Dengue fever has a huge impact on people’s physical, social, and economic lives in low-income locations worldwide. Researchers use epidemic models to better understand the transmission patterns of dengue fever in order to recommend effective preventative measures and give data for vaccine and treatment development. We use fractional calculus to organise the transmission phenomena of dengue fever, including immunisation, reinfection, therapy, and asymptomatic carriers. In addition, we focused our study on the dynamical behavior and qualitative approach of dengue infection. The existence and uniqueness of the solution of the suggested dengue dynamics are inspected through the fixed point theorems of Schaefer and Banach. The Ulam-Hyers stability of the suggested dengue model is established. To illustrate the contribution of the input factors on the system of dengue infection, the solution paths are studied using the Laplace Adomian decomposition approach. Furthermore, numerical simulations are used to show the effects of fractional-order, immunity loss, vaccination, asymptotic fraction, biting rate, and therapy. We have established that asymptomatic carriers, bite rates, and immunity loss rates are all important factors that might make controlling more challenging. The intensity of dengue fever may be controlled by reducing mosquito bite rates, whereas the asymptotic fraction is risky and can transmit the illness to noninfected regions. Vaccination, fractional order, index of memory, and medication can be employed as proper control parameters.

1. Introduction

Dengue infection is a well-known tropical illness transmitted by female Aedes aegypti mosquitoes and generated by dengue viruses. Due to global warming, dengue disease has extended to approximately 128 nations around the globe, posing a threat to public health and the economy [1]. Dengue virus causes headaches, joint pain, high fever, vomiting, nausea, lower back pain, extreme weakness, muscle pain, rash, bone pain, pain behind the eyes, red eyes, and extreme exhaustion. After sucking blood, the virus is transferred to a mosquito, which then distributes the infection to others. After then, the mosquitoes are diseased for the remainder of their lives, and there have been a few cases of vertical transmission.
dengue virus transmission [2, 3]. Dengue vaccines have been developed in large part due to the surge in popularity of dengue virus infection in recent decades. Vaccinations against dengue illness are available in some countries [4, 5], but completely effective vaccines have yet to be launched. The researcher proposes several control measures for preventing dengue illness, but additional research is required to develop viable techniques.

It is obvious that mathematical models play an important role in conceptualising infectious illness transmission processes and successfully investigating illness dynamics for control strategies [6]. The most important components of the transmission phenomena of various illnesses may be identified through mathematical modeling. Esteva and Vargas developed the basic dengue fever modelling approach; they used a changing human population in their model and evaluated the system's stability [7, 8]. The influence of vaccination and antibody-dependent enhancement (ADE) in the phenomena of dengue fever has been hypothesized in [9, 10], while the scientists in [11, 12] organised the transmissions of dengue and tested the stability of equilibrium of the suggested system. Asymptomatic carriers of dengue illness are frequently reported and developed by scientists in the suggested system. Asymptomatic carriers of dengue disease are frequently reported and developed by scientists in [13, 14]; these carriers are most dangerous for noninfected individuals. In [9, 10], while the scientists in [11, 12] organised the transmission phenomena of various illnesses may be hypothesized model. Memory is a key factor in the transmission phenomena of dengue fever, which can be accurately represented through fractional-calculus. To be more specific, the researchers focused on fractional systems due to its wide applications. The rudimentary notions are given as follows.

**Definition 1** (see [23]). Assume \( b(t) \) be a function with the condition \( b(t) \in L^1([g, h], R) \) and take the fractional order \( h \), then

\[
h\int_g^h b(t) = \frac{1}{\Gamma(h)} \int_0^h (t - r)^{h-1} b(r) dr
\]

represents fractional integral and \( 0 < h \leq 1 \).

**Definition 2** (see [23]). Consider \( b(t) \) be a function with \( b(t) \in C^n(g, h] \), then

\[
\frac{D_{g}^{h}}{[n-h]} b(t) = \frac{1}{\Gamma(n-h)} \int_0^n (t - r)^{n-h} b^n(r) dr
\]

represents the renowned derivative of Caputo.

**Lemma 3** (see [23]). Assume a function \( b(h) \) and take the below system

\[
\begin{cases}
\frac{D_{g}^{h}}{[n-h]} b(t) = \nu(t), t \in [0, \tau], n - 1 < h < n, \\
b(0) = v_0,
\end{cases}
\]

where \( \nu(t) \in C([0, \tau]) \), then

\[
b(t) = \sum_{i=0}^{n-1} d_i t^i, \text{for } i = 0, 1, \ldots, n - 1, \quad d_i \in R.\]

**Definition 4** (see [24]). The following is the Laplace transform for the Caputo operator.

\[
\mathcal{L} \left( \frac{D_{g}^{h}}{[n-h]} b(t) \right) = s^n b(s) - \sum_{k=0}^{n-1} s^{n-k-1} b^k(0),
\]

with \( n - 1 < h < n \). In addition to this, take the norm on \( \mathcal{X} \) as

\[
\|b\| = \max_{t \in [0, \tau]} \{ |b(t)|, \text{for all } b \in \mathcal{X} \}.
\]
\textbf{Theorem 5} (see [25]). Assume \( \mathcal{X} \) to be a Banach space in a way that \( G: \mathcal{X} \to \mathcal{X} \) is compact and continuous. Then, one can find a fixed point of \( G \), if
\[
\mathcal{X} = \{ b \in \mathcal{X} : b = \lambda G b, \lambda \in (0, 1) \}
\]  
(7)
is bounded.

\section*{3. Formulation of Dengue Dynamics}

In this formulation, we construct the interactions of female vectors \( N_v \) and hosts \( N_h \) to indicate the transmission process of dengue fever. The vector size is categorized into \((S_h)\) susceptible, \((E_h)\) exposed, and \((I_h)\) infected compartments while the host population is divided into \((S_{h1})\) susceptible, \((S_{h2})\) susceptible after losing immunity, \((E_{h1})\) exposed, \((I_{hA})\) asymptomatic, and \((R_h)\) recovered compartments. We indicated the rate of transmission from \( S_h \) to \( E_h \) by \(((b\beta/\mu E_h)(I_h + I_{hA}))\) while the rate of transmission from susceptible \((S_{h1})\) and susceptible \((S_{h2})\) to \( E_h \) are represented by \( b\beta_{h1}/\mu E_h \) and \( b\beta_{h2}/\mu E_h \). In addition to this, the transfer rate from \( E_h \) and \( I_h \) is symbolised by \( \rho_h \) and \( \rho_r \). The natural birth and mortality rates were assumed to remain constant for both populations, which are denoted by \( \mu_r \) and \( \mu_h \) for vector and host, accordingly.

A portion \( \psi \) is assumed to be asymptomatic, and recovery term is specified by \( \gamma \) from the infected classes. The terms \( r \) and \( p \) represent the treatment and vaccination rates, whereas \( \beta \) represents the vector bite rate. Furthermore, a term \( \nu \) of the \( R_h \) class loses immunity and becomes \( S_{h2} \) with a lower transmission rate \( \beta_{h2} \), resulting in \( \beta_{h2} < \beta_{h1} \). Then, we have below dynamics of dengue

\[
\begin{align*}
0_D^\beta S_{h1} &= \mu S_{h1} \frac{b\beta_{h1}}{\mu} - \beta_{h1} S_{h1} I_v - p S_{h1} - \mu S_{h1}, \\
0_D^\beta S_{h2} &= \mu S_{h2} - \beta_{h2} S_{h2} I_v - \mu S_{h2}, \\
0_D^\beta E_h &= \mu \frac{b\beta}{\mu} - \beta_{h1} I_v - \mu E_h, \\
0_D^\beta I_h &= (1 - \psi)\rho E_h - \left( r + \gamma + \mu \right) I_h, \\
0_D^\beta I_{hA} &= \psi \rho E_h - \left( \mu + \nu \right) I_{hA}, \\
0_D^\beta R_h &= p S_{h1} + r I_h + \gamma (I_h + I_{hA}) - \left( \mu + \nu \right) R_h, \\
0_D^\beta S_v &= \mu S_v - \beta \frac{b\beta}{\mu} (I_h + I_{hA}) S_v - \mu S_v, \\
0_D^\beta E_v &= \beta \frac{b\beta}{\mu} (I_h + I_{hA}) S_v - \left( \mu + \nu \right) E_v, \\
0_D^\beta I_v &= \mu E_v - \mu I_v,
\end{align*}
\]  

where
\[
\begin{align*}
S_h(0) &= S_{h1}(0) + S_{h2}(0) \\
E_h(0) &= E_{h1}(0) + E_{h2}(0) \\
I_h(0) &= I_{hA}(0) + I_h(0) \\
S_{h1}(0) &= 0, \\
S_{h2}(0) &= 0, \\
E_{h1}(0) &= 0, \\
E_{h2}(0) &= 0, \\
I_{hA}(0) &= 0, \\
I_h(0) &= 0.
\end{align*}
\]  

(9)

Furthermore, the strength of species is
\[
\begin{align*}
N_v &= S_v + E_v + I_v, \\
N_h &= S_{h1} + S_{h2} + E_h + I_h + I_{hA} + R_h.
\end{align*}
\]  

(10)

Liouville-Caputo’s operator is denoted by \( 0_L^\beta \), while the memory index is denoted by \( \theta \). Because biological processes are nonlocal, the outcomes of fractional systems are more dependable and precise; also, fractional systems contain hereditary properties and convey knowledge about their past and present states for the future. Because it is commonly known that Caputo’s derivative is more trustworthy and versatile for analysis, we used a fractional framework to depict the dynamics of dengue disease.

\textbf{Theorem 6.} The solutions \((S_{h1}, S_{h2}, E_h, I_h, I_{hA}, R_h, S_v, E_v, I_v)\) of the fractional model (8) of dengue infection are positive and bounded.

\textbf{Proof.} In order to obtain the required result, we proceed as follows:

\[
\begin{align*}
0_L^\beta S_{h1} \bigg|_{\theta=0} &= \mu S_{h1} \geq 0, \\
0_L^\beta S_{h2} \bigg|_{\theta=0} &= \mu S_{h2} \geq 0, \\
0_L^\beta E_h \bigg|_{\theta=0} &= \beta \frac{b\beta}{\mu} - \beta_{h1} S_{h1} I_v - \mu E_h \geq 0, \\
0_L^\beta I_h \bigg|_{\theta=0} &= \psi \rho E_h - \left( \mu + \nu \right) I_h \geq 0, \\
0_L^\beta I_{hA} \bigg|_{\theta=0} &= \rho \left( 1 - \psi \right) E_h \geq 0, \\
0_L^\beta R_h \bigg|_{\theta=0} &= p S_{h1} + r I_h + \gamma (I_h + I_{hA}) - \left( \mu + \nu \right) R_h \geq 0, \\
0_L^\beta S_v \bigg|_{\theta=0} &= \mu S_v \geq 0, \\
0_L^\beta E_v \bigg|_{\theta=0} &= \beta \frac{b\beta}{\mu} (I_h + I_{hA}) S_v - \left( \mu + \nu \right) E_v \geq 0, \\
0_L^\beta I_v \bigg|_{\theta=0} &= \mu E_v \geq 0.
\end{align*}
\]  

(11)
Hence, our fractional system (8) is positive. To show that the solution is bounded, we first add all the compartments of host population as

\[
0 \leq \mathcal{M} - \mu_{h}^{\beta}(S_{h1} + S_{h2} + E_{h} + I_{h} + I_{hA} + R_{h})
\]

Thus, our fractional system (8) is positive. To show that the solution is bounded, we first add all the compartments of host population as

\[
0 = \mathcal{M} - \mu_{h}^{\beta}(S_{h1} + S_{h2} + E_{h} + I_{h} + I_{hA} + R_{h})
\]

We get the following by asymptotic behavior of Mittag-Leffler function [23]:

\[
(S_{h1} + S_{h2} + E_{h} + I_{h} + I_{hA} + R_{h}) \leq \mathcal{M} \equiv \mathcal{M}_{1}.
\]

In the same way, we can take the compartments of vector population of the system (8); we have \(\mathcal{M}_{2} \equiv \mathcal{M}/\mu_{v}^{\beta} \), in which \(\mathcal{M}_{2} = \mathcal{N}/\mu_{v}^{\beta} \). Consequently, the solution of the system (8) is positive and bounded.

Our suggested fractional model (8) of dengue infection’s disease-free stable state is represented by \(\mathcal{E}_{0}(S_{h1}, S_{h2}, E_{h}, I_{h}, I_{hA}, R_{h}, S_{v}, E_{v}, I_{v})\) and is provided by

\[
\mathcal{E}_{0} = (\mathcal{L}_{1}, \mathcal{L}_{2}, 0, 0, 0, 0),
\]

where \(\mathcal{L}_{1} = \mu_{h}^{\beta}N_{h}/p^{\beta} + \mu_{h}^{\beta}, \mathcal{L}_{2} = \nu_{h}^{0}p^{\beta} + (\mu_{i}^{h} + \mu_{h}^{\beta}),\) and \(\mathcal{L}_{3} = \mu_{v}^{\beta}N_{v}^{0}/(p^{\beta} + \mu_{i}^{h}) (\nu_{v}^{0} + \mu_{h}^{\beta}).\) In this study, we mainly focused on the dynamical behavior and qualitative analysis of the infection; however, stability, sensitivity, bifurcation, and optimal control will be explored in our future work.

\[\square\]

4. Theory of Existence

The qualitative character of the suggested fractional model (8) of dengue disease will be examined in this phase of the study using existence theory. To accomplish so, we must follow the instructions outlined below:
We can also rewrite the system (16) as

\[
\begin{cases}
L^0_{t, \delta}x(t) = \mathcal{L}(x(t), \mathcal{P}(t)), & t \in [0, r], \\
\mathcal{P}(0) = \mathcal{P}_0, & 0 < \delta \leq 1,
\end{cases}
\]  

(17)

where

\[
\begin{align*}
\mathcal{P}(t) &= \begin{cases}
S_{h1}(t), \\
S_{h2}(t), \\
E_h(t), \\
I_h(t), \\
I_{ha}(t), \\
R_h(t), \\
S_v(t), \\
E_v(t), \\
I_v(t), \\
I_0(t),
\end{cases} \\
\mathcal{P}_0(t) &= \begin{cases}
S_{h10}, \\
S_{h20}, \\
E_{h0}, \\
I_{h0}, \\
I_{ha0}, \\
R_{h0}, \\
S_{v0}, \\
E_{v0}, \\
I_{v0}, \\
I_{00},
\end{cases}
\]

\[
\mathcal{L}(t, \mathcal{P}(t)) = \begin{cases}
\mathcal{P}_1(t, S_{h1}, S_{h2}, E_h, I_h, I_{ha}, R_h, S_v, E_v, I_v), \\
\mathcal{P}_2(t, S_{h1}, S_{h2}, E_h, I_h, I_{ha}, R_h, S_v, E_v, I_v), \\
\mathcal{P}_3(t, S_{h1}, S_{h2}, E_h, I_h, I_{ha}, R_h, S_v, E_v, I_v), \\
\mathcal{P}_4(t, S_{h1}, S_{h2}, E_h, I_h, I_{ha}, R_h, S_v, E_v, I_v), \\
\mathcal{P}_5(t, S_{h1}, S_{h2}, E_h, I_h, I_{ha}, R_h, S_v, E_v, I_v), \\
\mathcal{P}_6(t, S_{h1}, S_{h2}, E_h, I_h, I_{ha}, R_h, S_v, E_v, I_v), \\
\mathcal{P}_7(t, S_{h1}, S_{h2}, E_h, I_h, I_{ha}, R_h, S_v, E_v, I_v), \\
\mathcal{P}_8(t, S_{h1}, S_{h2}, E_h, I_h, I_{ha}, R_h, S_v, E_v, I_v), \\
\mathcal{P}_9(t, S_{h1}, S_{h2}, E_h, I_h, I_{ha}, R_h, S_v, E_v, I_v).
\end{cases}
\]

(18)

For the examination of our suggested system, we use the Lipschitz criteria listed as follows:

C1. For \( q \in [0, 1) \), there exists \( \mathcal{U}_X, \mathcal{V}_X \) with the following

\[
|\mathcal{L}(t, \mathcal{P}(t))| \leq \mathcal{U}_X|\mathcal{P}|^q + \mathcal{V}_X. 
\]

(20)

C2. We have \( M_X > 0 \), and all \( \mathcal{P}, \tilde{\mathcal{P}} \in \mathcal{X} \), with the condition

\[
|\mathcal{L}(t, \mathcal{P}) - \mathcal{L}(t, \tilde{\mathcal{P}})| \leq M_X \|\mathcal{P} - \tilde{\mathcal{P}}\|. 
\]

(21)

Introduce a map \( B \) on \( \mathcal{X} \) as given below:

\[
B\mathcal{P}(t) = \mathcal{P}_0(t) + \frac{1}{I(\delta)} \int_0^t (t - r)^q \mathcal{L}(r, \mathcal{P}(r)) dr. 
\]

(22)

There is at least a solution of (17), if the C1 and C2 holds true. To investigate the solution of our dengue system, we proceed as follows.

**Theorem 7.** There exists at least one solution of the suggested model (8) of dengue fever if the assumptions C1 and C2 satisfied.

**Proof.** We will utilize Schaefer’s fixed point theorem to show the needed result. We will demonstrate this theorem in four phases, as follows:

P1. In first phase, the continuity of the operator \( B \) will be established. Take here, \( \mathcal{P}_i \) is continuous for \( i = 1, 2, \ldots, 9 \); this gives us that \( \mathcal{L}(t, \mathcal{P}(t)) \) is continuous. In the upcoming steps, \( \mathcal{P}_i, \mathcal{P} \in \mathcal{X} \) such that \( \mathcal{P}_i \rightarrow \mathcal{P} \), we have \( B\mathcal{P}_i \rightarrow B\mathcal{P} \). In addition to this, assume

\[
\|B\mathcal{P}_i - B\mathcal{P}\| = \max_{r \in [0, r]} \left| \frac{1}{I(\delta)} \int_0^t (t - r)^q \mathcal{L}(r, \mathcal{P}(r)) dr \right|
\]

\[
- \frac{1}{I(\delta)} \int_0^t (t - s)^q \mathcal{L}(s, \mathcal{P}(s)) ds
\]

\[
\leq \max_{r \in [0, r]} \left| \frac{1}{I(\delta)} \int_0^t (t - r)^q \mathcal{L}(r, \mathcal{P}(r)) dr \right|
\]

\[
\leq \frac{t^q M_X}{I(\delta + 1)} \|\mathcal{P}_i - \mathcal{P}\| 
\rightarrow 0 \text{ as } j \rightarrow \infty.
\]

(23)

The continuity of \( B\mathcal{P}_i \rightarrow B\mathcal{P} \) is achieved as \( \mathcal{X} \) is continuous which insure the continuity of \( B \).

P2. In the second phase, the boundedness of \( B \) will be established. Let us take \( \mathcal{P} \in X \), then the following are satisfied through the operator \( B \):
\[\|B\mathcal{P}\| = \max_{t \in [0, \tau]} \left| \mathcal{P}_0(t) + \frac{1}{\Gamma(\theta)} \int_0^t (t-r)^{\theta-1} \mathcal{I}(r, \mathcal{P}(r)) \, dr \right| \]
\[\leq \|\mathcal{P}\| \max_{t \in [0, \tau]} \frac{1}{\Gamma(\theta)} \int_0^t (t-r)^{\theta-1} \|\mathcal{I}(r, \mathcal{P}(r))\| \, dr \]
\[\leq \|\mathcal{P}\| + \frac{\tau^\theta}{\Gamma(\theta+1)} \{U_x\|\mathcal{P}\|^\eta + V_x\}. \]

(24)

Next, the boundedness of \(B(T)\) will be established for a bounded subset \(T\) of \(\mathcal{X}\). Assume \(\mathcal{P} \in T\) as \(S\) is bounded; as a result of this, there is a \(U \geq 0\) such that
\[\|\mathcal{P}\| = U. \forall \mathcal{P} \in T. \tag{25}\]

As a result, we get the following through the above for any \(\mathcal{P} \in T:\)
\[\|BW\| \leq \|\mathcal{P}\| \leq \frac{\tau^\theta}{\Gamma(\theta+1)} \{U_x\|\mathcal{P}\|^\eta + V_x\}. \tag{26}\]

Consequently, the boundedness of the operator \(B(T)\) is obtained.

P3. For the equi-continuity, take \(t_1, t_2 \in [0, \tau]\) with \(t_1 \geq t_2\), then we have
\[|B\mathcal{P}(t_1) - B\mathcal{P}(t_2)| = \frac{1}{\Gamma(\theta)} \int_0^{t_1} (t_1-r)^{\theta-1} |\mathcal{I}(r, \mathcal{P}(r))| \, dr - \frac{1}{\Gamma(\theta)} \int_0^{t_2} (t_2-r)^{\theta-1} |\mathcal{I}(r, \mathcal{P}(r))| \, dr \leq \frac{\tau^\theta}{\Gamma(\theta+1)} \{U_x\|\mathcal{P}\|^\eta + V_x\} [t_1^\theta - t_2^\theta], \tag{27}\]

which goes to zero as \(t_1\) goes to \(t_2\). This insures the relative compactness of \(B(T)\) through Arzela-Ascoli theorem.

P4. In fourth phase, the following set is considered:
\[\mathcal{E} = \{ \mathcal{P} \in \mathcal{X} : \mathcal{P} = \lambda B\mathcal{P}, \lambda \in (0, 1) \}. \tag{28}\]

To show that set \(\mathcal{E}\) is bounded, we assume \(\mathcal{P} \in \mathcal{E}\); then for any \(t \in [0, \tau]\), the below condition satisfies
\[\|\mathcal{P}\| = \lambda \|B\mathcal{P}\| \leq \lambda \left[ \frac{\tau^\theta}{\Gamma(\theta+1)} \{U_x\|\mathcal{P}\|^\eta + V_x\} \right]. \tag{29}\]

This indicates that the set \(\mathcal{E}\) is bounded. As a result of Schaefer’s theorem, the operator \(B\) has a fixed point. Consequently, our suggested system (17) of dengue has at least one solution.

Remark 8. If \(C_1\) fulfills for \(q = 1\), then Theorem 7 can be proved for \((\tau^\theta U_x/\Gamma(\theta+1)) < 1\).

Theorem 9. If \((\tau^\theta U_x/\Gamma(\theta+1)) < 1\) is satisfied, then the dengue fever fractional system (17) has a unique solution.

Proof. For the proof, Banach’s contraction theorem is applied with the assumption \(\mathcal{P}, \bar{\mathcal{P}} \in \mathcal{X}\) as
\[\|B\mathcal{P} - B\bar{\mathcal{P}}\| \leq \frac{\max_{t \in [0, \tau]} \{U_x\|\mathcal{P}\|^\eta + V_x\}}{\Gamma(\theta+1)} \tag{30}\]
\[\leq \frac{\tau^\theta U_x}{\Gamma(\theta+1)} \|\mathcal{P} - \bar{\mathcal{P}}\|, \]

Thus, there is a unique fixed point of \(B\); therefore, a unique of model (17) of dengue fever exists.

5. Ulam-Hyers Stability

Here, our main focus is to investigate the suggested model of dengue for the Ulam-Hyers stability (UHS). First, Ulam proposed the concept of stability in 1940, and Hyers [26, 27] expanded it. Several researchers have applied the Ulam-Hyers stability concept to several fields of study [28–30]. The fundamental theory is as follows.

Let us consider \(\mathcal{H} : \mathcal{X} \rightarrow \mathcal{X}\) in a way that
\[\mathcal{H}\mathcal{V} = \mathcal{V} \text{ for } \mathcal{V} \in \mathcal{X}. \tag{31}\]

Definition 10. Upper mentioned (31) is UHS if for every solution \(\mathcal{P} \in \mathcal{X}\) and \(\zeta > 0\), one can find
\[\|\mathcal{V} - \mathcal{H}\mathcal{V}\| \leq \zeta \text{ for } t \in [0, \tau]. \tag{32}\]

Furthermore, there exists a unique solution \(\bar{\mathcal{P}}\) of the upper mentioned (31) in a way that \(0 < C_q\) and the following holds true
\[\|\mathcal{V} - \bar{\mathcal{P}}\| \leq C_q \zeta, \quad t \in [0, \tau]. \tag{33}\]

Definition 11. Let \(\mathcal{P}\) and \(\bar{\mathcal{P}}\) be solution of (31); then, the system (31) is generalized UHS if
\[\|\mathcal{V} - \bar{\mathcal{P}}\| \leq \mathcal{L}(\zeta), \tag{34}\]

in which the image of 0 is 0 and \(\mathcal{X} \in C(\mathbb{R},\mathbb{R})\).

Remark 12. If the solution \(\bar{\mathcal{P}} \in \mathcal{X}\) satisfies (33) and for all \(t \in [0, \tau]\) the below satisfied
(a) \(|\phi(t)| \leq \zeta, \text{ in which } \phi \in C([0, \tau] ; \mathbb{R})\)
(b) \(\mathcal{H}\mathcal{V}(T) = \mathcal{V} + \phi(T)\)
Then, system (17) after small changes becomes as

\[
\begin{aligned}
D_0^\frac{\tau}{\tau^3} \Psi(t) &= \mathcal{P}(t, \Psi(t)) + \omega(t), \\
\Psi(0) &= \Psi_0.
\end{aligned}
\]  

(35)

Lemma 13. System (35) also fulfills

\[
|\Psi(t) - T\Psi(t)| \leq a\xi, \quad \text{in which } a = \frac{\tau^3}{\Gamma(\theta + 1)}.
\]

(36)

Utilizing Lemma 3 and Remark 12, we can easily prove this theorem.

Theorem 14. If the condition \((\tau^3 L_p/\Gamma(\theta + 1)) < 1\) holds true, then the solution of (17) is UHS and generalized UHS on Lemma 13.

Proof. We assume the solutions \(\Psi \in X\) and \(\Psi \in X\) of the system (17) for the required proof, thus

\[
\begin{aligned}
|\Psi(t) - T\Psi(t)| &= |\Psi(t) - T\Psi(t)| \\
&\leq |\Psi(t) - T\Psi(t)| \\
&\leq a\xi + \frac{\tau^3 L_p}{\Gamma(\xi + 1)} |\Psi(t) - T\Psi(t)| \\
&\leq \frac{a\xi}{1 - (\tau^3 L_p/\Gamma(\xi + 1))}.
\end{aligned}
\]  

(37)

Thus, the UHS and GUHS of the suggested nonlinear system (17) of dengue fever are insured. □

Definition 15. The solution of (31) is the Ulam-Hyers-Rassias stable (UHRS) if for any \(\Psi \in \mathcal{X}\), we write

\[
||\Psi - \Psi^*|| \leq \Omega(t)\xi, \quad \text{for } t \in [0, \tau],
\]

(38)

where \(\Omega \in C[0, \tau, R]\) and \(\xi > 0\). If \(\xi > 0\), then there exists a unique solution \(\Psi^*\) of the system (31) satisfying

\[
||\Psi^* - \Psi|| \leq C_\Omega \Omega(t)\xi,
\]

(39)

for all \(t\) in \([0, \tau]\).

Definition 16. Take the unique solution \(\Psi^*\) and \(\mathcal{P}\) be any other solution of (31) such that

\[
||\Psi^* - \Psi|| \leq C_{\xi, \Omega} \Omega(t)\xi,
\]

(40)

in which \(t\) belongs to \([0, \tau]\), \(\Omega \in D([0, \tau, R])\) in a way that \(C_{\xi, \Omega}\) and \(\xi > 0\). This implies that the solution of (31) is generalized UHRS.

Remark 17. Take \(\Psi \in X\); this solution will satisfy (33) if \(\forall \ t \in [0, \tau]\), we write

(a) \(|\omega(t)| \leq \xi \Omega(t)\), in which \(\omega(t) \in \mathcal{C}([0, \tau]; R)

(b) \(\Psi \Psi^*(t) = \Psi^* + \omega(t)

Lemma 18. The perturb system (17) holds the conditions

\[
|\Psi(t) - T\Psi(t)| \leq a\xi, \quad \text{in which } a = \frac{\tau^3}{\Gamma(\theta + 1)}.
\]

(41)

Utilizing Lemma 3 and Remark 17, we can easily obtained the required proof.

Theorem 19. The solution of (17) is UHRS and generalized UHRS on Lemma 18 if \((\tau^3 L_p/\Gamma(\theta + 1)) < 1\) holds true.

Proof. Assuming a unique solution \(\Psi \in \mathcal{X}\) and any other system (17) solution \(\Psi \in \mathcal{X}\), we get that

\[
|\Psi(t) - T\Psi(t)| = |\Psi(t) - T\Psi(t)| \leq |\Psi(t) - T\Psi(t)| \\
\leq |\Psi(t) - T\Psi(t)| \\
\leq a\xi + \frac{\tau^3 L_p}{\Gamma(\xi + 1)} |\Psi(t) - T\Psi(t)| \\
\leq \frac{a\xi}{1 - (\tau^3 L_p/\Gamma(\xi + 1))}.
\]

(42)

As a result, UHRS and generalized UHRS are the solutions of (17).

6. Dynamical Behavior of the Model

Here, the dynamical behavior of the system of dengue infection will be investigated. The Laplace transform will be used to construct a scheme for the system (8). The method steps are given as

\[
\begin{aligned}
\mathcal{L}[S_{a1}(t)] &= \frac{S_{a10}}{s} + \frac{1}{s^3} \mathcal{L} \left[ \mu^b \nu_1 - \frac{\beta}{N_1} S_{a1} I_{a1} + \mu^a S_{a1} \right], \\
\mathcal{L}[S_{a2}(t)] &= \frac{S_{a20}}{s} + \frac{1}{s^3} \mathcal{L} \left[ \nu^b R_a - \frac{\beta}{N_1} S_{a2} I_{a2} - \mu^a S_{a2} \right], \\
\mathcal{L}[E_{a1}(t)] &= \frac{E_{a10}}{s} + \frac{1}{s^3} \mathcal{L} \left[ \beta S_{a1} I_{a1} + \beta I_{a1} - \left( \mu^a + \rho^a \right) E_{a1} \right], \\
\mathcal{L}[I_{a1}(t)] &= \frac{I_{a10}}{s} + \frac{1}{s^3} \mathcal{L} \left[ (1 - \psi) R_a - \left( \mu^a + \rho^a \right) I_{a1} \right], \\
\mathcal{L}[I_{a2}(t)] &= \frac{I_{a20}}{s} + \frac{1}{s^3} \mathcal{L} \left[ \psi R_a E_{a1} - \left( \mu^a + \rho^a \right) I_{a2} \right], \\
\mathcal{L}[R_a(t)] &= \frac{R_a0}{s} + \frac{1}{s^3} \mathcal{L} \left[ \beta^b S_{a1} I_{a1} + \nu^b I_{a1} + \left( \mu^a + \rho^a \right) R_a \right], \\
\mathcal{L}[S_{a1}(t)] &= \frac{S_{a10}}{s} + \frac{1}{s^3} \mathcal{L} \left[ \mu^b \nu_1 - \frac{\beta}{N_1} S_{a1} I_{a1} + \mu^a S_{a1} \right], \\
\mathcal{L}[S_{a2}(t)] &= \frac{S_{a20}}{s} + \frac{1}{s^3} \mathcal{L} \left[ \nu^b R_a - \frac{\beta}{N_1} S_{a2} I_{a2} - \mu^a S_{a2} \right], \\
\mathcal{L}[E_{a1}(t)] &= \frac{E_{a10}}{s} + \frac{1}{s^3} \mathcal{L} \left[ \beta S_{a1} I_{a1} + \beta I_{a1} - \left( \mu^a + \rho^a \right) E_{a1} \right], \\
\mathcal{L}[I_{a1}(t)] &= \frac{I_{a10}}{s} + \frac{1}{s^3} \mathcal{L} \left[ (1 - \psi) R_a - \left( \mu^a + \rho^a \right) I_{a1} \right], \\
\mathcal{L}[I_{a2}(t)] &= \frac{I_{a20}}{s} + \frac{1}{s^3} \mathcal{L} \left[ \psi R_a E_{a1} - \left( \mu^a + \rho^a \right) I_{a2} \right], \\
\mathcal{L}[R_a(t)] &= \frac{R_a0}{s} + \frac{1}{s^3} \mathcal{L} \left[ \beta^b S_{a1} I_{a1} + \nu^b I_{a1} + \left( \mu^a + \rho^a \right) R_a \right],
\end{aligned}
\]

(43)
where
\[
\begin{align*}
S_h(t) &= \sum_{j=0}^{\infty} S_{h,j}(t), \\
E_h(t) &= \sum_{j=0}^{\infty} E_{h,j}(t), \\
I_h(t) &= \sum_{j=0}^{\infty} I_{h,j}(t), \\
S(t) &= \sum_{j=0}^{\infty} S_j(t), \\
E(t) &= \sum_{j=0}^{\infty} E_j(t).
\end{align*}
\]
(44)

We use Adomian polynomials to decompose the nonlinear terms as

\[
D_j(t) = \frac{1}{P} \int \frac{dz}{dz} \left[ \sum_{k=0}^{j} z^k S_{h,k}(t) \sum_{l=0}^{j} z^l I_{h,l}(t) \right] z = 0,
\]
(45)

\[
S_h(t) I_h(t) = \sum_{j=0}^{\infty} E_j(t),
\]

(46)

Therefore, we get

\[
D_j(t) = \frac{1}{P} \int \frac{dz}{dz} \left[ \sum_{k=0}^{j} z^k S_{h,k}(t) \sum_{l=0}^{j} z^l I_{h,l}(t) \right] z = 0.
\]

\[
S_h(t) I_h(t) = \sum_{j=0}^{\infty} E_j(t),
\]

(45)

\[
E_j(t) = \frac{1}{P} \int \frac{dz}{dz} \left[ \sum_{k=0}^{j} z^k S_{h,k}(t) \sum_{l=0}^{j} z^l I_{h,l}(t) \right] z = 0.
\]
Thus, we have

\[
\begin{align*}
\mathcal{L}[S_{h11}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ \mu_h^6 N_h - \frac{b_1^6}{N_h} D_0(t) - p^6 S_{h11}(t) - \mu_h^6 S_{h11}(t) \right], \\
\mathcal{L}[S_{h21}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ v^6 R_{h0}(t) - \frac{b_2^6}{N_h} E_0(t) - \mu_h^6 S_{h20}(t) \right], \\
\mathcal{L}[E_{h1}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ \frac{\rho_{h1} b_1^6}{N_h} D_0(t) + \frac{\rho_{h2} b_2^6}{N_h} E_0(t) - \left( \mu_h^6 + \rho_h^6 \right) E_{h0}(t) \right], \\
\mathcal{L}[I_{h1}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ (1 - \psi) \rho_h^6 E_{h0}(t) - \left( \gamma^6 + \mu_h^6 \right) I_{hA0}(t) \right], \\
\mathcal{L}[I_{hA1}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ \psi \rho_h^6 E_{h0}(t) - \left( \gamma^6 + \mu_h^6 \right) I_{hA0}(t) \right], \\
\mathcal{L}[R_{h1}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ p^6 S_{h10}(t) + \tau^6 I_{h0}(t) + \gamma^6 (I_{h0}(t) + I_{hA0}(t)) - \left( \mu_h^6 + \nu^6 \right) R_{h0}(t) \right], \\
\mathcal{L}[S_{v1}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ \mu_v^6 N_v - \frac{b_1^6}{N_h} F_0(t) - \mu_v^6 S_{v0}(t) \right], \\
\mathcal{L}[E_{v1}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ \frac{b_2^6}{N_h} F_0(t) - \left( \mu_v^6 + \rho_v^6 \right) E_{v0}(t) \right], \\
\mathcal{L}[I_{v1}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ \rho_v^6 E_{v0}(t) - \mu_v I_{v0}(t) \right], \\
\mathcal{L}[S_{h12}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ \mu_h^6 N_h - \frac{b_1^6}{N_h} D_1(t) - p^6 S_{h11}(t) - \mu_h^6 S_{h11}(t) \right], \\
\mathcal{L}[S_{h22}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ v^6 R_{h1}(t) - \frac{b_2^6}{N_h} E_1(t) - \mu_h^6 S_{h21}(t) \right], \\
\mathcal{L}[E_{h2}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ \frac{b_1^6}{N_h} D_1(t) + \frac{b_2^6}{N_h} E_1(t) - \left( \mu_h^6 + \rho_h^6 \right) E_{h1}(t) \right], \\
\mathcal{L}[I_{h2}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ (1 - \psi) \rho_h^6 E_{h1}(t) - \left( \gamma^6 + \mu_h^6 + \nu^6 \right) I_{h1}(t) \right], \\
\mathcal{L}[I_{hA2}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ \psi \rho_h^6 E_{h1}(t) - \left( \gamma^6 + \mu_h^6 \right) I_{hA1}(t) \right], \\
\mathcal{L}[R_{h2}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ p^6 S_{h11}(t) + \tau^6 I_{h1}(t) + \gamma^6 (I_{h1}(t) + I_{hA1}(t)) - \left( \mu_h^6 + \nu^6 \right) R_{h1}(t) \right], \\
\mathcal{L}[S_{v2}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ \mu_v^6 N_v - \frac{b_1^6}{N_h} F_1(t) - \mu_v^6 S_{v1}(t) \right], \\
\mathcal{L}[E_{v2}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ \frac{b_2^6}{N_h} F_1(t) - \left( \mu_v^6 + \rho_v^6 \right) E_{v1}(t) \right], \\
\mathcal{L}[I_{v2}(t)] &= \frac{1}{s^8} \mathcal{L} \left[ \rho_v^6 E_{v1}(t) - \mu_v I_{v1}(t) \right].
\end{align*}
\]
Furthermore, we attain

\[ \mathcal{D} \left[ S_{h(j+1)}(t) \right] = \frac{1}{s^\theta} \mathcal{D} \left[ \rho^\theta N_h - \frac{b^\theta \beta_{h1}}{N_h} D_j(t) - \rho^\theta S_{h(j)}(t) - \mu^\theta S_{h1}(t) \right], \]

\[ \mathcal{D} \left[ S_{h2(j+1)}(t) \right] = \frac{1}{s^\theta} \mathcal{D} \left[ \rho^\theta N_h - \frac{b^\theta \beta_{h2}}{N_h} E_j(t) - \rho^\theta S_{h2}(t) - \mu^\theta S_{h1}(t) \right], \]

\[ \mathcal{D} \left[ E_{h(j+1)}(t) \right] = \frac{1}{s^\theta} \mathcal{D} \left[ \beta_{h1} b^\theta N_h D_j(t) + \beta_{h2} b^\theta N_h E_j(t) - \left( \rho^\theta + \mu^\theta \right) E_{h(j)}(t) \right], \]

\[ \mathcal{D} \left[ I_{h(j+1)}(t) \right] = \frac{1}{s^\theta} \mathcal{D} \left[ \left( 1 - \psi_1 \right) \rho^\theta R_{h(j)}(t) - \left( \gamma^\theta + \tau^\theta + \mu^\theta \right) I_{h(j)}(t) \right], \]

\[ \mathcal{D} \left[ I_{h1(j+1)}(t) \right] = \frac{1}{s^\theta} \mathcal{D} \left[ \psi \rho^\theta R_{h(j)}(t) - \left( \gamma^\theta + \mu^\theta \right) I_{h1(j)}(t) \right], \]

\[ \mathcal{D} \left[ I_{h2(j+1)}(t) \right] = \frac{1}{s^\theta} \mathcal{D} \left[ \psi \rho^\theta R_{h(j)}(t) - \left( \gamma^\theta + \mu^\theta \right) I_{h2(j)}(t) \right], \]

\[ \mathcal{D} \left[ I_{h3(j+1)}(t) \right] = \frac{1}{s^\theta} \mathcal{D} \left[ \rho^\theta N_v - \frac{b^\theta \beta_{v}}{N_h} F_j(t) - \mu^\theta S_{v}(t) \right], \]

\[ \mathcal{D} \left[ E_{v(j+1)}(t) \right] = \frac{1}{s^\theta} \mathcal{D} \left[ \rho^\theta N_v - \frac{b^\theta \beta_{v}}{N_h} E_j(t) - \left( \rho^\theta + \mu^\theta \right) E_{v(j)}(t) \right], \]

\[ \mathcal{D} \left[ I_{v(j+1)}(t) \right] = \frac{1}{s^\theta} \mathcal{D} \left[ \rho^\theta E_{v(j)}(t) - \mu^\theta I_{v(j)}(t) \right]. \]

Initial conditions are stated as

\[
\begin{align*}
S_{h10}(t) &= S_{h10}S_{h20}(t) = S_{h20}, \\
E_{h0}(t) &= E_{h0}I_{h0}(t) = I_{h0}, \\
I_{hA0}(t) &= I_{hA0}R_{h0}(t) = R_{h0}, \\
S_{v0}(t) &= S_{v0}E_{v0}(t) = E_{v0}, \\
I_{v0}(t) &= I_{v0}.
\end{align*}
\]
To further simplify it, we proceed as follows:

\[
\begin{align*}
S_{h1}(t) &= \mathcal{L}^{-1} \left[ \frac{1}{\gamma} \mathcal{L} \left[ \mu_h \sigma_n - \frac{b_h \beta_n}{N_h} D_0(t) - p_s S_{h10}(t) - \mu_h S_{h1}(t) \right] \right], \\
S_{h2}(t) &= \mathcal{L}^{-1} \left[ \frac{1}{\gamma} \mathcal{L} \left[ \gamma S_{h10}(t) - \frac{b_h \beta_n}{N_h} E_0(t) - \mu_h S_{h20}(t) \right] \right], \\
E_{h1}(t) &= \mathcal{L}^{-1} \left[ \frac{1}{\gamma} \mathcal{L} \left[ \frac{b_h \beta_n}{N_h} A_0(t) + \frac{b_h \beta_n}{N_h} E_0(t) - \left( \rho_h + \mu_h \right) E_{h1}(t) \right] \right], \\
I_{h1}(t) &= \mathcal{L}^{-1} \left[ \frac{1}{\gamma} \mathcal{L} \left[ (1 - \psi) \rho_h E_{h1}(t) - \left( \gamma + \mu_h + \varepsilon \right) I_{h0}(t) \right] \right], \\
I_{hA1}(t) &= \mathcal{L}^{-1} \left[ \frac{1}{\gamma} \mathcal{L} \left[ \psi \rho_h E_{h1}(t) - \left( \gamma + \mu_h \right) I_{hA0}(t) \right] \right], \\
R_{h1}(t) &= \mathcal{L}^{-1} \left[ \frac{1}{\gamma} \mathcal{L} \left[ p_s S_{h10}(t) + \varepsilon I_{h0}(t) + \gamma (I_{h0}(t) + I_{hA0}(t)) - \left( \mu_h + \varepsilon \right) R_{h0}(t) \right] \right], \\
S_{v1}(t) &= \mathcal{L}^{-1} \left[ \frac{1}{\gamma} \mathcal{L} \left[ \mu_v - \frac{b_h \beta_n}{N_h} F_0(t) - \mu_v S_{v0}(t) \right] \right], \\
E_{v1}(t) &= \mathcal{L}^{-1} \left[ \frac{1}{\gamma} \mathcal{L} \left[ \frac{b_h \beta_n}{N_h} F_0(t) - \left( \rho_v + \rho_v \right) E_{v0}(t) \right] \right], \\
I_{v1}(t) &= \mathcal{L}^{-1} \left[ \frac{1}{\gamma} \mathcal{L} \left[ \rho_v E_{v0}(t) - \mu_v I_{v0}(t) \right] \right].
\end{align*}
\]
Furthermore, we get that

\[
\begin{align*}
S_{h_1(t)}(t) &= \mathcal{L}^{-1} \left[ \frac{1}{s^3} \mathcal{L} \left[ \mu_h N_h - \frac{b_h}{N_h} D_j(t) - \rho_h S_{h_1}(t) - \mu_h S_{h_1}(t) \right] \right], \\
S_{h_2(t)}(t) &= \mathcal{L}^{-1} \left[ \frac{1}{s^3} \mathcal{L} \left[ \frac{b_h}{N_h} R_{h_1}(t) - \frac{b_h}{N_h} E_j(t) - \mu_h S_{h_2}(t) \right] \right], \\
E_{h_1(t)}(t) &= \mathcal{L}^{-1} \left[ \frac{1}{s^3} \mathcal{L} \left[ \frac{b_h}{N_h} A_j(t) + \frac{b_h}{N_h} B_j(t) - \left( \rho_h + \mu_h \right) E_{h_1}(t) \right] \right], \\
I_{h_1(t)}(t) &= \mathcal{L}^{-1} \left[ \frac{1}{s^3} \mathcal{L} \left[ \left( 1 - \nu \right) \rho_h E_{h_1}(t) - \left( \nu + \mu_h \right) I_{h_1}(t) \right] \right], \\
I_{h_2(t)}(t) &= \mathcal{L}^{-1} \left[ \frac{1}{s^3} \mathcal{L} \left[ \left( 1 - \nu \right) \rho_h E_{h_1}(t) + \gamma \left( I_{h_1}(t) + I_{h_2}(t) \right) - \left( \mu_h + \nu \right) I_{h_2}(t) \right] \right], \\
S_v(t) &= S_0(t) + S_{h_1}(t) + S_{h_2}(t) + S_{h_3}(t) + \ldots, \\
E_v(t) &= E_0(t) + E_{h_1}(t) + E_{h_2}(t) + E_{h_3}(t) + \ldots, \\
I_v(t) &= I_0(t) + I_{h_1}(t) + I_{h_2}(t) + I_{h_3}(t) + \ldots, \\
S_{h_1}(t) &= S_{h_0}(t) + S_{h_1}(t) + S_{h_2}(t) + S_{h_3}(t) + \ldots, \\
S_{h_2}(t) &= S_{h_0}(t) + S_{h_1}(t) + S_{h_2}(t) + S_{h_3}(t) + \ldots, \\
E_h(t) &= E_{h_0}(t) + E_{h_1}(t) + E_{h_2}(t) + E_{h_3}(t) + \ldots, \\
I_h(t) &= I_{h_0}(t) + I_{h_1}(t) + I_{h_2}(t) + I_{h_3}(t) + \ldots, \\
R_h(t) &= R_{h_0}(t) + R_{h_1}(t) + R_{h_2}(t) + R_{h_3}(t) + \ldots.
\end{align*}
\]  

Therefore, we have the following answer in series form:

\[
\begin{align*}
S_v(t) &= S_0(t) + S_{h_1}(t) + S_{h_2}(t) + S_{h_3}(t) + \ldots, \\
E_v(t) &= E_0(t) + E_{h_1}(t) + E_{h_2}(t) + E_{h_3}(t) + \ldots, \\
I_v(t) &= I_0(t) + I_{h_1}(t) + I_{h_2}(t) + I_{h_3}(t) + \ldots, \\
S_{h_1}(t) &= S_{h_0}(t) + S_{h_1}(t) + S_{h_2}(t) + S_{h_3}(t) + \ldots, \\
S_{h_2}(t) &= S_{h_0}(t) + S_{h_1}(t) + S_{h_2}(t) + S_{h_3}(t) + \ldots, \\
E_h(t) &= E_{h_0}(t) + E_{h_1}(t) + E_{h_2}(t) + E_{h_3}(t) + \ldots, \\
I_h(t) &= I_{h_0}(t) + I_{h_1}(t) + I_{h_2}(t) + I_{h_3}(t) + \ldots, \\
R_h(t) &= R_{h_0}(t) + R_{h_1}(t) + R_{h_2}(t) + R_{h_3}(t) + \ldots.
\end{align*}
\]  

The upper mentioned numerical technique is utilized to investigate the dynamics (8) of dengue infection. For numerical reasons, the parameter values in Table 1 are utilized. Here, we will perform distinct numerical scenario to illustrate the impact of input factors on the system of dengue. As a result of our findings, we will recommend effective control strategies that will minimize the frequency of dengue fever in population. Using simulations, we showed the time series of infected, exposed, and asymptomatic carriers in the host population, while we emphasized the time series of exposed and diseased people in the vector population.

The influence of vaccination on dengue transmission patterns has been depicted in the first simulation shown in Figure 1. The human and mosquito time series are indicated with varied vaccination \( p \) values. It has been discovered that vaccination can reduce the degree of infection; hence, it is advised that vaccine efficacy be improved in order to eliminate dengue infection. In Figure 2, we highlighted the influence of treatment \( \tau \) on the system of dengue in the second simulation. We proposed that therapy can be utilized as a control parameter to minimize infection levels in society. The role of mosquito bite rate is depicted in Figure 3 which illustrated that the biting rate is vital and can transmit the illness; therefore, controlling this aspect is crucial to avoid infection transmission.

The effect of the asymptotic carrier on the system is seen in the fourth scenario in Figure 4. It was demonstrated that this input component is crucial and can be a source of infection in dengue-endemic locations. In Figure 5, the role of the losing rate of immunity \( \nu \) has been visualised. This parameter is equally harmful, as shown by the results, and can enable dengue process more complicated. In Figure 6, we
Figure 1: Graphical view analysis of system (8) with distinct values of $p$, i.e., $p = 0.535, 0.435, 0.335$, and $0.235$, for dengue dynamics.
Figure 2: Graphical view analysis of system (8) distinct values of $\tau$, i.e., $\tau = 0.040, 0.035, 0.30,$ and $0.025,$ for dengue dynamics.
Figure 3: Plotting the time series of the system (8) with distinct values of $b$, i.e., $b = 0.55, 0.50, 0.45$, and $0.40$, for dengue dynamics.
Figure 4: Representation of the system (8) with distinct values of the input factor $\psi$, i.e., $\psi = 0.7, 0.6, 0.5,$ and $0.4$, for dengue fever.
Figure 5: Plotting the time series of the system (8) with distinct values of $\nu$, i.e., $\nu = 0.005, 0.010, 0.015,$ and $0.020$, for dengue fever.
Figure 6: Plotting of the system (8) with distinct values of memory, i.e., $\vartheta = 1.0, 0.9, 0.8,$ and $0.7$, for dengue fever.
graphically represented the influence of memory on the time series of dengue infection. The plot of the infected individuals was illustrated with various values of memory $\vartheta$. This parameter seems to be effective and recommended to the policymakers for better control of the infection.

Memory plays a crucial part in vector-borne disease transmission dynamics because vector-borne sickness has knowledge of previous stages and an associative learning experience [15, 16]. In dengue transmission, mosquitoes use their prior experience about the human's location, blood selection, colour, and the smell of humans sweat to reduce the contact rate between vector and hosts. Fractional-order models give information about the past and present states of biological systems for the future. Moreover, fractional-order models possess hereditary properties and represent the nonlocal behavior of biological systems. Therefore, the results of fractional models are more accurate than that of integer models, and the index of memory may be utilized as a control measure. Fractional-order systems may readily reflect these kinds of phenomena in mathematical models of infectious diseases. As a result, it is crucial to consider memory’s role in the spread of dengue disease. We primarily focused on such elements in our study to demonstrate their influence on infection dynamics. Our findings showed that the memory index can limit infection levels, which is something policymakers should consider. We proved that vaccination and diagnosis can help to stabilize dengue fever and the index of memory may be utilized as a control measure.

7. Conclusion

Dengue fever is a life-threatening and severe sickness that affects people all over the globe. Introducing effective techniques for the management of this viral infection is now a problem for policymakers, scientists, and public health professionals. Through a fractional framework, we developed a new for dengue fever that includes asymptotic carriers, immunisation, reinfection, and therapy. With the use of the fixed-point theorem, the existence and uniqueness of the solution of the hypothesized system are explored in the context of Banach’s and Schaefer’s. We created the necessary parameters for the Ulam-Hyers stability in our dengue system. The effect of various variables on the dynamics of dengue is evaluated using the LADM approach to describe the effect of various input factors on the time series of dengue. We found that biting rates, asymptomatic carriers, and immunity loss rates are significant characteristics that make control more challenging, but dengue infection can be eliminated by vaccine, memory index, and therapy. We demonstrated the impact of memory on the time series of dengue and proposed that it may be employed as a control parameter for infection management. We shall demonstrate the influence of incubation and maturation delay on dengue transmission in future studies.

Data Availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare that there is no conflict of interest regarding this work.

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