



OPEN

Analysis of a fractional-order model for dengue transmission dynamics with quarantine and vaccination measures

Muhammad Usman¹, Mujahid Abbas^{2,3}, Safeer Hussain Khan⁴✉ & Andrew Oname^{1,5}

A comprehensive mathematical model is proposed to study two strains of dengue virus with saturated incidence rates and quarantine measures. Imperfect dengue vaccination is also assumed in the model. Existence, uniqueness and stability of the proposed model are proved using the results from fixed point and degree theory. Additionally, well constructed Lyapunov function candidates are also applied to prove the global stability of infection-free equilibria. It is also demonstrated that the model is generalized Ulam-Hyers stable under some appropriate conditions. The model is fitted to the real data of dengue epidemic taken from the city of Espirito Santo in Brazil. For the approximate solution of the model, a non-standard finite difference(NSFD) approach is applied. Sensitivity analysis is also carried out to show the influence of different parameters involved in the model. The behaviour of the NSFD is also assessed under different denominator functions and it is observed that the choice of the denominator function could influence the solution trajectories. Different scenario analysis are also assessed when the reproduction number is below or above one. Furthermore, simulations are also presented to assess the epidemiological impact of dengue vaccination and quarantine measures for infected individuals.

Keywords Dengue, Strains, Mathematical model, Existence and uniqueness, Stability, Reproduction number, Nonstandard finite difference scheme

The virus that causes dengue fever is spread mostly by female *Aedes* mosquitoes, predominantly *Aedes aegypti* mosquitoes, and secondarily *Aedes albopictus* mosquitoes. Through mosquito bites, the dengue virus can infect humans and cause mild or severe sickness or death in some cases¹. The areas with a high *Aedes* mosquito population are most likely to experience a dengue outbreak. The disease is primarily spread when an adult female *Aedes* mosquito bites a person carrying the virus, catching it and then passing it on to an uninfected person. However, there are other less common ways to spread the virus including through breastfeeding and pregnancy and in extremely rare instances, through organ transplantation². Four Dengue serotypes (Dengue1-4) have spread quickly inside nations and across continents, resulting in epidemics and severe dengue fever, hyperendemicity of numerous Dengue serotypes in tropical nations, and autochthonous transmission in Europe and the USA^{3,4}. In the past, there was no specific treatments for dengue. The only possibility to control the disease was to control the vectors, which was very difficult. In 2015, a new vaccine for Dengue virus (*Dengvaxia* by Sanofi Pasteur) has been released⁵. *Dengvaxia* is a tetravalent vaccine whose efficacy varies by serotypes (54.7% for serotype 1, 43.0% for serotype 2, 71.6% for serotype 3 and 76.9% for serotype 4.)⁶.

Mathematical models using the classical integer-order derivative have been developed in studying the dynamics of infectious diseases^{7–18}. In particular, Ferguson et al.¹⁴ employed a PDE model that takes prior infection history into account. Using an agent-based dengue model. Hladish et al.¹⁵ investigated the effects of several vaccine scenarios on dengue transmission dynamics in Yucatan, Mexico. For the purpose of examining the effects of immunisation against the four dengue serotypes, Coudeville and Garnett¹⁶ considered an age-structured

¹Abdus Salam School of Mathematical Sciences, Government College University Katchery Road, Lahore 54000, Pakistan. ²Department of Mathematics, Government College University Katchery Road, Lahore 54000, Pakistan. ³Department of Medical Research, China Medical University Hospital, China Medical University, Taichung 40402, Taiwan. ⁴Department of Mathematics and Statistics, North Carolina A & T State University, Greensboro, NC 27411, USA. ⁵Department of Mathematics, Federal University of Technology, Owerri, Nigeria. ✉email: shkhan@ncat.edu

compartmental model. A similar (age-stratified) model was employed by Rodriguez-Barraquer et al.¹⁷ to assess the effectiveness of a vaccination that is only partially effective against three of the four dengue serotypes. Using compartmental and agent-based modelling techniques, Chao et al.¹⁸ demonstrated that a dengue vaccine with efficacy ranging from 70% to 90% against all four dengue serotypes has the potential to reduce the frequency and magnitude of dengue epidemics significantly in the short and medium terms.

These models, due to the integer nature of the derivative constitute certain limitations. Different fractional operators relying on power-law¹⁹, exponential²⁰, generalized Mittag-Leffler²¹ and other forms of kernels have emerged and their applications to modelling biological processes have gained much attraction in recent times. The fractional order operators have some advantages over classical order operator such as memory effect and better performance. Mathematical models using the fractional order operators have been successfully applied in investigating the dynamics of the infectious diseases^{22–27}. In particular, Fatmawati²⁷ studied the dengue dynamics with fractal-fractional Caputo-Fabrizio operator and employed real statistical data of dengue infection cases of East Java, Indonesia, from 2018 and parameterized the dengue model.

The Caputo fractional operator with a singular kernel offers advantages in modeling disease transmissions by providing a more flexible framework that can capture memory effects, non-local behavior, and complex dynamics. Memory effects means that it accounts for the history of the system. In disease transmission models, this can be particularly useful for capturing the impact of past infections, immunity, or interventions on the current state of the population. Unlike classical derivatives, fractional derivatives are non-local operators showing that the behavior of the system does not depend on the neighbourhood of a particular point but depends on its history over a range of time, which can be crucial for modeling the spread of infectious diseases where past interactions can influence future outcomes. The non standard finite difference (NSFD) scheme has a couple of favourable properties. It is explicit and due to its construction it reproduces important properties of the solution, like the number and location of fixed-points, the positivity, accuracy, stability and certain conservation laws. It offer valuable tools for numerical simulation and analysis across a wide range of scientific and engineering disciplines, where accurate and efficient approximation of model solutions is essential for understanding and predicting complex phenomena.

In this study, based on Caputo fractional operator, a comprehensive model for two strains of dengue is proposed, and validated using data from Brazilian state of Espirito Santo. Given that both vaccines have varying levels of efficacy, the suggested model also assumes separate immunization for strains-1 and strain-2. We have also included two co-infection compartments, which have not been considered in the existing models, for possible disease states, including exposed, asymptomatic, and symptomatic infections. We established the conditions for existence, uniqueness and stability of the model. In addition, we applied the nonstandard finite difference (NSFD) scheme to obtain approximate solution of the model. Then impact of different denominator functions on the approximated solution is also presented. To the best of our knowledge, the proposed model studied in this paper is novel and appropriate to study the co-circulation of two dengue strains using fractional calculus tools.

The paper is organized as follows: The model is formulated in “[Model formulation](#)”. The rigorous analysis of the sub-model and the full model is given in “[Analysis of the sub-models](#)” and “[Analysis of the complete model](#)”. Existence and uniqueness are proved in the “[Existence, uniqueness and Ulam–Hyers stability of the complete model](#)”. The Ulam–Hyers stability is presented in “[Ulam–Hyers stability](#)”. The model solution is approximated with the help of non standard finite difference scheme in “[Nonstandard finite difference scheme](#)”. The model fitting and the numerical assessments are given in “[Model fitting and numerical assessment](#)”. Finally “[Conclusion](#)” contains the concluding remark and some future directions.

Preliminaries

Definition 1.1 ¹⁹ The Caputo fractional derivative of a function f of order $\sigma \in (0, 1)$ is defined by

$${}^C D_t^\sigma f(t) = \frac{1}{\Gamma(n-\sigma)} \int_0^t (t-\wp)^{n-\sigma-1} f^{(n)}(\wp) d\wp, \quad (1)$$

where, $n = [\sigma] + 1$ and Γ stands for the Gamma function.

Definition 1.2 ¹⁹ The Riemann–Liouville fractional integral of a function f of order $\sigma \in (0, 1)$ is defined by

$${}^C I_t^\sigma f(t) = \frac{1}{\Gamma(\sigma)} \int_0^t (t-\wp)^{\sigma-1} f(\wp) d\wp, \quad t > 0, \quad (2)$$

Lemma 1.1 ¹⁹ The Laplace transform of Caputo fractional derivative is given by

$$\mathcal{L}\{{}^C D_t^\sigma f(t)\} = s^\sigma \mathcal{L}\{f(t)\} - s^{\sigma-1} f(0), \quad 0 < \sigma < 1, \quad (3)$$

where \mathcal{L} is the Laplace transform operator.

We now recall the following definitions from²⁸.

Definition 1.3 The Kuratowski measure of non-compactness $\mu : \mathbb{B} \rightarrow [0, \infty)$ is defined as:

$$\mu(\Omega) = \inf\{d > 0 : \Omega \in \mathbb{B} \text{ admits a finite cover by sets of diameter } \leq d\},$$

where \mathbb{B} denotes the family of all bounded subsets of E .

Recall that the Kuratowski measure μ has the property that $\mu(\Omega) = 0$ iff Ω is relatively compact.

Definition 1.4 A continuous and bounded function $T : S \rightarrow E$ is said to be μ -Lipschitz if there exist $k \geq 0$ such that

$$\mu(T(\Omega_0)) \leq k\mu(\Omega_0)$$

for all bounded subsets of $S \subseteq E$.

Definition 1.5 The mapping T is said to be μ -condensing if

$$\mu(T(\Omega_0)) < \mu(\Omega_0)$$

for all bounded subset of S .

Lemma 1.2 If A and B are μ -Lipschitz map with constant k and k' respectively then $A+B$ is also μ -Lipschitz with constant $k + k'$.

Lemma 1.3 If T is a compact map, then T is μ -Lipschitz with constant 0.

Lemma 1.4 If T is Lipschitz map with constant k , then T is μ -Lipschitz map with the same constant.

Theorem 1.1 Let $T : E \rightarrow E$ be μ -condensing and

$$\Theta = \{\zeta \in E : \exists \lambda \in [0, 1] \text{ such that } \zeta = \lambda T\zeta\}.$$

If Θ is a bounded set in E , that is there exists $r > 0$ such that $\Theta \subset B_r(0)$, then the degree

$$\mathbb{D}(I - \lambda T, B_r(0), 0) = 1, \quad \forall \lambda \in [0, 1].$$

Consequently, T has at least one fixed point and the set of the fixed points of T lies in $B_r(0)$.

Model formulation

To formulate the model, the human population at a given time t is denoted by $N(t)$ and is subdivided into following classes: vulnerable or uninfected persons $S_h(t)$, individuals vaccinated against Dengue $V_h(t)$, individuals exposed to Dengue strain 1 and strain 2 $E_{h1}(t)$, $E_{h2}(t)$, respectively, individuals exposed for co-infection $E_{h12}(t)$, Individuals infected with strain 1, strain 2 and both disease (Asymptomatic stage) $A_{h1}(t)$, $A_{h2}(t)$, $A_{h12}(t)$, respectively, Individuals infected with strain 1, strain 2 and both disease (Symptomatic stage) $I_{h1}(t)$, $I_{h2}(t)$, $I_{h12}(t)$, respectively, quarantine individuals suffering with strain 1, strain 2 and both disease $Q_{h1}(t)$, $Q_{h2}(t)$, $Q_{h12}(t)$, respectively, recovered individuals from strain 1, strain 2 and co-infection are $R_{h1}(t)$, $R_{h2}(t)$, $R_{h12}(t)$, respectively. The vector population is divided into: Susceptible vectors $S_v(t)$, Vectors exposed to strain 1 and strain 2 $E_{v1}(t)$, $E_{v2}(t)$, respectively, vectors infected with strain 1 and strain 2 $I_{v1}(t)$, $I_{v2}(t)$, respectively.

Based on the established knowledge about the epidemiology of dengue serotypes^{29–31}, the proposed model has the following assumptions:

- Susceptible individuals can get infections with dengue strain- i from infected vectors at the rate of $\frac{\beta_{hi}^\sigma I_{vi}}{1 + \alpha_i^\sigma I_{vi}}$ for $i = 1, 2$.
- The saturated form of incidence $\frac{\beta_{hi}^\sigma I_{vi}}{1 + \alpha_i^\sigma I_{vi}}$ for $i = 1, 2$ is adopted in this model. Basically we are adding the parameters α_i^σ to add some control in the transmission due to the crowding effect and inhabitation effect and behavioral change by the susceptible individuals. This has been used in some epidemiological models^{32–34}.
- Susceptible individuals are vaccinated at the rate ψ^σ . The dengue vaccine is assumed to have efficacy of ϕ_i^σ against strain i .
- Individuals infected with either strain 1 or strain 2 can get infected with the other strain.
- Natural death rate is assumed to be μ_h^σ for all human compartments.
- Symptomatic individuals are quarantined at the rate η_i^σ .
- Removal of vectors from the population is assumed at the rate μ_v^σ .
- The recovered individuals can lose their immunity and return to the susceptible state at the rate δ_{hi}^σ .

The model's parameters are described in Table 1 whereas the system's equations are presented in (4).

Parameter	Description	Value	References
β_{h1}^σ	Transmission rate for dengue Strain 1	$4.3892e-13 \text{ day}^{-1}$	Fitted
β_{h2}^σ	Transmission rate for dengue Strain 2	$5.1160e-07 \times 10^{-6} \text{ day}^{-1}$	Fitted
θ_{a1}^σ	Mortality rates for Asymptomatic Individuals of strain 1	0.0406 day^{-1}	Fitted
θ_{a2}^σ	Mortality rates for Asymptomatic Individuals of strain 2	$4.2968e-07 \text{ day}^{-1}$	Fitted
ψ^σ	Vaccination rate	$7.0002e-05 \text{ day}^{-1}$	Fitted
ζ_{a1}^σ	Recovery rates from Asymptomatic individuals of strain 1	0.0544 day^{-1}	Fitted
ζ_{a2}^σ	Recovery rates from Asymptomatic individuals of strain 2	$3.9726e-07 \text{ day}^{-1}$	Fitted
$\alpha_1^\sigma, \alpha_2^\sigma$	Saturated incidence rates	0.005 day^{-1}	Assumed
Λ_h^σ	Human recruitment rate	$\frac{4,000,000}{78 \times 365} \text{ day}^{-1}$	35
μ_h^σ	Natural death rates	$\frac{1}{78 \times 365} \text{ day}^{-1}$	35
δ_{h1}^σ	Immunity loss from individuals recovered from strain 1	0.026 day^{-1}	36
δ_{h2}^σ	Immunity loss from individuals recovered from strain 2	0.026 day^{-1}	36
δ_{h12}^σ	Immunity loss from individuals recovered from co-infection	0.026 day^{-1}	36
ϕ_1^σ	Vaccine efficacy against Strain 1	$[0.81, 0.88] \text{ day}^{-1}$	37
ϕ_2^σ	Vaccine efficacy against Strain 2	$[0.81, 0.88] \text{ day}^{-1}$	37
$\xi_{e1}^\sigma, \xi_{e2}^\sigma, \xi_{e12}^\sigma$	Progression rates from exposed to asymptomatic	0.1 day^{-1}	38
$\xi_{a1}^\sigma, \xi_{a2}^\sigma, \xi_{a12}^\sigma$	Progression rates from asymptomatic to symptomatic	0.1 day^{-1}	38
ζ_{a12}^σ	Recovery rates from Asymptomatic individuals of co-infection	$[0.11, 0.15] \text{ day}^{-1}$	37
$\zeta_{i1}^\sigma, \zeta_{i2}^\sigma, \zeta_{i12}^\sigma$	Recovery rates from Symptomatic individuals	$[0.11, 0.15] \text{ day}^{-1}$	37
$\zeta_{h1}^\sigma, \zeta_{h2}^\sigma, \zeta_{h12}^\sigma$	Recovery rates from Quarantine individuals	$[0.11, 0.15] \text{ day}^{-1}$	37
θ_{a12}^σ	Mortality rates for Asymptomatic Individuals of co-infection	0.001 day^{-1}	37
$\theta_{i1}^\sigma, \theta_{i2}^\sigma, \theta_{i12}^\sigma$	Mortality rates for Symptomatic individuals	0.001 day^{-1}	37
$\theta_{h1}^\sigma, \theta_{h2}^\sigma, \theta_{h12}^\sigma$	Mortality rates for Quarantine individuals	0.001 day^{-1}	37
$\eta_{i1}^\sigma, \eta_{i2}^\sigma, \eta_{i12}^\sigma$	Rates for which people are quarantine	0.05 day^{-1}	Assumed
Λ_v^σ	Mosquitos recruitment rate	$20,000 \text{ day}^{-1}$	Assumed
β_{v1}^σ	Transmission rate from human to vectors for dengue strain-1	$[0.60, 0.75] \text{ day}^{-1}$	37
β_{v2}^σ	Transmission rate from human to vectors for dengue strain-2	$[0.60, 0.75] \text{ day}^{-1}$	37
μ_v^σ	Rate of removal of vectors	$(\frac{1}{21} - \frac{1}{7}) \text{ day}^{-1}$	37
$\gamma_1^\sigma, \gamma_2^\sigma, \gamma_3^\sigma, \gamma_4^\sigma$	Saturated incidence rates for vectors	0.0005	Assumed
ω_{v1}^σ	Progression rates for vectors from Expose to infected with strain 1	0.1 day^{-1}	38
ω_{v2}^σ	Progression rates for vectors from Expose to infected with strain 2	0.1 day^{-1}	38
ρ_{v1}^σ	Mortality of infected vectors with strain 1	$\text{negligible day}^{-1}$	38
ρ_{v2}^σ	Mortality of infected vectors with strain 2	$\text{negligible day}^{-1}$	38

Table 1. Model (4) parameters’ description.

$$\begin{aligned}
{}^C\mathcal{D}_{0+}^\sigma S_h(t) &= \Lambda_h^\sigma - \frac{\beta_{h1}^\sigma I_{v1}}{1 + \alpha_1^\sigma I_{v1}} S_h - \frac{\beta_{h2}^\sigma I_{v2}}{1 + \alpha_2^\sigma I_{v2}} S_h - (\mu_h^\sigma + \psi^\sigma) S_h + \delta_{h1}^\sigma R_{h1} + \delta_{h2}^\sigma R_{h2} + \delta_{h12}^\sigma R_{h12}, \\
{}^C\mathcal{D}_{0+}^\sigma V_h(t) &= \psi^\sigma S_h - (1 - \phi_1^\sigma) \frac{\beta_{h1}^\sigma I_{v1}}{1 + \alpha_1^\sigma I_{v1}} V_h - (1 - \phi_2^\sigma) \frac{\beta_{h2}^\sigma I_{v2}}{1 + \alpha_2^\sigma I_{v2}} V_h - \mu_h^\sigma V_h, \\
{}^C\mathcal{D}_{0+}^\sigma E_{h1}(t) &= \frac{\beta_{h1}^\sigma I_{v1}}{1 + \alpha_1^\sigma I_{v1}} [S_h + (1 - \phi_1^\sigma) V_h] - (\mu_h^\sigma + \xi_{e1}^\sigma) E_{h1} - \frac{\beta_{h2}^\sigma I_{v2}}{1 + \alpha_2^\sigma I_{v2}} E_{h1}, \\
{}^C\mathcal{D}_{0+}^\sigma E_{h2}(t) &= \frac{\beta_{h2}^\sigma I_{v2}}{1 + \alpha_2^\sigma I_{v2}} [S_h + (1 - \phi_2^\sigma) V_h] - (\mu_h^\sigma + \xi_{e2}^\sigma) E_{h2} - \frac{\beta_{h1}^\sigma I_{v1}}{1 + \alpha_1^\sigma I_{v1}} E_{h2}, \\
{}^C\mathcal{D}_{0+}^\sigma E_{h12}(t) &= \frac{\beta_{h2}^\sigma I_{v2}}{1 + \alpha_2^\sigma I_{v2}} E_{h1} + \frac{\beta_{h1}^\sigma I_{v1}}{1 + \alpha_1^\sigma I_{v1}} E_{h2} - (\mu_h^\sigma + \xi_{e12}^\sigma) E_{h12}, \\
{}^C\mathcal{D}_{0+}^\sigma A_{h1}(t) &= \xi_{e1}^\sigma E_{h1} - (\mu_h^\sigma + \xi_{a1}^\sigma + \zeta_{a1}^\sigma + \theta_{a1}^\sigma) A_{h1} - \frac{\beta_{h2}^\sigma I_{v2}}{1 + \alpha_2^\sigma I_{v2}} A_{h1}, \\
{}^C\mathcal{D}_{0+}^\sigma A_{h2}(t) &= \xi_{e2}^\sigma E_{h2} - (\mu_h^\sigma + \xi_{a2}^\sigma + \zeta_{a2}^\sigma + \theta_{a2}^\sigma) A_{h2} - \frac{\beta_{h1}^\sigma I_{v1}}{1 + \alpha_1^\sigma I_{v1}} A_{h2}, \\
{}^C\mathcal{D}_{0+}^\sigma A_{h12}(t) &= \xi_{e12}^\sigma E_{h12} - (\mu_h^\sigma + \xi_{a12}^\sigma + \zeta_{a12}^\sigma + \theta_{a12}^\sigma) A_{h12} + \frac{\beta_{h2}^\sigma I_{v2}}{1 + \alpha_2^\sigma I_{v2}} A_{h1} + \frac{\beta_{h1}^\sigma I_{v1}}{1 + \alpha_1^\sigma I_{v1}} A_{h2}, \\
{}^C\mathcal{D}_{0+}^\sigma I_{h1}(t) &= \xi_{a1}^\sigma A_{h1} - (\mu_h^\sigma + \zeta_{i1}^\sigma + \theta_{i1}^\sigma + \eta_{i1}^\sigma) I_{h1} - \frac{\beta_{h2}^\sigma I_{v2}}{1 + \alpha_2^\sigma I_{v2}} I_{h1}, \\
{}^C\mathcal{D}_{0+}^\sigma I_{h2}(t) &= \xi_{a2}^\sigma A_{h2} - (\mu_h^\sigma + \zeta_{i2}^\sigma + \theta_{i2}^\sigma + \eta_{i2}^\sigma) I_{h2} - \frac{\beta_{h1}^\sigma I_{v1}}{1 + \alpha_1^\sigma I_{v1}} I_{h2}, \\
{}^C\mathcal{D}_{0+}^\sigma I_{h12}(t) &= \xi_{a12}^\sigma A_{h12} - (\mu_h^\sigma + \zeta_{i12}^\sigma + \theta_{i12}^\sigma + \eta_{i12}^\sigma) I_{h12} + \frac{\beta_{h2}^\sigma I_{v2}}{1 + \alpha_2^\sigma I_{v2}} I_{h1} + \frac{\beta_{h1}^\sigma I_{v1}}{1 + \alpha_1^\sigma I_{v1}} I_{h2}, \\
{}^C\mathcal{D}_{0+}^\sigma Q_{h1}(t) &= \eta_{i1}^\sigma I_{h1} - (\mu_h^\sigma + \zeta_{h1}^\sigma + \theta_{h1}^\sigma) Q_{h1}, \\
{}^C\mathcal{D}_{0+}^\sigma Q_{h2}(t) &= \eta_{i2}^\sigma I_{h2} - (\mu_h^\sigma + \zeta_{h2}^\sigma + \theta_{h2}^\sigma) Q_{h2}, \\
{}^C\mathcal{D}_{0+}^\sigma Q_{h12}(t) &= \eta_{i12}^\sigma I_{h12} - (\mu_h^\sigma + \zeta_{h12}^\sigma + \theta_{h12}^\sigma) Q_{h12}, \\
{}^C\mathcal{D}_{0+}^\sigma R_{h1}(t) &= \zeta_{a1}^\sigma A_{h1} + \zeta_{i1}^\sigma I_{h1} + \zeta_{h1}^\sigma Q_{h1} - (\mu_h^\sigma + \delta_{h1}^\sigma) R_{h1}, \\
{}^C\mathcal{D}_{0+}^\sigma R_{h2}(t) &= \zeta_{a2}^\sigma A_{h2} + \zeta_{i2}^\sigma I_{h2} + \zeta_{h2}^\sigma Q_{h2} - (\mu_h^\sigma + \delta_{h2}^\sigma) R_{h2}, \\
{}^C\mathcal{D}_{0+}^\sigma R_{h12}(t) &= \zeta_{a12}^\sigma A_{h12} + \zeta_{i12}^\sigma I_{h12} + \zeta_{h12}^\sigma Q_{h12} - (\mu_h^\sigma + \delta_{h12}^\sigma) R_{h12}, \\
{}^C\mathcal{D}_{0+}^\sigma S_v(t) &= \Lambda_v^\sigma - \frac{\beta_{v1}^\sigma (A_{h1} + I_{h1} + A_{h12} + I_{h12})}{1 + \gamma_1^\sigma A_{h1} + \gamma_2^\sigma I_{h1} + \gamma_3^\sigma A_{h12} + \gamma_4^\sigma I_{h12}} S_v - \frac{\beta_{v2}^\sigma (A_{h2} + I_{h2} + A_{h12} + I_{h12})}{1 + \gamma_1^\sigma A_{h2} + \gamma_2^\sigma I_{h2} + \gamma_3^\sigma A_{h12} + \gamma_4^\sigma I_{h12}} S_v - \mu_v^\sigma S_v, \\
{}^C\mathcal{D}_{0+}^\sigma E_{v1}(t) &= \frac{\beta_{v1}^\sigma (A_{h1} + I_{h1} + A_{h12} + I_{h12})}{1 + \gamma_1^\sigma A_{h1} + \gamma_2^\sigma I_{h1} + \gamma_3^\sigma A_{h12} + \gamma_4^\sigma I_{h12}} S_v - (\mu_v^\sigma + \omega_{v1}^\sigma) E_{v1}, \\
{}^C\mathcal{D}_{0+}^\sigma E_{v2}(t) &= \frac{\beta_{v2}^\sigma (A_{h2} + I_{h2} + A_{h12} + I_{h12})}{1 + \gamma_1^\sigma A_{h2} + \gamma_2^\sigma I_{h2} + \gamma_3^\sigma A_{h12} + \gamma_4^\sigma I_{h12}} S_v - (\mu_v^\sigma + \omega_{v2}^\sigma) E_{v2}, \\
{}^C\mathcal{D}_{0+}^\sigma I_{v1}(t) &= \omega_{v1}^\sigma E_{v1} - (\mu_v^\sigma + \rho_{v1}^\sigma) I_{v1}, \\
{}^C\mathcal{D}_{0+}^\sigma I_{v2}(t) &= \omega_{v2}^\sigma E_{v2} - (\mu_v^\sigma + \rho_{v2}^\sigma) I_{v2}.
\end{aligned}
\tag{4}$$

The model can be written in compact form as:

$$\begin{cases} {}^C\mathcal{D}_t^\sigma \Phi(t) = \mathcal{K}(t, \Phi(t)), \\ \Phi(0) = \Phi_0, \end{cases}
\tag{5}$$

The diagram of the model (4) is given in the Fig. 1.

Analysis of the sub-models

The complete model has 22 equations, which is quite complex to analyze qualitatively. We will therefore, consider the strain 1 and strain 2 sub-models for local and global stability analyses. The sub-models analyses results will help to examine the stability of the complete model.

Analysis of the strain 1 only sub-model

The strain 1-only sub-model is obtained by setting the classes $E_{h2} = E_{h12} = A_{h2} = A_{h12} = I_{h2} = I_{h12} = Q_{h2} = Q_{h12} = R_{h2} = R_{h12} = E_{v2} = I_{v2}$ equal to 0. The model is given by:

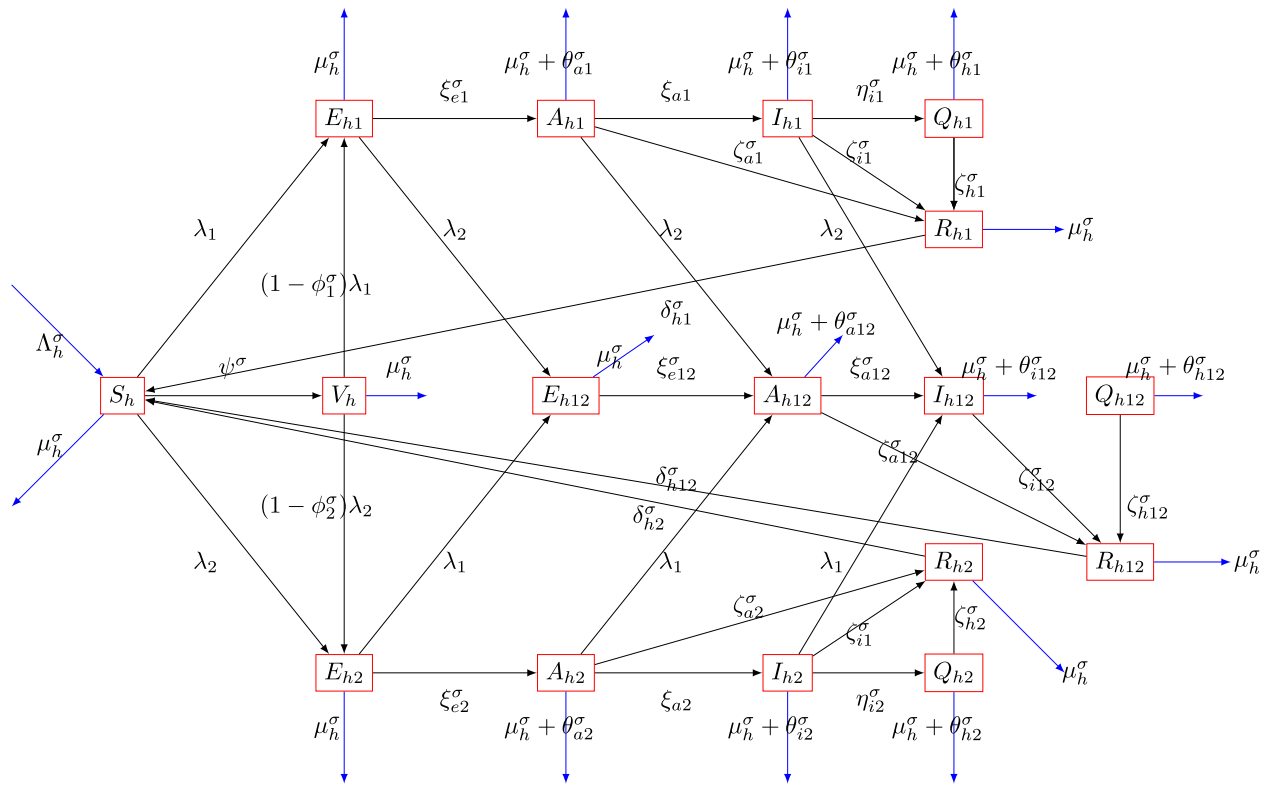


Figure 1. Schematic diagram of the model (4) where $\lambda_1 = \frac{\beta_{h1}^\sigma I_{v1}}{1 + \alpha_1^\sigma I_{v1}}$ and $\lambda_2 = \frac{\beta_{h2}^\sigma I_{v2}}{1 + \alpha_2^\sigma I_{v2}}$.

$$\begin{aligned}
 {}^C\mathcal{D}_{0+}^\sigma S_h(t) &= \Lambda_h^\sigma - \frac{\beta_{h1}^\sigma I_{v1}}{1 + \alpha_1^\sigma I_{v1}} S_h - (\mu_h^\sigma + \psi^\sigma) S_h + \delta_{h1}^\sigma R_{h1}, \\
 {}^C\mathcal{D}_{0+}^\sigma V_h(t) &= \psi^\sigma S_h - (1 - \phi_1^\sigma) \frac{\beta_{h1}^\sigma I_{v1}}{1 + \alpha_1^\sigma I_{v1}} V_h - \mu_h^\sigma V_h, \\
 {}^C\mathcal{D}_{0+}^\sigma E_{h1}(t) &= \frac{\beta_{h1}^\sigma I_{v1}}{1 + \alpha_1^\sigma I_{v1}} [S_h + (1 - \phi_1^\sigma) V_h] - (\mu_h^\sigma + \xi_{e1}^\sigma) E_{h1}, \\
 {}^C\mathcal{D}_{0+}^\sigma A_{h1}(t) &= \xi_{e1}^\sigma E_{h1} - (\mu_h^\sigma + \xi_{a1}^\sigma + \zeta_{a1}^\sigma + \theta_{a1}^\sigma) A_{h1}, \\
 {}^C\mathcal{D}_{0+}^\sigma I_{h1}(t) &= \xi_{a1}^\sigma A_{h1} - (\mu_h^\sigma + \zeta_{i1}^\sigma + \theta_{i1}^\sigma + \eta_{i1}^\sigma) I_{h1}, \\
 {}^C\mathcal{D}_{0+}^\sigma Q_{h1}(t) &= \eta_{i1}^\sigma I_{h1} - (\mu_h^\sigma + \zeta_{h1}^\sigma + \theta_{h1}^\sigma) Q_{h1}, \\
 {}^C\mathcal{D}_{0+}^\sigma R_{h1}(t) &= \zeta_{a1}^\sigma A_{h1} + \zeta_{i1}^\sigma I_{h1} + \zeta_{h1}^\sigma Q_{h1} - (\mu_h^\sigma + \delta_{h1}^\sigma) R_{h1}, \\
 {}^C\mathcal{D}_{0+}^\sigma S_v(t) &= \Lambda_v^\sigma - \frac{\beta_{v1}^\sigma (A_{h1} + I_{h1})}{1 + \gamma_1^\sigma A_{h1} + \gamma_2^\sigma I_{h1}} S_v - \mu_v^\sigma S_v, \\
 {}^C\mathcal{D}_{0+}^\sigma E_{v1}(t) &= \frac{\beta_{v1}^\sigma (A_{h1} + I_{h1})}{1 + \gamma_1^\sigma A_{h1} + \gamma_2^\sigma I_{h1}} S_v - (\mu_v^\sigma + \omega_{v1}^\sigma) E_{v1}, \\
 {}^C\mathcal{D}_{0+}^\sigma I_{v1}(t) &= \omega_{v1}^\sigma E_{v1} - (\mu_v^\sigma + \rho_{v1}^\sigma) I_{v1}.
 \end{aligned} \tag{6}$$

The strain-1 only infection-free equilibrium is given by:

$$\mathcal{D}_{01} = \left(\frac{\Lambda_h^\sigma}{\mu_h^\sigma + \psi^\sigma}, \frac{\psi^\sigma}{\mu_h^\sigma} \left(\frac{\Lambda_h^\sigma}{\mu_h^\sigma + \psi^\sigma} \right), 0, 0, 0, 0, 0, 0, \frac{\Lambda_v}{\mu_v}, 0 \right).$$

The matrix of new infection is given as:

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & \beta_{h1}^\sigma A_1^* \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_{v1}^\sigma A_2^* & \beta_{v1}^\sigma A_2^* & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \tag{7}$$

where $A_1^* = (S_h^* + (1 - \phi_1^\sigma) V_h^*)$, $A_2^* = S_v^*$.

The matrix for transfer of infection by all other means is given as:

$$V = \begin{bmatrix} K_1 & 0 & 0 & 0 & 0 & 0 \\ -\xi_{e1}^\sigma & K_2 & 0 & 0 & 0 & 0 \\ 0 & -\xi_{a1}^\sigma & K_3 & 0 & 0 & 0 \\ 0 & 0 & -\eta_{i1}^\sigma & K_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & K_5 & 0 \\ 0 & 0 & 0 & 0 & -\omega_{v1}^\sigma & K_6 \end{bmatrix}, \quad (8)$$

where $K_1 = (\mu_h^\sigma + \xi_{e1}^\sigma)$, $K_2 = (\mu_h^\sigma + \xi_{a1}^\sigma + \zeta_{a1}^\sigma + \theta_{a1}^\sigma)$, $K_3 = (\mu_h^\sigma + \zeta_{i1}^\sigma + \theta_{i1}^\sigma + \eta_{i1}^\sigma)$

$K_4 = (\mu_h^\sigma + \zeta_{h1}^\sigma + \theta_{h1}^\sigma)$, $K_5 = (\mu_v^\sigma + \omega_{v1}^\sigma)$, $K_6 = (\mu_v^\sigma + \rho_{v1}^\sigma)$.

The reproduction number \mathcal{R}_{01} for strain-1 is given as:

$$\mathcal{R}_{01} = \sqrt{\frac{\beta_{h1}^\sigma \beta_{v1}^\sigma A_1^* A_2^* \xi_{e1}^\sigma \omega_{v1}^\sigma (K_3 + \xi_{a1}^\sigma)}{K_1 K_2 K_3 K_5 K_6}}.$$

This can be written as:

$$\mathcal{R}_{01} = \sqrt{\mathcal{R}_{1H} \mathcal{R}_{1V}}.$$

where $\mathcal{R}_{1V} = \frac{\beta_{v1}^\sigma A_2^* \omega_{v1}^\sigma}{K_5 K_6}$ and $\mathcal{R}_{1H} = \frac{\beta_{v1}^\sigma A_2^* \xi_{e1}^\sigma}{K_1 K_2} + \frac{\beta_{v1}^\sigma A_2^* \xi_{e1}^\sigma \xi_{a1}^\sigma}{K_1 K_2 K_3}$.

The term \mathcal{R}_{1V} is the average number of new dengue strain-1 infections generated by one infected vector who progresses through the stages $\mathcal{E}_{v1} \rightarrow \mathcal{I}_{v1}$, in the susceptible human population. The term $\frac{\omega_{v1}^\sigma}{K_5}$ accounts for the average duration of infection in vectors in the exposed class while $\frac{1}{K_6}$ accounts for the average duration of infection in vectors in the infected stage. The term is product of the transmission rate of susceptible human by dengue infected mosquitoes (β_{v1}^σ) and the mean duration of infection in the mosquitoes $\frac{\omega_{v1}^\sigma}{K_5 K_6}$.

The term \mathcal{R}_{1H} is the average number of new dengue strain-1 infections in vectors generated by one infected individual who progresses through the stages $\mathcal{E}_{h1} \rightarrow \mathcal{A}_{h1}$ or $\mathcal{E}_{h1} \rightarrow \mathcal{A}_{h1} \rightarrow \mathcal{I}_{h1}$. The term $\frac{\beta_{v1}^\sigma A_2^* \xi_{e1}^\sigma \xi_{a1}^\sigma}{K_1 K_2 K_3}$ is the product of transmission rate in mosquitoes by a typical infected human β_{v1}^σ and the mean duration of infection in human $\frac{\xi_{e1}^\sigma \xi_{a1}^\sigma}{K_1 K_2 K_3}$. The explanation of the the term $\frac{\xi_{e1}^\sigma \xi_{a1}^\sigma}{K_1 K_2 K_3}$ is given as

- $\frac{\xi_{e1}^\sigma}{K_1}$ accounts for the duration of infection in human in the exposed class.
- $\frac{\xi_{a1}^\sigma}{K_2}$ accounts for the duration of infection in human in the asymptomatic class.
- $\frac{1}{K_3}$ accounts for the duration of infection in human in the infected (symptomatic) class.

Consider the Lyapunov function:

$$\mathcal{L}_1 = \left(\frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma \xi_{e1}^\sigma}{K_1 K_2 K_5 K_6} + \frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma \xi_{e1}^\sigma \xi_{a1}^\sigma}{K_1 K_2 K_3 K_5 K_6} \right) E_{h1} + \left(\frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma}{K_2 K_5 K_6} + \frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma \xi_{a1}^\sigma}{K_2 K_3 K_5 K_6} \right) A_{h1} \\ + \left(\frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma}{K_3 K_5 K_6} \right) I_{h1} + \left(\frac{\omega_{v1}^\sigma \mathcal{R}_{01}}{K_5 K_6} \right) E_{v1} + \left(\frac{\mathcal{R}_{01}}{K_6} \right) I_{v1}. \quad (9)$$

The time derivative of fractional order σ is given by:

$${}^C D_{0+}^\sigma \mathcal{L}_1 = \left(\frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma \xi_{e1}^\sigma}{K_1 K_2 K_5 K_6} + \frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma \xi_{e1}^\sigma \xi_{a1}^\sigma}{K_1 K_2 K_3 K_5 K_6} \right) {}^C D_{0+}^\sigma E_{h1} + \left(\frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma}{K_2 K_5 K_6} + \frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma \xi_{a1}^\sigma}{K_2 K_3 K_5 K_6} \right) {}^C D_{0+}^\sigma A_{h1} \\ + \left(\frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma}{K_3 K_5 K_6} \right) {}^C D_{0+}^\sigma I_{h1} + \left(\frac{\omega_{v1}^\sigma \mathcal{R}_{01}}{K_5 K_6} \right) {}^C D_{0+}^\sigma E_{v1} + \left(\frac{\mathcal{R}_{01}}{K_6} \right) {}^C D_{0+}^\sigma I_{v1}. \quad (10)$$

Substituting the values of the fractional derivatives for each compartments gives:

$${}^C D_{0+}^\sigma \mathcal{L}_1 = \left(\frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma \xi_{e1}^\sigma}{K_1 K_2 K_5 K_6} + \frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma \xi_{e1}^\sigma \xi_{a1}^\sigma}{K_1 K_2 K_3 K_5 K_6} \right) \left(\frac{\beta_{h1}^\sigma I_{v1}}{1 + \alpha_1^\sigma I_{v1}} [S_h + (1 - \phi_1^\sigma) V_h] - K_1 E_{h1} \right) \\ + \left(\frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma}{K_2 K_5 K_6} + \frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma \xi_{a1}^\sigma}{K_2 K_3 K_5 K_6} \right) \left(\xi_{e1}^\sigma E_{h1} - K_2 A_{h1} \right) + \left(\frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma}{K_3 K_5 K_6} \right) \left(\xi_{a1}^\sigma A_{h1} - K_3 I_{h1} \right) \\ + \left(\frac{\omega_{v1}^\sigma \mathcal{R}_{01}}{K_5 K_6} \right) \left(\frac{\beta_{v1}^\sigma (A_{h1} + I_{h1})}{1 + \gamma_1^\sigma A_{h1} + \gamma_2^\sigma I_{h1}} S_v - K_5 E_{v1} \right) + \left(\frac{\mathcal{R}_{01}}{K_6} \right) \left(\omega_{v1}^\sigma E_{v1} - K_6 I_{v1} \right), \quad (11)$$

which can also be written thus:

$$\begin{aligned}
{}^c D_{0+}^\sigma \mathcal{L}_1 \leq & \left(\frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma \xi_{e1}^\sigma}{K_1 K_2 K_5 K_6} + \frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma \xi_{e1}^\sigma \xi_{a1}^\sigma}{K_1 K_2 K_3 K_5 K_6} \right) \left(\beta_{h1}^\sigma I_{v1} (A_1^*) - K_1 E_{h1} \right) \\
& + \left(\frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma}{K_2 K_5 K_6} + \frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma \xi_{a1}^\sigma}{K_2 K_3 K_5 K_6} \right) \left(\xi_{e1}^\sigma E_{h1} - K_2 A_{h1} \right) + \left(\frac{A_2^* \beta_{v1}^\sigma \omega_{v1}^\sigma}{K_3 K_5 K_6} \right) \left(\xi_{a1}^\sigma A_{h1} - K_3 I_{h1} \right) \\
& + \left(\frac{\omega_{v1}^\sigma \mathcal{R}_{01}}{K_5 K_6} \right) \left(\beta_{v1}^\sigma (A_{h1} + I_{h1}) A_2^* - K_5 E_{v1} \right) + \left(\frac{\mathcal{R}_{01}}{K_6} \right) \left(\omega_{v1}^\sigma E_{v1} - K_6 I_{v1} \right),
\end{aligned} \quad (12)$$

which on simplification gives:

$${}^c D_{0+}^\sigma \mathcal{L}_1 \leq \mathcal{R}_{01} \left(\mathcal{R}_{01} - 1 \right) I_{v1} + \frac{\omega_{v1}^\sigma \beta_{v1}^\sigma A_2^*}{K_5 K_6} \left(\mathcal{R}_{01} - 1 \right) A_{h1} + \frac{\omega_{v1}^\sigma \beta_{v1}^\sigma A_2^*}{K_5 K_6} \left(\mathcal{R}_{01} - 1 \right) I_{h1}. \quad (13)$$

Clearly $\mathcal{R}_{01} \leq 1$ is satisfied since the above inequality with positive parameters shows that $D_t^\alpha \mathcal{L}_1$ is negative semi definite. It follows from the results in^{39,40} that the disease free equilibrium is globally asymptotically stable if $\mathcal{R}_{01} \leq 1$ and unstable if $\mathcal{R}_{01} > 1$.

Local stability for sub-model of strain-1

The stability of system (6) in the neighborhood of the DFE is analyzed by Jacobian of system (6) evaluated at DFE \mathcal{D}_{01} , which is given as:

$$\begin{bmatrix}
-K_0 & 0 & 0 & 0 & 0 & 0 & \delta_{h1}^\sigma & 0 & 0 & -\beta_{h1}^\sigma S_h^* \\
\psi_h^\sigma & -\mu_h^\sigma & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -(1-\phi_1^\sigma) \beta_{h1}^\sigma V_h^* \\
0 & 0 & -K_1 & 0 & 0 & 0 & 0 & 0 & 0 & \beta_{h1}^\sigma A_1^* \\
0 & 0 & \xi_{e1}^\sigma & -K_2 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \xi_{a1}^\sigma & -K_3 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \eta_{i1}^\sigma & -K_4 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \xi_{a1}^\sigma & \xi_{i1}^\sigma & \xi_{h1}^\sigma & -K_{13} & 0 & 0 & 0 \\
0 & 0 & 0 & -\beta_{v1}^\sigma A_2^* & -\beta_{v1}^\sigma A_2^* & 0 & 0 & -\mu_v^\sigma & 0 & 0 \\
0 & 0 & 0 & \beta_{v1}^\sigma A_2^* & \beta_{v1}^\sigma A_2^* & 0 & 0 & 0 & -K_5 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \omega_{v1}^\sigma & -K_6
\end{bmatrix}, \quad (14)$$

where

$$K_0 = \mu_h^\sigma + \psi_h^\sigma, K_{13} = \mu_h^\sigma + \delta_{h1}^\sigma$$

The “characteristic polynomial” is given by:

$$\begin{aligned}
& (\lambda + K_0)(\lambda + K_4)(\lambda + K_{13})(\lambda + \mu_h^\sigma)(\lambda + \mu_v^\sigma) \left((\lambda + K_1)(\lambda + K_2)(\lambda \right. \\
& \left. + K_3)(\lambda + K_5)(\lambda + K_6) - A_1^* A_2^* \beta_{h1}^\sigma \beta_{v1}^\sigma \xi_{e1}^\sigma (\lambda + K_3 + \xi_{a1}^\sigma) \omega_{v1}^\sigma \right) = 0.
\end{aligned} \quad (15)$$

This can be written as:

$$\begin{aligned}
& (\lambda + K_0)(\lambda + K_4)(\lambda + K_{13})(\lambda + \mu_h^\sigma)(\lambda + \mu_v^\sigma) \left(\lambda^5 + a_{11} \lambda^4 + a_{12} \lambda^3 \right. \\
& \left. + a_{13} \lambda^2 + a_{14} \lambda + K_1 K_2 K_3 K_5 K_6 \left(1 - R_{01}^2 \right) \right) = 0,
\end{aligned} \quad (16)$$

where

$$\begin{aligned}
a_{11} &= K_1 + K_2 + K_3 + K_5 + K_6 \\
a_{12} &= K_1 K_2 + K_1 K_3 + K_2 K_3 + K_1 K_5 + K_2 K_5 + K_3 K_5 + K_1 K_6 + K_2 K_6 + K_3 K_6 + K_5 K_6 \\
a_{13} &= K_1 K_2 K_3 + K_1 K_2 K_5 + K_1 K_3 K_5 + K_2 K_3 K_5 + K_1 K_2 K_6 \\
&+ K_1 K_3 K_6 + K_2 K_3 K_6 + K_1 K_5 K_6 + K_2 K_5 K_6 + K_3 K_5 K_6 \\
a_{14} &= K_1 K_2 K_3 K_5 + K_1 K_2 K_3 K_6 + K_1 K_2 K_5 K_6 + K_1 K_3 K_5 K_6 + K_2 K_3 K_5 K_6 - A_1^* A_2^* \beta_{h1}^\sigma \beta_{v1}^\sigma \xi_{e1}^\sigma \omega_{v1}^\sigma.
\end{aligned} \quad (17)$$

The eigenvalues are given by:

$$\lambda_1 = -\mu_v^\sigma, \quad \lambda_2 = -K_0, \quad \lambda_3 = -K_4, \quad \lambda_4 = -\mu_h^\sigma, \quad \lambda_5 = -K_{13},$$

and the solution of the equations is given by

$$\left(\lambda^5 + a_{11} \lambda^4 + a_{12} \lambda^3 + a_{13} \lambda^2 + a_{14} \lambda + K_1 K_2 K_3 K_5 K_6 \left(1 - R_{01}^2 \right) \right). \quad (18)$$

From the Routh–Hurwitz criterion, the Eq. (18) has roots with negative real parts if and only if $\mathcal{R}_{01} < 1$. Hence, the DFE is locally asymptotically stable if $\mathcal{R}_{01} < 1$.

Similar results can also be established for the Strain 2 only sub-model.

Analysis of the complete model

Invariant domain

Theorem 4.1 The closed set $\mathcal{D} = \mathcal{D}_h \times \mathcal{D}_v$, where

$$\begin{aligned} \mathcal{D}_h = & \left\{ (S_h(t), V_h(t), E_{h1}(t), E_{h2}(t), E_{h12}(t), A_{h1}(t), A_{h2}(t), A_{h12}(t), I_{h1}(t), I_{h2}(t), I_{h12}(t), Q_{h1}(t), Q_{h2}(t), Q_{h12}(t), \right. \\ & R_{h1}(t), R_{h2}(t), R_{h12}(t)) \in \mathfrak{R}_+^{17} : \\ & S_h(t) + V_h(t) + E_{h1}(t) + E_{h2}(t) + E_{h12}(t) + A_{h1}(t) + A_{h2}(t) + A_{h12}(t) + I_{h1}(t) + I_{h2}(t) + I_{h12}(t) + \\ & \left. Q_{h1}(t) + Q_{h2}(t) + Q_{h12}(t) + R_{h1}(t) + R_{h2}(t) + R_{h12}(t) \leq \frac{\Lambda_h^\sigma}{\mu_h^\sigma} \right\}, \\ \mathcal{D}_v = & \left\{ (S_v(t), E_{v1}(t), E_{v2}(t), I_{v1}(t), I_{v2}(t)) \in \mathfrak{R}_+^5 : \right. \\ & \left. S_v(t) + E_{v1}(t) + E_{v2}(t) + I_{v1}(t) + I_{v2}(t) \leq \frac{\Lambda_v^\sigma}{\mu_v^\sigma} \right\}, \end{aligned}$$

is positively invariant in relation to the system (4).

Proof Adding all the human components of the system (4), we have

$$\begin{aligned} {}^C_0 D_t^\sigma N_h = & \Lambda_h^\sigma - \mu N_h(t) - [\theta_{a1}^\sigma A_{h1} + \theta_{a2}^\sigma A_{h2} + \theta_{a12}^\sigma A_{h12} + \theta_{i1}^\sigma I_{h1} \\ & + \theta_{i2}^\sigma I_{h2} + \theta_{i12}^\sigma I_{h12} + \theta_{q1}^\sigma Q_{h1} + \theta_{q2}^\sigma Q_{h2} + \theta_{q12}^\sigma Q_{h12}]. \end{aligned} \quad (19)$$

From (19), we have

$${}^C_0 D_t^\sigma N_h < \Lambda_h^\sigma - \mu_h^\sigma N_h. \quad (20)$$

Applying Laplace transform on both sides of the inequality (20), we obtain that

$$s^\sigma \mathcal{L}\{N_h(t)\} - s^{\sigma-1} N_h(0) \leq \frac{\Lambda_h^\sigma}{s} - \mu_h^\sigma \mathcal{L}\{N_h(t)\},$$

which further implies that

$$\mathcal{L}\{N_h(t)\} \leq \frac{\Lambda_h^\sigma}{s(s^\sigma + \mu_h^\sigma)} + N_h(0) \frac{s^{\sigma-1}}{s^\sigma + \mu_h^\sigma}. \quad (21)$$

By partial fraction, the above expression reduces to

$$\mathcal{L}\{N_h(t)\} \leq \frac{\Lambda_h^\sigma}{\mu_h^\sigma} \left(\frac{1}{s} \right) - \left(\frac{\Lambda_h^\sigma}{\mu_h^\sigma} - N_h(0) \right) \frac{s^{\sigma-1}}{s^\sigma + \mu_h^\sigma}. \quad (22)$$

The inverse Laplace transform gives

$$N_h(t) \leq \frac{\Lambda_h^\sigma}{\mu_h^\sigma} - \left(\frac{\Lambda_h^\sigma}{\mu_h^\sigma} - N_h(0) \right) E_\sigma(-\mu_h^\sigma t^\sigma). \quad (23)$$

Since the “Mittag-Leffler function” has asymptotic behaviour, we have

$N_h(t) \leq \frac{\Lambda_h^\sigma}{\mu_h^\sigma}$ as $t \rightarrow \infty$. Following the arguments similar to those given above, we have $N_v(t) \leq \frac{\Lambda_v^\sigma}{\mu_v^\sigma}$ as $t \rightarrow \infty$. Therefore, system (4) has solutions in \mathcal{D} and hence is positively invariant.

The basic reproduction number of the model

The disease free equilibrium (DFE) of the model (4) is:

$$\begin{aligned} \mathcal{D}_0 = & (S_h^*, V_h^*, E_{h1}^*, E_{h2}^*, E_{h12}^*, A_{h1}^*, A_{h2}^*, A_{h12}^*, I_{h1}^*, I_{h2}^*, I_{h12}^*, Q_{h1}^*, Q_{h2}^*, Q_{h12}^*, R_{h1}^*, R_{h2}^*, R_{h12}^*, S_v^*, E_{v1}^*, E_{v2}^*, I_{v1}^*, I_{v2}^*) \\ = & \left(\frac{\Lambda_h^\sigma}{\mu_h^\sigma + \psi^\sigma}, \frac{\psi^\sigma}{\mu_h^\sigma} \left(\frac{\Lambda_h^\sigma}{\mu_h^\sigma + \psi^\sigma} \right), 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, \frac{\Lambda_v^\sigma}{\mu_v^\sigma}, 0, 0, 0, 0 \right). \end{aligned}$$

Following the approach from⁴¹,

the “basic reproduction number” of the model (4), is given by

$\mathcal{R}_0 = \max\{\mathcal{R}_{01}, \mathcal{R}_{02}\}$, where \mathcal{R}_{01} and \mathcal{R}_{02} are the associated “reproduction numbers” for dengue strain-1 and dengue strain-2, respectively are given by

$$\mathcal{R}_{01} = \sqrt{\frac{\beta_{h1}^{\sigma} \beta_{v1}^{\sigma} A_1^* A_2^* \xi_{e1}^{\sigma} \omega_{v1}^{\sigma} (K_3 + \xi_{a1}^{\sigma})}{K_1 K_2 K_3 K_5 K_6}}, \quad \mathcal{R}_{02} = \sqrt{\frac{\beta_{h2}^{\sigma} \beta_{v2}^{\sigma} A_3^* A_2^* \xi_{e2}^{\sigma} \omega_{v2}^{\sigma} (K_9 + \xi_{a2}^{\sigma})}{K_7 K_8 K_9 K_{11} K_{12}}}.$$

Also, the following result can be established for the full model:

Local asymptotic stability of the disease free equilibrium (DFE) of the model

Theorem 4.2 The system's DFE, \mathcal{D}_0 , is "locally asymptotically stable" (LAS) if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$.

Existence, uniqueness and Ulam–Hyers stability of the complete model

Existence

In this section, following the approach of²⁸, we study the necessary conditions for existence of solution of the proposed model (4).

Consider a Banach space $\mathbb{E} = \mathcal{C}[\mathcal{J}, \mathbb{R}^{22}]$ equipped with the norm:

$$\|\Phi\| = \sup_{t \in \mathcal{J}} |\Phi(t)|, \text{ where, } |\Phi(t)| = |\Phi_1(t)| + |\Phi_2(t)| + |\Phi_3(t)| + \dots + |\Phi_{21}(t)| + |\Phi_{22}(t)|.$$

The norm on $\mathcal{C}([\mathcal{J}, \mathbb{R}^{22}])$ or $\mathcal{C}([\mathcal{J}, \mathbb{R}])$ will be clear from its context. System (4) can be written in form of the Volterra integral equation given by

$$K(t) = K(0) + \frac{1}{\Gamma(\sigma)} \int_0^t (t - \wp)^{\sigma-1} \mathcal{K}(\wp, K(\wp)) d\wp. \quad (24)$$

Consider $\mathbf{B}_\eta = \{\Phi \in \mathbb{E} : \|\Phi\| \leq \eta\}$, where $\eta \geq |\Phi_0| + \Omega \|\Psi\|$, $\Phi_0 \in \mathbb{R}^{22}$ and $\Omega = \frac{b^\sigma}{\Gamma(\sigma+1)}$. Obviously \mathbf{B}_η is closed convex and bounded subset of \mathbb{E} .

Define operators $P_1, P_2 : \mathbf{B}_\eta \rightarrow \mathbb{E}$ by

$$\begin{aligned} (P_1 \Phi)(t) &= \frac{1}{\Gamma(\sigma)} \int_0^t (t - \wp)^{\sigma-1} \mathcal{K}(\wp, \Phi(\wp)) d\wp \quad \forall t \in \mathcal{J}, \\ (P_2 \Phi)(t) &= \Phi_0, \quad \forall t \in \mathcal{J}, \end{aligned}$$

respectively.

Lemma 5.1 The operator $(P_2 \Phi)$ is μ -Lipschitz with constant $k \in (0, 1)$.

Proof Since operator $(P_2 \Phi)(t)$ is constant so it is Lipschitz with Lipschitz constant $k \in (0, 1)$. By lemma(1.4) $(P_2 \Phi)(t)$ is μ -Lipschitz with constant $k \in (0, 1)$.

Lemma 5.2 If $|\mathcal{K}(t, \Phi(t))| \leq |\Psi(t)|$, for all $(t, \Phi(t)) \in \mathcal{J} \times \mathbb{R}^{22}$ and for some $\Psi \in \mathcal{C}(\mathcal{J}, \mathbb{R}_+)$ with $\|\Psi\| = \sup_{t \in \mathcal{J}} |\Psi(t)|$. Then the operator $(P_1 \Phi)$ is μ -Lipschitz with constant zero.

Proof As the function \mathcal{K} is continuous, so the operator P_1 is also continuous.

Now, for any $\Phi \in \mathbf{B}_\eta$, we have

$$\begin{aligned} \|(P_1 \Phi)\| &= \sup_{t \in \mathcal{J}} \left| P_1 \Phi(t) \right| \\ &= \sup_{t \in \mathcal{J}} \left| \frac{1}{\Gamma(\sigma)} \int_0^t (t - \wp)^{\sigma-1} \mathcal{K}(\wp, \Phi(\wp)) d\wp \right| \\ &\leq \sup_{t \in \mathcal{J}} \left| \frac{1}{\Gamma(\sigma)} \int_0^t (t - \wp)^{\sigma-1} |\Psi(\wp)| d\wp \right| \\ &\leq \frac{\|\Psi\|}{\Gamma(\sigma)} \sup_{t \in \mathcal{J}} \int_0^t (t - \wp)^{\sigma-1} d\wp \\ &\leq \frac{b^\sigma}{\Gamma(\sigma+1)} \|\Psi\| \\ &= \Omega \|\Psi\| \leq \eta. \end{aligned}$$

Thus, $P_1(\mathbf{B}_\eta) \subset \mathbf{B}_\eta$. As $\overline{P_1(\mathbf{B}_\eta)}$ is bounded and closed. To apply the Arzela Ascoli theorem, we now prove that $\overline{P_1(\mathbf{B}_\eta)}$ is equicontinuous.

For any $\Phi \in \mathbf{B}_\eta$, consider

$$\begin{aligned}
\left| (P_1\Phi)(t_2) - (P_1\Phi)(t_1) \right| &= \left| \frac{1}{\Gamma(\sigma)} \int_0^{t_2} (t_2 - \wp)^{\sigma-1} \mathcal{K}(\wp, \Phi(\wp)) d\wp \right. \\
&\quad \left. - \frac{1}{\Gamma(\sigma)} \int_0^{t_1} (t_1 - \wp)^{\sigma-1} \mathcal{K}(\wp, \Phi(\wp)) d\wp \right| \\
&= \frac{1}{\Gamma(\sigma)} \left[\left| \int_0^{t_1} [(t_2 - \wp)^{\sigma-1} - (t_1 - \wp)^{\sigma-1}] \mathcal{K}(\wp, \Phi(\wp)) d\wp \right| \right. \\
&\quad \left. + \int_{t_1}^{t_2} (t_2 - \wp)^{\sigma-1} \mathcal{K}(\wp, \Phi(\wp)) d\wp \right] \\
&\leq \frac{\|\Psi\|}{\Gamma(\sigma+1)} \left[(t_2^\sigma - t_1^\sigma) \right].
\end{aligned}$$

Clearly, the right hand side of the above inequality vanishes as $t_2 \rightarrow t_1$. Thus, $P_1\mathbf{B}_\eta$ is equicontinuous and so it $\overline{P_1(\mathbf{B}_\eta)}$. Hence, $\overline{P_1(\mathbf{B}_\eta)}$ being closed, bounded and equicontinuous is compact which gives that P_1 is a compact operator. Thus by lemma (1.3) P_1 is μ -Lipschitz with constant 0.

Theorem 5.1 Assume that the conditions of the lemmas (5.1) and (5.2) hold. Then the integral equation has at least one solution in E Moreover, the set of solutions of (24) is bounded in E .

Proof : By Lemma 5.1, P_2 is μ -Lipschitz with constant k , and by Lemma 5.2, P_1 is μ -Lipschitz with constant 0. Hence $\mathbf{P} = \mathbf{P}_1 + \mathbf{P}_2$ is μ -Lipschitz with constant k and hence $\mathbf{P}_1 + \mathbf{P}_2$ is μ -condensing. Define

$$G = \{\phi \in E : h \in [0, 1] \text{ such that } \phi = h\mathbf{P}(\phi)\}$$

Let $\phi \in E$, then we have

$$\|\phi\| = \|h\mathbf{P}(\phi)\| = h\|\mathbf{P}(\phi)\| = h\|K(0) + \frac{1}{\Gamma(\sigma)} \int_0^t (t-s)^{\sigma-1} \kappa(t, \phi(s)) ds\| \leq h[\phi_0 + \frac{b^\sigma \|\Psi\|}{\Gamma(\sigma+1)}] \leq \eta.$$

Thus $G \subseteq B_\eta$ and hence bounded and contained in $B_r(0)$. By Theorem (1.1), an operator \mathbf{P} has atleast one solution.

Uniqueness

Theorem 5.2 Suppose that the function $\mathcal{K} \in \mathcal{C}([\mathcal{J}, \mathbb{R}^{22}])$ satisfy the Lipschitz condition

$$|\mathcal{K}(t, \Phi_1(t)) - \mathcal{K}(t, \Phi_2(t))| \leq \mathcal{L}_\mathcal{K} |\Phi_1(t) - \Phi_2(t)|, \quad (25)$$

for all $t \in \mathcal{J}$ and each $\Phi_1, \Phi_2 \in \mathbb{E}$, $\mathcal{L}_\mathcal{K} > 0$. Then system (4), or its equivalent form (24) has unique solution whenever $\Omega\mathcal{L}_\mathcal{K} < 1$.

Proof Consider the operator $P : \mathbb{E} \rightarrow \mathbb{E}$ defined by

$$(P\Phi)(t) = \Phi_0 + \frac{1}{\Gamma(\sigma)} \int_0^t \mathcal{K}(\wp, \Phi(t))(t - \wp)^{\sigma-1} d\wp.$$

Now for any $\Phi_1, \Phi_2 \in \mathbb{E}$, we get

$$\begin{aligned}
\left\| (P\Phi_1) - (P\Phi_2) \right\| &\leq \sup_{t \in \mathcal{J}} \left\| \left[\Phi_0 + \frac{1}{\Gamma(\sigma)} \int_0^t (t-\varrho)^{\sigma-1} \mathcal{K}(\varrho, \Phi_1(\varrho)) d\varrho \right. \right. \\
&\quad \left. \left. - \left(\Phi_0 + \frac{1}{\Gamma(\sigma)} \int_0^t (t-\varrho)^{\sigma-1} \mathcal{K}(\varrho, \Phi_2(\varrho)) d\varrho \right) \right] \right\| \\
&\leq \sup_{t \in \mathcal{J}} \frac{1}{\Gamma(\sigma)} \int_0^t (t-\varrho)^{\sigma-1} \left| \mathcal{K}(\varrho, \Phi_1(\varrho)) \right. \\
&\quad \left. - \mathcal{K}(\varrho, \Phi_2(\varrho)) \right| d\varrho \\
&\leq \sup_{t \in \mathcal{J}} \frac{\mathcal{L}_{\mathcal{K}}}{\Gamma(\sigma)} \int_0^t (t-\varrho)^{\sigma-1} \left| \Phi_1(\varrho) - \Phi_2(\varrho) \right| d\varrho \\
&\leq \frac{\mathcal{L}_{\mathcal{K}}}{\Gamma(\sigma)} \left\| \Phi_1 - \Phi_2 \right\| \sup_{t \in \mathcal{J}} \int_0^t (t-\varrho)^{\sigma-1} d\varrho \\
&\leq \frac{b^\sigma}{\Gamma(\sigma+1)} \mathcal{L}_{\mathcal{K}} \left\| \Phi_1 - \Phi_2 \right\| \\
&= \Omega \mathcal{L}_{\mathcal{K}} \left\| \Phi_1(t) - \Phi_2(t) \right\|.
\end{aligned}$$

This implies that P is a contraction.

As $P(\Phi)(t) = P_1(\Phi)(t) + P_2(\Phi)(t)$, so $P\mathbf{B}_\eta \subset \mathbf{B}_\eta$. Since the set \mathbf{B}_η is closed, it follows from Banach contraction principle that the proposed model possess a unique solution.

Ulam–Hyers stability

The stability result for the fractional system is now studied in the frame-work of Ulam–Hyers (UH) stability^{42,43}. Let $\mathbb{E} = \mathcal{C}(\mathcal{J}, \mathbb{R}^{22})$ be space of “continuous functions” from \mathcal{J} to \mathbb{R}^{22} coupled with the norm $\|\Phi\| = \sup_{t \in \mathcal{J}} |\Phi(t)|$, where $\mathcal{J} = [0, b]$.

Definition 6.1 The model (4) or its transformed version given by

$$\begin{cases} {}^C D_t^\sigma \Phi(t) = \mathcal{K}(t, \Phi(t)), \\ \Phi(0) = \Phi_0, \end{cases} \quad (26)$$

is UH stable if $\exists k > 0$ such that for any $\varepsilon > 0$ and the given solution of (26) satisfying the following inequality

$$\| {}^C D^\sigma \bar{\Phi}(t) - \mathcal{K}(t, \bar{\Phi}(t)) \| \leq \varepsilon, \quad t \in \mathcal{J}, \quad \varepsilon = \max(\varepsilon_i)^T, \quad i = 1, 2, \dots, 22. \quad (27)$$

\exists unique solution $\Phi \in \mathbb{E}$ of system (26) in such a way that

$$\| \bar{\Phi}(t) - \Phi(t) \| \leq k\varepsilon, \quad t \in \mathcal{J}, \quad k = \max(k_j)^T, \quad j = 1, 2, \dots, 22.$$

Definition 6.2 System (26) is “generalized UH stable” if \exists a continuous function $\phi : \mathbb{R}^+ \rightarrow \mathbb{R}^+$ with $\phi(0) = 0$ such that for any other solution $\bar{\Phi} \in \mathbb{E}$ of the inequality (27), there exists a unique solution $\Phi \in \mathbb{E}$ satisfying the following:

$$\| \bar{\Phi}(t) - \Phi(t) \| \leq \phi(\varepsilon), \quad t \in \mathcal{J}, \quad \phi = \max(\phi_j)^T, \quad j = 1, 2, \dots, 22.$$

Remark 6.1 A function $\bar{\Phi} \in \mathbb{E}$ satisfies the inequality (27) if and only if there exists a function $h \in \mathbb{E}$ having the following properties:

- $\|h(t)\| \leq \varepsilon, \quad t \in \mathcal{J}.$
- ${}^C D^\sigma \bar{\Phi}(t) = \mathcal{K}(t, \bar{\Phi}(t)) + h(t), \quad t \in \mathcal{J}.$

Lemma 6.1 If $\bar{\Phi} \in \mathbb{E}$ holds for system (27), then $\bar{\Phi}$ also holds for the following:

$$\left\| \bar{\Phi}(t) - \left(\bar{\Phi}_0 + \frac{1}{\Gamma(\sigma)} \int_0^t (t-\varrho)^{\sigma-1} \mathcal{K}(\varrho, \bar{\Phi}(\varrho)) d\varrho \right) \right\| \leq \Omega \varepsilon \quad (28)$$

Proof Using (ii.) of the Remark 6.1, we have ${}^C D^\sigma \bar{\Phi}(t) = \mathcal{K}(t, \bar{\Phi}(t)) + h(t), \quad t \in \mathcal{J}$, which on applying Caputo integral gives that

$$\bar{\Phi}(t) = \bar{\Phi}_0 + \frac{1}{\Gamma(\sigma)} \int_0^t (t - \wp)^{\sigma-1} \mathcal{K}(\wp, \bar{\Phi}(\wp)) d\wp + \frac{1}{\Gamma(\sigma)} \int_0^t (t - \wp)^{\sigma-1} h(\wp) d\wp \quad (29)$$

Re-arranging and taking the norm on the both sides and applying the item (i.) of Remark 6.1, we obtain that

$$\begin{aligned} \left| \bar{\Phi}(t) - \left(\bar{\Phi}_0 + \frac{1}{\Gamma(\sigma)} \int_0^t (t - \wp)^{\sigma-1} \mathcal{K}(\wp, \bar{\Phi}(\wp)) d\wp \right) \right| & \leq \frac{1}{\Gamma(\sigma)} \int_0^t (t - \wp)^{\sigma-1} |h(\wp)| d\wp \\ & \leq \left(\frac{b^\sigma}{\Gamma(\sigma+1)} \right) \varepsilon \leq \Omega \varepsilon. \end{aligned}$$

Theorem 6.1 For all $\Phi \in \mathbb{E}$ and the Lipschitz mapping $\mathcal{K} : \mathcal{J} \times \mathbb{R}^{22} \rightarrow \mathbb{R}^{22}$ with Lipschitz constant $\mathcal{L}_{\mathcal{K}} > 0$ and $1 - \Omega \mathcal{L}_{\mathcal{K}} > 0$, where $\Omega = \frac{b^\sigma}{\Gamma(\sigma+1)}$, the model (26) is generalized UH stable.

Proof If $\bar{\Phi} \in \mathbb{E}$ satisfies the inequality given by (27) and $\Phi \in \mathbb{E}$ is a unique solution of (26). Then $\forall \varepsilon > 0$, $t \in \mathcal{J}$ together with Lemma 6.1, we have

$$\begin{aligned} \|\bar{\Phi}(t) - \Phi(t)\| &= \sup_{t \in \mathcal{J}} \left| \bar{\Phi}_0 + \frac{1}{\Gamma(\sigma)} \int_0^t (t - \wp)^{\sigma-1} \mathcal{K}(\wp, \bar{\Phi}(\wp)) d\wp + \frac{1}{\Gamma(\sigma)} \int_0^t (t - \wp)^{\sigma-1} h(\wp) d\wp \right. \\ &\quad \left. - \left(\Phi_0 + \frac{1}{\Gamma(\sigma)} \int_0^t (t - \wp)^{\sigma-1} \mathcal{K}(\wp, \Phi(\wp)) d\wp \right) \right| \\ &\leq \sup_{t \in \mathcal{J}} |\bar{\Phi}_0 - \Phi_0| + \sup_{t \in \mathcal{J}} \left[|h(t)| \left(\frac{1}{\Gamma(\sigma)} \int_0^t (t - \wp)^{\sigma-1} d\wp \right) \right] \\ &\quad + \sup_{t \in \mathcal{J}} \frac{1}{\Gamma(\sigma)} \int_0^t (t - \wp)^{\sigma-1} |\mathcal{K}(t, \bar{\Phi}(t)) - \mathcal{K}(t, \Phi(t))| d\wp \\ &\leq \Omega \varepsilon + \frac{\mathcal{L}_{\mathcal{K}} \|\bar{\Phi} - \Phi\|}{\Gamma(\sigma)} \sup_{t \in \mathcal{J}} \int_0^t (t - \wp)^{\sigma-1} d\wp \\ &\leq \Omega \varepsilon + \left(\frac{b^\sigma}{\Gamma(\sigma+1)} \right) \mathcal{L}_{\mathcal{K}} \|\bar{\Phi} - \Phi\| \\ &= \Omega \varepsilon + \Omega \mathcal{L}_{\mathcal{K}} \|\bar{\Phi}(t) - \Phi(t)\|. \end{aligned}$$

Thus, we have

$$\|\bar{\Phi} - \Phi\| \leq k \varepsilon, \quad (30)$$

where, $k = \frac{\Omega}{1 - \Omega \mathcal{L}_{\mathcal{K}}}$.

Thus, if we take $\phi(\varepsilon) = k \varepsilon$, then $\phi(0) = 0$ and hence the system (26) is both Ulam Hyers (UH) and generalized UH stable.

Nonstandard finite difference scheme

In order to analyze the disease's spread, we applied a nonstandard finite difference (NSFD) scheme⁴⁴ for the model that can ensure the solution's positivity and displays the right asymptotic behavior. Consider the Caputo derivative

$${}^c D_{0+}^\sigma f(t) = \frac{1}{\Gamma(1-\sigma)} \int_0^t f'(\theta) (t - \theta)^{-\sigma} d\theta.$$

The discretization of the domain $[0, T]$ is given by $t_j = jp$, $j = 0, 1, 2, \dots, N$, where p is the step size $p = \frac{T}{N}$ and T is the final time. The equation becomes for $t = t_{j+1}$

$${}^c D_{0+}^\sigma f(t)|_{t=t_{j+1}} = \frac{1}{\Gamma(1-\sigma)} \sum_{k=0}^j \int_{t_k}^{t_{k+1}} f'(\theta) (t_{j+1} - \theta)^{-\sigma} d\theta. \quad (31)$$

The approximation of $f'(\theta)$ is given as

$$\frac{df(\theta)}{d\theta} = f'(\theta) = \frac{f^{k+1} - f^k}{\Psi(p)},$$

where denominator function $\Psi(p)$ is defined as

$$\Psi(p) = \frac{p^{1-\sigma} (1 - E_\sigma(-(\mu_h^\sigma p)^\sigma))}{(E_\sigma(-(\mu_h^\sigma p)^\sigma)) \Gamma(2-\sigma) \mu_h^\sigma},$$

so that equation (31) becomes

$${}^c D_{0+}^\sigma f(t)|_{t=t_{j+1}} = \frac{1}{\Gamma(1-\sigma)} \sum_{k=0}^j \frac{f^{k+1} - f^k}{\Psi(p)} \int_{t_k}^{t_{k+1}} (t_{j+1} - \theta)^{-\sigma} d\theta, \quad (32)$$

which further gives

$${}^c D_{0+}^\sigma f(t)|_{t=t_{j+1}} = \frac{1}{\Gamma(2-\sigma)} \sum_{k=0}^j \frac{f^{k+1} - f^k}{\Psi(p)} A_{\sigma,j}^k, \quad (33)$$

where $A_{\sigma,j}^k = p^{1-\sigma} \left((j-k+1)^{1-\sigma} - (j-k)^{1-\sigma} \right)$.

Now following the⁴⁴ and using (33), the NSFD scheme for the model 4 is given by the following equations:

$$\begin{aligned} S_h^{j+1} &= \frac{p^{1-\sigma} S_h^j + \Gamma(2-\sigma) \Psi(p) (\Lambda_h^\sigma + \delta_{h1}^\sigma R_{h1}^j + \delta_{h2}^\sigma R_{h2}^j + \delta_{h12}^\sigma R_{h12}^j) - \sum_{k=0}^{j-1} (S_h^{k+1} - S_h^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) \left(\frac{\beta_{h1}^\sigma I_{v1}^j}{1+\alpha_1^\sigma I_{v1}^j} + \frac{\beta_{h2}^\sigma I_{v2}^j}{1+\alpha_2^\sigma I_{v2}^j} + (\mu_h^\sigma + \psi^\sigma) \right)}, \\ V_h^{j+1} &= \frac{p^{1-\sigma} V_h^j + \Gamma(2-\sigma) \Psi(p) (\psi^\sigma S_h^{j+1}) - \sum_{k=0}^{j-1} (V_h^{k+1} - V_h^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) \left((1-\phi_1^\sigma) \frac{\beta_{h1}^\sigma I_{v1}^j}{1+\alpha_1^\sigma I_{v1}^j} + (1-\phi_2^\sigma) \frac{\beta_{h2}^\sigma I_{v2}^j}{1+\alpha_2^\sigma I_{v2}^j} + \mu_h^\sigma \right)}, \\ E_{h1}^{j+1} &= \frac{p^{1-\sigma} E_{h1}^j + \Gamma(2-\sigma) \Psi(p) \left(\frac{\beta_{h1}^\sigma I_{v1}^j}{1+\alpha_1^\sigma I_{v1}^j} [S_h^{j+1} + (1-\phi_1^\sigma) V_h^{j+1}] \right) - \sum_{k=0}^{j-1} (E_{h1}^{k+1} - E_{h1}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) \left((\mu_h^\sigma + \xi_{e1}^\sigma) + \frac{\beta_{h2}^\sigma I_{v2}^j}{1+\alpha_2^\sigma I_{v2}^j} \right)}, \\ E_{h2}^{j+1} &= \frac{p^{1-\sigma} E_{h2}^j + \Gamma(2-\sigma) \Psi(p) \left(\frac{\beta_{h2}^\sigma I_{v2}^j}{1+\alpha_2^\sigma I_{v2}^j} [S_h^{j+1} + (1-\phi_2^\sigma) V_h^{j+1}] \right) - \sum_{k=0}^{j-1} (E_{h2}^{k+1} - E_{h2}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) \left((\mu_h^\sigma + \xi_{e2}^\sigma) + \frac{\beta_{h1}^\sigma I_{v1}^j}{1+\alpha_1^\sigma I_{v1}^j} \right)}, \\ E_{h12}^{j+1} &= \frac{p^{1-\sigma} E_{h12}^j + \Gamma(2-\sigma) \Psi(p) \left(\frac{\beta_{h2}^\sigma I_{v2}^j}{1+\alpha_2^\sigma I_{v2}^j} E_{h1}^{j+1} + \frac{\beta_{h1}^\sigma I_{v1}^j}{1+\alpha_1^\sigma I_{v1}^j} E_{h2}^{j+1} \right) - \sum_{k=0}^{j-1} (E_{h12}^{k+1} - E_{h12}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) \left((\mu_h^\sigma + \xi_{e12}^\sigma) \right)}, \\ A_{h1}^{j+1} &= \frac{p^{1-\sigma} A_{h1}^j + \Gamma(2-\sigma) \Psi(p) (\xi_{e1}^\sigma E_{h1}^{j+1}) - \sum_{k=0}^{j-1} (A_{h1}^{k+1} - A_{h1}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) \left((\mu_h^\sigma + \xi_{a1}^\sigma + \zeta_{a1}^\sigma + \theta_{a1}^\sigma) + \frac{\beta_{h2}^\sigma I_{v2}^j}{1+\alpha_2^\sigma I_{v2}^j} \right)}, \\ A_{h2}^{j+1} &= \frac{p^{1-\sigma} A_{h2}^j + \Gamma(2-\sigma) \Psi(p) (\xi_{e2}^\sigma E_{h2}^{j+1}) - \sum_{k=0}^{j-1} (A_{h2}^{k+1} - A_{h2}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) \left((\mu_h^\sigma + \xi_{a2}^\sigma + \zeta_{a2}^\sigma + \theta_{a2}^\sigma) + \frac{\beta_{h1}^\sigma I_{v1}^j}{1+\alpha_1^\sigma I_{v1}^j} \right)}, \\ A_{h12}^{j+1} &= \frac{p^{1-\sigma} A_{h12}^j + \Gamma(2-\sigma) \Psi(p) (\xi_{e12}^\sigma E_{h12}^{j+1} + \frac{\beta_{h2}^\sigma I_{v2}^j}{1+\alpha_2^\sigma I_{v2}^j} A_{h1}^{j+1} + \frac{\beta_{h1}^\sigma I_{v1}^j}{1+\alpha_1^\sigma I_{v1}^j} A_{h2}^{j+1}) - \sum_{k=0}^{j-1} (A_{h12}^{k+1} - A_{h12}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) \left((\mu_h^\sigma + \xi_{a12}^\sigma + \zeta_{a12}^\sigma + \theta_{a12}^\sigma) \right)}, \\ I_{h1}^{j+1} &= \frac{p^{1-\sigma} I_{h1}^j + \Gamma(2-\sigma) \Psi(p) (\xi_{a1}^\sigma A_{h1}^{j+1}) - \sum_{k=0}^{j-1} (I_{h1}^{k+1} - I_{h1}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) \left((\mu_h^\sigma + \zeta_{i1}^\sigma + \theta_{i1}^\sigma + \eta_{i1}^\sigma) + \frac{\beta_{h2}^\sigma I_{v2}^j}{1+\alpha_2^\sigma I_{v2}^j} \right)}, \\ I_{h2}^{j+1} &= \frac{p^{1-\sigma} I_{h2}^j + \Gamma(2-\sigma) \Psi(p) (\xi_{a2}^\sigma A_{h2}^{j+1}) - \sum_{k=0}^{j-1} (I_{h2}^{k+1} - I_{h2}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) \left((\mu_h^\sigma + \zeta_{i2}^\sigma + \theta_{i2}^\sigma + \eta_{i2}^\sigma) + \frac{\beta_{h1}^\sigma I_{v1}^j}{1+\alpha_1^\sigma I_{v1}^j} \right)}, \\ I_{h12}^{j+1} &= \frac{p^{1-\sigma} I_{h12}^j + \Gamma(2-\sigma) \Psi(p) (\xi_{a12}^\sigma A_{h12}^{j+1} + \frac{\beta_{h2}^\sigma I_{v2}^j}{1+\alpha_2^\sigma I_{v2}^j} I_{h1}^{j+1} + \frac{\beta_{h1}^\sigma I_{v1}^j}{1+\alpha_1^\sigma I_{v1}^j} I_{h2}^{j+1}) - \sum_{k=0}^{j-1} (I_{h12}^{k+1} - I_{h12}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) \left((\mu_h^\sigma + \zeta_{i12}^\sigma + \theta_{i12}^\sigma + \eta_{i12}^\sigma) \right)}, \\ Q_{h1}^{j+1} &= \frac{p^{1-\sigma} Q_{h1}^j + \Gamma(2-\sigma) \Psi(p) (\eta_{i1}^\sigma I_{h1}^{j+1}) - \sum_{k=0}^{j-1} (Q_{h1}^{k+1} - Q_{h1}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) \left((\mu_h^\sigma + \zeta_{h1}^\sigma + \theta_{h1}^\sigma) \right)}. \end{aligned} \quad (34)$$

$$\begin{aligned}
Q_{h2}^{j+1} &= \frac{p^{1-\sigma} Q_{h2}^j + \Gamma(2-\sigma) \Psi(p) (\eta_{i2}^\sigma I_{h2}^{j+1}) - \sum_{k=0}^{j-1} (Q_{h2}^{k+1} - Q_{h2}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) ((\mu_h^\sigma + \zeta_{h2}^\sigma + \theta_{h2}^\sigma))}, \\
Q_{h12}^{j+1} &= \frac{p^{1-\sigma} Q_{h12}^j + \Gamma(2-\sigma) \Psi(p) (\eta_{i12}^\sigma I_{h12}^{j+1}) - \sum_{k=0}^{j-1} (Q_{h12}^{k+1} - Q_{h12}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) (\mu_h^\sigma + \zeta_{h12}^\sigma + \theta_{h12}^\sigma)}, \\
R_{h1}^{j+1} &= \frac{p^{1-\sigma} R_{h1}^j + \Gamma(2-\sigma) \Psi(p) (\zeta_{a1}^\sigma A_{h1}^{j+1} + \zeta_{i1}^\sigma I_{h1}^{j+1} + \zeta_{h1}^\sigma Q_{h1}^{j+1}) - \sum_{k=0}^{j-1} (R_{h1}^{k+1} - R_{h1}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) (\mu_h^\sigma + \delta_{h1}^\sigma)}, \\
R_{h2}^{j+1} &= \frac{p^{1-\sigma} R_{h2}^j + \Gamma(2-\sigma) \Psi(p) (\zeta_{a2}^\sigma A_{h2}^{j+1} + \zeta_{i2}^\sigma I_{h2}^{j+1} + \zeta_{h2}^\sigma Q_{h2}^{j+1}) - \sum_{k=0}^{j-1} (R_{h2}^{k+1} - R_{h2}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) (\mu_h^\sigma + \delta_{h2}^\sigma)}, \\
R_{h12}^{j+1} &= \frac{p^{1-\sigma} R_{h12}^j + \Gamma(2-\sigma) \Psi(p) (\zeta_{a12}^\sigma A_{h12}^{j+1} + \zeta_{i12}^\sigma I_{h12}^{j+1} + \zeta_{h12}^\sigma Q_{h12}^{j+1}) - \sum_{k=0}^{j-1} (R_{h12}^{k+1} - R_{h12}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) (\mu_h^\sigma + \delta_{h12}^\sigma)}, \\
S_v^{j+1} &= \frac{p^{1-\sigma} S_v^j + \Gamma(2-\sigma) \Psi(p) (\Lambda_v^\sigma) - \sum_{k=0}^{j-1} (S_v^{k+1} - S_v^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) \left(\frac{\beta_{v1}^\sigma (A_{h1}^{j+1} + I_{h1}^{j+1} + A_{h12}^{j+1} + I_{h12}^{j+1})}{1 + \gamma_1^\sigma A_{h1}^{j+1} + \gamma_2^\sigma I_{h1}^{j+1} + \gamma_3^\sigma A_{h12}^{j+1} + \gamma_4^\sigma I_{h12}^{j+1}} + \frac{\beta_{v2}^\sigma (A_{h2}^{j+1} + I_{h2}^{j+1} + A_{h12}^{j+1} + I_{h12}^{j+1})}{1 + \gamma_1^\sigma A_{h2}^{j+1} + \gamma_2^\sigma I_{h2}^{j+1} + \gamma_3^\sigma A_{h12}^{j+1} + \gamma_4^\sigma I_{h12}^{j+1}} + \mu_v^\sigma \right)}, \\
E_{v1}^{j+1} &= \frac{p^{1-\sigma} E_{v1}^j + \Gamma(2-\sigma) \Psi(p) \left(\frac{\beta_{v1}^\sigma (A_{h1}^{j+1} + I_{h1}^{j+1} + A_{h12}^{j+1} + I_{h12}^{j+1})}{1 + \gamma_1^\sigma A_{h1}^{j+1} + \gamma_2^\sigma I_{h1}^{j+1} + \gamma_3^\sigma A_{h12}^{j+1} + \gamma_4^\sigma I_{h12}^{j+1}} S_v^{j+1} \right) - \sum_{k=0}^{j-1} (E_{v1}^{k+1} - E_{v1}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) (\mu_v^\sigma + \omega_{v1}^\sigma)}, \\
E_{v2}^{j+1} &= \frac{p^{1-\sigma} E_{v2}^j + \Gamma(2-\sigma) \Psi(p) \left(\frac{\beta_{v2}^\sigma (A_{h2}^{j+1} + I_{h2}^{j+1} + A_{h12}^{j+1} + I_{h12}^{j+1})}{1 + \gamma_1^\sigma A_{h2}^{j+1} + \gamma_2^\sigma I_{h2}^{j+1} + \gamma_3^\sigma A_{h12}^{j+1} + \gamma_4^\sigma I_{h12}^{j+1}} S_v^{j+1} \right) - \sum_{k=0}^{j-1} (E_{v2}^{k+1} - E_{v2}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) (\mu_v^\sigma + \omega_{v2}^\sigma)}, \\
I_{v1}^{j+1} &= \frac{p^{1-\sigma} I_{v1}^j + \Gamma(2-\sigma) \Psi(p) (\omega_{v1}^\sigma E_{v1}^{j+1}) - \sum_{k=0}^{j-1} (I_{v1}^{k+1} - I_{v1}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) (\mu_v^\sigma + \rho_{v1}^\sigma)}, \\
I_{v2}^{j+1} &= \frac{p^{1-\sigma} I_{v2}^j + \Gamma(2-\sigma) \Psi(p) (\omega_{v2}^\sigma E_{v2}^{j+1}) - \sum_{k=0}^{j-1} (I_{v2}^{k+1} - I_{v2}^k) A_{\sigma,j}^k}{p^{1-\sigma} + \Psi(p) \Gamma(2-\sigma) (\mu_v^\sigma + \rho_{v2}^\sigma)}.
\end{aligned}$$

(35)

Model fitting and numerical assessment

Demographic data related to Brazil have been used for the simulations. The initial conditions are set as: $S_h(0) = 3,600,000$, $V_h(0) = 400,000$, $E_{h1}(0) = 125$, $E_{h2}(0) = 1,26$, $E_{h12}(0) = 0$, $A_{h1}(0) = 500$, $A_{h2}(0) = 500$, $A_{h12}(0) = 0$, $I_{h1}(0) = 500$, $I_{h2}(0) = 500$, $I_{h12}(0) = 0$, $Q_{h1}(0) = 500$, $Q_{h2}(0) = 600$, $Q_{h12}(0) = 0$, $R_{h1}(0) = 500$, $R_{h2}(0) = 500$, $R_{h12}(0) = 0$, $S_v(0) = 48,000$, $E_{v1}(0) = 600$, $E_{v2}(0) = 600$, $I_{v1}(0) = 1000$, $I_{v2}(0) = 1000$. For the fitting of model to data available records for reported Dengue cases in Espirito Santo Brazil for 36 weeks⁴⁵, the fractional model is fitted to real data. The fitting, which is shown in Fig. 2 reveals that our model behaves very well to data when the order of the Caputo derivative is taken as: $\sigma = 0.975$.

Sensitivity analysis of reproduction numbers

Sensitivity analysis is carried out in this section to analyse the influence of the different parameters involved in the reproduction numbers of model 4. We employed the PRCC techniques separately for both reproduction number to show the role of the parameters in the reproduction number. It can be observed from Fig. 3a,b, that transmission rates for human as well as vectors, vaccination rates and vaccine efficacy are very sensitive to the reproduction number. To be more specific transmission rates are positively correlated with the reproduction numbers. It is observed in the Fig. 4a–h that, with the increment in the transmission rates from vectors to human, the reproduction number is also increased. Vaccine efficacy is negatively correlated with respect to the reproduction number. In the Fig. 4b,f, it can be seen easily that increment in the vaccine efficacy lower the reproduction number which means we can control the disease by introducing the vaccines that have stronger efficacy. The Fig. 4c,g describe the behaviour of reproduction numbers for both of the disease depending upon the transmission rate and removal of vectors. It is shown that we can also control the reproduction number and hence the disease by removing the more vectors from the environment. On the similar fashion, the Fig. 4a,e describes the dependence of reproduction numbers upon the transmission rate and progression rates. It can be seen that progression rate is positively correlated with respect to the reproduction number. Similarly, the other parameters involved in reproduction number like progression rates, recovery rates and quarantine measures have also great impact on reproduction numbers. We also presented the pie charts for both of the reproduction numbers in Fig. 3c,d that gives the percentage influence of all the parameters.

Numerical assessment

To obtain the correct long-time behaviour of the model with NSFD, some denominator functions available in the literature were explored. The impact of these denominator functions for all the compartments of the model is shown in the Fig. 5a–f. The different denominator functions are $\Phi_1 = h^\sigma$, $\Phi_2 = \frac{h^{1-\sigma} (1 - E_\sigma(-(\mu_h^\sigma p)^\sigma))}{(E_\sigma(-(\mu_h^\sigma p)^\sigma)) \Gamma(2-\sigma) \mu_h^\sigma}$,

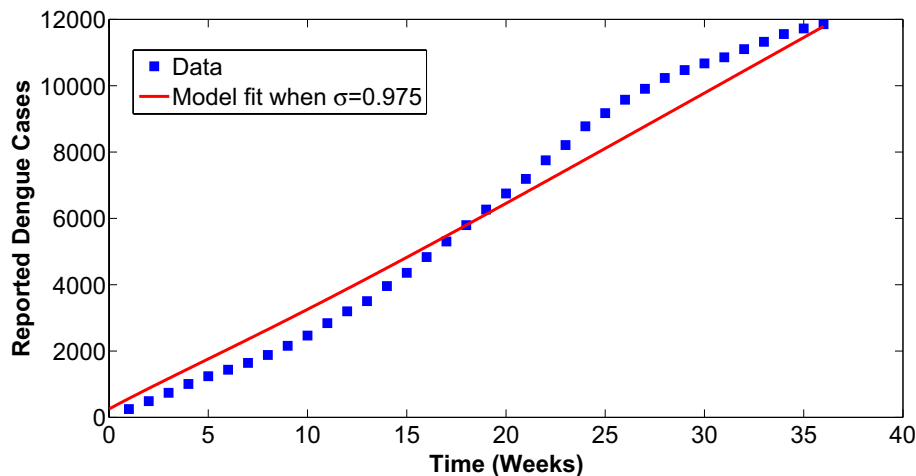


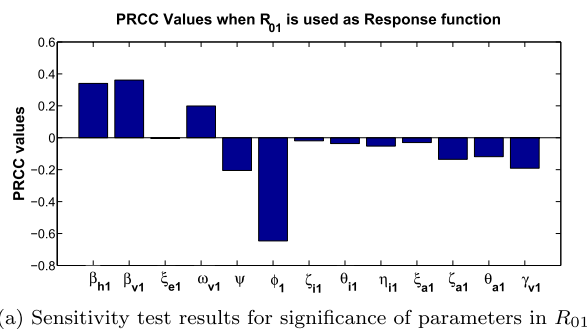
Figure 2. Fitting the model to data.

$\Phi = \frac{e^{\mu_h^\sigma} - 1}{\mu_h^\sigma}$ are used for the simulations. The reproduction number is the quantity that plays an important role for the disease to die out or spread. Different scenarios for the reproduction number of both diseases are listed below and for these scenarios different simulations are carried out shown in the Fig. 8a–d. These simulations are showing that when reproduction number is greater than 1 the disease is spreading and when reproduction number is less than 1 the disease eventually die out.

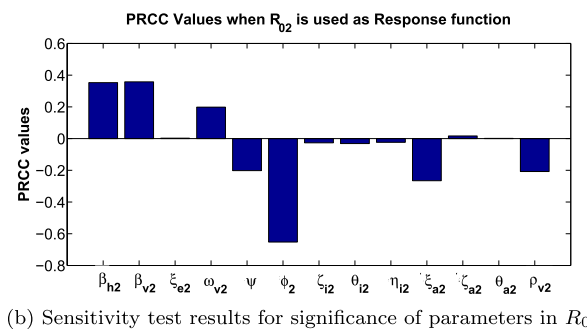
Different scenarios are considered: Scenario-1: $\mathcal{R}_{01} \geq 1, \mathcal{R}_{02} \geq 1$, Scenario-2: $\mathcal{R}_{01} \leq 1, \mathcal{R}_{02} \geq 1$, Scenario-3: $\mathcal{R}_{01} \geq 1, \mathcal{R}_{02} \leq 1$ and Scenario-4: $\mathcal{R}_{01} \leq 1, \mathcal{R}_{02} \leq 1$.

In Fig. 9a–d, the epidemiological affect of quarantine measure is assessed. It can be observed that the quarantine measure has great impact in averting new dengue strains infections, Specifically, maximum number of cases averted is recorded when quarantine rates are as much as $\eta_{i1} = 0.05, \eta_{i1} = 0.10$ and $\eta_{i1} = 0.20$.

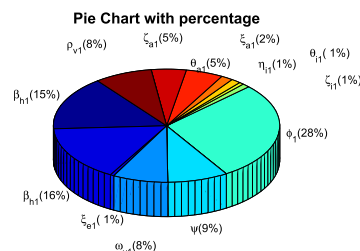
In Fig. 10a–c, simulations of the infected compartments are presented when vaccine parameters ψ and ϕ_1 are varied. It is interesting that maximum number in the dengue strain-1 infection averted is recorded when $\psi = 0.20$ and $\phi_1 = 0.85$. Similar conclusion can be reached for strain-2 for maximum number dengue strain-2 infection averted and this accounts when $\psi = 0.20$ and $\phi_2 = 0.85$ in Fig. 10d–f. Hence to keep the co-circulation of both dengue strain as low as possible vaccination rate must be stepped up to 0.20 per day while keeping effectiveness of vaccine against strain-1 and strain-2 infections at 0.85.



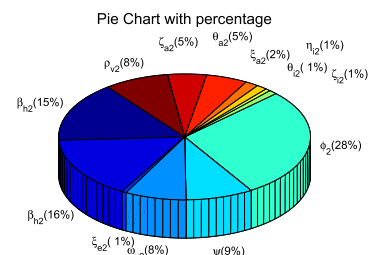
(a) Sensitivity test results for significance of parameters in R_{01}



(b) Sensitivity test results for significance of parameters in R_{02}



(c) Impact of parameters on R_{01} through Pie chart



(d) Impact of parameters on R_{02} through Pie chart

Figure 3. Illustration of the influence of parameters on reproduction number through PRCC and Pie Chart.

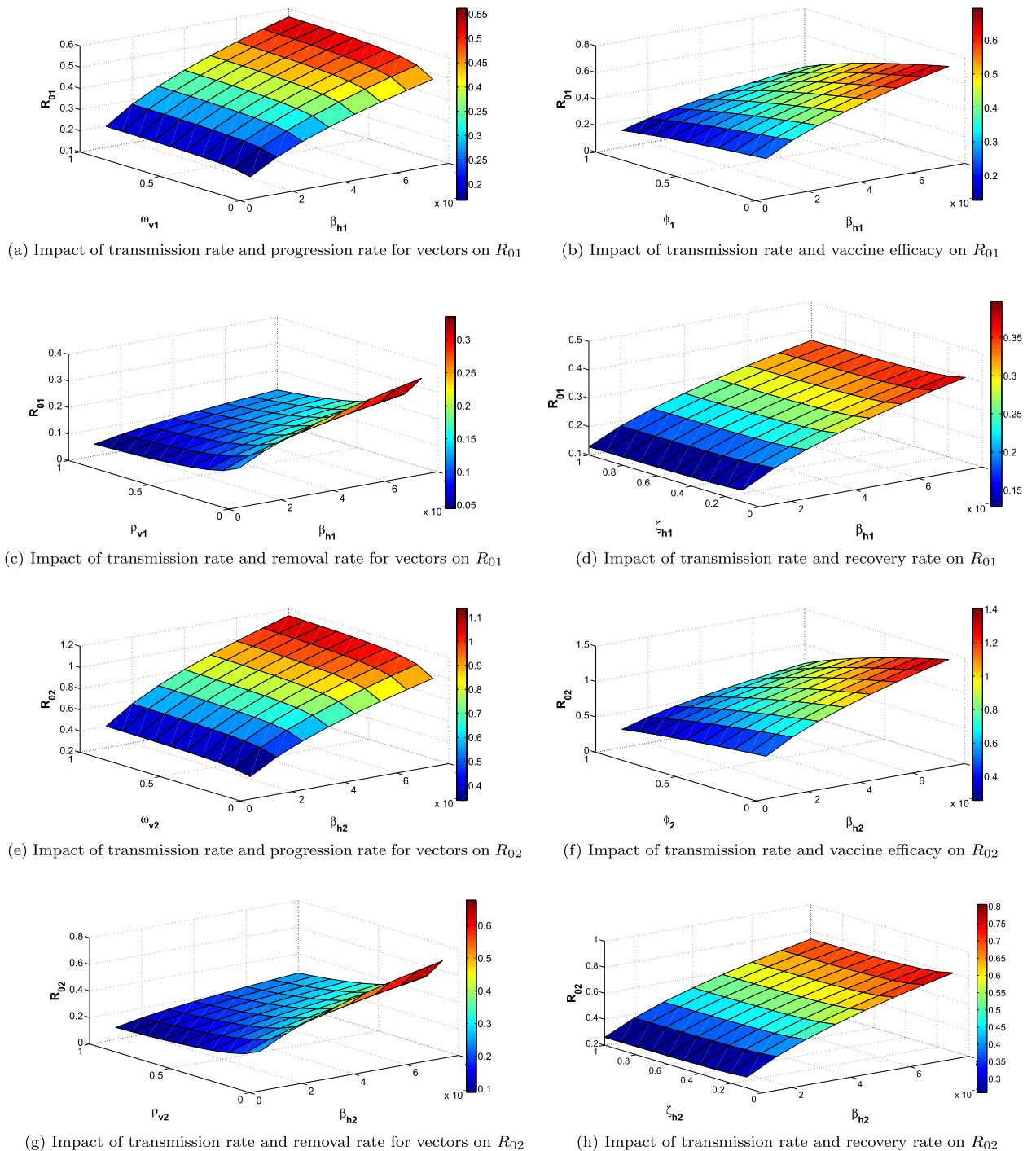


Figure 4. Surface plots to show the impact of different parameters involved in reproduction numbers.

The phase portraits of the exposed, Asymptomatic infected and Symptomatic infected at different initial conditions and for different cases of reproduction numbers are presented in Fig. 11a–f, respectively. In Fig. 11a–c it can be observed that the solution paths for all the infected classes (Exposed, Asymptomatic and Symptomatic infected) tend towards the infection free equilibrium when reproduction number is less than 1 irrespective of the initial conditions and order of the derivative. Similarly it can also be observed in Fig. 11d–f that the solution paths for all the infected classes tend towards the endemic equilibrium when reproduction number is greater than 1 irrespective of the initial conditions and order of the derivative.

Conclusion

In this paper, a comprehensive mathematical model is proposed for two strains of dengue virus with saturated incidence rates and quarantine measures. Imperfect dengue vaccination is also assumed in the model. Existence, uniqueness and stability of the new model are established using some results from fixed point, measure theory

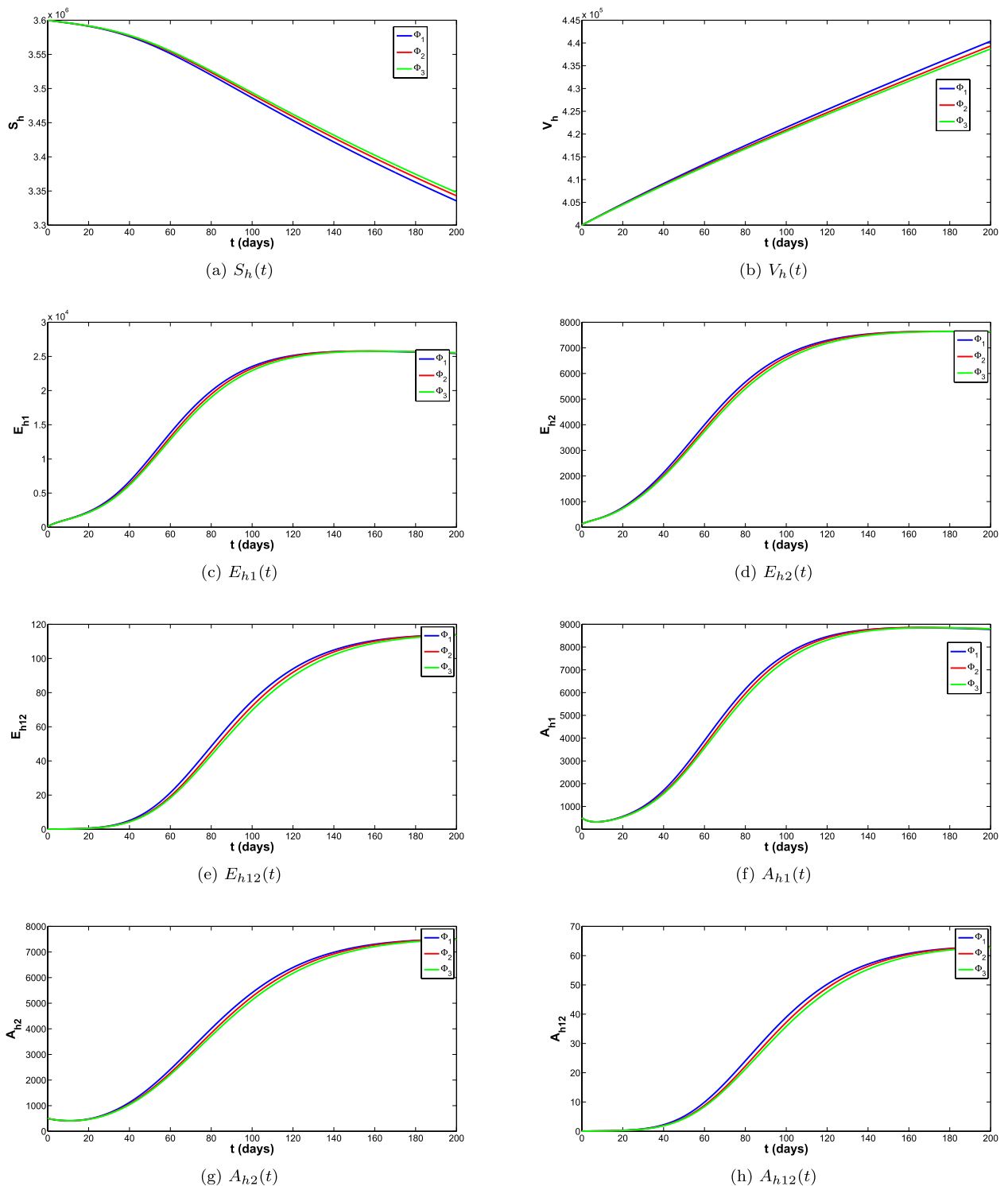


Figure 5. Comparison of all classes for different denominator functions.

and degree theory. Additionally, well constructed Lyapunov function candidates are also applied to prove the global stability of infection-free and endemic equilibria. It is also demonstrated that the model system is generalized Ulam–Hyers stable under certain appropriate conditions. The model is fitted to the real data for dengue epidemic for the city of Espirito Santo in Brazil. For the approximate solution of the model, a non-standard finite difference(NSFD) approach is applied. The behaviour of the NSFD is also assessed under different denominator functions and it is observed that the choice of the denominator function could influence the solution trajectories. Different scenario analysis are also assessed when the reproduction number is below or above one. Furthermore, simulations are also presented to assess the epidemiological impact of dengue vaccination and quarantine measures for infected individuals. Some of the major highlights of the qualitative analysis are as follows:

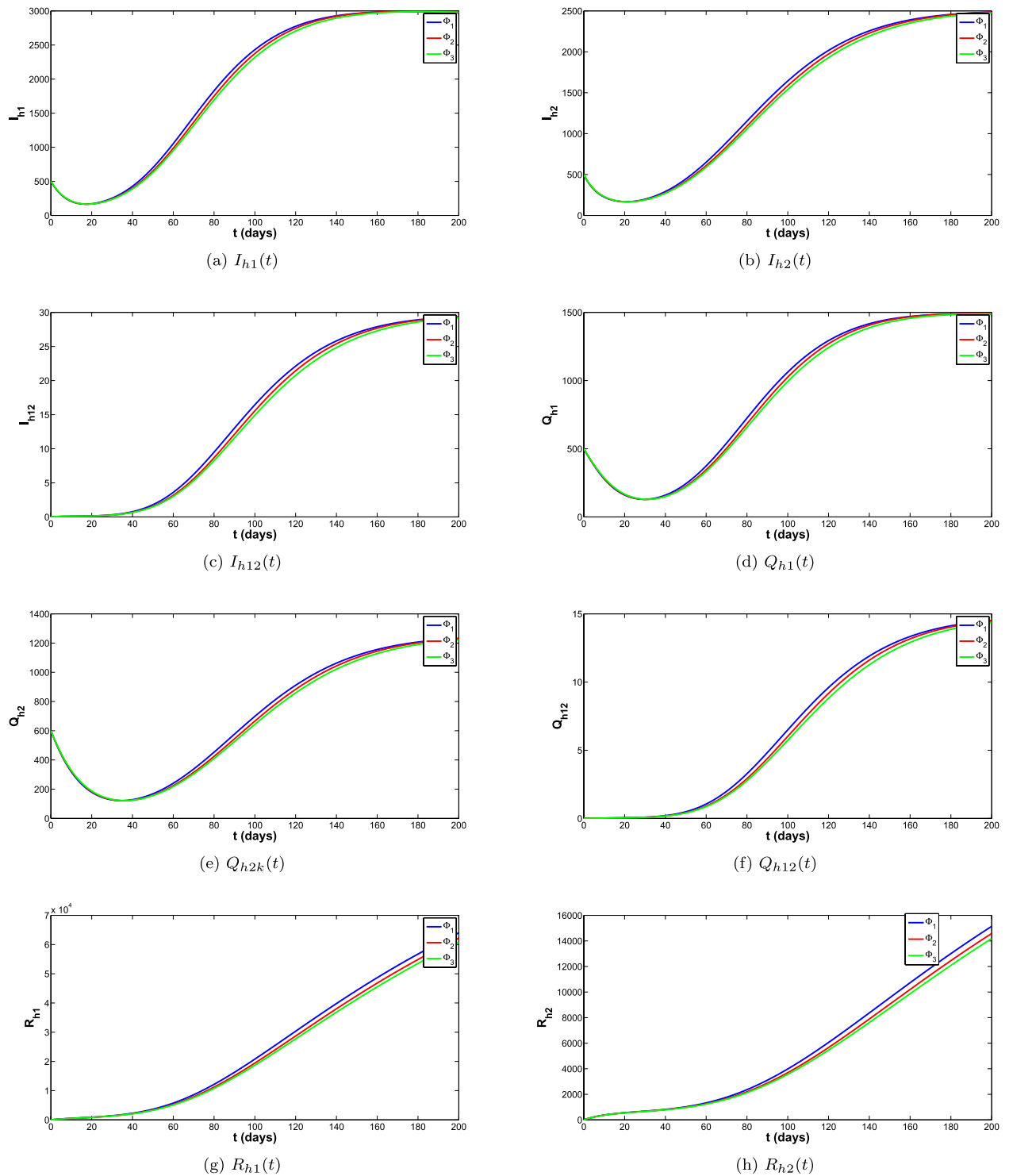


Figure 6. Comparison of all classes for different denominator functions.

- (i) The strain 1 and strain 2 sub-models are qualitatively analyzed, investigating the stability in the sense of Lyapunov which are presented in “[Analysis of the sub-models](#)”.
- (ii) The full model’s infection-free equilibrium is proved to be locally stable, as presented in Theorem 4.2.
- (iii) Existence, uniqueness and stability of the complete model are presented in “[Existence, uniqueness and Ulam–Hyers stability of the complete model](#)” with the help of results from fixed point theory and degree theory.

The major highlights of the numerical analysis which are carried out using the non-standard finite difference scheme and are given in “[Nonstandard finite difference scheme](#)” are presented below:

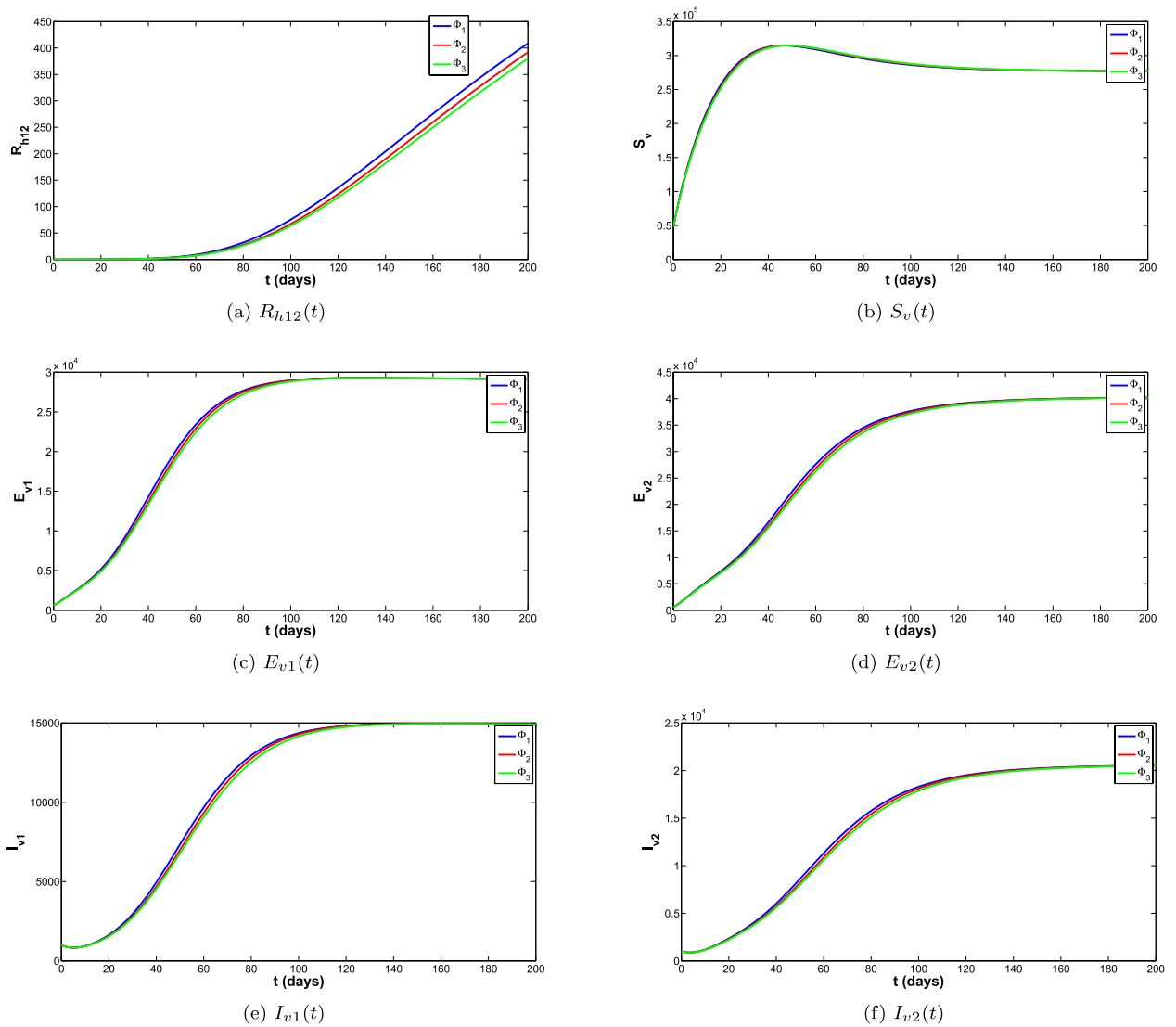


Figure 7. Comparison of all classes for different denominator functions.

- (i) The model is fitted to the real data for the city of Espirito Santo in Brazil.
- (ii) The choice of the denominator function influences the behaviour of the solution under consideration.
- (iii) Sensitivity analysis of the reproduction number for both strains are carried out to show the sensitive parameters and the result is shown with the help of pie chart,
- (iv) The solution profiles when the reproduction numbers of both strains are either below or greater than one as well as when one reproduction number dominates the other, are also investigated.
- (v) Different scenario analyses to investigate the epidemiological impact of dengue vaccination and quarantine for infected individuals shows that these two measures could greatly reduce the co-spread of both strains within a population.

The research in this paper can be extended in the following ways: One could consider stochastic equivalence as well as fractal fractional form of the current model for a possible research problem. Approximate solution of the model using some other novel numerical schemes that can yield the better results can also be considered. Moreover, one could also establish the existence, uniqueness and stability results using some novel fixed point theorems

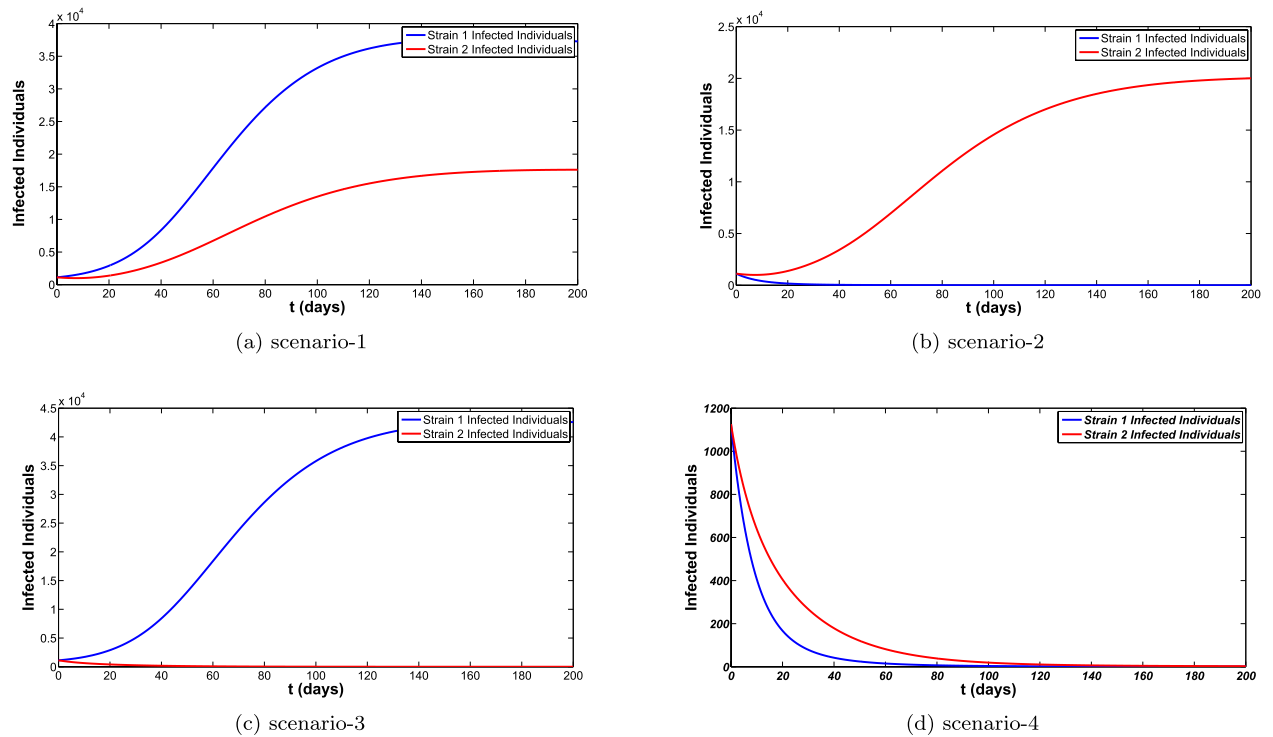


Figure 8. Comparison of infected individuals for different scenarios of reproduction number.

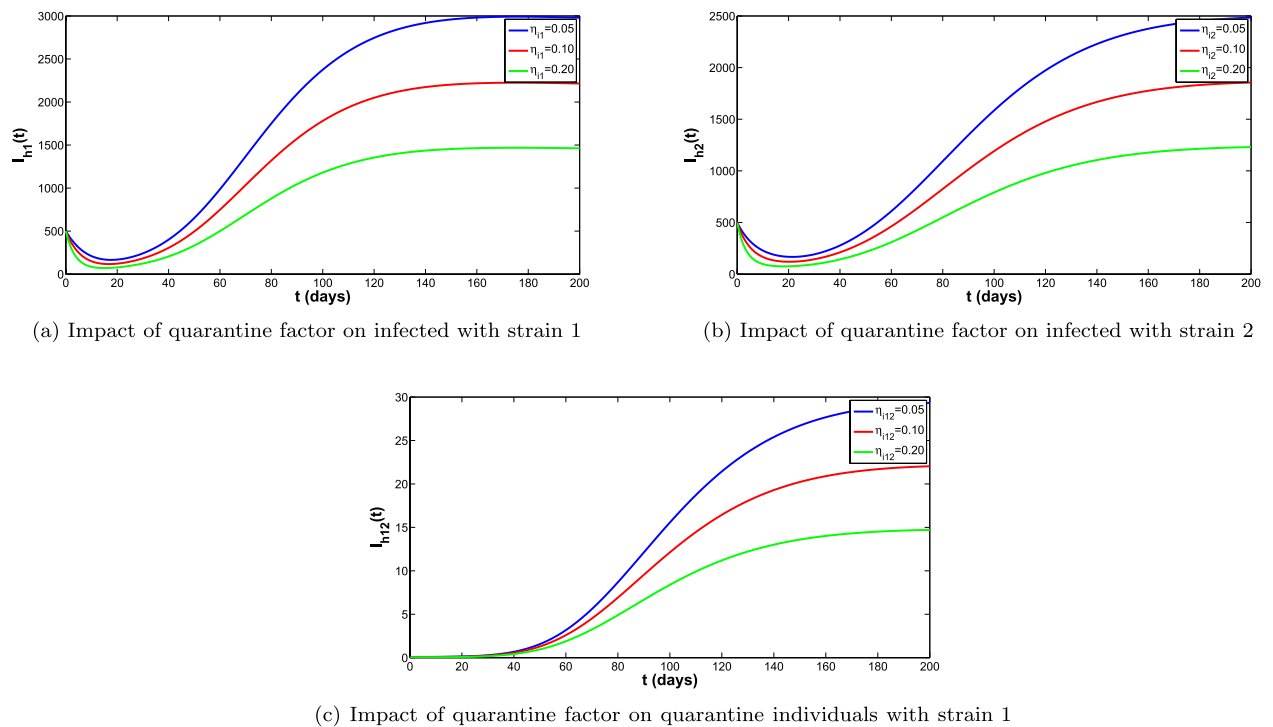
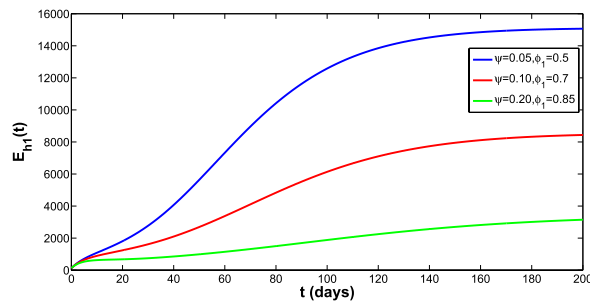
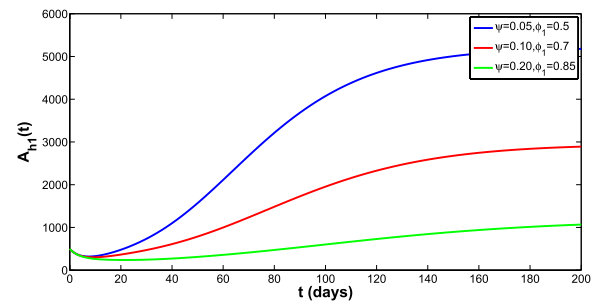


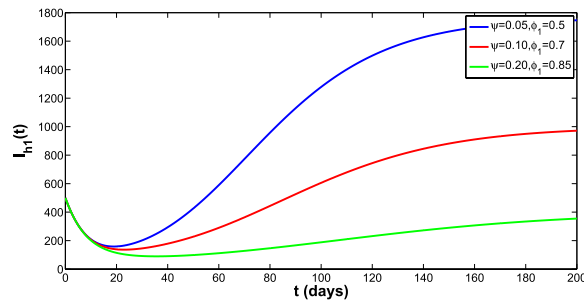
Figure 9. Impact of quarantine measures on infected and quarantine individuals.



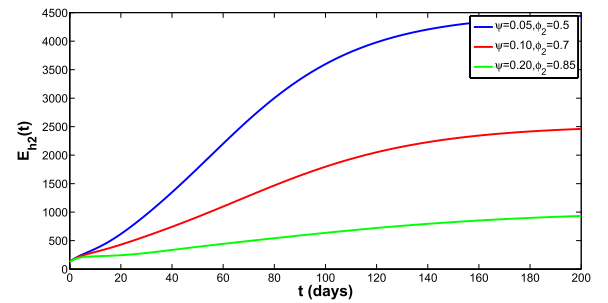
(a) Impact of Vaccination on exposed for strain 1



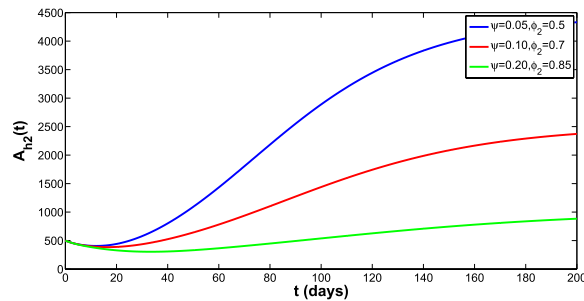
(b) Impact of Vaccination on asymptomatic with strain 1



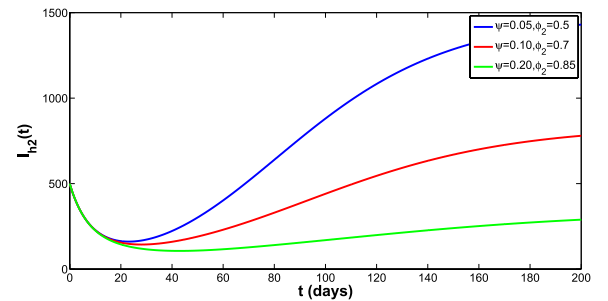
(c) Impact of Vaccination on symptomatic with strain 1



(d) Impact of Vaccination on exposed for strain 2



(e) Impact of Vaccination on asymptomatic with strain 2



(f) Impact of Vaccination on symptomatic with strain 2

Figure 10. Impact of vaccination and vaccine efficacy on infected compartments.

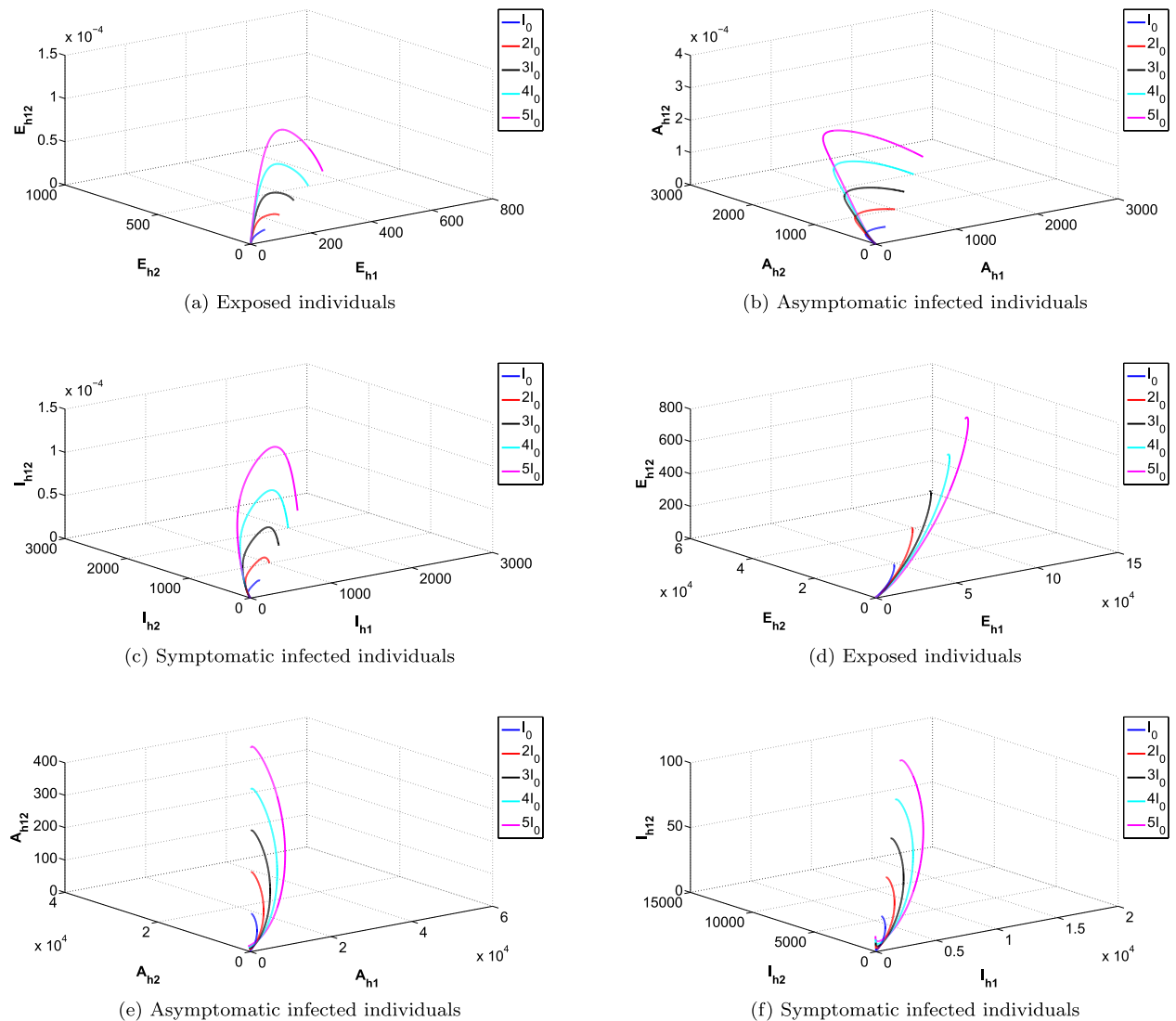


Figure 11. Simulations of infected classes separately with co-infection at different initial conditions.

Data availability

The datasets used and/or analysed during the current study available from the corresponding author on reasonable request.

Received: 22 February 2024; Accepted: 21 May 2024

Published online: 25 May 2024

References

- Dengue and severe dengue. <https://www.who.int/news-room/fact-sheets/detail/dengue-and-severe-dengue> (accessed Nov. 02, 2022).
- CDC, Dengue transmission—CDC, Centers for Disease Control and Prevention, Sep. 26, 2019. <https://www.cdc.gov/dengue/transmission/index.html> (accessed Nov. 02, 2022).
- Masyeni, S., Yohan, B. & Sasmono, R. T. Concurrent infections of dengue virus serotypes in Bali, Indonesia. *BMC. Res. Notes* **12**(1), 129. <https://doi.org/10.1186/s13104-019-4164-9> (2019).
- Guzman, M. G. & Harris, E. Dengue. *Lancet*. **385**(9966), 453–465. [https://doi.org/10.1016/S0140-6736\(814\)960572-9](https://doi.org/10.1016/S0140-6736(814)960572-9) (2015)
- Vannice, K. S., Durbin, A. & Hombach, J. Status of vaccine research and development of vaccines for dengue. *Vaccine*. **34**, 2934–2938 (2016).
- Immunization, Vaccines and Biologicals. World Health Organization. <https://www.who.int/news-room/questions-and-answers/item/dengue-vaccines>. Accessed: December 2016.
- Ali, A., Khan, S. U., Ali, I. & Khan, F. U. On dynamics of stochastic avian influenza model with asymptomatic carrier using spectral method. *Math. Methods Appl. Sci.* **45**(13), 8230–8246. <https://doi.org/10.1002/mma.8183> (2022).
- Rihan, F. A., & Alsakaji, H. J. Dynamics of a stochastic delay differential model for COVID-19 infection with asymptomatic infected and interacting people: Case study in the UAE. *Res. Phys.* 104658 (2021)
- Rihan, F. A., Alsakaji, H. J. & Rajivganthi, C. Stochastic SIRC epidemic model with time-delay for COVID-19. *Adv. Differ Equ.* **2020**, 502. <https://doi.org/10.1186/s13662-020-02964-8> (2020).

10. Oname, A. & Abbas, M. Modeling SARS-CoV-2 and HBV co-dynamics with optimal control. *Phys. A* **615**, 128607. <https://doi.org/10.1016/j.physa.2023.128607> (2023).
11. Hezam, I. M., Foul, A. & Alrasheedi, A. A dynamic optimal control model for COVID-19 and cholera co-infection in Yemen. *Adv. Differ. Equ.* **2021**, 108. <https://doi.org/10.1186/s13662-021-03271-6> (2021).
12. Saha, S. & Samanta, G. Analysis of a host-vector dynamics of a dengue disease model with optimal vector control strategy. *Math. Comput. Simul.* **195**, 31–55 (2022).
13. Saha, S., Das, M. & Samanta, G. Analysis of an SIRS Model in Two-Patch Environment in Presence of Optimal Dispersal Strategy. *Axioms* **13**, 94. <https://doi.org/10.3390/axioms13020094> (2024).
14. Ferguson, N. M., Rodriguez-Barraquer, I., Dorigatti, I., Mier-y-Teran-Romero, L., Laydon, D. J., & Cummings, A. T. Benefits and risks of the Sanofi-Pasteur dengue vaccine: Modeling optimal deployment. *Science* **353**, 6303 (2016).
15. Hladish, T. J., Pearson, C. A. B., Chao, D. L., Rojas, D. P., Recchi, G. L., Gomez-Dantes, H., Elizabeth Halloran, M., Pulliam, J. R. C., & Longini, I. M. Projected impact of dengue vaccination in Yucatan, Mexico. *PLOS Negl. Trop. Dis.* **10**(5), e0004661 (2016).
16. Coudeville, L. & Garnett, G. P. Transmission dynamics of the four dengue strains in southern Vietnam and the potential impact of vaccination. *PLoS ONE* **7**(12), e51244 (2012).
17. Rodriguez-Barraquer, I., Mier-y-Teran-Romero, L., Schwartz, I. B., Burke, D. S. & Cummings, A. T. Potential opportunities and perils of imperfect dengue vaccines. *Vaccine* **32**, 514–520 (2014).
18. Chao, D. L., Halstead, S. B., Halloran, M. E. & Longini, I. M. Jr. Controlling dengue with vaccines in Thailand. *PLoS Negl. Trop. Dis.* **6**, e1876 (2012).
19. Caputo, M. Linear models of dissipation whose Q is almost frequency independent. *Ann. Geophys.* **19**(4), 383–393 (1966).
20. Caputo, M. & Fabrizio, M. A new definition of fractional derivative without singular kernel. *Prog. Fract. Differ. Appl.* **1**(2), 1–3 (2015).
21. Atangana, A. & Baleanu, D. New fractional derivatives with nonlocal and non-singular kernel: Theory and applications to heat transfer model. *Therm. Sci.* **20**(2), 763–769 (2016).
22. Peter, O. J., Fahrani, N. D. & Chukwu, C. W. A fractional derivative modeling study for measles infection with double dose vaccination. *Healthcare Anal.* **4**, 100231 (2023).
23. Peter, O. J. Transmission dynamics of fractional order brucellosis model using caputo-fabrizio operator. *Int. J. Differ. Equ.* **1**, 1–11 (2020).
24. Peter, O. J. *et al.* Fractional order of pneumococcal pneumonia infection model with caputo fabrizio operator. *Res. Phys.* **29**, 104581 (2021).
25. Peter, O. J. *et al.* A mathematical model analysis of meningitis with treatment and vaccination in fractional derivatives. *Int. J. Appl. Comput. Math.* **8**(3), 117 (2022).
26. Das, M. & Samanta, G. Optimal control of a fractional order epidemic model with carriers. *Introd. J. Dyn. Control* **10**, 598–619. <https://doi.org/10.1007/s40435-021-00822-3> (2022).
27. Fatmawati, A. *et al.* Analysis of dengue model with fractal-fractional Caputo-Fabrizio operator. *Adv. Differ. Equ.* **2020**, 422. <https://doi.org/10.1186/s13662-020-02881-w> (2020).
28. Khan, R. A., Gul, S., Jarad, F. & Khan, H. Existence results for a general class of sequential hybrid fractional differential equations. *Adv. Differ. Equ.* **1**, 284 (2021).
29. Okuneye, K., Velasco, H. & Jorge, & Gumel A., The “unholy” Chikungunya-Dengue-Zika Trinity: A Theoretical Analysis. *J. Biol. Syst.* <https://doi.org/10.1142/S0218339017400046> (2018).
30. Garba, S. M., Gumel, A. B. & Abu Bakar, M. R. Backward bifurcations in dengue transmission dynamics. *Math. Biosci.* **215**(1), 11–25 (2008).
31. Oname, A., Rwezaura, H., Diagne, M. L., Inyama, S. C. & Tchuente, M. COVID-19 and dengue co-infection in Brazil: optimal control and cost-effectiveness analysis. *Eur. Phys. J. Plus.* **136**, 1090. <https://doi.org/10.1140/epjp/s13360-021-02030-6> (2021).
32. Zhang, J., Jia, J. & Song, X. Analysis of an SEIR epidemic model with saturated incidence and saturated treatment function. *Sci. World J.* **1**, 910421. <https://doi.org/10.1155/2014/910421> (2014).
33. Ojo, M., Gbadamosi, B., Olukeyode, A. & Oluwaseun, O. Sensitivity Analysis of Dengue Model with Saturated Incidence Rate. *Open Access Library J.* **5**, 1–17. <https://doi.org/10.4236/oalib.1104413> (2018).
34. Umdekar, S., Sharma, P. K. & Sharma, S. An SEIR model with modified saturated incidence rate and Holling type II treatment function. *Comput. Math. Biophys.* **11**(1), 20220146. <https://doi.org/10.1515/cmb-2022-0146> (2023).
35. https://www.indexmundi.com/brazil/demographics_profile.html. Accessed: Jan 1, 2022.
36. COVID-19 and dengue co-infection in Brazil: optimal control and cost-effectiveness analysis.
37. Okuneye, K., Velasco-Hernandez, J. X. & Gumel, A. B. The unholy chikungunya-dengue-zika trinity: A theoretical analysis. *J. Biol. Syst.* **25**, 545–585 (2017).
38. Garba, S. M. *Backward bifurcations in dengue transmission dynamics* (Elsevier, 2008).
39. Krasovskii, N. N. *Nekotorye zadachi teorii ustoychivosti dvizheniya* (Gosudarstv. Izdat. Fiz.-Mat. Lit, Moscow, 1959).
40. Lasalle, J.P. Some extensions of Liapunov's second method. *IRE Trans. CT-7*, pp. 520–527 (1960).
41. van den Driessche, P. & Watmough, J. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Math. Biosci.* **180**(1), 29–48 (2002).
42. Ulam, S. M. Vol. 29 (New York, 1960).
43. Ulam, S. M. Problem in modern mathematics (Courier Co., 2004).
44. Hamou, M., Ehrhardt, M., & Tabharit, L. A nonstandard finite difference scheme for a time-fractional model of zika virus transmission (2022).
45. <https://www.citypopulation.de/en/brazil/cities/espíritossanto/>. Accessed: Aug 1, 2023.

Acknowledgements

The author: S.H. Khan acknowledges the support from North Carolina A & T State University, Greensboro, USA for the Start-up grant used to publish this paper. All the authors are grateful to the handling editor and anonymous reviewers whose constructive criticisms and queries have greatly helped to improve the quality of the manuscript's presentation.

Author contributions

All authors contributed equally to bring the manuscript into the current shape.

Funding

The author: S H Khan acknowledges the funding from North Carolina A & T State University, Greensboro, USA for the Start-up grant used to publish this paper.

Competing interests

The authors declare no competing interests.

Additional information

Correspondence and requests for materials should be addressed to S.H.K.

Reprints and permissions information is available at www.nature.com/reprints.

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.



Open Access This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by/4.0/>.

© The Author(s) 2024