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RESEARCH ARTICLE

Discrete (q,τ)-nabla fractional modeling of Dengue dynamics with stability and optimal control

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ABSTRACT

Utilizing the (q,τ)-nabla fractional operator, we develop a discrete-time Dengue transmission model incorporating memory and time-scale deformation effects. Stability, endemic equilibrium, and bifurcation behavior are analyzed through Jacobian eigenvalue methods and fractional threshold conditions. A Lyapunov-based feedback strategy and a memory-weighted optimal control framework are introduced to regulate infected and hospitalized populations. Necessary optimality conditions are derived using a generalized Pontryagin maximum principle adapted to the (q,τ)-fractional setting. Numerical simulations confirm the theoretical results and demonstrate the influence of the parameters q, τ, and α on memory dynamics and intervention efficiency. The proposed framework provides a flexible approach for modeling and controlling epidemic systems with nonlocal memory effects.

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1. Introduction

Fractional quantum calculus is a potent method for modeling memory-dependent processes in engineering,[1–3] physics,[4,5] and biology.[6,7] Among various formulations,[8–12] the fractional nabla offers a discrete-time framework that is enhanced with nonlocal memory and time-scale deformation.[13–15] This makes it particularly suitable for modeling systems in which the dynamics of the present are influenced to varying degrees by the past states.[16–18] This is especially relevant in epidemiological designs, since delayed immune responses, behavioral patterns, and intervention effects compound with time.

Dengue fever, a virus carried by mosquitoes, remains a major public health concern in tropical and subtropical regions.[19–22] By neglecting control feedback delays and historical influences, conventional epidemic models often generate oversimplified estimates.[23–27] The mathematical mechanism of dengue fever is usually described through a compartmental epidemic model, where the human and mosquito populations are divided into interacting classes. The transmission dynamics are governed by nonlinear differential equations that represent infection, recovery, and vector-host interactions.[28,29]

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We propose a discrete-time Dengue model controlled by a (q,τ)-nabla fractional operator to get over these limitations. The scale-sensitive transmission dynamics and memory implications found in real epidemics can be represented using this model.[30–32] The novelty of this work is the development of a discrete-time Dengue epidemic framework driven by the (q,τ)-nabla fractional operator, which simultaneously incorporates nonlocal memory effects, discrete-time deformation, and time-scale sensitivity within a unified epidemiological model. Unlike classical integer-order and standard fractional epidemic models, the proposed formulation introduces the deformation parameter q and the temporal scaling parameter τ, allowing the system to capture memory-dependent transmission dynamics and irregular temporal evolution. From the mathematical viewpoint, this work establishes new theoretical results for the proposed (q,τ)-fractional Dengue system, including the existence and uniqueness of solutions, positivity of trajectories, fractional threshold conditions, local stability analysis via the Jacobian matrix, and bifurcation behavior under memory-dependent dynamics. A generalized Lyapunov framework adapted to the (q,τ)-nabla setting is further developed to analyze asymptotic stability in different epidemiological regimes. Another important novelty is a memory-weighted optimal control problem governed by the (q,τ)-fractional dynamics. A generalized Pontryagin maximum principle is derived specifically for the (q,τ)-nabla operator, leading to new optimality conditions and feedback control laws that incorporate historical disease evolution and time-scale deformation. The resulting control strategy combines vaccination, treatment, fumigation, and awareness mechanisms while penalizing excessive intervention through a nonlocal memory-dependent cost functional. In addition, the work introduces a general bifurcation-map formulation for the (q,τ)-fractional Dengue model and demonstrates how the parameters q, τ, and α affect oscillatory behavior, disease persistence, stability transitions, and eradication thresholds. Therefore, the proposed framework extends existing epidemic models by integrating fractional memory, quantum-type deformation, optimal control, and bifurcation analysis into a single mathematically consistent system, providing a new direction for memory-aware epidemiological modeling and control.

2. Preliminaries

In this section, we present the following concepts, which are the generalization of the well-known quantum[33–35] and can be found in.[36–39]

Definition 1. *Let $0<q<1$, $\tau>0$, and define the (q,τ)-Gamma function as*

$$\Gamma_{q,\tau}(x) := (1-q)^{1-x}\prod_{k=0}^{\infty}\frac{1-q^{\tau(k+1)}}{1-q^{\tau x+\tau k}}.$$

The basic Gamma function is generalized to $q\to 1$ and $\tau\to 1$. Additionally, the definition of the (q,τ)-fractional binomial coefficient is

$$\binom{\alpha}{k}_{(q,\tau)} := \frac{\Gamma_{q,\tau}(\alpha+1)}{\Gamma_{q,\tau}(k+1)\Gamma_{q,\tau}(\alpha-k+1)}.$$

Definition 2. *Let $f:\mathbb{N}_0\to\mathbb{R}$, and let $0<q<1$, $\tau>0$, $0<\alpha<1$. The fractional (q,τ)-nabla operator of order α using the (q,τ)-Gamma function is defined by*

$$\nabla^{\alpha}_{q,\tau}f(n) := \frac{1}{\Gamma_{q,\tau}(1-\alpha)}\sum_{k=0}^{n-1}\frac{f(k)-f(k+1)}{[(n-k)_{(q,\tau)}]^{\alpha}},$$

where

$$(n-k)_{(q,\tau)} := \frac{1-(q\tau)^{n-k}}{1-q},$$

and $\Gamma_{q,\tau}(x)$ is the (q,τ)-Gamma function satisfying the recurrence

$$\Gamma_{q,\tau}(x+1) = (x)_{(q,\tau)}\Gamma_{q,\tau}(x), \quad \Gamma_{q,\tau}(1)=1.$$

Proposition 1. *Let $f,g:\mathbb{N}_0\to\mathbb{R}$, and $a,b\in\mathbb{R}$. Let $0<q<1$, $\tau>0$, and $0<\alpha<1$. Define the fractional (q,τ)-nabla operator using the (q,τ)-Gamma function as:*

$$\nabla^{\alpha}_{q,\tau}f(n) = \frac{1}{\Gamma_{q,\tau}(1-\alpha)}\sum_{k=0}^{n-1}\frac{f(k)-f(k+1)}{[(n-k)_{(q,\tau)}]^{\alpha}},$$

where $(n-k)_{(q,\tau)} = \frac{1-(q\tau)^{n-k}}{1-q}$.

Then the following properties hold:

(1) Linearity:

$$\nabla^{\alpha}_{q,\tau}[af(n)+bg(n)] = a\nabla^{\alpha}_{q,\tau}f(n) + b\nabla^{\alpha}_{q,\tau}g(n).$$

(2) Consistency with Integer Order: If $\alpha\to 1^{-}$, then

$$\lim_{\alpha\to 1^{-}}\nabla^{\alpha}_{q,\tau}f(n) = \nabla^{*}_{q,\tau}f(n) = \frac{f(n)-f(n+1)}{(n)_{(q,\tau)}}.$$

(3) Vanishing on Constants: If $f(n)=C$ for all n, then

$$\nabla^{\alpha}_{q,\tau}f(n) = 0.$$

(4) Monotonicity Preservation: If $f(n)$ is monotonically decreasing, then $\nabla^{\alpha}_{q,\tau}f(n)\geq 0$.

Proof. (1) Linearity follows directly from the linearity of the sum and difference:

$$\nabla^{\alpha}_{q,\tau}(af+bg)(n) = \frac{1}{\Gamma_{q,\tau}(1-\alpha)} \sum_{k=0}^{n-1}\frac{a(f(k)-f(k+1))+b(g(k)-g(k+1))}{[(n-k)_{(q,\tau)}]^{\alpha}}. \tag{1}$$

(2) As $\alpha \to 1^-$, we use the limit $\Gamma_{q,\tau}(1-\alpha) \to \Gamma_{q,\tau}(0) \to \infty$ in a scaled form and isolate the leading term $k = n-1$, recovering:

$$\nabla_{q,\tau} f(n) = \frac{f(n) - f(n+1)}{(n)_{(q,\tau)}}.$$

(3) For a constant function $f(n) = C$, the difference $f(k) - f(k+1) = 0$, so the entire sum vanishes.

(4) If $f(k) \geq f(k+1)$, then each term in the sum is non-negative, hence the entire operator is non-negative.

□

Example 1. ***Tables 1*** *and* ***2*** *show how the fractional (q,τ)-nabla operator $\nabla^{\alpha}_{q,\tau} f(n)$ behaves when applied to two exponential functions with contradictory patterns: the decaying function $f(n) = e^{-0.3n}$ and the growing function $f(n) = e^{0.3n}$. Using the dominant-term approximation at $k = n-1$, both are assessed with fractional order $\alpha = 0.9$, deformation parameters $q = 0.5$, and $\tau = 1.2$. For the decaying case $f(n) = e^{-0.3n}$, the operator produces negative values which correspond to the flattening of the exponential curve and thedecrease in magnitude with increasing n. The operator is reduced to a discrete fractional backward difference in this approximation since the kernel $(n-k)^{\alpha}_{(q,\tau)}$ is equal to 1 for all n. A typical feature of models with fatigue, decay, or immunity is the memory-aware slowing of dynamics, which is captured by this structure. In contrast, the operator values in the rising case $f(n) = e^{0.3n}$ are strictly positive and increase rapidly with n, reflecting the function's rapid growth. The growth pattern is thus amplified by the (q,τ)-nabla operator, which also shows how sensitive memory-based fractional dynamics are to monotonic trends. Collectively, these findings show that the (q,τ)-nabla operator offers an interpretable, memory-sensitive generalization of classical differences and accurately depicts both attenuation and amplification dynamics based on its fundamental pattern. This characteristic is crucial when modeling processes such as infection growth or decay in biological or epidemiological systems that exhibit nonlocal history dependence (see* ***Figure 1****).*

Table 1. Approximation of (q,τ)-nabla operator $\nabla^{\alpha}_{q,\tau} f(n)$ for $f(n) = e^{-0.3n}$, with $q = 0.5$, $\tau = 1.2$, and $\alpha = 0.9$

n	$f(n)$	$f(n-1)$	$(n-k)^{\alpha}_{(q,\tau)}$	$\nabla^{\alpha}_{q,\tau} f(n)$
1	0.740818	1.000000	1.000	−0.259182
2	0.548812	0.740818	1.000	−0.192007
3	0.406570	0.548812	1.000	−0.142242
4	0.301194	0.406570	1.000	−0.105375
5	0.223130	0.301194	1.000	−0.078064

Table 2. Approximation of (q,τ)-nabla operator $\nabla^{\alpha}_{q,\tau} f(n)$ for $f(n) = e^{0.3n}$, with $q = 0.5$, $\tau = 1.2$, and $\alpha = 0.9$

n	$f(n)$	$f(n-1)$	$(n-k)^{\alpha}_{(q,\tau)}$	$\nabla^{\alpha}_{q,\tau} f(n)$
1	1.349859	1.000000	1.000	0.349859
2	1.822119	1.349859	1.000	0.472260
3	2.459603	1.822119	1.000	0.637484
4	3.320117	2.459603	1.000	0.860514
5	4.481689	3.320117	1.000	1.161572

Remark 1. *The use of the (q,τ)-deformed discrete fractional calculus is motivated by the need to simultaneously capture memory-dependent dynamics and nonuniform temporal scaling in epidemic systems. Classical discrete epidemic models assume uniform time evolution and local interactions, while many real biological processes, particularly Dengue transmission, depend strongly on delayed immune responses, accumulated infection history, behavioral adaptation, and time-varying intervention effects. Standard fractional operators incorporate memory through nonlocal kernels, but they still rely on fixed temporal structures and cannot fully describe deformation-sensitive dynamics. The proposed (q,τ)-nabla framework extends the classical and fractional discrete models by introducing the deformation parameter q, which controls discrete scaling effects, and the temporal parameter τ, which regulates the memory scale and nonlocal*

interaction structure. Consequently, the (q,τ)-operator provides a unified mechanism for modeling discrete-time evolution, long-memory accumulation, and scale-sensitive transmission behavior within a single framework. This formulation allows the model to reproduce richer epidemic phenomena such as delayed stabilization, oscillatory persistence, bifurcation transitions, and memory-dependent control responses that are difficult to capture using standard integer-order or classical fractional models. Moreover, the framework remains mathematically consistent with existing theories since the limits $q \to 1$ and $\tau \to 1$ recover the standard fractional discrete operator, while $\alpha \to 1$ yields the classical discrete difference model.

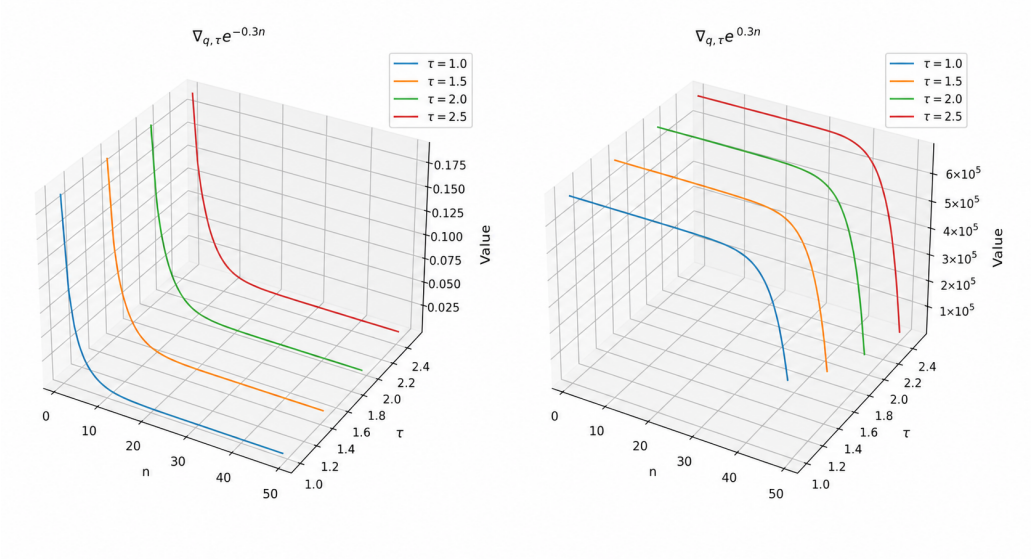


Figure 1. 3D surface plots of the (q,τ)-nabla operator $\nabla_{q,\tau} f(n)$ for $f(n) = e^{-0.3n}$ (left) and $f(n) = e^{+0.3n}$ (right), under varying $\tau = 1.0,\ 1.5,\ 2.0,\ 2.5$ with fixed $q = 0.5$. These illustrate how the (q,τ)-nabla derivative evolves as both function type and memory parameter τ change.

3. Solvability fractional (q,τ)-nabla Dengue symptom model

We examine the development of Dengue symptoms using a discrete-time compartmental model that incorporates (q,τ)-nabla fractional derivatives. The resulting equations are as follows:

$$\begin{aligned} \nabla^{\alpha}_{q,\tau} S_n &= -\beta S_n I_n, \\ \nabla^{\alpha}_{q,\tau} E_n &= \beta S_n I_n - \delta E_n, \\ \nabla^{\alpha}_{q,\tau} I_n &= \delta E_n - (\gamma+\mu) I_n, \\ \nabla^{\alpha}_{q,\tau} R_n &= \gamma I_n, \\ \nabla^{\alpha}_{q,\tau} H_n &= \mu I_n - \xi H_n, \end{aligned} \tag{2}$$

where all parameters $\beta, \delta, \gamma, \mu, \xi$ are non-negative constants, and $\nabla^{\alpha}_{q,\tau}$ indicates the fractional (q,τ)-nabla operator of order $0 < \alpha \leq 1$. These are the initial conditions: $S_0, E_0, I_0, R_0, and\ H_0 \in [0,1]$. The fractional (q,τ)-nabla calculus is used in the model **(2)** to explain the development of Dengue symptoms. **Table 3** presents a definition of the variables and parameters.

Theorem 1 (Existence and Uniqueness of Solutions)**.** *Let the functions on the right-hand side of* **(2)** *be locally Lipschitz and assume that the initial values $(S_0, E_0, I_0, R_0, H_0) \in [0,1]^5$. Then, there exists a unique solution*

$$(S_n, E_n, I_n, R_n, H_n)_{n=0}^{N}$$

to the system **(2)** *for each $N \in \mathbb{N}$.*

Proof. We reformulate system **(2)** in its fractional (q,τ)-nabla integral form:

$$x_n = x_0 + \frac{1}{\Gamma_{q,\tau}(\alpha)} \sum_{k=0}^{n-1} (n-k-1)^{\alpha-1}_{q,\tau} f_k(x_k),$$

where $f_k(x_k)$ represents the right-hand side function for each compartment (e.g., $-\beta S_k I_k$ for S_k) and $(n-k-1)^{\alpha-1}_{q,\tau}$ is the (q,τ)-rising factorial. Define the Banach space:

$$\mathcal{B} = \left\{ x : \mathbb{N}_0 \to \mathbb{R}^5 \mid \|x\|_{\infty} := \sup_n \|x_n\| < \infty \right\},$$

equipped with the supremum norm. We now define an operator $\mathcal{T}$ on $\mathcal{B}$ by:

$$\mathcal{T}[x](n) := x_0 + \frac{1}{\Gamma_{q,\tau}(\alpha)} \sum_{k=0}^{n-1} (n-k-1)^{\alpha-1}_{q,\tau} f_k(x_k).$$

To prove that $\mathcal{T}$ is a contraction, suppose that each f_k is Lipschitz continuous with Lipschitz constant L. Then for $x, y \in \mathcal{B}$,

$$\|\mathcal{T}[x](n) - \mathcal{T}[y](n)\| = \left\| \sum_{k=0}^{n-1} \frac{(n-k-1)_{q,\tau}^{\alpha-1}}{\Gamma_{q,\tau}(\alpha)} \left(f_k(x_k) - f_k(y_k)\right) \right\|$$
$$\leq \frac{L}{\Gamma_{q,\tau}(\alpha)} \sum_{k=0}^{n-1} (n-k-1)_{q,\tau}^{\alpha-1} \|x_k - y_k\|.$$

Taking the supremum over n and define:

$$M := \sup_n \sum_{k=0}^{n-1} (n-k-1)_{q,\tau}^{\alpha-1}.$$

Then we have

$$\|\mathcal{T}[x] - \mathcal{T}[y]\|_\infty \leq \frac{LM}{\Gamma_{q,\tau}(\alpha)} \|x - y\|_\infty.$$

Selecting L and α such that: $\frac{LM}{\Gamma_{q,\tau}(\alpha)} < 1$. Then $\mathcal{T}$ is a contraction, and by Banach's Fixed-Point Theorem, $\mathcal{T}$ has a unique fixed point in $\mathcal{B}$, which is the solution to **(2)**. □

Next, we use the spectral features of the linearized (q,τ)-nabla system to investigate the local asymptotic stability of the disease-free equilibrium (DFE) of the system **(2)**.

Theorem 2 (General Local Stability via the Jacobian Matrix)**.** *Consider the (q,τ)-nabla fractional Dengue system*

$$\begin{aligned}
\nabla_{q,\tau}^{\alpha} S_n &= -\beta S_n I_n, \\
\nabla_{q,\tau}^{\alpha} E_n &= \beta S_n I_n - \delta E_n, \\
\nabla_{q,\tau}^{\alpha} I_n &= \delta E_n - (\gamma + \mu) I_n, \\
\nabla_{q,\tau}^{\alpha} R_n &= \gamma I_n, \\
\nabla_{q,\tau}^{\alpha} H_n &= \mu I_n - \xi H_n,
\end{aligned}$$

where $0 < \alpha \leq 1$ and all parameters are nonnegative.

Let

$$X_n = (S_n, E_n, I_n, R_n, H_n)^T$$

and write the system in the compact form

$$\nabla_{q,\tau}^{\alpha} X_n = F(X_n).$$

Let

$$X^* = (S^*, E^*, I^*, R^*, H^*)$$

be an equilibrium point satisfying $F(X^*) = 0$. Then the local behavior of the system near X^* is determined by the Jacobian matrix

$$J(X^*) = \left[\frac{\partial F_i}{\partial X_j}(X^*) \right]_{i,j=1}^{5}.$$

For the proposed Dengue model, this Jacobian matrix is given by

$$J(X^*) = \begin{pmatrix}
-\beta I^* & 0 & -\beta S^* & 0 & 0 \\
\beta I^* & -\delta & \beta S^* & 0 & 0 \\
0 & \delta & -(\gamma+\mu) & 0 & 0 \\
0 & 0 & \gamma & 0 & 0 \\
0 & 0 & \mu & 0 & -\xi
\end{pmatrix}.$$

If every eigenvalue λ_i of $J(X^*)$ satisfies the fractional stability condition

$$|\arg(\lambda_i)| > \frac{\alpha\pi}{2}, \qquad i = 1, \ldots, 5,$$

then the equilibrium X^* is locally asymptotically stable. If at least one eigenvalue violates this condition, then X^* is locally unstable.

Proof. Let $Y_n = X_n - X^*$ be a small perturbation around the equilibrium point. Since $F(X^*) = 0$, Taylor expansion gives

$$F(X_n) = F(X^* + Y_n) = J(X^*)Y_n + \mathcal{O}(\|Y_n\|^2).$$

Neglecting the higher-order terms, the linearized system becomes

$$\nabla_{q,\tau}^{\alpha} Y_n = J(X^*)Y_n.$$

Therefore, the local stability of the nonlinear system is determined by the spectrum of the matrix $J(X^*)$. For a discrete fractional system of order $0 < \alpha \leq 1$, the equilibrium is locally asymptotically stable whenever the eigenvalues of the linearized matrix satisfy $|\arg(\lambda_i)| > \frac{\alpha\pi}{2}$. Hence, if this condition holds for every eigenvalue of $J(X^*)$, then $Y_n \to 0$ as $n \to \infty$, which implies $X_n \to X^*$. Therefore, X^* is locally asymptotically stable. Conversely, if one eigenvalue does not satisfy the fractional stability sector condition, perturbations in the corresponding eigendirection do not decay, and the equilibrium is locally unstable. □

Theorem 3 (Local Stability of the Disease-Free Equilibrium)**.** *Consider the (q,τ)-nabla fractional Dengue model*

$$\begin{aligned}
\nabla_{q,\tau}^{\alpha} S_n &= -\beta S_n I_n, \\
\nabla_{q,\tau}^{\alpha} E_n &= \beta S_n I_n - \delta E_n, \\
\nabla_{q,\tau}^{\alpha} I_n &= \delta E_n - (\gamma + \mu) I_n, \\
\nabla_{q,\tau}^{\alpha} R_n &= \gamma I_n, \\
\nabla_{q,\tau}^{\alpha} H_n &= \mu I_n - \xi H_n,
\end{aligned}$$

where $0 < \alpha \leq 1$ and

$$\beta, \delta, \gamma, \mu, \xi > 0.$$

Then the disease-free equilibrium

$$X_0^* = (1, 0, 0, 0, 0)$$

is locally asymptotically stable with respect to the infected variables (E_n, I_n, H_n) whenever

$$R_0 := \frac{\beta}{\gamma + \mu} < 1.$$

If $R_0 > 1$, then the disease-free equilibrium is locally unstable.

Table 3. Description of variables, parameters, and operators in the (q,τ)-nabla fractional Dengue model

Symbol	Description
S_n	Number of susceptible individuals at time step n.
E_n	Number of exposed (infected but not yet symptomatic) individuals at time step n.
I_n	Number of infectious (symptomatic) individuals at time step n.
R_n	Number of recovered individuals at time step n.
H_n	Number of hospitalized or severely symptomatic individuals at time step n.
β	Transmission rate of the virus (contact between susceptible and infectious individuals).
δ	Rate of progression from exposed to infectious stage.
γ	Recovery rate from the infectious state.
μ	Rate at which infected individuals develop severe symptoms requiring hospitalization.
ξ	Discharge or death rate from the hospitalized compartment.
$\nabla^{\alpha}_{q,\tau}$	Fractional (q,τ)-nabla operator of order $\alpha \in (0,1]$, defined on a discrete-time domain.
α	Fractional order of the derivative, representing memory effects.
$q \in (0,1)$	Quantum deformation parameter.
$\tau > 0$	Time-scaling (nonlocal memory deformation) parameter.
$(n)_{q,\tau}$	(q,τ)-deformed integer: $(n)_{q,\tau} := \dfrac{1-q^{n\tau}}{1-q^{\tau}}$.
$\Gamma_{q,\tau}(\cdot)$	Generalized (q,τ)-Gamma function used in the definition of the fractional sum and derivative.
$(n-k-1)^{\alpha-1}_{q,\tau}$	(q,τ)-falling factorial of order $\alpha-1$, used as the kernel in the discrete fractional sum.
$\mathcal{B}$	The Banach space of bounded sequences $x : \mathbb{N}_0 \to \mathbb{R}^5$ with norm $\|x\|_{\infty} := \sup_n \|x_n\|$.
$\mathcal{T}$	The operator defined on $\mathcal{B}$ representing the right-hand side of the integral form of the system.
M	A finite constant defined as $M := \sup_n \sum_{k=0}^{n-1} (n-k-1)^{\alpha-1}_{q,\tau}$.
L	Lipschitz constant of the nonlinear functions in the system.
$\mathcal{R}_0 = \dfrac{\beta}{\gamma+\mu}$	Basic reproduction number, representing the average number of new infections caused by one infectious individual in a fully susceptible population.

Proof. Let

$$X_n = (S_n, E_n, I_n, R_n, H_n)^T$$

and write the model as

$$\nabla^{\alpha}_{q,\tau} X_n = F(X_n).$$

The Jacobian matrix of F is

$$J(X) = \begin{pmatrix} -\beta I & 0 & -\beta S & 0 & 0 \\ \beta I & -\delta & \beta S & 0 & 0 \\ 0 & \delta & -(\gamma+\mu) & 0 & 0 \\ 0 & 0 & \gamma & 0 & 0 \\ 0 & 0 & \mu & 0 & -\xi \end{pmatrix}.$$

At the disease-free equilibrium $X_0^* = (1,0,0,0,0)$, this becomes

$$J(X_0^*) = \begin{pmatrix} 0 & 0 & -\beta & 0 & 0 \\ 0 & -\delta & \beta & 0 & 0 \\ 0 & \delta & -(\gamma+\mu) & 0 & 0 \\ 0 & 0 & \gamma & 0 & 0 \\ 0 & 0 & \mu & 0 & -\xi \end{pmatrix}.$$

The variables E_n and I_n determine the local growth or decay of the disease. Hence, the infected subsystem is governed by the matrix

$$J_I = \begin{pmatrix} -\delta & \beta \\ \delta & -(\gamma+\mu) \end{pmatrix}.$$

The characteristic polynomial of J_I is

$$\det(\lambda I - J_I) = \lambda^2 + (\delta+\gamma+\mu)\lambda + \delta(\gamma+\mu-\beta).$$

If $R_0 = \frac{\beta}{\gamma+\mu} < 1$, then $\delta+\gamma+\mu > 0$ and $\delta(\gamma+\mu-\beta) > 0$. Therefore, by the Routh–Hurwitz criterion for a second-order polynomial, both eigenvalues of J_I have negative real parts. Using the

standard sector stability condition for the linearized (q,τ)-nabla fractional system, the infected subsystem is locally asymptotically stable whenever the eigenvalues satisfy $|\arg(\lambda_i)| > \frac{\alpha\pi}{2}$, $i = 1, 2$.

Since the eigenvalues of J_I have negative real parts, this condition is satisfied for every $0 < \alpha \leq 1$. Hence, $E_n \to 0$, $I_n \to 0$. Moreover, the hospitalized equation satisfies

$$\nabla^{\alpha}_{q,\tau} H_n = \mu I_n - \xi H_n.$$

Since $I_n \to 0$ and $\xi > 0$, it follows that $H_n \to 0$.

Thus, the disease-free equilibrium is locally asymptotically stable with respect to the infected and hospitalized compartments. On the other hand, if $R_0 > 1$, then $\delta(\gamma + \mu - \beta) < 0$. Thus, the characteristic polynomial has one positive eigenvalue. Therefore, small perturbations in the infected direction grow, and the disease-free equilibrium is locally unstable. □

We suggest the Dengue symptom progression system with control inputs $u_n = (u_n^{(1)}, u_n^{(2)}, u_n^{(3)}) \in \mathcal{U}$, where $\mathcal{U}$ is the admissible control set such that

$$\mathcal{U} := \left\{ u_n^{(i)} \in [0, u_{\max}^{(i)}] \subset \mathbb{R} \right\}.$$

The state dynamics are governed via the next (q,τ)-nabla fractional discrete system:

$$x_{n+1} = x_n + \frac{1}{\Gamma_{q,\tau}(\alpha)} \sum_{k=0}^{n} (n-k)^{\alpha-1}_{q,\tau} f_k(x_k, u_k), \qquad x_0 = x^{(0)},$$

where $x_n \in \mathbb{R}^5$ is the state vector and f_k indicates the nonlinear functions for the Dengue model. The performance index (cost functional) is formulate by:

$$\mathcal{J}(u) = \sum_{n=0}^{N} \left[L(x_n, u_n) \right],$$

with $L : \mathbb{R}^5 \times \mathcal{U} \to \mathbb{R}_+$ being continuously differentiable.

Theorem 4 (Generalized Pontryagin Maximum Principle for the (q,τ)-Nabla Fractional Dengue System)**.** *Consider the controlled (q,τ)-nabla fractional Dengue system*

$$X_{n+1} = X_0 + \frac{h^{\alpha}}{\Gamma_{q,\tau}(\alpha+1)} \sum_{k=0}^{n} \omega^{(q,\tau,\alpha)}_{n-k} F(X_k, u_k),$$

where

$$X_n = (S_n, E_n, I_n, R_n, H_n)^T, \qquad u_n \in U,$$

and

$$\omega^{(q,\tau,\alpha)}_{n-k} \geq 0$$

denotes the (q,τ)-fractional memory kernel. Let the cost functional be

$$J(u) = \sum_{n=0}^{N} L(X_n, u_n).$$

Suppose that:

(1) $F(X,u)$ and $L(X,u)$ are continuously differentiable,
(2) the admissible control set $U \subset \mathbb{R}^m$ is convex and closed,
(3) the state system admits a unique solution for each admissible control.

If u_n^* is an optimal control with associated state trajectory X_n^*, then there exists an adjoint sequence

$$\lambda_n \in \mathbb{R}^5$$

such that the Hamiltonian

$$H_n(X_n, u_n, \lambda_{n+1}) = L(X_n, u_n) + \lambda_{n+1}^T F(X_n, u_n)$$

satisfies the following conditions:

(1) State equation:

$$X^*_{n+1} = X_0 + \frac{h^{\alpha}}{\Gamma_{q,\tau}(\alpha+1)} \sum_{k=0}^{n} \omega^{(q,\tau,\alpha)}_{n-k} F(X_k^*, u_k^*).$$

(2) Adjoint equation:

$$\lambda_n = \frac{\partial H_n}{\partial X_n} + \frac{h^{\alpha}}{\Gamma_{q,\tau}(\alpha+1)} \sum_{k=n}^{N} \omega^{(q,\tau,\alpha)}_{k-n} \left(\frac{\partial F}{\partial X_n} \right)^T \lambda_{k+1}.$$

(3) Terminal condition:

$$\lambda_{N+1} = 0.$$

(4) Optimality condition:

$$H_n(X_n^*, u_n^*, \lambda_{n+1}) = \min_{u_n \in U} H_n(X_n^*, u_n, \lambda_{n+1}).$$

Proof. Let u_n^* be an optimal control and consider the perturbation

$$u_n^{\varepsilon} = u_n^* + \varepsilon v_n,$$

where v_n is an admissible variation and $|\varepsilon| \ll 1$. Let

$$X_n^{\varepsilon} = X_n^* + \varepsilon Z_n + o(\varepsilon),$$

where Z_n denotes the first variation of the state variable. Substituting into the (q,τ)-fractional state equation gives

$$X^{\varepsilon}_{n+1} = X_0 + \frac{h^{\alpha}}{\Gamma_{q,\tau}(\alpha+1)} \sum_{k=0}^{n} \omega^{(q,\tau,\alpha)}_{n-k} F(X_k^{\varepsilon}, u_k^{\varepsilon}).$$

Using Taylor expansion around (X_k^*, u_k^*),

$$F(X_k^{\varepsilon}, u_k^{\varepsilon}) = F(X_k^*, u_k^*) + \varepsilon \left(\frac{\partial F}{\partial X_k} Z_k + \frac{\partial F}{\partial u_k} v_k \right) + o(\varepsilon).$$

Therefore, the first variation satisfies

$$Z_{n+1} = \frac{h^{\alpha}}{\Gamma_{q,\tau}(\alpha+1)} \sum_{k=0}^{n} \omega^{(q,\tau,\alpha)}_{n-k} \left(\frac{\partial F}{\partial X_k} Z_k + \frac{\partial F}{\partial u_k} v_k \right).$$

Next, the variation of the cost functional is

$$\delta J = \sum_{n=0}^{N} \left(\frac{\partial L}{\partial X_n} Z_n + \frac{\partial L}{\partial u_n} v_n \right).$$

Introduce the adjoint sequence λ_n and define the Hamiltonian

$$H_n = L(X_n, u_n) + \lambda_{n+1}^T F(X_n, u_n).$$

Using the discrete fractional summation-by-parts argument and collecting the coefficients of Z_n, we obtain the adjoint relation

$$\lambda_n = \frac{\partial H_n}{\partial X_n} + \frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)} \sum_{k=n}^{N} \omega_{k-n}^{(q,\tau,\alpha)} \left(\frac{\partial F}{\partial X_n} \right)^T \lambda_{k+1},$$

together with the transversality condition

$$\lambda_{N+1} = 0.$$

Substituting the adjoint equation into δJ eliminates the state variation terms and yields

$$\delta J = \sum_{n=0}^{N} \frac{\partial H_n}{\partial u_n} v_n.$$

Since u_n^* minimizes the cost functional over the admissible control set U, the first-order necessary condition implies

$$\frac{\partial H_n}{\partial u_n} = 0$$

for interior controls, or equivalently,

$$H_n(X_n^*, u_n^*, \lambda_{n+1}) = \min_{u_n \in U} H_n(X_n^*, u_n, \lambda_{n+1}).$$

Therefore, the state equation, adjoint equation, terminal condition, and Hamiltonian minimization condition constitute the generalized Pontryagin Maximum Principle for the (q,τ)-nabla fractional Dengue system. □

Example 2 (Implementation of the Optimal Control Problem)**.** *The (q,τ)-nabla fractional system governs the Dengue symptom progression model. We also include control techniques that reflect hospitalization, treatment, and immunization. Let the state variables be $x_n = (S_n, E_n, I_n, R_n, H_n)$ and control variables be $u_n = (u_n^{(1)}, u_n^{(2)}, u_n^{(3)})$ with bounded values:*

$$0 \le u_n^{(i)} \le u_{\max}^{(i)}.$$

The controlled state equations over $n = 0, 1, \ldots, N$ are suggested as follows:

$$\begin{aligned}
S_{n+1} &= S_n - \left(\beta S_n I_n + u_n^{(1)} S_n \right), \\
E_{n+1} &= E_n + \left(\beta S_n I_n - \delta E_n \right), \\
I_{n+1} &= I_n + \left(\delta E_n - (\gamma + \mu + u_n^{(2)}) I_n \right), \\
R_{n+1} &= R_n + \left(\gamma I_n + u_n^{(1)} S_n + u_n^{(2)} I_n \right), \\
H_{n+1} &= H_n + \left(\mu I_n - \xi H_n - u_n^{(3)} H_n \right),
\end{aligned}$$

where the parameters $\beta, \delta, \gamma, \mu, \xi$ represent transmission, progression, recovery, hospitalization, and discharge rates, respectively.

We define the fractional memory kernel:

$$(n-k)_{q,\tau}^{\alpha-1} := \left(\frac{1 - q^{(n-k)\tau}}{1 - q^\tau} \right)^{\alpha-1}, \quad \text{with } \alpha \in (0,1].$$

A safe normalization is applied:

$$(n-k)_{q,\tau}^{\alpha-1} := 1 \quad \text{for } n = k,$$

to avoid zero raised to negative powers.

The Cost Functional can be considered as follows: the performance index is defined as:

$$\mathcal{J}(u) = \sum_{n=0}^{N} [A_1 I_n + A_2 H_n + \frac{1}{2}(B_1 (u_n^{(1)})^2 + B_2 (u_n^{(2)})^2 + B_3 (u_n^{(3)})^2)],$$

where A_1, A_2 are weights on symptomatic and severe cases and B_1, B_2, B_3 are penalties for the control efforts.

We simulate the model over $N = 30$ time steps with the following initial data:

$$S_0 = 0.99, \quad E_0 = 0.005, \quad I_0 = 0.005, \quad R_0 = 0, \quad H_0 = 0,$$

and control guesses $u_n^{(i)} = 0.3$. The parameters used are:

$$\beta = 0.4, \quad \delta = 0.3, \quad \gamma = 0.2, \quad \mu = 0.1, \quad \xi = 0.1.$$

The total cost functional evaluated from the simulation, when $q = 0.9, \tau = 1, \alpha = 0.9$ is: $\mathcal{J} \approx 0.04685$.

We suggest the Dengue symptom design under fractional (q,τ)-nabla dynamics with control variables $u_n^{(1)}$, $u_n^{(2)}$, and $u_n^{(3)}$, where $0 < \alpha \le 1$, $q \in (0,1)$, and $\tau > 0$. Let the design be:

$$\begin{aligned}
\nabla_{q,\tau}^\alpha S_n &= -\beta S_n I_n - u_n^{(1)} S_n, \\
\nabla_{q,\tau}^\alpha E_n &= \beta S_n I_n - \delta E_n, \\
\nabla_{q,\tau}^\alpha I_n &= \delta E_n - (\gamma + \mu + u_n^{(2)}) I_n, \\
\nabla_{q,\tau}^\alpha R_n &= \gamma I_n + u_n^{(1)} S_n + u_n^{(2)} I_n, \\
\nabla_{q,\tau}^\alpha H_n &= \mu I_n - \xi H_n - u_n^{(3)} H_n.
\end{aligned}$$

Theorem 5 (Lyapunov-Based). *Let the Lyapunov function be formulated by*

$$V_n := \frac{1}{2}I_n^2 + \frac{1}{2}H_n^2.$$

Then, the feedback control laws

$$u_n^{(2)} = \max\left(0, \frac{\delta(I_n^2+H_n^2)+\delta I_n E_n+\mu H_n I_n}{I_n^2} - (\gamma+\mu)\right),$$

$$u_n^{(3)} = \max\left(0, \frac{\delta(I_n^2+H_n^2)+\mu H_n I_n}{H_n^2} - \xi\right),$$

guarantee that

$$\nabla_{q,\tau}^{\alpha} V_n \le -\delta V_n,$$

for all $n \in \mathbb{N}_0$, provided that $I_n, H_n \neq 0$.

Proof. From the definition of V_n, we compute its (q,τ)-nabla derivative utilizing linearity:

$$\nabla_{q,\tau}^{\alpha} V_n = I_n \nabla_{q,\tau}^{\alpha} I_n + H_n \nabla_{q,\tau}^{\alpha} H_n.$$

Substituting from the design

$$\nabla_{q,\tau}^{\alpha} I_n = \delta E_n - (\gamma+\mu+u_n^{(2)})I_n,$$
$$\nabla_{q,\tau}^{\alpha} H_n = \mu I_n - (\xi+u_n^{(3)})H_n,$$

we have

$$\nabla_{q,\tau}^{\alpha} V_n = I_n\left(\delta E_n - (\gamma+\mu+u_n^{(2)})I_n\right) + H_n\left(\mu I_n - (\xi+u_n^{(3)})H_n\right)$$
$$= \delta I_n E_n - (\gamma+\mu+u_n^{(2)})I_n^2 + \mu H_n I_n - (\xi+u_n^{(3)})H_n^2.$$

Now suggest the target inequality:

$$\nabla_{q,\tau}^{\alpha} V_n \le -\delta V_n = -\delta\left(\frac{1}{2}I_n^2 + \frac{1}{2}H_n^2\right).$$

Multiply both sides by 2:

$$2\nabla_{q,\tau}^{\alpha} V_n \le -\delta(I_n^2+H_n^2).$$

From the previous expression, we have

$$2\nabla_{q,\tau}^{\alpha} V_n = 2\delta I_n E_n - 2(\gamma+\mu+u_n^{(2)})I_n^2 +$$
$$2\mu H_n I_n - 2(\xi+u_n^{(3)})H_n^2.$$

To ensure the inequality holds, we require

$$2\delta I_n E_n + 2\mu H_n I_n - 2(\gamma+\mu+u_n^{(2)})I_n^2 -$$
$$2(\xi+u_n^{(3)})H_n^2 \le -\delta(I_n^2+H_n^2).$$

Rewriting the inequality, we obtain

$$-2u_n^{(2)}I_n^2 - 2u_n^{(3)}H_n^2 \le -\delta(I_n^2+H_n^2) -$$
$$2\delta I_n E_n - 2\mu H_n I_n + 2(\gamma+\mu)I_n^2 + 2\xi H_n^2.$$

Thus, to satisfy this inequality, choose:

$$u_n^{(2)} \ge \frac{\delta(I_n^2+H_n^2)+2\delta I_n E_n+2\mu H_n I_n-2(\gamma+\mu)I_n^2-2\xi H_n^2}{2I_n^2},$$

$$u_n^{(3)} \ge \frac{\delta(I_n^2+H_n^2)+2\mu H_n I_n-2(\gamma+\mu)I_n^2-2\xi H_n^2-2\delta I_n E_n}{2H_n^2}.$$

We utilize conservative overestimates and simplify to:

$$u_n^{(2)} = \max\left(0, \frac{\delta(I_n^2+H_n^2)+\delta I_n E_n+\mu H_n I_n}{I_n^2} - (\gamma+\mu)\right),$$

$$u_n^{(3)} = \max\left(0, \frac{\delta(I_n^2+H_n^2)+\mu H_n I_n}{H_n^2} - \xi\right).$$

These control laws ensure that $\nabla_{q,\tau}^{\alpha} V_n \le -\delta V_n$. □

Example 3. *Over $N = 30$ discrete time steps, we model the fractional Dengue symptom model using Lyapunov-based feedback control rules that stabilize hospitalization H_n and infection I_n.*

The definition of the feedback controls is:

$$u_n^{(1)} = \kappa_1 I_n \quad \textit{(awareness control)},$$

$$u_n^{(2)} = \max\left(0, \frac{\delta(I_n^2+H_n^2)+\delta I_n E_n+\mu H_n I_n}{I_n^2} - (\gamma+\mu)\right),$$

$$u_n^{(3)} = \max\left(0, \frac{\delta(I_n^2+H_n^2)+\mu H_n I_n}{H_n^2} - \xi\right),$$

where $\kappa_1 = 0.5$, $\delta = 0.1$, and the system parameters are:

$$\beta = 0.4, \quad \delta = 0.3, \quad \gamma = 0.2, \quad \mu = 0.1,$$
$$\xi = 0.1, \quad q = 0.9, \quad \tau = 1.0, \quad \alpha = 0.9.$$

Initial conditions are:

$$S_0 = 0.99, \quad E_0 = 0.005, \quad I_0 = 0.005,$$
$$R_0 = 0, \quad H_0 = 0.$$

*We present the first few steps of the simulation in **Table 4**.*

*The controls effectively lower hospitalization and infection rates without necessitating constant, intense effort. In response to infection levels, the awareness control $u_n^{(1)}$ is smoothly triggered. Hospitalization and treatment are only initiated when necessary, protecting resources and guaranteeing stability. In the context of fractional (q,τ)-nabla calculus, the simulation results in **Table 4** show how well Lyapunov-based feedback control suppresses symptomatic and hospitalized compartments. The parameters $q = 0.9$, $\tau = 1.0$, and $\alpha = 0.9$ are crucial in describing the simulation's memory and scale-deformation characteristics.*

(1) Fractional Order α: The value $\alpha = 0.9$ governs the strength of memory in the dynamical response. Since $\alpha < 1$, the system exhibits a fading memory effect—recent states contribute more to current dynamics, enabling the control law to adjust to disease progression with partial historical influence. This enhances stabilization without overreacting to transient fluctuations.

(2) Quantum Deformation Parameter q: With $q = 0.9$, the (q,τ)-factorial structure smoothens the growth of fractional sums, maintaining near-classical dynamics while still accommodating quantum-like deformation. This avoids oscillatory instability and ensures the weighted summation of previous steps remains stable and convergent.

(3) Time-Scaling Parameter τ: The choice $\tau = 1.0$ implies a standard scale for the memory kernel. Larger values of τ would stretch memory over

longer intervals, whereas smaller τ would localize the response. Setting $\tau = 1.0$ maintains consistency with physical time and matches the natural scale of symptom progression.

The values of (q, τ, α) collectively determine the behavior of the fractional operator $\nabla^{\alpha}_{q,\tau}$, impacting the incorporation of memory and the propagation of control actions over time. These variables were specifically selected for this simulation in order to strike a balance between maintaining numerical stability and capturing nonlocal effects. The appropriateness of this parameter regime for epidemiological modeling with memory and feedback is confirmed by the steady performance over time steps and the presence of constrained and smoothly triggered controls.

For $\tau = 2$, the system maintains its historical effect in ***Table 5****, resulting in a smoother but delayed decline in I_n. Although it may provide robustness to noise, this prolonged memory attenuates abrupt control responses, necessitating more forceful intervention to attain the same suppression rate. This sensitivity is further demonstrated by the behavior of H_n. The hospitalization curve shows a slower convergence and more oscillatory behavior for $\tau = 2$. According to this, higher levels of τ may intensify the impacts of previous infections, necessitating careful adjustment of feedback gains to prevent instability or control saturation. In the end, higher τ enhances long-term memory integrity, but it may also decrease responsiveness. Thus, in epidemic control techniques with memory, τ is a crucial design parameter that controls an equilibrium between stability, responsiveness, and resilience.*

Table 4. Selected results from Lyapunov-based control simulation

Time	S_n	E_n	I_n	R_n	H_n	$u_n^{(1)}$	$u_n^{(2)}$	$u_n^{(3)}$
0	0.990000	0.005000	0.005000	0.000000	0.000000	0.002500	0.0000	0.0000
1	0.985545	0.005480	0.005000	0.003475	0.000500	0.002500	0.0000	11.000
2	0.981110	0.005807	0.005144	0.006939	−0.004550	0.002572	0.0000	0.0000
3	0.976568	0.006084	0.005343	0.010491	−0.003581	0.002671	0.0000	0.0000
4	0.971872	0.006346	0.005565	0.014169	−0.002688	0.002783	0.0000	0.0000

Table 5. Simulation with fractional (q, τ)-memory, $\tau = 2$, $q = 0.9$, $\alpha = 0.9$

Time	S_n	E_n	I_n	R_n	H_n
0	0.990000	0.005000	0.005000	0.000000	0.000000
1	0.985545	0.005480	0.005000	0.003475	0.000500
2	0.985230	0.005431	0.005080	0.003723	-0.002530
3	0.984828	0.005454	0.005098	0.004038	-0.001436
4	0.984393	0.005484	0.005113	0.004378	-0.000933

4. Bifurcation and chaos control analysis

In this section, we discuss the possible emergence of bifurcation and chaotic oscillations in the (q, τ)-nabla fractional Dengue system. The transmission rate β is selected as the main bifurcation parameter, while the remaining parameters are kept fixed. Biologically, increasing β strengthens the infection feedback mechanism and may change the long-term behavior of the infected population from convergence to oscillation and then to irregular fluctuation. Let I_n denote the infected human population at time step n. Numerical bifurcation analysis is performed by recording the asymptotic values of I_n after discarding the transient iterations. For small values of β, the infection trajectory approaches an equilibrium. As β increases, the equilibrium may lose stability and a periodic orbit appears. Further increase of β may generate a sequence of period-doubling transitions, leading to chaotic-type oscillations. The memory parameters q, τ, and α affect the location of the critical bifurcation point. In particular, smaller fractional order α and larger memory scale τ may delay damping and produce richer oscillatory behavior. To suppress chaotic oscillations, we introduce a Pyragas-type delayed feedback control of the form

$$u_n^c = -K(I_n - I_{n-\Delta}),$$

where $K > 0$ is the feedback gain and Δ is the delay length. This control is non-invasive on a Δ-periodic orbit, because it vanishes whenever $I_n = I_{n-\Delta}$. Hence, the control does not change the desired periodic solution but can stabilize it by damping deviations from it.

Theorem 6 (Local Bifurcation and Pyragas Control)**.** *Consider the discrete (q,τ)-nabla fractional Dengue system and assume that the infection dynamics can be written in the reduced form*

$$I_{n+1} = F_\beta(I_n, I_{n-1}, \dots, I_0),$$

where F_β is continuously differentiable with respect to the infection history and the bifurcation parameter β. Let I^* be a positive endemic equilibrium. Suppose that the linearized characteristic multiplier $\lambda(\beta)$ associated with I^* satisfies

$$\lambda(\beta_c) = -1, \qquad \frac{d\lambda}{d\beta}(\beta_c) \neq 0.$$

Then the endemic equilibrium undergoes a local period-doubling bifurcation at $\beta = \beta_c$. Moreover, assume that for $\beta > \beta_c$ the uncontrolled system possesses an unstable periodic orbit of period Δ. If the Pyragas-type control

$$u_n^c = -K(I_n - I_{n-\Delta})$$

is added to the infection equation, then there exists an interval $K \in (K_1, K_2)$ such that the corresponding Δ-periodic orbit becomes locally asymptotically stable.

Proof. Let I^* be an endemic equilibrium of the infection subsystem. Linearizing the reduced infection equation around I^* gives

$$\eta_{n+1} = A(\beta)\eta_n + \text{higher-order terms},$$

where $\eta_n = I_n - I^*$ and $A(\beta)$ is the effective linear multiplier generated by the (q,τ)-nabla memory kernel and the epidemiological parameters. A period-doubling bifurcation occurs when the dominant multiplier crosses the value -1. By assumption, $\lambda(\beta_c) = -1, \quad \lambda'(\beta_c) \neq 0$. The second condition is the transversality condition, which means that the multiplier crosses the unit circle instead of merely touching it. Therefore, by the standard discrete bifurcation theorem, the equilibrium loses stability at $\beta = \beta_c$ and a local period-two orbit is generated. This proves the first part. Now suppose that the uncontrolled system admits an unstable periodic orbit satisfying $I_n^p = I_{n-\Delta}^p$. For this orbit, the Pyragas control satisfies $u_n^c = -K(I_n^p - I_{n-\Delta}^p) = 0$. Hence, the controller is non-invasive and does not change the periodic solution. Let $e_n = I_n - I_n^p$ be a perturbation from the periodic orbit. Linearizing the controlled system around the periodic orbit gives a delayed error equation of the form

$$e_{n+1} = a_n e_n - K b_n (e_n - e_{n-\Delta}) + \text{higher-order terms},$$

where a_n and b_n are bounded coefficients along the periodic orbit. The term $-K(e_n - e_{n-\Delta})$ penalizes deviations from the desired periodic motion. For suitable values of K, the characteristic multipliers of the linearized delayed system lie inside the unit disk. Hence,

$$e_n \to 0 \quad \text{as} \quad n \to \infty.$$

Therefore, the periodic orbit becomes locally asymptotically stable under the Pyragas feedback control. □

4.1. General bifurcation map

To address the bifurcation behavior in a general form, we consider the transmission rate β as the main bifurcation parameter, while the remaining parameters are fixed. The (q,τ)-nabla fractional Dengue system may be written as

$$\nabla_{q,\tau}^{\alpha} X_n = F(X_n; \beta), \qquad X_n = (S_n, E_n, I_n, R_n, H_n)^T.$$

Using the equivalent memory-dependent recurrence form, one obtains

$$X_{n+1} = X_0 + \frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)} \sum_{k=0}^{n} \omega_{n-k}^{(q,\tau,\alpha)} F(X_k; \beta),$$

where

$$\omega_{n-k}^{(q,\tau,\alpha)} = \left((n-k+1)_{q,\tau}^{\alpha} - (n-k)_{q,\tau}^{\alpha}\right),$$

$$(n-k)_{q,\tau} = \frac{1-q^{(n-k)\tau}}{1-q^\tau}.$$

For each value of β, the asymptotic values of a selected state variable, usually the infected or hospitalized class, are recorded after removing transient iterations. Hence, the general bifurcation map is defined by

$$\mathcal{B}_I = \{(\beta, I_n) : \beta \in [\beta_{\min}, \beta_{\max}],\ n = N_{\text{tr}}, \dots, N\},$$

or, equivalently, for the hospitalized class,

$$\mathcal{B}_H = \{(\beta, H_n) : \beta \in [\beta_{\min}, \beta_{\max}],\ n = N_{\text{tr}}, \dots, N\}.$$

Here, N_{tr} denotes the number of transient iterations discarded before plotting the long-term dynamics. Therefore, the bifurcation map is not tied to a single numerical example, but is generated by the general solution operator

$$\Phi_{q,\tau,\alpha}^{\beta} : X_0 \mapsto X_n(\beta),$$

where

$$X_n(\beta) = \Phi_{q,\tau,\alpha}^{\beta}(X_0).$$

The bifurcation diagram is then obtained by plotting

$$\beta \longmapsto I_n(\beta) \quad \text{or} \quad \beta \longmapsto H_n(\beta)$$

for the post-transient values of n. A local bifurcation may occur at a critical value $\beta = \beta_c$ when the dominant characteristic multiplier of the linearized memory map crosses the stability boundary. In particular, a period-doubling bifurcation occurs if

$$\lambda(\beta_c) = -1, \qquad \frac{d\lambda}{d\beta}(\beta_c) \neq 0.$$

Equivalently, for the Jacobian matrix $J(X^*;\beta)$ evaluated at an equilibrium X^*, the critical values are determined by

$$\det\left(\lambda I - J(X^*;\beta)\right) = 0,$$

together with the boundary condition

$$|\lambda| = 1$$

in the discrete case, or by the corresponding fractional sector condition

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

for the fractional linearized system. Thus, the general bifurcation set is given by

$$\mathcal{C} = \{\beta_c : \exists \lambda \in \sigma(J(X^*;\beta_c)) \text{ such that } \lambda \text{ lies on the stability boundary}\}.$$

This provides a general mathematical construction of the bifurcation map for the present (q,τ)-fractional Dengue system.

4.2. Example: Local bifurcation and pyragas control

Consider the (q,τ)-nabla fractional Dengue system

$$\begin{aligned}
\nabla^{\alpha}_{q,\tau} S_n &= -\beta S_n I_n,\\
\nabla^{\alpha}_{q,\tau} E_n &= \beta S_n I_n - \delta E_n,\\
\nabla^{\alpha}_{q,\tau} I_n &= \delta E_n - (\gamma+\mu) I_n,\\
\nabla^{\alpha}_{q,\tau} R_n &= \gamma I_n,\\
\nabla^{\alpha}_{q,\tau} H_n &= \mu I_n - \xi H_n.
\end{aligned}$$

Here, H_n represents hospitalized or severe symptomatic individuals. We take β as the bifurcation parameter and assume that all other parameters are fixed. To control oscillations in the severe-symptom class, we introduce the Pyragas feedback $u^c_n = -K(H_n - H_{n-\Delta})$, where $K > 0$ is the feedback gain and Δ is the delay. The controlled hospitalized equation becomes

$$\nabla^{\alpha}_{q,\tau} H_n = \mu I_n - \xi H_n - K(H_n - H_{n-\Delta}).$$

For illustration, choose

$$\alpha = 0.85, \quad q = 0.6, \quad \tau = 1.2, \quad \delta = 0.25,$$
$$\gamma = 0.12, \quad \mu = 0.04, \quad \xi = 0.15.$$

Let the initial data be

$$S_0 = 0.90, \quad E_0 = 0.05, \quad I_0 = 0.04,$$
$$R_0 = 0, \quad H_0 = 0.01.$$

For small transmission rate, for example $\beta = 0.8$, the infected and hospitalized populations approach a stable equilibrium. When β is increased, the endemic equilibrium may lose stability. Near a critical value $\beta = \beta_c$, the dominant multiplier of the linearized system crosses -1, producing a local period-doubling bifurcation. For larger values such as $\beta = 2.8$, the sequence H_n may display irregular oscillations. When the feedback gain is applied, for example $K = 0.35$, $\Delta = 2$, the control term suppresses the difference between H_n and H_{n-2}. Thus, the chaotic or irregular hospitalized trajectory is stabilized into a period-two orbit. If instead $\Delta = 1$, the same controller may stabilize the trajectory toward an equilibrium.

Theorem 7 (Local Control of Severe Dengue Oscillations)**.** *Assume that the reduced infection–hospitalization subsystem has a positive endemic equilibrium and that the dominant characteristic multiplier satisfies $\lambda(\beta_c) = -1$, $\lambda'(\beta_c) \neq 0$. Then the system undergoes a local period-doubling bifurcation at $\beta = \beta_c$. Furthermore, if the hospitalized sequence H_n admits an unstable periodic orbit of period Δ for $\beta > \beta_c$, then the Pyragas controller*

$$u^c_n = -K(H_n - H_{n-\Delta})$$

locally stabilizes this periodic orbit for a suitable interval of gains $K \in (K_1, K_2)$.

Proof. The first claim follows from the standard discrete period-doubling criterion. After linearization around the endemic equilibrium, the stability is determined by the characteristic multipliers of the infection–hospitalization subsystem. When the dominant multiplier crosses the unit circle through -1 and the transversality condition $\lambda'(\beta_c) \neq 0$ holds, a local period-two orbit is generated. For the control part, let H^p_n be a periodic orbit of period Δ. Then $H^p_n = H^p_{n-\Delta}$. Hence, $u^c_n = -K(H^p_n - H^p_{n-\Delta}) = 0$. Therefore, the controller is non-invasive on the target orbit. Let $e_n = H_n - H^p_n$. Linearizing the controlled hospitalization equation gives

$$\nabla^{\alpha}_{q,\tau} e_n = -\xi e_n - K(e_n - e_{n-\Delta}) + \mu\eta_n,$$

where $\eta_n = I_n - I^p_n$. For suitable $K > 0$, the delayed feedback term damps the error $e_n - e_{n-\Delta}$. If the corresponding linearized multipliers lie inside the unit disk, then

$$e_n \to 0 \quad \text{as} \quad n \to \infty.$$

Thus, the periodic severe-symptom orbit is locally asymptotically stable. □

Theorem 8 (Positivity of Solutions)**.** *Assume that*

$$S_0, E_0, I_0, R_0, H_0 \geq 0, \qquad \beta, \delta, \gamma, \mu, \xi \geq 0.$$

Let the discrete (q,τ)-fractional scheme be written in the form

$$X_{n+1} = X_n + \frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)} \sum_{k=0}^{n} \omega_{n-k}^{(q,\tau,\alpha)} F(X_k),$$

where

$$\omega_{n-k}^{(q,\tau,\alpha)} \geq 0.$$

If the step size and parameters satisfy the positivity restrictions

$$\frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}\beta I_n \leq 1,$$
$$\frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}\delta \leq 1,$$
$$\frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}(\gamma+\mu) \leq 1,$$

and

$$\frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}\xi \leq 1,$$

then every solution starting from nonnegative initial data remains nonnegative:

$$S_n, E_n, I_n, R_n, H_n \geq 0, \qquad n \geq 0.$$

Proof. The proof follows by induction. Assume that

$$S_k, E_k, I_k, R_k, H_k \geq 0, \qquad 0 \leq k \leq n.$$

Since the (q,τ)-fractional weights satisfy

$$\omega_{n-k}^{(q,\tau,\alpha)} = \left((n-k+1)_{q,\tau}^{\alpha} - (n-k)_{q,\tau}^{\alpha}\right), \qquad 0 \leq k \leq n,$$

where

$$(m)_{q,\tau} = \frac{1-q^{m\tau}}{1-q^\tau}, \qquad m \geq 0.$$

the memory contribution is a nonnegative weighted accumulation of previous states. For the susceptible class, the update has the form

$$S_{n+1} = S_n - \frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}\beta S_n I_n.$$

Hence, we obtain

$$S_{n+1} = S_n\left(1 - \frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}\beta I_n\right).$$

By the assumed restriction,

$$1 - \frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}\beta I_n \geq 0.$$

Therefore, $S_{n+1} \geq 0$. Similarly,

$$E_{n+1} = E_n + \frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}\left(\beta S_n I_n - \delta E_n\right),$$

so

$$E_{n+1} = E_n\left(1 - \frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}\delta\right) + \frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}\beta S_n I_n.$$

Both terms are nonnegative; hence $E_{n+1} \geq 0$. For the infected class,

$$I_{n+1} = I_n + \frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}\left(\delta E_n - (\gamma+\mu) I_n\right),$$

which gives

$$I_{n+1} = I_n\left(1 - \frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}(\gamma+\mu)\right) + \frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}\delta E_n.$$

Thus, $I_{n+1} \geq 0$. For the recovered class,

$$R_{n+1} = R_n + \frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}\gamma I_n \geq 0.$$

Finally, for the hospitalized class,

$$H_{n+1} = H_n + \frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}\left(\mu I_n - \xi H_n\right),$$

or equivalently,

$$H_{n+1} = H_n\left(1 - \frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}\xi\right) + \frac{h^\alpha}{\Gamma_{q,\tau}(\alpha+1)}\mu I_n.$$

Hence, $H_{n+1} \geq 0$. Therefore, by induction,

$$S_n, E_n, I_n, R_n, H_n \geq 0$$

for all $n \geq 0$. Thus, the nonnegative cone $\mathbb{R}_+^5$ is positively invariant under the proposed (q,τ)-fractional Dengue dynamics. □

5. Fractional threshold stability

Theorem 9 (Fractional Threshold Stability)**.** *Consider the (q,τ)-nabla Dengue model with memory order $0 < \alpha \leq 1$, and initial conditions $S_0 \in (0,1), I_0 > 0$, and $H_0 \geq 0$. Define the fractional threshold parameter as*

$$\mathcal{R}_0^{(q,\tau,\alpha)} := \frac{\beta \cdot M_{q,\tau}(\alpha)}{\gamma+\mu},$$

where $M_{q,\tau}(\alpha)$ is the effective memory accumulation kernel. Then:

(1) If $\mathcal{R}_0^{(q,\tau,\alpha)} < 1$, the disease-free equilibrium $(S_n, I_n, H_n) = (1,0,0)$ is locally asymptotically stable.
(2) If $\mathcal{R}_0^{(q,\tau,\alpha)} > 1$, the disease persists and nonzero endemic equilibria may emerge.

Proof. We linearize the (q,τ)-nabla Dengue model around the disease-free equilibrium $S_n = 1, I_n = 0, H_n = 0$. The infected equation under memory becomes:

$$\nabla_{q,\tau}^{\alpha} I_n = \sum_{j=1}^{n} w_j^{(q,\tau,\alpha)}\left[\delta\beta S_{n-j} I_{n-j} - (\gamma+\mu) I_{n-j}\right],$$

where $w_j^{(q,\tau,\alpha)} := \left(\frac{1-q^{j\tau}}{1-q^\tau}\right)^{\alpha-1}$ are the fractional weights. At the disease-free equilibrium, $S_{n-j} \approx 1$,

so we approximate:

$$\nabla^{\alpha}_{q,\tau} I_n \approx \sum_{j=1}^{n} w_j^{(q,\tau,\alpha)} \left[(\beta-\gamma-\mu) I_{n-j}\right].$$

We define the convolutional memory operator:

$$M_{q,\tau}(\alpha) := \sum_{j=1}^{\infty} w_j^{(q,\tau,\alpha)}.$$

Assuming a perturbation $I_n = \varepsilon \rho^n$, the characteristic equation becomes

$$\nabla^{\alpha}_{q,\tau} I_n \approx \varepsilon \rho^n (\beta-\gamma-\mu) M_{q,\tau}(\alpha).$$

Stability requires $|\rho| < 1$, which holds if

$$\beta M_{q,\tau}(\alpha) < \gamma+\mu \quad \Leftrightarrow \quad \mathcal{R}_0^{(q,\tau,\alpha)} < 1.$$

In this instance, all perturbations decrease in norm, and the memory-averaged reproduction rate is not high enough to support growth. As a result, the infection-free state On the other hand, if $\mathcal{R}_0^{(q,\tau,\alpha)} > 1$, memory accumulation causes the system to permit nonzero attractors or oscillatory states. □

Theorem 10 (Fractional Threshold and Endemic Equilibrium)**.** *Suppose the S_n, I_n, H_n represent the susceptible, infected, and hospitalized populations in a fractional (q,τ)-nabla Dengue system of order $0 < \alpha \leq 1$. Define the memory-adjusted reproduction number:*

$$\mathcal{R}_0^{(q,\tau,\alpha)} := \frac{\beta M_{q,\tau}(\alpha)}{\gamma+\mu},$$

where

$$M_{q,\tau}(\alpha) = \sum_{j=1}^{\infty} \left(\frac{1-q^{j\tau}}{1-q^{\tau}}\right)^{\alpha-1}.$$

Then:

(1) If $\mathcal{R}_0^{(q,\tau,\alpha)} < 1$, the disease-free equilibrium $(S^*, I^*, H^*) = (1,0,0)$ is locally asymptotically stable.

(2) If $\mathcal{R}_0^{(q,\tau,\alpha)} > 1$, there exists a unique endemic equilibrium (S^*, I^*, H^*) approximately given by:

$$S^* \approx \frac{1}{\mathcal{R}_0^{(q,\tau,\alpha)}}, \quad I^* \approx \frac{\eta(\mathcal{R}_0^{(q,\tau,\alpha)}-1)}{\beta\delta}, \quad H^* \approx \frac{\mu}{\eta} I^*.$$

Proof. Examine the initial Dengue system that is still in memory, where a (q,τ)-nabla fractional difference operator is involved in every dynamic equation:

$$\nabla^{\alpha}_{q,\tau} X_n := \sum_{j=1}^{n} w_j^{(q,\tau,\alpha)} \Delta X_{n-j},$$

with weights $w_j^{(q,\tau,\alpha)} = \left(\frac{1-q^{j\tau}}{1-q^{\tau}}\right)^{\alpha-1}$. Setting the fractional derivatives to zero will allow us to investigate the equilibrium points:

$$\nabla^{\alpha}_{q,\tau} S_n = \nabla^{\alpha}_{q,\tau} I_n = \nabla^{\alpha}_{q,\tau} H_n = 0.$$

From the Dengue model equations:

$$\begin{aligned} 0 &= -\delta\beta S^* I^*, \\ 0 &= \delta\beta S^* I^* - (\gamma+\mu) I^*, \\ 0 &= \mu I^* - \eta H^*. \end{aligned}$$

From the first equation, either $S^* = 0$, $I^* = 0$, or $\delta\beta = 0$. We discard trivial cases, so assume $I^* > 0$. Then:

$$\delta\beta S^* = \gamma+\mu \quad \Rightarrow \quad S^* = \frac{\gamma+\mu}{\delta\beta}.$$

Substitute into the definition of $\mathcal{R}_0^{(q,\tau,\alpha)}$:

$$\mathcal{R}_0^{(q,\tau,\alpha)} = \frac{\beta M_{q,\tau}(\alpha)}{\gamma+\mu} \quad \Rightarrow \quad S^* = \frac{1}{\mathcal{R}_0^{(q,\tau,\alpha)}}.$$

Now, solve for I^*, we have

$$I^* = \frac{\delta\beta S^* I^*}{\gamma+\mu} \quad \Rightarrow \quad I^* = \frac{\eta(\mathcal{R}_0^{(q,\tau,\alpha)}-1)}{\beta\delta},$$

as $S^* = 1/\mathcal{R}_0^{(q,\tau,\alpha)}$ implies $\delta\beta S^* = \gamma+\mu$.

From the third equation:

$$H^* = \frac{\mu}{\eta} I^*.$$

Hence, if $\mathcal{R}_0^{(q,\tau,\alpha)} > 1$, then $I^* > 0$, $H^* > 0$, and the point (S^*, I^*, H^*) defines a biologically feasible endemic equilibrium. Local stability can be studied by computing the Jacobian matrix of the full fractional system and analyzing its memory-weighted spectral radius. For small perturbations around (S^*, I^*, H^*), if the eigenvalues (adjusted for (q,τ)-weights) lie inside the unit disk, the equilibrium is stable. □

We linearize the (q,τ)-nabla Dengue system around the endemic equilibrium point

$$(S^*, I^*, H^*) = \left(\frac{\gamma+\mu}{\delta\beta}, I^*, \frac{\mu}{\eta} I^*\right).$$

The right-hand side of the system is:

$$\begin{aligned} f_1 &= -\delta\beta SI, \\ f_2 &= \delta\beta SI - (\gamma+\mu) I, \\ f_3 &= \mu I - \eta H. \end{aligned}$$

The Jacobian matrix J with respect to variables (S, I, H) is given by:

$$J = \begin{bmatrix} -\delta\beta I & -\delta\beta S & 0 \\ \delta\beta I & \delta\beta S - (\gamma+\mu) & 0 \\ 0 & \mu & -\eta \end{bmatrix}.$$

Substituting the equilibrium values

$$S^* = \frac{\gamma+\mu}{\delta\beta}, \quad H^* = \frac{\mu}{\eta} I^*,$$

we obtain

$$J = \begin{bmatrix} -\delta\beta I^* & -(\gamma+\mu) & 0 \\ \delta\beta I^* & 0 & 0 \\ 0 & \mu & -\eta \end{bmatrix}.$$

The characteristic polynomial is obtained from $\det(J-\lambda I)=0$, yielding eigenvalues:

$$\lambda_1 = -\eta,$$
$$\lambda_{2,3} = -\frac{\delta\beta I^*}{2} \pm \frac{1}{2}\sqrt{\delta\beta I^* \left(\delta\beta I^* - 4(\gamma+\mu)\right)}.$$

5.1. Stability of the endemic equilibrium

The Jacobian matrix's $\lambda_{2,3}$ eigenvalues determine how stable the endemic equilibrium is. The endemic equilibrium is locally asymptotically stable and exhibits spiral sink behavior if $\delta\beta I^* < 4(\gamma+\mu)$. This is because the square root term becomes imaginary, resulting in complex conjugate eigenvalues with negative real parts. On the other hand, the eigenvalues are real and one may turn positive if $\delta\beta I^* > 4(\gamma+\mu)$, which could cause local instability or the start of bifurcation. Because the reproduction number $\mathcal{R}_0^{(q,\tau,\alpha)}$ makes the infected equilibrium value I^* memory-dependent, the memory effects represented by (q,τ,α) are crucial in determining the stability action of the system.

Example 4 (Numerical Example: Jacobian and Stability at Endemic Equilibrium)**.** *We consider the (q,τ)-nabla fractional Dengue model with the following parameters:*

$$\delta = 0.4, \quad \beta = 0.6, \quad \gamma = 0.1, \quad \mu = 0.05,$$
$$\eta = 0.2, \quad I^* = 0.12.$$

Substituting into the Jacobian matrix evaluated at the endemic equilibrium yields:

$$J = \begin{bmatrix} -0.0288 & -0.15 & 0 \\ 0.0288 & 0 & 0 \\ 0 & 0.05 & -0.2 \end{bmatrix}.$$

The eigenvalues of J are:

$$\lambda_1 = -0.0144 + 0.0641i, \quad \lambda_2 = -0.0144 - 0.0641i,$$
$$\lambda_3 = -0.2.$$

Every eigenvalue has a real part that is negative. Rapid decline in the hospitalized class is confirmed by $\lambda_3 = -0.2$, whereas damped oscillatory behavior near the endemic equilibrium is indicated by the complex conjugate pair $\lambda_{1,2}$. The endemic equilibrium is locally asymptotically stable under the specified conditions. The system is not destabilized by memory or fractional effects, although they do affect the rate of convergence.

Under the chosen parameters, all variables converge to a stable endemic equilibrium (**Figure 2**). The infected population I_n exhibits mild transient oscillations before stabilizing, while the hospitalized class H_n initially rises and then decays toward equilibrium. The susceptible population S_n decreases and levels off due to depletion and memory effects. The phase portraits (**Figure 3**) confirm stability: the I_n–S_n trajectory shows a damped spiral toward a fixed point, and the H_n–I_n plane reflects delayed hospitalization dynamics driven by memory. These results validate the theoretical stability analysis and highlight the role of (q,τ) scaling and fractional memory α in shaping long-term epidemic behavior.

5.2. Optimal control theorem for the (q,τ)-nabla Dengue model

In a controlled (q,τ)-nabla Dengue model of order $0<\alpha\le 1$, let S_n, I_n, H_n be the susceptible, infected, and hospitalized populations. The control $u_n \in [0, u_{\max}]$ represents an intervention attempt (e.g., vaccine or fumigation). The dynamics of the state are:

$$\nabla^\alpha_{q,\tau} S_n = -\delta\beta(1-u_n)S_n I_n,$$
$$\nabla^\alpha_{q,\tau} I_n = \delta\beta(1-u_n)S_n I_n - (\gamma+\mu)I_n,$$
$$\nabla^\alpha_{q,\tau} H_n = \mu I_n - \eta H_n.$$

We define the cost functional:

$$J[u] = \sum_{n=0}^{T} \left[AI_n^2 + Bu_n^2\right](n)^\alpha_{(q,\tau)},$$

where $A, B > 0$ are weights, and $(n)^\alpha_{(q,\tau)}$ is the generalized (q,τ)-power to model memory-weighted costs.

Theorem 11 (Fractional Optimal Control—Necessary Conditions)**.** *Let u_n^* be an optimal control minimizing $J[u]$, subject to the dynamics and bounds $0 \le u_n \le u_{\max}$. Then there exist adjoint variables $\lambda_n^S, \lambda_n^I, \lambda_n^H$ satisfying the backward (q,τ)-adjoint system:*
$\nabla^\alpha_{q,\tau}\lambda_n^S = \delta\beta(1-u_n)I_n(\lambda_n^S - \lambda_n^I)$,
$\nabla^\alpha_{q,\tau}\lambda_n^I = -2AI_n(n)^\alpha_{(q,\tau)} + \delta\beta(1-u_n)S_n(\lambda_n^S-\lambda_n^I) + (\gamma+\mu)\lambda_n^I - \mu\lambda_n^H$,
$\nabla^\alpha_{q,\tau}\lambda_n^H = \eta\lambda_n^H$,
with transversality conditions $\lambda_T^S = \lambda_T^I = \lambda_T^H = 0$, and the optimal control satisfies the pointwise minimum condition:

$$u_n^* = \min\left\{\max\left\{0, \frac{\delta\beta S_n I_n(\lambda_n^I - \lambda_n^S)}{2B}\right\}, u_{\max}\right\}.$$

Proof. We form the Hamiltonian:

$$\mathcal{H}_n = AI_n^2(n)^\alpha_{(q,\tau)} + Bu_n^2(n)^\alpha_{(q,\tau)} + \lambda_n^S\left[-\delta\beta(1-u_n)S_nI_n\right]$$
$$+\lambda_n^I\left[\delta\beta(1-u_n)S_nI_n - (\gamma+\mu)I_n\right] + \lambda_n^H\left[\mu I_n - \eta H_n\right].$$

The following prerequisites must be met for optimality in the discrete-time fractional framework: Joint dynamics and state dynamics (previously provided): The optimality criterion is to minimize $\mathcal{H}_n$ with respect to u_n pointwise in n. The backward (q,τ)-nabla difference of the costate is equal to minus the partial derivative of $\mathcal{H}_n$ with respect to the state variables. Computing gradients:

$$\frac{\partial \mathcal{H}_n}{\partial u_n} = 2Bu_n(n)^{\alpha}_{(q,\tau)} + \delta\beta S_n I_n(\lambda^S_n - \lambda^I_n).$$

Setting $\frac{\partial \mathcal{H}_n}{\partial u_n} = 0$ gives the unconstrained minimizer:

$$u_n = \frac{\delta\beta S_n I_n(\lambda^I_n - \lambda^S_n)}{2B(n)^{\alpha}_{(q,\tau)}}.$$

After enforcing the box constraint $0 \le u_n \le u_{\max}$, we obtain:

$$u^*_n = \min\left\{\max\left\{0, \frac{\delta\beta S_n I_n(\lambda^I_n - \lambda^S_n)}{2B(n)^{\alpha}_{(q,\tau)}}\right\}, u_{\max}\right\}.$$

The adjoint equations follow by setting:

$$\nabla^{\alpha}_{q,\tau}\lambda^X_n = -\frac{\partial \mathcal{H}_n}{\partial X_n}, \quad X \in \{S, I, H\}.$$

□

Example 5. *Assume **Table 6** describing our application.*

The simulation results in **Table 7** show that, during the initial time steps, the susceptible population S_n gradually decreases while the infected population I_n increases and then begins to stabilize, with a corresponding rise in the hospitalized class H_n. The optimal control u_n remains inactive in the early stages, reflecting a delayed intervention strategy driven by the (q,τ)-memory effect, which balances intervention cost against infection reduction. As the infection grows, control is activated illustrating that the fractional memory-based framework yields more efficient and cost-effective intervention strategies compared to classical models.

5.3. Biological interpretation of compartments and parameters

The proposed (q,τ)-fractional Dengue model divides the human population into five epidemiological compartments. The susceptible population S_n represents individuals who are healthy but vulnerable to Dengue infection through contact with infectious mosquitoes or infected individuals. The exposed compartment E_n consists of individuals who have been infected but are still in the incubation stage and are not yet fully symptomatic. The infected class I_n represents actively infectious individuals capable of transmitting the disease. The recovered population R_n contains individuals who have gained temporary or permanent immunity after recovery. The hospitalized compartment H_n describes severe or critical cases requiring medical treatment or hospitalization.

The parameter β denotes the effective transmission rate of the disease and measures the interaction strength between susceptible and infectious individuals. The parameter δ represents the progression rate from the exposed stage to the infectious stage, reflecting the average incubation period of the virus. The recovery rate is denoted by γ, while μ represents the hospitalization or severe-symptom progression rate from the infected class. The parameter ξ corresponds to the discharge or removal rate from hospitalization.

The fractional order $\alpha \in (0,1]$ characterizes the memory strength of the epidemic process. Smaller values of α imply stronger memory effects and slower fading of past infection history. The deformation parameter $q \in (0,1)$ governs the discrete scaling and quantum-like deformation of the temporal dynamics, whereas the parameter $\tau > 0$ controls the temporal memory scale and the degree of nonlocal time deformation. Together, the parameters (q,τ,α) regulate how previous epidemic states influence the current disease evolution and intervention response.

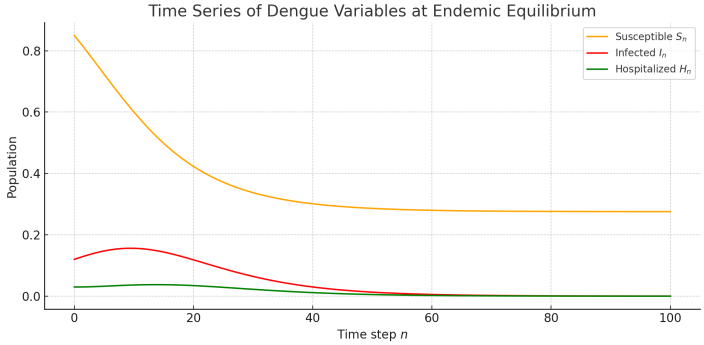


Figure 2. Dengue system variables' time series across 100 iterations: hospitalized H_n, infected I_n, and susceptible S_n. Local stability is confirmed when the trajectories converge to the endemic equilibrium.

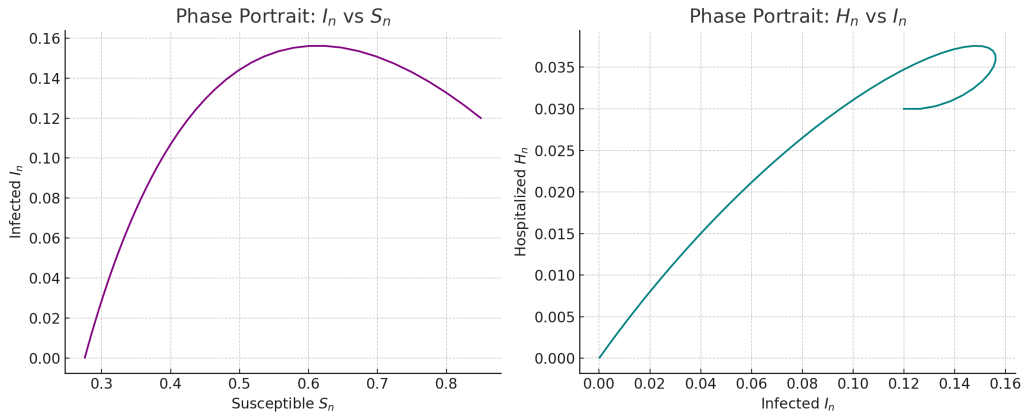


Figure 3. Dengue system phase portraits as follows: (Left) Hospitalized vs. Infected (H_n vs. I_n); (Right) Infected vs. Susceptible (I_n vs. S_n). The endemic equilibrium is shown to be approached via convergence in both graphs.

Table 6. Simulation parameters, initial conditions, and cost weights for the optimal control problem

Parameter	**Value**	**Description**
δ	0.4	Effective contact rate
β	0.6	Transmission efficiency
γ	0.1	Natural recovery rate
μ	0.05	Hospitalization rate
η	0.2	Hospital recovery rate
q	0.9	Quantum time scale deformation parameter
τ	1.0	Fractional shift parameter
α	0.9	Order of the fractional nabla operator
A	1.0	Weight for infected individuals in cost functional
B	0.1	Weight for control effort in cost functional
$u_{\max}$	1.0	Maximum allowed control level
T	50	Time horizon (steps)
S_0	0.85	Initial susceptible population
I_0	0.12	Initial infected population
H_0	0.03	Initial hospitalized population

Table 7. Optimal control simulation of the (q, τ)-nabla fractional Dengue desing over the first 10 time steps.

$\boldsymbol{n}$	$\boldsymbol{S_n}$	$\boldsymbol{I_n}$	$\boldsymbol{H_n}$	$\boldsymbol{u_n}$
0	0.850000	0.120000	0.030000	0.000
1	0.825520	0.126480	0.030000	0.000
2	0.800461	0.132567	0.030324	0.000
3	0.774994	0.138149	0.030888	0.000
4	0.749298	0.143122	0.031617	0.000
5	0.723562	0.147397	0.032455	0.000
6	0.697972	0.150904	0.033362	0.000
7	0.672699	0.153593	0.034312	0.000
8	0.647902	0.155432	0.035282	0.000
9	0.623720	0.156408	0.036256	0.000
10	0.600270	0.156520	0.037223	0.000

6. Conclusion

In this study, we proposed a discrete-time fractional Dengue model based on the (q, τ)-nabla operator, incorporating both time-scale deformation and nonlocal memory effects. We established existence and stability of solutions, analyzed the endemic equilibrium via the Jacobian, and derived a fractional threshold condition for disease persistence. A Lyapunov-based approach ensured stability of infected and hospitalized populations, while a memory-aware optimal control problem was formulated using an extended Pontryagin principle. Numerical results validated the theory and demonstrated the effectiveness of the control strategy. Future work will focus on efficient numerical schemes and real-data calibration to enhance practical applicability.

Future research directions may focus on extending the proposed (q, τ)-nabla fractional Dengue framework toward data-driven and multi-scale epidemic intelligence systems. One possible direction is the integration of real epidemiological datasets, climate variability, and mosquito mobility patterns to improve predictive accuracy and parameter identification for q, τ, and α. The model may also be generalized to stochastic and spatially distributed epidemic networks, where uncertainty, migration, and regional heterogeneity play significant roles in disease transmission. Another important extension involves coupling the (q, τ)-fractional dynamics with machine learning and physics-informed neural networks to construct adaptive forecasting and real-time control strategies. In addition, future work may investigate multi-strain Dengue interactions, co-infection mechanisms, vaccination optimization, and hospital resource allocation under memory-dependent dynamics. From the mathematical viewpoint, further theoretical developments may include global bifurcation analysis, synchronization phenomena, entropy-based stability criteria, and efficient numerical algorithms for large-scale (q, τ)-fractional systems with long-memory effects.

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Conflicts of Interest

The author declares that there are no competing interests, financial or otherwise.

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Availability of data

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AI tools statement

All authors confirm that no AI tools were used in the preparation of this manuscript.

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