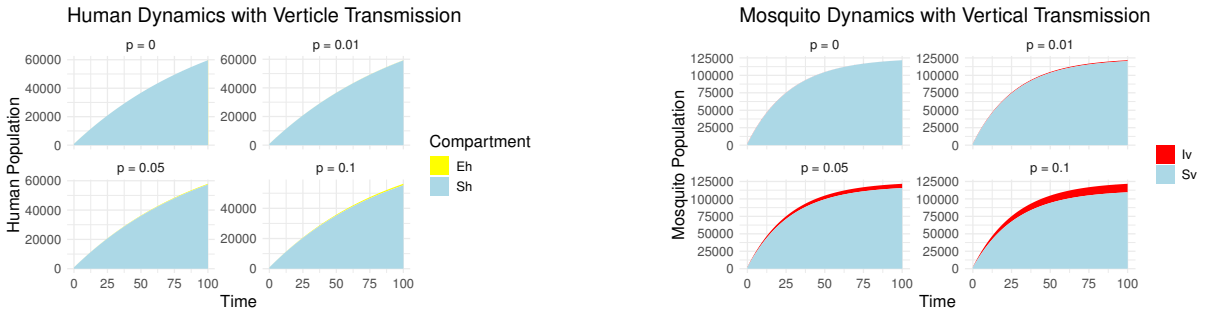
(a) (b)

Figure 8. Impact of transmission rate $\beta_v = 0.001, 0.002, 0.003, 0.004$ on vector compartments.



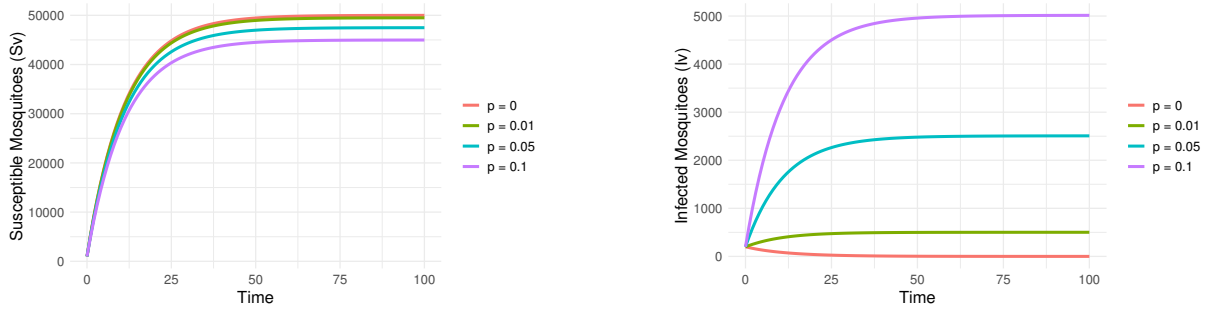
(a) (b)

Figure 9. Impact of vector control $c = 0.01, 0.05, 0.1$ on vector compartments.



(a) (b)

Figure 10. Impact of VT rate $p = 0, 0.01, 0.05, 0.1$ on human and vector compartments.



(a) (b)

Figure 11. Impact of VT rate $p = 0, 0.01, 0.05, 0.1$ on vector compartments.

7. Conclusion

This paper presents the analysis of the influence and effects of the fractional-order parameter α, transmission rates (β_h, β_v), control measures c and vertical transmission probability p on vector-borne disease dynamics. The findings suggest that lower values of α create memory effects that retard the rate of disease progression, whereas larger values promote infection spread. An increase in β_h and β_v is associated with an increase in the rate of decline of susceptible and an increase in the rate of rise of infections, which further supports and strengthens the hypothesis of human-to-mosquito and mosquito-to-human infections. Control measures, c, appear to reduce the population of susceptible mosquitoes and control the rate of infection growth, thus reinforcing the rationale for vector-targeted control efforts. Vertical transmission, p, maintains the infections in both populations and makes elimination of the disease difficult.This study emphasizes the need of fractional-order models in accurately capturing the dynamics of disease transmission. Future work should focus on effective interventions and application of real-world epidemiological data for effective disease management.

Declarations

Ethics approval and consent to participate. Not applicable.

Consent for publication. Not applicable.

Availability of data and materials. The data that support the study's findings are available upon reasonable request from the corresponding author.

Competing interests. The authors state that they do not have any conflicts of interest.

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