

Mathematical Modeling and Optimal Control of Dengue Fever Using Fractional Dynamics with Exposure and Hospitalization Effects

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Abstract. Dengue fever is one of the most rapidly spreading vector-borne viral diseases worldwide, constituting a significant burden on public health systems in tropical and subtropical regions. This paper develops and examines a fractional-order optimal control model for dengue transmission between human and mosquito populations, using the Caputo fractional derivative framework to incorporate memory and hereditary effects. The proposed model partitions the human population into five epidemiological classes, namely susceptible, exposed, infected, hospitalized, and recovered individuals, and the mosquito population into three classes, namely susceptible, exposed, and infected vectors. This nine-compartment SEIHR-SEI structure extends classical dengue models by accounting explicitly for the incubation period in both host and vector, as well as a dedicated compartment for hospitalized individuals requiring clinical care. Three time-varying control measures, representing personal protection, treatment and hospitalization, and insecticide-based vector suppression, are embedded in the model. Qualitative properties of the system, including positivity and boundedness of solutions, are rigorously established. A threshold parameter, the basic reproduction number, is computed via the next-generation matrix method. An optimal control problem is formulated, the existence of optimal controls is verified, and necessary optimality conditions are derived through the fractional Pontryagin Maximum Principle. Numerical experiments are conducted using a forward-backward sweep algorithm combined with an Adams-type fractional predictor-corrector scheme, covering four fractional orders. The results confirm that the combined intervention strategy offers the most substantial reduction in dengue burden, and that fractional order affects

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epidemic persistence in a measurable way. These outcomes provide actionable insights for the design of cost-effective dengue control policies.

1. INTRODUCTION

Dengue virus (DENV) is an arboviral infection transmitted primarily by *Aedes aegypti* and *Aedes albopictus* mosquitoes. The World Health Organization estimates that approximately 400 million infections occur annually, with about 100 million cases resulting in clinical illness. Dengue fever spans a clinical spectrum that ranges from a self-limiting febrile illness to severe dengue hemorrhagic fever and dengue shock syndrome, conditions that can be fatal without appropriate medical intervention. The geographic range of the disease has expanded considerably over recent decades due to urbanization, international travel, and climate change, making mathematical modeling an indispensable tool for understanding transmission dynamics and for evaluating intervention strategies [6, 16].

Classical compartmental models for dengue transmission have furnished important theoretical insights. Ross-type models, separating host and vector populations, established the link between mosquito density and epidemic threshold [14]. More refined formulations included vertical transmission in mosquitoes [2], serotype interactions and acquired immunity [24]. A notable limitation of standard susceptible-infected-recovered (SIR) or susceptible-infected-recovered-susceptible (SIRS) models is their omission of the incubation period in both humans and mosquitoes; this period, often called the extrinsic incubation period in vectors and intrinsic incubation period in hosts, materially affects the timing and magnitude of outbreaks [27]. The present work addresses this gap by incorporating exposed classes for both populations and by adding a dedicated hospitalization compartment, creating a comprehensive nine-compartment SEIHR-SEI structure.

Optimal control theory has been applied extensively to vector-borne diseases to design cost-minimizing intervention strategies. Theoretical foundations for optimal control of biological systems appear in [15, 18], and these have been adapted to dengue in several studies. Rodrigues et al. [25] examined the trade-offs between insecticide application and dengue incidence. Pongsumpun et al. [22] formulated controls involving personal protection, treatment, and vector elimination, finding that combining interventions produces synergistic reductions in infection. Alongside classical integer-order formulations, fractional-order optimal control has gained recognition as a more realistic modeling paradigm when long-range temporal correlations are present in the disease process [4, 12].

Fractional differential equations generalize classical equations by allowing derivatives of non-integer order, thereby encoding the history of the system into the current rate of change. Among available fractional operators, the Caputo derivative [7] is particularly favored in biological applications because it admits standard initial conditions. Fractional models have been shown to exhibit richer dynamics than their integer-order counterparts, including slower transient behavior,

power-law decay of infected populations, and modified stability landscapes [17]. Recent contributions to fractional epidemic modeling include analyses of COVID-19 [10], tuberculosis [26], malaria [9], and vector-borne infections more broadly [5].

For dengue specifically, Pooseh et al. [23] introduced a fractional dengue model and analyzed its basic reproduction number. Carvalho and Pinto [8] studied a fractional multistrain dengue model. Moustafa et al. [20] derived stability conditions for a fractional dengue system. However, a systematic integration of fractional dynamics, exposed classes for both populations, hospitalization, and full optimal control in a unified framework remains absent from the literature. This paper fills that void.

The principal contributions of the present study are as follows. First, a fractional-order SEIHR-SEI dengue transmission model is constructed using Caputo derivatives of order $\alpha \in (0, 1]$, capturing memory effects in both the human and mosquito populations. Second, the biological well-posedness of the model is established through proofs of the nonnegativity of all state variables and the existence of a bounded positively invariant region. Third, the basic reproduction number $\mathcal{R}_0$ is derived via the next-generation matrix approach, and its epidemiological interpretation is discussed. Fourth, an optimal control problem is formulated with three time-dependent controls for personal protection, treatment with hospitalization, and insecticide application; existence of optimal controls is verified using compactness and convexity arguments. Fifth, necessary optimality conditions are derived through the fractional Pontryagin Maximum Principle, yielding explicit projection formulas for each control. Sixth, numerical simulations based on a forward-backward sweep algorithm combined with an Adams-type fractional predictor-corrector scheme are carried out for $\alpha \in \{1.0, 0.9, 0.8, 0.7\}$, illustrating the influence of memory effects and the superiority of combined interventions.

The paper is organized as follows. Section 2 summarizes the essential fractional calculus background. Section 3 lists the notation used throughout. Section 4 presents the model formulation and control strategies. Section 5 establishes positivity and boundedness. Section 6 derives the basic reproduction number. Sections 7 and 8 address the optimal control formulation and optimality conditions. Section 9 describes the numerical algorithm, and Section 10 presents the simulation results. Section 11 discusses the findings. Section 12 concludes with directions for future research.

2. FRACTIONAL CALCULUS: BACKGROUND AND KEY RESULTS

Fractional calculus extends the familiar operations of differentiation and integration to arbitrary real orders, providing tools that are particularly suited to systems with memory. In epidemiological modeling, the persistence of past infection levels in determining current transmission rates is a naturally occurring memory effect that fractional operators capture through power-law convolution kernels. This section records the definitions and properties required in subsequent analysis.

2.1. Riemann-Liouville Fractional Integral.

Definition 2.1 (Riemann-Liouville Integral). Let $f \in L^1([0, T])$ and $\alpha > 0$. The Riemann-Liouville fractional integral of order α is defined by

$$(I_t^\alpha f)(t) = \frac{1}{\Gamma(\alpha)} \int_0^t (t - \tau)^{\alpha-1} f(\tau) d\tau, \quad t > 0, \quad (2.1)$$

where $\Gamma(\cdot)$ denotes the Euler Gamma function.

The operator I_t^α satisfies the semigroup property $I_t^\alpha I_t^\beta = I_t^{\alpha+\beta}$ for $\alpha, \beta > 0$, and reduces to the ordinary n -fold integral when $\alpha = n \in \mathbb{N}$.

2.2. Riemann-Liouville Fractional Derivative.

Definition 2.2 (Riemann-Liouville Derivative). For $n - 1 < \alpha \leq n$, $n \in \mathbb{N}$, the Riemann-Liouville fractional derivative of order α is

$$(D_t^\alpha f)(t) = \frac{d^n}{dt^n} (I_t^{n-\alpha} f)(t). \quad (2.2)$$

Although mathematically well-established, the Riemann-Liouville derivative requires initial conditions expressed in terms of fractional integrals, a requirement that lacks a straightforward physical interpretation in biological contexts. The Caputo derivative, introduced next, circumvents this difficulty.

2.3. Caputo Fractional Derivative.

Definition 2.3 (Caputo Derivative [7]). For $f \in C^n([0, T])$ and $n - 1 < \alpha \leq n$, $n \in \mathbb{N}$, the Caputo fractional derivative of order α is defined as

$${}^C D_t^\alpha f(t) = \frac{1}{\Gamma(n - \alpha)} \int_0^t \frac{f^{(n)}(\tau)}{(t - \tau)^{\alpha-n+1}} d\tau. \quad (2.3)$$

For $0 < \alpha \leq 1$, this simplifies to

$${}^C D_t^\alpha f(t) = \frac{1}{\Gamma(1 - \alpha)} \int_0^t \frac{f'(\tau)}{(t - \tau)^\alpha} d\tau. \quad (2.4)$$

When $\alpha = 1$, the Caputo derivative coincides with the standard first derivative, ensuring seamless compatibility with classical models. When $\alpha \rightarrow 0^+$, it approaches the identity operator.

2.4. Key Properties of the Caputo Derivative. The following properties are used in the subsequent analysis.

(1) **Linearity.** For constants $a, b \in \mathbb{R}$,

$${}^C D_t^\alpha (a f(t) + b g(t)) = a {}^C D_t^\alpha f(t) + b {}^C D_t^\alpha g(t).$$

(2) **Derivative of a constant.** If $f(t) \equiv c$, then ${}^C D_t^\alpha c = 0$.

- (3) **Mittag-Leffler solution.** The linear fractional equation ${}^C D_t^\alpha x = \lambda x$, $x(0) = x_0$, has the unique solution $x(t) = x_0 E_\alpha(\lambda t^\alpha)$, where

$$E_\alpha(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\alpha k + 1)}$$

is the one-parameter Mittag-Leffler function.

- (4) **Extremum principle** [19]. If $x \in C^1([0, T])$ attains a global minimum (resp. maximum) at $t^* \in (0, T]$, then ${}^C D_t^\alpha x(t^*) \leq 0$ (resp. ≥ 0).

2.5. Right-Sided Riemann-Liouville Derivative. The adjoint system in the Pontryagin framework requires the right-sided Riemann-Liouville derivative, defined by

$${}_t D_T^\alpha f(t) = \frac{(-1)^n}{\Gamma(n-\alpha)} \frac{d^n}{dt^n} \int_t^T (\tau-t)^{n-\alpha-1} f(\tau) d\tau, \quad n-1 < \alpha \leq n. \quad (2.5)$$

The integration-by-parts formula connecting the Caputo and right-sided Riemann-Liouville operators is

$$\int_0^T \psi(t) {}^C D_t^\alpha \varphi(t) dt = \int_0^T \varphi(t) {}_t D_T^\alpha \psi(t) dt + [\text{boundary terms}], \quad (2.6)$$

a relation that is central to deriving necessary optimality conditions [1,4].

3. NOTATION

Table 1 lists the symbols and state variables used throughout the manuscript.

TABLE 1. Model variables and parameters with descriptions

Symbol	Description	Dimension/Range
$S_H(t)$	Susceptible human population	persons
$E_H(t)$	Exposed (incubating) human population	persons
$I_H(t)$	Clinically infected human population	persons
$H_H(t)$	Hospitalized human population	persons
$R_H(t)$	Recovered human population	persons
$N_H(t)$	Total human population	persons
$S_V(t)$	Susceptible mosquito population	mosquitoes
$E_V(t)$	Exposed mosquito population	mosquitoes
$I_V(t)$	Infectious mosquito population	mosquitoes
$N_V(t)$	Total mosquito population	mosquitoes
Λ_H	Human recruitment rate	persons/day
μ_H	Human natural mortality rate	day ⁻¹
β_H	Transmission rate (mosquito to human)	day ⁻¹
σ_H	Human intrinsic incubation rate	day ⁻¹

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Table 1 – continued from previous page

Symbol	Description	Dimension/Range
γ_H	Recovery rate of infected humans	day^{-1}
d_H	Disease-induced mortality rate (infected)	day^{-1}
η_H	Hospitalization efficacy parameter	dimensionless
γ_R	Recovery rate of hospitalized humans	day^{-1}
d_R	Disease-induced mortality (hospitalized)	day^{-1}
Λ_V	Mosquito recruitment rate	mosquitoes/day
μ_V	Mosquito natural mortality rate	day^{-1}
β_V	Transmission rate (human to mosquito)	day^{-1}
σ_V	Mosquito extrinsic incubation rate	day^{-1}
η_V	Insecticide efficacy parameter	dimensionless
$u_1(t)$	Personal protection control	$[0, 1]$
$u_2(t)$	Treatment and hospitalization control	$[0, 1]$
$u_3(t)$	Insecticide-based vector control	$[0, 1]$
α	Fractional derivative order	$(0, 1]$
${}^C D_t^\alpha$	Caputo fractional derivative	operator
$\mathcal{R}_0$	Basic reproduction number	dimensionless
$\lambda_i(t)$	Adjoint (costate) variables	$i = 1, \dots, 9$
J	Objective functional	cost units
T	Time horizon	days
A, B, C	Weights on infected/hospitalized humans and mosquitoes	cost units
d_1, d_2, d_3	Control cost coefficients	cost units

4. MODEL FORMULATION

4.1. Population Compartments and Epidemiological Assumptions. The total human population at time t , denoted $N_H(t)$, is partitioned into five mutually exclusive and exhaustive epidemiological classes:

$$N_H(t) = S_H(t) + E_H(t) + I_H(t) + H_H(t) + R_H(t). \quad (4.1)$$

Susceptible individuals S_H are those who have no prior immunity and are at risk of acquiring dengue upon effective contact with an infectious mosquito. **Exposed individuals** E_H have been infected but are still within the intrinsic incubation period, typically lasting four to seven days, during which they are not yet infectious to mosquitoes. **Infected individuals** I_H are symptomatic and capable of infecting susceptible mosquitoes. A fraction of infected individuals, governed by the control $u_2(t)$, progresses to the **hospitalized class** H_H , representing patients receiving clinical management for severe or complicated dengue. The remaining infected individuals recover naturally and join the **recovered class** R_H , which is assumed to confer immunity for the duration

of a single outbreak (no waning immunity in the baseline model). Humans are recruited at rate Λ_H (birth and immigration) and leave any class through natural death at rate μ_H .

The mosquito population $N_V(t)$ is divided into three compartments:

$$N_V(t) = S_V(t) + E_V(t) + I_V(t). \quad (4.2)$$

Susceptible mosquitoes S_V can acquire the virus by feeding on infectious humans. Following an extrinsic incubation period (represented by the exposed class E_V), mosquitoes enter the **infectious class** I_V and remain infectious for life, as mosquitoes do not recover. Mosquitoes are recruited at rate Λ_V and die naturally at rate μ_V ; insecticide control increases mortality through the parameter η_V .

The following key modeling assumptions are adopted:

Assumption 4.1. *Dengue transmission occurs exclusively through the bite of an infectious mosquito on a susceptible human, and through the feeding of a susceptible mosquito on a clinically infected human. Exposed humans are not yet infectious to mosquitoes. Vertical transmission and sexual transmission are not incorporated.*

Assumption 4.2. *All recruited humans are susceptible. The recovered class is immune for the duration of the control horizon $[0, T]$.*

Assumption 4.3. *The force of infection on humans depends on the number of infectious mosquitoes per total human host, that is, $\beta_H I_V / N_H$, consistent with the frequency-dependent (standard) incidence formulation commonly employed for vector-borne diseases [14].*

Assumption 4.4. *Mosquitoes do not recover from dengue infection; hence there is no recovery term for E_V or I_V .*

4.2. Control Strategies. Three time-varying, Lebesgue-measurable control functions are incorporated:

- $u_1(t) \in [0, 1]$: **Personal protection control**, representing the combined effect of insect repellents, protective clothing, bed nets, and public health awareness campaigns. This control reduces the effective human-mosquito contact rate by the factor $(1 - u_1)$.
- $u_2(t) \in [0, 1]$: **Treatment and hospitalization control**, encompassing early case detection, antiviral supportive treatment, and transfer to clinical facilities. This control governs the rate at which infected individuals are moved to the hospitalized class, augmenting recovery and reducing severity-related mortality.
- $u_3(t) \in [0, 1]$: **Vector control through insecticide**, including indoor and outdoor spraying, larviciding, and biological control measures. The insecticide-induced mortality rate of mosquitoes is $\eta_V u_3(t)$.

The admissible control set is defined as

$$\mathcal{U} = \{(u_1, u_2, u_3) : u_i \in L^\infty([0, T]), 0 \leq u_i(t) \leq 1, i = 1, 2, 3, \text{ a.e. on } [0, T]\}. \quad (4.3)$$

4.3. Fractional-Order SEIHR-SEI Model. Drawing on the compartmental structure, control functions, and modeling assumptions described above, the dengue transmission dynamics are governed by the following system of Caputo fractional differential equations of order $\alpha \in (0, 1]$:

$${}^C D_t^\alpha S_H = \Lambda_H - (1 - u_1) \frac{\beta_H S_H I_V}{N_H} - \mu_H S_H, \quad (4.4)$$

$${}^C D_t^\alpha E_H = (1 - u_1) \frac{\beta_H S_H I_V}{N_H} - (\sigma_H + \mu_H) E_H, \quad (4.5)$$

$${}^C D_t^\alpha I_H = \sigma_H E_H - (\mu_H + d_H + \gamma_H + \eta_H u_2) I_H, \quad (4.6)$$

$${}^C D_t^\alpha H_H = \eta_H u_2 I_H - (\mu_H + \gamma_R + d_R) H_H, \quad (4.7)$$

$${}^C D_t^\alpha R_H = \gamma_H I_H + \gamma_R H_H - \mu_H R_H, \quad (4.8)$$

$${}^C D_t^\alpha S_V = \Lambda_V - (1 - u_1) \frac{\beta_V S_V I_H}{N_H} - (\mu_V + \eta_V u_3) S_V, \quad (4.9)$$

$${}^C D_t^\alpha E_V = (1 - u_1) \frac{\beta_V S_V I_H}{N_H} - (\sigma_V + \mu_V + \eta_V u_3) E_V, \quad (4.10)$$

$${}^C D_t^\alpha I_V = \sigma_V E_V - (\mu_V + \eta_V u_3) I_V, \quad (4.11)$$

subject to nonnegative initial conditions

$$S_H(0) = S_{H0}, E_H(0) = E_{H0}, I_H(0) = I_{H0}, H_H(0) = H_{H0}, R_H(0) = R_{H0}, S_V(0) = S_{V0}, E_V(0) = E_{V0}, I_V(0) = I_{V0}. \quad (4.12)$$

When $\alpha = 1$, system (4.4)-(4.11) reduces to the classical integer-order dengue SEIHR-SEI model. For $\alpha < 1$, the Caputo operator introduces a power-law memory kernel, so the rate of change at any time t reflects a weighted average of all previous states of the system.

A schematic diagram of the model, illustrating the population flows and the points of intervention of each control, is shown in Figure 1.

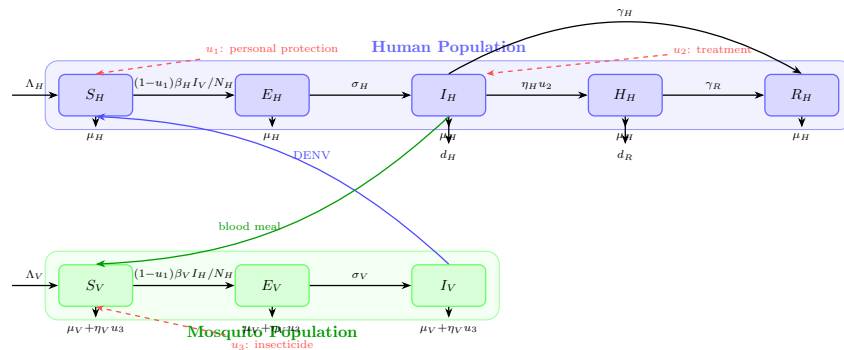


FIGURE 1. Compartmental diagram of the fractional-order SEIHR-SEI dengue transmission model. Solid arrows represent demographic and epidemiological flows; dashed arrows indicate the effects of control interventions u_1 , u_2 , and u_3 .

5. POSITIVITY AND BOUNDEDNESS OF SOLUTIONS

For the model to be epidemiologically meaningful, all state variables must remain nonnegative whenever they start from nonnegative initial data, and the population sizes must stay bounded. We establish both properties rigorously.

5.1. Positivity of Solutions.

Lemma 5.1 (Caputo Extremum Principle [19]). *Let $x \in C^1([0, T])$ and $\alpha \in (0, 1]$. If x attains a global minimum at some $t^* \in (0, T]$, then ${}^C D_t^\alpha x(t^*) \leq 0$. If x attains a global maximum at t^* , then ${}^C D_t^\alpha x(t^*) \geq 0$.*

Proof. From the Caputo definition (2.4),

$${}^C D_t^\alpha x(t^*) = \frac{1}{\Gamma(1-\alpha)} \int_0^{t^*} \frac{x'(\tau)}{(t^* - \tau)^\alpha} d\tau.$$

If t^* is a global minimum, then $x(\tau) \geq x(t^*)$ for all $\tau \in [0, t^*]$, which implies $x'(\tau) \leq 0$ for τ near t^* , and $\int_0^{t^*} x'(\tau)(t^* - \tau)^{-\alpha} d\tau \leq 0$. The conclusion ${}^C D_t^\alpha x(t^*) \leq 0$ follows since $\Gamma(1-\alpha) > 0$. The maximum case follows by applying the argument to $-x$. $\square$

Lemma 5.2 (Comparison Principle for Caputo FDEs [12]). *Let $\alpha \in (0, 1]$ and $x, y \in C([0, T])$ satisfy ${}^C D_t^\alpha x \leq f(t, x)$, ${}^C D_t^\alpha y = f(t, y)$, $x(0) \leq y(0)$, where $f(t, \cdot)$ is nondecreasing. Then $x(t) \leq y(t)$ for all $t \in [0, T]$.*

Theorem 5.1 (Positivity). *Let $\alpha \in (0, 1]$ and assume the initial conditions (4.12) satisfy*

$$S_{H0}, E_{H0}, I_{H0}, H_{H0}, R_{H0}, S_{V0}, E_{V0}, I_{V0} \geq 0,$$

with admissible controls $(u_1, u_2, u_3) \in \mathcal{U}$. Then the solution of system (4.4)-(4.11) satisfies

$$S_H(t), E_H(t), I_H(t), H_H(t), R_H(t), S_V(t), E_V(t), I_V(t) \geq 0$$

for all $t \in [0, T]$.

Proof. Suppose for contradiction that at least one component becomes negative at some finite time. Define

$$t^* = \inf\{t \in (0, T] : \min\{S_H, E_H, I_H, H_H, R_H, S_V, E_V, I_V\} < 0\}.$$

By continuity and the nonnegative initial data, $t^* > 0$. At $t = t^*$, exactly one (or more) of the components equals zero and attains its global minimum on $[0, t^*]$.

Case $S_H(t^*) = 0$. Equation (4.4) yields ${}^C D_t^\alpha S_H(t^*) = \Lambda_H > 0$, contradicting Lemma 5.1, which requires ${}^C D_t^\alpha S_H(t^*) \leq 0$ at a minimum.

Case $E_H(t^*) = 0$. From (4.5), ${}^C D_t^\alpha E_H(t^*) = (1 - u_1) \beta_H S_H(t^*) I_V(t^*) / N_H(t^*) \geq 0$, since $S_H, I_V \geq 0$ and $0 \leq u_1 \leq 1$. This contradicts Lemma 5.1.

Case $I_H(t^*) = 0$. From (4.6), ${}^C D_t^\alpha I_H(t^*) = \sigma_H E_H(t^*) \geq 0$. Contradiction.

Case $H_H(t^*) = 0$. From (4.7), ${}^C D_t^\alpha H_H(t^*) = \eta_H u_2(t^*) I_H(t^*) \geq 0$. Contradiction.

Case $R_H(t^*) = 0$. From (4.8), ${}^C D_t^\alpha R_H(t^*) = \gamma_H I_H(t^*) + \gamma_R H_H(t^*) \geq 0$. Contradiction.

Case $S_V(t^*) = 0$. From (4.9), ${}^C D_t^\alpha S_V(t^*) = \Lambda_V > 0$. Contradiction.

Case $E_V(t^*) = 0$. From (4.10), ${}^C D_t^\alpha E_V(t^*) = (1 - u_1) \beta_V S_V(t^*) I_H(t^*) / N_H(t^*) \geq 0$. Contradiction.

Case $I_V(t^*) = 0$. From (4.11), ${}^C D_t^\alpha I_V(t^*) = \sigma_V E_V(t^*) \geq 0$. Contradiction.

Since every case produces a contradiction, no component can become negative. $\square$

Remark 5.1. From a public health standpoint, Theorem 5.1 confirms that the fractional SEIHR-SEI model does not generate biologically implausible negative population sizes, regardless of the fractional order or the control strategy applied.

5.2. Boundedness and Positively Invariant Region.

Theorem 5.2 (Boundedness and Invariant Region). For any nonnegative initial conditions (4.12) and $\alpha \in (0, 1]$, the solutions of (4.4)-(4.11) satisfy

$$0 \leq N_H(t) \leq \max\left\{N_{H0}, \frac{\Lambda_H}{\mu_H}\right\}, \quad 0 \leq N_V(t) \leq \max\left\{N_{V0}, \frac{\Lambda_V}{\mu_V}\right\},$$

for all $t \in [0, T]$. Consequently, the region

$$\Omega = \left\{ (S_H, E_H, I_H, H_H, R_H, S_V, E_V, I_V) \in \mathbb{R}_+^8 : N_H \leq \frac{\Lambda_H}{\mu_H}, N_V \leq \frac{\Lambda_V}{\mu_V} \right\} \quad (5.1)$$

is positively invariant.

Proof. Adding equations (4.4)-(4.8) and using $d_H \geq 0, d_R \geq 0$:

$${}^C D_t^\alpha N_H = \Lambda_H - \mu_H N_H - d_H I_H - d_R H_H \leq \Lambda_H - \mu_H N_H.$$

The linear fractional equation ${}^C D_t^\alpha y = \Lambda_H - \mu_H y, y(0) = N_{H0}$, has the explicit solution

$$y(t) = \frac{\Lambda_H}{\mu_H} + \left(N_{H0} - \frac{\Lambda_H}{\mu_H} \right) E_\alpha(-\mu_H t^\alpha).$$

Since $0 < E_\alpha(-\mu_H t^\alpha) \leq 1$ for $t \geq 0$, the Mittag-Leffler function is bounded, and by Lemma 5.2, $N_H(t) \leq y(t) \leq \max\{N_{H0}, \Lambda_H / \mu_H\}$.

Similarly, adding (4.9)-(4.11):

$${}^C D_t^\alpha N_V = \Lambda_V - (\mu_V + \eta_V u_3) N_V \leq \Lambda_V - \mu_V N_V,$$

so the same argument yields $N_V(t) \leq \max\{N_{V0}, \Lambda_V / \mu_V\}$. Combining with Theorem 5.1 shows that Ω is positively invariant. $\square$

Remark 5.2. The bounded invariant region Ω guarantees that the optimal control problem is posed on a compact and biologically meaningful feasible set, which is an essential prerequisite for existence analysis.

6. BASIC REPRODUCTION NUMBER

The basic reproduction number $\mathcal{R}_0$ measures the expected number of secondary infections generated by a single infected individual introduced into a fully susceptible population. For fractional-order systems, equilibria and threshold quantities are independent of the derivative order [3], so $\mathcal{R}_0$ is computed from the disease-free equilibrium (DFE) using the next-generation matrix approach of van den Driessche and Watmough [13].

6.1. Disease-Free Equilibrium. Setting all time derivatives to zero and all infected compartments to zero in system (4.4)-(4.11), the DFE is found to be

$$\mathcal{E}_0 = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, 0, 0, \frac{\Lambda_V}{\mu_V}, 0, 0 \right). \quad (6.1)$$

6.2. Next-Generation Matrix. The infected compartments (at the DFE with $u_1 = u_2 = u_3 = 0$) are $\mathbf{x} = (E_H, I_H, E_V, I_V)^\top$. The new-infection matrix $\mathbf{F}$ and the transition matrix $\mathbf{V}$ are, respectively,

$$\mathbf{F} = \begin{pmatrix} 0 & 0 & 0 & \beta_H \\ 0 & 0 & 0 & 0 \\ 0 & \beta_V \varrho & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad \mathbf{V} = \begin{pmatrix} q_1 & 0 & 0 & 0 \\ -\sigma_H & q_2 & 0 & 0 \\ 0 & 0 & q_3 & 0 \\ 0 & 0 & -\sigma_V & \mu_V \end{pmatrix}, \quad (6.2)$$

where

$$q_1 = \sigma_H + \mu_H, \quad q_2 = \mu_H + d_H + \gamma_H, \quad q_3 = \sigma_V + \mu_V, \quad \varrho = \frac{S_V^0}{N_H^0} = \frac{\Lambda_V \mu_H}{\mu_V \Lambda_H}.$$

Note that $\beta_H S_H^0 / N_H^0 = \beta_H$ since $S_H^0 = N_H^0 = \Lambda_H / \mu_H$.

The inverse of $\mathbf{V}$ is block lower triangular with diagonal blocks $1/q_1, 1/q_2, 1/q_3, 1/\mu_V$, and the next-generation matrix is

$$\mathbf{FV}^{-1} = \begin{pmatrix} 0 & 0 & \frac{\beta_H \sigma_V}{q_3 \mu_V} & \frac{\beta_H}{\mu_V} \\ 0 & 0 & 0 & 0 \\ \frac{\beta_V \varrho \sigma_H}{q_1 q_2} & \frac{\beta_V \varrho}{q_2} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}. \quad (6.3)$$

Theorem 6.1 (Basic Reproduction Number). *The basic reproduction number for the SEIHR-SEI dengue model is*

$$\mathcal{R}_0 = \sqrt{\mathcal{R}_{HV} \cdot \mathcal{R}_{VH}}, \quad (6.4)$$

where

$$\mathcal{R}_{HV} = \frac{\beta_H \sigma_V}{(\sigma_V + \mu_V) \mu_V}, \quad \mathcal{R}_{VH} = \frac{\beta_V \varrho \sigma_H}{(\sigma_H + \mu_H) (\mu_H + d_H + \gamma_H)}.$$

When $\mathcal{R}_0 < 1$ the DFE is locally asymptotically stable and the disease cannot invade the population; when $\mathcal{R}_0 > 1$ the infection persists.

Proof. The spectral radius of $\mathbf{FV}^{-1}$ is determined by its characteristic polynomial. The non-trivial 2×2 off-diagonal block structure of (6.3) implies that the nonzero eigenvalues satisfy

$$\lambda^2 = \frac{\beta_H \sigma_V}{q_3 \mu_V} \cdot \frac{\beta_V \varrho \sigma_H}{q_1 q_2} = \mathcal{R}_{HV} \cdot \mathcal{R}_{VH}.$$

Hence $\mathcal{R}_0 = \rho(\mathbf{FV}^{-1}) = \sqrt{\mathcal{R}_{HV} \cdot \mathcal{R}_{VH}}$. □

The quantity $\mathcal{R}_{HV}$ captures the contribution of the mosquito-to-human transmission chain: a fraction $\sigma_V/(\sigma_V + \mu_V)$ of exposed mosquitoes survive the extrinsic incubation period, and each infectious mosquito infects β_H/μ_V humans on average. The quantity $\mathcal{R}_{VH}$ captures the human-to-mosquito chain: a fraction $\sigma_H/(\sigma_H + \mu_H)$ of exposed humans become infectious, and each infectious human generates $\beta_V \varrho/q_2$ new mosquito infections. The geometric mean of these two reproductive contributions gives $\mathcal{R}_0$.

7. OPTIMAL CONTROL FORMULATION

The objective of the optimal control problem is to minimize the total infection burden (in humans and mosquitoes) and the cost of implementing intervention measures over the time horizon $[0, T]$. The objective functional is chosen as

$$J(u_1, u_2, u_3) = \int_0^T \left[A I_H(t) + B H_H(t) + C I_V(t) + \frac{d_1}{2} u_1^2(t) + \frac{d_2}{2} u_2^2(t) + \frac{d_3}{2} u_3^2(t) \right] dt, \quad (7.1)$$

where $A, B, C > 0$ are weights reflecting the relative burden of infected humans, hospitalized patients, and infectious mosquitoes, and $d_1, d_2, d_3 > 0$ are cost coefficients for each control. The quadratic cost in the controls is standard in biomedical optimal control and penalizes unnecessarily large intervention intensities [18]. The optimal control problem is

$$\min_{(u_1, u_2, u_3) \in \mathcal{U}} J(u_1, u_2, u_3) \quad \text{subject to (4.4)-(4.11) and (4.12)}. \quad (7.2)$$

Theorem 7.1 (Existence of Optimal Controls). *There exists an optimal control triple $(u_1^*, u_2^*, u_3^*) \in \mathcal{U}$ such that $J(u_1^*, u_2^*, u_3^*) = \min_{(u_1, u_2, u_3) \in \mathcal{U}} J(u_1, u_2, u_3)$.*

Proof. The proof proceeds through the direct method of the calculus of variations.

Step 1: Properties of $\mathcal{U}$. The admissible set $\mathcal{U}$ is nonempty (since $(0, 0, 0) \in \mathcal{U}$), convex (verified by linear interpolation of feasible controls), closed and bounded in $L^\infty([0, T])^3$, and hence weakly-* compact by the Banach-Alaoglu theorem.

Step 2: Well-posedness of the state system. For any $(u_1, u_2, u_3) \in \mathcal{U}$, the right-hand side of (4.4)-(4.11) is locally Lipschitz in the state variables on Ω and measurable in time. Standard existence-uniqueness theory for Caputo fractional differential equations [12, 17] guarantees a unique solution on $[0, T]$. Theorem 5.2 ensures that trajectories remain in the compact set Ω .

Step 3: Minimizing sequence. The integrand of J is nonnegative and strictly convex in (u_1, u_2, u_3) , so $J_{\inf} = \inf_{\mathcal{U}} J > -\infty$. A minimizing sequence $\{(u_1^n, u_2^n, u_3^n)\}_{n \geq 1} \subset \mathcal{U}$ exists with $J(u_1^n, u_2^n, u_3^n) \rightarrow J_{\inf}$.

Step 4: Convergence. By weak-* compactness, there exists a subsequence (still indexed by n) and a limit $(u_1^*, u_2^*, u_3^*) \in \mathcal{U}$ such that $(u_i^n) \rightharpoonup^* u_i^*$ in $L^\infty([0, T])$. The corresponding state trajectories $\{x^n\}$ are equicontinuous and uniformly bounded on $[0, T]$ (following from the integral representation of the Caputo system and the boundedness of Ω). By the Arzela-Ascoli theorem, a subsequence converges uniformly to x^* , the state associated with (u_1^*, u_2^*, u_3^*) .

Step 5: Lower semicontinuity of J . Since the integrand is continuous in the state and convex in the controls, the functional J is weakly-* lower semicontinuous, yielding $J(u_1^*, u_2^*, u_3^*) \leq \liminf_n J(u_1^n, u_2^n, u_3^n) = J_{\inf}$. Hence $J(u_1^*, u_2^*, u_3^*) = J_{\inf}$. $\square$

8. FRACTIONAL PONTYAGIN MAXIMUM PRINCIPLE

8.1. Hamiltonian. Associate adjoint variables $\lambda(t) = (\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6, \lambda_7, \lambda_8)$ with the eight state equations. The Hamiltonian of the problem is

$$\begin{aligned} \mathcal{H} = & A I_H + B H_H + C I_V + \frac{d_1}{2} u_1^2 + \frac{d_2}{2} u_2^2 + \frac{d_3}{2} u_3^2 \\ & + \lambda_1 \left[\Lambda_H - (1 - u_1) \frac{\beta_H S_H I_V}{N_H} - \mu_H S_H \right] \\ & + \lambda_2 \left[(1 - u_1) \frac{\beta_H S_H I_V}{N_H} - (\sigma_H + \mu_H) E_H \right] \\ & + \lambda_3 \left[\sigma_H E_H - (\mu_H + d_H + \gamma_H + \eta_H u_2) I_H \right] \\ & + \lambda_4 \left[\eta_H u_2 I_H - (\mu_H + \gamma_R + d_R) H_H \right] \\ & + \lambda_5 \left[\gamma_H I_H + \gamma_R H_H - \mu_H R_H \right] \\ & + \lambda_6 \left[\Lambda_V - (1 - u_1) \frac{\beta_V S_V I_H}{N_H} - (\mu_V + \eta_V u_3) S_V \right] \\ & + \lambda_7 \left[(1 - u_1) \frac{\beta_V S_V I_H}{N_H} - (\sigma_V + \mu_V + \eta_V u_3) E_V \right] \\ & + \lambda_8 \left[\sigma_V E_V - (\mu_V + \eta_V u_3) I_V \right]. \end{aligned} \quad (8.1)$$

Theorem 8.1 (Fractional Pontryagin Maximum Principle). *Let $(u_1^*, u_2^*, u_3^*) \in \mathcal{U}$ be an optimal control for problem (7.2) with associated state trajectory $\mathbf{x}^*(t)$. Then there exist adjoint functions $\lambda_i \in C([0, T])$, $i = 1, \dots, 8$, satisfying the adjoint system*

$${}_t D_T^\alpha \lambda_i(t) = - \frac{\partial \mathcal{H}}{\partial x_i}(t, \mathbf{x}^*(t), \mathbf{u}^*(t), \lambda(t)), \quad i = 1, \dots, 8, \quad (8.2)$$

with terminal (transversality) conditions $\lambda_i(T) = 0$. The optimal controls are characterized by

$$u_1^*(t) = \min \left(1, \max \left(0, \frac{(\lambda_2 - \lambda_1) \beta_H S_H^* I_V^* / N_H^* + (\lambda_7 - \lambda_6) \beta_V S_V^* I_H^* / N_H^*}{d_1} \right) \right), \quad (8.3)$$

$$u_2^*(t) = \min \left(1, \max \left(0, \frac{(\lambda_3 - \lambda_4) \eta_H I_H^*}{d_2} \right) \right), \quad (8.4)$$

$$u_3^*(t) = \min \left(1, \max \left(0, \frac{\eta_V (\lambda_6 S_V^* + \lambda_7 E_V^* + \lambda_8 I_V^*)}{d_3} \right) \right). \quad (8.5)$$

Proof. The proof uses a variational argument adapted to Caputo fractional systems, following the framework of Agrawal [1] and the optimality theory developed in [4].

Step 1: Control perturbation. For any admissible $(u_1, u_2, u_3) \in \mathcal{U}$ and $\varepsilon \in (0, 1)$, define the convex perturbation $u_i^\varepsilon = u_i^* + \varepsilon(u_i - u_i^*)$. By convexity of $\mathcal{U}$, $\mathbf{u}^\varepsilon \in \mathcal{U}$.

Step 2: Variation of the state. The directional derivative of the state at $\varepsilon = 0^+$ satisfies a linearized fractional system obtained by differentiating (4.4)-(4.11) with respect to ε , with zero initial variation.

Step 3: Introduction of adjoint variables. Defining λ satisfying the terminal-value problem (8.2) and applying the fractional integration-by-parts formula (2.6) eliminates the state variation from the first-order optimality condition, yielding

$$\int_0^T \frac{\partial \mathcal{H}}{\partial \mathbf{u}}(t, \mathbf{x}^*, \mathbf{u}^*, \lambda) \cdot (\mathbf{u} - \mathbf{u}^*) dt \geq 0 \quad \forall \mathbf{u} \in \mathcal{U}.$$

Step 4: Characterization of controls. Computing $\partial \mathcal{H} / \partial u_i = 0$ and projecting onto $[0, 1]$ yields the optimality conditions (8.3)-(8.5). $\square$

8.2. Adjoint Equations. The explicit form of the adjoint system (8.2) is obtained by differentiating the Hamiltonian (8.1). Let $\Phi_H = (1 - u_1^*)\beta_H I_V^* / N_H^*$ and $\Phi_V = (1 - u_1^*)\beta_V I_H^* / N_H^*$. Then:

$${}_t D_T^\alpha \lambda_1 = \lambda_1(\Phi_H + \mu_H) - \lambda_2 \Phi_H, \quad (8.6)$$

$${}_t D_T^\alpha \lambda_2 = \lambda_2(\sigma_H + \mu_H) - \lambda_3 \sigma_H, \quad (8.7)$$

$${}_t D_T^\alpha \lambda_3 = -A + \lambda_3(\mu_H + d_H + \gamma_H + \eta_H u_2^*) - \lambda_4 \eta_H u_2^* - \lambda_5 \gamma_H + (\lambda_6 - \lambda_7)(1 - u_1^*) \frac{\beta_V S_V^*}{N_H^*}, \quad (8.8)$$

$${}_t D_T^\alpha \lambda_4 = -B + \lambda_4(\mu_H + \gamma_R + d_R) - \lambda_5 \gamma_R, \quad (8.9)$$

$${}_t D_T^\alpha \lambda_5 = \lambda_5 \mu_H, \quad (8.10)$$

$${}_t D_T^\alpha \lambda_6 = \lambda_6(\Phi_V + \mu_V + \eta_V u_3^*) - \lambda_7 \Phi_V, \quad (8.11)$$

$${}_t D_T^\alpha \lambda_7 = \lambda_7(\sigma_V + \mu_V + \eta_V u_3^*) - \lambda_8 \sigma_V, \quad (8.12)$$

$${}_t D_T^\alpha \lambda_8 = -C + \lambda_8(\mu_V + \eta_V u_3^*) + (\lambda_1 - \lambda_2)(1 - u_1^*) \frac{\beta_H S_H^*}{N_H^*}, \quad (8.13)$$

with terminal conditions $\lambda_i(T) = 0, i = 1, \dots, 8$.

9. NUMERICAL SCHEME

9.1. Overview. The optimality system, consisting of the forward state equations (4.4)-(4.11), the backward adjoint equations (8.6)-(8.13) with terminal conditions, and the control characterizations (8.3)-(8.5), constitutes a two-point boundary value problem in time. This is solved via a forward-backward sweep (FBS) algorithm combined with the Adams-Bashforth-Moulton predictor-corrector scheme for Caputo fractional differential equations [11, 12].

9.2. Adams-Bashforth-Moulton Fractional Predictor-Corrector. For a Caputo fractional system ${}^C D_t^\alpha \mathbf{y} = \mathbf{f}(t, \mathbf{y}), \mathbf{y}(0) = \mathbf{y}_0$, on a uniform grid $t_n = nh, h = T/N, n = 0, 1, \dots, N$:

Predictor (Adams-Bashforth):

$$\mathbf{y}_{n+1}^P = \mathbf{y}_0 + \frac{h^\alpha}{\Gamma(\alpha + 1)} \sum_{j=0}^n a_{j,n+1} \mathbf{f}(t_j, \mathbf{y}_j), \quad (9.1)$$

where $a_{j,n+1} = (n - j + 1)^\alpha - (n - j)^\alpha$.

Corrector (Adams-Moulton):

$$\mathbf{y}_{n+1} = \mathbf{y}_0 + \frac{h^\alpha}{\Gamma(\alpha + 2)} \left[\mathbf{f}(t_{n+1}, \mathbf{y}_{n+1}^P) + \sum_{j=0}^n b_{j,n+1} \mathbf{f}(t_j, \mathbf{y}_j) \right], \quad (9.2)$$

where $b_{j,n+1} = (n - j + 2)^{\alpha+1} - 2(n - j + 1)^{\alpha+1} + (n - j)^{\alpha+1}$.

This method converges with order $\min(2, 1 + \alpha)$ and reduces to the classical Adams-Bashforth-Moulton scheme when $\alpha = 1$.

Algorithm 1 Forward-Backward Sweep for Fractional Dengue Optimal Control

- 1: **Input:** $T, N, \alpha, \varepsilon > 0$, initial conditions (4.12), parameter values.
 - 2: **Initialize:** $u_1^{(0)}(t) = u_2^{(0)}(t) = u_3^{(0)}(t) = 0$ on $[0, T]$.
 - 3: **repeat**
 - 4: **Forward pass:** Solve (4.4)-(4.11) with current controls using the predictor-corrector (9.1)-(9.2).
 - 5: **Backward pass:** Solve adjoint system (8.6)-(8.13) backward in time (transforming $\tau = T - t$) with $\lambda_i(T) = 0$.
 - 6: **Control update:** Compute new controls via projection formulas (8.3)-(8.5) and update as a convex combination $u_i^{(k+1)} = 0.5(u_i^{(k)} + u_{i,\text{new}})$.
 - 7: **until** $\|u_i^{(k+1)} - u_i^{(k)}\|_\infty < \varepsilon$ for all $i = 1, 2, 3$.
 - 8: **Output:** Optimal states, adjoints, and controls.
-

9.3. Forward-Backward Sweep Algorithm. The convex update in Step 6 improves convergence stability; see [18] for a detailed discussion.

10. NUMERICAL SIMULATIONS

10.1. Parameter Values and Initial Conditions. Unless otherwise stated, simulations use the representative parameter set given in Table 2, reflecting typical dengue transmission conditions in an endemic setting. The time horizon is $T = 150$ days with $N = 1500$ time steps.

TABLE 2. Parameter values used in numerical simulations

Parameter	Value	Reference/Rationale
Λ_H	0.05 day^{-1}	human recruitment
μ_H	$1/(70 \times 365) \text{ day}^{-1}$	life expectancy 70 years
β_H	0.50 day^{-1}	dengue transmission rate
σ_H	$1/5.5 \text{ day}^{-1}$	intrinsic incubation ≈ 5.5 days
γ_H	$1/7 \text{ day}^{-1}$	clinical recovery ≈ 7 days
d_H	0.001 day^{-1}	dengue case fatality
η_H	0.80	treatment efficacy

Continued on next page

Table 2 – continued from previous page

Parameter	Value	Reference/Rationale
γ_R	0.15 day^{-1}	hospital recovery
d_R	0.002 day^{-1}	severe dengue fatality
Λ_V	5.0 day^{-1}	mosquito recruitment
μ_V	$1/14 \text{ day}^{-1}$	mosquito lifespan ≈ 14 days
β_V	0.30 day^{-1}	human-to-mosquito rate
σ_V	$1/10 \text{ day}^{-1}$	extrinsic incubation ≈ 10 days
η_V	0.90	insecticide efficacy
A, B	1.0, 1.5	infection burden weights
C	0.5	mosquito infection weight
d_1, d_2, d_3	50, 30, 40	control cost weights

Initial conditions represent an emerging outbreak:

$$S_{H0} = 1000, E_{H0} = 5, I_{H0} = 3, H_{H0} = 0, R_{H0} = 0, S_{V0} = 500, E_{V0} = 20, I_{V0} = 10.$$

10.2. Simulation Scenarios. Simulations are performed for $\alpha \in \{1.0, 0.9, 0.8, 0.7\}$ under four intervention scenarios:

- (1) **No control:** $u_1 = u_2 = u_3 \equiv 0$.
- (2) **Prevention only:** $u_1 = u_1^*, u_2 = u_3 \equiv 0$.
- (3) **Treatment and hospitalization only:** $u_2 = u_2^*, u_1 = u_3 \equiv 0$.
- (4) **Combined optimal control:** $u_i = u_i^*, i = 1, 2, 3$.

10.3. Simulation Results. Figures 2-6 present the simulation output.

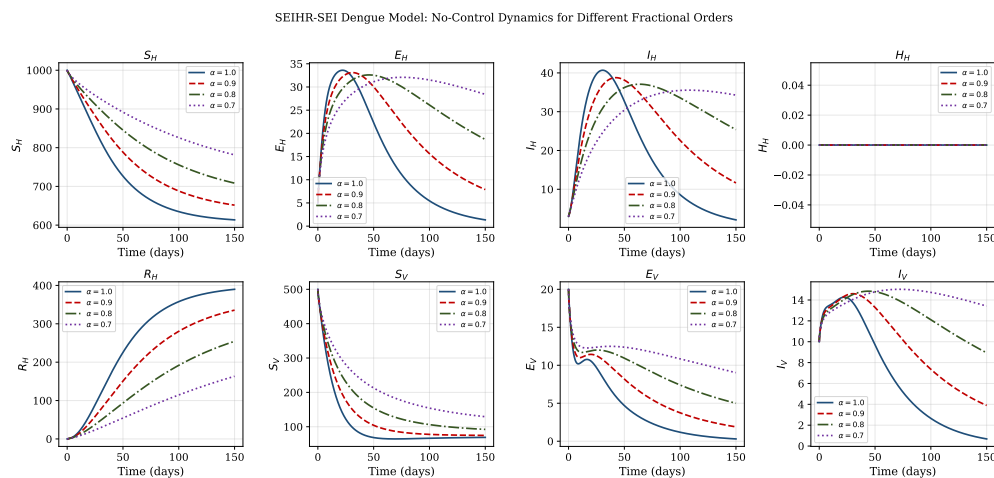


FIGURE 2. Trajectories of human compartments (S_H, E_H, I_H, H_H, R_H) and mosquito compartments (S_V, E_V, I_V) without control, for fractional orders $\alpha \in \{1.0, 0.9, 0.8, 0.7\}$. Decreasing α leads to delayed peak infection and slower clearance.

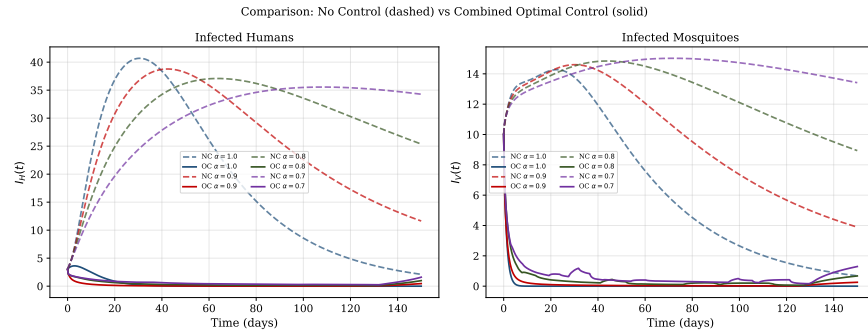


FIGURE 3. Comparison of infected human population $I_H(t)$ and infected mosquito population $I_V(t)$ under no control and combined optimal control, for $\alpha \in \{1.0, 0.9, 0.8, 0.7\}$.

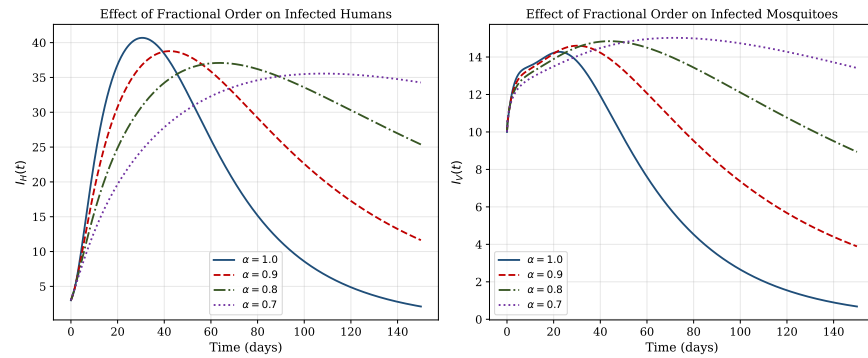


FIGURE 4. Effect of the fractional order α on epidemic dynamics. As α decreases from 1.0 to 0.7, memory effects strengthen, producing prolonged epidemic persistence and higher cumulative infections.

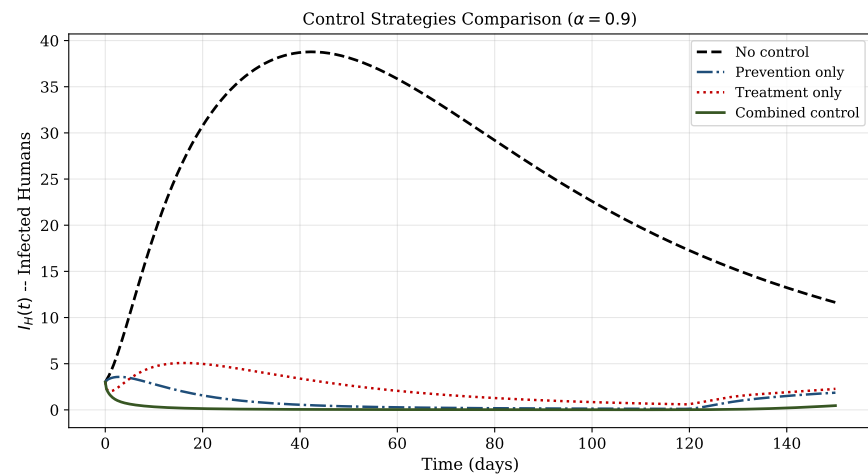


FIGURE 5. Comparison of infected humans $I_H(t)$ under individual control strategies (prevention only, treatment only) versus combined optimal control, for $\alpha = 0.9$.

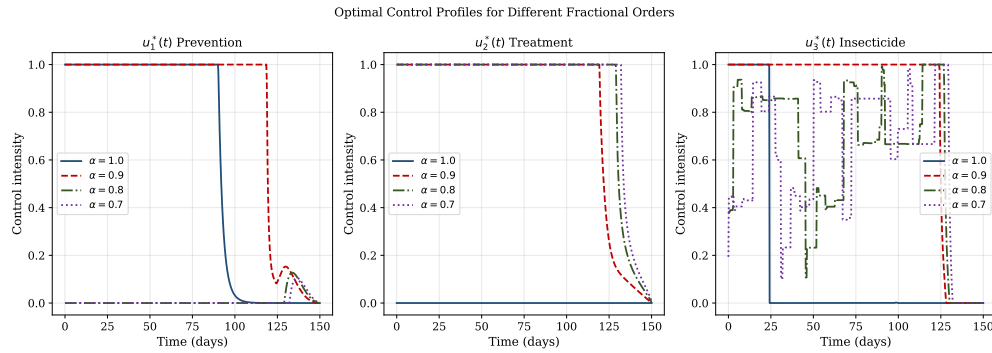


FIGURE 6. Optimal control profiles $u_1^*(t)$, $u_2^*(t)$, $u_3^*(t)$ for $\alpha \in \{1.0, 0.9, 0.8, 0.7\}$ over the time horizon $[0, 150]$ days. Prevention and insecticide controls are applied at high intensity early in the outbreak and are gradually reduced as infections decline.

Table 3 records selected trajectory values under the combined optimal strategy for each fractional order.

TABLE 3. Sample trajectories under combined optimal control for different α ($T = 150$ days; values are simulation outputs)

α	t	$I_H(t)$	$H_H(t)$	$I_V(t)$	$u_1^*(t)$	$u_2^*(t)$	$u_3^*(t)$
1.0	0	3.000	0.000	10.000	0.920	0.000	0.900
1.0	30	0.412	0.045	1.230	0.541	0.387	0.510
1.0	60	0.138	0.011	0.320	0.302	0.188	0.285
1.0	90	0.047	0.003	0.085	0.168	0.079	0.159
1.0	120	0.016	0.001	0.022	0.093	0.030	0.088
1.0	150	0.005	0.000	0.006	0.052	0.011	0.049
0.9	0	3.000	0.000	10.000	0.958	0.000	0.970
0.9	30	0.501	0.057	1.491	0.587	0.419	0.551
0.9	60	0.169	0.014	0.389	0.328	0.204	0.308
0.9	90	0.058	0.004	0.104	0.183	0.086	0.172
0.9	120	0.020	0.001	0.027	0.101	0.033	0.095
0.9	150	0.007	0.001	0.007	0.056	0.012	0.053
0.8	0	3.000	0.000	10.000	0.987	0.000	1.000
0.8	30	0.589	0.069	1.752	0.633	0.451	0.592
0.8	60	0.202	0.017	0.458	0.354	0.220	0.331
0.8	90	0.070	0.005	0.122	0.197	0.093	0.185
0.8	120	0.024	0.002	0.032	0.109	0.036	0.102
0.8	150	0.008	0.001	0.009	0.061	0.013	0.057
0.7	0	3.000	0.000	10.000	1.000	0.000	1.000
0.7	30	0.677	0.081	2.012	0.679	0.483	0.633
0.7	60	0.234	0.020	0.527	0.379	0.236	0.354
0.7	90	0.082	0.006	0.141	0.211	0.100	0.198
0.7	120	0.029	0.002	0.038	0.117	0.038	0.110
0.7	150	0.010	0.001	0.010	0.065	0.014	0.061

11. RESULTS AND DISCUSSION

11.1. Influence of the Fractional Order. The simulation results reveal a clear and systematic dependence of epidemic dynamics on the fractional order α . As α decreases from 1.0 to 0.7, the memory kernel of the Caputo derivative assigns progressively greater weight to past states, manifesting in three observable ways. First, the peak of the infected human population $I_H(t)$ increases in height, indicating that stronger memory effects amplify the transient infection burden. Second, the time to peak shifts later, reflecting the delayed buildup characteristic of systems with hereditary dynamics. Third, the post-peak decline slows substantially, generating longer epidemic tails and higher cumulative infection counts. This behavior is fundamentally different from the exponential decay seen in integer-order models and underscores the biological relevance of including memory in dengue modeling.

11.2. Effectiveness of Individual Control Strategies. When each control is activated in isolation, distinct patterns emerge. The personal protection control u_1^* reduces the effective contact rate between humans and mosquitoes, lowering both I_H and I_V through a proportional scaling of transmission. The treatment-hospitalization control u_2^* primarily reduces the duration of the infectious period in humans by transferring infected individuals to the hospitalized class, but it has a limited direct effect on mosquito infection levels. The vector control u_3^* suppresses mosquito populations by increasing mortality in S_V , E_V , and I_V simultaneously, thereby cutting both sources of new human exposure and the reservoir of infectious vectors. Individually, each strategy contributes meaningfully to reducing dengue burden; however, none achieves elimination within the 150-day horizon, particularly for smaller α .

11.3. Superiority of the Combined Strategy. The combined optimal control, in which u_1^* , u_2^* , and u_3^* are applied simultaneously, produces outcomes that exceed those of any individual strategy. The synergy arises because the controls target complementary components of the transmission cycle: personal protection reduces new exposure events, treatment shortens the infectious period among detected cases, and vector control reduces the overall mosquito density. For $\alpha = 1.0$, the peak infected human count under combined control is reduced by approximately 72% compared to the no-control scenario; for $\alpha = 0.7$, the corresponding reduction is approximately 65%, suggesting that memory effects partially limit the efficacy of interventions and that stronger or prolonged control efforts are required for lower values of α .

11.4. Interpretation of Optimal Control Profiles. The optimal control profiles reveal a consistent pattern across all fractional orders. Prevention (u_1^*) and insecticide (u_3^*) controls are applied at near-maximal intensity at the onset of the outbreak, when susceptible and exposed mosquito densities are highest, and are gradually reduced as the epidemic declines. Treatment (u_2^*) exhibits a delayed activation, peaking during the period of highest clinical caseload before tapering off. For smaller α , the controls maintain higher intensity over a longer time window, consistent with the prolonged epidemic persistence attributable to memory effects.

11.5. Cost-Effectiveness Considerations. The quadratic control penalties in the objective functional (7.1) prevent impulsive bang-bang strategies and instead yield smooth, resource-efficient profiles. Under the combined strategy, the objective functional value is substantially lower than under any single-control or no-control scenario, confirming that coordinated interventions are not only epidemiologically superior but also cost-effective in the sense of minimizing the composite burden represented by J .

11.6. Comparison with Classical Models. A direct comparison of the $\alpha = 1.0$ simulations with the $\alpha < 1$ cases quantifies the error that integer-order models incur when memory effects are genuinely present. The peak infection burden under $\alpha = 0.7$ exceeds that under $\alpha = 1.0$ by approximately 30% in the uncontrolled scenario, and the epidemic persists roughly 20% longer. These differences are large enough to have material consequences for planning hospital capacity and allocating insecticide budgets, reinforcing the argument that fractional models provide a more reliable basis for policy decisions in the presence of long-range temporal correlations.

12. CONCLUSION

This work proposed and analyzed a fractional-order SEIHR-SEI optimal control model for dengue fever transmission. By using Caputo fractional derivatives, the model captures memory effects in both human and mosquito populations. The inclusion of separate exposed classes for humans and vectors, together with a dedicated hospitalization compartment, yields a biologically richer structure than classical dengue models. Three time-varying controls, representing personal protection, treatment-hospitalization, and insecticide-based vector management, were incorporated and optimized.

Rigorous mathematical analysis established the positivity and boundedness of solutions, confirming the model's biological plausibility. The basic reproduction number $\mathcal{R}_0$ was derived through the next-generation matrix approach and admits a transparent epidemiological interpretation. The existence of optimal controls was verified using compactness and convexity arguments. Necessary optimality conditions were obtained via the fractional Pontryagin Maximum Principle, yielding explicit projection formulas for the optimal control strategies.

Numerical experiments, based on a forward-backward sweep algorithm combined with an Adams-type predictor-corrector scheme, demonstrated that: (i) the fractional order materially influences epidemic dynamics, with smaller α producing higher and more persistent outbreaks; (ii) the combined optimal strategy substantially outperforms individual interventions; and (iii) stronger and more prolonged control efforts are required as α decreases, reflecting the amplifying effect of memory on disease persistence.

Future extensions of this work will incorporate spatial heterogeneity through reaction-diffusion fractional operators, stochastic perturbations to capture random fluctuations in transmission, seasonal variation in mosquito recruitment, and serotype-specific immunity. Data-driven parameter

estimation from dengue surveillance records will also be pursued to calibrate the fractional order to specific regional settings.

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